Hand-washing and its impact on child health in Kathmandu, Nepal

LANGFORD, REBECCA,MARY

Hand-washing and its impact on child health in Kathmandu, Nepal

Rebecca Langford

Department of Anthropology

October 2009

Thesis submitted to Durham University for the degree of Doctor of Philosophy

For Apsana

This thesis is dedicated to my friend, Apsana Giri, who was an unending source of support and inspiration to me during this project. She was killed in a car accident on January 2nd 2008.

Declaration of Copyright

I confirm that no part of the material presented in this thesis has been previously submitted by me or any other person for a degree in this or any other university. In all cases, where it is relevant, material from the work of others has been acknowledged.

Signed:

Rebecca Langford

Date:

Acknowledgements

My first thanks must, of course, go to all the mothers and children of Shanti Nagar, Palpakot, Sinamangal, Jhagriti Tol, Khadiparka, Pathibhara, Ramhiti and Sankhamul who participated in this study. They were incredibly welcoming to this strange *bideshi* who invaded their communities every few weeks. I thank them for their laughter, hospitality and generosity.

My thanks too, to my hard-working team of field assistants. Apsana Giri was my confidante, inspiration and friend: none of this would have been possible without her unending cheeriness and optimism. The help of Laxmi Bhadra was also essential and I thank her for her incredible efficiency in getting things done! Neeru Maharjan admirably took over Apsana's translating duties when she left the country and was a real pleasure to work with.

A big thanks to the Community Motivators - Subba Maya Pariyar, Bhanumati Das, Subhadra Pokhrel, Sita Parajuli and Krishna Kumari Subba – and my fieldworkers - Sunita Mahat, Saraswoti Pokhrel, Laxmi Lama, Paru Chimoriya, Samjhana Sunuwar, Yamuna Adhikari, Rojina Tamang, Divya Joshi, Mingma Thapa, Jyoti Magar, Pabitra Dahal, Kamala Lama and Nirjala Khadga. Thanks also to the numerous other people who helped out in various ways along the way – Tulsi Dawadi, Makhan Maharjan, Ram Chhetri, Kamala Sigdel, Prakash, Krishna Shah Yatri, JB Ruwali, Juna and Pinky Chimoriya, Ajay Das, Ranju Buddhathoki, Ganga Karki and Kamal Basnet. And of course to the many friends I made in Nepal this time, especially Katie Kilcline, Sean Hanlon, Caleen Kennedy, Brian Roedl, Alex Owen, Rajesh Maharjan and Kiran and Geri Subba.

My thanks to Dr Rachel Hawksworth and her colleagues from Anandaban for giving up their time to do the health camps at the end of the project. Thanks too, to Murdo MacDonald, Lesley Oakden, Annie Sanders, Katie Oven and Rob Dunford for helping me deliver all my samples back to the UK without too much mishap! I am also very grateful to my collaborator, Dr Peter Lunn, of Cambridge University for undertaking the analysis of these samples.

I will be forever grateful to Dr Sanja Stanojevic and Dr Clare Chandler for their advice and support on the statistical analyses – you were both incredibly generous and patient! Also thanks to Dr Rob Dunford for his help on using STATA.

Thanks are also due to my fellow post-grads – Mathilde Matthijsse, Caroline Jones, Lane Volpe, Steve Emery, Julian Kotze, Teresa Platz, Julia Chase-Grey and Katie Oven. You provided me with interesting and stimulating conversation, debate and gossip. You (almost) tirelessly listened to my continual rants against neo-liberal economics and you made absolutely excellent drinking partners – usually on a Friday afternoon when we'd all had enough of work.

I must also thank my friends and family who supported me through this PhD – especially my parents, Clare Wynn-Mackenzie, Vic Jupp, Matt Durey, Matt Guest, Ralph Wood and Sue, Tim, Charlie, Sam and Tilly Nicholls. You've all been wonderful!

I am very grateful to the ESRC and MRC for the studentship they provided, to the Nepal Health Research Council for giving permission to do this work and to the Biosocial Society and the Parkes Foundation for fieldwork funding.

Finally, I must also thank my supervisors – Prof. Catherine Panter-Brick and Dr Andrew Russell. I couldn't have done this with without you and I am so grateful for all your time and support. I am particularly grateful to Catherine who gave me the opportunity to go to Nepal in the first place and has been a great support and brilliant friend ever since. I'll never forget having supervision with her whilst bouncing on a trampoline!

THANK YOU ALL

Hand-washing and its impact on child health in Kathmandu, Nepal

Abstract

Gut damage, resulting in maldigestion or malabsorption of food and stimulation of the immune system, has been linked to growth faltering in young children in the developing world. Gut damage occurs along a spectrum, with only the more severe damage resulting in visible symptoms such as diarrhoea; most gut damage in young children is sub-clinical but chronic, and over time it can have a significant impact on a child's growth rate. Hand-washing with soap has been found to reduce the risk of diarrhoea by 42-47%. Would this simple intervention also reduce the sub-clinical yet chronic form of gut damage associated with childhood growth faltering? Framed within the bio-cultural research paradigm, and theoretically informed by insights from Critical Medical Anthropology, this study used a mixed-method, longitudinal approach in order to investigate this question.

Eighty-eight children aged 3-12 months were recruited from eight slum communities in Kathmandu, Nepal. Each community was allocated to a control or intervention group (n=43 and 45 children, respectively). In intervention areas, a community-based hand-washing with soap programme was devised and implemented for six months; in control areas, mothers continued their normal practices. The intervention was evaluated by comparing five outcomes: rates of maternal hand-washing, levels of child morbidity, gut damage, immune stimulation and growth faltering in the two groups.

Hand-washing rates increased amongst intervention mothers: by the end of the intervention, mothers living in hand-washing areas were more likely to report hand-washing with soap after cleaning the baby's bottom and before cooking, eating or feeding the child (for all, $P<.01$). As a result, children in the intervention areas experienced a decrease in both the number of diarrhoeal episodes (3.0 vs. 4.3 episodes, $P=.049$) and the number of days with diarrhoeal symptoms over the period of study (9.67 vs. 16.33 days, $P=.023$).

Yet, despite reducing diarrhoeal morbidity, hand-washing had no impact on the biochemical or growth status of the children: there was no significant reduction in levels of gut damage or immune stimulation in children from intervention areas over the period of

the study. Consequently there was no improvement in growth rates for these children, as measured by height-for-age, weight-for-age and weight-for-height z-scores.

This study concludes that when children live in highly contaminated, over-crowded environments, with poor access to clean water and sanitation, selective interventions focusing on one small behavioural change are unlikely to have an impact. In such highly contaminated environments, faecal contamination of hands is just one of the many pathways by which these children are exposed to pathogenic organisms. The biggest threat to the health of these children is not poor hygiene behaviour, but life in the slum. Comprehensive strategies to provide basic services and raise general standards of living in the slums are the best way in which to have a significant impact: piecemeal interventions focusing on single issues risk being ineffective both in terms of health impact and cost-effectiveness. This point is situated within the literature on effective and sustainable health interventions and the wider social and political debates surrounding global public health policy and practice in the 21st Century.

Table of Contents

CHAPTER 8

CONCLUSIONS AND IMPLICATIONS: PRIORITIES FOR THE 21ST CENTURY 229

List of Tables

List of Figures

List of Boxes

Abbreviations and Acronyms

AGP	α-1-acide glycoprotein
AIDS	Acquired Immune Deficiency Syndrome
ARI	Acute Respiratory Infection
ATP	Adenosine-5'-triphosphate disodium salt
BMI	Body Mass Index
BMR	Basal Metabolic Rate
CDC	Center for Disease Control
CI	Confidence Interval
CM	Community Motivator
CRP	C-Reactive Protein
CSDH	Commission for the Social Determinants of Health
DFID	Department for International Development
ESRC	Economic and Social Research Council
FG	Focus Group
GD	Gut Damage
GF	Growth Faltering
GDP	Gross Domestic Product
HAZ	Height-for-Age- z-score
Hb	Haemoglobin
HIV	Human Immunodeficiency Virus
HK-6GP	Hexokinase-glucose-6 phosphate dehydrogenase
HW	Hand-Washing
HWWS	Hand-Washing With Soap
IDR	Incidence Density Ratio
IgG	Immunoglobulin G
IQ	Inter-quartile range
IS	Immune Stimulation

KTM	Kathmandu
L:C	Lactose:Creatinine ratio
MDG	Millennium Development Goal
MRC	Medical Research Council
NADP	Nicotinamide Adenine Dinuculotide Phosphate
NCHS	National Center for Health Statistics
NGO	Non-Governmental Organisation
NWSC	Nepal Water Supply Corporation
PUFA	n-3 poly-unsaturated fatty acids
RCT	Randomised Controlled Trial
RR	Relative Risk
Rs	Rupees (Nepali)
SD	Standard Deviation
SES	Socio-economic status
SPSS	Statistical Package for Social Science
STI	Sexually Transmitted Infection
TE	Triethlanolamine
UK	United Kingdom
UN	United Nations
UNESCO	United Nations Educational, Scientific and Cultural Organization
UNICEF	United Nations Children's Fund
US/USA	United States (of America)
UV-A	Ultra-Violet rays A
WAZ	Weight-for-Age- z-score
WHO	World Health Organization
WHZ	Weight-for-Height- z-score

CHAPTER 1

Growth Faltering, Infections and Hand-washing

Introduction

This thesis is about the impact of a hand-washing intervention on the health and growth of young children living in the slums of Kathmandu, Nepal. The research sought to document, understand and influence the phenomenon of childhood growth faltering both in terms of its biological causes and consequences *and* its wider social, economic and environmental influences. In order to do this, I employed a mixed-method approach to design, implement and evaluate a community-based hand-washing intervention in the slums of Kathmandu, Nepal. Thus I sought to integrate quantitative research methods located within the biomedical paradigm with qualitative approaches that provided meaning and context for the research. Human health and well-being are complex phenomena - the products of an intricate and dynamic interplay between human biology and human society. The best way to understand human health must therefore be through an approach that seeks to understand this dynamic interaction by integrating different research methodologies.

This thesis was conceptualised, conducted and analysed using a theoretical approach influenced by Critical Medical Anthropology (Singer and Baer 1995) and under the broad paradigm of 'bio-cultural' research (Goodman and Leatherman 1998). Originally posited as a means of bridging the growing chasm between biological and socio-cultural fields of anthropology, the bio-cultural paradigm seeks to understand human biology in terms of its relationship with both the natural, physical environment and the broader socio-cultural context. Critical Medical Anthropology exhorts anthropologists to further widen their theoretical scope to include a consideration of the impact of power (or lack thereof) and political economy on human health and well-being, spanning from the micro- to the macro-level of influence.

In this introductory chapter I present the research background to my study, discussing the issue of growth faltering and its relationship to infectious disease as a pressing issue for global public health community. Chapter 2 describes both the study setting – the slums of

Kathmandu – and the methods used in the research. In Chapter 3, I describe the way in which the hand-washing intervention was designed and implemented in the slum communities. Chapters 4 and 5 present a profile of the behavioural, socio-economic and health characteristics of the communities studied, and the impact of the intervention on the health and growth of the children. Chapter 6 evaluates the implementation and impact of the intervention, whilst Chapter 7 discusses the wider constraints on child health and well-being in the slums. Chapter 8 – the conclusion to the thesis – examines the wider implications of this study's findings for global public health practice in the 21st Century.

1.1 Childhood growth faltering: where, when and why does it matter?

The relationship between health and growth in childhood is very simple: healthy children grow well, sick children do not. Because of its remarkable sensitivity to environmental insults, growth is perhaps the best global indicator of child well-being. Despite notable reductions in the prevalence of childhood growth faltering since the 1980s (de Onis, Frongillo et al. 2000) it remains an insidious and dangerous problem for millions of children throughout the developing world. For this reason, in September 2000, world leaders made a commitment to tackle this global issue, and pledged to halve the number of under-weight children by 2015 as part of the Millennium Development Goals. At the baseline year of 1990, globally 33% of children were under-weight; by 2006 progress had been made and this figure had dropped to 26% (United Nations Statistics Division 2008). Yet with just six years to go until the 2015 deadline, it is apparent that the set target is unlikely to be met (United Nations 2008).

1.1.1 Global distribution of child growth faltering

The latest global figures from UNICEF state that just under a third (31%) of children are stunted (low height-for-age), one in four is under-weight (low weight-for-age) and one in ten is wasted (low weight-for-height) (UNICEF 2009); almost all of these children live in the developing world. South Asia bears the heaviest burden of child growth faltering with over half of the world's underweight children (78 million) living in this region, mostly in India, Pakistan and Bangladesh. The astonishingly high levels of growth faltering seen in

this region are difficult to explain and this problem has been dubbed 'the Asian enigma' (Ramalingaswami, Jonsson et al. 1996): as Bhutta notes, 'although levels of poverty and agricultural production in South Asia are similar to those in sub-Saharan Africa, rates of malnutrition in South Asia are significantly and persistently higher' (2000:809). Nepal is no exception to this South Asian pattern; just under half of all the children in Nepal are significantly stunted or under-weight (Figure 1.1).

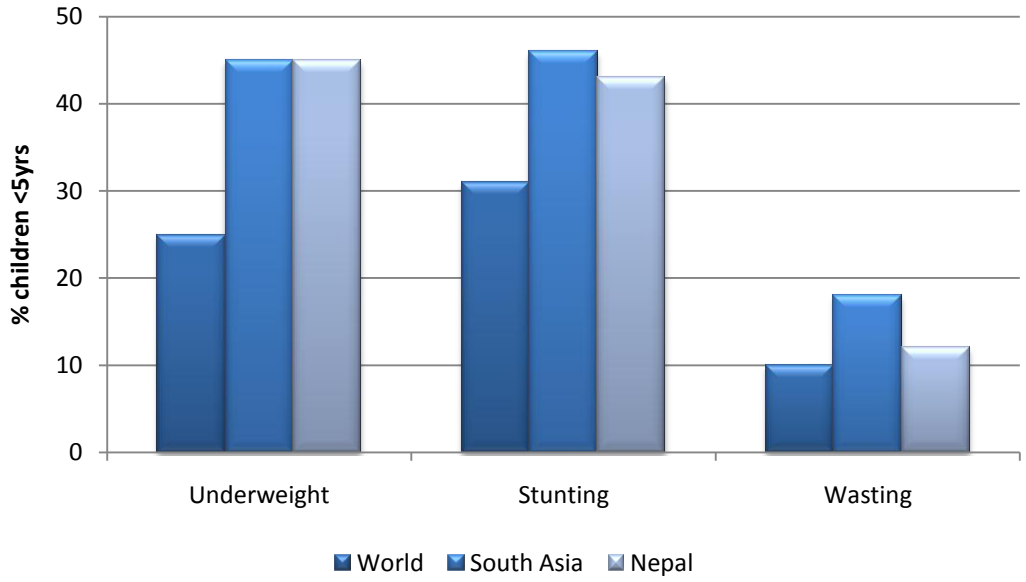


Figure 1.1 Levels of underweight, stunting and wasting in children under five years of age for the world, South Asia and Nepal. Data taken from UNICEF (2009).

1.1.2 Timing of child growth faltering

The process of growth faltering starts early in life and appears to follow a near universal pattern. A recent review of growth data from 39 nationally representative datasets from across the world revealed surprisingly similar trajectories of growth faltering in children, not only within specific regions, but also at a global level (Shrimpton, Victora et al. 2001).

At birth, average length-for-age, weight-for-age and weight-for-length z-scores fall close to the National Centre for Health Statistics (NCHS) reference (Figure 1.2). The process of stunting starts at birth and continues into the third year of life. Weight starts to falter at approximately three months of age and declines rapidly until 12 months, followed by a markedly slower decline until 18 months. Wasting, however, appears to be restricted to the first 15 months of life. For all three measures of growth faltering (stunting, underweight and wasting), Asian children show consistently and considerably worse trajectories

compared to their African and Latin American counterparts (Shrimpton, Victora et al. 2001).

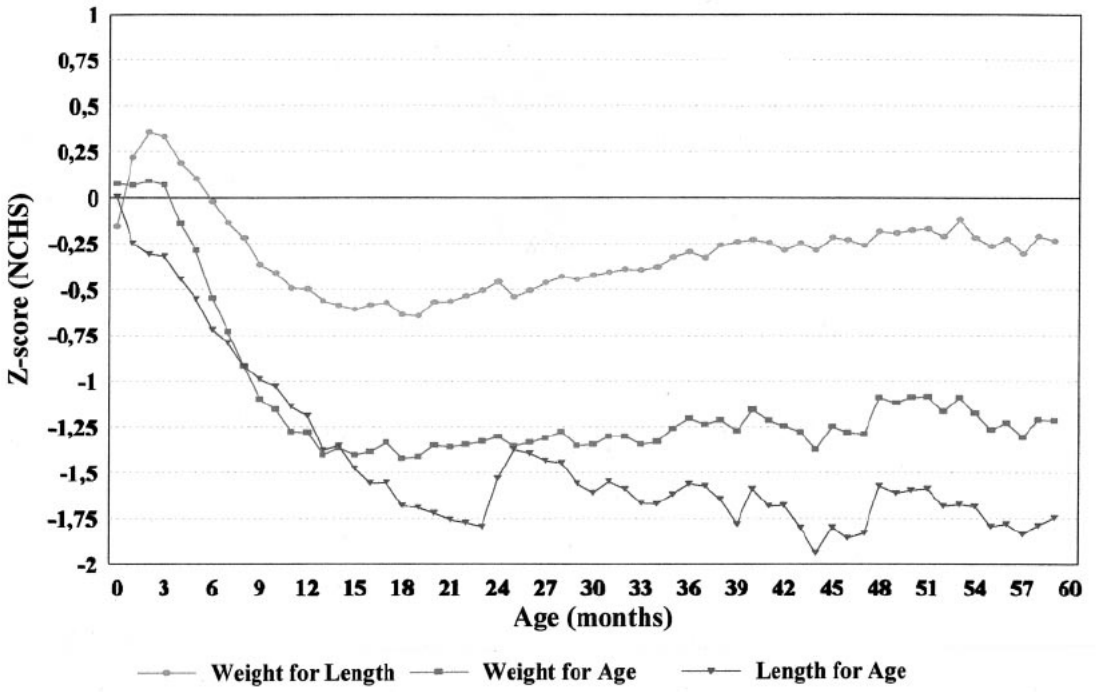


Figure 1.2 The timing of growth faltering: mean anthropometric z-scores by age, relative to the National Centre for Health Statistics reference. Taken from Shrimpton et al. (2001)[1].

Clearly, the first two-to-three years of a child's life are crucial in determining his/her growth trajectory. Shrimpton et al. (2001) argue that interventions should therefore concentrate on the earliest periods of life – the pre-natal period, infancy and early childhood – in order to have the greatest chance of preventing childhood growth faltering. However, for those children who have already experienced growth faltering, there is considerable debate as to whether or not this constitutes a permanent situation or whether there can be catch-up growth in later childhood and adolescence.

Martorell et al. (1990) argued that stunting is 'a condition resulting from events in early childhood and which, once present, remains for life', and suggest that no catch-up growth occurs to compensate for these early insults in later childhood and adolescence. There are numerous studies that support this argument reporting very high correlations between

[1]NB. The sudden increase in length-for-age at 24 months is artefactual and is the result of the disjunction of the two datasets that make up the NCHS reference curve (Shrimpton et al. 2001).

height at three years and final adult height (for example, Mills, PH et al. 1986; Satyanarayana, Prasanna et al. 1986; Binkin, Yip et al. 1988; Martorell, Rivera et al. 1992). Golden (1994:s58) notes, 'these data could be interpreted to show that a period of malnutrition in the first two-to-three years of childhood irrevocably changes the child so that he is 'locked into' a lower growth trajectory with a lower potential for future growth'.

Clearly a child's potential to catch-up on growth is strongly related to the timing and severity of the faltering (Golden 1994). However, numerous supplementation and adoption studies provide evidence to suggest that at least partial recovery from stunting in young children is possible. For example, in a study of over 2000 Filipino children who were followed from two to twelve years of age, Adair (1999) found evidence of moderate recovery from stunting; at twelve years, 63% of children were stunted, but this figure had dropped to 50% by twelve years of age. Similarly, a study conducted by Proos et al. (1991) demonstrated catch-up growth in Indian children adopted by Swedish parents. These children experienced significant catch-up in height within two years of arriving in Sweden and by puberty were only 0.3 z-scores below the NCHS reference curve. However, because these children experienced an early puberty, ultimately their final adult height fell 1.4 z-scores below the reference. In his comprehensive review of the literature on this issue, Golden concludes that although catch-up growth in height is possible, it rarely occurs since the conditions required for this are rarely satisfied: 'in most populations the environment and diet, associated with poor growth performance initially, do not change' (1994:s58).

1.1.3 Consequences of childhood growth faltering

The impact of growth faltering on health and well-being is severe and long-lasting. It is associated with numerous negative sequelae that track across the life span and even across generations. Children with poor growth status have been found to be at greater risk of diseases such as diarrhoea and respiratory infections (Bhandari, Bhan et al. 1989; Zaman, Baqui et al. 1997). Such infections are also likely to be more severe and of longer duration in poorly growing children (James 1972; Tomkins 1988). Consequently, these children experience much higher mortality rates, with poor growth status being associated with over half of all childhood deaths (Calder and Jackson 2000). If they survive their first five years, the early insults to their growth continue to impact on their health and well-being into adolescence and adulthood. Growth stunting in early childhood has been shown to impair

physiological, motor function and intellectual development (Grantham-McGregor 1995; Oberhelman 1998; Mendez 1999; Berkman, Lescano et al. 2002). It is also known to affect levels of physical activity and work capacity, diminishing income-generating abilities. In women it can result in reduced fertility and higher rates of mortality during child birth (Norgan 2000). In addition, the negative effects of growth retardation can span the generations: women who experience poor growth in their own childhood are more likely to give birth to under-weight babies. These infants not only have a dramatically reduced chance of survival in the short-term, they also appear to be predisposed to long-term health risks such as hypertension, insulin resistance, type II diabetes and cardiovascular disease (Barker 1998).

Growth faltering during childhood can therefore have negative impacts that are dramatic, long-term and inter-generational. Finding ways to tackle this endemic problem and reduce levels of growth faltering is a key priority in the battle to save and improve the lives of millions of children throughout the developing world.

1.2 Causes of childhood growth faltering

Growth is a highly complex and sensitive process – the product of an intricate interaction between genes and the environment (Bogin 1999). On an individual level, genetic make-up plays an important role in determining adult height and weight. However, though it was originally believed that genes were also responsible for much of the global variation in growth at the population level, it is now thought that the influence of ethnicity on growth is minimal. New global data collected by the WHO show that given the optimum start in life, 'children born anywhere in the world…have the potential to develop to within the same range of height and weight' (WHO 2006). Although there are individual differences among children, across large populations (both regionally and globally) the average growth of well-off children is remarkably similar. On a global level therefore, it appears that the environment, rather than the genes, plays the greatest role in determining growth and two of the most important environmental factors are nutrition and infection.

1.2.1 Role of nutrition

Inadequate nutrition is an obvious culprit for growth faltering: children who do not receive adequate levels of essential nutrients will inevitably not be able to sustain growth at the

expected normal levels. Countries with the highest levels of food scarcity and insecurity therefore also experience the highest levels of growth faltering, and there are numerous studies documenting the impact of inadequate intake of protein and/or calories on growth. For example, Kapur et al.'s (2005) study focusing on young children (less than three years of age) living in an urban slum in Delhi, found that nutritional intake was grossly inadequate, supplying just 13-56% of the recommended daily allowances for ten key nutritional items. Levels of growth faltering were correspondingly very high; three-quarters of children were underweight and/or stunted (<-2 WAZ/HAZ), with over a third being severely affected (<-3 WAZ/HAZ).

However, adequate levels of protein and calories alone are not sufficient to stave off growth faltering; deficiencies in one or more micro-nutrients can also have significant deleterious effects on growth. There is now strong evidence for the contribution of zinc deficiency to growth faltering among children, with even mild to moderate deficits affecting growth (Rivera, Hotz et al. 2003). Deficiencies in vitamin A and iron are also noted to impair growth in young children, though only when such deficiencies are severe (*ibid*).

The role of nutrition in growth faltering therefore is well established and well documented. The 1960s and '70s saw numerous macro- and micro-nutrient feeding programmes implemented throughout many poor developing countries with the aim of improving child growth and health. Yet in general such food supplementation programmes have been unsuccessful at reducing levels of growth faltering (Schilling 1990). For example, Figure 1.3 below shows the results from a five-year supplementation trial implemented by the UK Medical Research Council in The Gambia.

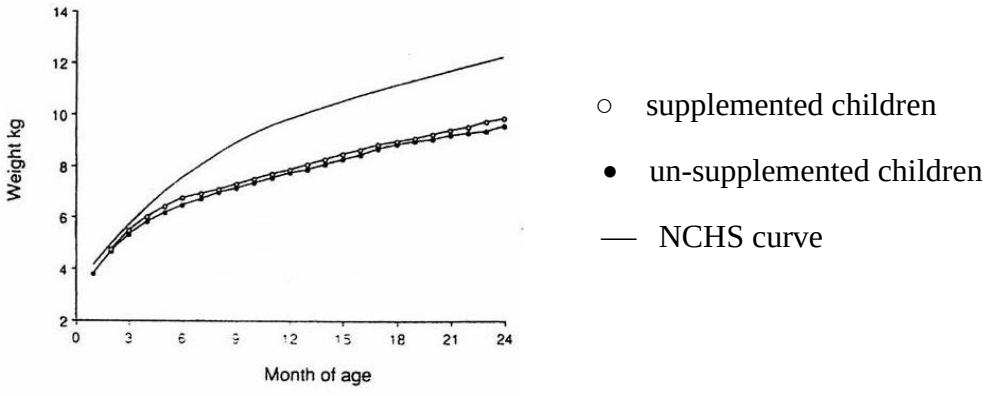


Figure 1.3 Weight gain in supplemented and un-supplemented Gambian children. Taken from Prentice et al. (1993).

Children in this programme were given massive dietary supplementation including twice the recommended amount for energy, 2.5 times the recommended amount for protein, and substantial vitamins and minerals (Hoare, Poppitt et al. 1996). Although the intervention did produce short-term catch-up growth in supplemented children, this was reversed as soon as the child left the feeding study (Rowland, Rowland et al. 1988; Sullivan, Lunn et al. 1992). Ultimately, supplemented and non-supplemented cohorts of infants showed very little difference in their growth profiles.

The lack of success of these programmes therefore revealed an important message: adequate nutrition is undeniably necessary, *but not sufficient* to ensure proper linear or ponderal growth. Clearly within many developing countries growth is constrained not merely by nutrition, but by other important environmental factors (Panter-Brick, Lunn et al. 2009).

1.2.2 Role of infection

As a result of this conclusion attention has turned once more to the role of infection in growth faltering. It is now widely accepted that infectious diseases in childhood can have a significant detrimental impact on growth velocity and are at least as important as nutritional deficiencies in the causation of growth faltering (Martorell and Ho 1984). Indeed some go even further and argue that infection is *the* primary cause of the majority of growth faltering observed in children of the developing world (Mata, Kromal et al. 1977). The ways in which infection can lead to growth faltering in a child are summarised in Figure 1.4 and are discussed below.

Infection and reduced nutrient intake

Many childhood infections are characterised by a loss of appetite in the child: local inflammation at the site of infection provokes a systemic inflammatory response leading to an increase in plasma cytokines known to induce anorexia (Northrop-Clewes, Rousham et al. 2001). This can lead to what can be a dramatic reduction in both macro- and micro-nutrients: Molla et al. (1983) found that Bangladeshi children less than five years-old consumed 40% less energy during the acute stages of diarrhoea, compared with after recovery; Duggan et al. (1986) reported a 75% reduction in energy consumption in young Kenyan children with measles. The effects of infection-induced anorexia are in many countries further exacerbated by cultural beliefs and practices regarding the withholding of food during sickness (Calder and Jackson 2000).

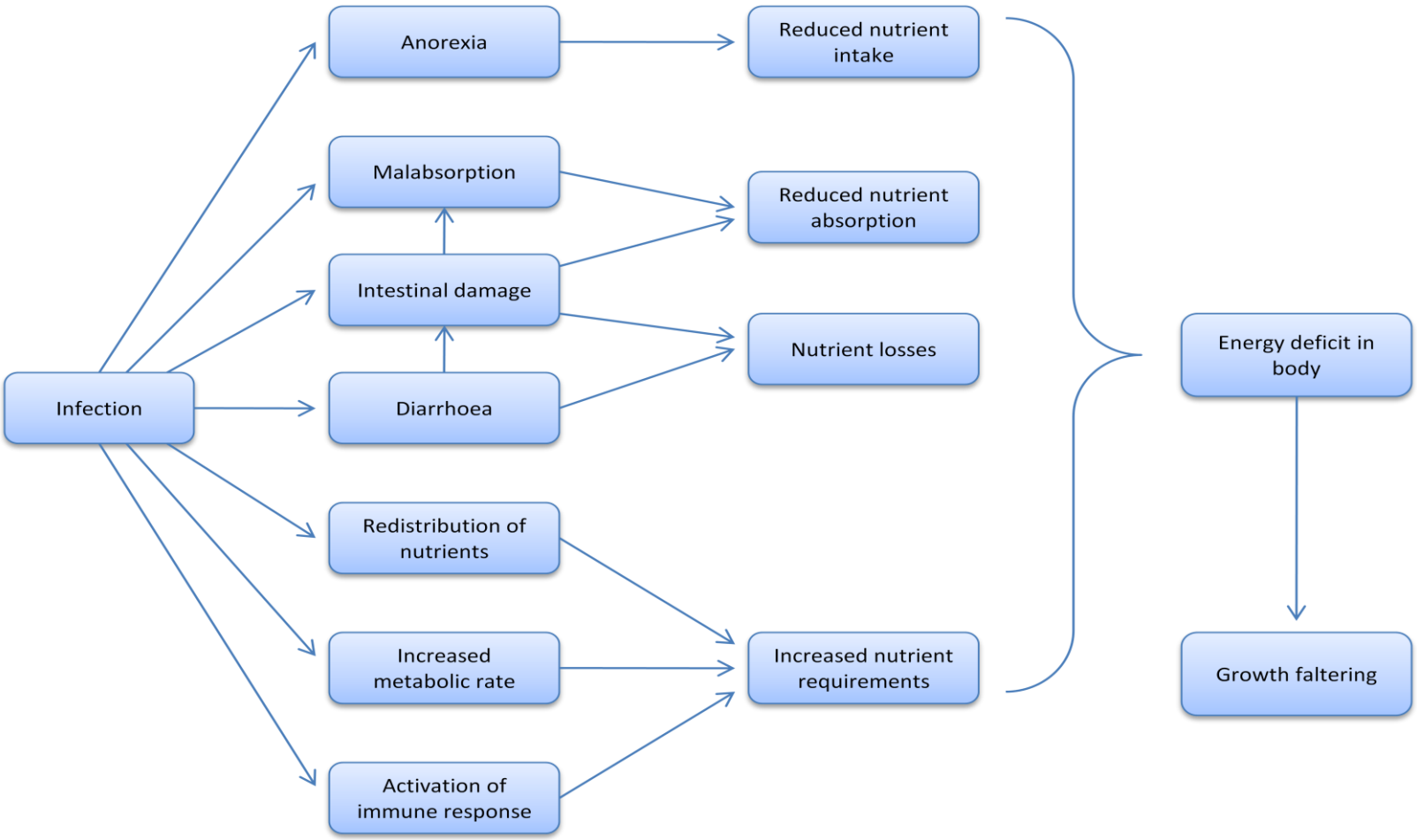


Figure 1.4 Effects of infection on the host leading to energy deficits and growth faltering. Adapted from Calder & Jackson (2000:5)

Infection and reduced nutrient absorption and nutrient losses

Diarrhoea, measles and helminths infections can all cause damage to the intestinal wall resulting in malabsorption of nutrients from both breast-milk and complementary foods. Absorption levels in Bangladeshi children suffering from rotavirus diarrhoea averaged only 43% for nitrogen, 42% for fat, 74% for carbohydrates and 55% for total energy (Molla, Molla et al. 1983). Infections have also been shown to block absorption of specific micro-nutrients such as iron, copper, zinc and vitamin A (Cartwright, Lauritsen et al. 1946; Sivakumar and Reddy 1975; Castillo-Duran, Vial et al. 1988). Some infections are also known to cause direct nutrient loss via the faeces as a result of severe damage to the intestinal wall: a study in Bangladesh found that 65% of patients with enterotoxic *E. coli* and 40% of those with rota-virus diarrhoea had excessive losses of protein in their faeces (Scrimshaw and SanGiovanni 1997).

Infection and increased nutrient requirement

In addition to causing a reduction in intake and absorption of food, infections also actually increase nutritional requirements, further exacerbating energy deficits. For each 1°C increase in temperature, there is a corresponding 13% increase in basal metabolic rate (Calder and Jackson 2000). Consequently, infections inducing fever can dramatically increase the energy requirements of a child during periods of sickness.

1.2.3 Diarrhoeal infections

Infectious diseases can therefore cause a negative energy balance within the child which, if un-checked, can quickly lead to growth faltering. A number of infectious diseases, including malaria and pneumonia, have been found to have a negative impact on growth (Rowland, Cole et al. 1977; Victora, Barros et al. 1990). However, perhaps not surprisingly, the bulk of research into this area has focused on the role of diarrhoeal disease in growth faltering.

Diarrhoea is the second biggest killer of children under five years, accounting for more deaths than malaria, measles and HIV combined (UNICEF/WHO 2006). In absolute figures this equates to over 1.87 million deaths per year: approximately one child dying every 17 seconds (WHO 2005). The non-fatal morbidity level for diarrhoea is obviously much higher and it has been estimated that in developing countries children under five

years of age suffer on average 3.2 episodes of diarrhoea per child year, though in some areas this figure is significantly higher (Kosek, Bern et al. 2003).

The relationship between diarrhoea and growth faltering is known to be bi-directional and synergistic (Scrimshaw, Taylor et al. 1968). Diarrhoea adversely affects a child's nutritional status through reductions in dietary intake, impairment of intestinal absorption and increases in nutrient requirements. Conversely, children with poor nutritional status are predisposed to infection because of impaired skin and mucous membranes that provide a barrier against pathogens, and changes in immune functioning (Calder and Jackson 2000; Brown 2003). Because of the enormous potential of diarrhoea to cause childhood growth faltering, it is worth reviewing this relationship in some detail.

Incidence of diarrhoea

A number of studies suggest that children with poor growth status experience an increased incidence of diarrhoeal disease. Several studies have found an association between low weight-for-age and diarrhoeal incidence (Gordon, Gúzman et al. 1964; James 1972; Bhandari, Bhan et al. 1989). Sepúlveda et al.'s (1988) study of children below two years of age in urban areas of Mexico found diarrhoeal incidence rate almost doubled to 6.0 episodes per year in moderately underweight children, compared to just 3.3 episodes in children of normal weight. Tomkins (1981) noted a similar association with wasting in Nigerian children, with children less than 80% standard weight-for-height experiencing 47% more episodes of diarrhoea than better nourished children. However, a number of other studies have failed to find such an association (Black, Brown et al. 1984a; Bairagi, Chowdhury et al. 1987; Henry, Alam et al. 1987). Indeed, Chen et al. (1981) designed a study specifically to test the hypothesis linking poor growth status and incidence of diarrhoea in Bangladeshi children and failed to find any evidence to support it.

Severity of diarrhoea

There is better agreement within the literature to suggest that once infected, children with poor growth status experience infections of greater severity. Children with poor growth status often suffer increased duration of diarrhoeal episodes. Black et al. (1984a) found that duration of diarrhoea in Bangladeshi children less than 80% of the NCHS weight-for-height standard was 56% longer compared to children with better growth status. In Nigeria, diarrhoeal episodes were 33% longer in underweight children, 37% longer in stunted children, and 79% longer in wasted children (Tomkins 1981). In addition, poor growth

status is associated with increased risk of admission to hospital or mortality following a diarrhoeal infection. Brazilian children with low birth weights were 1.95 times more likely to be admitted to hospital for diarrhoeal infections than children with satisfactory birth-weight (Victora, Barros et al. 1990). In Filipino children, each one unit decrease in weight-for-age z-scores (WAZ) was associated with a 1.6 fold increased risk of mortality (Yoon, Black et al. 1997).

Impact on short-term growth

Diarrhoeal infections impact negatively on short-term weight gain in children by causing anorexia and damage to the intestinal walls, leading to malabsorption and nutrient losses. Numerous studies conducted in different countries across the world have found an association between significant reductions in short-term ponderal growth and diarrhoeal infection (measurement intervals ranged from one to three months: Rowland, Cole et al. 1977; Black, Brown et al. 1984a; Bairagi, Chowdhury et al. 1987; Briend, Hasan et al. 1989; Becker, Black et al. 1991; Walker, Grantham-McGregor et al. 1992).

Evidence to suggest that diarrhoea may also affect short-term height gain is less consistent, though this is perhaps not surprising given that, unlike weight, height measurements cannot decrease (Stephenson 1999). Nonetheless, a higher frequency of diarrhoea has been associated with reduced short-term height increments in a number of studies in The Gambia, Jamaica and Bangladesh (Rowland, Cole et al. 1977; Black, Brown et al. 1984a; Walker, Grantham-McGregor et al. 1992). Other studies, however, have failed to find such an association. Bairagi et al. (1987), for example, found that in Bangladeshi children aged between one and four years, diarrhoeal infections resulted in significantly reduced increments in weight over a two month interval, but had no effect on linear growth rates.

Impact on long-term growth

Whilst the short-term impact of diarrhoea on ponderal, if not linear, growth is well established, its long-term impact remains a much debated and highly contested issue. Some studies suggest that diarrhoeal disease has a long-term detrimental impact on both height and weight, whilst others suggest that catch-up growth after periods of illness generally averts any long-term growth faltering. What constitutes ‘long-term’ in this context is highly debateable, though extremely important. As Checkley et al. (2003) point out, relatively short ‘long-term’ intervals may over estimate the impact of diarrhoea since it may not be possible to detect in this small interval any catch-up growth; on the other hand,

such intervals may under estimate any impact because they do not allow enough time to detect possible delayed effects.

Studies using various 'long-term' intervals of different lengths have suggested diarrhoea does have a long-term impact on growth. Kossmann et al. (2000) assessed the impact of diarrhoea on child growth over a six month period in Sudanese pre-school children. They found that attained height over this period was 17mm lower in children who had experienced diarrhoea compared to children who had not. Bairagi et al. (1987) found that diarrhoea had an impact on both attained height and weight over an eight-month period in Bangladeshi children: children who suffered from diarrhoea more than 10% of days in this time-period achieved only 70% weight and 75% height velocity compared to children who were healthy during this period. Other studies have found noticeable effects of diarrhoea on linear and/or ponderal growth over an interval of one year (Condon-Paoloni, Cravioto et al. 1977; Black, Brown et al. 1984b; Torres, Peterson et al. 2000). Moore et al. (2001) related diarrhoeal burden during the first two years of life to growth faltering two to seven years later in a cohort of 119 Brazilian children: even after controlling for nutritional status in infancy, family income and maternal education, early childhood diarrhoeal burden was significantly associated with linear growth faltering in later childhood.

Many authors, however, suggest that although diarrhoea can affect growth in the short-term, such deficits are transitory and are usually quickly made up through catch-up growth. In their view diarrhoea therefore has no long-term effect on child growth retardation. Moy et al. (1994) set out specifically to test the hypothesis that diarrhoea was important cause of growth faltering in young children. In their study of 204 Zimbabwean infants recruited at <12 months of age, they found little difference in the growth status between children with frequent and infrequent diarrhoea. Diarrhoea appeared to have only a transient effect on weight gain: weight loss associated with each episode was small (approximately 2%) and return to the child's trend was 90% complete within a month.

Briend et al. (1989) conducted a study on rural Bangladeshi children aged 6-35 months. Though short-term reductions in height and weight were associated with diarrhoea, catch-up growth was found to occur and deficits in weight gain and linear growth were no longer apparent a few weeks later. They concluded that the effect of diarrhoea on child growth is

Study	Sample	Key findings
Martorell et al. (1975) Guatemala	716 children, 0-7 years	Children who had a low prevalence of diarrhoea had substantially larger increments in length and weight than children who were sick more frequently.
Condon-Paolini et al. (1977) Mexico	276 children recruited from birth	High frequency of diarrhoea in first three years of life was significantly associated with reduced weight gain, but had no affect on height.
Mata et al. (1977) Guatemala	30 children recruited from birth	Diarrhoea had a negative impact on both height and weight increments, often persisting for weeks or months.
Black et al. (1984a) Bangladesh	157 children, 6-48 months	Diarrhoea had a significant impact on height, but not weight, increments over a 12 month period.
Zumrawi et al. (1987) Sudan	439 children recruited from birth	After three month of age an episode of diarrhoea in any two-week period reduced the gain in that period to less than 50% of that found in uninfected children. Diarrhoea did not always lead to faltering, but it was an initiating factor in about half of those children who did falter.
Bairagi et al. (1987) Bangladesh	1000 children, 1-4 years	Acute diarrhoea reduced short-term weight increments, but had no effect on long-term (eight-month) height or weight increments. Chronic diarrhoea, however, reduced both height and weight increments in the long term.
Rowland et al. (1988) The Gambia	126 children recruited from birth	Diarrhoea accounted for half of the observed deficits in weight gain in children by 12 months. Diarrhoea reduced weight gain in weaned infants by 14.4g/day. No impact was observed for length gain.
Lutter et al. (1989) Colombia	241 children recruited from birth	Diarrhoea was negatively associated with length at 36 months in un-supplemented children, but had no effect on children who had received nutritional supplementation.
Briend et al. (1989) Bangladesh	6-35 months, n= not given	Analysis of three-month intervals suggested that though diarrhoea slowed weight gain and linear growth, the effects were transitory and were no longer apparent a few weeks later.

Study	Sample	Key findings
Becker et al. (1991) Bangladesh	70 children, 5-18 months	Monthly changes in weight were inversely related to diarrhoea. However the authors conclude that increasing nutritional intake to the recommended WHO standards would have a greater impact on weight gain than eliminating diarrhoea.
Walker et al. (1992) Jamaica	161 children, 9-24 months	Analysis of two-month intervals revealed that diarrhoea had a negative effect on weight gain. Diarrhoea also reduced height increments over a four month period if diarrhoea occurred in the first two months of the interval.
Moy et al. (1994) Zimbabwe	154 children, <12 months	Little observed difference in average rates of growth between children with frequent diarrhoea and infrequent diarrhoea. Diarrhoea had only a transient effect on weight gain.
Poskitt et al. (1999) The Gambia	1190 children, 0-2 years	Over a 15-year period diarrhoeal incidence and prevalence fell by 23% and 24% respectively, yet had no impact on HAZ and WAZ of infants at 12 and 24 months.
Kossman et al. (2000) Sudan	28,753 pre-school children	Childhood infections were negatively associated with both height and weight increments over a six-month period. Attained height was on average 11mm lower in children who experience diarrhoea in the previous six months.
Torres et al. (2000) Bangladesh	182 children, 5-11 years	Total number of days with diarrhoea was negatively associated with annual weight gain, but had no impact on annual height gain.
Alam et al. (2000) Bangladesh	584 children, 6-48 months	Over a three-month interval both diarrhoea and dysentery were associated with lower weight gain. Dysenteric diarrhoea was also associated with lower annual height and weight gain.
Moore et al. (2001) Brazil	119 children, 0-24 months	Even after controlling for nutritional status, family income and maternal education, diarrhoeal burden between birth and two years was significantly associated with growth faltering in height at ages 2-7 years.
Checkley et al. (2003) Peru	224 children recruited from birth	Diarrhoea during the first six months of life resulted in long-term height deficits that tended to be permanent. Diarrhoea after six month of age showed only transient effects.

Table 1.1 Findings from observational studies investigating the impact of diarrhoea on child growth

transitory and efforts to reduce childhood diarrhoeal rates are unlikely to improve children's nutritional status.

This conclusion is supported by data from a 15-year study in the Gambia. Childhood diarrhoeal incidence and prevalence between 1979 and 1993 were reduced by 23% and 24% respectively in a rural Gambian community. This reduction, however, resulted in no change in either height-for-age or weight-for-age z-scores of children aged 12 and 24 months (Poskitt 1999). The results from these and other relevant studies are summarised in Table 1.1 on the previous page.

There are perhaps a number of methodological reasons for these equivocal data on the role of diarrhoea in causing long-term growth faltering. Firstly, a universal and rigorously applied definition of diarrhoea is lacking from much of the literature; future studies should adhere to the WHO definition of diarrhoea – at least three unusually loose or watery stools in a 24-hour period (WHO 2005). Secondly, many studies have failed to control properly for important variables that might influence growth, such as initial nutritional status, socio-economic variables and age (Bairagi, Chowdhury et al. 1987). Thirdly, most studies fail to differentiate between aetiological types of diarrhoeal infection. Alam et al. (2000) have shown that clinical type of diarrhoea (ordinary or dysenteric) is important in determining the long-term impact on growth faltering, yet most studies fail to make this distinction. Finally, few studies include children aged less than six months, though Checkley et al.(2003) found that diarrhoea in these very young children was likely to lead to permanent growth deficits, whereas the impact of infection in children older than six months was often transitory.

1.3. Sub-clinical infections

As the above review demonstrates, diarrhoea is clearly important in causing childhood growth faltering. However, it is also clear that it cannot account for all the growth faltering observed in young children in developing countries. This has therefore led to new focus on the role of *sub-clinical* (i.e. asymptomatic) infection in growth faltering.

It is worth noting at this point that diarrhoea is not a disease in itself; rather, it is a symptom of an underlying pathology - usually the result of significant damage to the

mucosal lining of the small intestine (Lunn, Northrop-Clewes et al. 1991). The pictures below show normal (left) and damaged (right) sections of the jejunal epithelium (Figure 1.5).

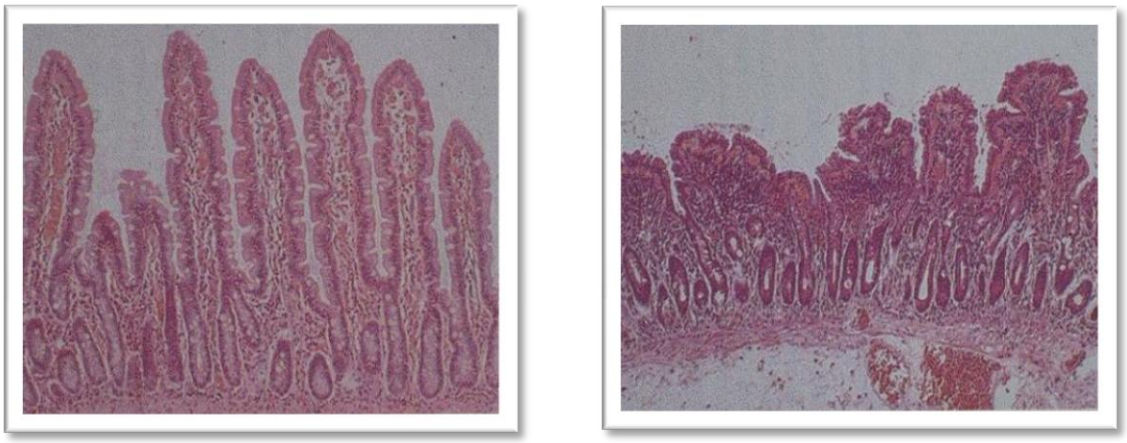


Figure 1.5 Normal and damaged jejunal epithlia. Taken from Peter and Gilles (1995).

Ingestion of enteric pathogens can cause significant damage to the villi and microvilli lining the walls of the small intestinal mucosa. Diarrhoeal symptoms are the result of significant levels of intestinal damage which impairs the host's ability to digest and absorb food. Yet, clearly the severity and extent of intestinal damage can vary widely along a continuum, from very little to very extensive levels of damage. More severe levels of damage result in clinically visible symptoms in the host such as diarrhoea, anorexia and fever. However, a significant amount of intestinal damage occurs at a sub-clinical level, i.e. it does not produce any clinically visible symptoms in the host (such as diarrhoea) but nonetheless results in some level of functional impairment.

1.3.1 Gut damage, immune stimulation and growth

In recognition of this fact, a new theory has been proposed by Dr Peter Lunn (Cambridge University), emphasising the role of intestinal damage (rather than diarrhoea per se), and its associated stimulation of the immune system, in causing childhood growth faltering (Figure 1.6).

Lunn's (2000) argument is that pathogens in food and/or water enter a young child's body and cause damage to the small intestinal mucosa. This leads to villous atrophy and erosion

of the enterocytes that cover the surface of the villi in the small intestine. Contained within these enterocytes are the many enzymes needed to digest food stuffs; degradation of such cells therefore causes maldigestion of nutrients. In addition, villous atrophy leads to a decrease in the surface area and absorptive capacity of the intestinal mucosa, similarly reducing nutrient intake. Of particular importance in young children consuming large amounts of breast milk, is the loss of the enzyme lactase which is required to digest lactose – the principle component of human breast milk. Being located in an exposed position on the intestinal villi, lactase is especially vulnerable to pathogenic damage Yet, as noted by Lunn, even in children aged 12 months, breast milk provides a substantial part of the total energy intake (20-30%), and thus any impairment in the child's ability to digest and absorb lactose could result in a significant impact on nutrient intake and growth performance (*ibid*).

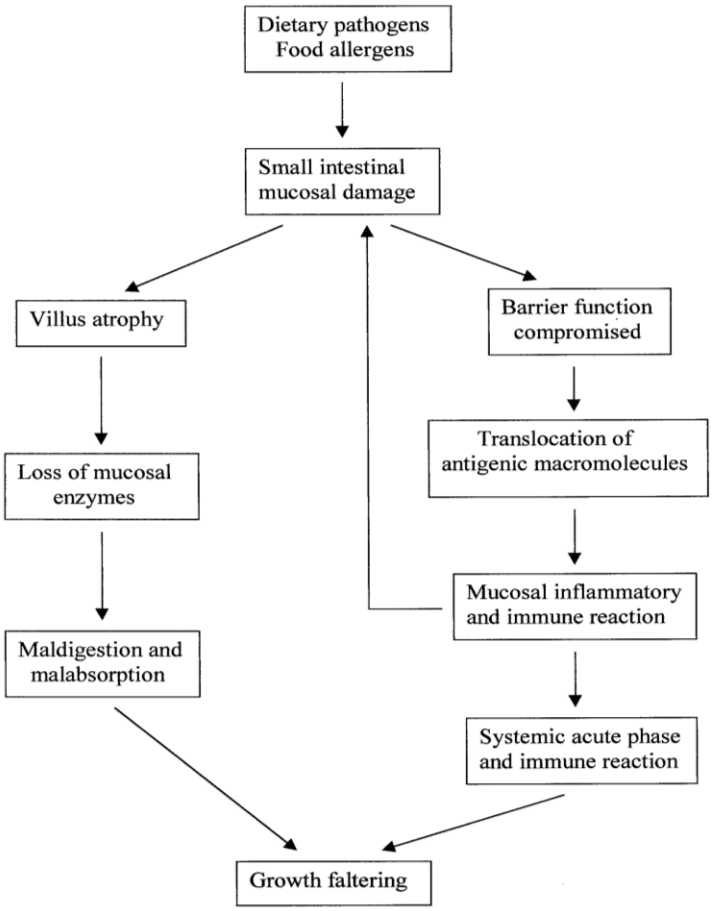


Figure 1.6 Diagrammatic representation of the mechanisms leading to poor growth children. Taken from Lunn (2000:152).

Lunn also suggests a second 'pathway' connecting intestinal damage and growth faltering in young children. Living in highly contaminated, pathogenic environments, children in the

developing world are known to have higher than expected plasma concentrations of immunoglobulins (*ibid*). Elevated levels of these and other acute phase proteins have been linked to reductions in growth in young children (*ibid*; Hautvast et al. 2000). However, high levels of these plasma protein markers of infection have also been related to increases in intestinal permeability, suggesting that immune stimulation and damage to the mucosa of the small intestine may be linked. As Lunn (2000) explains, in healthy children the intestinal mucosa plays an important role in the immune system by acting as a barrier against environmental pathogens and other macro-molecules. Damage to the mucosal lining compromises this function and allows pathogens, macromolecules and food particles to translocate across in to the body, whereupon they cause a local and systemic inflammatory and immune system response. Consequently, energy is diverted away from growth and into an immune response – fighting off infection and repairing the damaged tissues of the gut wall.

Importantly, gut damage and its associated immunostimulation can occur even in the absence of diarrhoeal symptoms. In most developing countries poor environmental conditions mean that children are frequently exposed to infection and the prevalence of subclinical infection is likely to be high (Adelekan, Northrop-Clewes et al. 2003). Thus, perpetual exposure to pathogens can lead to persistent gut damage and chronic stimulation of the immune system, preventing normal growth even in the presence of adequate nutrition and the absence of diarrhoea. This conclusion is supported by Checkley et al. (1997) in their study of the protozoan *Cryptosporidium parvum* among peri-urban Peruvian children. Although children with symptomatic cyrptosporidiosis gained less weight than those children with non-symptomatic cyrptosporidiosis, the latter form of infection was twice as common, leading the authors to conclude that asymptomatic infections may have a greater overall adverse effect on child growth.

This theory linking intestinal damage, immune stimulation and growth faltering has been most thoroughly tested in The Gambia among infants aged between 2-15 months (Lunn, Northrop-Clewes et al. 1991). During the first three months of life, Gambian infants were found to have similar intestinal permeability (gut damage) levels to UK children. At three months however, (potentially contaminated) weaning foods are introduced and by six months about 50% showed elevated intestinal permeability values – indicating significant levels of gut damage - rising to 96% by ten months. By the end of the first year of life, intestinal permeability levels were more than five times the normal UK values. This rise in

intestinal permeability levels coincided with a fall in growth performance in these children, both within and between individuals (see Figure 1.7). Indeed, over the nine-month period of study, intestinal permeability values were able to predict 43% of length and 40% of weight growth faltering. Similar increases in plasma proteins indicating stimulation of the immune system were also noted from three months and showed a close negative correlation with age (*ibid*). Further investigations revealed that intestinal enteropathy experienced in The Gambia persisted into adulthood (Campbell, Lunn et al. 2002): though permeability values did improve with age, they never fell into the 'normal' UK range. In addition, level of intestinal permeability was found to be negatively associated with adult height: the greater the level of intestinal damage, the shorter the final attained height.

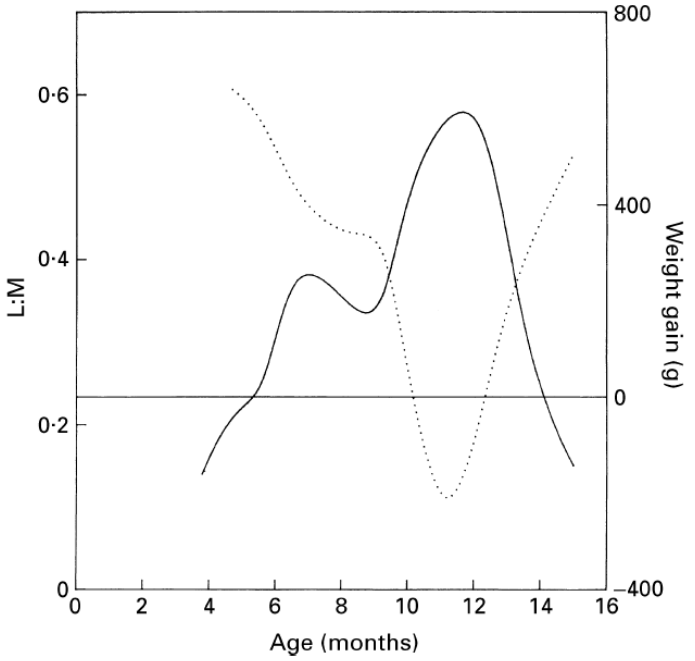


Figure 1.7 'Mirror image' relationship between growth and intestinal permeability (L:M) in Gambian infants. Taken from Lunn (2000:148).

Studies in other countries have found similar results. In an intervention study investigating the effect of helminth infections on child growth, Northrop-Clewes et al. (2001) found intestinal permeability was significantly associated with changes in height-for-age (HAZ) and weight-for-age (WAZ) z-scores in 123 Bangladeshi children (aged between two and five years). Similarly, in a study of 246 Guatemalan infants, poor weight-for-age (<-1.5 z-scores) was associated with higher levels of intestinal damage; 24% of children with WAZ >-1.5 displayed elevated intestinal permeability values, compared to 43% of children with WAZ <-1.5 (Goto, Chew et al. 1999).

The first study to examine this relationship in Nepal reported very high levels of intestinal damage (92% of children had high levels of intestinal permeability, compared to normal UK values), but found no significant relationship between intestinal permeability and growth (Goto, Panter-Brick et al. 2002). However, as the authors note, the children in this study were relatively old (mean age was 45 months); the majority of children had therefore past the age at which most growth faltering occurs (6-24 months). In addition, the study was cross-sectional and so may have been unable to detect the relationship between gut damage and growth faltering.

In light of these limitations, a second study was conducted in 2005 in Kathmandu (Panter-Brick, Lunn et al. 2009). This project, (of which I was project manager), followed two cohorts of middle-class (n=38) and slum (n=48) children aged between 3-18 months over a period of seven months. In this study, a significant negative relationship between intestinal permeability and growth performance was found and explained 9% and 19% of the deficits in height and weight in these children (ΔHAZ, $P = 0{\cdot}004$; ΔWAZ, $P < 0{\cdot}001$). Blood markers of immune stimulation (α-1-acid-glycoprotein and Immunoglobulin G) were also found to be inversely related to the children's z-scores for both HAZ and WAZ – i.e. growth was poorer when these markers of immunostimulation were elevated.

1.4 Interventions to reduce levels of gut damage and immune stimulation

Gut damage and immune stimulation evidently play an important role in growth faltering in developing countries, accounting for up to 40% of faltering in young Gambian children (Lunn 2000). Finding strategies to reduce levels of gut damage and immune stimulation may therefore be a very important strategy in tackling the global issue of childhood growth faltering. A number of interventions that aimed to do this have been suggested and I briefly outline them below.

1.4.1 Nutritional supplementation

A number of studies have suggested that micronutrient deficiencies increase the incidence and/or severity of intestinal damage and thus supplementation interventions might help to alleviate the problem. Berrant et al. (1992) found that children who suffered iron deficiency had significantly higher-than-normal intestinal permeability levels, compared to

children with adequate levels of iron. Similarly, Roy et al. (1992) found that zinc supplementation had a beneficial effect on gut integrity in children suffering from acute or persistent diarrhoea and aided their recovery.

Vitamin A is known for its anti-infective properties and for its role in the maintenance and repair of epithelia surfaces (McCullough, Northrop-Clewes et al. 1999). Thurnham et al. (2000) have suggested that it may therefore play an important role in improving gut integrity in young children. They note that 'sub-clinical infection was at its lowest, growth was least impaired and gut integrity was at its best' in Gambian infants from April to June (*ibid:*s25). These three months coincide with the mango season - a fruit rich in vitamin A – and the authors suggest that this boost of Vitamin A in the diet may help to protect the gut against damage and aid its recovery. In addition, they report on two studies conducted in India (community and hospital-based) that found gut integrity improved more rapidly in Vitamin A-supplemented children than in children acting as controls. However, a later study found that high doses of vitamin A had no beneficial effect on intestinal permeability levels and in fact the proportion of infants aged between two and seven months with abnormal intestinal permeability levels was greater in the high-dose group than the lower dose group who were given the standard amount recommended by the WHO (Darboe, Thurnham et al. 2007).

The role of supplementary n-3 polyunsaturated fatty acids (PUFA) on maintaining intestinal epithelial integrity is currently under investigation by a student at the London School of Tropical Medicine and Hygiene (van de Merwe 2006). Essential fatty acids are an important structural component of cell membranes and are also known to have significant physiological anti-inflammatory effects via modulation of inflammation and immunity reactions. Van de Merwe suggests that the high levels of intestinal permeability and growth failure seen in young Gambian children may be explained, in part, by deficiencies in PUFAs, and that dietary supplementation might ameliorate this damage by reducing gastro-intestinal inflammation. The results of a dietary PUFA supplementation intervention are currently being analysed, but no data are available to date.

1.4.2 Eradication of intestinal helminths and Giardia intestinalis

The protozoan parasite *Giardia intestinalis* is the most common water-born parasite in both the developed and developing world and is known to cause damage to the mucosa of the small intestine (Farthing 1984). It can cause morphological and functional changes in the

intestinal mucosa leading to villous atrophy and malabsorption of nutrients such as lactose, fat and vitamin A (*ibid*). It has been associated with raised intestinal permeability levels in Nepali children (0-5yrs) (Goto, Panter-Brick et al. 2002). Similarly, other intestinal parasites such as ascaris, trichuris and hook worm are also known to cause malabsorption, chronic inflammation and loss of nutrients in the host (Scrimshaw, Taylor et al. 1968). It has therefore been suggested that eradication of these pathogens could reduce levels of gut damage and improve child growth.

To test this theory a regular de-worming intervention study was carried out on 123 Bangladeshi children aged between two and five years (Northrop-Clewes, Rousham et al. 2001). No significant changes in intestinal permeability or immune-stimulation markers were observed after de-worming, and there was no difference in growth between the intervention and control children at the end of the study. The authors concluded that only when helminth infections are particularly intense would such a programme have a positive impact on child growth.

In her recent longitudinal study, Goto (2006; Goto, Mascie-Taylor et al. 2009) examined the impact of anti-giardia (Secnidazole), anti-helminthic (Albendazole) or a combination of both treatments on levels intestinal permeability, acute phase proteins and growth faltering in 298 Bangladeshi children. None of the treatment types were found to have any impact on the children's z-scores or any of the biochemical variables. The author suggests that this lack of impact may have been due to continuous re-infection with *Giardia* following treatment.

1.4.3 Prevention of diarrhoea

Given the limited success to-date of nutritional or medical interventions in reducing or preventing gut damage in young children, it is worth moving from interventions that act on the host's physiological and internal defences to interventions that focus on primary prevention – i.e. those interventions that aim to prevent or reduce exposure to the pathogens that cause mucosal damage and its associated immune stimulation. There is a huge body of literature dedicated to interventions that seek to reduce diarrhoeal morbidity in young children. As diarrhoea is merely a more extreme form of intestinal damage – severe enough to be produce clinically visible symptoms – it would seem likely that

interventions that reduce diarrhoeal disease might also result in decreases in sub-clinical forms of gut damage and immune stimulation.

There are estimated to be over a billion episodes of diarrhoea every year, and the source of virtually all of these is human or animal excrement. This is also the source of shigellosis, typhoid, cholera, other common endemic gastro-enteric infections, as well as some respiratory infections (Curtis, Scott et al. 2005:9). In just one gram of faeces there are up to 10^{12} viruses and 10 million bacteria, though not all are pathogenic (Curtis 2001). The 'F-diagram' below (Figure 1.8) was produced by Wagner and Lanoix in 1958 and outlines the ways in which faecal pathogens can pass through the environment into a new host.

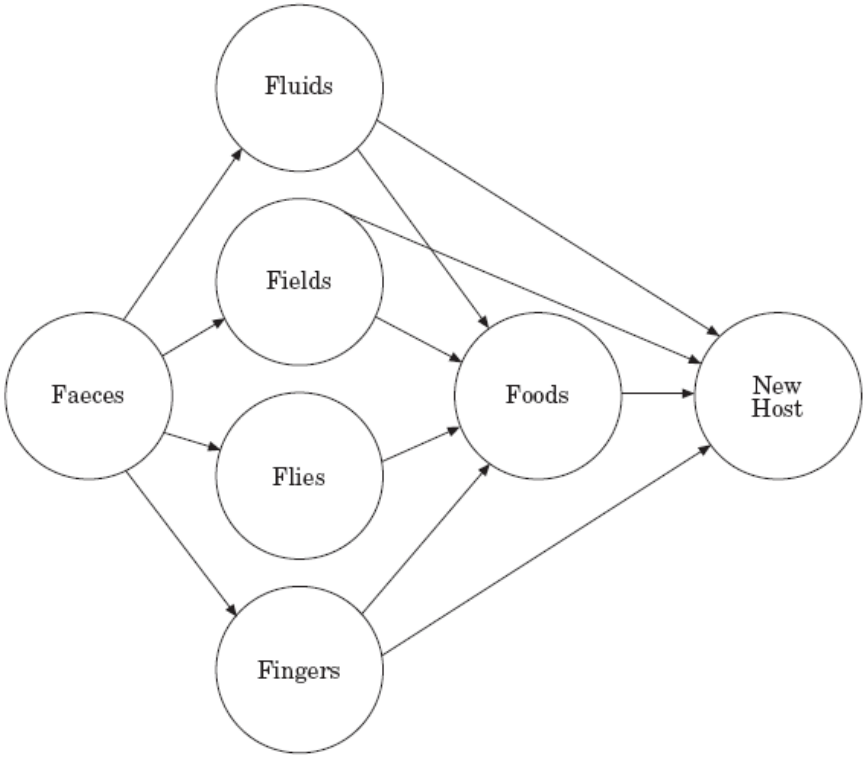


Figure 1.8 The F-diagram. Original diagram by Wagner & Lanoix (1958), reproduced from Curtis et al. (2000).

Two forms of intervention can interrupt the faecal-oral transmission route: primary barriers that prevent faeces from contaminating the environment through safe disposal of stools; and secondary barriers that prevent pathogens that have contaminated the environment from multiplying and infecting new hosts. In the following sections I outline the success of some of these primary and secondary interventions for the prevention of diarrhoea in

young children, assuming that such a reduction would also result in a reduction in sub-clinical gut damage and immune stimulation.

Improvements in water and sanitation

As implied by the F-diagram, if human and animal stools were safely disposed of so that any future contact with human hosts was impossible, the vast majority of diarrhoea would be prevented. One of the most effective ways of preventing childhood diarrhoea (and presumably also sub-clinical gut damage) would be to provide adequate sanitation and sewerage systems to every community. Esrey et al. (1991) reviewed over 140 water and sanitation intervention studies conducted throughout the world to assess their impact on morbidity and mortality rates from diarrhoea, trachoma, ascariasis, schistosomiasis, dracunculiasis and hookworm. Interventions focusing on improved sanitation resulted in a 22% reduction in diarrhoea morbidity, increasing to a 36% reduction when only the most methodologically rigorous studies were considered. Studies that combined improvements in sanitation and water supply together resulted in a median reduction in diarrhoeal morbidity of 20%, or 30% when considering only the more rigorous studies. Only one study reported the impact on diarrhoeal mortality: Habicht et al. (1988) found that provision of toilets and water supply resulted in an 82% reduction in mortality in Malaysian infants, when compared to children without access to these services.

Improvements in water supply can also have a substantial impact on diarrhoeal incidence. In the 1970s and 80s, it was assumed that improving bacteriological quality of drinking water was key in reducing diarrhoeal morbidity. Esrey et al.'s review (1991) suggested that in fact this was not the case, with the quantity, not quality, of water being far more important: improvements in water quality resulted in a 16% reduction in diarrhoeal morbidity, as opposed to a 27% reduction when water quantity alone was improved. Curtis et al. (2000:27) suggest this is because greater water supplies facilitate changes in hygiene behaviour (such as hand-washing) that interrupt pathogenic transmission; only when other sources of transmission of faecal pathogens are eliminated (by safe sanitation) would water quality become relatively more important.

Surprisingly the review conducted by Esrey et al. (1991), and a more recent review by Fewtrell et al. (2005) both suggested that multiple interventions that combined water, sanitation and educational measures in one comprehensive programme were no more effective than those interventions with a single focus. However, this finding is in contrast

to that of Esrey (1996). In this study he examined the impact of water and sanitation improvements on child diarrhoeal morbidity and growth for 16,880 children from eight developing countries using information provided by the Demographic and Health Surveys conducted at the end of the 1980s. He concluded that improvements in sanitation had a much greater effect on diarrhoeal incidence and anthropometric status than improvements in water supplied. However, when optimal water and sanitation services were provided *together*, their impact appeared to be synergistic, producing larger impacts than either intervention alone.

Improvements in water and sanitation services as a primary means of preventing contact with faecal pathogens therefore appears to be highly effective in reducing diarrhoeal (and other) disease, and would be likely to have a similar effect on sub-clinical gut damage too.

Improvements in child feeding practices

If, however, primary means of preventing faecal contamination of the environment have failed or were not present, we must rely on secondary measures to interrupt faecal pathogen transmission. Improvements in child feeding practices that minimise exposure to pathogenic organisms and prevent pathogen survival and multiplication are therefore potentially important in the prevention of diarrhoea.

Breastfeeding is considered particularly important in preventing diarrhoeal disease in young children, particularly in the developing world (de Zoysa, Rea et al. 1991; Huttly, Morris et al. 1997). Infants receiving no breast milk are at significantly greater risk of diarrhoeal disease: in their review of 35 studies, Feachem & Koblinsky (1984) found that compared to children who were exclusively breastfed, the median relative risk for diarrhoeal morbidity in children receiving no breast milk were between 3.5 to 4.9 in the first six months of life. Diarrhoeal episodes are likely to be much more severe in non-breastfed children (de Zoysa, Rea et al. 1991; Huttly, Morris et al. 1997) and correspondingly, diarrhoeal mortality rate is also significantly increased for these children: non-breastfed children were 25 times more likely to die than exclusively breastfed children during the first six months of life (Feachem and Koblinsky 1984).

The reasons for this protective effect of breastfeeding are two-fold. Firstly, breast milk has anti-infective properties that protect the infant's intestinal mucosa from damage by killing or suppressing the growth of pathogenic organisms that attempt to colonize the intestinal tract (Akre 1989:31). Secondly, by promoting exclusive breastfeeding for the first six

months of life, the child has limited exposure to pathogenic organisms that cause diarrhoea, which are commonly found in contaminated bottle milk and other weaning foods (de Zoysa, Rea et al. 1991).

Careful preparation and storage of weaning foods are probably also important in reducing diarrhoeal disease in young children. As noted by Barrell & Rowland (1979) in many parts of the world it is common to cook food in sufficient quantities for the whole day, rather than cooking afresh for each meal. In such cases, bacterial colonies in stored food can increase to dangerous levels (Esrey and Feachem 1989) and Barrell & Rowland (1979) suggest this may be a cause of infant diarrhoea, especially during the rainy season. In addition, uncovered food can be further contaminated by flies that spread pathogens via their feet, their faeces and the digestive fluids they regurgitate onto food (Curtis, Cairncross et al. 2000). It is worth noting, however, that though biologically plausible, data linking safer food hygiene practices and reduction in diarrhoeal disease are generally sparse and inconclusive (*ibid*).

Finally, boiling of water to kill pathogenic organisms has been suggested at a means of preventing diarrhoeal disease in young children and this message is frequently included in health education interventions in the developing world (Nichter 1996; McLennan 2000). However, as Gillman & Skillimore (1985) point out, this strategy is financially impossible for most people in the developing world. In their study conducted in rural villages in Bangladesh they calculated that the poorest families already spent almost a quarter (22%) of their annual income on fuel; boiling all drinking water for a year would require an 11% increase in the household budget. Even for the richest households, boiling water would require an increase in household budget of 3%. Thus, for many people living in the poorest parts of the world, this recommendation remains unachievable.

Improvements in hand-washing practices

Hand-washing is another important intervention that can interrupt the faecal-oral transmission route and can result in significant reductions in diarrhoeal (and other) disease. In many parts of the world, Asia especially, the left hand is used to clean the anus after defecation, resulting in significant contamination of the hands with faecal matter (Han, Oo et al. 1986). Even where toilet paper is used, hands can still become contaminated through several sheets of tissue paper (Hutchinson 1956). Hands can be similarly contaminated after cleaning a baby's bottom; in many countries a mother may clean her child's bottom

directly using her hand, but Sprunt et al. (1973) noted that changing soiled nappies can also result in significant contamination of the caregiver's hands.

While viruses excreted in human and animal faeces are not capable of reproducing themselves outside their host, some can survive in the environment and remain infective for some time (Curtis, Cairncross et al. 2000). Enteric bacteria, however, can multiply rapidly outside of the human host when provided with adequate warmth and nutrients (for example on hands) and can survive in the environment for long periods: in his study of a shigellosis outbreak in Southampton, Hutchinson (1956) found that *Shigella sonnei* survived on contaminated hands for at least three hours and other studies report similar findings (Pether and Gilbert 1971; Casewell and Phillips 1977). Faecally contaminated hands can therefore remain infective for several hours and enteric pathogens can easily be transferred from hands to food, environmental surfaces or directly to other people (Han et al. 1986).

Hand-washing is a very effective means of removing these enteric pathogens from contaminated hands. Feachem (1984) reviewed a number of studies that tested the effectiveness of different methods of hand-washing and reported that overall hand-washing with soap removed between 90-100% of bacteria on hands. Hand-washing with water alone removed a considerable number of bacteria, but was not as effective as hand-washing with soap. In clinical settings there has been much debate regarding the benefits of using alcohol-based hand-sanitizers, rather than washing hands with soap to prevent hospital-acquired infections. Alcohol-based solutions have been shown to be more effective in reducing bacterial contamination of hands than un-medicated soap (Kac, Podglajen et al. 2005) and anti-bacterial soap (Girou, Loyeau et al. 2002) and a recent review concluded that as well as being more effective, such hand-rubbing preparations were quicker to use and irritated skin less than hand-washing with soap (Picheansathian 2004).

However, in the majority of community settings in the developing world, alcohol-based sanitizers are not available and the vast majority of people use plain, un-medicated soap to cleanse hands, if they use any product at all. In a recent review, Aiello et al. (2007) identified four community-based randomised intervention studies that compared the effectiveness of anti-bacterial soap (containing triclosan or triclocarban) to that of plain soap (Luby, Agboatwalla et al. 2002; Larson, Lin et al. 2004; Luby, Agboatwalla et al. 2004; Luby, Agboatwalla et al. 2005). None of the studies noted any difference in

symptoms of illness between households using anti-bacterial or plain soap. While a number of non-community based studies have found anti-bacterial soaps effective in significantly reducing bacterial counts on hands, in most cases the soap used contained a relatively higher amount of triclosan (>1% wgt/vol) than that found in most consumer anti-bacterial soaps (0.1-0.45% wt/vol), and significant reductions were often only observed after multiple hand-washing episodes (Aiello, Larson et al. 2007). In addition, most of these studies tested the efficacy of soap after hand-washing episodes lasting for more than 30 seconds. As the authors note, 'it is unlikely that a ≥ 30s duration reflects the normal hand-washing practices in the community setting. Even health care professionals generally wash their hands for much shorter duration, and studies of hand washing in the community setting indicate sub-optimal hand-washing practices' (*ibid*:S144-5).

It is generally accepted therefore that in a community setting, hand-washing with ordinary, un-medicated soap is sufficient to remove the majority of pathogenic organisms from the hands and prevent disease (Curtis, Scott et al. 2005). In a classic study, Price (1938) examined the efficacy of different methods of hand cleansing and concluded that bacterial removal was not affected by type of soap used, temperature of the water, or indeed the bacteriological quality of the water. In fact, Sprunt et al. (1973) suggest that the effectiveness of hand-washing with soap is probably largely due to the abrasive rubbing action facilitated by the soap agent followed by thorough rinsing which removes organisms from the hands. As Feachem concludes, 'the effectiveness of hand-washing is determined more by its thoroughness (time taken and attention to all parts of the hands) than by the types of soap or water used.' (Feachem 1984:469).

Given its effectiveness at removing pathogens, hand-washing with soap is potentially a very effective intervention when practiced after contact with faeces (as a primary barrier) and before handling food (as a secondary barrier). Numerous studies have been conducted to examine the impact of hand-washing on reducing diarrhoeal morbidity and these studies will be reviewed in the next section.

1.5 Hand-washing with soap

1.5.1 Impact on diarrhoeal disease

The vast majority of hand-washing or hand-disinfection studies have taken place in hospitals and focused on the prevention of nosocomial infections (see Naikoba and Hayward 2001). Intervention trials that have focus on changing hand-washing behaviour in a community setting are much less common. However, there are a number of studies conducted in developing countries that have demonstrated such interventions can have a significant impact on improving hand hygiene and preventing disease in young children.

Curtis et al. (2001) conducted several qualitative and quantitative studies on childhood diarrhoea in Bobo-Dioulasso in Burkina Faso over a number of years. The findings from these studies were then used to design a large-scale community-based intervention to improve hand-washing rates amongst mothers of young children. Using a variety of methods including monthly home visits, street theatre, local discussion groups and slots on local radio stations, they promoted the message that hands should be washed with soap after contact with stools and children's faeces should be safely disposed of in latrines. The project, starting in August 1995, ran for three years and resulted in significant increases in hand-washing behaviour, though overall levels of hand-washing still remained low. The number of mothers observed to wash their hands with soap after using the latrine increased from a baseline level of just 1% to a post-intervention of level of 17%. Increases in hand-washing using water alone were more dramatic with rates rising from 33% to 67% over the period of the intervention. Hand-washing after cleaning the baby's bottom also increased from 13% to 31% for women using soap and from 35% to 74% for those using water alone. The authors conclude that well-designed and executed studies based on local practices and culture can result in significant improvements in hygiene behaviour.

Curtis et al.'s study (2001) focused simply on measuring increases in hand-washing behaviour and did not include any evaluation of the intervention's impact on reducing childhood disease. However, there are numerous other hand-washing studies that report significant reductions in diarrhoeal disease as a result of community-based interventions. Shahid et al. (1996) implemented a simple hand-washing intervention in a peri-urban village on the outskirts of Dhaka, Bangladesh and reported significant reductions in diarrhoeal disease in the intervention group. The study village was naturally separated into four distinct areas or 'paras'; two of these paras were allocated to the intervention group,

while the remaining two acted as controls. In the intervention area, each family was given half a bar of soap twice a week in addition to a pitcher to facilitate the use of water in the home. The families were encouraged to wash hands with soap after defecation or urination and before eating or handling food. Control families were not asked to change their hygiene behaviour in any way. The intervention and diarrhoeal surveillance of children and adults in both communities continued for a year. Figure 1.9 below shows the incidence density per person years for diarrhoea during the intervention period by age groups for both intervention and control areas.

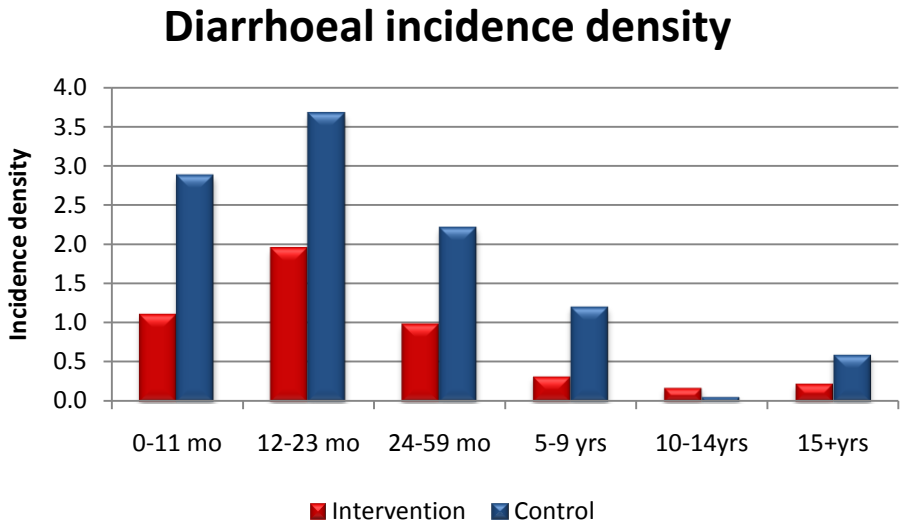


Figure 1.9 Diarrhoea incidence density over the period of study by age group. Data taken from Shahid et al. (1996).

For all age groups, people living in the hand-washing areas experienced significantly lower rates of diarrhoea over the period of study, with the magnitude of reduction ranging from 47% for children aged between 12-23 months to 73% for children aged between five and nine years. This simple, low-cost intervention focusing on improving hand-hygiene therefore appears to have been extremely effective in reducing diarrhoeal incidence in this community setting.

A number of other studies conducted in countries throughout Asia and Africa have reported similar success from hand-washing studies; these studies and their reported impact on diarrhoeal morbidity are presented in more detail in Chapter 3 and Table 3.2. A recent systematic review by Curtis and Cairncross (2003) of 17 hand-washing studies from across the world confirms the importance of hand-washing in the prevention of diarrhoeal

disease. From meta-analyses of these studies they calculate that hand-washing with soap is associated with a 42-47% decreased risk of diarrhoeal disease, and a 48-59% reduced risk for more severe outcomes.

1.5.2 Impact on other infectious diseases

Hand-washing with soap has also recently been found to be effective in reducing other prevalent childhood diseases such as impetigo and respiratory infections such as pneumonia – the other major killer of young children in the developing world. Acute respiratory infections (ARIs) kill over four million children every year and account for 30-50% of all child visits to health services and 20-40% of child admissions to hospitals (Hudelson, Huanca et al. 1995). Until now, there has been no obvious means of preventing the disease and public health interventions have focused on prompt anti-biotic treatment rather than prevention. However, there is growing evidence to suggest that a considerable number of these infections could be prevented by hand-washing with soap.

The logic behind this hypothesis is simple: pathogens that cause diarrhoeal disease can also cause respiratory symptoms. Enteric viruses are invasive and if they cause irritation to the epithelial cells in the gut, they are also likely to cause a similar reaction in the epithelial cells of the lungs and respiratory tract (Cairncross 2003). Pathogens excreted in human faeces may therefore be responsible not only for diarrhoeal episodes but respiratory infections as well. As noted above, these pathogens can survive on hands and environmental surfaces for several hours and only very small numbers are needed to cause an infection. Children can therefore easily pick up virus particles by touching objects and surfaces that have been contaminated by infected people and it follows that hand-washing could play an important role in interrupting this transmission.

To-date, most of the evidence suggesting hand-washing can reduce respiratory infections comes from developed countries. Ryan et al. (2001) report results from a simple hand-washing program implemented among new recruits at a Navy training centre in Illinois. The Commanding Officer at the training base issued a directive that recruits must wash their hands with soap at least five times a day and this was accompanied by monthly educational sessions and increased provision of liquid soap at all sinks. When rates of respiratory illness for the two years of intervention (1997-8) were compared with rates from the year immediately preceding the study, respiratory illnesses were found to have

fallen by an impressive 45%. Other studies conducted in the US and UK with adults in hospital settings (Isaacs, Dickson et al. 1991) and long-term care facilities (Makris, Morgan et al. 2000) and with children in elementary school (White, Shinder et al. 2001) and day-care centres (Niffenegger 1997; St Sauver, Khurana et al. 1998) report similar associations between increased hand-washing and reductions in respiratory infections.

Until recently no studies had considered the impact that hand-washing could have on respiratory infections in community-settings in the developing world. However, in 2005, a randomised control trial was conducted in Pakistan looking at the impact of hand-washing on diarrhoea, respiratory infections and impetigo (Luby, Agboatwalla et al. 2005). The study was conducted in 36 adjoining squatter settlements in Karachi, with 25 communities being assigned to the hand-washing intervention and 11 communities acting as controls. In the intervention areas 600 households were asked to take part in the study, with a further 306 households selected in the control areas; in both cases each household had to have at least two children under 15 years of age. Households in the intervention area were provided with a regular supply of soap and educational and community activities took place to promote the importance of hand-washing after contact with faeces or before handling food. The intervention ran for one year and was accompanied by the collection of weekly morbidity reports. The intervention appeared to be highly successful in reducing risk of respiratory disease in young children: children under five years of age in the intervention areas had a 50% lower incidence of pneumonia over the study period when compared to controls (95% CI -65% to -34%). The intervention also had a significant impact on diarrhoeal disease and impetigo: children under 15 years of age living in the hand-washing areas had a 53% lower incidence of diarrhoea (95% CI -65% to -41%) and a 34% lower incidence of impetigo (95% CI -52% to -16%) than their control counterparts.

A recent review of eight hand-washing interventions revealed risk reductions for ARIs of between 6-44% (Rabie and Curtis 2006). From meta-analyses they calculated a relative risk of 1.19, indicating that hand-washing can cut the risk of respiratory infection by 16%. All of these studies were conducted in developed countries, yet the findings from Luby et al.'s (2005) study suggest that this simple behaviour change intervention could potentially play an important role in reducing respiratory infections in the poorest countries as well.

1.6 Research question of present study

Hand-washing with soap appears to be a simple, low-cost intervention that has the potential to reduce diarrhoeal disease by almost 50% and respiratory infections by a lesser, but still significant amount. Its impact on these two leading killers of young children therefore makes it an extremely important public health intervention. What remains unknown, however, is the impact it may have on sub-clinical levels of infection. Does hand-washing with soap reduce levels of gut damage and immune stimulation in young children and therefore potentially also reduce growth faltering?

1.6.1 Aims and objectives of the study

This project aimed to assess the impact of a hand-washing with soap campaign on levels of reported morbidity, gut damage, immune stimulation and growth in young children living in the slums of Kathmandu. Previous work in Nepal (of which I was project manager) had already established a link between intestinal damage and growth faltering with both middle-class and slum children in Kathmandu, with the highest levels of gut damage, immune stimulation and growth faltering observed in the slum children. This current project therefore focused exclusively on slum children as the study population.

The four specific objectives of the project were to:

1. Collate information on socioeconomic status, childcare and hygiene practices in families of young children living in slum settlements in Kathmandu, using pre-tested questionnaires, structured observations of behaviour, semi-structured interviews and focus groups.
2. Design a culturally relevant community-based hygiene intervention aimed at improving hand-washing practice at five key junctures: after going to the toilet or cleaning the baby's bottom, and before cooking, eating or feeding the baby.
3. Implement the hand-washing campaign in intervention areas for six months, with other slum communities acting as controls.
4. Assess the intervention's impact through comparisons of the health and growth status of children living in the intervention and control areas.

The impact of the intervention assessed changes in the following specific outcome variables:

- *Hand-washing behaviour:* as reported by mothers at the start and end of the intervention.
- *Soap usage:* as reported by mothers on a weekly basis.
- *Child morbidity*: as reported by mothers, focusing on symptoms of diarrhoea, colds and fevers.
- *Gut damage:* as measured by a urine sample analysed for lactose:creatinine ratio (L:C).
- *Immune stimulation*: as measured by finger-prick blood-drop samples analysed for levels of α-1-acid glycoprotein (AGP) and immunoglobulin G (IgG).
- *Biochemical nutritional status:* as measured by blood-drop samples analysed for albumin and haemoglobin (Hb).
- *Growth faltering*: as measure by height-for-age, weight-for-age and weight-for-height z-scores.

1.6.2 Specific Hypotheses

The project was based upon a theoretical model linking pathogen exposure, morbidity, sub-clinical infection and growth faltering, depicted in Figure 1.10. The model suggests that, as a result of the hand-washing intervention, mothers in the intervention group would increase their hand-washing behaviour at the five key junctures mentioned in the intervention message (Level I). As a result of this change in hygiene behaviour, children in the intervention group would have lower exposure to pathogens, resulting in a reduction in clinical morbidity (mother-reported episodes of diarrhoea, colds and fevers), when compared to their control counterparts (Level II). Reduced exposure to pathogens would also result in less sub-clinical infection in children from the intervention areas, as measured by levels of gut damage and immune stimulation (Level III). Finally, the reduction in both clinical and sub-clinical infection would result in children living in intervention areas experiencing lower levels of growth faltering, as compared to children from control areas (Level IV). The specific hypotheses for this study are outlined below.

By the end of the intervention period:

Level I:

- Mothers in intervention areas will report washing their hands with soap at the five key junctures more frequently than their counterparts in control areas.
- Mothers in intervention areas will report using a greater number of bars of soap per month than their counterparts in control areas.

Level II:

- Children in intervention areas will experience fewer episodes and fewer days of sickness (diarrhoea, colds and fevers) than their counterparts in control areas.

Level III:

- Children in intervention areas will experience less intestinal damage than their counterparts in control areas, as measured by the lactose:creatinine urine test.
- Children in intervention areas will have lower levels of immune stimulation than their counterparts in control areas, as measured by levels of AGP and IgG in their blood.
- Children in intervention areas will have better biochemical nutritional status than their counterparts in control areas, as measured by levels of albumin and haemoglobin in their blood.

Level IV:

- Children in intervention areas will have better growth status than their counterparts in control areas, as measured by height-for-age, weight-for-age and weight-for-height z-scores.

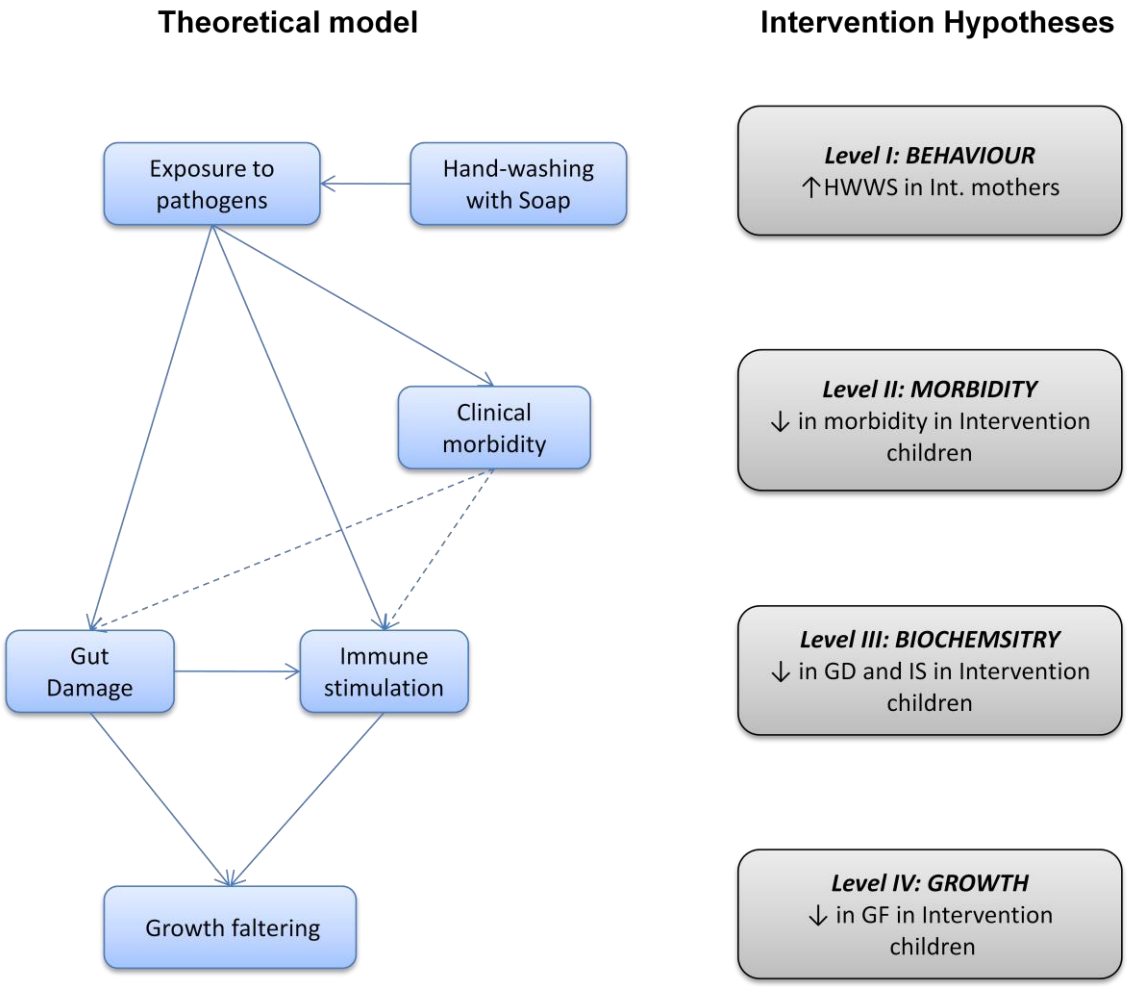


Figure 1.10 Theoretical model and hypotheses regarding the impact of the hand-washing intervention.

Summary

This chapter identified the problem of childhood growth faltering as a significant global health problem. It discussed the relationship between nutrition and infection in causing growth faltering in young children, and also highlighted the importance of sub-clinical infections. The theory linking intestinal damage and stimulation of the immune system to growth faltering was presented and methods by which these could be prevented were discussed. Hand-washing with soap was presented as a potentially very effective means of reducing sub-clinical infections and the aim and hypotheses of this particular study were detailed. The following chapter will present details of the study setting, the methods employed during the study and the statistical methods used to analyse the data.

CHAPTER 2
Project design and methods

Introduction

This chapter starts by describing the study setting – the slums of Kathmandu. It outlines the rapid increase in the slum population of Kathmandu over recent years and goes on to describe the living conditions experienced in these settlements. It continues by outlining the project design and the four phases of the research: preparation, design, implementation and evaluation. The design and implementation of the research instruments is then discussed. Finally, the management and analysis of the data is presented.

2.1 Study setting: Kathmandu, Nepal

Bordered to the south and north by two of the fastest growing economies – India and China – Nepal remains one of the poorest and least developed countries in the world; its Gross Domestic Product (GDP) per capita is just $1000, ranking it 212th out of 229 countries (Central Intelligence Agency 2009). Using the recently recalibrated global poverty lines, the World Bank estimates that over half (55%) of Nepal's 28.5 million citizens live on less than $1.25 per day and over three-quarters (78%) survive on less than $2 per day (World Bank 2008).

Nepal is one of the least urbanised countries in the world; 86% of the population live in rural areas, with agricultural activities accounting for almost half of the country's GDP (Sengupta and Sharma 2006). Yet paradoxically, Nepal is also one of the world's most rapidly urbanising countries (Pradhan 2004). In the second half of the last century, Nepal's total population almost tripled from 8m to 23m; the rate of urban expansion, however, was almost five times as fast, with the number of people living in cities increasing from less than 250,000 to over 3.2 million in just five decades (Figure 2.1). Although this urban expansion has occurred in a number of cities, Kathmandu has increased most rapidly, with its annual rate of population increase being the highest in all Asian cities for this time period (Asian Development Bank 2001).

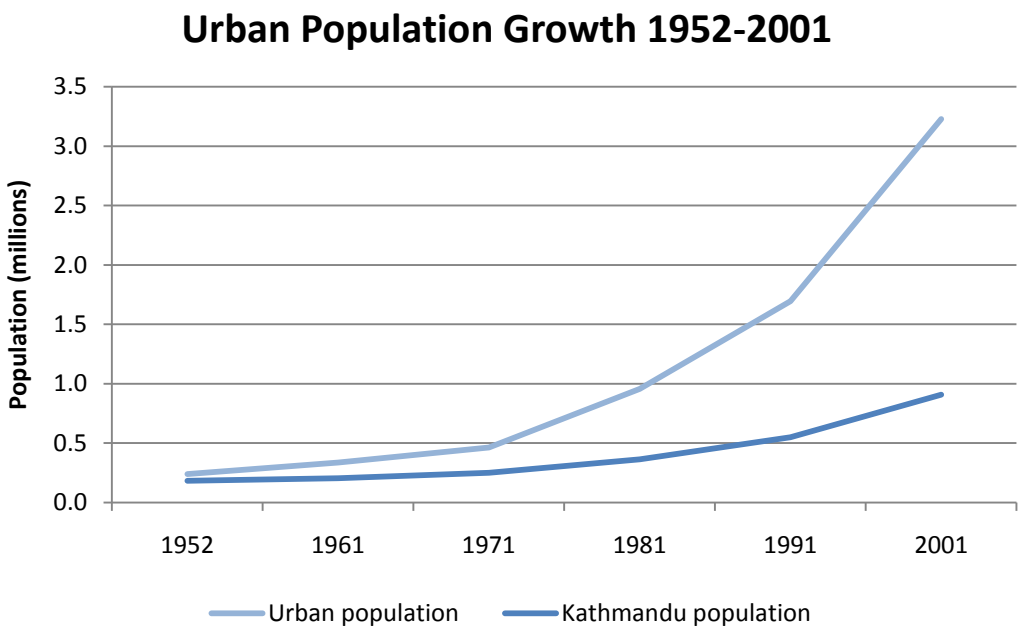


Figure 2.1 Population growth between 1952-2001 for all urban areas and Kathmandu. Taken from Sengupta and Sharma (2006), quoting Pradhan (2004).

Many of the earliest migrants to the urban centres were the poorest families pushed out from rural areas due to a combination of natural disasters and deforestation, shortages in inheritable land and poverty. More recently, the economic and social benefits of city life have drawn in many more migrants seeking better paid and more secure employment, as well as numerous other benefits such as access to education and healthcare facilities (Tanaka 2009). The recent Maoist uprising and ensuing civil war which raged for over a decade and killed more than 12,000 people also 'triggered a massive exodus from remote rural areas to urban centres to escape…violence and extortion' (Sengupta and Sharma 2006:109). Although there are no exact figures as yet, it is estimated that between 50-70,000 people fled their homes to escape violence and intimidation from both Maoist rebels and the Nepali army during the insurgency (Internal Displacement Monitoring Centre 2008). Since the peace treaty signed in 2006, few of these displaced people have returned to their natal villages, and the rate of urban migration does not appear to have fallen since hostilities have ceased (Tanaka 2009).

2.1.1 The growth of squatter and slum settlements in Kathmandu

As in many other parts of the developing world (Ooi and Phua 2007), the rapidly increasing urban population of Nepal – and more specifically, Kathmandu – has massively

outstripped the ability of local authorities to provide affordable housing, basic services and health infrastructure (Sengupta and Sharma 2006). In an already over-crowded city, spiralling land and construction costs mean that many new migrants cannot afford their own home, and rental costs in the city have also escalated dramatically in recent years (Gallagher 1992). Consequently, many of the poorest families moving into the city have been forced to take up occupancy in the rapidly growing slum and squatter settlements in Kathmandu.

Although some of the squatter settlements within the capital city are now well-established, with the oldest dating back about 60 years, there has been a dramatic rise in both the number of settlements and their total population over the past few decades. In 1985 there were just 17 squatter settlements within the city; by 2003 this number had almost quadrupled to 63 settlements and the population had increased seven-fold to 15,000 (Sengupta and Sharma 2006). Combined with those squatting in public buildings rather than in camps themselves, the total squatter population of Kathmandu is estimated to be just under 20,000 (Figure 2.2). In addition to the squatters, there are also many thousands more living in slum dwellings throughout the city, though there are currently no accurate figures as to the total number of residents occupying slum dwellings.

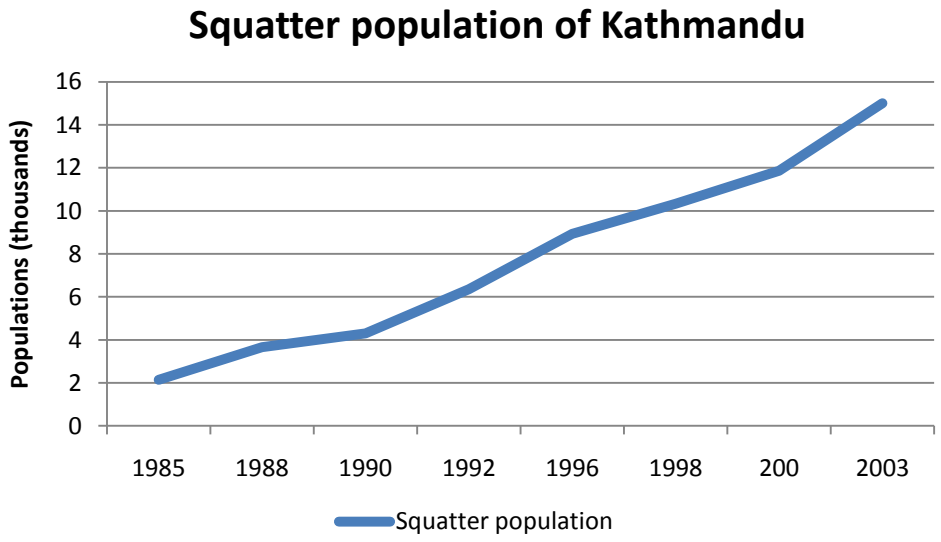


Figure 2.2 Population of people living in squatter settlements in Kathmandu between 1985-2003. Taken from Sengupta & Sharma, (2006).

It is worth at this point clarifying what is meant by the terms ‘slum’ and ‘squatter’. In the context of Nepal, a distinction is made between these two types of settlement based upon

their legal status. The term ‘slum’ is a catch-all word used to describe communities experiencing significant physical and social deprivation (described in more detail below). However, though squatter settlements fall into this general category of ‘slum’, they are also distinct in that, unlike other slum areas, they are *illegal* settlements: residents occupy unused, marginal government land but possess no legal right or entitlement to the land on which they live and thus could potentially face eviction by the local authorities at any time (Lumanti 2001; Shrestha and Shrestha 2005). However, although the Nepali government has occasionally carried out forced evictions and demolitions of squatter settlements in the past, many of the city’s squatter camps have now existed for several decades and are well-established and tolerated by the local community and government alike. For these larger and older settlements, their legal status is less of an issue in terms of the threat of eviction, than in the inability to access municipal government services (such as electricity, water and sanitation) and political representation. Although squatter communities face the additional problems of their illegal status, in terms of environmental quality, level of deprivation and access to basic services there is little difference between the illegal squatter settlements and legal slum communities. For the purposes of simplification, hereafter I refer to both the legal and illegal settlements using the generic term ‘slum’.

2.1.2 Description of Kathmandu’s slums

The term ‘slum’ is a complex and multidimensional concept that includes not only the physical characteristics of a settlement (such as over-crowding or poor quality housing), but also legal and social dimensions as well. As such, it is difficult to produce a clear definition of what is meant by the term. Numerous definitions have been employed by national and local governments, aid agencies and non-governmental organisations (NGOs). Whilst none of these definitions is exactly the same, there are some common characteristics of the slums that are generally agreed upon (for example, see Fry, Cousens et al. 2002; UN-Habitat 2003; Sclar, Garau et al. 2005; WHO 2005; Ooi and Phua 2007; Vlahov, Fruedenberg et al. 2007). Below I present a brief description of the slums of Kathmandu referring to these criteria. First, however, I offer a short vignette based on actual sights and quotes from some of the people I met while working in the slums, in order to give a ‘thick description’ of the field work setting.

Box 2.1 A walk through the slums

Apsana, my field assistant, and I walk onto the worryingly rickety metal bridge that traverses the Bagmati river and leads to the settlement. Halfway over, I stop for a moment and peer over the side into the river beneath. The last time I was in Nepal a friend took me out of the city and up to Sundharijul so that I could see the Bagmati River as it enters the valley. Up there, the Bagmati is a glorious river that sparkles as it rushes over the rocks and pebbles down the hills and into the valley. Looking down at the murky waters below it seems hard to imagine this is the same river. Now, at the height of the dry season and having travelled through half of the city already, it has been reduced to a pathetic brown stream that seems to ooze, rather than flow, past us, accompanied by an almost overpowering stench of sewage. The exposed banks are littered with all kinds of rubbish and small islands of plastic debris have formed at various intervals along its route.

This river marks the lower boundary of slum settlement, with houses balancing precariously on the edges of the steep slopes that run down to these fetid waters. Beyond this first row of houses, the settlement sprawls out, away from the river in a crazy and haphazard way. From this angle it is impossible to see the narrow alleyways that cross-cut the entire settlement – all you can see is a mass of corrugated iron roofs and plastic sheeting weighed down with heavy rocks here and there. Having crossed the bridge, we turn down one of these narrow paths and enter the settlement. I've walked through this area a number of times now, visiting the mothers, and yet I still seem to get hopelessly lost every time; the paths wind and twist and I lose my bearings almost immediately. Some of the paths are quite wide, others are barely more than half a metre across and at times we find ourselves squeezing through tiny gaps between two rows of houses, turning on our sides and walking with a sort of awkward, shuffling side-step.

Wandering these paths inbetween the ramshackle and impossibly small houses we eventually end up at the far edge of the settlement again where the houses meet the river banks. There are several toilets situated right on the edge of the steep banks – tiny shacks with a wooden door and flapping, torn plastic sheeting forming the walls. These toilets consist of nothing more than a couple of bricks where you place your feet and a long plastic tube which drains off the waste directly into the stinking river below. A little further

up we see a group of women gathered around the communal rower pump – a small clearing in the midst of these sprawling houses. One woman is bathing – a wet lungi[2] *pulled up over her chest and her thick, dark hair filled with soap suds. Another woman, squatting on her haunches, draws up water from the rower pump and sets about scouring her metal pots, cups and plates with gritty, green dish soap.*

Behind them, in the doorway to one of the dark houses sit two women – one younger, one older. The younger one – a girl aged 19 years, though she looks much younger – holds a tiny baby in her arms, swaddled in many layers of cloth. This is her first child – a son – born just two weeks ago. He looks a strong child, we tell her, being careful not to compliment him too much for fear of attracting the evil eye.

We continue on and are spotted by Deepa Subba – one of the mothers enrolled in our programme – who calls us to come have tea with her. Entering her house, we both have to stoop low to get through the doorway. I'm not even sure you could really call it a house – it's simply a tiny part of a large, sprawling area covered by corrugated iron sheets supported at intervals by wooden struts. Each 'household' is separated from its neighbours by perhaps another few sheets of iron if they're lucky or, more often, just a thin layer of plastic sheeting. You can hear every word, every sound from the other families that surround this small home.

There's practically nothing in this house - a bed where Deepa and her husband sleep and a cot for their young son, a small table, a kerosene stove and some cooking utensils set neatly in the corner. That's it, nothing else.

It's swelteringly hot in here and dark too, as the only light that enters is from the open doorway. I ask Deepa about living here in this house and she sighs,

> *'It's too hot in the summer, too cold in the winter and the roof leaks during the monsoon. Last year we had to move our bed several times to keep the water from dripping onto it all the time.'*

She continues,

> *'But at least we don't live right next to the river though. Oh! The smell, the flies! It smells bad enough up here, but further down it's even worse, especially in the dry season. And then when the monsoon comes the river rises and often floods those houses. You've seen what it's like down there – that water is filthy*

[2] lungi – cloth garment usually worn around the waist like a skirt.

– that's where all our toilets flow into after all.'

We drink our tea, which is thick and sweet. Eventually we leave to meet other women from our study. Every now and again I see things that jar me, that seem so out of place in the slums. Most women here wear simple clothes – cotton saris, lungis, kurta suruwal[3]. For the most part their clothes look old and worn, but clean. But then suddenly, from one of the dark doorways, emerges a young teenage girl wearing tight jeans and a short, tight top. Donning big sun glasses, teetering along in impossibly high-heeled sandals and with her straight, dark hair flowing behind her she looks like she has just stepped off the set of a Bollywood movie.

When our work is done, we leave the settlement and walk up the hill to catch a bus on the ring-road that skirts the city. As I ride home I get chatting to the guy sitting next to me. He's surprised and then delighted to find that I speak Nepali and he asks me what I am doing here. I explain that I'm a student and I'm working with women and children who live in the slums. He frowns at me and then says,

'Ah! Those sukumbasi[4]! They're all liars you know – none of them are actually landless. They've all got homes and land back in the villages. They just want to come here to get a good job but are too lazy to pay proper rent somewhere. We all have to pay rent, why shouldn't they? The government should throw them all out!'

Substandard housing and overcrowding

As described in the above vignette, the housing standards in Kathmandu's slums are generally very poor, though quality can vary greatly both within and between settlements. The quality of housing structures and building materials tend to be closely related to the age of the slum settlement: upon moving into the slum new residents quickly erect flimsy, non-permanent structures made from a patchwork of scrounged materials such as scrap metal, plastic sheeting, bamboo and brick. Over the years, families gradually improve their homes, such that the older settlements in the city now largely comprise of permanent (though often poorly constructed and dilapidated) brick buildings. However, even these brick constructions can fail to provide adequate protection against the elements; often

[3] Kurta suruwal – traditional Nepali dress of trousers and tunic.
[4] Sukumbasi – pejorative term for the slums, literally meaning 'landless'.

houses are poorly constructed, letting in the cold during the winter and rain and flood water during the monsoon. Inside the houses are often dark, damp and poorly ventilated. The settlements are also very over-crowded, often with entire families crowded into a single room, used for cooking, sleeping and living. Such overcrowding has significant implications for health as it allows for the rapid spread of infectious diseases throughout entire communities.

Poor infrastructure and access to basic services

Kathmandu's slums are also characterised by a lack of access to, or very poor quality, basic services. The majority of households in the slums now have an electricity connection, though in most cases the connection, wiring and meters required were paid for by the local community themselves, rather than being provided by the government. For most slum dwellers, access to water and sanitation services is a much greater problem.

The most pressing issue for the majority of residents in the slums is access to safe drinking water. In three of the eight settlements I worked in during this project, there was a government-provided water supply. This usually took the form of public water taps supplying drinking water from the Nepal Water Supply Corporation (NWSC). However, despite this government-provided facility, access to drinking water was still very limited; water was often available at these taps for just one or two hours on alternate days. As a result, residents would have to wait in long queues in order to collect water, and were often unable to collect quantities sufficient for their families' needs. In this case, women and children would walk to public taps which had better supplies in communities located some distance away. For the five communities who had no access to government water supplies, most residents either collected water on foot from another area, or used water from public or privately-owned tube wells and deep wells. Although in some areas tube well water was perceived by local residents to be of sufficient quality for drinking, many women complained that this water was not good to drink as it tasted bad, was yellowish in colour and contained a lot of grit and sediment.

Lack of access to drinking water was cited as a major problem in every slum community I worked in. However, water used for other domestic purposes (such as washing, cooking and hygiene practices) was generally always available from tube wells and deep wells or, in some cases, from near-by rivers and streams. Though water levels diminished during the

height of the dry season (March-May), residents reported that water was almost always available from these sources.

Over the past decade, aid from local NGOs and the actions of private landlords in the slums have dramatically improved the sanitary facilities in the slums. In the past, most of the slum residents had no access to sanitation at all and simply defecated on the riverbanks. Now, however, most houses in the slums have access to a toilet. Often a house will consist of multiple households each renting a single room from the house owner; thus a single toilet may be used by a large number of people and the cleanliness and upkeep of these facilities can vary dramatically. In some areas, these toilets drain into septic tanks which are then emptied by a private company on a regular basis. More commonly, the toilets drain directly into a nearby stream. Many slum residents therefore find themselves living on the banks of fetid, open sewers producing an overpowering stench and swarms of flies during the hot, summer months and a risk of sewage-contaminated flood waters during the monsoon.

Poverty and social exclusion

Slums are the most visible 'physical and spatial manifestation of urban poverty and intra-city inequality' characterised by concentrated areas of poverty, social exclusion and deprivation (UN-Habitat 2003:xxvi). Although there can be much heterogeneity within settlements, the majority of slum residents can be classified as poor or low income. Unemployment or under-employment rates are high and those who are employed are often engaged in poorly paid and insecure work (Lumanti 2001). Many slum residents are from socially deprived groups such as the low-caste *dalit* [untouchable caste] community or people internally displaced from their natal homes by natural disasters, violence and war. There is also a very high proportion of people living in rented accommodation in slum areas. The illegal status of the squatter settlements, in particular, can lead to intimidation and abuse from neighbouring communities and property developers and the local term for squatter settlements – *sukumbasi* – is often used in a highly pejorative way. Living in a squatter or slum community is therefore often associated with considerable shame and social stigma.

2.2 Project Design

This project employed a mixed-method, longitudinal design to investigate the impact of a community-based hand-washing with soap intervention on the health and growth of young children living in deprived areas of Kathmandu, Nepal.

The project consisted of four distinct stages, as outlined in Figure 2.3. In the preparatory stage, structured observations were conducted in mothers' homes to identify current hand-washing practices. In-depth interviews and focus groups were conducted with mothers to investigate local perceptions of child health and hygiene. Baseline data on demography, socio-economic status and hygiene, feeding and child-care practices were collected for every family.

The second stage concerned the design of the intervention programme. In each intervention area, a well-respected local woman was selected to act as a Community Motivator (CM) to help implement the intervention. Interview and focus group data were analysed by members of the research team (two research assistants, five Community Motivators and myself) to identify the most compelling way to couch and promote the hand-washing message.

The third stage commenced with a baseline health check of all children in the intervention and control groups, assessing the children's levels of morbidity, gut damage (urine sample), immune stimulation (blood-drop sample) and growth status. This was followed by the launch of the hand-washing programme in the intervention areas only. The importance of hand-washing with soap at five key junctures (after going to the toilet or cleaning a baby's bottom and before cooking, eating or feeding the baby) was promoted through educational sessions, posters and songs. Adoption of this practice was encouraged through the provision of soap to each household, daily home visits by the Community Motivators and mothers' meetings. These activities continued for six months.

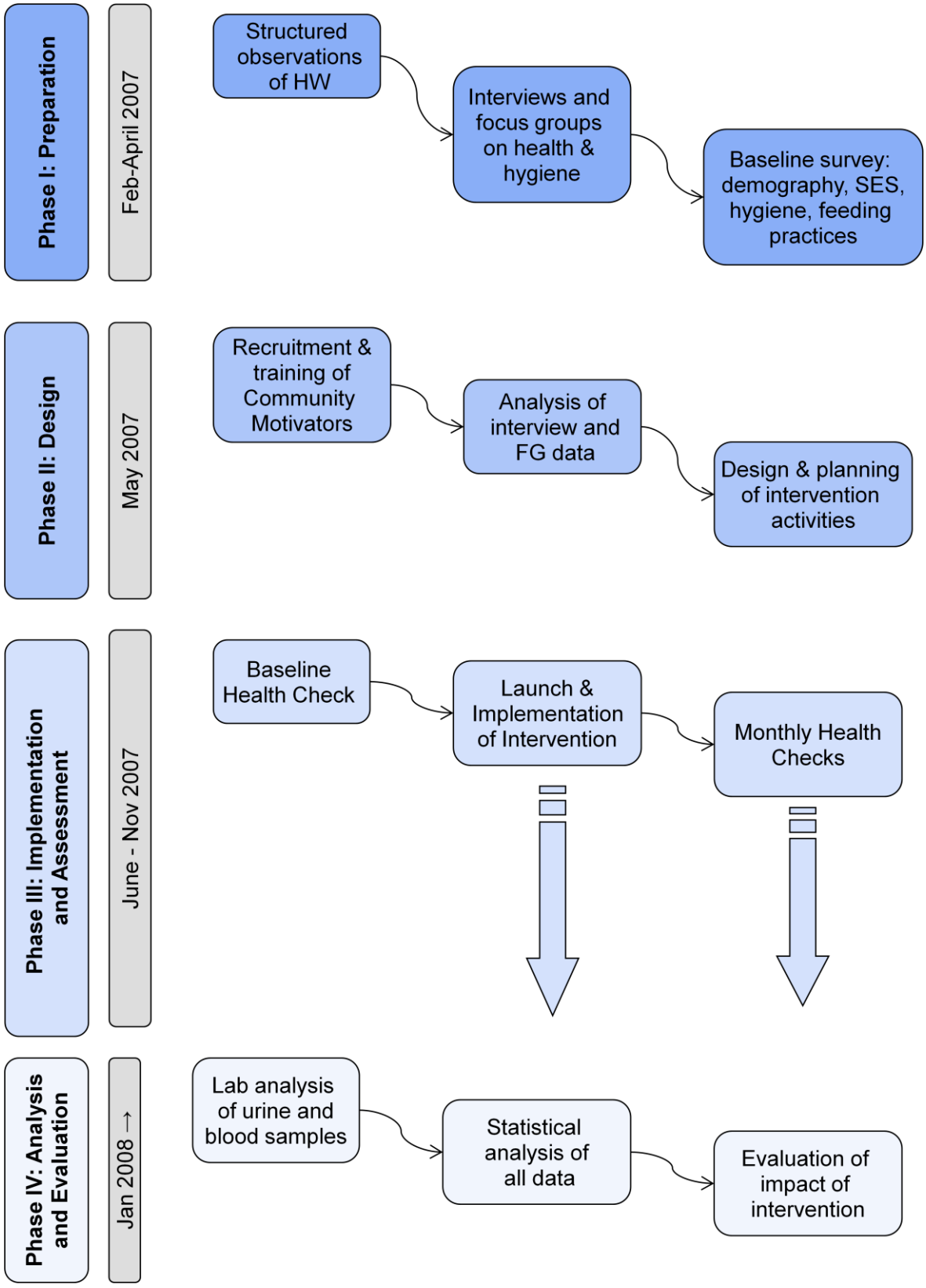


Figure 2.3 Outline of the four stages of the project and the activities undertaken.

The impact of the intervention was assessed through monthly health checks on all children and weekly reports on child morbidity and family soap usage. Towards the end of the intervention period, further in-depth interviews were conducted with mothers from both intervention and control groups whose children had been identified as growing poorly or well, in order to elucidate factors that may have influenced their child's health. The hygiene questionnaire was also administered for a second time, to assess changes in reported hand-washing practices. The final stage involved the analysis of the biological samples in the UK and statistical comparisons of levels of morbidity, gut damage, immune stimulation and growth between the two groups to assess the intervention's impact.

2.3 Study sample

2.3.1 Sample population

This study was based on a sample of breast-fed children living in the slum settlements of Kathmandu, who were aged 3-12 months on the 1st June 2007. As described in Chapter 1, a previous study conducted in 2005 confirmed children of this age range living in these poor urban communities experienced high rates of morbidity, gut damage, immune stimulation and growth faltering (Panter-Brick, Lunn et al. 2009).

2.3.2 Sample size

Data from the 2005 study (Panter-Brick, Lunn et al. 2009) were used to calculate the required sample size, using the following formula, based on a test with 80% power, a 95% level of significance and a 30% reduction in levels of gut damage, immune stimulation and growth faltering (Table 2.1). For the latter, only WAZ was used as the 2005 study found no significant relationship between gut damage and HAZ or WHZ in slum children (*ibid*).

$$n = \frac{2\sigma^2 [z_{1-\alpha} + z_{1-\beta}]}{(\mu_1 - \mu_2)^2}$$

[n = sample size, σ = standard deviation, $[z_{1-\alpha} + z_{1-\beta}] = 7.85$ based on a test with 80% power and a 95% level of significance]

Health Markers	Original values from 2005 study (*n*=48)		Expected value after intervention (30% reduction)	Required sample size
	μ_1	σ	μ_2	*n*
Intestinal Damage:				
Lactose:Creatinine	0.16	0.08	0.11	88
Immune stimulation:				
AGP g/L	0.63	0.12	0.44	12
IgG g/L	7.53	1.74	5.27	18
Growth:				
WAZ	-2.02	0.99	-1.38	84

Table 2.1 Required sample size for growth and biochemical variables, based on data from Panter-Brick et al. (2009).

A total of 88 children (44 per group) were required in order to detect significant changes in biochemical and growth status as a result of the intervention. As slum communities tend to be fairly transitory in nature the target sample size was increased to 100 children to mitigate the effect of attrition.

2.3.3 Sampling strategy

For logistical reasons the number of field sites needed to be kept as low as possible; therefore only slum settlements with the largest populations were selected. However, a number of the largest sites were excluded for several reasons: inaccessibility in three cases; unwillingness amongst community leaders to participate in one case; or incomparability with other sites due to recent NGO work that had dramatically improved access to water facilities (Table 2.2).

House-to-house surveys were conducted by a research assistant and a local woman in each selected area to determine the number of children in the target age-range. All eligible children were invited to participate in the study. Thus, the sampling strategy consisted of a purposive sampling of the largest slum settlements in Kathmandu, followed by a total sample of children in the target age-range from these communities.

Area	HHs with no water connection	No. of Households	Total population	Comments
Palpakot	96%	262	1068	Selected
Pathibhara	unknown	unknown	1000	Selected
Ramhiti	100%	114	684	Selected
Jhagriti Tol	99%	124	634	Selected
Khadipakha	100%	134	630	Selected
Sinarnangal	100%	111	522	Selected
Sankhamul	100%	101	459	Selected
Shanti Nagar-A	100%	135	603	Selected
Shanti Nagar–C	98%	101	453	Selected
Shanti Nagar –B	100%	78	332	Selected
Bansinghat	100%	98	459	Declined
Dyola Galli	27%	60	394	Excluded
Dhoukel	50%	64	343	Excluded
Jadibuti-A	100%	257	909	Inaccessible
Koteshwor-D	100%	125	455	Inaccessible
Palpakot-A	100%	122	424	Inaccessible
Pashupati Nagar Marg	91%	80	359	Not found
Bhim Muktoshwor	100%	82	407	Too few children

Table 2.2 Slum population data taken from Shrestha and Shrestha (2005).

2.3.4 Allocation to intervention and control groups

Originally, each community was to be randomly allocated to either intervention or control group. However, many of the slum sites in the south-east of the city were very close together – sometimes just separated by a mud road or small stream. As these neighbouring communities were well acquainted with each other, the control areas could easily be 'contaminated' by talking to friends living in the intervention areas.

The communities were therefore grouped into two geographical areas. The first group comprised of the five communities in the south-east of the city. The second group comprised of the sites to the north-east of the city. In addition, one extra site was added to this group to equalise numbers; though this site was close to the south-east group, it was sufficiently distant (15 minutes by car) that the possibility of 'contamination' was very low. These two geographical groups were then randomly allocated (by flipping a coin) to control or intervention groups. Table 2.3 shows the distribution of children across the field sites for the 88 children in the final dataset and Figure 2.4 shows the geographical location of the sites.

NORTH-EAST: CONTROL		SOUTH-EAST: INTERVENTION	
Area	N Children	Area	N Children
Jhagriti Tol	9	Shanti-Nagar A, B, C	29
Khadiparka	12	Sinamangal	6
Pathibhara	9	Palpakot	10
Ramhiti	6		
Sankhamul	7		
TOTAL	**43**	**TOTAL**	**45**

Table 2.3 Recruited children from intervention and control sites.

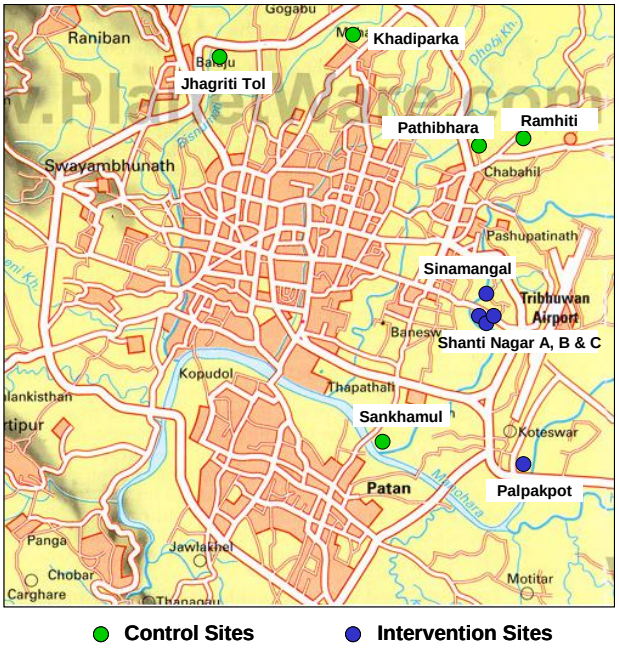


Figure 2.4 Map showing location of field sites in Kathmandu, Nepal.

2.4 Research Instruments

A mixture of qualitative and quantitative methods was used to design, implement and evaluate the impact of the intervention on the health and growth of the children.

2.4.1 Structured observations of hand-washing behaviour

Baseline data were collected on hand-washing practices in both intervention and control areas through structured observations – the recommended 'gold standard' method for assessing hand-washing behaviour (Curtis, pers. comm.) The observation schedule employed was one developed by an international hygiene intervention programme, which

was then specifically adapted for use in this context (The Hand-Washing Handbook: Curtis, Scott et al. 2005) (Appendix 1). It focused on identifying hand-washing behaviour before or after certain key events, such as defecation, cleaning the baby's bottom, cooking food or feeding the baby. In each instance, whether hand-washing occurred was recorded, noting whether or not soap was used.

Instrument design

My research assistant and I initially trialled the observation schedule in local slum households, with further modifications carried out during the fieldworker training process. In each area a young local woman was selected to undertake the structured observations. These fieldworkers underwent a week of training which included practice observations carried out in one of the centrally located field-sites in households that had a child less than two years of age but were not enrolled in the study[5]. In total, five days of trial observations were carried out, with fieldworkers working in rotating pairs each day to ensure a consistent approach across the whole team. After each observation session, I met with all the fieldworkers to discuss any problems they had encountered, clarifying and (where necessary) modifying the schedule. Each pairs' results were also checked for consistency, and where discrepancies arose the reasons for this were discussed and resolved. This practice proved invaluable for modifying the observation schedule and ensuring consistency and accuracy across the fieldworkers. The final schedule was back-translated into English to check accuracy and consistency of meaning.

Data collection

Due to time constraints, observations could not be undertaken in all households; instead a random sample of two-thirds of recruited households was selected, namely 75 of the original 109 households recruited (41 intervention, 34 control).

Fieldworkers were instructed to visit the selected mother the day before and seek her consent to undertake the observation the following morning. To avoid 'reactivity' the mothers were simply told that these observations were to learn more about the life and work of Nepali women; hand-washing was never mentioned. If the mother agreed, the fieldworker arrived at the house the following morning as soon as the mother woke up

[5] As all eligible children (<12 mo) from this area had been enrolled, in practice this meant that trial observations were carried out in household of children aged between 12-24 months**.**

(usually about 7am), and spent three hours observing her (and her family's) behaviour. This morning period was chosen as most of the activities of interest (such as personal hygiene, cooking and eating) took place during this period.

The mother was asked to go about her normal morning routine. Fieldworkers were instructed to sit quietly in the house during the observation period, and to engage in conversation with the family as little as possible. In compensation for the inconvenience of these observations, the mothers were offered 100Rs (approximately 80p) as a token of thanks. Completed forms were collected and checked for missing data. The coded results were then entered into an Excel file, and later converted into an SPSS file.

2.4.2 In-depth interviews and focus groups

A number of semi-structured interviews and focus groups were conducted with mothers enrolled in the programme. During Phase I, these interviews and focus groups were used to investigate local perceptions and understandings of hygiene and health/disease in order to inform a) the intervention message and b) the design of the morbidity reports. At the end of the intervention period, further in-depth interviews were conducted with mothers from both intervention and control areas whose children had been identified as growing poorly or well, in order to elucidate factors that may have influenced their child's health.

I conducted all interviews in Nepali, using a translator where necessary to clarify points. In order to make the interviews less formal, they were conducted in the mothers' own homes, which had the advantage of allowing informal observations to be carried out at the same time. Notes were taken during the interview and were written up into comprehensive field notes as soon as possible on the same day.

Three focus groups, concentrating on local perceptions of hygiene and cleanliness, were conducted in the intervention areas. For each focus group, between six and eight mothers were randomly selected and invited to attend. Due to the linguistic demands of this method, moderation of the focus groups was conducted by a research assistant specifically trained for this task. Specially designed 'flash cards' were created depicting scenes where hand-washing might occur. These were used with the mothers to stimulate discussion and identify when hand-washing occurred, how hands were washed (with water alone, or with soap), and why they did this.

Another research assistant and I took comprehensive notes during the discussions. After the focus group had concluded (usually 1.5-2 hours), the moderator, note-taker and myself met to discuss the pertinent findings and tackle any problematic issues in preparation for the next session. It is recognised that full transcription and analysis of focus group material is considered best practice. However, due to both time and economic constraints this was not attempted here since similar research conducted in other countries has not found this to be necessary (Biran 2005).

Because the interviews and focus groups were not fully transcribed all sections including the mothers' dialogue in subsequent chapters are close approximations of their comments derived from my detailed interview notes, rather than direct word-for-word quotations. As far as possible I have tried to keep to the language and sentence structure used by the women during the interviews and focus groups.

2.4.3 Questionnaires

A total of four questionnaires were designed and administered to all mothers enrolled in the study. These questionnaires referred to: demographic and socio-economic data; pregnancy, feeding and child-care practices; hygiene practices; and child morbidity.

Demography and household socio-economic status

These questions were adapted from official government surveys conducted in previous years and were administered to all mothers during Phase I of the project. Demographic data collected included: age and sex of index child; place of birth of index child; age, literacy, education levels, place of birth, employment status, caste and religion of index child's parents. Socio-economic status (SES) was assessed through questions relating to parental education levels, the family's housing situation, access to certain facilities (such as sanitation), household income and ownership of valuable material possessions (Appendix 2). These data were used to create a composite SES score for each family. Details of the construction of this index are outlined in Table 2.4.

Variable	Categories	Score
Maternal education	None	0
	Primary	1
	Secondary or above	2
Paternal education	None	0
	Primary	1
	Secondary or above	2
House ownership	Rent	0
	Own	1
House size	1 room	0
	2+ rooms	1
Toilet type	Public/shared	0
	Private	1
Fuel type	Firewood	0
	Kerosene	1
	Gas	2
Possessions[6]	None	0
	1-2	1
	3+	2
Income (per person per month)	<1000Rs	0
	100-1499Rs	1
	≥1500Rs	2
	MAXIMUM SCORE	**13**

Table 2.4 Components of the composite socio-economic score.

Pregnancy, feeding and child-care practices

Adapting questions used in the Nepal Demographic and Health Survey (2006), mothers were asked about their last pregnancy and feeding and child-care practices in order to identify other possible child health 'risk' factors. These included questions regarding: aspects of antenatal care and the pregnancy itself; breastfeeding and complementary feeding practices; and other specific child-care practices and beliefs (Appendix 3). The questionnaire was administered to all mothers during Phase I of the project and the data were used to construct a risk index for each child. The construction of this index is detailed in Table 2.5; the higher the score, the greater the number of risk factors.

[6] From a list of seven items: radio, television, bike, motorbike, mobile phone and fridge.

Variable	Categories	Score
Mother's age at birth of index child	Between 18-35 years	0
	<18 or >35 years	1
Smoked cigarettes during last pregnancy	No	0
	Yes	1
Drank alcohol during last pregnancy	No	0
	Yes	1
Saw HCP[7] at least 4 times during pregnancy	Yes	0
	No	1
Premature birth	No	0
	Yes	1
Child born in hospital	Yes	0
	No	1
Breastfed within 60 minutes of birth	Yes	0
	No	1
Fed child colostrum	Yes	0
	No	1
Fed child pre-lacteal	No	0
	Yes	1
Treat baby's drinking water[8]	Yes	0
	No	1
Weaning age of child[9]	Timely	0
	Early/late	1
Child vaccinated	Yes	0
	No	1
Correct knowledge about liquids during sickness[10]	Yes	0
	No	1
Correct knowledge about foods during sickness[9]	Yes	0
	No	1
	MAXIMUM SCORE	**15**

Table 2.5 Components of the composite risk score.

Hygiene practices

In addition to structured observations of hand-washing practices, self-reports of hand-washing behaviour were also collected from each mother. It is well documented that self-reports of hand-washing behaviour are not a reliable indicator of *actual* hand-washing practice (Cousens, Kanki et al. 1996). However, such reports are useful in determining people's *knowledge* about when hand-washing should occur. This hygiene questionnaire

[7] Health Care Professional
[8] Boiled or SODIS (sun-treated) water
[9] Timely = 6 months, early = <6 months, late = >6 month.
[10] Mothers were asked if children should be give more, less or the same amount of liquids or food during sickness such as diarrhoea. Mothers indicating that children should be give 'less' or the 'same amount' of liquid or food were given a score of 1 for this variable.

was administered to every mother during Phase I of the project (baseline) and again at the end of the project (endline) in order to assess changes in reported hand-washing behaviour over the intervention period (Appendix 4).

Mothers were asked to list times when they washed their hands during the day (before and after which activities). These junctures were written on a form, and for each juncture mentioned, the mother was asked what she washed her hands with (water, soap, mud/ash). If the mother had not spontaneously mentioned all of the five key hand-washing junctures (after defecation, after cleaning baby's bottom, before cooking, feeding or feeding baby), she was then asked specifically about these occasions; did she ever wash her hands at these times, and if so, what did she used to wash her hands with? If the mother replied that she washed her hands with soap (for any juncture), she was then asked why she did this and what type of soap she usually used for this.

In addition, to assess increases in soap usage, mothers were asked how many *new* bars of soap they had started using each week. As all types of soap (dish, laundry and body) were reportly used for hand-washing, this question referred to any type of soap started during the previous week. This question was administered by fieldworkers at the same time as the weekly morbidity reports.

Child morbidity

The design of the child morbidity questionnaire was informed by data collected during in-depth interviews during Phase I. The morbidity report was used to record the presence of the most commonly-reported symptoms (colds, fevers and diarrhoea) and their duration, with extra space to record any other symptoms the child had experienced during that week (Appendix 5). The morbidity report was administered to every mother in both intervention and control groups on a weekly basis by a local fieldworker from baseline (May 2007), throughout the intervention until November 2007.

Some semantic issues arose in the development of this questionnaire since the mothers' definition of diarrhoea differed from the biomedical definition. Mothers did not consider the child to have diarrhoea unless they were passing at least four or five loose stools a day, as opposed to the biomedical definition of 'three loose stool in a 24-hour period' (WHO 2005). In addition, mothers did not consider *'hariyo phij'* (loose, green, frothy stools) to be

diarrhoea at all, regardless of the number of stools passed in a day. Instead this symptom was attributed most commonly to '*chiso'* ('cold' entering the child) or more rarely to the child being attacked by the spirit of a child who had recently died. The morbidity form therefore did not ask the mothers directly if the child had suffered from diarrhoea in the previous week. Rather it asked specifically about the consistency and frequency of the child's stools during the week; only those children who fitted the biomedical definition were recorded as having diarrhoea. Mothers were also asked if there was any blood in the stools to differentiate between watery diarrhoea and dysenteric diarrhoea.

For colds, mothers were asked to describe all symptoms suffered by the child in the previous week and these were ticked off from a symptom list. The fieldworker also asked whether the child had suffered from a fever at all in the previous seven days. The mother was then asked if the child had suffered from any other symptoms not already mentioned and the details of these symptoms were noted in the blank section at the bottom of the form. For each symptom (diarrhoea, cold, fever or other symptom) the number of days the child had had the symptom was noted, as well as whether s/he was still suffering on the day of interview.

Local fieldworkers were trained in how to complete morbidity questionnaires with the mothers, firstly through role-playing with each other and then in a field setting. Once again training took place at a centrally located field site with mothers of children less than two years of age who were not recruited into the project. Each fieldworker interviewed each mother separately about all symptoms experienced by her child in the preceding seven days. The forms for each mother were then compared between fieldworkers to check consistency. This provided an opportunity to identify any areas of misunderstanding or confusion and ensured that all fieldworkers were using the same method and technique to elicit the most accurate information from the mothers. There followed two weeks of further training in the fieldworkers' own areas. At the end of each week, I met with all fieldworkers to check the forms for inconsistencies and missing data. Spot-checks were also conducted at random during the initial stages of the project to check the method and accuracy of the fieldworkers' technique.

Data collection

The socio-demographic, child-care and hygiene questionnaires were carried out with every mother after the completion of the structured observations. The questionnaires were

conducted verbally (in Nepali) in either the mother's own home or in a convenient central location. All answers were recorded on a pre-coded form and were subsequently entered into an Excel datasheet and then converted to an SPSS file.

Morbidity and soap-usage reports were conducted on a weekly basis by the local fieldworkers with the mother of the child. These reports commenced four weeks prior to the start of the intervention, to provide baseline morbidity data, and continued on a weekly basis thereafter until the close of the project.

2.4.4 Health measures

A baseline health check was conducted with all children in May 2007 prior to the start of the intervention. Thereafter, health checks were held for all children on a monthly basis until November 2007.

Anthropometry

Lengths and weights of children were measured using standard anthropometric techniques, as described by Lohman et al. (1988). Lengths were measured using a SECA stadiometer (Milton Keynes Scales, Leighton Buzzard, Bedfordshire, UK) to the nearest 0.5cm. Recognising the difficulty in taking such measurements, all children were measured twice by the same fieldworkers; where there were discrepancies between the two measurements, the smaller value was used. Weights were measured using a SECA baby-scale (Milton Keynes Scales, Leighton Buzzard, Bedfordshire, UK) to the nearest 0.01 kg. Infants were measured naked, save for the nappy containing the urine pad, the weight of which was subtracted to give a final measurement for the child.

Duplicate measures of height and weight for 20 children were taken to calculate technical errors of measurement (Ulijaszek and Kerr 1999) which yielded coefficients of reliability of .99 and .98 for weight and height, respectively.

Gut damage biomarker: lactose:creatinine urine test

Gut damage (intestinal permeability) was assessed using the lactose:creatinine urinary test (L:C) – a method which had been successfully employed in a previous study in Nepal (Panter-Brick, Lunn et al. 2009).

Breastfeeding infants ingest lactose from their mother's breast milk and the flux of this sugar in the body is known to achieve a steady state (Northrop-Clewes, Lunn et al. 1997). In a healthy child, lactose is hydrolyzed by the mucosal enzyme lactase and the resultant monomers are then passively absorbed across the mucosal wall into the bloodstream; only very small amounts of undigested lactose are absorbed into the body and excreted in the urine.

However, because of its exposed position on the brush border membrane of the mucosal villi, lactase is highly vulnerable to pathogenic damage. With the loss of this enzyme, less lactose is hydrolyzed, allowing more undigested lactose to be absorbed into the body and excreted in greater quantities in the urine. Thus, an increased quantity of lactose recovered in the urine is indicative of higher levels of intestinal damage.

As uptake and excretion of a probe molecule such as lactose can be affected by a number of factors other than intestinal permeability, a ratio of probes is often used to correct for this, based on the assumption that both probes are equally affected by abnormalities in the intestinal tract (Lifschitz 1985). In the absence of any dietary source, creatinine is metabolised and excreted in the urine at a constant rate of approximately 1g per 17.9-20kg of muscle mass (Borsook and Dubnoff 1947; Graystone 1968). Because the rate of creatinine excretion is known, the ratio of urinary lactose to creatinine (adjusted for body musculature) provides an accurate measure of the excretion rate of lactose in the child.

Until recently, most studies assessing levels of intestinal permeability relied upon the lactulose:mannitol test (Lunn, Northrop-Clewes et al. 1991; Northrop-Clewes, Rousham et al. 2001; Goto, Panter-Brick et al. 2002; Campbell, Elia et al. 2003; Goto 2006; Goto, Mascie-Taylor et al. 2009). However, this test has numerous disadvantages in field settings. The child must be dosed with the lactulose:mannitol solution and food withheld for at least two hours after ingestion. The test also requires the total amount of urine over a five-hour period to be collected, which places a considerable strain on mothers who must wait at the test centre for the duration. Full urine collections are difficult to obtain due to leakages of the urine bags or contamination by faecal matter. Even in controlled hospital conditions the collection of a total urine sample is extremely difficult to achieve (Kukuruzovic, Haase et al. 1999) and incomplete urine samples can significantly compromise the reliability of the lactulose:mannitol test.

By contrast, the L:C test avoids the need for dosing the child and can be calculated from a single, un-timed 'spot' urine sample, making it highly suitable for field conditions. The L:C test has been shown to correlate strongly with the L:M test for intestinal permeability (Beasley 2003; Beasley and Lunn 2004)) and has been successfully employed in our previous fieldwork in Nepal in 2005 (Panter-Brick, Lunn et al. 2009).

Urine sample collection

Health checks were conducted in the morning (between 7-9.30am) when it was cooler, making it easier to collect urine samples from the children. Upon arrival, the mother was asked to verify that the child had not been fed any cow or dairy milk that morning (which would invalidate the urine test). The child's bottom and genital area was thoroughly cleaned and dried before fitting the child with a locally-purchased plastic nappy, containing a sterile urine pad (Newcastle Urine Collection Pack, Ontex UK Ltd, Corby, UK). This pad was then checked by the mother or fieldworkers every five-to-ten minutes; once wet, it was removed from the child and two 2ml samples of urine were extracted using a sterile syringe. These samples were preserved with one or two drops of bacteriostat (chlorhexidine digluconate, 2g/L solution) and then frozen at -20˚C until shipment to the UK. As faecal contamination of the urine would invalidate the samples, any pads that contained faecal matter were rejected and the child was fitted with a new pad. Mothers were encouraged to breastfeed their children to promote urination.

Immune stimulation and nutritional biomarkers: finger-prick blood-spots

Levels of immunostimulation were assessed through analysis of protein markers in dried blood spots: α-1-acid glycoprotein (AGP), immunoglobulin G (IgG). In addition, haemoglobin (Hb) and albumin were also measured, providing information on the nutritional status of the child.

In response to tissue damage, inflammation or infection, the acute phase response is activated within the body, characterised by an increase in liver-synthesised proteins in the blood and tissue. Concentrations of these acute phase proteins and immunoglobulins in the blood rise dramatically and can therefore be used as an indicator of the severity and extent of immune-system stimulation. Previously whole blood samples were required for such tests, limiting their applicability outside clinical settings. However, recently developed assay methods allow for analysis of proteins in dried blood spots alone (see Panter-Brick,

Lunn et al. 2001). This minimally invasive method allows samples to be easily collected and stored under fieldwork conditions.

AGP is one of the major acute phase proteins in humans (Fournier, Medjoubi-N et al. 2000). In response to systemic tissue injury, inflammation or infection, cytokines are released causing the increased synthesis of AGP in the liver (*ibid*). Serum concentrations of AGP increase three- or four-fold within days of infection, with a half-life of five and a half days (Laurell 1985). Although C-reactive protein (CRP) is known to be a more sensitive indicator of the acute phase response, responding earlier and rising higher (1000-fold), its half-life is much shorter, with declines in concentrations occurring after 24-48 hours (*ibid*). Thus, unless blood sampling occurs within this 24-48 hour period no impact would be detected. With its longer half-life, AGP proves to be a more reliable indicator of recent infection.

Immunoglobulins (Ig), also known as 'antibodies', are glycoproteins made by B cells as part of the immune response. There are five different types of immunoglobulins in the body – IgG, A, M, D and E – with IgG being the most abundant in the blood. IgG is known to develop cumulatively in the body in response to infection and therefore is a useful indicator of long-term exposure to pathogens and general environmental quality.

Albumin, the most abundant plasma protein, is a useful indicator of nutritional status. Hypoalbuminemia is indicative of impaired nutritional status, specifically indicating an inadequacy of protein in the diet (Fuhrman 2002). However, it also acts as a 'negative acute phase protein', decreasing in serum concentration by 80-90% in the five days following infection or injury (Fleck 1989). Thus it also serves as another measure of immune status in young children.

As with albumin, haemoglobin levels provide information as to the nutritional status of the child. Haemoglobin contains iron which binds to oxygen molecules in the lungs and transports them to tissues in the body. Low levels of iron in the body lead to anaemia which has been shown to have negative impacts on health, particularly in young children; low haemoglobin levels have been associated with increased risk of gastro-intestinal and respiratory infections in young children and delays in cognitive development and behavior during infancy and childhood (Ryan 1997). Using the WHO definition, children with Hb levels <110g/L were defined as anaemic (WHO 2008).

Blood drop sample collection

Up to five blood drops were collected on 903 protein saver collection cards (Whatman Plc, Maidstone, Kent, UK) from the child's finger-tip. Mothers were asked to sit on a chair holding the child on her lap while the fieldworker cleaned the child's fingers with alcohol, drying them thoroughly with cotton wool. A single-use lancet (Hemocue Ltd, Dronfield, Derbyshire, UK) was then applied to the fingertip. The first drop of blood was wiped away and the second was used to fill a microcuvette which was placed in a Hemocue (Hemocue Ltd, Dronfield, Derbyshire, UK) to obtain an on-the-spot haemoglobin result. The subsequent blood drops were collected on the collection card which was then left to dry for up to 12 hours, before being placed in a plastic zip-lock bag with desiccant and frozen at -20˚C until shipment to the UK.

2.5 Research process

2.5.1 Research team

The research team consisted of: myself; a field co-ordinator who helped with the running and organisation of the intervention and health checks; a research assistant who acted as translator during interviews and focus groups, and assisted with the design of the intervention and training of Community Motivators; ten local fieldworkers who undertook the structured observations and weekly morbidity reports; five Community Motivators (intervention areas only) who were employed to run the intervention and encourage adoption of hand-washing practices; an additional field worker who assisted during the health checks. All fieldwork activities were closely supervised by myself throughout, including unannounced spot-checks and regular meetings with team members.

2.5.2 Data collection schedule

Fieldwork was conducted between February 2007 and January 2008, with data collection running from March-December 2007. The table below summarises the activities undertaken, the sampling strategy employed and the frequency of the activities.

	Activity	Sample	Frequency
Phase I Preparation	Structured observations	Random selection of two-thirds of households, n=75	Once
	In-depth Interviews	Random selection of intervention mothers only, n=26	Once
	Focus groups	Random selection of intervention mothers only, n=3 with each focus group consisting of 6-8 mothers	Once
	Questionnaires - socio-demographic - child-care practices - hygiene	All mothers in both intervention and control areas	Once
Phase II Design	Design of hand-washing intervention - see Chapter 3 for details		
Phase III Implementation	Intervention activities: - home visits, group visits - distribution of posters, soap etc	All intervention areas	On-going
	Health checks: - anthropometry - urine and blood samples	All children in intervention and control areas	Monthly
	Questionnaires: - morbidity and soap usage - hygiene	All children in intervention and control areas	Weekly Once
	In-depth interviews	Purposive selection from intervention and control groups, n=27	Once
Phase IV Evaluation	Laboratory analysis of urine and blood samples Data entry and cleaning Statistical and qualitative analysis of data		

Table 2.6 Data collection schedule.

2.5.3 Ethical considerations

The study complied with the six core principles outlined in the *ESRC*'s Research Ethics Framework, as outlined below:

1. Research should be designed, reviewed and undertaken to ensure integrity and quality.

The project design was discussed with my supervisors and other colleagues in relevant fields (Dr Peter Lunn, Cambridge University; Dr Valerie Curtis, London School of Hygiene and Tropical Medicine) prior to fieldwork. It was also subject to internal departmental review in the form of a first year viva to assess the validity and feasibility of the research proposal.

Official ethical permission for this study was granted by Durham Anthropology Department Ethics Committee and the Nepal Health Research Council, Kathmandu, Nepal. In addition, verbal permission was given by local community leaders in each area.

2. Research staff and subjects must be informed fully about the purpose, methods and intended possible uses of the research, what their participation in the research entails and what risks, if any, are involved.

The purpose of the research project was fully explained to all research staff and appropriate training was provided for staff members specific to their own particular roles.

Mothers were invited to attend an information session about the project in their local area at the start of the study. In order to reduce reactivity in the later parts of the project, no mention was made of hand-washing or hygiene at this point in the study; the mothers were simply told that the project wished to study the health and growth of young Nepali children. All measures (height, weight, urine and blood-drop samples) were demonstrated to the mothers and they were encouraged to ask questions about the study.

At the end of the project, mothers of children in the control areas were invited to attend educational sessions promoting hand-washing-with-soap, similar to those provided in the intervention areas earlier in the year. The mothers were also provided with free samples of soap at this time.

3. *The confidentiality of information supplied by research subjects and the anonymity of respondents must be respected.*

All data provided by participants were held under secure conditions. Care was taken to protect the anonymity of participants by the use pseudonyms, where necessary. Consent was gained from care-givers for the use of photographs of children in this and other publications. The importance of maintaining confidentiality was emphasised to all staff throughout the project.

4. Research participants must participate in a voluntary way, free from any coercion.

Mothers consenting to participate in the project were read a consent form (in Nepali) that emphasised their right to withdraw from the study, at any point, without having to provide any explanation (Appendix 6). In addition, verbal consent was gained from each mother at other appropriate stages of the project – i.e. before structured observations, interviews or health checks.

At each health check, mothers were offered 200Rs (approximately £1.50, equivalent to a day's wages). This monetary gift was a way of compensating mothers for the time they gave up during the health checks, rather than a means of encouraging participation.

At the end of the study all children were presented with a gift of clothing and were checked by a team of doctors and nurses from Anandaban Hospital in Kathmandu, who provided medication and packets of fortified cereal to children requiring these.

5. Harm to participants must be avoided.

Strict safely procedures for the collection of biological samples were followed at all times. My research assistant and I collected all urine and finger-prick blood-drop samples, strictly adhering to hygiene and safety procedures. Used needles and hemocue cuvettes were stored and disposed of safely at a local hospital.

6. The independence of the research must be clear; any conflicts of interest or partiality must be explicit.

Funding for the research was provided by a joint studentship from the Economic and Social Research Council and the Medical Research Council. Additional funds were

provided by grants from The Biosocial Society and The Parkes Foundation. There were no conflicts of interest or partiality.

2.6 Data analyses

2.6.1 Urine sample analysis

All urine samples were analysed at the Department of Biological Anthropology, Cambridge University, by Dr Peter Lunn. Urinary concentrations of lactose were measured using automated enzymatic assay methods, as described by Northrop et al. (1990) and Beasley (2003). Two ELISA plates were prepared with 20 µl of undiluted urine: to the first was added 25µl of triethlanolamine (TE) buffer; to the second, 25µl of β-galactosidase dissolved in the TE buffer. The plates were incubated at 37˚C for two hours and were then placed in the ELISA plate reader. The reagent reservoir in the plate reader contained an enzyme-buffer cocktail of TE buffer, adenosine-5'-triphosphate disodium salt (ATP), nicotinamide adenine dinuculotide phosphate (NADP), hexokinase-glucose-6 phosphate dehydrogenase (HK-6GP) and water (Sigma Aldrich Company Ltd, Dorset, UK). 150ml of this enzyme-buffer cocktail was added to each ELISA plate and the optical density change was recorded for up to seven minutes, giving an index of lactose concentration in the sample (Beasley 2003).

Creatinine concentration was analysed using the Jaffe technique (Randox creatinine assay kit, Crumlin, Co. Antrim, UK). This colorimetric method is based upon analysis of the colour difference of the creatinine-pictrate reaction before and after treating the sample with sulphuric and acetic acid. Both assays were performed using Labsystems iEMS ELISA Plate Reader and its accompanying Ascent Software (Labsystems iEMS, Cambridge, UK).

2.6.2 Blood-drop sample analysis

With the exception of the on-the-spot haemoglobin reading, all blood analyses were undertaken at the Department of Biological Anthropology, Cambridge University, by Dr. Peter Lunn. Six millimetre discs were punched out of the dried blood spots and the plasma constituents were eluted by immersion in 1.25 ml of a phosphosaline buffer (0.01M

sodium phosphate, 0.5M sodium chloride, pH 7.2) containing 1% Tween 20 for 24 hours at 4˚C. Concentration of blood proteins in the eluate were then assessed through standard assay techniques (Panter-Brick, Lunn et al. 2001). Albumin was assessed through a turbidmetric technique with reagents supplied by DakoCytomation (Ely, Cambs, UK). A double-sandwich ELISA technique was used to determine levels of IgG and AGP, using antibodies from DakoCytomation and Insight Biotechnology (London, UK), respectively. Haemoglobin concentration was determined using a cyanmethaemoglobin technique (Randox haemoglobin assay kit, Randox, Crumlin, Co. Antrim, UK). To correct for any possible elution errors, a correction factor was calculated using the ratio between original Hb values (as measured by the Hemocue in Nepal) and Hb concentrations in the dried blood spots (see Panter-Brick, Lunn et al. 2009).

2.6.3 Data management

Growth data (heights and weights) were used to calculate height-for-age, weight-for-age and weight-for-height z-scores using the Epi-Info computer package (using growth reference curves from the CDC 2000 data set). Almost all mothers knew their child's exact date-of-birth in the Nepali calendar. This date was converted into its Gregorian equivalent using an official Nepali-Gregorian calendar. Two mothers knew the month and year of birth, but not the actual day; these children were assigned to the 15th of the month in question.

All continuous variables (growth and biochemical variables) were checked for skewness using Cox's test (coefficient of skewness divided by standard error of skewness). Intestinal permeability values (L:C) showed positive skewing and were normalised using log transformations (log10). In order to prevent negative values, +2 was added to each individual score before transformation, as recommended by Tabachnick & Fadell (1996).

Outliers (+/- 2SD) for all growth and biochemical variables were carefully checked; those outliers that were biologically plausible were left in the dataset. One child displayed extremely low weight-for-age z-scores (range -6.03 to -5.38). Although extremely low, these weights were carefully checked in the field for errors and accurately reflected this child's growth status. As this child was so thin, a medical doctor was asked to examine her; apart from being very under-nourished, she was otherwise healthy and her data were kept in the final dataset.

In addition, one AGP and three L:C values proved to be very high. In each case the child's biochemical and morbidity profile were studied carefully but in all cases it was felt that these values were too high to be biologically plausible. Rather than eliminate these children altogether (and thus further reducing the sample size) these abnormal values were removed; the child's mean value for that variable was substituted. These substitutions would not unduly affect statistical relationships.

2.6.4 Statistical analyses

Statistical analyses were conducted using Statistical Package for Social Sciences for Windows, versions 14 and 15 (SPSS, Chicago, Illinois) and Stata (Intercooled) Version 8.2 (StataCorp, College Station, Texas)**.** Relationships between categorical variables were assessed using χ^2 tests, and between continuous variables using two-tailed independent t-tests and linear regression. Non-normally distributed morbidity variables were analysed using non-parametric Mann-Whitney U tests. The statistical significance level was set at .05.

For analysis of the biochemical (L:C, IgG, AGP, albumin and Hb) and growth variables (HAZ, WAZ, WHZ) I employed a three-step analytical strategy. Firstly, I examined these variables on a cross-sectional basis, using linear regression to look for relationships between gut damage, immune stimulation and growth month-by-month for the seven months of data collection[11].

Secondly, relationships between these variables *over the whole period of the intervention* were assessed, following methods used by Panter-Brick et al. (2009). Mean values were created for each biochemical variable to assess average levels of gut damage (L:C) and immune stimulation (AGP, IgG) and biochemical nutritional status (albumin, Hb) over the whole study period. Mean growth status for HAZ, WAZ and WHZ was also calculated. Linear regression analysis was then used to assess the relationships, firstly between mean biomarkers, and secondly between mean biomarkers and mean growth status.

[11] The intervention ran for six months (June-Nov 07), but was preceded by a baseline health check (May 07); therefore there are seven data points for each variable.

Thirdly, I conducted more sophisticated analyses employing a multi-level time series approach to analyse the effect of the intervention longitudinally. One of the assumptions underpinning ordinary linear regression is that for any two observations the residual terms should be independent (Field 2005). In longitudinal or time-series studies where each subject is measured on multiple occasions, this assumption is violated: repeated data points collected for each individual child are clearly not independent of one another and consequently their residual values will be correlated. For this reason, time-series data need to be analysed using a technique that can take into account this lack of independence between data points, by controlling for the shared variation expected within each child. In time-series analysis variation is partitioned into within-subject variation (Level 1) and between-subject variation (Level 2) (Figure 2.5). Regression models created using this technique can therefore examine between-subject variation, whilst adjusting for the clustering of variation that occurs within each subject[12].

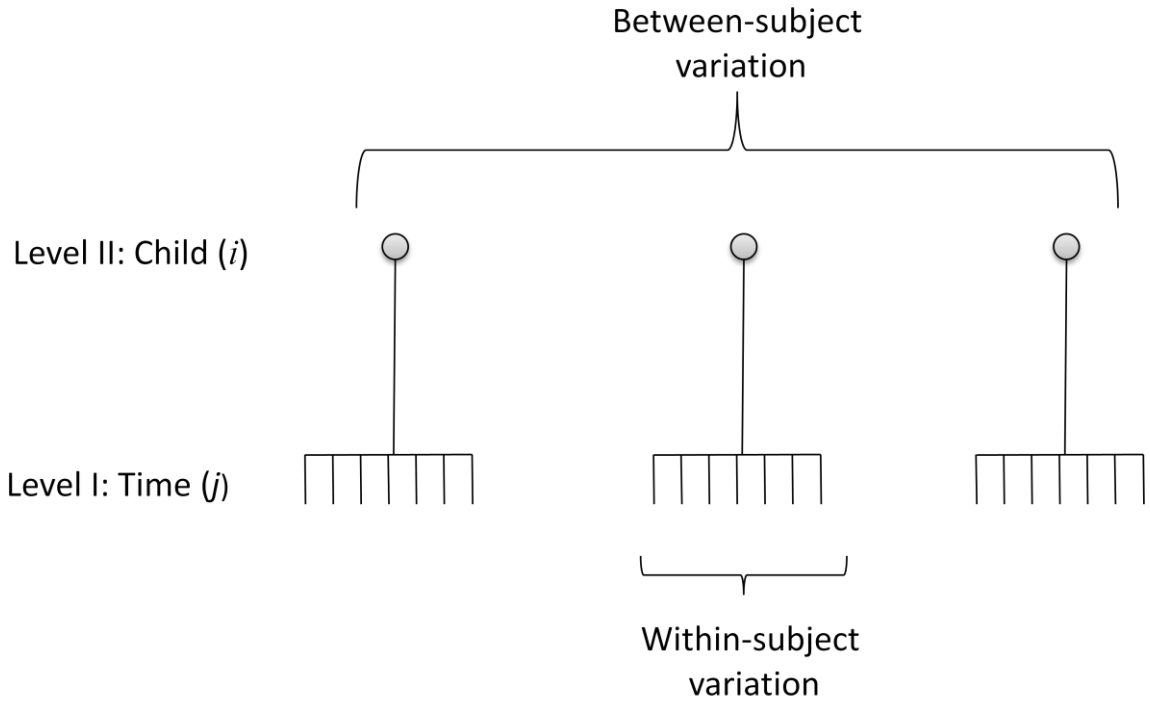


Figure 2.5 Diagram depicting the different levels of variation for cross-sectional time series analysis.

[12] In time series analysis, between-subject variation is denoted by Rho which can range from 0 to 1.

For each of the three types of analysis, relationships between dependent and predictor variables were first assessed through univariate models. Multivariate models were then constructed using predictor variables that were significant (P <.05) in the univariate analysis. The impact of relevant demographic and socio-economic variables on the model was tested at each stage. Age showed a strong relationship with both growth and biochemical variables and thus was included as a covariate in all further analyses. However, controlling for other variables (such as gender, maternal age, maternal body mass index [BMI], caste, SES and household size) had no bearing on the relationships between predictor and outcome variables and thus were excluded from the final analyses.

Because I was interested in assessing how the two groups changed in levels of biomarkers and growth over the period of the intervention I also tested for significant interactions between time and group: a significant interaction would suggest that the two groups were changing in different ways over the period of the intervention. Where a significant interaction was observed between time and group, I controlled for *baseline age*, rather than *age*. All terms included in the interaction (time and group) must be included as independent predictors in the model. However, because *age* and *time* are closely correlated (r=.60) including both of these in the model could lead to collinearity, violating an important assumption of the test. Baseline age was therefore substituted into the model to avoid this.

2.6.5 Sample attrition

The required sample size was calculated to be 88 children, which was increased to 100+ to accommodate potential attrition. Originally, 109 children were enrolled into the study, but ten children dropped out between recruitment and the start of the intervention in May 2007. Of the 99 children who completed the study, 11 had incomplete profiles and were removed from the final analyses, leaving a final sample size of 88 children (45 intervention, 43 control).

There were no differences between the attrition (n=11) and study (n=88) group for any variables (Appendix 7), with the exception of toilet type: none of the attrition group had a private toilet compared to 18% of the complete group (cell count was too low to perform a χ^2 test on this variable.) Here after all analyses refer to the 88 children with complete data profiles.

Summary

This chapter described the study setting and the sampling technique used to recruit the 88 children in the study. It outlined in detail the design and implementation of the different qualitative and quantitative research instruments and set out the data management and statistical analytical strategy employed. The following chapter will describe in detail the design and implementation of the hand-washing intervention.

CHAPTER 3

Design and Implementation of a Community-Based Hand-Washing-With-Soap Intervention

Introduction

This chapter will describe the design and implementation of the community-based hand-washing-with-soap intervention. It starts with a theoretical discussion about the best ways to change and influence behaviour. The theoretical model of behaviour change that underpinned the intervention is presented and discussed. Key features of successful interventions are identified and the methods used in other hand-washing interventions are briefly reviewed, before going on to describe the specific preparation, design and implementation of this study's intervention.

3.1 Theoretical model of behaviour change

A fundamental principle in public health is that a substantial proportion of morbidity and mortality from a wide range of diseases is due to particular patterns of behaviour and that these behaviours can (at least theoretically) be modified (Conner and Norman 2005:1). Take for example some of the biggest health concerns of the 21st Century: HIV/AIDS, malaria, lung cancer, coronary heart disease. Each of these diseases could be dramatically reduced through simple changes in behaviour; by using condoms, using bed nets, giving up smoking and eating healthily and increasing exercise, respectively. As discussed in Chapter 1, major childhood killers such as diarrhoea and acute respiratory infections are no exception to this principle: in both cases, the pathogens that cause these diseases could effectively be reduced through the simple act of washing hands with soap at appropriate junctures.

However, if these simple behavioural solutions – none of which require expensive technical equipment or expertise – are so effective, why have we not seen a dramatic decrease in morbidity and mortality rates for these diseases? The simple reason is that

knowledge alone is not enough. Human beings are not rational automatons who weigh-up each decision they make in an exclusively objective and logical way. Every choice we make is influenced not only by the knowledge we possess, but by numerous other cognitive, emotional, psychological, social, cultural and environmental factors that interact in complex and dynamic ways. Thus, despite the knowledge that, for example, smoking causes lung cancer - and perhaps also despite an *intention* to quit – many people continue to smoke cigarettes. It is at the gaps between knowledge, intention and behaviour, where public health interventions so often fail: it is far easier to change people's knowledge and intentions than it is to actually translate these intentions into sustained behavioural change.

Successful interventions are the ones that manage to bridge these gaps – the ones that move from being informative to persuasive to *compelling* (Panter-Brick, Clarke et al. 2006). So what makes an intervention compelling? Before we can answer that, we first require an understanding of the process of behavioural change in human beings. The past thirty years has seen a burgeoning of research into this area, with many researchers trying to elucidate how and why people act as they do. Numerous models of behaviour change have been proposed and have been tested in the field with varying degrees of success. It is beyond the scope of this thesis to offer a comprehensive review of all these theories. Instead, some of the major theoretical models of behaviour change have been summarised in Table 3.1

.

Models	Key References	Summary
Health Belief Model	Rosenstock (1966; 1974); Becker (1977)	An individual's behaviour depends upon on how *susceptible* they feel to a particular disease and how *severe* they perceive the consequences of that disease to be, with these perceptions being modified by demographic and social factors and inherent personality traits. Their actions to counteract this threat will depend on the evaluation of alternative options, weighing up how effective they think the action will be at protecting them from the disease threat, and the perceived barriers (physical, psychological, social, financial) to undertaking that action.
Health Locus of Control Model	Wallston & Wallston (1978); Wallston et al. (1981)	This model has its origins in social learning theory (Rotter, 1954) and centres on how an individual perceives their ability to influence and control their own life. People's perceptions are measured along three dimensions focusing on the extent to which they believe their health is predominantly influenced by their own actions, powerful others (such as healthcare professionals) or by fate or chance. The model predicts that people with a strong sense of control over their lives will be more likely to engage in health promoting/protecting behaviours.
Socio-cognitive Model	Bandura,(1977; 1986; 1997)	This model focuses on the interaction between an individual and their social environment in order to understand what motivates behaviour. Behaviour is thought to be predicted by an interaction between incentives, outcome expectancy and efficacy expectancy. Incentives are the value that an individual places on the outcome or consequence of behaviour. Outcome expectancy refers to the belief that a certain action will produce a certain outcome. Efficacy expectancy refers to the extent to which an individual feels capable of carrying out the required action.
Theory of Planned Behaviour	Fishbein & Ajzen (1975); Ajzen & Fishbein (1980); Ajzen (1991)	This model is an extension of Fishbein & Ajzen's *Theory of Reasoned Action* (1975). It suggests that the proximal determinant of behavioural change is an 'intention', although a trigger is also necessary in order to move an individual from intention into action. Intention to perform a certain behaviour is determined by an individual's attitudes (referring to their overall evaluation of the behaviour), subjective norms (referring to whether they think significant others think s/he should engage in this behaviour) and self efficacy (referring to their perceived ability to perform the required behaviour).

Models	Key References	Summary
Protection Motivation Theory	Rogers (1975; 1983)	This model suggests that an individual's health behaviour is the result of two appraisal processes. Threat appraisal refers to an individual's perception of their susceptibility to a health threat and their assessment of the severity of their threat. The coping appraisal refers to an individual's process of assessing behavioural alternatives to diminish this health threat. This is made up of action-outcome efficacy – the extent to which the individual believes the action will remove or diminish the threat – and self-efficacy – the extent to which the individual believes they are capable of executing the required course of action.
Stages of Change Model	Prochaska & DiClemente (1983) ; Prochaska et al. (1992); Prochaska & Velicer (1997)	This model suggests that behavioural change does not occur instantly but rather an individual passes through a number of specific stages when changing behaviour or adopting a new one. In the *pre-contemplation* stage, the individual is content with their existing behaviour and feels no motivation to change. In *contemplation,* a change of behaviour is considered. In the *action* stage, a new (or changed) behaviour is attempted. This behaviour is then sustained in the *maintenance* stage. Following this, there are two possibilities: either the individual continues with the new behaviour until it becomes entrenched and the process of change is said to be complete; or, the person relapses to the old behaviour and returns to the contemplation stage.
Diffusion of idea	Rogers (1983)	This model describes the spread of new behaviours through communities. It suggests that, at first, the take-up of a new idea is very slow and is only adopted by a few (the innovators). The idea then starts to 'diffuse' throughout the community and more and more people start to try it. Diffusion finally slows as only the resistant or 'hard-to-reach' groups are left practising the old behaviour. The model suggests that different approaches must be used depending on whether you are attempting to introduce a new idea, encourage the spread of an existing idea, or trying to influence the 'hard-to-reach' groups.

Table 3.1 Theoretical models of behaviour change.

The theoretical model that under-pinned the design and implementation of this intervention programme is depicted in Figure 3.1. This model is primarily based on Fishbein and Ajzen's *Theory of Planned Behaviour* (Fishbein and Ajzen 1975; Ajzen and Fishbein 1980; Ajzen 1991), but also incorporates ideas from several other models described in Table 3.1.

The model suggests that an intention to perform a certain behaviour is a product of the interaction between a person's attitudes, subjective norms and perceptions of self-efficacy. 'Attitudes' refers to the person's overall positive or negative feeling towards personally performing the behaviour in question (Fishbein and Yzer 2003). This attitude is formed by two elements. Firstly, *outcome expectancy* refers to how certain the actor is that the potential threat (e.g. contracting an STI) will occur if they do not perform the target behaviour (e.g. using a condom) and how severe they believe this threat to be. Secondly, *outcome efficacy* refers to the actor's certainty that the target behaviour will have the desired effect (e.g. reduce the risk of STIs). Together, these beliefs contribute to a person's overall positive or negative attitude towards the behaviour in question.

The concept of the subjective norm moves us from what the actor him/herself feels to a consideration of the perceived social influences upon the actor. It refers to the actor's belief that important others (i.e. friends, family, authority figures) think s/he should perform the behaviour, combined with the actor's motivation to comply with these expectation.

Finally, self-efficacy (or perceived behavioural control, in some models) refers to the actor's self-perceived ability to perform the behaviour in question. It refers to the actor's belief that s/he has the skills, confidence, resources etc (enablers) needed in order to achieve the desired goal and the belief that s/he can over-come obstacles (barriers) to that behaviour (such as time, money, location etc).

Clearly attitudes, subjective norms and perceptions of self-efficacy vary between people because they are influenced by numerous individual characteristics, such as demography, personality type and environment. In the model these are termed *external factors* as they are (usually) not modifiable by the intervention process but are none-the-less important predictive factors.

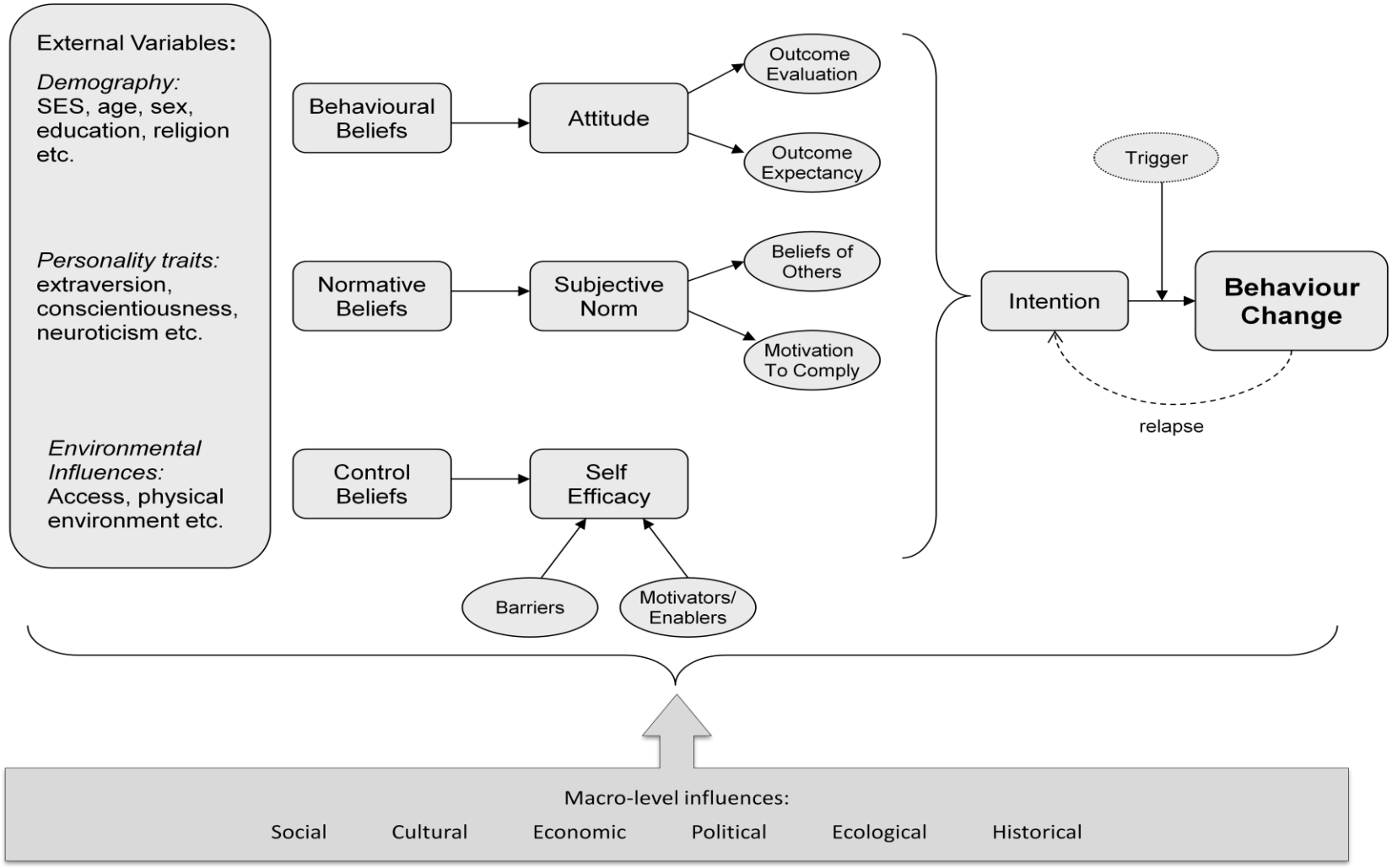


Figure 3.1 Theoretical model of behaviour change, adapted from Fishbein (2000).

An addition made to Fishbein & Ajzen's (1975) original model is the role of macro-level factors that influence every section of the behavioural model. All human behaviour takes place within specific social, cultural, political, economic, historical and ecological contexts. All of these factors interact dynamically with each other to profoundly influence people's behavioural intentions and their ability to act upon these intentions. It is easy to see how historical factors shape and influence current societal normative beliefs, or how prevailing political and economic systems can potentially hinder one's ability to successfully effect behavioural change by limiting access to resources or power. However, these macro-level influences also affect even the more immutable factors in the model – such as demography, for example – by influencing the *way* in which gender, age or religion is interpreted within any given culture.

Taking into account these external factors and the wider macro-level influences, it is the complex interactions between these factors and an individual's attitudes, norms and perceptions of self-efficacy that leads to an individual making a decision (on a conscious or unconscious level) about the behaviour. Once an intention is formed, something must 'trigger' the actor to move from intention into action. The model also acknowledges behaviour change as a dynamic process, whereby relapse and renewed attempts are a recognised part of the process until a new habit is formed.

Thus, according to this model, for an intervention to be compelling it must identify and then effectively manipulate the attitudes, social norms and perceptions of self-efficacy in the target population to stimulate behavioural change. Such information is best discovered through in-depth, qualitative preparatory work in the target community.

3.2 Key features of successful interventions

Having a theoretical model of behaviour change that underpins and informs the design and implementation of an intervention is seen as essential element of success (Fishbein and Ajzen 1975; Gallant and Maticka-Tyndale 2004; Panter-Brick, Clarke et al. 2006). Too many interventions have failed because they have been developed without adequately identifying and addressing local beliefs, attitudes and constraints on human agency (Cornwall and Jewkes

1995; Jongpiputvanich, Veeravongs et al. 1998). Reviewing the most successful intervention programmes reveals a list of the best strategies by which to identify and influence attitudes and social norms and address issues of self-efficacy. Some of these key features of effective interventions are depicted in Figure 3.2 and are discussed below.

The most successful interventions are those founded on the principles of community participation, mobilisation and empowerment. First heralded at the International Conference on Primary Health Care in Alma Ata (now Almaty) in 1978, the idea of community participation in health planning is one that has received enormous attention in the rhetoric, if not the actual practice, of public health. If, as the present theoretical model suggests, an intervention must be based upon local attitudes and beliefs and be cognisant of local social and environmental constraints on behaviour, who better to identify these than local people themselves? Including local people in the planning and implementation of interventions means that they are much more likely to meet local priorities, be culturally appropriate and identify and promote the messages and strategies that are most compelling for the local audience.

Other essential features include building upon local practices, targeting those members of the community most receptive to change and focusing on outcomes that are most relevant to the target audience. This last feature is particularly important. Many interventions have failed because of the conceptual gap between how planners think about a health issue and how it is envisaged by the local population (de Koning and Martin 1996). For example, in a study of hand-washing in Burkina Faso, Curtis (1997) notes that childhood diarrhoea was only rarely seen as being related to hygiene by local mothers, with most episodes being attributed to the 'evil eye', teething or the transgression of social taboos. Attempting to get mothers to increase hand-washing by promoting its effects on reducing diarrhoea would simply not have made sense to these women. Instead, ethnographic investigation revealed that a message revolving around the importance of hygiene as a social virtue tapped into local attitudes and norms and was therefore much more compelling (*ibid*).

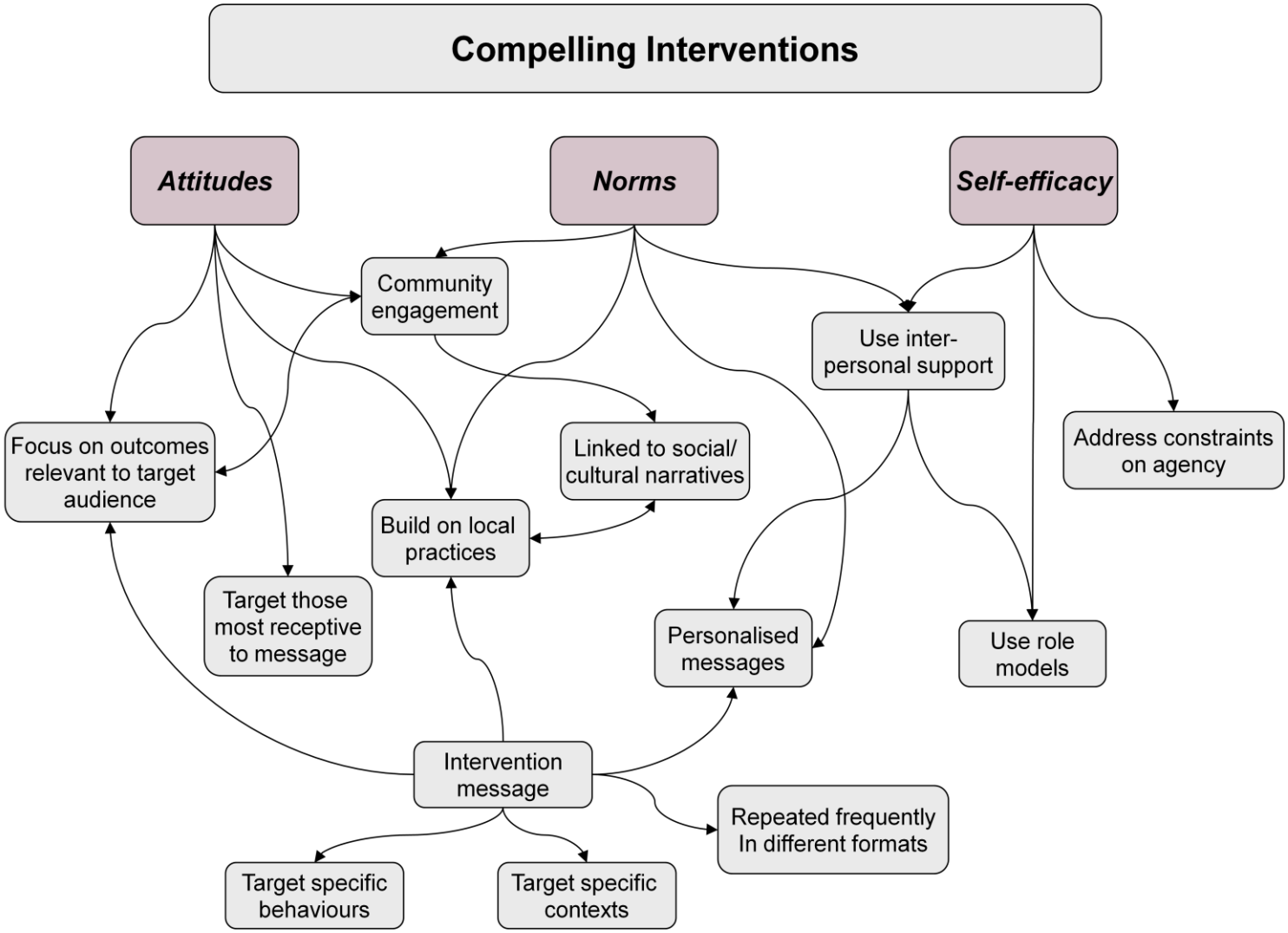


Figure 3.2 Features of culturally compelling interventions and links to attitudes, norms and perceptions of self-efficacy.

It is not only the content of the message that requires attention, but also the media through which it is promoted. Galavotti et al. (2001) argued that intervention messages must be promoted in a way that links in with local social and cultural narratives. Messages that are relevant to the everyday lives of the target audience should be promoted through popular local media, such as drama, songs, poetry and dance. Such formats are not only familiar to the audience, but are also designed to entertain and thus stimulate an affective connection with the intervention message, rather than just a cognitive understanding (*ibid*; Panter-Brick et al. 2006).

Bolstering perceptions of self-efficacy is, as we have seen, also crucial to the success of an intervention and several strategies for achieving this have been suggested. Obviously, there is a very practical side to this where, as far as possible, interventions must identify and address local constraints on human agency, whether these be financial, environmental or social. But interventions must also work on an individual level too by boosting the skills and confidence of local people to ensure they can initiate and sustain behaviour change. The use of inter-personal support systems and role models has been successful in many studies. Galavotti et al. (2001) argued that role models can be a powerful way of changing people's attitudes towards a behaviour by modelling the steps needed to achieve such change, practically demonstrating the way in which obstacles can be overcome and providing living evidence of how the behaviour can enhance people's lives. Thus, the use of local role models to promote the intervention message can increase people's confidence in their own ability to affect such a change.

Finally, we should consider the specific content of the intervention message itself. Fishbein (2000) argues that intervention messages have the most impact when they are directed at specific behaviours rather than more general behavioural categories. For example, an effective intervention message would promote 'eating five portions of fruit and vegetables every day', rather than the more generic message of 'eat healthily'. In addition, he suggest that the most effective messages include at least three elements: an action (e.g. using), a target (e.g. condoms) and a context (e.g. during sex). In this way, a simple, clear and unambiguous message is delivered. Finally, messages should be promoted frequently and in many different

formats in order to be most effective (Loevinsohn 1990; Pinfold and Horan 1996; Panter-Brick, Clarke et al. 2006)

3.3 Key features of hand-washing interventions

Whilst the above strategies and principles are useful for informing the general design of this study's intervention, it is worth reviewing in more depth the methods and messages used in other interventions specifically designed to promote hand-washing. Table 3.2 reviews the target group, messages, methods, outcomes and impact of hand-washing interventions across the developing world.

In most of these interventions the **target** group was the mothers or families of young children. All interventions aimed to improve hand-washing rates at key events (e.g. before cooking, eating or feeding and after defecation or cleaning a baby's bottom), though most also included other messages regarding safe disposal of faeces and food hygiene. The majority of the interventions specifically encouraged the use of soap, although one (not providing free soap to the participants) did not do so for fear of alienating the poorest families (Pinfold and Horan 1996). The studies in Burkina Faso (Curtis, Kanki et al. 2001) and Thailand (Pinfold and Horan 1996) both refrained from specifically mentioning diarrhoea in the intervention messages, emphasising instead the social virtue of cleanliness. All other studies however involved at least some basic form of education regarding the faecal-oral route of transmission.

A wide variety of intervention **methods** were used in these studies. Many held community meetings to raise interest and awareness of the programme. These were often followed up with small discussion groups that met on a regular basis to discuss the importance of hygiene and any barriers to behaviour change, as well as offering support and encouragement. Regular home visits by fieldworkers to provide additional support to participants were also a common feature. Several studies made use of traditional forms of theatre, songs, proverbs and poems to help deliver the intervention message. Intervention materials included posters, pamphlets, videos, slide-shows, educational materials for school children and, in some cases, provision of soap and/or water containers to facilitate hand-washing practices.

Intervention	*Target Group*	*Message*	*Methods*	*Outcomes*	*Impact*
Khan (1982) Bangladesh	• Families of patients with Shigellosis diagnosis • 4 groups: soap and water; soap only; water only; control	• HW before cooking, eating and feeding • HW after defecation • Aim to reduce secondary infection rates	• Provision of soap and/or water to appropriate intervention groups • Intervention carried out for just 10 days	• Secondary infection rates of Shigellosis	• 84% reduction in secondary infection in soap and water group, compared to control group
Sircar et al. (1987) India	• 740 households in two Calcutta slums	• HW after defecation and before cooking/eating	• Soap provided to participants • Home visits emphasising importance of HW • No posters, slides or visual materials were used	• Diarrhoeal and dysentery morbidity rates	• Incidence of dysentery in individuals over five years was significantly higher(*P*=0.05) in control than intervention sites
Stanton and Clemens (1987) Bangladesh	• Women and children in 25 intervention and 25 control villages	• Prevention of open defecation by children • HW before cooking, eating and feeding • HW after defecation and cleaning baby's bottom • Proper disposal of rubbish and faeces	• Group discussions for women and children • Large community demonstrations of behaviours • Community-wide planning and action meetings • Stories and games to reinforce message	• Diarrhoeal incidence reports	• Diarrhoeal incidence was found to be 26% lower in intervention than control area
Alam et al. (1989) Bangladesh	• Mothers in households with children <5 years (n=314 and 309 intervention and control children, respectively)	• Variety of messages including use of hand-pump water, safe water storage, safe disposal of faeces and HW with ash or soap after defecation	• Female health works conducted home visits, group discussions and demonstrations	• Diarrhoeal morbidity reports	• Significant reduction in diarrhoea episodes in intervention children – 3.4 vs. 4.1 episodes per year (*P*<.001)

Intervention	*Target Group*	*Message*	*Methods*	*Outcomes*	*Impact*
Han & Hlaing (1989) Burma	• 494 mothers of children <5 years	• HW before cooking, eating and feeding • HW after defecation and cleaning baby's bottom	• Soap provided to participants • Daily visits by intervention staff	• Incidence density ratios (IDR) for diarrhoea and dysentery	• IDR for diarrhoea was significantly lower in intervention than control children (IDR=0.70, 95% CI 0.54-0.92)) • 40% reduction in dysentery in children < 2 years
Wilson et al. (1991) Indonesia	• 130 mothers in 2 Indonesian villages	• HWWS before preparing/eating food, after defecation	• Mothers given soap and explanation of faecal oral route • Repeated and reinforced fortnightly • Placebo intervention in control area	• Diarrhoeal morbidity reports	• Children of intervention mothers experienced an 89% reduction in diarrhoeal episodes
Pinfold and Horan (1996) Thailand	• Households in 37 villages allocated to different groups as follows: - 12 villages = control - 13 villages = low cost intervention - 12 villages = high cost intervention	• Washing dishes immediately after eating • HW before cooking, eating and feeding • HW after defecation and cleaning baby's bottom • Diarrhoea not mentioned specifically in the intervention message • Motivation given as avoiding 'germs' and having healthy children	• Variety of media used to communicate message: posters, stickers, slideshows, T-shirts • Songs in traditional folk style designed and played via loudspeakers in village • Plastic containers distributed to facilitate HW • Soap distributed in high-cost area only • Local workshops and community meetings • School activities and poster competitions	• Knowledge and adoption score of key behaviours • Fingertip impression in agar plate for sub-sample of 45 households • Morbidity reports	• After intervention HW knowledge scores expressed as a percentage were 44%, 55% and 60% for control, low-cost and high-cost intervention groups respectively • Mean differences between fingertip contamination before and after intervention were 34%, 55% and 65% for control, low-cost and high-cost intervention groups respectively • 39% reduction in diarrhoeal rates in intervention area

Intervention	*Target Group*	*Message*	*Methods*	*Outcomes*	*Impact*
Ahmed et al. (1993) Bangladesh	• Households with children <19 months • 185HH in both intervention and control groups	• Numerous messages focusing on ground sanitation, personal and domestic hygiene and food hygiene	• Intervention messages translated into simple action messages based on local proverbs, poems and songs • Community lectures and demonstrations • Weekly educational sessions for groups of 3-5 women • Germ theory taught and women helped to identify own problems and solutions • Intervention run for 6 months	• Questions re hygiene knowledge and adoption of behaviours • Weekly morbidity reports • Assessment of cleanliness of environment, mother and child • Anthropometry	• Percentage of children and household environments rated clean increased by 54% in intervention area and just 4% in control • Reported diarrhoeal morbidity was lower in intervention than control area (data not provided) • Percentage of severely malnourished children after intervention was less in intervention than control area
Haggerty et al. (1994) Zaire	• Households with children aged 3-35 months • 9 intervention and 9 control villages	• Proper disposal of child and animal faeces • HW before cooking, eating and feeding • HW after defecation and cleaning baby's bottom	• Non-formal education sessions held with mothers • Village meetings • Home visits by fieldworkers • Use of songs, stories, proverbs and poems • Placebo intervention implemented in control site with equal intensity	• Child morbidity reports	• Few differences in diarrhoeal incidence noted between intervention and control groups • At peak diarrhoeal time, intervention children experienced 11% less diarrhoea • Evidence suggests intervention children experience diarrhoeal of shorter duration
Shahid et al. (1996) Bangladesh	• 1366 people in two slum areas of Dhaka	• HWWS before eating and after defecation or urination	• Soap and water provided to participants • Reinforcement visits every 2 days	• Diarrhoeal morbidity reports	• 43-73% reduction in diarrhoea in intervention area
Peterson et al. (1998) Malawi	• 402 households in a refugee camp in Malawi	• No educational message included	• 200g of soap distributed to households on a monthly basis	• Diarrhoeal morbidity reports	• 27% fewer episodes of diarrhoea in households when soap was present compared to when no soap was present (RR=0.73, 95%CI: .054-.098)

Intervention	*Target Group*	*Message*	*Methods*	*Outcomes*	*Impact*
Curtis et al. (2001) Burkina Faso	• Mothers • Older sisters • 'Maids' • School children	• Safe stool disposal • HWWS after defecation or cleaning baby's bottom • Emphasis on hygiene as social virtue, rather than link with disease and diarrhoea	• Launched with municipal ceremony, mass clean up of public areas and radio phone in • Monthly house-to-house visits • Participatory discussion groups with Health Centre staff • Neighbourhood meetings • Youth theatre plays • Comic radio spots • Curriculum and materials for 6 primary school hygiene lessons • Project ran for 3 years and cost $302,507	• Observations of stool disposal • Observations of hand-washing after defecation and cleaning baby's bottom	• Increase in children using potty from 74% to 82% • Increase in no. mothers HWWS after using latrine from 1% to 17% (using just water from 33% to 67%) • Increase in no. mothers HWWS after cleaning baby's bottom from 13% to 31% (using just water 35% to 74%) • No change in the use of latrines for the disposal of faeces
Luby et al. (2005) Pakistan	• Households with children <15 years • Separated into 25 intervention communities and 11 control communities	• HWWS before food preparation, eating or feeding child • HWWS after defecation or cleaning baby's bottom	• Initial meeting held in small groups to show video, slides and pamphlets • Local meetings 2-3 times a week for mothers, reducing to once weekly from 2-9 months and fortnightly in the last 3 months • Monthly meetings for 1st 3 months for men • Weekly home visits by fieldworkers • Provision of soap • Placebo intervention implemented with equal intensity in controls	• Morbidity reports for diarrhoea, pneumonia and impetigo	• Intervention children had 53%, 50% and 34% lower incidence rate for diarrhoea, pneumonia and impetigo respectively than control children

Table 3.2 Key features of community-based hand-washing interventions in developing countries

All but one intervention used child morbidity rates as an **outcome** variable, and some also included anthropometric assessments of growth performance over the intervention period. All interventions reported improvements in the outcome variables after the intervention period, though this is perhaps reflective of the reluctance of researchers and journal editors to write or publish papers where interventions have failed (Cave and Curtis 1999). Curtis et al. (2001) reported a 39% improvement in hand-washing rates after cleaning a baby's bottom, though the improvement for hand-washing *with soap* was less significant (18%). Impressive reductions in incidence rate were observed for diarrhoeal infections (in the range of 11-89%), as well as for other diseases such as pneumonia and impetigo.

3.4 Specific design of this study

The intervention for this project aimed to promote hand-washing amongst mothers of young children at five key junctures where faecal contamination could occur. The design of this intervention was informed by the theoretical model of behaviour change described in Section 3.1 and sought to change attitudes and social norms and increase self-efficacy in order to promote hand-washing. A variety of strategies and methods were employed in order to achieve this behaviour change, drawing on ideas from other hand-washing interventions in developing countries.

The intervention comprised of three phases: a preparatory stage – where in-depth interviews and focus groups were conducted to understand local perceptions of and attitudes towards hygiene and child health; a planning stage – where these data were analysed and the intervention message and activities were formulated; and finally, the implementation stage, lasting for six months. Figure 3.3 graphically depicts the intervention design and Table 3.3 explains how these activities fulfil the key features of successful interventions identified in section 3.2.

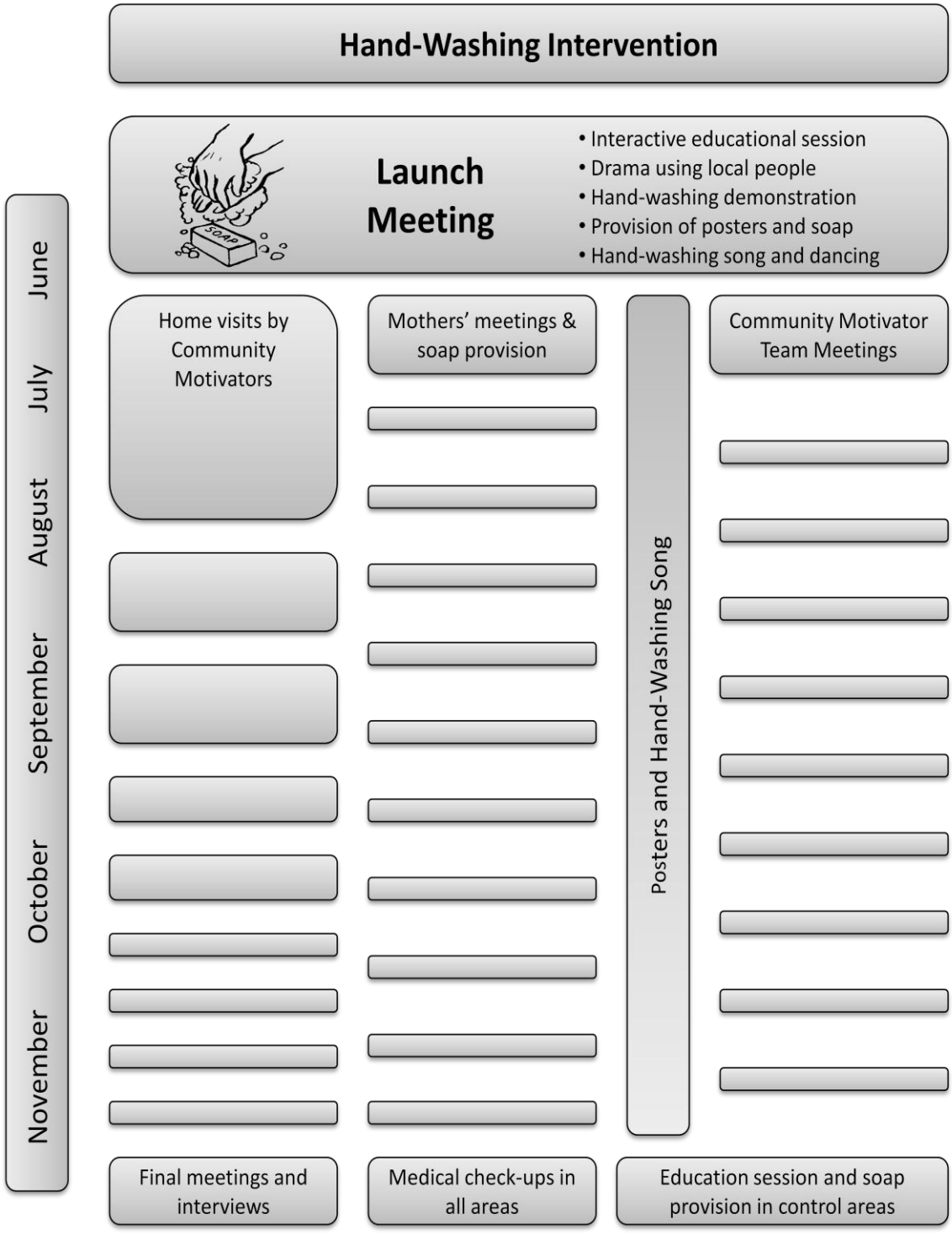


Figure 3.3 Hand-washing intervention design. Bars represent the relative frequency of each activity.

Key Features of Compelling Interventions	How this was achieved in this Intervention
Principles	
Underpinned by theory of behaviour change	Intervention underpinned by theoretical model outlined in Figure 3.1
Community engagement and participation	Local women employed as Community Motivators to design and implement intervention; use of local people in drama
Strategies	
Build upon local practices	Local hand-washing practices identified through observations, interviews and focus groups
Focus on outcomes relevant to target audience	Relevant outcomes identified through interviews and focus groups
Linked to social and cultural narratives	Identified by interviews and focus groups and used in songs and drama
Use inter-personal support and role models	Community motivators employed to act as role models and support change; mothers' groups formed to offer mutual support
Address constraints on human agency	Soap provided to all families on regular basis; support of mothers-in-law and husbands developed
Target those most receptive to message	Intervention primarily targeted mothers but also extended to husbands, mothers-in-law and older children
Messages	
Personalised messages	Daily home visits by Community Motivators to each mother
Target specific behaviours at specific contexts	Targeted hand-washing with soap at five key junctures
Repeated frequently in different formats	Message repeated at educational sessions, home visits and group meetings through discussion, drama, songs and posters

Table 3.3 Key features of compelling interventions and how these were achieved in this study.

3.5 Phase I: Preparation

If one is to effectively and compellingly influence local attitudes and norms to increase hand-washing practices, one must first understand *what* people currently do and *why* they do this. The preparatory stage of the intervention therefore aimed to:

- Identify current hand-washing practices amongst the mothers.
- Investigate the mothers' perceptions of hand-washing in terms of attitudes, subjective norms and perceptions of self-efficacy.

3.5.1 Current hand-washing behaviour

Current hand-washing practices of the mothers were identified through i) structured observations and ii) self-reported behaviour recorded during interviews and focus groups.

i. Observations of hand-washing behaviour[13]

Hand-washing with soap did not appear to be a routine practice amongst the observed mothers. Only a fifth of mothers were observed to wash hands with soap after defecation and only 14% used to soap after cleaning the baby's bottom. Hand-washing with soap before cooking or feeding the baby was almost never practiced: of the 75 mothers observed, only two were seen to wash hands with soap before handling food and none washed her hands with soap before feeding the child. Thus, structured observations suggested that hand-washing with soap at the five key junctures was very low and could stand to be substantially improved.

ii. Self-reports of hand-washing behaviour

Self-reports of behaviour are often unreliable, subject to poor memory recall and over-reporting of 'correct' behaviour (Cousens, Kanki et al. 1996). Not surprisingly therefore, in this study self-reports of hand-washing behaviour were consistently much higher than observed rates. Whilst producing less reliable results on *actual* practice, these reports are

[13] The results from the structured observations are presented in full in Chapter 4, but are briefly summarised here to provide context for the intervention design.

nonetheless useful in that they provide important information on what people think *should* be happening. Thus, they provide an important insight into 'ideal' hand-washing behaviour.

During interviews and focus groups mothers were asked to name all junctures (before and after which activities) at which they washed their hands. For each juncture mentioned, they were asked to specify how they washed their hands – with water, soap, mud/ash etc.

Hands were reportedly washed with water alone after getting up in the mornings; before and after eating food; after cleaning or doing housework; and before leaving the house to go somewhere. A few mothers also mentioned washing hands with water before cooking food, but this practice was generally uncommon. During focus groups, it emerged that most mothers felt that washing hands before cooking was unnecessary. As their first task was to wash the rice in cold water, they felt this action was sufficient to remove dirt from their hands before touching other foods.

Whilst most mothers felt that they washed their hands with water many times in the day, hand-washing with soap occurred less frequently and only at specific junctures; after coming into contact with faeces and when hands were visibly dirty. Soap was used at these junctures because it was most effective at removing faecal matter, dirt, germs and bad smells; water alone was simply not sufficient to clean hands when they were so soiled.

In contrast to the observational results where only a fifth of mothers were seen to wash hands with soap after defecation, virtually every mother claimed to always hand-wash with soap after defecation; only two mothers from the intervention areas admitted they did not always do this. Similarly, most mothers also said that they washed hands with soap after cleaning the baby's bottom, though during observations only 14% were observed to have done so. The mothers explained that they only washed their hands with soap if they had used their hands to clean the bottom; in most cases the mothers said they simply wiped the child's bottom with a rag and so did not feel that it was necessary to wash hands at this time. As one mother

explained, she only washed her hands with soap if she felt that they had faeces on them, otherwise it was not necessary[14].

Admittedly, using a cloth to wipe the bottom would reduce the chances of faecal contamination. However, there was still an often considerable risk of faecal contamination of the hands even when using a cloth; the cloths used were usually very thin and torn and the often liquid state of the child's stools (either from being exclusively breast-fed or from diarrhoea) meant that faecal matter could easily soak through the cloth and contaminate the hands, as demonstrated by the case study below.

Box 3.1 Interview with Sarala Karki.

Sarala is the mother of five month-old Alok. On the day of the interview, Alok is sick. He has had severe vomiting and diarrhoea for the past five days. He seems dehydrated and lethargic. Sarala is holding Alok in her arms, and as we talk he has another bout of diarrhoea – the stools are thin, watery and yellow. Alok is wrapped in a shawl but the stools run out from underneath this onto the floor. Sarala uses the shawl to wipe up the stools from her son's bottom and legs but as it is so watery it penetrates the cloth and contaminates her hand. She obviously notices this as she rubs her fingers together and then rubs them on her own sari. The interview continues and later the child is playing with his mother's fingers and chews and sucks on them. This is the same hand that she used to wipe his bottom earlier…. We conclude the interview early as the mother is going to take Alok to the hospital today.

In addition to washing after contact with faecal matter, mothers also indicated that hand-washing occurred when hands were visibly dirty or greasy - for example, after cleaning the

[14] The structured observations did not distinguish between hand-washing after cleaning the baby's bottom with the hand or with a rag and so it is not possible to compare these self-reports of hand-washing with actual observations.

house, working in the garden, or eating greasy food. At these times the hands looked or felt particularly dirty; soap was therefore necessary, since water alone would be unable to remove this dirt.

3.5.2 Attitudes, norms and self-efficacy: Local perceptions of hand-washing

Following the theoretical model described in Figure 3.1, interviews and focus groups were used to identify local attitudes, subjective norms and perceptions of self-efficacy in relation to hand-washing in order to inform the design and implementation of the intervention.

i. Attitudes

As specified by the theoretical model, it was important to understand the mothers' overall attitudes towards hand-washing with soap, in order to identify what promotes or prevents this behaviour. Eliciting the mothers' attitudes towards hand-washing involved identifying both the positive outcomes they believed would arise from performing this behaviour and the negative outcomes that might have occurred should they fail to act (outcome evaluation), as well as exploring their confidence and certainty that hand-washing with soap could achieve these outcomes (outcome expectancy).

The strongest motivators for hand-washing with soap were framed in the negative – i.e. by referring to negative outcomes that would occur if they did not wash their hands with soap. Prevention of disease was by far the most commonly and strongly cited reason for hand-washing. All mothers interviewed stated that they washed their hands with soap after defecation because failure to do so would result in sickness – both in oneself and in one's family. The types of diseases the mothers felt hand-washing with soap could prevent centred on the most commonly-experienced diseases in the area – diarrhoea, dysentery, vomiting, stomach-aches, colds, pneumonia etc. About a fifth of mothers could not name any disease that would be prevented by hand-washing but still emphatically stated that it could prevent sickness. Some of the more educated mothers were able to provide fairly accurate accounts of how hand-washing with soap prevents sickness, with about a third of mothers specifically mentioning bacteria (*kitanu*) in describing the link between hand-washing and health. As one mother explained,

You must wash your hands before you eat food or you will get sick. Your hands may have bacteria on them and if they get inside you, you will get diarrhoea…Bacteria are small kiraa [insects/organisms] – *so small you can't see them… It's because you can't see them on your hands that you eat them.*

Sarita Limbu, mother of Jyoti Limbu

For most mothers, protecting the health of their family was the primary motivation for hand-washing with soap after defecation or contact with children's faeces. Because they were primarily responsible for child care and the feeding of the family it was especially important that they follow this practice. Several mothers explained that because they lived in such poor and dirty areas good hygiene was all the more important: simply by living in such a bad area they were endangering their health, and so every effort should be made to protect and enhance it. Some women also added that it was better to take a bit of trouble now to be clean, than to have to find money for treatment when someone fell sick. Thus good hygiene was seen as a way of averting future costs.

Hand-washing was also strongly motivated by notions of disgust. The mothers explained that failing to wash hands with soap after defecation would make them feel 'disgusting', 'wrong', 'sick', 'dirty', 'uncomfortable'. Hand-washing with soap after defecation was therefore clearly motivated by internal feelings of disgust and the desire to be clean. Similar sentiments were expressed regarding the need to wash hands after contact with their child's faeces, although the revulsion at failing to do so was not as strong, as mothers generally held the belief that children's faeces were less dirty and less harmful than adults'. By contrast, using soap to clean hands made them feel 'nice', 'clean', 'fresh', 'light', 'at ease'. Only soap could give them that 'really clean' feeling and many mothers mentioned having soft, nice-smelling hands as a positive outcome of hand-washing**.**

With regard to outcome expectancy, most mothers were confident that hand-washing with soap (and good hygiene in general) could reduce diseases such as diarrhoea in both themselves and their child. However, there were times when this connection seemed less concrete in their minds. Though virtually all mothers made a link between health and hygiene, many also cited examples when this link did not seem to be so clear-cut. For example, one woman explained,

I don't understand it. I am so careful about my children. I pay great attention to keeping them clean, washing their hands, their faces, giving them good food, clean clothes – and yet they still get sick.

Sita Gurung, mother of Durga Gurung

Similarly, another woman said**,**

You see these children running around. They never wash their hands after going to the toilet, they wear filthy clothes…and yet they never seem to get sick…I'm so surprised by this. I don't understand it.

Aruna Poudyal, mother of Ajay Poudyal

Interviews with the mothers regarding child health and illness revealed that although good hygiene was seen as a way of preventing sickness, children could contract diseases such as diarrhoea for many different reasons, many of which the mothers had no control over at all. Diarrhoea, colds, fevers and other diseases were commonly attributed to changes in the weather, the cold (*chiso* – see below) or evil spirits. Belief in evil spirits was particularly strong in Shanti Nagar, a settlement sited downstream from the holy Pashupatinath temple – the holiest Hindu temple in Nepal. Bodies are often cremated at the temple and the ashes are swept into the Bagmati River below that runs directly through the Shanti Nagar settlement. Many mothers attributed the high frequency of sickness and diarrhoea in the area to the influence of spirits that come down the river to the settlement. Similar beliefs in evil spirits were held in other settlements too; one woman believed a particularly severe episode of diarrhoea in her child was the result of him being touched by a woman who had recently lost her own child – the spirit of the dead child was believed to have been transferred into the little boy and had made him sick. Narratives such as this were commonly repeated by mothers from all areas. However, there was disagreement between the mothers as to how much childhood sickness could be attributed to these supernatural causes. Some believed that most sickness was simply a result of poor hygiene, with spiritual sicknesses occurring only infrequently. Others believed that much of the disease burden in the area was caused by spirits and therefore hygiene and hand-washing were unlikely to do much to prevent such diseases in children.

ii. Subjective norms

Hygienic behaviour, including hand-washing with soap, was also motivated by strongly felt social norms and expectations. It was important for the women to be thought of as good mothers who looked after their families well and achieving this required high standards of hygiene. The mothers felt they should be clean and well-presented at all times: old, worn clothes were acceptable, but dirty clothes were not. Similarly, their children's hands and faces should be washed whenever they were dirty and they should be dressed in clean clothing. Hand-washing with soap after contact with faecal matter was also an essential part of being seen as a good, clean, responsible mother. Thus, it was clear that for these mothers cleanliness carried with it strong moral connotations. When asked to describe what a person who did not wash their hands after defecation would be like, the mothers firstly focused on the person's physical appearance (unkempt, dirty, smelly) but swiftly moved on to describe her in more morally-loaded terms. For example, a 'dirty' mother had children who were wild and out-of-control; she did not take care of them properly because she spent all day watching films or gossiping with her friends; she and/or her husband were probably alcoholics; she was the victim of domestic abuse, etc.

Most mothers said they knew people who did not wash their hands after defecation – including other mothers enrolled in the project – but, as mentioned above, only two mothers admitted to this themselves. The fact that there was a large discrepancy between observed and self-reported hand-washing with soap rates after defecation (19% vs. 96%) under-scores the social value placed on hygiene: the mothers clearly knew they should be washing their hands with soap at this juncture, even if they did not always do so.

Being thought of as a clean person fostered a sense of pride and self-respect in these mothers, as demonstrated by the example below.

Box 3.2 **Interview with Nirmala Tamang**

Nirmala lives in a tiny rented room – probably measuring just six-by-four feet – with her husband and two children. What strikes me as we enter the dark room is just how incredibly tidy and well-ordered it is. The room contains minimal furniture – just a bed, some shelves, two water buckets and some cooking equipment – but absolutely everything is neatly and exactly placed. All the family's clothes and possessions (few though they are) are neatly stacked on the shelves. The bed is neatly made, the pots are shiny and freshly scrubbed and the floor is completely spotless. I wonder if she had tidied especially for us, but then remember that we were not supposed to be interviewing her today so she could not have known we were coming.

I comment about how tidy the room is and she says that she makes a special effort to be clean and tidy at all times. The other people who rent rooms in this house are very dirty but she likes everything to be exactly right. Beaming with pride she explains that the house-owner will never take tea or food with the other families when he comes to visit but he is always happy to accept food from her as he knows she keeps such a clean house. She says it gives her a sense of pride and respect for being known as someone who keeps a clean house.

iii. Self efficacy

When asked if they felt they could increase the number of times in a day when they washed hands with soap, most mothers were confident that they could achieve this. Neither the extra water nor soap required for hand-washing was presented by the mothers as a significant barrier to hand-washing. Although drinking water was scarce, none of the women reported a shortage of water used for domestic purposes, even in the height of the dry season; water fit for hand-washing purposes was always available from local tube wells or deep wells. Similarly, lack of soap was not reported as a barrier to hand-washing: soap was a standard household item

possessed by even the poorest families. However, it was noted by some mothers that if the entire family started washing their hands with soap more frequently they would require a greater amount which, for the poorest families, could be a strain on tight resources. As one woman explained, the poorest families were forced to live from one day to the next and if it was a choice between spending ten rupees on soap or ten rupees on food, they would choose food.

However, by far the greatest barrier to hand-washing was the widely held belief that hand-washing with soap was simply not necessary before cooking, eating or feeding the baby. Very few mothers (12%) reported washing their hands with soap before starting to cook a meal; 86% claimed they washed hands with water before cooking, but as mentioned above, in most cases this simply comprised of the hands being 'passively' washed whilst they were rinsing and soaking the rice. Hand-washing with soap was similarly uncommon before eating or feeding the baby: in both cases the mothers usually washed their hands with water alone.

When questioned about this practice, the mothers explained that hands were usually not perceived to be very dirty at these times and so water alone was sufficient to cleanse the hands. If hands were visibly soiled soap would be used, but in the majority of cases this was deemed to be unnecessary. Even those mothers who knew about bacteria and were aware that they were too small to be seen by the naked eye reported this opinion. These mothers knew that even clean-looking hands could be covered in germs, and yet they felt soap was not necessary at these junctures. Furthermore, unlike hand-washing after defecation, there was clearly no social expectation or compulsion to use soap before cooking, eating or feeding the baby. As one woman explained,

> *We wash our hands with soap after we go to the toilet because it is our habit. It is what we were taught to do. But before cooking – it isn't necessary. It isn't our habit to use soap then.*
>
> Meena Rai, mother of Sunita Rai

Similarly, hand-washing with soap before feeding the child was deemed to be unnecessary. Not only were hands not seen as particularly dirty, but many mothers also tended to feed the

child with a spoon rather than with her hands. Since there was no contact between the hands and food, hand-washing was felt to be redundant.

Other barriers to hand-washing with soap were that, compared to washing with water alone, it took greater time and effort. Whereas washing hands with water was perceived as quick and easy, hand-washing with soap was often deemed to be a bit of a chore: you had to go outside, it took much longer to clean hands properly and required much greater amounts of water to rinse all the suds away. Although mothers said they did this after defecation, it seemed like too much effort to go through all this at other times (like before cooking) when it simply did not seem necessary.

Poverty and difficult circumstances were also seen as a barrier to hand-washing and good hygiene in general. The women commented that mothers in the poorest families often had to work all day, leaving their children unattended, and often received little or no support from their husbands. In such difficult circumstances, priority was given to simple survival rather than good hygiene and hand-washing practices.

Finally, a fear of '*chiso*' or 'cold' was sometimes mentioned as a barrier to increased hand-washing. There was a widely held belief amongst the mothers that many childhood (and adult) sicknesses were caused by *chiso* entering the body. One way in which *chiso* could enter a child was through the mother's breast milk. If the mother spent a great deal of time with her hands in cold water, the *chiso* could enter her body and be transmitted through the breast milk into the child, where it could cause fevers, diarrhoea, vomiting or coughs and colds. Some mothers felt that if they spent much more time washing their hands they would be more susceptible to catching this *chiso* and passing it onto their child.

3.6 Phase II: Design of intervention message and activities

3.6.1 Community involvement

As noted in section 3.2, the most successful interventions are those that engage with the local community, drawing on their ideas and enthusiasm for change. It was therefore crucial that local people were involved in the planning and implementation of the intervention programme. Following a strategy that had been used in a number of successful hand-washing interventions (Han and Hlaing 1989; Haggerty, Muladi et al. 1994; Shahid, Greenough et al. 1996; Luby, Agboatwalla et al. 2005), a local woman was selected from each intervention site to act as a Community Motivator. It was the responsibility of these women to implement the intervention in their local area, encouraging and promoting hand-washing with soap amongst the mothers enrolled in the study. As it was these women who would be primarily responsible for the implementation and success of the intervention, their input and advice into the design of the programme was crucial to the success of the project. The intervention's message and activities were therefore designed by an intervention team consisting of the five Community Motivators, two research assistants/translators and myself. It was also informed by the information gathered during the observations, interviews and focus groups combined with the local knowledge and experience of the Community Motivators.

The Community Motivators needed to be well-respected, well-known local women who were active in their community and who would have the appropriate skills and attitudes needed in order to promote behaviour change in the mothers. During the interviews and focus groups, mothers were asked to suggest local women who would fit these criteria. These women were then approached and interviewed informally regarding the position of Community Motivator and a woman was selected for each of the intervention sites[15].

The Community Motivators underwent two weeks of interactive training with myself and the other research assistants. The first week of training included educational sessions on germ theory, motivational techniques, theoretical models of behaviour change, communication skills,

[15] As there were considerably greater numbers of women in Shanti Nagar, three women were recruited to cover this area. The settlement was naturally divided into three areas (upper, lower and bridge) and the Community Motivators were each allocated to an area.

problem solving and ethics. During the second week the qualitative data from the interviews and focus group were analysed by the whole research team and the specific intervention messages and activities were designed and planned.

3.6.2 Focus of intervention message

The specific content of the intervention message was determined by the need to reduce the children's exposure to faecal material and therefore concentrated on promoting hand-washing with soap at the five key junctures previous mentioned. However, this message needed to tap into the most compelling motivators for hand-washing, change social norms and address any barriers to behaviour change. The way in which the message was couched was therefore informed by the beliefs and ideas expressed by the mothers during the interviews and focus groups conducted in Phase I. The final formulation of the intervention message is summarised in Figure 3.4.

The strongest motivating factor for hand-washing with soap identified by the mothers was to protect and promote health (*swastha*). This was mentioned by every mother as the key motivator for hand-washing behaviour and so became the primary motivating message for the intervention. Mothers were encouraged to wash their hands at the five key junctures to prevent their children (and themselves) from coming into contact with faecal matter that could cause diarrhoea, coughs/colds and fevers. This message was summed up in the slogan *'haat dhaau, swastha rachau'* which translates roughly as, 'Let's wash hands – it makes us healthy'.

As many mothers believed that diarrhoea was an almost inevitable part of childhood, often caused by evil spirits attacking the child, special attention was paid to this issue during the intervention. Educational sessions were held with the mother to teach them about the faecal-oral transmission route and to help them identify for themselves risky practices. As the belief in evil spirits was so strongly entrenched in most areas it was decided that the intervention message should not set itself up in direct opposition to these beliefs. Therefore, the mothers were told that although some diarrhoea could be caused by spirits, the majority was caused by lack of hygiene and could therefore be prevented. Thus, by not simply rejecting the notion of

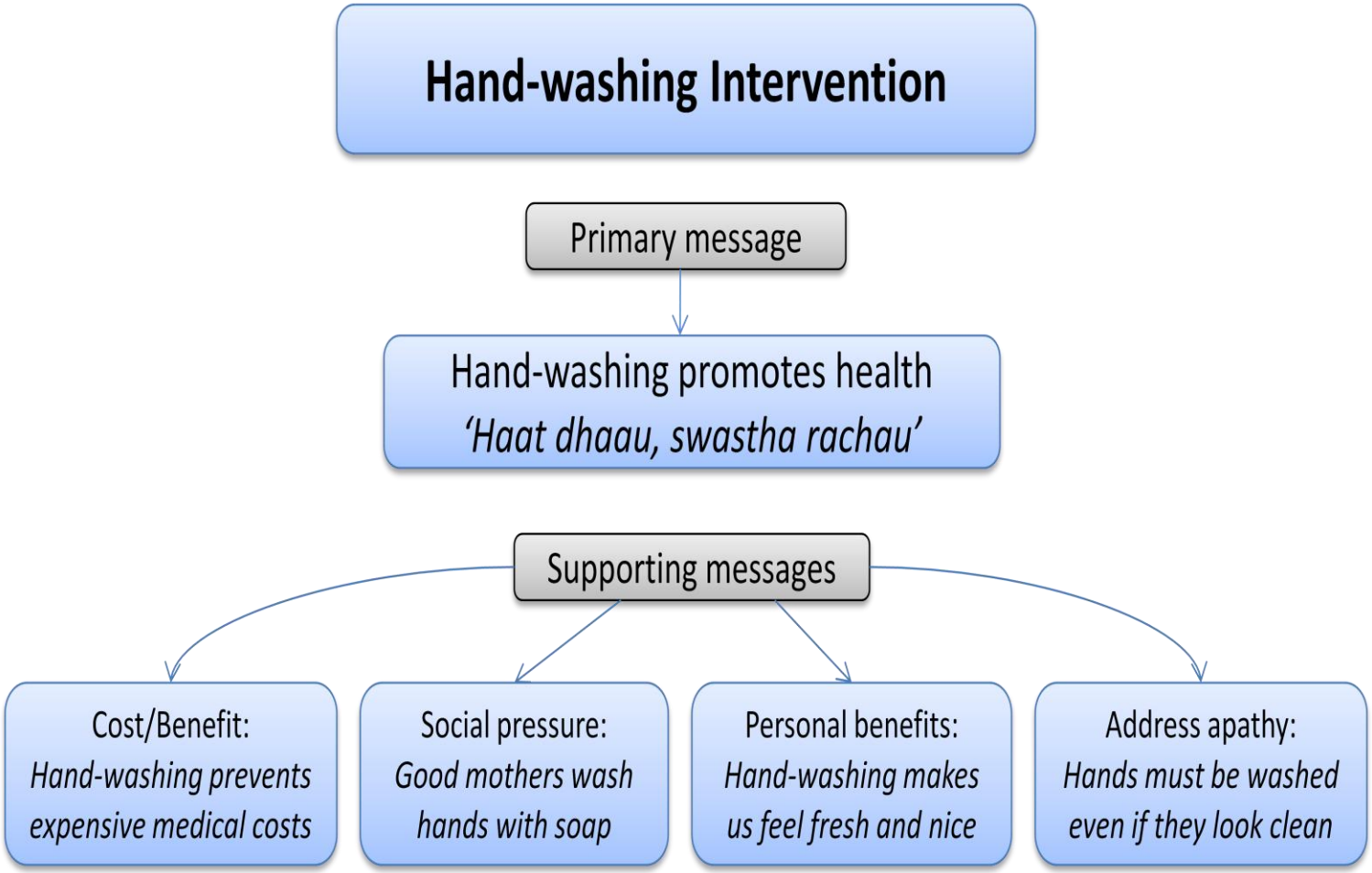


Figure 3.4 The primary and supporting intervention messages.

diarrhoea caused by evil spirits, the intervention was made more acceptable and credible to the mothers.

In addition, following comments made by several mothers, it was decided that the message needed to emphasise the financial benefits of improved hygiene: prevention of sickness through hand-washing would save money on consultations with doctors and purchase of medication when a family member fell sick. Given the financially-vulnerable position of many families living in the slums, this risk-averse strategy proved quite compelling.

Though protection and promotion of health was presented as the primary motivator for hand-washing, the interviews and focus groups had also identified other important motivating factors that influenced hand-washing behaviour and so the intervention sought to incorporate these messages too as subsidiary and supporting motivators. The first of these aimed to promote and encourage the social pressure to hand-wash with soap by emphasising the attitude that this was what 'good', 'clean' mothers do and that eating without first washing ones hands with soap, for example, was simply disgusting. Several mothers commented that seeing other mothers take extra care with hygiene and cleanliness would motivate them to improve their own practice as they would not want people suggesting they weren't as clean as others. The Community Motivators felt that harnessing this sense of social competitiveness and rivalry – by promoting a sense of 'keeping up with the neighbours' – could be a very effective way of increasing hand-washing practices amongst the mothers.

Secondly, the intervention sought to stress the positive personal benefits of hand-washing with soap. The intervention stressed how cleanliness, and hand-washing in particular, made one feel 'good', 'clean' and 'right'. Hand-washing was therefore a way of raising the self-esteem in the mothers and creating a 'demand' for good hygiene amongst these mothers.

The qualitative interviews also identified a number of beliefs, attitudes and norms that might act as barriers to hand-washing with soap and which needed to be addressed by the intervention campaign. As explained above, most mothers simply did not believe that hands were dirty enough to warrant the use of soap before handling food. The intervention therefore aimed specifically to address this belief by educating the mothers about germ theory and how hands could still be highly contaminated even when they looked clean. It also specifically

reminded the mothers that hand-washing with soap was necessary before feeding the child, even if they used a spoon, since the mothers often used their fingers to wipe up dribbled food from the child's mouth and contamination could occur this way. Similarly, it was emphasised to the mothers that hands also needed to be washed with soap after wiping the child's bottom with a cloth, since faecal material could inadvertently get onto the hands without the mother realising it.

3.6.3 Strategies for improving hand-washing rates

The specific activities to promote hand-washing with soap amongst the mothers were designed by the intervention team and were informed by the theoretical model of behaviour change, the strategies used in other hand-washing interventions, the data collected during the interviews and focus groups, and the experiences and ideas of the Community Motivators and mothers themselves.

The mothers (during the interviews and focus groups) and the Community Motivators provided many suggestions for making hand-washing with soap easier to do. The mothers suggested that they would need someone to convince them of the need for hand-washing and that it would be useful to have someone who would come and remind them to do it in the early stages as they were creating this new habit. Both the mothers and Community Motivators also felt that it would be useful to create a group of women who were trying to increase hand-washing rather than just having each mother attempt to change her behaviour on her own. These groups would enable the mothers to get to know each other and they would be able to offer each other encouragement and support. The Community Motivators also noted that such groups would be good at creating a new hand-washing 'norm' for these women by providing them with a new social group that expected and encouraged hand-washing.

Other practical suggestions were made in order to facilitate hand-washing. Some mothers suggested that having taps in their own houses would make hand-washing much easier. At present most women had to use water poured from a jug for hand-washing. This was difficult because it meant you could only wash one hand at a time whilst holding the jug in the other. Keeping the soap and a water container near the toilet was also suggested as a way to promote

hand-washing. For most women, soap was kept in their house and they had to remember to collect it before visiting the toilet, which could be some distance from the house. Having both soap and water within easy reach of the toilet would both remind and facilitate hand-washing.

These suggestions, though eminently sensible, were not feasible activities for this project. Providing running water in the houses would undoubtedly assist hygiene behaviours but was completely beyond the scope and budget of this intervention. Similarly, provision of soap and water in each toilet was not possible. The majority of families shared access to their toilet with other families and, as the mothers themselves pointed out, no-one would be prepared to be responsible for maintaining the supply of soap and water for these communal toilets. In addition, the mothers explained that any soap and/or containers in the toilets would almost certainly be stolen, destroyed or lost by children playing with them.

3.7 Phase III: Implementation of intervention

The final design of the intervention activities was based upon:

a) An **initial launch meeting** which introduced the mothers to the intervention programme, and promoted hand-washing with soap through inter-active educational sessions, hand-washing demonstrations and a short play.

b) **Repeated activities** sustained throughout the six months of the intervention period that included home visits, mothers' group meetings, soap provision and intervention team meetings.

c) **Other means of reinforcing the message** including the use of posters and songs.

3.7.1 Launch meeting

The Community Motivators felt that it was important to have a special event to mark the launch of the intervention. This event would provide an opportunity to for all the mothers to meet one another and their local Community Motivator, as well as providing an ideal time to introduce the hand-washing message and act as a trigger for behaviour change. Launch

meetings were therefore held in each community in a local communal space (usually a school room) at the beginning of June 2007. All mothers from the local area enrolled in the project were invited to attend. Since family support for the intervention was important in supporting and sustaining behaviour change, mothers-in-law, sisters, husbands and children were also invited to attend.

i. Educational session

The launch meeting started with me welcoming the families and introducing the research team and the Community Motivator for that area. The Community Motivator then gave a short and interactive educational presentation explaining how germs can be transmitted into the body and how hand-washing with soap could prevent this. For this presentation the Community Motivators made use of an educational flip chart that had been specially designed by UNICEF to promote hand-washing with soap in non- or semi-literate populations in Nepal. During the training week the Community Motivators had worked together on the delivery of this presentation, making it as lively and interactive as possible. The five key hand-washing junctures were reiterated several times during this session and the Community Motivators promoted the message using the agreed upon motivators – that hand-washing with soap was what 'good' mothers did and would protect and promote the health of their children and families.

ii. Hand-washing demonstration

Because so many mothers felt that hand-washing with soap was unnecessary before handling food, it was decided that this belief needed to be explicitly addressed during the meeting. A comedic 'skit' therefore followed the Community Motivator's presentation demonstrating how even clean hands can be covered in germs and how these can easily be transferred to food. In this skit, red paint was used to represent the bacteria on the hands of a mother (played by myself) after failing to wash her hands after defecation. As this mother set about cleaning her face and preparing food, red paint was transferred to everything she touched. Thus it visually (and amusingly) represented to the mothers how easily bacteria could be transferred to food which was then fed to her child and husband. Following this demonstration the mothers were shown correct way to wash their hands to ensure maximum bacteria removal and were invited to practice this technique.

iii. Hand-washing play

There then followed a short play performed by local children and adults. Street theatre has been found to be a very effective method for engaging people, imparting information and encouraging behavioural change (Galavotti, Pappas-DeLuca et al. 2001). The Community Motivators had seen this method used in their communities by other health initiatives in the past and knew that this was a very effective and well-received method: student nurses working in the area had used theatre to address issues of alcoholism and domestic abuse and people had talked for weeks about what they had seen. Using local people was also suggested by the Community Motivators as being more effective since people would recognise their own friends and family in the cast. It was not possible to use local people from each area due to time and logistical constraints. However, the actors for the drama were selected from the largest intervention community - Shanti Nagar - and so were 'local' for the majority of mothers.

A drama teacher from a Kathmandu secondary school with experience of directing educational street theatre was commissioned to write and produce a short (15 minute) play to promote hand-washing at the five key junctures in a stimulating and amusing way. Local adults and children were selected through informal auditions to act in the play and rehearsals were held in the week prior to the intervention's launch. The play featured a small boy who had been suffering from diarrhoea for several days and who had been experiencing nightmares about monsters making a home in his body and making him sick. His grandmother was convinced that her neighbours had put the evil eye on the child but when the *dhami jhankri* (shaman) arrived to examine the boy, he realised that in fact this was a simple case of diarrhoea caused by the whole family failing to wash their hands with soap. During the play the five key junctures when hand-washing with soap should occur were reiterated several times to help promote the intervention message. The play was a useful way of promoting the hand-washing message in an informal and amusing way and was very well-received by the audiences in each intervention site.

3.7.2 Regular activities throughout intervention period

i. Daily home visits

The launch meetings were followed the next day by the start of the home visits by the Community Motivators. As mentioned above, several of the mothers suggested that having someone come and remind them to wash their hands would help them establish this new habit. The primary role of the Community Motivator therefore was to visit the mothers and their families in their own home and to promote and encourage this new practice. Initially the Community Motivator visited each household every day for a period of two weeks, and then on alternate days for a further two weeks. This intensive contact was felt necessary at the start of the intervention to keep up momentum and help establish the new habits and routines. Later on, the frequency of these visits decreased until the mothers were visited just once or twice a week. The Community Motivators were encouraged to visit the mothers at different times of the day and to establish an informal and friendly relationship with the families. The Community Motivators discussed the intervention message with the mothers and other family members, focusing on key motivating factors identified during the interviews and focus groups. They discussed with the mothers any issues and practicalities that made hand-washing easier or more difficult. The Community Motivators worked with the mothers to identify solutions to problems and shared with them ideas and solutions that other women had found useful. During these home visits the Community Motivators established close relationships with the mothers and were also able to assess accurately those mothers who had taken on board the message and changed their behaviour and those who had not yet changed. The Community Motivators spent extra time with these mothers in order to promote and establish new hand-washing behaviours as far as possible.

The Community Motivators also made a point of talking to the husbands and mothers-in-law of the women to ensure that they understood the importance of hand-washing with soap and would encourage this practice within their own household. It was important to have the support of these key family members, since they have a great amount of influence over the mother and her behaviour. If the mother-in-law had felt that extra hand-washing was unnecessary and wasteful, the mother would have been severely limited in her ability to change her behaviour. Thus, by also specifically targeting key members of the family, the

Community Motivators aimed to increase the confidence and self-efficacy of the mothers to effect this behavioural change.

ii. Mothers' meetings

Drawing on an idea that was used successfully used in other interventions (Luby et al. 2005 in Pakistan; Ahmed et al. 1993 and Stanton and Clemens, 1987 in Bangladesh), all mothers were invited to attend a mothers' meeting every two weeks in their local area. These meetings provided an opportunity for all the mothers to meet with each other and their Community Motivator in an informal and sociable setting. The hand-washing intervention was discussed by the women, with each meeting usually focusing on a different theme. For example, the mothers might discuss how washing hands had made them feel and what they thought were the personal benefits from doing so or share their own strategies for how they remembered to wash hands before cooking food. It was at these meetings that the soap was distributed to all the mothers, though if any mother could not attend a meeting for any reason, the Community Motivators would ensure that she still received her bar of soap the following day.

iii. Provision of soap

At the end of the launch meeting the mothers were given a bar of soap to encourage hand-washing practices. A new bar of soap was provided to each family every two weeks thereafter. The choice and distribution of soap for the intervention families had been discussed in detail by the research team. The vast majority of mothers used laundry soap to wash their hands as this was easily and cheaply available. In the interests of sustainability of the behavioural change after the close of the intervention, there was an argument that the mothers should be provided with the soap they already used for hand-washing. However, the Community Motivators felt very strongly that providing laundry soap for the mothers would not be a big enough incentive to change. They explained that the mothers would simply use this laundry soap for washing clothes, saving money by not having to buy as many bars of soap in a month because of the intervention: hand-washing rates would be unlikely to change at all. Almost all mothers said that if they had a choice they would prefer to use body soap for hand-washing since it left the hands smelling nice and the skin soft. We decided therefore to provide mothers with free bars of body soap throughout the intervention. Mindful of the need for sustainability, we chose one of the popular yet cheaper brands – Lifebuoy (14Rs, approximately 10p) – and

also emphasised throughout the intervention (through home visits and mothers' meetings) that *any* soap was equally as good at ridding the hands of germs.

In addition, the team discussed how much and how frequently soap should be provided to the families. Recognising that body soap would also inevitably be used by the families for bathing and hair washing, we decided to provide each family one bar of soap every two weeks, following recommendations used in other hand-washing interventions (Han and Hlaing 1989; Shahid, Greenough et al. 1996). The team did discuss whether family size should be considered in allocating bars of soap. However, the Community Motivators felt that providing families with different amounts of soap according to the size of their family could become very complicated (since family members often came and went between Kathmandu and their natal villages) and might cause resentment among the mothers. One bar of soap every two weeks was a generous allowance which would ensure that no family ran out of soap during the intervention period.

iv. Research team meetings

In addition to the mothers' meetings, the whole intervention team met every two weeks in a central location. These meetings provided an opportunity for the Community Motivators to report back on their activities over the previous two weeks, discuss any problems they had encountered and share ideas between themselves. These meetings were very useful for monitoring the general success of the intervention so far. As the Community Motivators spent time with the mothers they started to build up an idea of those mothers who had increased hand-washing and those who had not yet done so. This therefore allowed them to spend more time with these mothers convincing them of the importance of hand-washing and addressing any barriers to change. The meetings were also very useful as the Community Motivators could provide feedback from the mothers regarding our monthly health checks, allowing us to identify and resolve problems as they arose. For example, in one area some of the mothers were concerned about what the blood drop samples that were being collected were to be used for. Although the purpose of the health checks was explained at the start of the study some women had heard rumours that you could sell blood and were worried about what was happening to their children's samples. Having been told this by the Community Motivators, we were able to call a meeting with all the mothers and explain again what the samples were

used for and how they were stored, as well as emphasising the mothers' right to withdraw from the study altogether at any point if they so wished.

3.7.3 Other means of reinforcing the intervention message

i. Poster

At the end of the launch meeting, the mothers were provided with two copies of a poster reminding them of the five key junctures when they should wash hands with soap. One poster was to be placed on their toilet door and the other on the wall of their kitchen or cooking area. The Community Motivators also displayed the posters in prominent locations throughout the area such as in schools, health centres and local shops.

Although UNICEF hand-washing posters depicting Nepali families were available for use by this project, the team felt that a poster designed specifically for this project would be more meaningful and therefore more compelling for the mothers. Various ideas and sketches were produced and discussed by the team. The final poster drew upon an idea that linked the five key junctures with the five fingers of the hand (see Figure 3.5 below) under the intervention's slogan of '*haat dhaau, swastha rachau'*. A local artist who had been commissioned to make the flash cards used in the focus groups was commissioned to produce the poster using the same cartoon-style design. A draft of the poster was shown to some local mothers not enrolled in the study to gain feedback before final production.

Figure 3.5 A local mother and her child standing next to the intervention poster.

ii. Song

Song and dance are very popular in Nepal and most festivals, community and family events involve much singing and dancing. The Community Motivators suggested re-writing the lyrics of a popular Nepali folk tune to promote the hand-washing message to the mothers. All the mothers and their family members were taught this song by the Community Motivators at the launch meeting for the intervention. The song was also often used by the Community Motivators to open or close the mothers' meeting and mothers would sometimes also start singing the song during the monthly health checks while they were waiting with their children. It was popular with the mothers, and an effective reminder of the intervention message because of its catchy tune and repetitive lyrics.

About six weeks after the launch of the intervention, the research team felt another activity was required to remind and renew the mothers' interest in the intervention. A mini-parade of local children was therefore organised in the different areas, with the children and research team marching through the local community singing this song and dancing for the mothers and their neighbours. This proved to be a very effective way of re-stimulating interest in the

project in the local communities and getting people talking about hand-washing. The lyrics of the song are presented in Box 3.3 below.

Box 3.3

The Hand-Washing Song

Lyrics by Sita Parajuli, to the tune of 'yi saano ***nani le'***

yi saano nani le	*These small children*
haat dhunchhan sabun paani le	*Wash their hands with soap and water*
khaana khaana basau hai sab satha	*Let's all sit together to eat*
khaana khaana agaaDi dhau haat la hai	*But before we eat we must wash our hands*
sabun paani le	*With soap and water*
yi saano nani le	*These small children*
haat dhunchhan sabun paani le	*Wash their hands with soap and water*
bachau aba kiTaanu baTaa	*Let's save ourselves from bacteria*
Toilet baTaa niskesi dhau haat la hai	*After going to the toilet we must wash our hands*
sabun paani le	*With soap and water*
yi saano nani le	*These small children*
haat dhunchhan sabun paani le	*Wash their hands with soap and water*
haat na dhoi khaanu ni pardaina	*Without washing our hands - we cannot eat*
sabun binaa kiTaanu mardaina la hai	*Without soap - the bacteria won't die*
sabun paani le	*Soap and water*
yi saano nani le	*These small children*
haat dhunchhan sabun paani le	*Wash their hands with soap and water*
swastha rahau birami pariela	*We must stay healthy, we might fall sick*
haat na dhoi khaaena mariela	*If we don't wash hands before we eat we may die*
sabun paani le	*Soap and water*
yi saano nani le	*These small children*
haat dhunchhan sabun paani le	*Wash their hands with soap and water*

Timetable of implementation

The intervention was launched in the three intervention sites (Shanti Nagar, Sinamangal and Palpakot) on the 8th and 9th June 2007. These launch meetings were then immediately followed by the start of the home visits by the Community Motivators, which decreased in

frequency throughout the intervention period. After two weeks the first mothers' groups were held and continued on a two-weekly basis until the close of the project at the end of November 2007. During the six months of the intervention, in both intervention and control sites, weekly morbidity reports were carried out by the morbidity fieldworkers, while health checks (growth, urine and blood-drop samples) were conducted every month. The project closed with final meetings held in all areas where the mothers were thanked for their participation and were provided with gifts for their children.

Summary

This chapter described the design and implementation of the hand-washing intervention programme. The intervention design was based upon a theoretical model of behaviour change, influenced by Fishbein & Ajzen's (1975) Theory of Planned Behaviour. During the preparatory stage of the intervention, observations, interviews and focus groups were carried out with local mothers in order to understand the mothers' attitudes, subjective norms and perceptions of self-efficacy with regard to hygiene and hand-washing practices. The intervention message and activities were design by a research team including local women who acted as Community Motivators for the project. The intervention used a variety of strategies to promote hand-washing with soap including home visits, group meetings, drama, posters and songs. The following chapter describes the socio-demographic, behavioural and health characteristics of the population sample as they were at baseline.

CHAPTER 4

Socio-demographic and health characteristics of sample

Introduction

This chapter provides an overview of the characteristics of the families and infants enrolled in the study as they were at baseline. It starts by describing demographic and socio-economic profiles of the sample and their feeding, child-care and hygiene practices. The baseline morbidity, biochemical and growth profile of the sample is then presented. Where appropriate, comparisons are made between this sample and Nepal as a whole, using data from the Demographic and Health Survey (MOHP, Ministry of Health and Population (MOHP) [Nepal] et al. 2007).

4.1 Socio-demographic characteristics at baseline

As expected, the families enrolled in the study were poor and experienced significant financial, environmental and social deprivation. Here I present a profile of the families at baseline (Table 4.1) and comment on the few differences between the intervention and control groups.

4.1.1 Demographic characteristics

Mean age of the children at baseline (May 2007) was 7.60 months (range 3.29-11.96, SD 2.38) with a male:female sex ratio of 1:1.1. Most children in the study were the first (35%) or second-born (39%) child in the family. On average, mothers and fathers were 24 (4.60 SD) and 28 (5.54 SD) years of age, respectively. All fathers and most mothers (73%) were born outside Kathmandu and had been in their current residence for an average of six and four years, respectively.

The majority of families (61.4%) were from the intermediate-level *Baishya* caste which comprises of the ethnic Hill Tribes (Gurung, Rai, Limbu, Tamang etc.) and the Newars (the

original inhabitants of the Kathmandu Valley). Just over a quarter of families (26.1%) were from the high caste *Bahun-Chhetri* group, and 12.5% were from the *dalit* (untouchable) group, broadly reflecting the overall proportions within the Nepali population (MOHP, Ministry of Health and Population (MOHP) [Nepal] et al. 2007).

4.1.2 Housing

Just over half (55%) of the families in the study owned their own house (but not the deeds to the land), while the rest lived in rented accommodation. Almost two-thirds (65%) of families also owned land in their natal villages and women would often return to the villages during harvest time or for important festivals. Most families had moved to Kathmandu to seek work, though some had left their villages to escape violence during the Maoist insurgency or because they had lost their lands in natural disasters (such as land-slides).

Most houses were simple brick constructions roofed with corrugated iron sheets, though some houses were only walled with woven bamboo or flimsy plastic sheeting. The houses were generally small, dark and over-crowded. Over half (57%) of families in the study lived in just one room, which served as kitchen, bedroom and general living area for the entire family.

4.1.3 Sanitation

The majority of field sites were located on disused government land by the banks of rivers. Most families (82%) did not have access to a private toilet, but instead shared sanitary facilities with several families or used public toilets. In most cases waste from the latrines flowed directly into the nearby river. During the dry season, when the rivers were low, this caused problems with bad smells and swarms of flies and mosquitoes. During the monsoon, houses closest to the river banks were at risk of flooding In addition, faecal matter was often seen on the ground in the settlements – either from animals (dogs, pigs, ducks, chickens) or from children who were too young to use the latrines.

4.1.4 Water supply

As previously described in Chapter 2, water for washing or hygiene purposes was generally always available in all field sites, even during the height of the dry season. Drinking water was much more of a problem for these families; government water taps only operated for a few hours each day and where there was no government supply women would walk some distance (up to 30 minutes) to a water source (tube well or stone tap) that they considered 'safe' to collect drinking water. The quality of drinking water in Kathmandu is known to be very poor with high bacteriological contamination even in these supposedly 'safe' water sources (Maharjan and Sharma 2000; Joshi and Maharjan 2003; Warner, Levy et al. 2008). Once collected, drinking water was usually stored in the house in traditional *gagris* or plastic jerry cans.

4.1.5 Cooking fuel

Less than a third of households (30%) could afford to use the easiest-to-use but most expensive form of cooking fuel – bottled gas. Most households had to rely on cheaper forms of fuel such as kerosene or fire wood collected locally. As fieldwork coincided with a prolonged period of significant political disruption in the south of the country (the Terai), many commodities such as gas, kerosene and food in general, were subject to severe shortages and significant price hikes. Many mothers commented during interviews on the difficulties this was causing them and how they had had to adapt to this new situation by using cheaper sources of food and fuel.

4.1.6 Valuable possessions and income

Families owned few valuable possessions; the median number was two items, with almost a quarter (24%) of families owning none at all. The most commonly owned items were televisions and radios, with 64% and 50% of families owning these items, respectively.

It was not possible to do a full income-expenditure analysis for every family. Instead, each family was asked to estimate their average monthly income. For some families, living on irregular remittances sent from abroad, this was difficult to do but these data provide some

indication of the average levels of wealth in these communities. Median monthly income for these families (after adjusting for family size) was just 4500Rs (IQ range: 3000, 6300Rs), equivalent to about £33 per month.

The World Bank currently employs two monetary cut-off points for identifying people living in poverty: people living on less than $2 per day are said to be living in poverty, while those living on less than $1.25 per day are categorised as being *extremely* poor (World Bank, 2008). These two cut-off points equate to approximately 79Rs and 154Rs per day, or 2370Rs and 4620Rs a month, respectively. Using these estimates, over half (52%) of the families were living in poverty, with about 8% being extremely poor.

4.1.7 Employment

Very few of the mothers were currently in employment as they were still caring for their young children. Of those mothers who were working (15%), most were engaged in home-based activities such as sewing or weaving wool or were employed to wash clothes and dishes in other people's homes nearby.

Almost all fathers (97%) were employed, with over half (52%) working in unskilled jobs such as labourers, drivers of microbuses or taxis, house painters or factory workers. Much of this work was on a casual basis so actual monthly income was variable depending on how much work the father had managed to find that month. Just under a fifth (18%) of fathers were employed as skilled or semi-skilled workers (e.g. tailors, carpenters, welders, chefs) and a further 16% were involved in some kind of professional work – soldiers, policemen or business owners.

4.1.8 Literacy and education

Over half (53%) of the mothers enrolled in the study were illiterate[16] having received no formal education at all. As expected, paternal literacy rates were, in comparison, much higher:

[16] Literacy was defined as self-reported ability to read and write.

almost three-quarters (73%) of the fathers were literate. These literacy rates are high in comparison to the national average of 42% and 69% for women and men, respectively (UNESCO Institute for Statistics 2006) and probably reflect the greater educational opportunities available in urban centres.

Of those mothers who had received some formal education (47%), 18% had received only primary level education, while 28% had attended at least some secondary level schooling. For fathers, over half (52%) had received some secondary level education, 21% had received primary level schooling and just over a quarter (27%) had received no formal education at all.

4.1.9 Socio-demographic differences between groups

Three significant differences between the intervention and control groups were noted at baseline. Firstly, the two groups differed slightly in family composition: there were more extended families in control areas, resulting in a significant difference between the two groups in terms of the number of adults in the household (Mann-Whitney U, P=.01). Secondly, over-crowding (indicated by number of rooms used by the family) was greater in intervention than control areas: 69% of intervention families lived in just one room, compared to 44% of control families (χ^2, P=.017). Thirdly, families living in intervention areas were more likely to use the cheapest source of cooking fuel – firewood – compared to control families (47% vs. 23% for intervention and control, respectively. χ^2, P=.02).

These final two differences suggest that families from the intervention areas were somewhat poorer than those living in control areas. However, aggregate socio-economic status score, summing single SES indicators, showed no significant difference between the two groups (P=.08), indicating that the two groups were broadly comparable in terms of socio-economic status. In particular, the two groups were similar in terms of parental education levels, access to sanitation and monthly income which are arguably the most important variables that may influence child health status.

Index Child		**All (n=88)**		**Control (n=43)**		**Intervention (n=45)**		**Test**	***P***
Age (months)	mean (SD)	7.6	(2.38)	7.5	(2.45)	7.7	(2.34)	t=-0.364	0.72
Sex %	male	48.0		46.5		48.9		χ^2=0.05	0.50
	female	52.0		53.5		51.1			
Birth order	median (IQ range)	2.0	(1-3)	2.0	(1-2)	1.0	(1-2)	U=874	0.41
Mother									
Age (years)	mean (SD)	24.4	(4.60)	23.7	(4.09)	25.1	(5.01)	t=-1.462	0.15
Age (years) at marriage	mean (SD)	18.7	(3.48)	18.1	(2.49)	19.3	(4.13)	t=-1.710	0.10
Years residency	median (IQ range)	4.0	(1.63-7)	3.0	(2-7)	4.0	(1-6.5)	U=966	0.99
Place of birth %	inside KTM	7.0		7.0		6.7		χ^2=0.003	0.64
	outside KTM	93.0		93.0		93.3			
Literacy %		46.6		48.8		44.4		χ^2=0.171	0.42
Education %	none	53.4		51.1		55.6			
	primary	18.2		18.6		17.8		χ^2=0.186	0.91
	secondary+	28.4		30.2		26.6			
Employment %		15.0		16.3		17.8		χ^2=0.035	0.54

Father						
Age (years)	mean (SD)	28.1 (5.54)	27.1 (4.15)	29.2 (6.51)	t=-1.857	0.07
Age (years) at marriage	mean (SD)	22.7 (4.37)	21.8 (3.27)	23.6 (5.11)	t=-1.977	0.05
Years residency	median (IQ range)	6.0 (3-15)	8.5 (2.75-20)	5.0 (3-12)	U=803	0.23
Place of birth %	inside KTM	13.6	16.3	11.1	χ^2=0.499	0.37
	outside KTM	86.4	83.7	88.9		
Literacy %		72.7	74.4	71.1	χ^2=0.121	0.46
Education %	none	27.3	25.6	28.9		
	primary	20.5	11.6	28.9	χ^2=5.071	0.08
	secondary+	52.2	62.8	42.2		
Employment %	Employed	96.0	97.7	93.3	χ^2=0.955	0.33
	Unskilled	52.3	48.8	55.6		
	(Semi) Skilled	18.2	18.6	17.8	χ^2=1.779	0.62
	Professional	15.9	20.9	11.1		
	Other	13.6	11.6	15.5		

Household		**All (n=88)**	**Control (n=43)**	**Intervention (n=45)**	**Test**	***P***
Household size	median (IQ range)	4.0 (4-5)	5.0 (4-6)	4.0 (4-5)	U=846.5	0.30
Adults in house	median (IQ range)	2.0 (2-3)	2.0 (2-5)	2.0 (2-2)	U=690	0.01
Children 5-15yrs	median (IQ range)	1.0 (0-1.75)	1.0 (1-1)	1.0 (0-2)	U=961	0.95
Children <5 yrs	median (IQ range)	1.0 (1-2)	1.0 (1-2)	1.0 (1-2)	U=772.5	0.06
Ethnicity %	Dalit	12.5	11.6	13.3		
	Baishaya	61.4	67.4	55.6	χ^2=1.429	0.49
	Bahun-Chhetri	26.1	20.9	31.1		
Religion %	Hindu	76.1	74.4	77.8		
	Buddhist	18.2	20.9	15.6	χ^2=0.539	0.76
	Other	5.7	4.7	6.7		
Own house %		54.5	53.5	55.6	χ^2=0.038	0.51
Land outside KTM %		64.8	62.8	66.7	χ^2=0.145	0.44
Rooms in house %	1 room	56.8	44.2	68.9		
	2+ rooms	43.2	55.8	31.3	χ^2=5.469	0.02
Toilet %	Own	18.2	16.3	20.0		
	Shared/Public	81.8	83.7	80.0	χ^2=0.205	0.43

Household		**All (n=88)**		**Control (n=43)**		**Intervention (n=45)**		**Test**	***P***
Fuel type %	Firewood	35.6		23.3		67.7			
	Kerosene	34.5		34.9		34.1		χ^2=7.740	0.02
	Gas	29.9		41.9		18.2			
Income per month (Rs)	median (IQ range)	4500	(3000-6300)	4500.0	(3000-7200)	4000.0	(3000-5300)	U=912.5	0.65
Possessions %	median (IQ range)	2.0	(1-3)	2.0	(1-3)	1.0	(1-3)	U=795	0.14
	0	23.9		20.9		26.7			
	1-2	42		37.2		46.7		χ^2=2.26	0.32
	3+	34.1		41.9		26.7			
SES Score	median (IQ range)	5	(3-9)	6.00	(4-10)	5	(3-7,5)	U=761	0.083

Table 4.1 Socio-demographic characteristics of sample. Differences between intervention and control groups analysed by χ^2, t-test or Mann-Whitney U tests, where appropriate.

4.2 Other data relevant to maternal-child health

4.2.1 Pregnancy and ante-natal care

For the majority of mothers, this was their first or second child. On average, women in this sample got married at 19 (3.48 SD) years of age and had their first child a year later (Table 4.2). Living in the capital city, antenatal care for these women was better than for women living in rural areas. Most women (68%) took iron tablets during their pregnancy: this is higher than the national average of 59%, but lower than the average for urban centres of 75% (MOHP, Ministry of Health and Population (MOHP) [Nepal] et al. 2007). The WHO recommends that women should be seen by a health-care professional for antenatal checks at least four times during their pregnancy (WHO, cited by MOHP, Ministry of Health and Population (MOHP) [Nepal] et al. 2007): 64% of mothers in this sample achieved this – a figure considerably higher than both the national average (29%) and the average for urban areas (52%) (MOHP, Ministry of Health and Population (MOHP) [Nepal] et al. 2007). This impressive figure is probably due to the fact that several of the field sites were located within walking distance of local hospitals (Thapathali Maternity Hospital, Kathmandu Medical Centre or Kanti Children's Hospital). About 11% of mothers smoked and 19% drank some alcohol during their pregnancy. Just under half (47%) of the children in the study were born in a hospital or health-care facility, in-line with the average for urban populations in Nepal (48%) (*ibid*). As with the national data for Nepal, first-time mothers were more likely to deliver in hospital than at home; 74% of primiparous mothers gave birth in hospital, as opposed to 32% of multiparous mothers. (χ^2, P <0.001).

4.2.2 Nutritional status of mothers

Mothers enrolled in the study were, on average, 151cm (5.40 SD) tall and weighed 48.33kg (8.28 SD). Women who are particularly short are known to be at risk of complicated deliveries (due to their small pelvis size) and are more likely to give birth to low birth-weight babies. Cut-off points for the height at which mothers are considered to be at risk varies, but the most recent Demographic & Health Survey of Nepal used a cut-off point of 145cm. In this study, 11% of the mothers fell below this cut-off point, compared to a national figure of 14% for Nepal (MOHP, Ministry of Health and Population (MOHP) [Nepal] et al. 2007).

Mean Body Mass Index for the mothers in this study was 21.17 kg/m^2, slightly higher than the national average of 20.6 kg/m^2 for Nepal (*ibid*). About 15% of the women were under-weight (BMI<18.5 kg/m^2) and 13% were over-weight (BMI>25 kg/m^2), compared to national figures of 24% and 9% for under- and over-weight, respectively.

4.2.3 Breastfeeding and complementary feeding practices

All children were breastfed from birth. The majority of children (83%) were fed colostrum; mothers who discarded the colostrum stated that they had been told (by their mother-in-law, friends etc.) that this milk was dirty and would harm the baby. The use of pre-lacteals (usually sugar-water) in this sample was unusual – just 14% of mothers said that they had given their child a pre-lacteal feed, compared to a national average of 37% (MOHP, Ministry of Health and Population (MOHP) [Nepal] et al. 2007).

Median age for the introduction of complementary foods was five months (IQ Range: 4, 6); this usually consisted of *lito* (cereals mixed with milk or water) or *jaulo* (soft mix of rice and lentils). Although most mothers had been advised not to start complementary foods until the child was six months old, they chose to introduce food earlier in response to their child crying all the time and appearing hungry. (This has also been documented in another study in Nepal – see Moffat 2001). The child's diet increased in quantity and variety over the following months, until it eventually resembled the basic family diet of rice, lentils and small amounts of vegetables. Very spicy or oily foods were usually withheld from young or sick children. Meat, fish and eggs were not consumed regularly – usually only very small amounts were given to older children, once every one or two weeks. The majority of mothers (86%) stated that they only gave their child treated (boiled or solar-treated) water, although water added to food was rarely boiled beforehand.

4.2.4 Health-seeking behaviour

Almost all children had been vaccinated, primarily due to the introduction of a free-of-charge childhood vaccination programme in Nepal (Sharma 2002). When a child became sick enough to be regarded as in need of treatment mothers employed a variety of treatment options, largely depending of the type and severity of the symptoms. Some ailments were treated by the local *dhami jhankri* (shaman) through herbs, amulets and

mantras. However, mothers would also commonly take advice from local pharmacists or doctors at the local hospital and saw no contradiction in consulting both traditional and biomedical practitioners for the same sickness. Most mothers had heard of Oral Rehydration Salts (known locally by their brand name of *jeevanjal*) and had given their child these during episodes of diarrhoea. However, during interviews it became clear that many mothers did not understand the purpose of ORS (to replace lost fluids) and became frustrated when this 'medicine' did not appear to stop the diarrhoea at all. Therefore, almost all mothers also reported giving their children anti-diarrhoeal syrups to stop the symptoms. Antibiotic syrups were also purchased, either on prescription from a doctor or, more commonly, on the advice of a pharmacist. When asked about these antibiotics, mothers displayed a lack of knowledge about what they were, how often to give them to the child and the importance of finishing the antibiotic course. The misuse of antibiotics in Nepal is widespread and has been documented by Watcher et al. (1999).

4.2.5 Differences between groups

Mothers from the control areas were significantly more likely to have seen a health-care professional at least four times during their pregnancy, compared to women in intervention areas (*P*=.03). This difference is probably due to the fact that two of the control sites were located close to Thapathali Maternity Hospital or Kanti Children's hospital, therefore making access to clinics much easier for these women. Intervention mothers were significantly more likely to give their child increased amount of food during episodes of sickness, than control mothers (*P*=.01). There were no other differences between the two groups.

As described in Chapter 2, data collected on pregnancy, ante-natal care, health seeking behaviour and feeding practices were used to construct an index of risk for each child: the higher the score, the greater the number of risk factors for that child. The results from this index are displayed at the bottom of Table 4.2. Overall, children in this sample scored four out of a possible 15 for the risk score, with no significant difference between the two groups detected (*P*=.22).

	All (n=88)		Control (n=43)		Intervention (n=45)			
RISK SCORE INDEX	**%**		**%**		**%**		**Test**	***P***
Maternal age < 18.5 yrs	14.8		9.3		20		χ^2=0.167	0.52
> 35 yrs	12.5		14		11.1			
During pregnancy:								
Smoked	11.4		4.7		17.8		χ^2=3.762	0.52
Drank alcohol	19.3		20.9		17.8		χ^2=0.140	0.46
Saw a HCP at least 4 times	63.6		74.4		53.5		χ^2=4.225	0.03
Took iron tablets	68.2		76.7		60		χ^2=2.842	0.07
Premature birth	3.4		2.3		4.4		χ^2=0.30	0.52
Gave birth in a health facility	46.6		55.8		37.8		χ^2=2.875	0.07
First breastfeed >60mins after birth	61.4		62.8		60		χ^2=0.072	0.48
Child fed prelacteal	15.9		11.6		20		χ^2=1.152	0.22
Child fed colostrum	83		86		80		χ^2=0.569	0.32
Correct weaning age	23.9		32.6		15.6		χ^2=3.499	0.05
Treat drinking water for child	86.4		83.7		88.9		χ^2=0.499	0.35
Vaccinated child	98.9		0		2.2		χ^2=0.967	0.51
Correct knowledge about liquids	87.5		88.4		86.7		χ^2=0.058	0.53
Correct knowledge about food	55.7		41.9		68.9		χ^2=6.509	0.01
	median	**I-Q range**	**median**	**I-Q range**	**median**	**I-Q range**	**Test**	***P***
Risk Score	4	(3-5)	3	(3-5)	4	(3-6)	U=822.5	0.22

	All (n=88)		Control (n=43)		Intervention (n-45)			
	mean	**(SD)**	**mean**	**(SD)**	**mean**	**(SD)**	**Test**	***P***
Age at birth of 1st child (years)	20.3	3.38	19.78	2.69	20.8	3.91	t=-1.364	0.18
Age at birth of index child (years)	24.4	4.6	23.7	4.09	25.1	5.01	t=-1.462	0.15
Maternal weight (kg)	48.33	8.28	49.29	8.28	47.4	8.02	t=1.065	0.29
Maternal height (cm)	151	54	150	61	151	47	t=-0.949	0.35
Maternal BMI (kg/m^2)	21.2	3.19	21.7	2.91	20.7	3.38	t=0.425	0.13
	median	**I-Q Range**	**median**	**I-Q Range**	**median**	**I-Q Range**	**Test**	***P***
Age complementary foods introduced (months)	5	(4-6)	5	(4-6)	5	(3-7)	U=865.5	0.88

Table 4.2 Pregnancy, feeding and child care-giving data for the sample, including risk score. Differences between intervention and control groups analysed by χ^2, t-test or Mann-Whitney U tests, where appropriate.

4.3 Hand-washing practices

Hand-washing practices were assessed through structured observations, self-reports of behaviour and weekly measures of soap usage.

4.3.1 Observations of hand-washing practice

The structured observations focused on hand-washing (or lack thereof) at key junctures where faecal contamination could occur: after cleaning the baby's bottom, after defecation, and before cooking food or feeding the baby. Because the observed number of junctures where hand-washing might have occurred was generally very low, statistical comparisons between the intervention and control groups were not possible. The data here are therefore presented as a whole, to provide a descriptive picture of baseline hand-washing practices within the sample. At each juncture, observers noted if hand-washing took place or not, and if it did, whether this was with soap or just water alone (Table 4.3 and Figure 4.1). In total, observations were conducted in 75 households (41 intervention, 34 control).

During these 75 observations, only 29 children were seen to defecate. In the majority of cases (95%), the mother was responsible for cleaning the child's bottom after defecation, though occasionally this was done by the child's grandmother (data not shown). In over two-thirds (69%) of cases, the mother/carer did *not* wash her hands with either water or soap after cleaning the child's bottom. Although the mother/carer often cleaned the child's bottom with a rag, rather than directly with her hand, there is still considerable potential for faecal contamination of her hands at this juncture. Of those mothers/carers who did wash their hands, 17% used water alone and only 14% used soap.

Only 14 mothers were observed to go for defecation[17] during the observation period: 50% of these mothers did not wash their hands at all after defecation; 29% washed their hands with water alone and only a fifth (21%) used soap. Hand-washing behaviour after defecation was also observed for other family members (i.e. fathers, grandmothers, older siblings). In total 24 family members were observed to go for defecation during the

[17] Obviously it was difficult to know for sure if the mother visited the toilet for urination or defecation. Fieldworkers were instructed to make educated guesses about whether the mother had defecated, depending on the length of time spent in the lavatory. Although not ideal, this was felt to be the best solution to the problem; asking the mother directly would have been intrusive and would have drawn attention to the focus of the observation, thereby possibly changing the mother's behaviour.

observation period: of these, 42% washed hands with soap, 21% washed hands with just water and 38% did not wash their hands at all.

Hand-washing rates before contact with food were also very low. Cooking of food occurred in 72% of the observations and in most cases (85%) was done by the mother. As mentioned in the previous chapter, hands were not washed at all before starting cooking in over three-quarters (78%) of the observations. Ten people (19%) washed their hands with water before cooking and only two people (one mother, one grandmother) used soap to clean hands before preparing food.

The child was fed in 35% of the observations and in all but two cases s/he was fed by the mother. Despite the fact that in half of the cases the mother used her fingers to feed the child, none was seen to wash hands with soap before feeding the child: most mothers (65%) did not wash their hands at all, while 35% washed hands with water.

The limitations[18] of structured observations of hygiene behaviour notwithstanding, there seemed to be clear evidence that hand-washing with soap was not routinely practiced at any of the important junctures and therefore children were potentially at high risk of coming into contact with faecal matter.

	N events observed	% Hands NOT Washed	% Hands washed with WATER	% Hands washed with SOAP
After cleaning baby's bottom	29	69	17	14
After maternal defecation	14	50	29	21
After other family member defecation	24	38	21	42
Before feeding baby	26	65	35	0
Before cooking	54	78	19	4

Table 4.3 Structured observations of hand washing behaviour at baseline

18 Discussed in greater detail in Chapter 6.

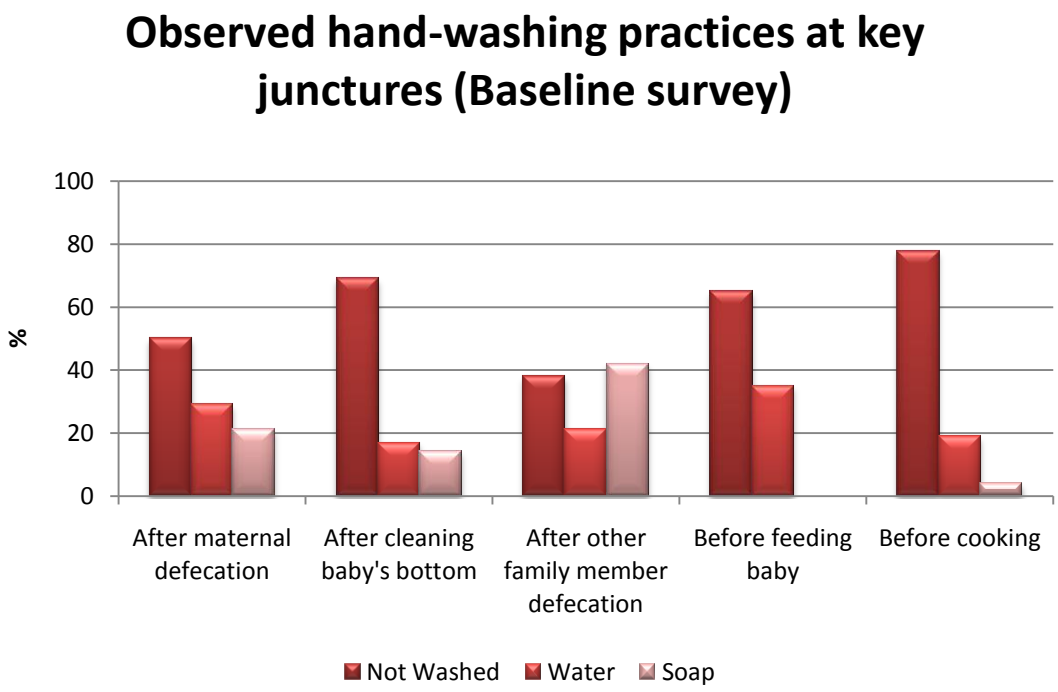


Figure 4.1 Baseline observations of hand-washing behaviour at key junctures.

4.3.2 Self-reports of hand-washing practice

After completion of the observations, all mothers were specifically asked about their own hand-washing behaviour at the five key junctures. As expected, self-reports of hand-washing with soap were much higher than those observed. Almost all mothers (96%) reported hand-washing with soap after defecation and 81% reported using soap after cleaning a child's bottom.

Mothers reported much lower rates of hand-washing before contact with food. Only 12% of mothers reported washing hands with soap before cooking food (compared to 4% seen to do so during observations). The vast majority (81%) of mothers said they merely washed hands with water before cooking, while 7% said they did not wash hands at all at this juncture. About a quarter (26%) of mothers said they washed hands with soap before feeding a child, while two-thirds (66%) said they only used water and 8% did not wash their hands at all at this juncture.

When comparing reported hand-washing rates between the intervention and control groups no differences were found for the first four hand-washing junctures. However, intervention

mothers were significantly more likely to report hand-washing with soap before eating, than their control counterparts (χ^2 = 5.78, *P*=.027, data not shown).

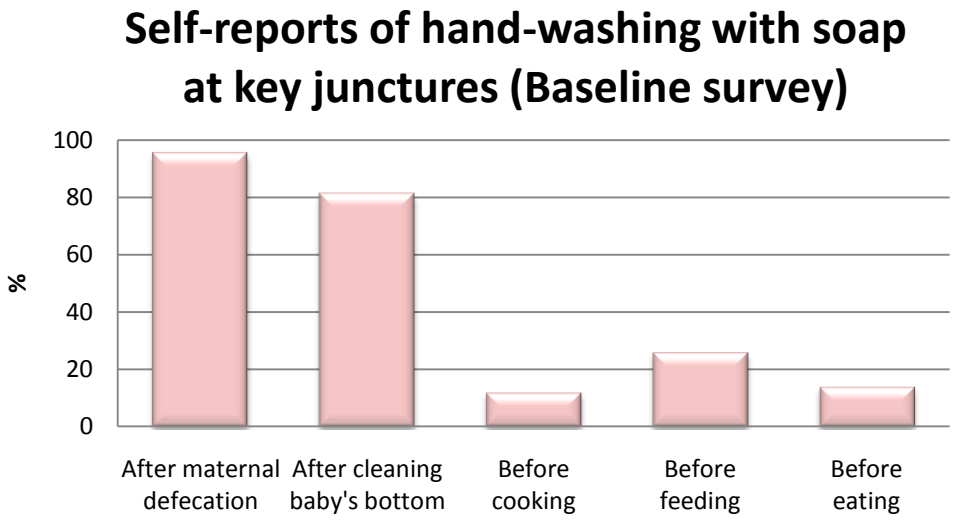


Figure 4.2 Baseline reports of hand-washing behaviour at key junctures.

4.3.3 Monthly soap usage

Spot-check observations confirmed the presence of soap in every household in the study. The median number of bars of soap used per month at baseline (May 2007) was 4.6 bars for both intervention and control groups. The most commonly used soap for hand-washing was laundry soap.

4.4 Health and growth status of children at baseline

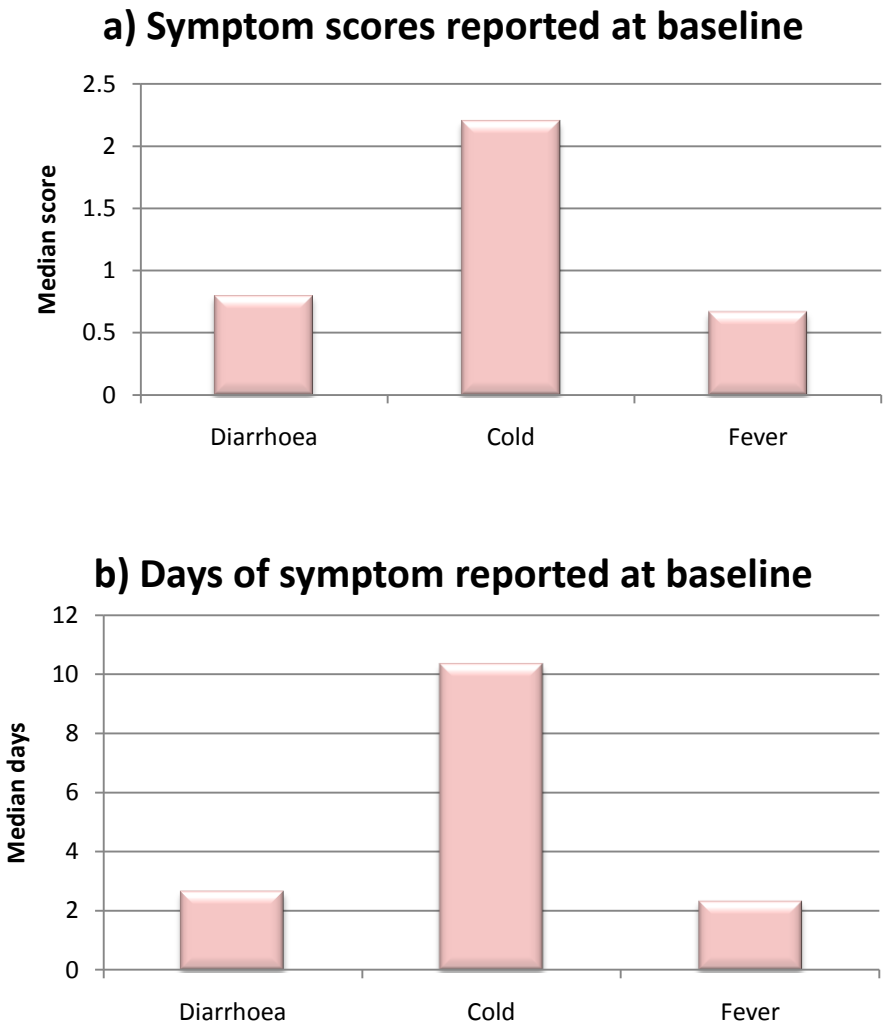
This section presents the morbidity, biochemical and growth status of the children at baseline (May 2007).

4.4.1 Morbidity

Baseline data for child morbidity were collected for four weeks (May 2007) prior to the launch of the hand-washing programme in the intervention sites. The data collected consisted of two measures:

1. Symptom score: referring to the total number of weeks per month (28 days) where the child experienced a particular symptom. (For example, a child who reported diarrhoeal symptoms for two weeks in a month would have symptom score of two).
2. Days of symptom: referring to the total number of days in the month the child experienced that symptom.

During the baseline period, the children experienced a median of 0.8 episodes of diarrhoea, lasting 2.67 days (Figures 4.3 a-b). Colds were much more common with most children reporting colds on two out of four weeks of survey, totalling a median of ten days in the month. Fevers were less common; children experienced 0.67 episodes and two days of fever during this baseline month. There were no significant differences between the two groups for either the number of reports or the number of days of any symptom.



Figures 4.3a-b. Symptom scores and days of sickness at baseline for diarrhoea, colds, and fevers.

The table below shows the percentage of children experiencing diarrhoea or fever over a two week period for this study and for the whole of Nepal (no data were available for the prevalence of colds). Data for Nepal are taken from the Demographic and Health Survey of Nepal for 2006 (MOHP, Ministry of Health and Population (MOHP) [Nepal] et al. 2007) while the data from this study uses only the first two weeks of the baseline period for comparative purposes. It was not possible to disaggregate the DHS morbidity data across rural and urban areas, so the two samples are not directly comparable. However, this limitation notwithstanding, the data suggest that children from the slum areas had considerably higher rates of diarrhoea and fevers than the national average.

Children experiencing symptoms in previous two weeks	% Nepal (MOPH, 2007) n=978	% This study n=88
Diarrhoea	16.6	39.5
Fever	20.3	35.8

Table 4.4 Comparison of percentage of children (<12 months) experiencing diarrhoea or fever in the previous two weeks for Nepal (MOHP, Ministry of Health and Population (MOHP) [Nepal] et al. 2007) and this study.

4.4.2 Growth status

At baseline (May 2007) the children in this study were, on average, mildly stunted (HAZ= -1.27) and underweight (WAZ= -1.07), but not wasted (WHZ= 0.13). Just under a fifth of children were moderate-to-severely stunted and underweight (18% and 17%, respectively); only three children were moderate-to-severely wasted at baseline. There were no significant differences in growth status between the intervention and control groups at baseline

The growth status of children in this study was compared to national data taken from the Demographic and Health Survey (MOHP, Ministry of Health and Population (MOHP) [Nepal] et al. 2007) as shown in Figure 4.4a-c. Growth status for children in this study declined progressively by age group for all three growth measures (HAZ, WAZ and WHZ), following the national trend. For all three age groups, children in this study were considerably *more* stunted but *less* wasted than children in Nepal as a whole. For under-weight, children in this study fared better than the national average below six months of age, but worse at 10-12 months of age.

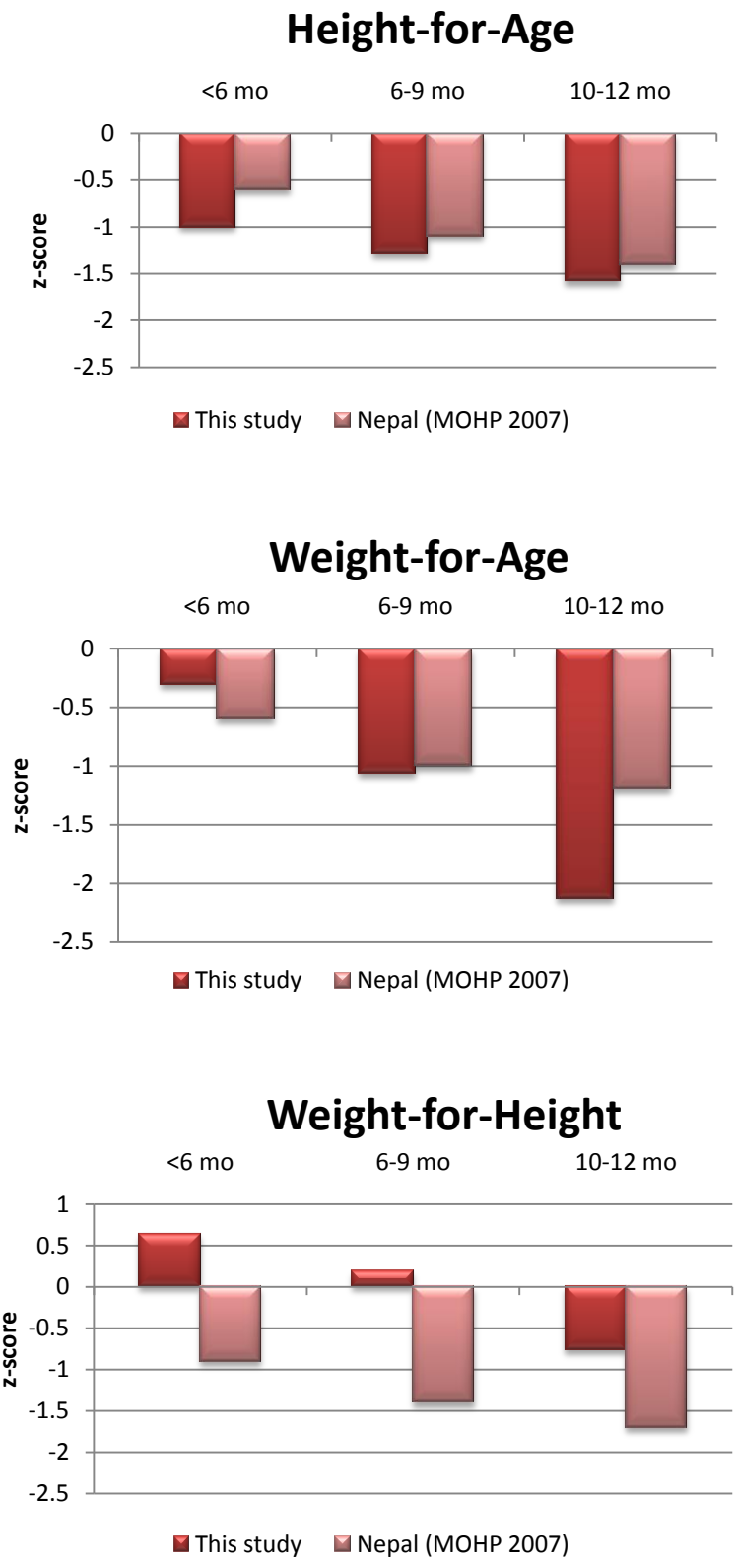


Figure 4.4a-c. Chart comparing growth status (HAZ, WAZ, WHZ) of children in this study with data for Nepal. (Data for Nepal taken from Nepal Demographic & Health Survey, MOHP et al. 2007).

4.4.3 Biochemical status

Gut damage was assessed through the lactose:creatinine (L:C) urine test (normalised by log transformation) with a threshold value of .32 set as an indicator of significant levels of gut damage[19]. At baseline, mean L:C was .33 (.02 SD) with 69% children experiencing gut damage. There was no difference between the two groups in mean L:C values, nor the percentage of children with gut damage at baseline.

According to the WHO (2008) definition of anaemia (< 110g/L), the children in this study were mildly anaemic at baseline, with a mean haemoglobin level of 104g/L (8.8 SD). There were no differences in Hb levels between the two groups.

Three markers of immune function were assessed: IgG, AGP and albumin. For all three indicators, children in this study showed elevated immune stimulation relative to 'normal' ranges seen in healthy children from the developed world.

Meites (1989) gives 'normal' IgG values for infants of different ages with means ranging from 3.3g/L for three month-olds up to 5.9g/L for children aged 10-12 months. Mean IgG for children in this study was considerably higher at 6.71g/L (2.3 SD).

Mean AGP was .85g/L (.33SD) at baseline. There are currently no data available for 'normal' AGP levels in children, but the mean value for these children falls into the upper range for normal adult values of .5-1.0g/L (Calvin, Neale et al. 1988).

Albumin is an indicator of both immune stimulation and poor nutritional status, lowering in circulating plasma levels in response to both. Mean albumin for these children was 33.12g/L (7.19 SD). Hicks and Boeckx (1984) give 95% reference limits for children aged 4-12 months as 49-51g/L. Thus, the fact that albumin levels in these children were outside the 95% reference limits probably reflects both their higher exposure to pathogens and their poor growth status.

It is clear that the children in this study displayed elevated levels of immune stimulation compared to healthy children from the developed world, reflecting their highly contaminated environment and high morbidity load. However, baseline levels of immune

---

[19] Panter-Brick et al. (2009) set the threshold L:C ratio for gut damage at 0.1. As explained in Chapter 2, this valued was normalised by log transformation, after adding '2' to each value to prevent negative values. Thus, after normalisation, the new threshold value becomes 0.32.

stimulation were *not* equal in both groups: for all three variables, significant differences between intervention and control groups were observed. Levels of IgG and AGP were significantly higher (indicative of greater immune stimulation) in intervention children (mean IgG: 6.09 and 7.31g/L for control and intervention groups, respectively, P=.01; mean AGP: 0 .76 and 0.93 for control and intervention groups, respectively, P=.02). Levels of albumin were also higher in intervention children (31.39 and 34.75g/L for control and intervention groups, respectively, P=.03), which (in this negative acute phase protein) would suggests *less* immune stimulation, thus contradicting the results for IgG and AGP. However, this discrepancy may be accounted for by slight differences in nutritional status at baseline: children from the control areas had slightly lower weight-for-height z-scores, which may account for their lower levels of albumin (WHZ: .15 and .10 for intervention and control groups, respectively).

These differences between the two groups at baseline were unexpected since other variables (such as socio-economic status, growth and morbidity) were comparable. Ideally baseline biological samples would have been analysed immediately to check for such differences, but it was not possible to ship the samples back to the UK for analysis during the fieldwork period. However, the multi-level statistical modelling used to analyse these data takes into account individual baseline differences for each child, and thus these group differences do not present a problem for analysis.

4.5 Impact of socio-economic and demographic variables on child health and growth status

The impact of socio-economic and demographic variables on child health and growth over the intervention period was analysed using cross-sectional time-series analysis, as described in Chapter 2. Demographic variables thought to potentially have an impact on child health were entered independently as predictor variables into time series regression models. These included: maternal age, maternal BMI, birth order, caste, household size and number of adults in the family. The composite SES and risk scores were similarly entered into regression models to determine their impact on health and growth status. The results of these regression models are presented in Table 4.5 (only models with significant results are presented).

As expected, socio-economic status had a significant impact on child growth status: children from families with higher SES scores had better height-for-age and weight-for-age z-scores (*P*=.026 and .019, for HAZ and WAZ, respectively; Table 4.5). These children also had significantly higher Hb levels than children from poorer families (*P*=.011). Socio-economic status had no effect, however, on WHZ, L:C, IgG, AGP and albumin. Children with higher scores for the composite risk variable had significantly higher levels of AGP (*P*=.035). They also had higher levels of gut damage, but this just failed to reach significance (*P* = .05).

Maternal BMI was positively associated with child growth: mothers with higher BMIs had children with significantly better HAZ, WAZ and WHZ scores (*P*=.021, <.001 and .002, respectively). The children of these women also had significantly lower levels of gut damage over the intervention period (*P*=.003), although along with age, maternal BMI only accounted for about a fifth of the variation observed in L:C values.

Maternal age, the child's birth order and caste grouping had no impact on any biochemical or growth variable. Household size and number of adults in the family, however, had a positive impact on growth. In larger households children had significantly better weight-for-age z-scores (*P*=.036). Similarly, in families where there were more adults, children had significantly better HAZ and WAZ scores (*P*=.013 and .011), with the association being stronger for HAZ (coef. = .146 and .116 for HAZ and WAZ, respectively).

However, larger families consisting of more adults potentially mean a greater number of wage-earners, in which case these results may simply reflect the better socio-economic status of larger families. These analyses were therefore re-run controlling for SES. After controlling for SES, the relationship between the number of adults and child growth (HAZ and WAZ) disappeared, suggesting that the positive benefit to child growth in having more adults around is explained by their contribution to household SES. However, household size *per se* seemed to have a beneficial impact on child growth even after controlling for SES, since the association with WAZ remained in this new analysis. A possible explanation for this may that in larger households there are potentially a greater number of older children who can assist with child-care activities, even though they do not directly contribute to the household's socio-economic status.

	Predictor	Coef.	Std. Err.	z	P	95% CI		Rho
	age	-0.098	0.005	-19.430	0.000	-0.107	-0.088	
	SES	0.064	0.029	2.220	0.026	0.008	0.120	0.942
	constant	-0.856	0.209	-4.090	0.000	-1.266	-0.447	
	age	-0.098	0.005	-19.440	0.000	-0.107	-0.088	
	Mat. BMI	0.071	0.031	2.300	0.021	0.011	0.131	0.942
	constant	-1.963	0.661	-2.970	0.003	-3.259	-0.667	
HAZ	age	-0.097	0.005	-19.410	0.000	-0.107	-0.088	
	HH size	0.083	0.061	1.350	0.177	-0.037	0.203	0.941
	SES	0.061	0.029	2.140	0.032	0.005	0.117	
	constant	-1.234	0.348	-3.540	0.000	-1.917	-0.552	
	age	-0.097	0.005	-19.420	0.000	-0.107	-0.088	
	adults	0.113	0.062	1.800	0.071	-0.010	0.235	0.940
	SES	0.044	0.030	1.440	0.150	-0.016	0.103	
	constant	-1.050	0.232	-4.530	0.000	-1.504	-0.595	
	age	-0.153	0.006	-25.240	0.000	-0.165	-0.141	
	SES	0.075	0.032	2.350	0.019	0.012	0.137	0.932
	constant	-0.395	0.233	-1.700	0.090	-0.852	0.062	
	age	-0.153	0.006	-25.260	0.000	-0.165	-0.141	
	Mat. BMI	0.116	0.033	3.520	0.000	0.051	0.180	0.927
	constant	-2.390	0.709	-3.370	0.001	-3.778	-1.001	
WAZ	age	-0.153	0.006	-25.230	0.000	-0.164	-0.141	
	HH size	0.134	0.067	1.990	0.046	0.002	0.265	0.929
	SES	0.070	0.031	2.250	0.024	0.009	0.132	
	constant	-1.005	0.382	-2.630	0.009	-1.754	-0.256	
	age	-0.153	0.006	-25.250	0.000	-0.165	-0.141	
	adults	0.126	0.069	1.830	0.067	-0.009	0.262	0.929
	SES	0.052	0.034	1.550	0.121	-0.014	0.118	
	constant	-0.611	0.258	-2.370	0.018	-1.117	-0.106	
	age	-0.075	0.008	-9.500	0.000	-0.091	-0.060	
WHZ	Mat. BMI	0.082	0.027	3.030	0.002	0.029	0.134	0.827
	constant	-1.083	0.586	-1.850	0.065	-2.232	0.065	

	Predictor	Coef.	Std. Err.	z	P	95% CI		Rho
L:C	age	-0.001	0.000	-2.300	0.021	-0.002	0.000	
	Mat. BMI	-0.001	0.000	-2.950	0.003	-0.002	0.000	0.214
	constant	0.370	0.011	33.000	0.000	0.348	0.392	
AGP	age	0.006	0.005	1.150	0.250	-0.004	0.015	
	RISK	0.017	0.008	2.110	0.035	0.001	0.033	0.075
	constant	0.679	0.063	10.710	0.000	0.555	0.804	
Hb	age	0.772	0.145	5.330	0.000	0.488	1.056	
	SES	0.635	0.251	2.530	0.011	0.143	1.127	0.533
	constant	95.101	2.398	39.670	0.000	90.402	99.800	

Table 4.5 Impact of demographic and socio-economic status on growth and biochemical variables using time series analysis.

Summary

This chapter provided a socio-demographic description of the study sample at baseline (May 2007). The families enrolled in this project were generally poor, with over half living on less than $2 per day. Houses were often poorly constructed and over-crowded and access to basic services such as drinking water and sanitation were limited. The children were mildly stunted and underweight, but reported high levels of morbidity and had correspondingly high levels of gut damage and immune stimulation. Socio-economic status, maternal BMI and household size were important predictors of certain growth and biochemical variables over the period of study. The following chapter will discuss the impact of the hand-washing intervention on the outcome variables: hand-washing behaviour, morbidity, gut damage, immune stimulation and growth.

CHAPTER 5
Impact of the Intervention

Introduction

This chapter analyses the impact of the intervention at a number of different levels. It starts by examining its impact on hand-washing practices by comparing reported hand-washing rates between mothers from intervention and control areas. It then goes on to consider the effect of the intervention on reported morbidity amongst the children. The relationship between biomarkers and growth status is described and the underlying hypotheses on which the intervention was based are tested. Finally, the impact of the intervention on levels of gut damage, immune stimulation and growth faltering over the period of study is assessed.

5.1 Intervention hypotheses and levels of evaluation

Based upon a theoretical model linking pathogen exposure, morbidity, sub-clinical infection and growth, predictions were made as to the impact of the intervention on four different levels. The specific hypotheses were set out in Chapter 1, but briefly they suggested that:

Level I: Mothers from the intervention areas would increase their hand-washing behaviour.

Level II: Children from the intervention areas would have lower levels of morbidity.

Level III: Children from the intervention areas would have lower levels of gut damage and immune stimulation.

Level IV: Children from the intervention areas would have lower levels of growth faltering.

The following sections of this chapter will examine each of these hypotheses in turn and evaluate the impact of the intervention at the four different levels.

5.2 Level I: Changes in hand-washing behaviour

Changes in hand-washing behaviour were measured by reported hand-washing behaviour and measures of monthly soap usage per household[20].

5.2.1 Reported hand-washing practices

As reported in Chapter 4, at baseline (May 2007) reported hand-washing with soap was high after contact with faecal material, but relatively low before contact with food (Figure 5.1a). Only one difference was detected between the two groups at baseline, with mothers in the intervention area being more likely to report hand-washing with soap before eating food, than their control counterparts (χ^2=5.78, *P*=.027).

Six months later (November 2007), hand-washing rates had improved dramatically in the intervention areas, but dropped slightly amongst mothers from the control groups (Figure 5.1b). *All* mothers living in the intervention sites now reported washing their hands with soap after defecation or cleaning the baby's bottom, compared to 91% and 84% of mothers from control areas for these two junctures, respectively. The difference between the two groups just failed to reach significance for hand-washing after defecation (χ^2=4.39, *P*=.053), but was highly significant for hand-washing after cleaning the baby's bottom (χ^2=7.96, *P*=.005). Hand-washing with soap before cooking, eating or feeding the baby, was also much higher in the intervention areas, with differences between the two groups being highly significant in all cases (*P*<.001 for all three junctures).

[20] Structured observations of behaviour could not be used to assess changes in hand-washing practices, for reason discussed in Chapter 6.

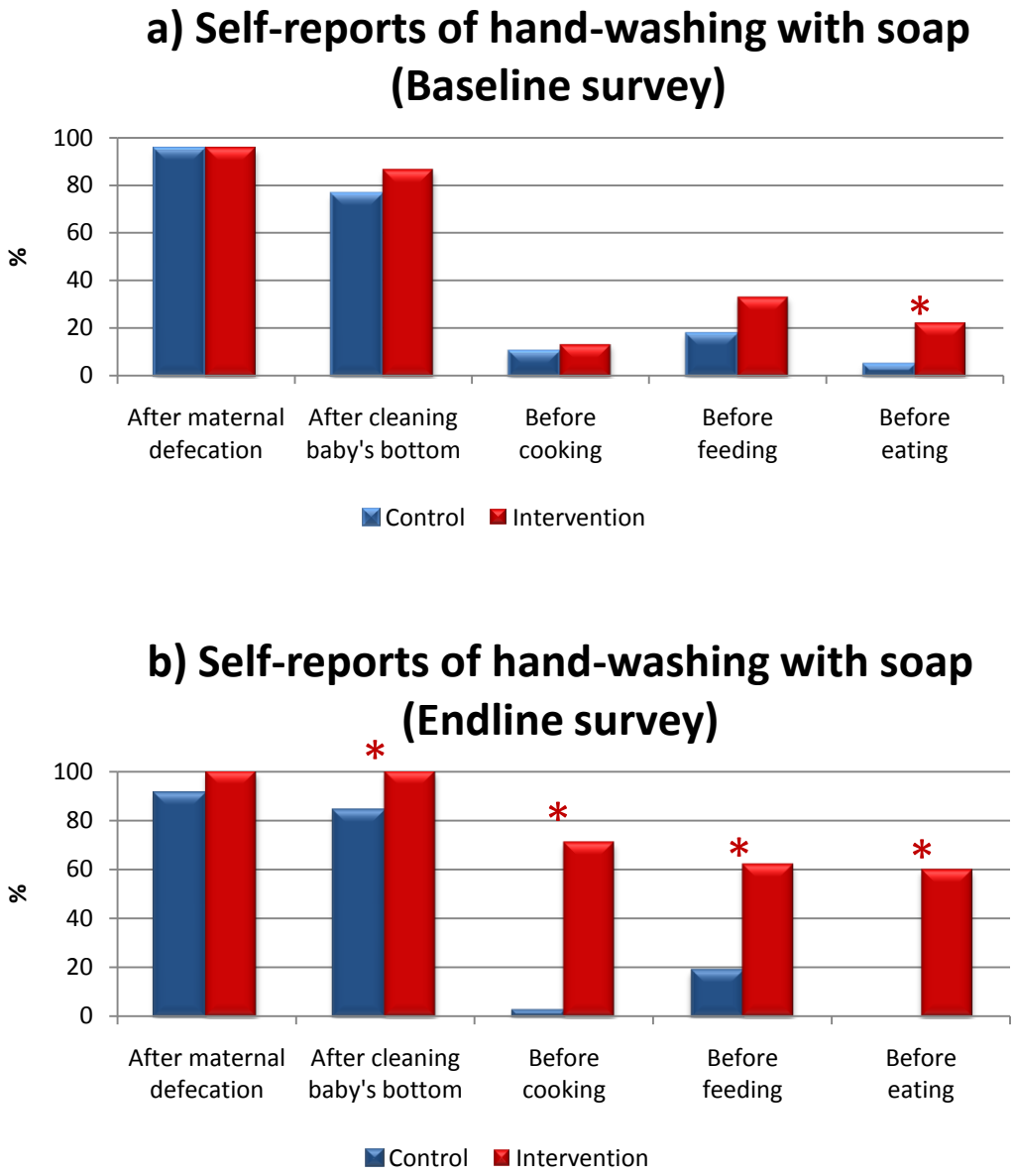


Figure 5.1 a-b. Self-reported hand-washing with soap at five key junctures at baseline (May 07) and endline (Nov 07) for intervention and control groups. * indicates significant difference between groups (*P*<.05), by χ^2 test.

No changes in reported behaviour from baseline to endline were detected for any hand-washing juncture for mothers in the control areas; as predicted, their hand-washing practice did not change over the period of study (Table 5.1). For intervention mothers, however, significant changes (increases) in reported hand-washing practice from baseline to endline were detected for four junctures (after cleaning baby's bottom, before cooking, eating or feeding baby). Although reported hand-washing after defecation also increased in

intervention mothers over the study period, this difference was not significant because baseline levels of reported hand-washing were already so high.

McNemar's test of change in hand-washing behaviour		
	Control	Intervention
Hand-washing Juncture	*P*	*P*
After toilet	0.625	0.500
After cleaning baby	0.549	0.031
Before cooking	0.125	<0.001
Before feeding	0.500	0.004
Before eating	0.100	0.003

Table 5.1 McNemar's test to examine changes in reported hand-washing with soap practice from baseline to endline.

5.2.2 Reported soap usage

It was hypothesised that mothers living in intervention areas would increase hand-washing with soap practice, and therefore their monthly usage of soap, compared to those mothers living in control areas. Median monthly soap usage[21] for intervention and control groups is displayed in Figure 5.2 below. At baseline (May), families from both intervention and control areas reported using 4.6 bars of soap (IQ range: 3.4, 6.33) per month. After baseline, families in intervention areas consistently reported higher soap usage every month; however, none of these differences reached significance (although it approaches significance in August, *P*=.052). The total amount of soap used across the whole intervention period was also calculated: families in intervention areas used a greater amount of soap across the whole study period, but again this difference failed to reach significance (*P*=.239).

[21] As larger families inevitably use greater amounts of soap, monthly soap usage was divided by the total number of family members and multiplied by the median family size (four people) to give a standardised value.

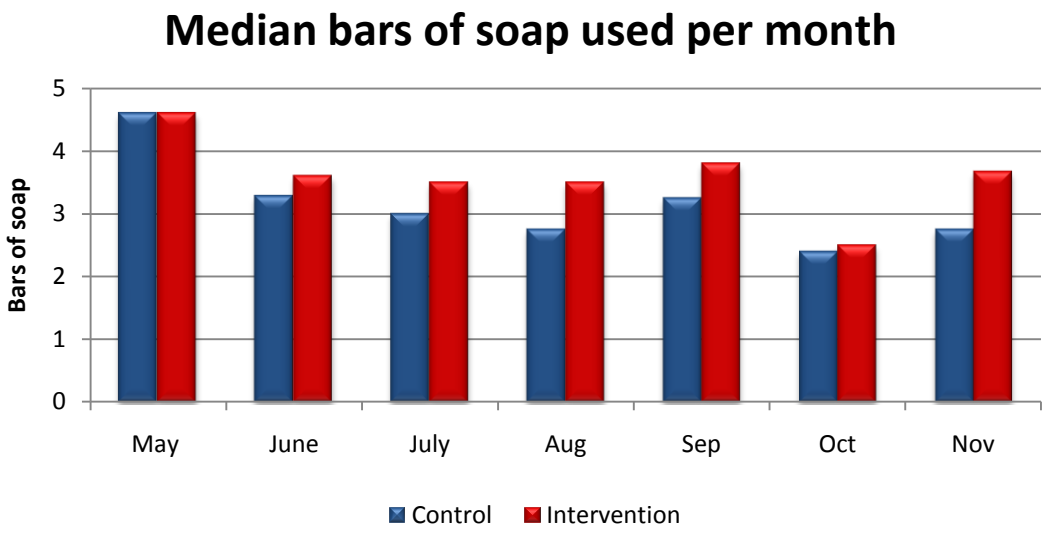


Figure 5.2 Median number of bars of soap used per month by intervention and control groups, after adjusting for family size.

It is interesting to note that reported soap usage actually decreased in both groups over the study period. This unexpected finding might have arisen from the mothers becoming better at remembering how many bars of soap they started each week; thus during the first few weeks, when they were unused to the question being asked, they probably over-estimated how much soap they actually used.

5.3 Level II: Changes in reported morbidity

Morbidity data were first analysed cross-sectionally, comparing both reports and days of sickness (for diarrhoea, colds and fevers) between intervention and control groups on a month-by-month basis using Mann-Whitney U tests. Morbidity data were then analysed over the entire study period by summing all the monthly reports of sickness or days of sickness for each symptom.

5.3.1 Morbidity reports

No significant differences in reported sickness between the intervention and control groups were recorded for any of the three symptoms at any month, with the exception of one month: in September, children from control areas reported significantly more fevers than intervention children (U = 755, *P*=.044) (Appendix 8).

Over the whole study period, children from the intervention areas reported fewer episodes of sickness for each type of symptom than children in the control areas (Figure 5.3). However, this difference only reached significance for diarrhoeal sickness (U=732, *P*=.049). On average, children from intervention areas experienced 31% fewer episodes of diarrhoea than control counterparts.

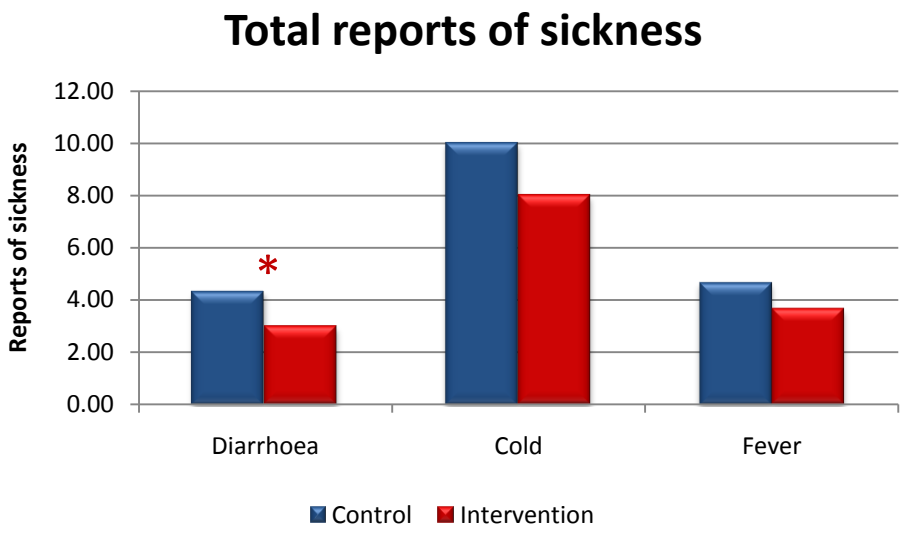


Figure 5.3. Total reports of sickness over whole study period. * indicates significant difference between groups (*P*<.05), by Mann-Whitney U test.

5.3.2 Days of sickness

Month-by-month, there were no differences between children in intervention and control groups in terms of the number of days of diarrhoeal symptoms. However, children from intervention areas reported significantly fewer days of colds in June and October (*P*=.041, for both months) and significantly fewer days of fevers in September (*P*=.049) (Appendix 9).

Figure 5.4 shows the total number of days of sickness for each symptom over the entire study period. Children in the intervention group experienced significantly fewer days of diarrhoea than children living in control areas, (9.67 vs. 16.33 days for intervention and control groups, respectively, *P*=.023) representing a 41% reduction in the number of days with diarrhoeal symptoms for these children. Intervention children also reported fewer days of colds and fevers over the study period but these differences did not reach significance. (The difference between the two groups does, however, approach significance for days of colds, *P*=.062).

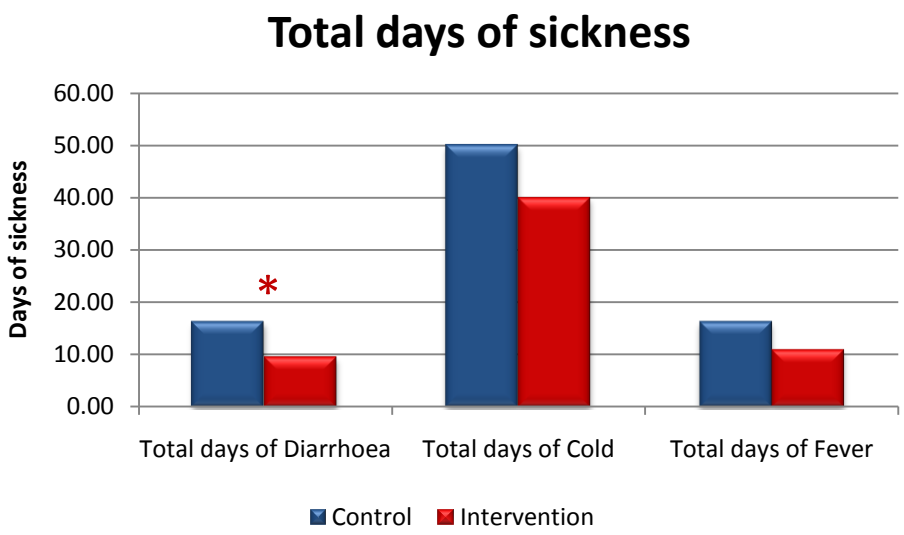


Figure 5.4 Total days of sickness over whole study period. * indicates significant difference between groups (*P*<.05), by Mann-Whitney U test.

5.4 Assessing underlying hypotheses

The previous two sections have indicated that maternal hand-washing practices increased in the intervention areas and, in concordance with results from other hand-washing studies, resulted in a significant reduction in diarrhoeal symptoms in children from these areas. The aim of this study, however, was to move beyond assessing the intervention's impact purely in terms of clinical morbidity, to determine how hand-washing affects levels of gut damage and immune stimulation (Level III) and growth faltering (Level IV) in young children. Underpinning this intervention therefore were the following hypotheses:

- Exposure to pathogens causes damage to the mucosal lining of the small intestine and/or stimulation of the immune system in the child. This may occur at a clinical or sub-clinical level.

- Increases in mucosal damage (L:C) result in increased immune stimulation (IgG, AGP), as the child repairs damaged tissue and fights off infection.

- Higher levels of mucosal damage and immune stimulation result in less energy being available for growth and therefore may result in poorer biochemical nutritional status (albumin, Hb) and growth retardation in the child (HAZ, WAZ, WHZ).

These hypotheses are depicted diagrammatically in Figure 5.5. As discussed in Chapter 1, these hypotheses have been confirmed in previous studies. However, before examining the impact of the intervention at Levels III and IV, it is first necessary to explore the relationships between biochemical and growth variables and determine whether these underlying hypotheses are supported by the data from this study.

The first hypothesis outlined in Figure 5.5 cannot be empirically tested: no reliable data on pathogen exposure could be collected for each child. For the purposes of this study, being in the hand-washing group is taken as a proxy for reduced exposure to pathogens. However, it was possible to test the other hypotheses regarding a) the relationship between biomarkers of gut damage and immune stimulation and b) the relationship between these biomarkers and growth faltering in children.

As described in Chapter 2, I employed a three-step analytical strategy. Firstly, relationships were assessed on a monthly, cross-sectional basis. Secondly, relationships between *mean* biochemical and growth variables over the whole intervention period were analysed. Finally, time-series analysis was used to examine relationships between outcome variables longitudinally. All univariate models are presented in Appendices10-18: here I present only multivariate models.

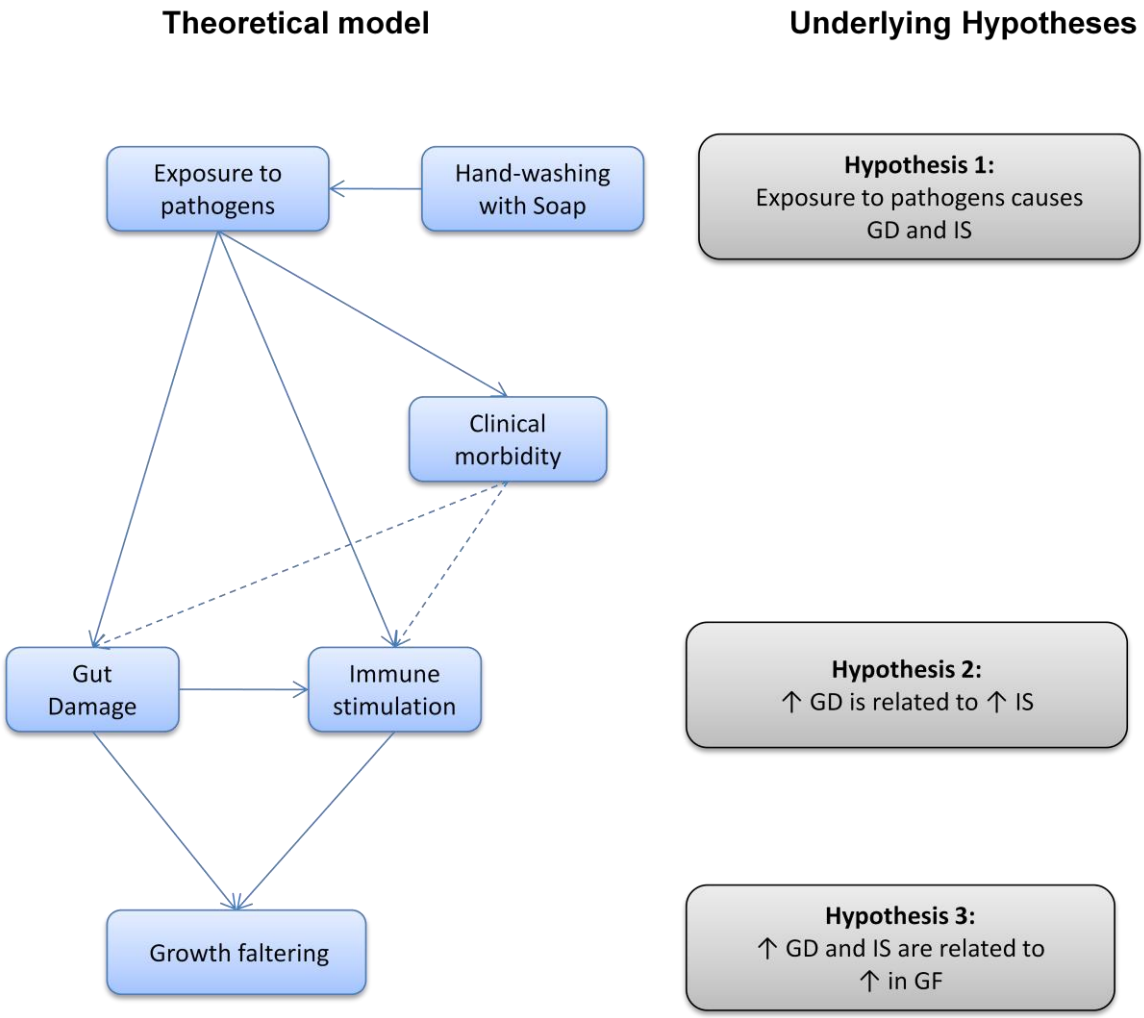


Figure 5.5 Theoretical model of this study and the underlying hypotheses on which it is based.

5.4.1 Relationships between gut damage and immune biomarkers

Step 1: Monthly cross-sectional analyses

Cross-sectional relationships between biochemical variables are presented in Appendix 10 and are summarised diagrammatically in Figure 5.6. As expected, the two biomarkers that measure immune stimulation - AGP and IgG - were significantly related to each other at each month (with the exception of November, P=.13): children with higher levels of AGP also had higher levels of IgG (R^2 ranged from .08 to .28). A significant, but weak, positive relationship was also observed between albumin and IgG at each month (P<.01 for all months). Children with higher levels of albumin were also found to have significantly higher levels of haemoglobin on five of the seven months of study, although Hb explained very little of the variation in albumin at any given month – R^2 values ranged from just .05

to .13. Gut damage (L:C) was not associated with any blood variable at any month. It is possible that there is a biological time-lag between increased levels of mucosal damage and a corresponding rise in blood markers of immune stimulation; thus, cross-sectional analyses might fail to detect a relationship between these biomarker variables.

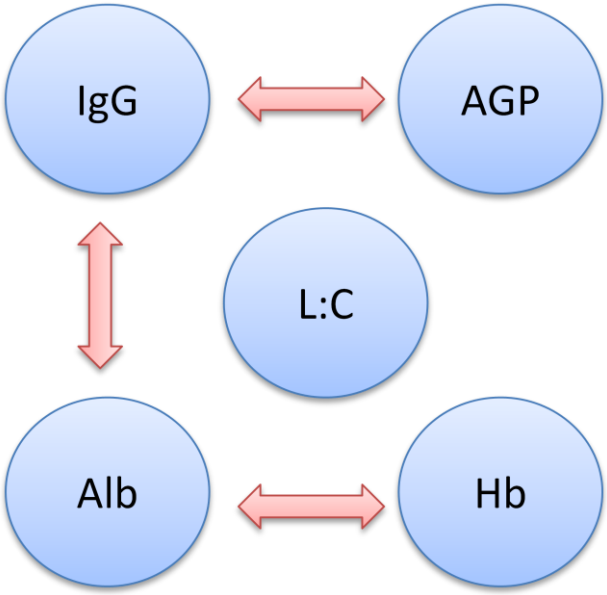


Figure 5.6 Diagram representing the relationships between biomarkers on a monthly cross-sectional basis. Red arrows indicate a positive relationship between variables.

Step 2: Relationship between biomarkers over period of study

In step two, relationships between biomarkers were investigated over the whole intervention period using mean values (Table 5.2, Figure 5.7, Appendix 11). Neither albumin nor Hb were found to be associated with L:C, IgG or AGP in univariate models. They were, however, significantly associated with each other: children with better iron levels were also found to have higher levels of albumin (*P*=.02).

After controlling for age, L:C significantly predicted both IgG and AGP: in both cases, children who experienced a higher mean level of L:C over the period of the intervention had correspondingly elevated levels of IgG and AGP (*P*=.046 and .004, respectively). A significant positive relationship was also found between IgG and AGP (*P*=.006).

In a multivariate analysis model for IgG, the association between AGP and IgG remained (*P*=.024), while L:C was no longer a significant predictor of IgG (*P*=.203). The association between L:C and AGP, however, remained, in the multivariate model, even after controlling for IgG.

	Predictor	Coef.	Std. Err.	t	P	95% CI		Adj. R-sq
mean IgG	Mean age	0.256	0.072	3.560	0.001	0.113	0.399	0.217
	Mean L:C	15.481	12.044	1.290	0.202	-8.469	39.431	
	Mean AGP	2.599	1.133	2.290	0.024	0.347	4.852	
	constant	-2.715	3.930	-0.690	0.492	-10.529	5.100	

	Predictor	Coef.	Std. Err.	t	P	95% CI		Adj. R-sq
mean AGP	Mean age	0.008753	0.007147	1.22	0.224	-0.00546	0.022964	0.160
	Mean L:C	2.694025	1.097636	2.45	0.016	0.511256	4.876795	
	Mean IgG	0.022693	0.009889	2.29	0.024	0.003027	0.042359	
	constant	-0.33428	0.366382	-0.91	0.364	-1.06287	0.394313	

Table 5.2 Multivariate linear regression model assessing the relationships between mean biomarkers after controlling for age.

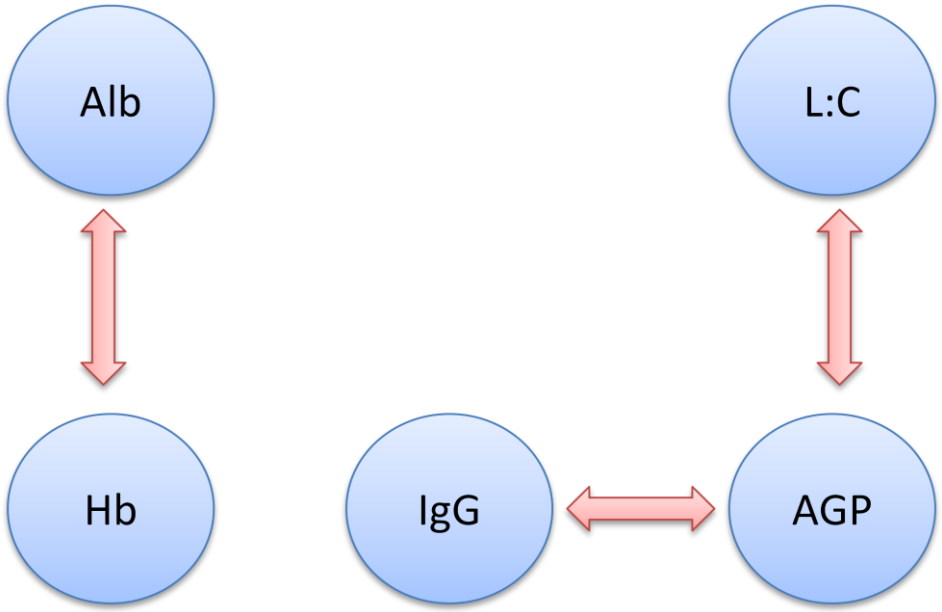


Figure 5.7 Diagram representing the relationships between mean biomarkers. Red arrows indicate a positive relationship between variables.

Step 3: Time series analyses

In multivariate time series analysis, both AGP and albumin were associated with elevated IgG levels (*P*<.01) (Table 5.3, Appendix 12). Together with age, these variables accounted for 45.9% of the between-subject variation in IgG over the intervention period. Though IgG, L:C and albumin were all significantly associated with AGP in univariate analysis, in the multivariate model only IgG remained a significant predictor (although L:C approached significance, *P*=.055). Higher levels of albumin were associated with correspondingly higher levels of Hb (*P*<.001). The relationships between biomarkers as analysed by time-series analysis are depicted in Figure 5.8 below.

	Predictor	Coef.	Std. Err.	z	P	95% CI		Rho
IgG	age	0.448	0.033	13.760	0.000	0.384	0.512	
	AGP	1.598	0.215	7.450	0.000	1.177	2.018	
	Alb	0.130	0.011	12.140	0.000	0.109	0.151	0.459
	Hb	0.015	0.009	1.640	0.101	-0.003	0.034	
	constant	-4.839	0.979	-4.950	0.000	-6.757	-2.921	

	Predictor	Coef.	Std. Err.	z	P	95% CI		Rho
AGP	age	-0.012	0.006	-2.040	0.041	-0.023	0.000	
	IgG	0.043	0.006	6.730	0.000	0.030	0.055	
	L:C	1.036	0.540	1.920	0.055	-0.023	2.095	0.092
	Alb	0.000	0.002	-0.050	0.964	-0.004	0.004	
	constant	0.287	0.201	1.430	0.153	-0.107	0.681	

Table 5.3 Multivariate time series regression models assessing the relationships between biomarkers over the period of the intervention.

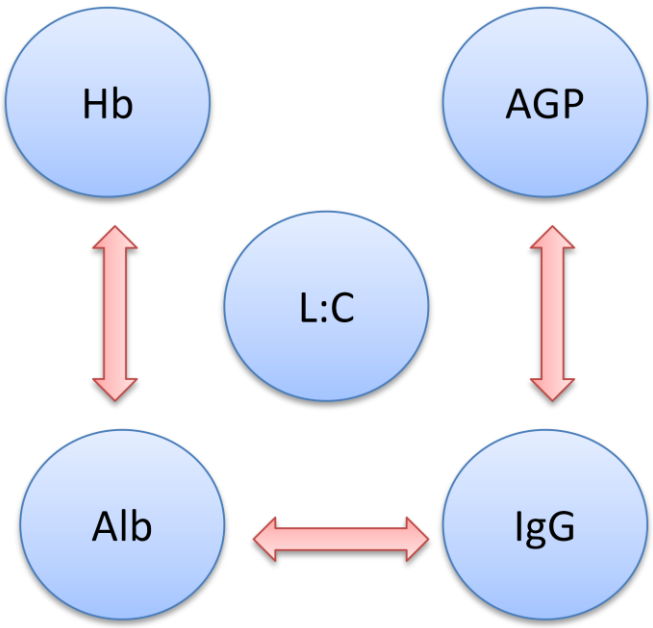


Figure 5.8 Diagram representing relationships between biomarkers using cross-sectional time series analysis. Red arrows indicate a positive relationship between variables.

Summary

The results of this analysis on biomarker variables confirm some expected patterns but not others. Markers of immune stimulation were consistently associated with one another: children with higher levels of AGP also had higher levels of IgG (P<.001). However, the hypothesis predicting higher levels of gut damage (L:C) would be associated with raised levels of immune stimulation was largely unsupported by the data: no association was found between IgG and L:C in any of the analyses, and L:C was only found to be related to AGP when analysing mean values (R^2 = .12, P=.004).

Markers of biochemical nutritional status were, as expected, positively associated with one another: higher levels of albumin were found in children with better Hb levels (P<.001). Higher levels of albumin are generally indicative of better nutritional status or lower immune stimulation (Fuhrman, 2002). The finding that children with higher levels of long-term immune stimulation (IgG) also had higher levels of albumin is therefore unexpected.

5.4.2 Relationships between biomarkers and growth

The next stage was to analyse the relationships between biomarkers and child growth. As before, the relationships were analysed in a three-step process.

Step 1: Monthly cross-sectional analyses

Monthly cross-sectional analyses indicate that blood biomarkers were very poor predictors of growth status at any given month, but gut damage (L:C) was fairly consistently associated with poorer growth in height-for-age, weight-for-age and weight-for-height (Appendix 13).

Children with higher levels of gut damage (L:C) had significantly lower height-for-age z-scores for five of the months of study (June-Oct); however, L:C accounted for only a very small amount of the variation observed for HAZ – just 6-11%. L:C was the strongest and most consistent predictor of WAZ: with the exception of the baseline month, higher levels of gut damage were associated with significantly poorer weight-for-age. Together with age, L:C accounted for between a quarter to a third of the variation observed for WAZ (R^2 ranged from .25 to .38). L:C was also significantly associated with WHZ for four months of study (July-Nov) with higher gut damage levels associated with poorer WHZ for these four months. Together with age, L:C accounted for between 28-39% of the variation observed.

Children with elevated levels of AGP in August had significantly poorer WHZ scores in that month (*P*=.02), and elevated levels in October were associated with lower HAZ and WAZ (*P*=.001 for both). Higher levels of albumin were associated with better HAZ scores for just one month (August, *P*=.02), with the relationship being non-significant at all other times. IgG and Hb were not associated with HAZ or WAZ at any month of study, but unexpectedly, for one month only (September), children with higher IgG values had better WHZ scores (*P*=.01).

Step 2: Relationship between biomarkers and growth over period of study

The relationship between mean biomarkers and growth status of over the whole intervention period are presented in Appendix 14. For all three growth variables (HAZ, WAZ, WHZ) the only significant predictor variable was L:C: in all cases, higher levels of gut damage were associated with poorer growth. Gut damage had the strongest effect on WAZ (coef.= -30.35) and, together with age, accounted for over 40% of the variation in weight-for-age. The relationship between L:C and WHZ was weaker (coef.= -18.60) but

explained a similar amount of the variation in WHZ (R^2 = .40). The relationship between L:C and HAZ was weaker still (coef.= -18.1) and explained just 10% of the variation in this variable. There was no relationship between blood biomarkers and any growth variable.

Step 3: Time series analyses

In multivariate analysis both L:C and IgG were significant predictors of HAZ (*P*=.01 and .03, respectively) (Table 5.4, Appendix 15). L:C showed the strongest relationship with HAZ (coef.= -1.16) and together with IgG and age, these variables accounted for a very significant amount of variation in HAZ between children (Rho= .95).

L:C, AGP and albumin were all highly significant predictors of WAZ in multivariate analysis (*P*<.01 for all) and accounted for 94% of the between-subject variation in WAZ over the period of intervention. Both L:C and AGP showed negative relationships with WAZ indicating that higher levels of gut damage and immune stimulation were associated with poorer weight-for-age, with L:C showing a stronger relationship than AGP (coef.= -1.93 and -.22 for L:C and AGP, respectively). Albumin showed a positive, but weak, relationship with WAZ (coef.= .01, *P*=.001).

L:C, AGP and albumin were also significant predictors of WHZ in multivariate analysis. L:C showed a strong, negative relationship with WHZ (coef.= -1.50), but was the least significant of the three biochemical variables (*P*=.03). AGP was also negatively related to WHZ and was highly significant (coef.= -.03, *P*<.001), whilst albumin showed a weak, but significant, positive relationship with WHZ (coef.= .01, *P*=.002). The relationships between biomarkers and growth are presented in Figure 5.9 below.

	Predictor	Coef.	Std. Err.	z	P	95% CI		Rho
HAZ	age	-0.093	0.006	-16.260	0.000	-0.104	-0.082	0.945
	L:C	-1.162	0.452	-2.570	0.010	-2.049	-0.276	
	IgG	-0.011	0.005	-2.180	0.029	-0.021	-0.001	
	constant	-0.047	0.191	-0.250	0.805	-0.422	0.328	
	Predictor	Coef.	Std. Err.	z	P	95% CI		Rho
WAZ	age	-0.159	0.006	-27.090	0.000	-0.170	-0.147	0.939
	L:C	-1.932	0.525	-3.680	0.000	-2.960	-0.904	
	AGP	-0.219	0.036	-6.110	0.000	-0.289	-0.149	
	Alb	0.006	0.002	3.450	0.001	0.003	0.009	
	constant	0.739	0.224	3.300	0.001	0.300	1.178	

	Predictor	Coef.	Std. Err.	z	P	95% CI		Rho
WHZ	age	-0.081	0.008	-10.360	0.000	-0.096	-0.066	
	L:C	-1.499	0.705	-2.130	0.034	-2.881	-0.117	
	AGP	-0.251	0.048	-5.210	0.000	-0.346	-0.157	0.844
	Alb	0.007	0.002	3.040	0.002	0.002	0.012	
	constant	1.163	0.277	4.190	0.000	0.619	1.707	

Table 5.4 Multivariate time series regression models assessing the relationships between biomarkers and growth over the period of the intervention.

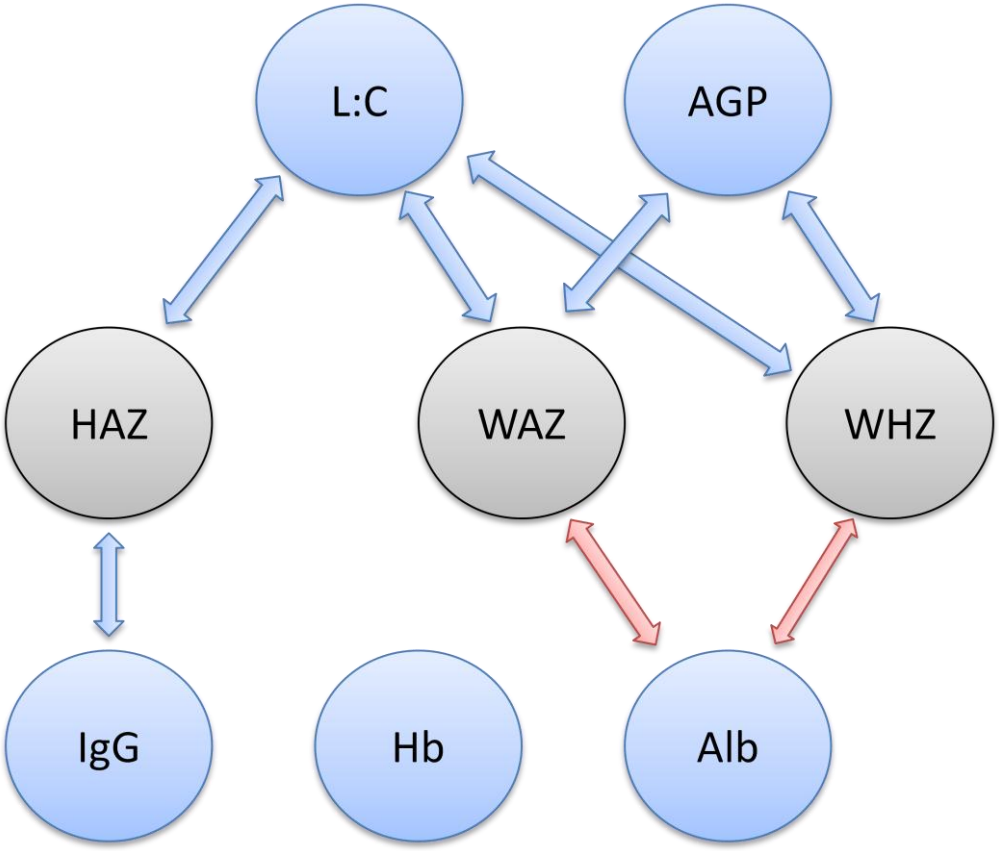


Figure 5.9 Diagram representing relationships between biomarkers and growth using time series analysis. Red arrows indicate a positive relationship between variables, blue arrows indicate a negative relationship.

Summary

All three stages of analysis found significant relationships between gut damage and poorer growth: higher levels of gut damage (L:C) were consistently associated with lower height-for-age, weight-for-age and weight-for-height z-scores. As one would expect, gut damage had the greatest impact on children's ponderal growth (L:C coef.= -1.93 and -1.50 for

WAZ and WHZ, respectively). However, gut damage also had a significant, but weaker, impact on linear growth (L:C coef.= -1.16).

No relationship between elevated immune stimulation and poorer growth was found during the first two stages of analysis; in the more powerful, time series analysis, however, both IgG and AGP were associated with poorer growth. In comparison to gut damage, however, both IgG and AGP had a much smaller impact on growth.

The hypothesis that higher levels of gut damage and immune stimulation are associated with poor growth was therefore confirmed by this study. Figure 5.10 summarises the overall relationships between biomarkers and growth for this study.

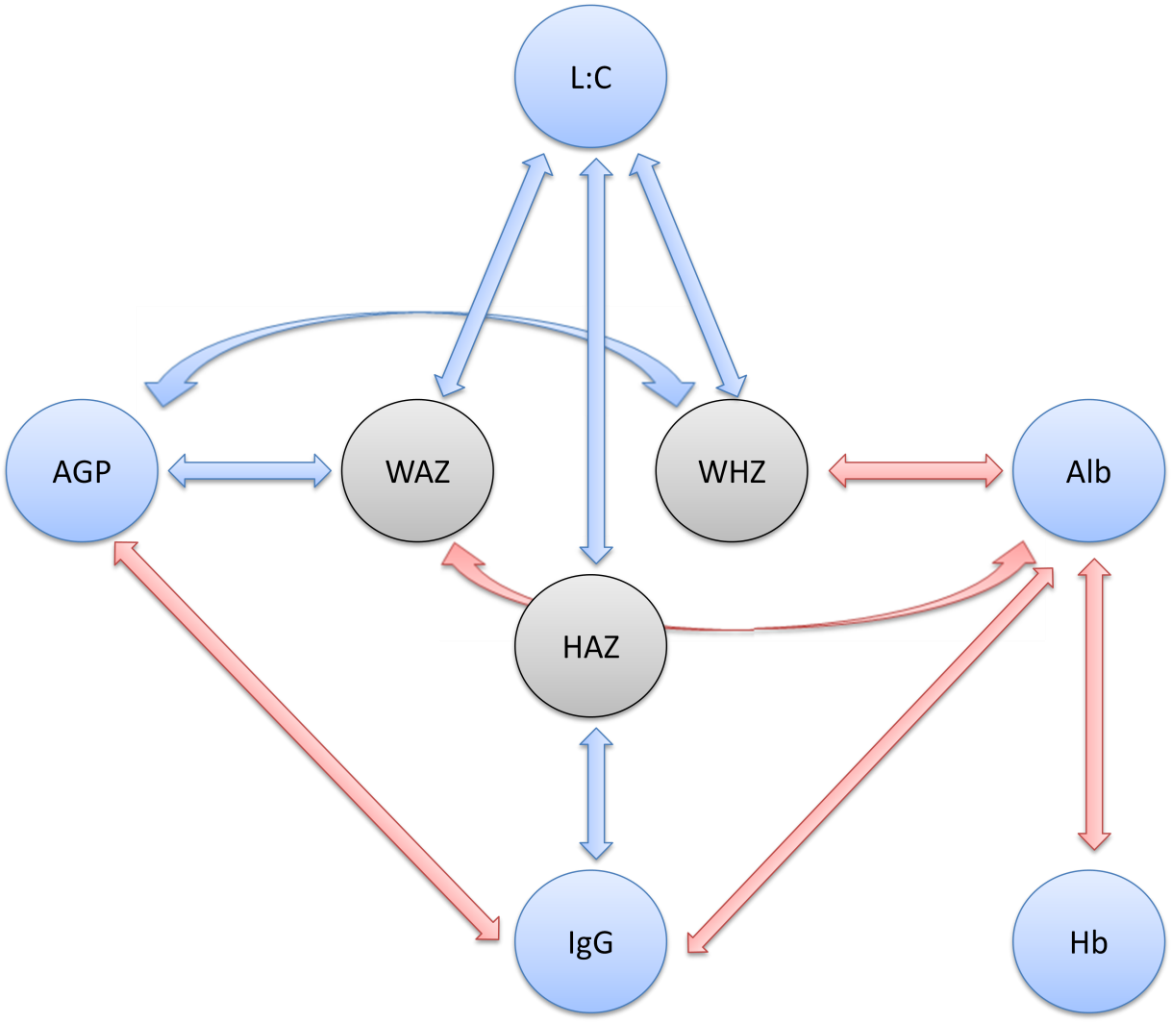


Figure 5.10 Model showing the relationships between biomarkers and growth variables, as analysed by time-series analysis. Red arrows indicate positive relationships, blue arrow indicate negative relationships.

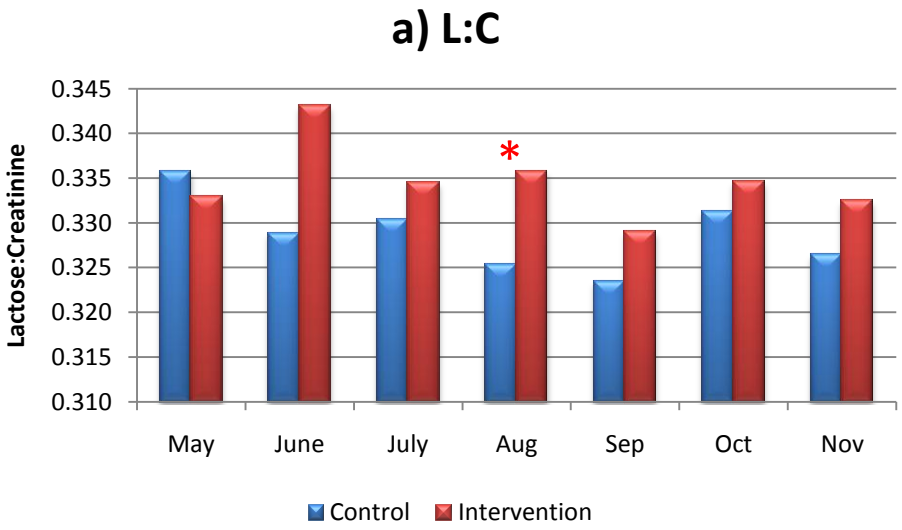
5.5 Level III and IV: Changes in biochemical and growth status

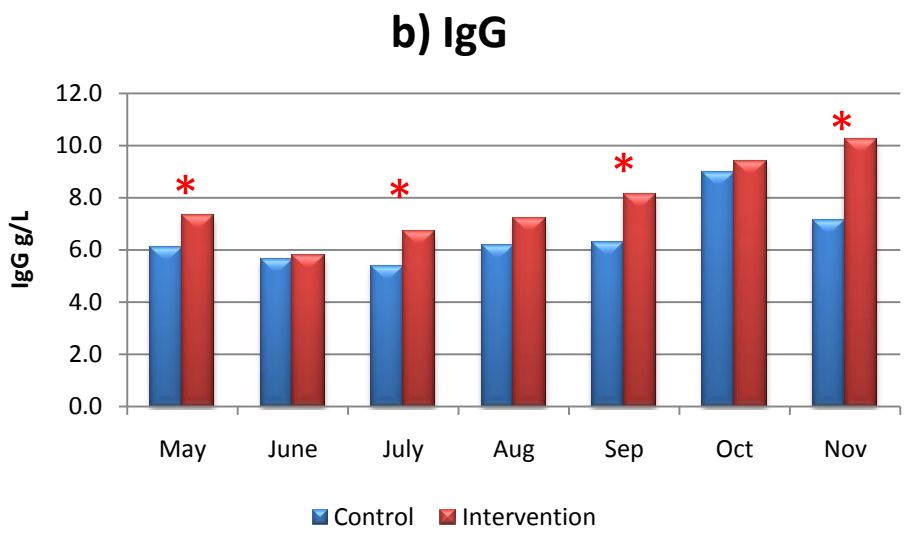
Having confirmed the relationships between the biochemical and growth variables, the impact of the hand-washing intervention on levels of gut damage and immune stimulation and growth faltering was assessed using the same three-step process.

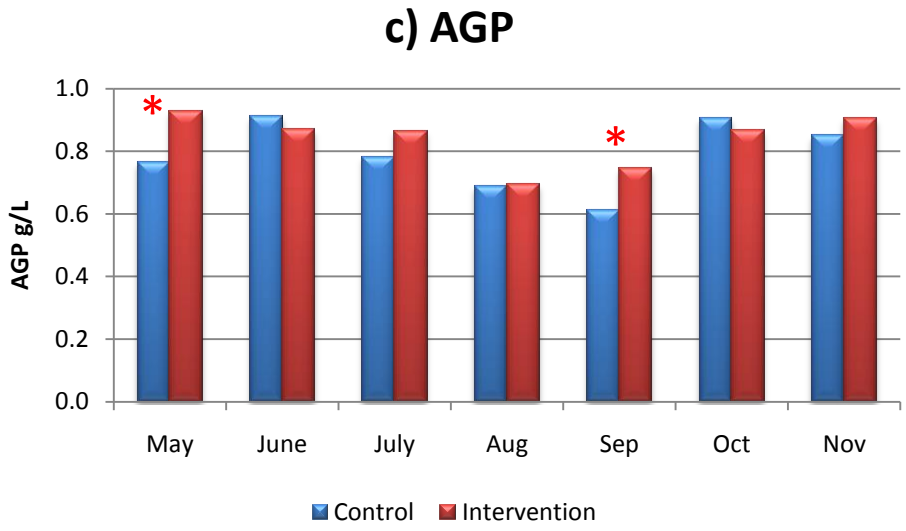
Step 1: Monthly cross-sectional analyses

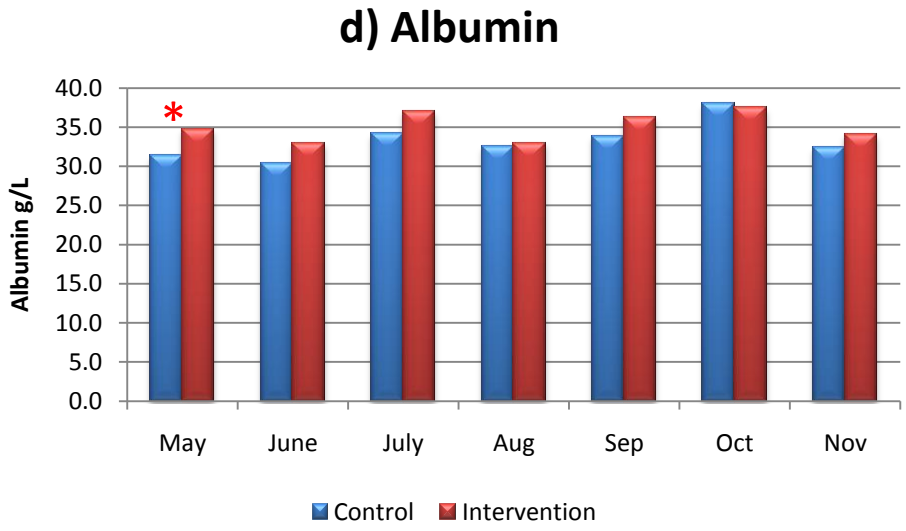
As noted in Chapter 4, baseline differences between the two groups had been observed for three of the biochemical variables. Looking for absolute differences between the two groups was therefore inappropriate: any differences observed may simply have reflected the different starting points of the children, rather than the effect of the intervention. To address this issue, regression models controlled not only for age, but also for baseline differences observed for IgG, AGP and albumin.

Monthly differences between intervention and control groups for all biomarker variables are shown in Figure 5.11a-e. Unexpectedly, children in the intervention areas displayed worse health status relative to their control counterparts, though these differences only reached significance at a few points in time (Appendix 16). For example, for four months of the study, children in the intervention areas displayed elevated levels of IgG (*P*=.001 and .015 for May and July, and <.001 for September and November). They also displayed elevated levels of albumin at baseline (May, *P*=.024), L:C in August (*P*=.03) and AGP in May (*P*=.02) and September (*P*=.006). There were no significant differences between the two groups for levels of haemoglobin.




b) IgG
IgG g/L
12.0
10.0
8.0
6.0
4.0
2.0
0.0
May
June
July
Aug
Sep
Oct
Nov
Control
Intervention


c) AGP
AGP g/L
1.0
0.8
0.6
0.4
0.2
0.0
May
June
July
Aug
Sep
Oct
Nov
Control
Intervention


d) Albumin
Albumin g/L
40.0
35.0
30.0
25.0
20.0
15.0
10.0
5.0
0.0
May
June
July
Aug
Sep
Oct
Nov
Control
Intervention

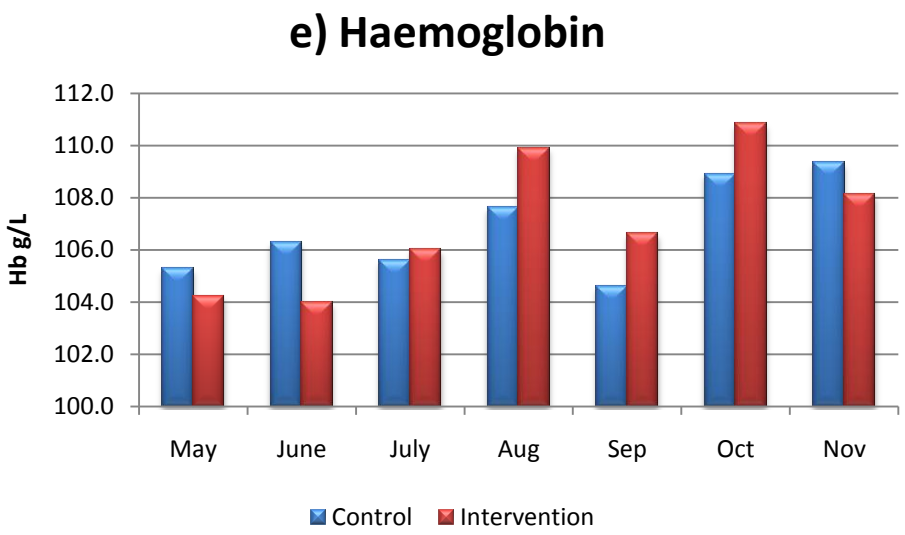


Figure 5.11a-e. Charts showing mean values for all biomarker variables at each month for intervention and control groups. * indicates significant (*P* <.05) differences between groups.

Figure 5.12a-c shows the mean HAZ, WAZ and WHZ scores for the two groups at each month. For all three measures of growth status, children in both groups showed a steady decline over the period of the intervention. Children in the intervention group displayed poorer z-scores throughout; however, at no point do the differences between intervention and control children reach significance (Appendix 16).

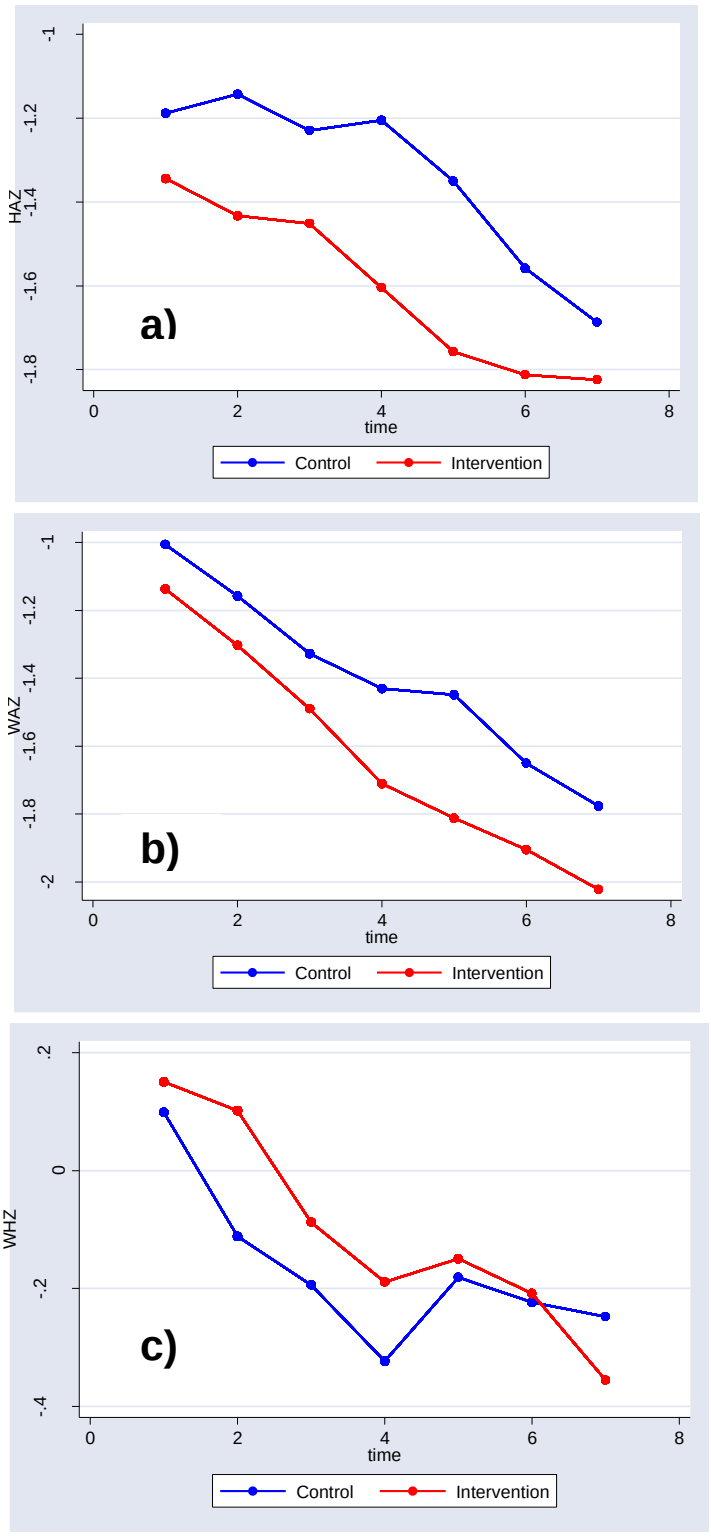


Figure 5.12a-c. Charts showing mean z-scores at each month for intervention and control groups.

Step 2: Mean differences in intervention and control groups over period of study

There were no differences between the two groups for mean levels of L:C, AGP , albumin or Hb over the period of study, nor did the groups differ in mean HAZ, WAZ or WHZ (Appendix 17). Children from the intervention group, however, had significantly higher mean levels of IgG than control counterparts (*P*=.007), as shown in Table 5.5 below.

	Predictor	Coef.	Std. Err.	z	P	95% CI		Adj. R-sq
IgG	IgG (baseline)	0.464	0.062	7.490	0.000	0.341	0.587	0.558
	mean age	0.107	0.057	1.860	0.066	-0.007	0.221	
	group	0.718	0.259	2.770	0.007	0.203	1.233	
	constant	2.598	0.582	4.460	0.000	1.440	3.756	

Table 5.5 Linear regression models assessing the relationships between mean biomarkers after controlling for mean age and baseline IgG.

Step 3: Time-series analysis

Time series models for each variable are presented in Appendix 18. Models with significant interactions between time and group are presented in Table 5.6 below.

Gut damage

There was a significant, yet weak, relationship between gut damage and age (coef.= -.001, *P*=.045), indicating that gut integrity improved as children got older. Children from the intervention areas had slightly higher overall levels of gut damage (coef.= .006) but this difference failed to reached significance (*P*=.05). The hand-washing intervention therefore failed to reduce sub-clinical levels of gut damage in these children.

Immune stimulation

As one would expect, children with high levels of IgG at baseline, had higher IgG levels over the whole period of study (coef= .46, *P*<.001). A significant interaction between group and time was observed for IgG (*P*=.002), indicating that children in the intervention and control groups were changing in significantly different ways over the period of the intervention (Figure 5.13, Table 5.6). IgG levels increased significantly in both groups over the period of the intervention, but the slope of the regression line for the intervention group is steeper indicating that, even after correcting for baseline difference between the two groups, IgG

levels were increasing at a faster rate in the intervention group (coef.= .621, *P*<.001) than in the control group (coef.= .384, *P*<.001). Despite this difference in trajectory, overall the two groups were not significantly different from one another (group coef.= -.235, *P*=.56). The intervention therefore appeared to have no impact on reducing levels of long-term immune stimulation, as measured by IgG.

Similarly, the intervention did not have any effect on reducing levels of AGP in children from the intervention group. After controlling for both age and baseline AGP levels, there were no differences in AGP levels over the period of study between the two groups (coef.= .10, *P*=.74).

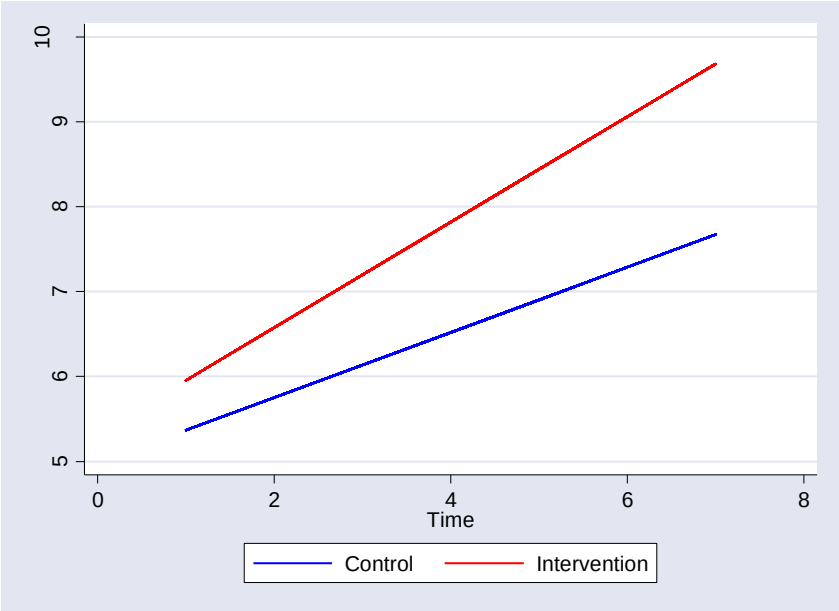


Figure 5.13 Regression slopes for intervention and control groups for IgG.

Nutritional biomarker status

After controlling for baseline values, there was no difference between the two groups in levels of albumin over the period of study (coef.= 1.06, *P*=.09). Haemoglobin increased with age but there were no differences observed between groups (coef.= .198, *P*=.91). Once again, the intervention appeared to have no effect on increasing the biochemical indicators of nutritional status in intervention children.

Growth indices

There was a significant relationship between age and growth (*P*<.001), with older children having significantly poorer scores for all three growth indices. The relationship was strongest with WAZ and WHZ (coef.= -.263 and -.225, respectively) and weakest for HAZ (coef.= -.098).

There were no significant differences between the two groups for HAZ (coef.= -.252, *P*=.21). For both WAZ and WHZ however, the two groups changed in significantly different ways over time (*P*=.012 and .019, respectively for interaction terms). Both groups experienced significant decreases in WAZ over the period of study (Figure 5.14, Table 5.6): children from the intervention areas declined at a faster rate than the control group (coef.= -.149 and -.027, for intervention and control groups, respectively). Despite these different trajectories, overall WAZ scores for the two groups were not significantly different from one another (group coef.= -.068, *P*=.76).

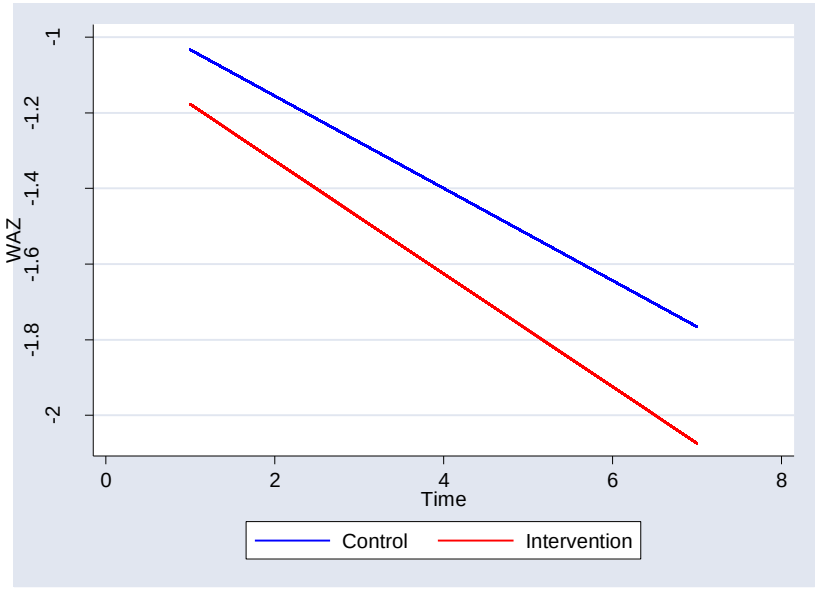


Figure 5.14 Regression slopes for intervention and control groups for WAZ.

WHZ declined over time in both groups, but at a gentler rate than for WAZ (Figure 5.15). Children from intervention areas showed a faster decline than control counterparts (coef.= -.079 and -.034, for intervention and control groups, respectively), but the overall difference between the two groups was non-significant (group coef.= .241, *P*=.162).

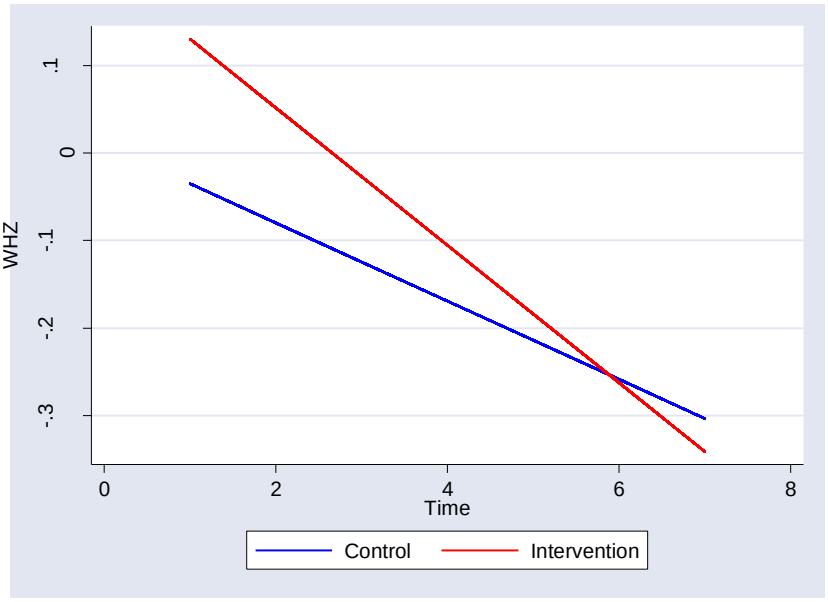


Figure 5.15 Regression slopes for intervention and control groups for WHZ.

	Predictor	Coef.	Std. Err.	z	P	95% CI		Rho
IgG	IgG (baseline)	0.463	0.06	7.67	0.000	0.345	0.582	0.178
	age (baseline)	0.108	0.056	1.93	0.054	-0.002	0.218	
	group	-0.235	0.397	-0.59	0.555	-1.013	0.544	
	time	0.384	0.055	7.02	0.000	0.277	0.491	
	time*group	0.237	0.077	3.1	0.002	0.087	0.387	
	constant	1.349	0.514	2.62	0.009	0.341	2.357	
WAZ	age (baseline)	-0.263	0.045	-5.82	0.000	-0.352	-0.175	0.932
	group	-0.068	0.219	-0.31	0.755	-0.497	0.361	
	time	-0.122	0.008	-15.67	0.000	-0.137	-0.107	
	time*group	-0.027	0.011	-2.5	0.012	-0.049	-0.006	
	constant	1.065	0.374	2.85	0.004	0.333	1.798	
WHZ	age (baseline)	-0.225	0.034	-6.57	0.000	-0.292	-0.158	0.814
	group	0.241	0.172	1.4	0.162	-0.097	0.579	
	time	-0.045	0.01	-4.33	0.000	-0.065	-0.024	
	time*group	-0.034	0.014	-2.35	0.019	-0.062	-0.006	
	constant	1.699	0.285	5.96	0.000	1.14	2.258	

Table 5.6 Time series models analysing difference between intervention and control groups

Summary

The results presented in this chapter show the intervention was successful in achieving behavioural change amongst mothers living in intervention areas: reported levels of hand-washing with soap at the five key junctures increased dramatically by the end of the intervention. This resulted in a 31% reduction in the number of episodes of diarrhoea and a 41% reduction in the total number of days of diarrhoeal symptoms in children living in intervention areas. However, the intervention had no impact on reducing levels of gut damage or immune stimulation: there were no significant differences in the levels L:C, IgG and AGP between the two groups over the period of the study. Similarly, there was no improvement in either albumin or haemoglobin, nor in growth status for intervention children. Thus, although hand-washing was effective in reducing diarrhoeal morbidity, it did not have any impact on sub-clinical levels of infection, nor on child growth rates.

CHAPTER 6
Evaluation of the Intervention

Introduction

As demonstrated in the previous chapter, the hand-washing intervention resulted in a reduction in diarrhoeal morbidity in children living in the intervention areas, but had no effect on levels of sub-clinical gut damage and immune stimulation or growth faltering. This chapter will critically analyse the results of the intervention, exploring what might account for these findings.

6.1 Evaluation of health interventions

Despite the burgeoning number carried out across the world over the past few decades, there are remarkably few examples of truly successful and sustainable behavioural interventions in public health. With the exception of some HIV/AID programmes, few community-based *behavioural* interventions have resulted in more than a modest impact on health outcomes and many have failed to have any impact at all (Merzel and D'Afflitti 2003). Rychetnik et al. (2002) following Hawe (2000), noted that when it comes to evaluating unsuccessful public health interventions, it is necessary to identify the ultimate reason for the lack of success. As they explain,

> *'The evaluation of evidence must distinguish between the fidelity of the **evaluation process** in detecting the success or failure of an intervention, and the relative success or **failure of the intervention** itself. Moreover, if an intervention is unsuccessful, the evidence should help to determine whether the intervention was **inherently faulty** (that is, failure of intervention concept or theory), or **badly delivered** (failure of implementation)'*
>
> Rychetnik et al. (2002:119, my emphasis)

This advice is summarised in Figure 6.1 below. I use this framework here to evaluate the hand-washing intervention, considering the possible reasons for the intervention's lack of impact. Firstly, I will examine the possibility of *implementation failure*: the possibility that implementation of the intervention failed to increase hand-washing practice amongst the mothers. Secondly, I will consider the possibility of *evaluation failure*: the possibility that hand-washing behaviour increased, but the study failed to detect its impact on the health and growth of the children. Thirdly, I will consider the possibility of *intervention failure*, distinguishing between the *efficacy* of the intervention – concerned with establishing a plausible, biological link between the intervention and outcome variables – and the *effectiveness* of the intervention – concerned with the effect of the intervention when implemented under real-world conditions.

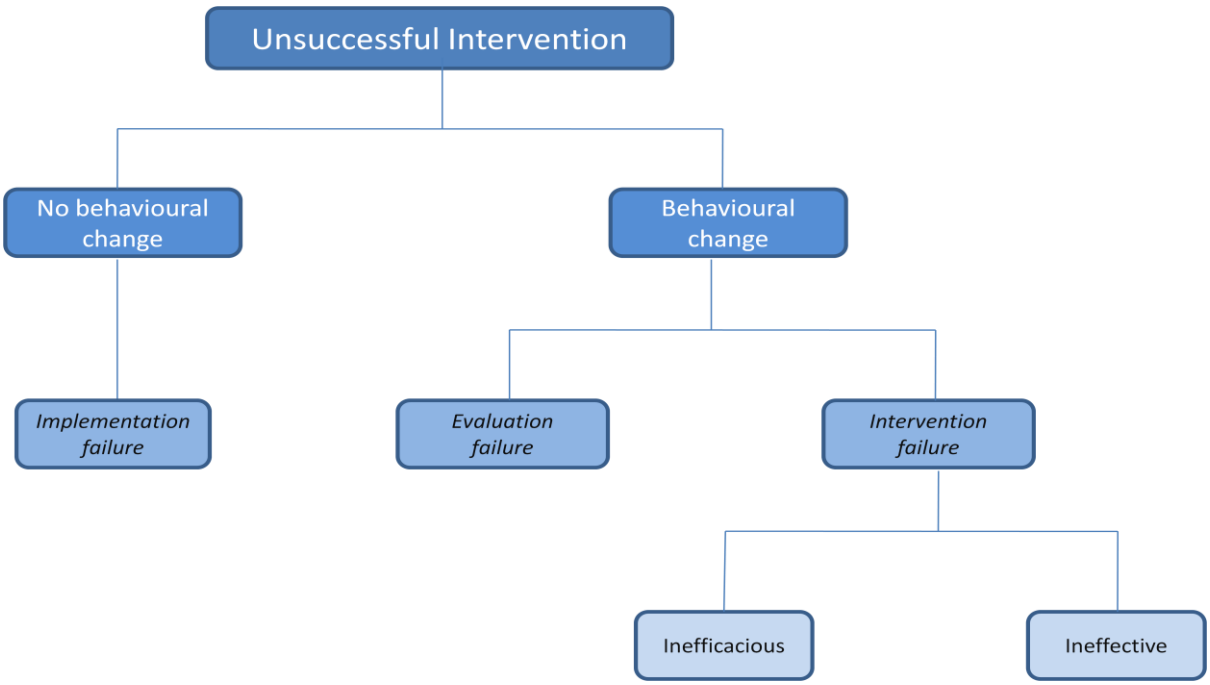


Figure 6.1 Model depicting different types of intervention failure.

6.2 Implementation failure

Was the intervention successful in increasing maternal hand-washing practices at the five key junctures identified by the intervention message? Perhaps the reason why the study revealed

no significant differences between the two groups in terms of gut damage, immune stimulation and growth was because the intervention failed to motivate behavioural change. Assessing changes in behaviour in a simple, yet reliable, way is a difficult task, especially so when the behaviours in question are personal hygiene practices which often take place out of sight and carry strong moral connotations (Curtis, Biran et al. 2003). This study employed a number of methods to assess changes in hand-washing behaviour, following techniques used in other hand-washing studies described in Chapter 3. Here I consider the validity of each of these methods and discuss the methodological difficulties in accurately documenting changes in behaviour following an intervention programme.

6.2.1 Observations of hand-washing behaviour

Opportunities for informal observations of hand-washing and hygiene behaviour occurred frequently throughout the duration of the intervention – during interviews, health-checks, group meetings and community events etc. These observations were particularly useful at the start of the project, helping to determine when and where hand-washing occurred. However, such observations provided only a rough indication of when hand-washing occurred. In order to collect quantifiable data on both the frequency of hand-washing and the junctures at which it was most likely to occur I conducted structured observations of hygiene behaviours in the mothers' own houses.

Initially, I planned to conduct structured observations in both intervention and control households before the start of the intervention (May 2007) and again at the end of the project (November 2007). This would have allowed for direct comparisons between the two groups, as well as documenting changes in hand-washing behaviour over the period of the intervention. However, numerous methodological problems were encountered that meant that this plan could not be carried out and thus the usefulness of the observations that were conducted was limited. Below, I briefly outline some of the difficulties.

Though arguably more reliable than self-reports in measuring behavioural change, observations have the down-side of being difficult to implement, time consuming, intrusive and expensive. A sub-sample of households were randomly selected and observed for a three-hour period in the morning, following the recommendations of Curtis et al. (1993), Cousens et

al. (1996) and Biran et al. (2005). However, in practice, the number of junctures where hand-washing might have occurred during this three-hour period, was generally very low; out of the 75 observations carried out, children were seen to defecate only 29 times, and mothers only 14 times. Feeding of the child was also relatively infrequent, occurring during just 26 of the observations. Consequently, very little data on hand-washing behaviours were collected, not because it was not practiced by the mothers, but simply because the opportunity to observe this behaviour occurred infrequently. The conclusions that could be drawn as to the prevalence of hand-washing behaviours in these communities were therefore limited. In addition, the low number of events observed meant that statistical comparisons between the two groups were not possible.

As a result of these problems I decided not to conduct a second round of observations at the end of the intervention period. Because so few hand-washing events were observed, statistical comparisons comparing rates of hand-washing between the two groups, or changes over the period of study would not have been possible. In addition, by the end of the intervention period I felt that any data collected during observations would have been hopelessly biased due to the problem of behavioural 'reactivity'.

Reactivity – whereby the actors modify their behaviour as a result of being observed – is one of the major limitations of structured observations (Cousens, Kanki et al. 1996). Various precautions were taken at the beginning of the study to limit the impact of reactivity as much as possible: mothers were not informed of the focus of observations beforehand and local fieldworkers were used, having been well-trained in the unobtrusive collection of these data. However, had the observations been conducted again at the end of the intervention, in the intervention areas at least, the mothers would have known full well the fieldworkers were interested in observing hand-washing practices and would likely have increased their hand-washing practices correspondingly[22].

As a result of the limitations mentioned above, I was not able to use structured observations as a means of evaluating the impact of the project on hand-washing rates. In an ideal scenario, I

[22] Curtis et al.'s (2001) hand-washing study in Burkina Faso did not suffer from these limitations to the same extent, as their study was conducted over a much longer time period. Baseline observations were conducted at the start of the study and then repeated over three years later. Thus, the mothers were unlikely to connect the two sets of observations and modify their behaviour.

would have conducted observations in *every* household, on *repeated* occasions, and for a *longer* period of time. Cousens et al. (1996) showed that observations conducted repeatedly within a household significantly diminish reactivity, as the actors 'acclimatise' to the presence of the observer. This, combined with a longer observational period, would have ensured that enough hand-washing junctures were observed to allow for statistical comparisons.

In this study, however, increasing the length and number of observations would have incurred significant additional financial cost, disrupted the very tight research schedule and would have proved unpopular with both the mothers and fieldworkers alike. The observations tended to run from about 6am-9am, at which point the mother would usually start getting her older children ready for school. Extending the time period of the observations would therefore probably not have produced many more opportunities for the observation of hand-washing since the mother would soon be leaving the house. In addition, the fieldworkers often found sitting for several hours in cold, dark and often smoky atmospheres difficult and I believe that asking them to observe for longer than three hours would have resulted in a loss of concentration and a decline in data quality. It should also be noted that though a greater number of observations of longer duration might have solved some of the problems encountered, it would not have solved the issue of reactivity at the end of the project and thus would still not have been able to produce reliable data by which to assess changes in behaviour over the intervention period.

6.2.2 Self reports of hand-washing behaviour

As a result of these issues, I had to rely on self-report of hand-washing behaviour in order to assess the impact of the intervention. Questionnaires based on self-reported data are commonly employed as an evaluative tool in interventions as they are both simple and inexpensive to implement. However, there are two significant limitations to this method. Firstly, self-reports of behaviour are often subject to high levels of recall error: it is difficult to remember accurately one's behaviour in a given situation. Secondly, self-reports may be subject to recall bias: participants may not give honest answers, leading to significant over- or under-reporting of the behaviour in question (Cousens, Kanki et al. 1996). The problem of recall bias is exacerbated when the behaviour in question is personal, sensitive or morally

loaded - hand-washing being a prime example of this. In-depth interviews conducted during the preparatory stage of this project revealed that mothers regarded cleanliness as extremely important and a lack of hygiene and cleanliness was associated with negative moral connotations. Given these connotations, it is unlikely that the mothers gave completely accurate reports of their hand-washing practice, since few mothers would be prepared to admit openly a failure to comply with this societal norm. The problem of recall bias was almost certainly compounded further in the intervention areas where, after six months of the intensive hygiene promotion, the mothers would have known to supply the 'correct' answer in response to questions about their hand-washing behaviour.

The unreliability of self-reports of hygiene behaviours has been noted in numerous other studies and it is now generally acknowledged that self-reports consistently show poor correlations with other markers of behaviour. In a project that aimed to investigate maternal hygiene behaviours, Curtis et al. (1993) found discordance between mothers reports of behaviour and structured observations of their actions over a period of three-hours: though 75% of mothers said that the child defecated in a pot, only 66% of children were seen to do so (kappa score = 0.25) . In addition, 67% mother reported disposing of their children's faeces in the toilet, but observations revealed only 56% actually did so (kappa score = 0.28). Stanton et al. (1987) set out to investigate the accordance between self-reports of hygiene behaviour based on 24-hour recall, and direct observation lasting between three-to-five hours in Dhaka, Bangladesh. The authors found very low agreement (kappa scores < 0.10) between 24-hour recall reports and the observations for a number of hygiene behaviours; in each case the discrepancy arose because of the mothers' tendency to over-report the 'correct' behaviour. The agreement between reports and observations of hand-washing after defecation was extremely low – the kappa score being just 0.01.

Although reported and observed hand-washing practices were not recorded specifically for every mother, this current study also found discrepancies between what mothers said they did and what they were actually observed to do. Whilst 96% of mothers claimed to wash their hands with soap after defecation, during three-hour observations conducted at baseline on a randomly selected sub-sample of mothers (n=75), only 50% were seen to wash their hands at all, and just 17% used soap. Similarly, while 81% of mothers reported washing hands with soap after cleaning the baby's bottom, just 14% of the observed mothers were seen to do this.

(An additional 17% washed their hands with water alone). Given the significant doubts regarding the validity of self-reports, the dramatic increase in reported hand-washing with soap in mothers from the intervention areas by the end of the intervention, cannot be regarded as proof that behaviour change actually did occur.

Given these limitations, why were self-reports of behaviour included in this study? Firstly, the discrepancy between the self-reports and the observations of behaviour that were conducted during the preparatory stage of the intervention provided interesting and informative data regarding the cultural pressure to perform hand-washing and/or the hygiene knowledge of the mothers. For example, at baseline, reported hand-washing with soap after contact with faeces was near universal, but hand-washing before handling food was much less common. This suggested either that hand-washing was not regarded as necessary before contact with food, or that there was considerably less cultural pressure to wash hands at these junctures, or both. In terms of designing the intervention this was valuable information to know: clearly special attention needed to be paid to the promotion of hand-washing before contact with food.

Secondly, changes in reported behaviour provided important evidence to suggest increases in the knowledge about when hand-washing should take place. The significant increases in reported hand-washing amongst mothers from the intervention areas over the period of study, whilst not providing reliable evidence of *actual* behavioural change, did provide compelling evidence of an increase in *knowledg*e about the importance of hand-washing at these five key junctures. Though it is well known that knowledge alone is not enough to initiate changes in behaviour it is probably often an important precursor to such change.

6.2.3 Measures of soap usage

A number of studies described in Chapter 3 measured soap usage to monitor compliance with hand-washing practices: Khan (1982) and Shahid et al. (1996) both reported inspecting soap bars for use during regular visits to participating households; Han & Hlaing (1989) weighed bars of soap with electronic scales to determine usage; Sircar et al. (1987) reported measuring the dimensions of the soap every two weeks throughout the intervention period. It is interesting to note, however, that none of these papers report the results from these

inspections; it appears that rather than being quantifiable measures of hand-washing practice, these methods were used as a qualitative means of assessing compliance.

Because of the limitations of both self-reports and observations, I wanted to measure consumption of soap as an additional indicator of hand-washing practice. However, weighing or measuring the dimensions of soap bars in each household seemed too complicated a method to employ and would have required the purchase of several highly sensitive weighing-scales. Instead, we employed a simple and pragmatic approach: we assessed soap consumption simply by asking the mother how many *new* bars of soap she had started that week (including laundry, dish and body soap, since interview data had indicated that all three types of soap were used for hand-washing), adjusting this value for household size.

However, as seen in Chapter 5, the intervention failed to increase household consumption of soap in the intervention areas: at baseline the two groups both used 4.3 bars of soap per month and at no point thereafter did a significant difference in soap consumption emerge. Does this indicate that hand-washing practices did not increase amongst the intervention mothers? This is certainly possible. However, it should be noted that this was a very crude approach to measuring soap consumption and was unable to distinguish between soap used for hand-washing and soap used for other hygiene purposes. Thus, it may be that this indicator was simply not sensitive enough to detect real changes in soap consumption between the two groups as a result of changes in hand-washing behaviour.

6.2.4 Morbidity levels in children

By far the most common outcome measure used to detect the effectiveness of a hand-washing intervention is child morbidity levels. Every intervention described in Chapter 3, with the exception of Curtis et al. (2001), used child morbidity as the main indicator of the intervention's success. Given that a reduction in diarrhoeal morbidity is usually the ultimate aim of a hand-washing campaign, it seems somewhat circular to use this outcome, simultaneously, as an indicator of changes in hand-washing practice. Nonetheless, a reduction in morbidity in the absence of any other obvious change in environmental conditions would seem to provide quite compelling evidence of the intervention's effectiveness in instigating behavioural change in the mothers.

Following other hand-washing studies, child morbidity was used as a key outcome variable in this current study. As discussed in the previous chapter, the intervention was effective in reducing child morbidity: children in the intervention areas experienced 31% fewer episodes of diarrhoea and 41% fewer days with diarrhoeal symptoms over the entire intervention period, as compared with their control counterparts. These data therefore seem to provide good evidence that the intervention was successful in increasing hand-washing behaviour amongst intervention mothers, resulting in a reduction in child morbidity. However, though it is tempting to suggest that morbidity reports are more objective and reliable than either self-reports or observation of behaviour, it should be noted that this method is also not without its limitations.

Most community intervention studies in the developing world at least, tend to rely on child morbidity data collected via interviews with the child's mother or primary care-taker. Rousham et al. (1998) found close associations between maternal morbidity reports and biochemical indicators of infection in Bangladeshi children. On the other hand, Panter-Brick et al. (2001) found considerable under-reporting of morbidity in rural Nepali boys in comparison to pathogenic exposure indicated by objective markers of immune response ascertained from their blood tests.

Because of the possibility of over- or under-reporting of symptoms using this method, some researchers advocate assessing morbidity via clinical assessment. This approach has the advantage of identifying asymptomatic conditions (such as anaemia or helminthiasis) that the patient/carer may be unaware of, as well collecting more accurate data on the prevalence of chronic conditions which are often under-reported in lay interviews (Ross and Vaughan 1986). However, since the main symptoms of interest to this project (diarrhoea, colds and fevers) are easily recognised and reported by mothers, physician examination and diagnosis was not felt to be necessary.

However, it is possible that the difference in morbidity between the children from intervention and control areas was the result of differences in reporting practices, rather than the impact of the intervention in increasing hand-washing practice. The intervention message promoted to the mothers in the intervention areas explained that hand-washing with soap could reduce sickness in their children. In addition, there was a strong moral component to the intervention

message: during the home visits and group meetings, the Community Motivators promoted the idea that hand-washing was something that 'good', 'clean' mothers did. Given the moral connotations of these messages, it is possible that the mothers in the intervention areas purposely under-reported sickness in their children: reporting sickness in their child might have reflected badly on them by suggesting that they had not been washing their hands. If this were the case, the reduction in morbidity in intervention children would have been due to this social pressure to under-report sickness, rather than any true biological effect on morbidity rates.

Attempts were made during the collection of morbidity data to mitigate such reporting bias. I chose young, local women to collect the morbidity data on a weekly basis. Unlike the Community Motivators or myself, these fieldworkers were unlikely to have been seen as 'senior' to the mothers, and were thus arguably more likely to be able to elicit truthful responses regarding morbidity levels. In addition, none of these fieldworkers was ever involved in any aspect of promoting the intervention message to the mothers; their role was entirely confined to structured observations, morbidity reports and helping to organise the monthly health checks.

Based on my experience of talking with the mothers during the intervention, I judge that mothers in the intervention areas were unlikely to have purposely under-reported morbidity levels. Although most mothers saw a clear connection between dirt and disease, they also strongly believed that many sicknesses experienced in childhood were unconnected to issues of hygiene. Mothers frequently attributed diarrhoea, fevers and colds to episodes of teething, attacks by evil spirits and changes in the season or weather. Given that sicknesses could have occurred in their child for any of these reasons, it seems unlikely that there would have been much social pressure to purposely under-report morbidity. I therefore believe that the reduction in diarrhoeal morbidity amongst the children in the intervention areas is a result of improved hand-washing by their mothers, suggesting that the intervention was successful at promoting behavioural change.

6.2.5 Qualitative evidence for behavioural change

This study aimed to collect several lines of evidence to document behavioural change in the mothers from the intervention areas. None, however, provided unequivocal evidence to suggest that behavioural change occurred. The difficulty in accurately recording behavioural change is a well-documented and significant challenge to behavioural hygiene interventions. Literature on hygiene and hand-washing interventions frequently notes there is currently no 'ideal' method for assessing changes in hygiene behaviour that is accurate, reliable, simple to implement and cost-effective (Kaltenthaler and Pinfold 1995; Cousens, Kanki et al. 1996; Curtis, Biran et al. 2003; Curtis and Cairncross 2003). In their systematic review of hand-washing intervention papers, Curtis and Cairncross (2003) state that all of the studies reviewed were methodologically flawed and none provided adequate data on compliance with hand-washing practices. Clearly this is an issue that requires further research into appropriate tools for evaluation. It is likely, however, that the 'ideal' method will remain elusive. At present, efforts must be made to use the methods we do have in the most scientifically rigorous way, with multiple lines of evidence allowing us to build up a general, if not definitive, picture of whether change had occurred (Kirkwood, Cousens et al. 1997).

To this end, it is worth considering one final line of evidence: that of the qualitative data collected from the Community Motivators responsible for implementing the intervention. Meetings were held with all Community Motivators every two weeks throughout the intervention period, and I had the opportunity to meet more informally with each of them as I spent time in the communities. During these meetings we discussed how the intervention was progressing in each community, how receptive they felt the mothers were to the intervention message and whether they were increasing hand-washing practices as a result. The Community Motivators visited every mother on a regular basis, getting to know them well over the intervention period. As such, they were perhaps better placed than anyone to assess the success of the intervention in instilling better hand-washing practices.

The Community Motivators were generally very positive about the impact of the intervention; they felt that most of the mothers were very interested in and receptive to the intervention message. During the first few weeks they spent time talking with the mothers about the importance of hand-washing, encouraging them and reminding them to use soap at the five key junctures. After the first few weeks, they felt quite confident that most mothers were now

washing their hands with soap much more often than they had been before. They conducted informal spot checks of the soap bars to see if they were being used, and reported that they often saw women using the soap for hand-washing when they visited their houses or simply as they walked through the community. The Community Motivators also said they had heard the mothers themselves telling their neighbours about the project and promoting the importance of hand-washing. The Community Motivators also talked to the husbands and children in each household and it was clear that many of the mothers were very enthused about this project and had got the whole household to increase their hand-washing practices.

It could be argued that the Community Motivators had a vested interest in providing positive feedback about the success of the intervention, since this reflected well on their own performance. However, there are a number of reasons why I do not suspect the Community Motivators of over-reporting hand-washing behaviour. Firstly, they admitted they had had greater success at encouraging hand-washing after contact with faeces, than with instilling the need to wash hands before contact with food. Their assessment of the mothers behavioural change therefore fitted closely with the results we obtained from the self-reports of behaviour at the end of the intervention. Secondly, rather than suggesting that *all* the mothers had improved their hand-washing practices, the Community Motivators were able to discuss at length the success of the intervention for each individual mothers. Thus, from their own observations, they could confidently name those mothers who consistently remembered to wash their hands at each juncture, those who remembered most of the time, and those few mothers who had not improved their hygiene practices at all. This conversation would have been unlikely amongst people with a vested interest in reporting wholesale behavioural change.

6.2.6 Summary

Significant reductions in diarrhoeal morbidity amongst children in the intervention areas, the qualitative data collected from Community Motivators and the mothers' reports of their own behaviour all indicate that the intervention was successful in increasing hand-washing practice amongst mothers in the intervention areas. The intervention's failure to reduce levels of gut damage, immune stimulation and growth faltering in the children, is therefore unlikely to be due to a failure to successfully implement the intervention.

6.3 Evaluation failure

Was the study suitably designed and powered in order to be able to detect the effect of the intervention on the outcome variables? This section will examine some of the limitations in study design and methodology that may have compromised the ability of this study to detect the true impact of the intervention on the various measures of child health. It will start with a discussion regarding study design in community-based behavioural interventions. It will then focus in particular on sampling issues and the length of the intervention period.

6.3.1 Study design

Given the inherent complexity of community-based behavioural interventions, careful consideration must be given to the most appropriate study design in order to create a project that is scientifically rigorous whilst at the same time being logistically feasible to implement. Double-blind, randomised controlled trials (RCTs) are deemed to be the 'gold standard' for evaluating the impact of health interventions because of their ability to minimise bias and avoid false conclusions (Kirkwood, Cousens et al. 1997; Stephenson and Imrie 1998). Though widely applied in the evaluation of new drugs or medical treatments, they have been less consistently and less successfully used in evaluating community-based health interventions that focus on behavioural change (Lambert, Gordon et al. 2006). In a review of 57 non-RCT community health intervention studies, Smith et al. (1997) found that over 40% of the studies presented could and should have been analysed using RCT methodology, and concluded that RCT study design was underused in community health research resulting in many interventions not being evaluated in a scientifically rigorous manner.

As Smith et al.'s review suggests, RCT methodology and community-based health interventions are not incompatible. There are, however, numerous reasons why an RCT approach is considerably more challenging (or impossible, in some settings) in community-based behavioural research. Drugs trials are generally much more suited to evaluation using RCT methodology; intervention protocols can be precisely defined, implemented in a standardised way and adherence closely monitored (Glasgow, Klesges et al. 2004). In addition, blinding of both the participants and researchers is fully possible. Community-based

behavioural trials, however, present significant challenges to RCT methodology. Interventions tend to be highly complex and context specific (Campbell, Fitzpatrick et al. 2000; Rychetnik, Frommer et al. 2002); analysis is often conducted at community level with significant implications for sample size requirements (Kirkwood, Cousens et al. 1997); logistical and/or political issues often determine allocation to intervention groups, rather than being truly randomised (*ibid*); monitoring of 'compliance' can be difficult (Glasgow et al. 2004); and blinding of participants and researchers is often impossible (Smith, Moffat et al. 1997; Davidson, Goldstein et al. 2003) .

Many of these issues were pertinent to this study and prevented the use of an RCT study design. Firstly, it was not possible to randomly allocate each slum community to the intervention or control group. As explained in Chapter 2, several of the slum communities were very close together and had to be treated as one single group in order to prevent 'contamination' of the hand-washing message from intervention to control areas. Thus, two 'clusters' of communities were created – one in the south-east and one in the north-east. It was these 'clusters' that were randomly allocated to intervention or control conditions. Grouping communities into clusters is a common practice in large-scale community interventions, with resulting data analysed at the community levels using summary scores created for each community (Kirkwood, Cousens et al. 1997). However, because analysis in this study remained at the level of the individual, this cannot be considered a cluster-randomised controlled trial.

Secondly, it was not possible in this study to blind either the participants or the researchers to their intervention condition. Blinding minimises bias and ensures that errors in measurement outcomes will occur with equal frequency in both group. This provides a conservative estimate of the intervention's impact since it will tend to mask, rather than exaggerate, any impact (Kirkwood, Cousens et al. 1997). Blinding is clearly not possible for an intervention focusing on hand-washing behaviour. However, efforts were made to minimise bias as far as possible: fieldworkers were rigorously trained in standardised data collection procedures and none of the fieldworkers involved in collecting hygiene or morbidity data was ever involved in the promotion of the hand-washing intervention.

Thirdly, although a control group was included in the study, this was not necessary a rigorous enough control for an RCT design. Although many behavioural RCTs use 'usual care' or 'usual practice' as their control condition, this practice has been criticised as being inadequate (Schwartz et al. 1997). A 'placebo' intervention that has no impact on the outcomes of interest must be implemented with equal intensity in control sites (Schwartz et al. 1997) in order to control for (Schwartz, Chesney et al. 1997)(Schwartz, Chesney et al. 1997)a 'Hawthorne effect', whereby changes in outcome variables occur not because of the intervention content per se, but as a result of participating in the research and the frequent contact and monitoring this involves. However, as Davidson et al. note, 'in contrast to drug interventions in which active drugs and placebos look exactly the same, it is difficult to achieve a behavioural placebo that has the same appearance and credibility as the active treatment' (2003:166). In their hand-washing intervention study in rural Zaire (now the Democratic Republic of Congo), Haggerty et al. (1994) implemented an educational intervention in their control sites to promote i) continued breastfeeding of the child during episodes of diarrhoea and ii) the use of oral rehydration salts. This was deemed to be a suitable placebo intervention, since neither of these messages would directly impact on the incidence of diarrhoeal disease.

I had originally considered the need for a placebo intervention for the control sites, and planned to follow Haggerty et al. (1994) in promoting the use of ORS as the placebo message. However, upon arrival in Kathmandu this idea had to be abandoned for a number of reasons. Initial interviews regarding childhood morbidity revealed that virtually every mother already knew about and used ORS, thus rendering this an inappropriate placebo message. As Haggerty et al. (1994) also found, it proved difficult to determine a suitable placebo intervention message that would not impact on childhood morbidity, whilst still remaining credible to the mothers. Luby et al. (2005) provided educational materials such as pens and paper etc. to control families as a placebo intervention, but we rejected this idea on two grounds: firstly, the children enrolled in this study were too young to benefit from such educational materials; secondly we felt that the mothers would find it incongruous for us to be promoting an intervention that had no health message at all, whilst at the same time implementing weekly morbidity reports and monthly health checks on all children.

In addition, implementing a credible placebo intervention at the same intensity and with the same level of resources as the hand-washing intervention would have proved logistically and

financially impossible. Any placebo intervention would have required proper formative research and detailed planning in order to be credible and properly executed. It simply was not possible to conduct such formative research on top of all the other activities that were being carried out in relation to the real hand-washing intervention. Financially it would also have required employing an extra five Community Motivators to work in the control sites – a burden that could not have been sustained on the limited budget available, on top of the salaries of the 18 other teams members.

As a result of the logistical, financial and time constraints mentioned above, this study was not able to apply a strict RCT design, and instead employed a simple intervention vs. control comparison to assess the impact of the intervention. Ideally, pre- and post-intervention comparisons would also have been done, but this was not possible for two reasons. Firstly, given the time constraints of the project, it was not possible to collect several months of baseline data before the start of the intervention. Secondly, if several months of baseline data had been collected, the children would have grown older during this time and many of them would have then fallen outside of the target age range of the study. Instead, only one month of baseline data was collected in order to assess the comparability of the two groups before the start of the intervention.

6.3.2 Sampling issues

As explained in Chapter 2, the required sample size for this study was calculated using data collected from slum children in Kathmandu in a previous study (Panter-Brick, Lunn et al. 2009). However, sample size calculations determined using previously-collected data can only ever provide an approximate guide to the number of participants required to detect statistically significant effects in a new study. One reason why the intervention failed to have any impact on levels of gut damage, immune stimulation and growth faltering in this current study may be that the sample was too small, resulting in a statistically under-powered study.

Originally, this study planned to recruit only children aged between six and twelve months, as our previous research had identified this as the key period for growth faltering (Panter-Brick, Lunn et al. 2009). The required sample size was calculated to be 88 children, increased to a target of 100+ in order to accommodate the possibility of children being lost to follow-up.

However, having conducted a survey of the number of children aged between six and twelve months living in the largest slum settlements, it became clear that the required sample size could not be achieved using this age-range criterion; across eight field sites only 65 children fell into this age range. Including other field sites to boost numbers was deemed to be impractical; because of the small size of most of the settlements we would have had to work in 15-20 different sites to achieve this sample, with only a few children (two or three) living in most of these settlements.

Instead, I decided to widen the age-range, extending it to include children aged from three months; these children would move into the key period for growth faltering (6-12 months) during the six month intervention period. However, younger children tend to have better z-scores and lower levels of gut damage and immune stimulation than older children. Including these children in the sample would therefore presumably have affected the sample size calculation by increasing the means and standard deviations on which the calculation was based. Re-calculating the sample size prediction using Panter-Brick et al.'s (2009) data for children aged 3-12 month, resulted in the following sample size predictions:

Health Markers	Original values from 2005 study (n=23)		Expected value after intervention (30% reduction)	Required sample size
	μ_1	σ	μ_2	N
L:C	0.19	0.08	0.13	68
Albumin	35.94	3.30	25.16	4
AGP	0.64	0.12	0.45	12
IgG	7.12	1.50	4.99	16
HAZ	-0.79	0.82	-0.55	370
WAZ	-1.44	0.92	-1.01	140
WHZ	-0.78	0.74	-0.55	314

Table 6.1 Recalculation of sample size based on data from children aged 3-12 months taken from Panter-Brick et al. (2009).

For all of the biochemical variables, widening the sample age-range had no impact on the required sample size. However, it did have a significant impact on the sample size required to detect differences in growth. In the original sample size calculation, HAZ and WHZ were not included since no significant relationships between either gut damage or immune stimulation and these two growth variables had been found in the original study. For WAZ, however, a significant relationship had been found and the original calculation had determined a sample

size of 84 children would be required for this variable. By increasing the age range to include children as young as three months, the required sample size increases from 84 to 140 children. Thus, the achieved sample of 88 children was likely inadequate to detect differences in weight-for-age occurring as a result of the intervention.

There is another reason to suspect that the sample size resulted in a statistically under-powered study. The sample size calculation for this study was based on the expectation of a 30% reduction in levels of gut damage, immune stimulation and growth faltering. In doing so, this study was in line with the majority of community-level interventions that anticipate a medium effect size as a result of the intervention (Fishbein 1996). However, it is possible that this level of effect size was considerably over-optimistic. As Merzel & D'Afflitti (2003) note, reviews of the impact of numerous health promotion programmes indicate changes in outcome variables were usually less than 5% and certainly no larger than 15%. Fishbein (1996) suggests therefore that most public health interventions are woefully under-powered since they mistakenly assume community-level interventions are capable of producing effect sizes similar to those seen in medical drugs trials. By way of comparison, he notes that manufacturers who spend enormous sums of money on advertising would be extremely satisfied with an increased market share of 1-2%. In calculating sample sizes based on the expectation of a change in the range of 20-30%, he concludes that 'we often set ourselves up for failure' (1996:1075). Bearing this in mind, if the sample size for this study were recalculated with the expectation of a 15% reduction, the required sample size to detect changes in growth or gut damage soars dramatically. Thus it is possible that the intervention may have had an effect on these outcome variables, but the study simply lacked the statistical power to be able to detect it.

6.3.3 Length of intervention

It is also possible that the study failed to detect any impact from the intervention simply because it was not run long enough to be able to initiate and detect these changes. In the absence of further exposure to pathogenic organisms, the crypt cells in the epithelial lining of the small intestine could be expected to regenerate fairly quickly – within about six weeks. In discussion with my collaborator – Dr Peter Lunn (Cambridge University) – we decided that an

intervention period of six months would be sufficient to detect changes in levels of gut damage and immune stimulation. However, it is possible that six months was inadequate to detect the impact of the intervention on all outcome variables for two reasons: firstly, because of a 'biological time-lag' between the start of the intervention and its impact on the outcome variables; and secondly because of different patterns in which the intervention was taken up by the mothers.

Once hand-washing practices increased, one would expect a certain delay before observing the impact of this behavioural change at a biological level. The length of this 'biological time-lag' would not be the same for each outcome variable – certain variables would theoretically be expected to react more quickly to a reduction in pathogen exposure. For example, one would expect morbidity and levels of gut damage to be reduced first. Following this, one would anticipate a corresponding reduction in immune stimulation and thus a fall in circulating AGP levels. IgG is known to rise cumulatively with age so IgG would be expected to continue to increase but at a slower rate, reflecting the reduction in pathogen exposure. Only once these

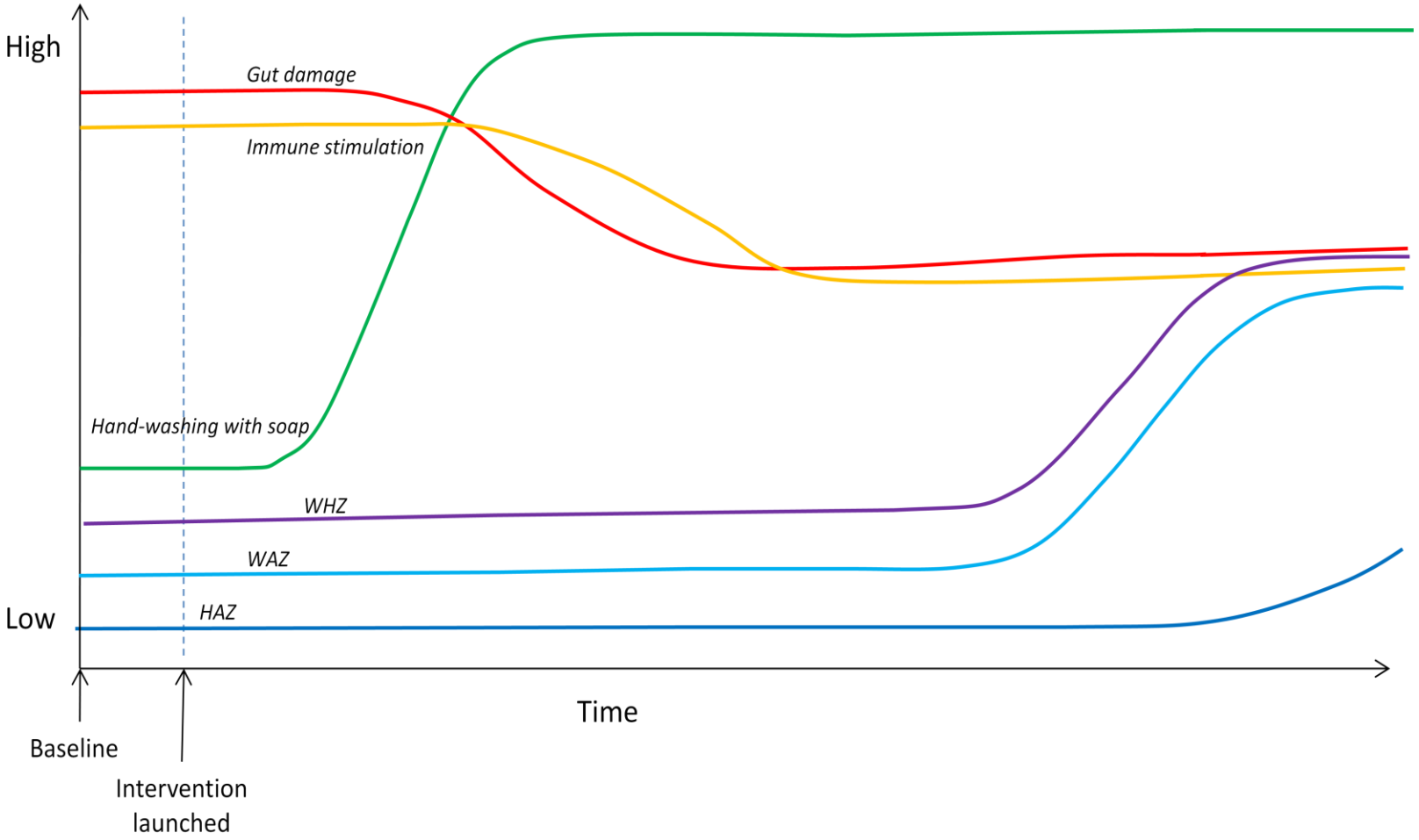


Figure 6.2 Expected time-lag between up-take of hand-washing practices and impact on the outcome variables.

changes in morbidity, gut damage and immune stimulation had been established would one expect to see any impact on growth. Since weight is more sensitive to environmental changes (being able to increase *and* decrease in response to stimuli), one would expect weight-for-height and weight-for-age z-scores to be the first to improve. Finally, height-for-age z-scores would improve in the children, but because stunting reflects longer-term changes, the impact of the intervention on height-for-age may not have been noticeable for several months. The differing biological time-lags that may have occurred in these children are depicted in Figure 6.2. It may be that this six-month intervention only covered the first half of this series of changes and needed to be run for longer in order capture the intervention's full impact on growth variables.

This situation is complicated further if we consider the ways in which behavioural change can occur, depicted in Figure 6.3 below. Here, the red line assumes that behavioural change occurred shortly after the launch of the intervention and, once established, was sustained throughout the entire intervention period as the practice became habitual. However, it is also possible that an initial increase in hand-washing occurred, followed by a gradual decrease over the intervention period as the mothers forgot about or became 'immune' to the intervention message (green line). Or perhaps there was a much longer 'run-in' to successful up-take of the intervention message; it may be that during the first few months the mothers had not fully established the new habit so it took several months before this new practice took hold and started to have an impact on outcome variables (blue line). Each of these different scenarios would have significant implications for the timing of the expected impact of the intervention and its ability to detect changes during the six month period. Because of the problems in demonstrating and documenting behavioural change, it is difficult to know for sure which scenario is most likely. However, unless the first scenario (red line) was achieved (rapid and sustained up-take) a six-month evaluation period may not have been long enough to detect real changes arising as a result of the intervention.

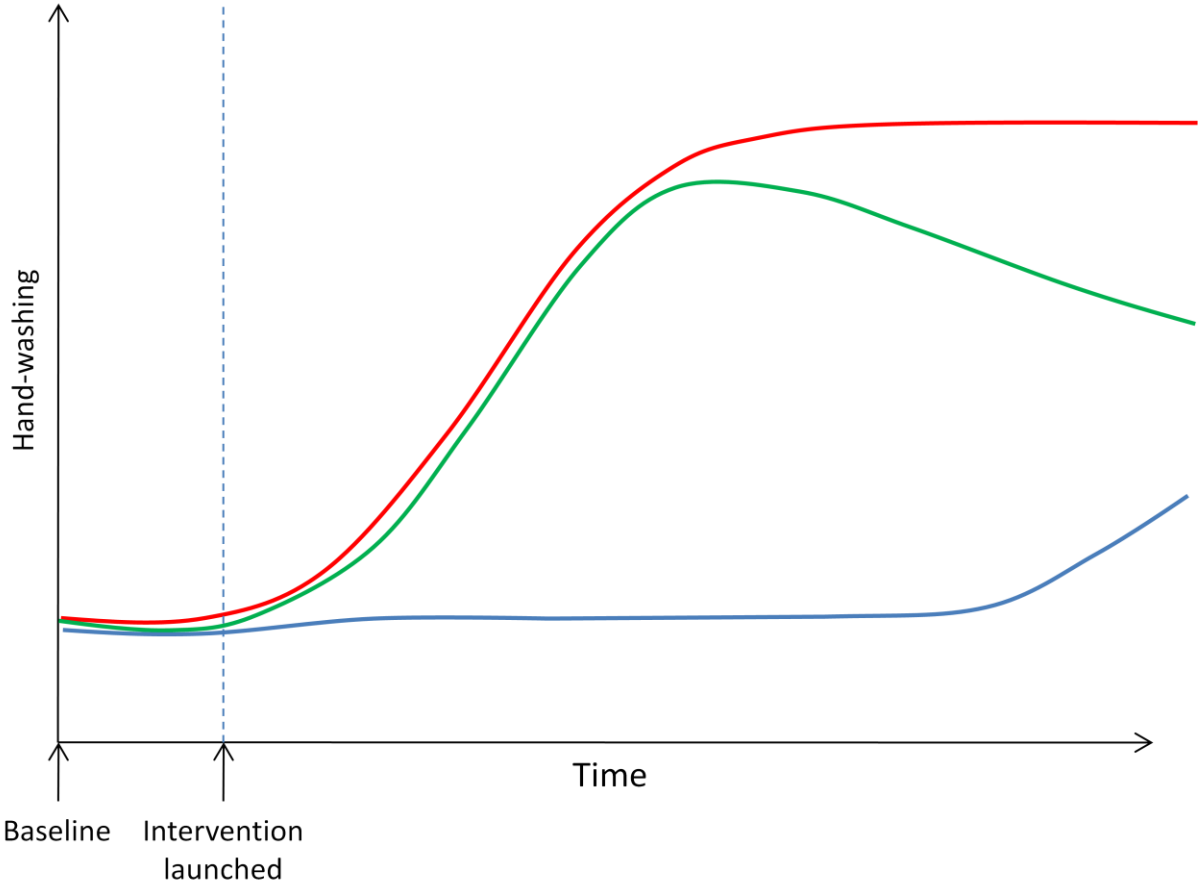


Figure 6.3 Different up-take rates of hand-washing practice: Rapid and sustained up-take (red line); rapid up-take followed by a decline (green line); and slow and gradual up-take (blue line).

6.3.4 Summary

Financial, logistical and time constraints on fieldwork meant that this study could not employ an RCT study design in order to test the hypotheses linking hand-washing, sub-clinical infection and growth. In addition, the study was limited in terms of its sample size and length of evaluation. Thus it is possible that the true effect of the intervention on outcome variables was not detected. However, it should be noted that the sample size required to detect significant changes in gut damage (L:C) and immune stimulation (AGP and IgG) were small and easily met by the achieved sample size of 88 children retained over six months for the longitudinal study. In addition, as shown in Figure 6.2, one would expect markers of gut damage and immune stimulation to react fairly quickly to changes in hygiene behaviour. Although the study was under-powered to detect changes in child growth, it should have been able to detect changes in gut damage and immune stimulation; yet no such differences were found suggesting that the hand-washing intervention failed to have an impact on these outcome variables.

6.4 Intervention failure

The final scenario to consider is that the intervention itself failed: mothers from the intervention areas increased their hand-washing practices, but though this led to a reduction in diarrhoeal morbidity, it had no effect on sub-clinical gut damage, immune stimulation or growth faltering in these children. Two possibilities could account for this result. It may be that the intervention failed on a theoretical level and there is no biological link between hand-washing and the outcome variables. Alternatively, it may be that there is a plausible biological link between hand-washing and outcome variables, but this effect was not observed because its impact was constrained by other external factors. Thus in the case of intervention failure, it is necessary to distinguish between the intervention being inefficacious versus ineffective.

Efficacy refers to the ability of an intervention to produce a positive effect when delivered under optimum conditions. An efficacious intervention therefore establishes an empirical link between the intervention and outcome variables. However, public health interventions are rarely delivered under optimal conditions. Of much greater significance to public health

specialists is the *effectiveness* of an intervention, determining whether the intervention can have a positive impact when delivered under real world conditions.

In this section, I will consider first the possibility that the intervention was inefficacious and that hand-washing does not have any effect in reducing levels of gut damage, immune stimulation and growth faltering. Secondly, I will discuss the evidence to suggest that the there may be a biological link between hand-washing and outcome variables, but that this effect was masked by the numerous other factors that could cause gut damage and immune stimulation in children living in these communities.

6.4.1 Efficacy of the intervention

This hand-washing intervention appears to have been efficacious only in terms of reducing diarrhoeal morbidity; it did not result in a reduction in levels of gut damage, immune stimulation and growth faltering in these children. Because of the issues noted above we cannot categorically state that there is no link between hand-washing and these outcomes: it is possible that the original hypothesis is correct, but that this study was not run long enough or did not have enough statistical power to detect this effect. However, there may be some reasons for thinking that hand-washing would not be an effective intervention, even under the best conditions.

As noted in Chapter 1, Esrey et al.'s (1991) review of water, sanitation and hygiene interventions aimed at reducing diarrhoeal disease found that such programmess had a greater effect on reducing the severity, rather than the frequency, of infections: reductions in overall mortality rates from diarrhoeal disease were greater than reductions in its incidence or prevalence. This suggests that though the overall rate of infection may have only been moderately reduced, the infections that did occur were less severe and resulted in fewer deaths. Following this argument, Curtis and Cairncross argue that hand-washing with soap might cause the incidence of severe infections to fall before that of mild ones (2003:278).

If this were the case, one could argue that hand-washing is unlikely to have a considerable effect on levels of gut damage and immune stimulation in young children. Much of the gut damage experienced by young children occurs at a *sub-clinical* level. The damage to the small intestine is not necessarily severe enough to produce clinically visible symptoms such as

diarrhoea, but nonetheless compromises the child's ability to digest and absorb nutrients into the body, whilst also resulting in low level (but often chronic) stimulation of the immune system. In their original study of Gambian infants, Lunn and colleagues (1991) reported that children experienced diarrhoeal symptoms 7% of the time, but were found to have high levels of intestinal permeability (i.e. gut damage) 76% of the time. Discrepancies between levels of diarrhoea and gut damage in children were also observed in this study: in any given month just over a third of children (40%) reported diarrhoeal symptoms, yet almost two-thirds of them (61%) were found to have gut damage.

Damage to the gut that occurs at a sub-clinical level is, by definition, less severe than gut damage that results in the clinically visible symptoms of diarrhoea. Thus, if hand-washing with soap is more effective in reducing incidence of severe infections, it is possible that it could result in a reduction in diarrhoeal symptoms but with little or no effect on sub-clinical gut damage in young children. Sub-clinical gut damage levels would remain high, immune stimulation would remain chronic, and growth would be unlikely to improve.

However, though Esrey et al. (1991) and Curtis and Cairncross (2003) may be right in suggesting that hand-washing with soap has a greater effect on severe infections, this does not necessarily imply it would not have *any* effect on the less severe infections that cause sub-clinical infection. Any reduction in pathogen exposure, via improvements in hand-washing practice, would presumably lead to less pathogens being ingested by the child, whether they were virulent strains that cause severe infections or less virulent strains that cause low-level, yet persistent intestinal damage. I suspect that the reason why the intervention had no effect on sub-clinical infections is not because the intervention was inefficacious (i.e. based on a flawed theory), but rather because its potential effectiveness was constrained by wider environmental factors

6.4.2 Effectiveness of the intervention

There is no doubt that hand-washing with soap is an effective way of preventing diarrhoea in young children; children from intervention areas in this study reported 41% fewer days of diarrhoeal sickness over the period of the intervention than their control counterparts – an impact similar to that predicted by Curtis and Cairncross' (2003) meta-analysis of hand-

washing interventions. However, for children living in poor slum conditions there are still numerous other ways in which they can be exposed to infectious bacteria and viruses. It is possible therefore that the intervention was ineffective because any positive impact resulting from improved hand hygiene was masked by the children's continued exposure to pathogens via other pathways.

The slum environment presents multiple opportunities for exposure to pathogenic organisms, particularly for young children who combine the least developed immune systems with a lack of learned hygiene behaviours. Faecal contamination of hands is just one way in which children can be exposed to enteric pathogens that cause intestinal damage and immune stimulation. Children are also put at risk of infectious diseases through consumption of contaminated food and water, as well as living in poor quality and over-crowded environments that promote the rapid spread of diseases through a community. Hand-washing may interrupt one particular route of transmission, but if children continued to be exposed to pathogens via a multitude of other pathways, it is unlikely that any significant reduction in gut damage and immune stimulation would occur. In the following chapter I discuss this issue in greater detail.

Summary

This chapter reviewed the possible reasons why the intervention failed to have any apparent impact on levels of gut damage, immune stimulation and growth. Multiple lines of evidence suggested that the majority of mothers in the intervention areas increased their hand-washing behaviour, and thus the lack of impact is unlikely to have been due to a failure to correctly implement the intervention programme. Various weaknesses in the study design were noted, which may have affected the study's ability to detect the true impact of the intervention on child biomarkers and growth. However, I suggest that the intervention may have been ineffective because the positive impact of hand-washing was constrained by the wider environmental conditions in which these children live. The following chapter will discuss this issue in greater detail.

CHAPTER 7

Constraints on Health and Effectiveness of Intervention

Introduction

This chapter considers the ways in which the effectiveness of the hand-washing intervention may have been constrained by the highly pathogenic environment in which the children lived. It outlines how children in the slums remain exposed to pathogenic organisms even in the presence of improvements to hand-hygiene. This is followed by a discussion of the wider socio-economic factors that affect the health and well-being of children in the slums. Using ethnographic case studies drawn from interviews with the mothers, I focus a number of factors that can insulate children against the negative effects of slum environments, or exacerbate these problems even further.

7.1 Multiple pathways of infection

As argued at the end of the previous chapter, the hand-washing intervention may have failed to reduce sub-clinical infection and growth faltering because its impact was constrained by other pathways of infection that continued to expose children to pathogenic organisms, despite improvements in hand-washing behaviour. In this section I describe some potential pathways of infection common in the slum areas, before considering their epidemiological impact on the hand-washing programme. I focus specifically on the impact of the pathogenic contamination of water, food and the general living environment.

7.1.1 Contaminated water supply

Kathmandu's water supplies are highly contaminated with coliform bacteria. This results in numerous outbreaks of diarrhoea, dysentery, typhoid and occasionally cholera (Pokhrel and

Viraraghavan 2004). Municipal water supplies are provided by the Nepal Water Supply Corporation (NWSC) and are ostensibly treated before being piped to residents' houses or public stand points (Joshi and Maharjan 2003). However, such treatment is rarely adequate to kill all the coliforms present in the water: NWSC supplies have frequently been found to be contaminated beyond the acceptable levels laid down by the WHO (Ono, Rai et al. 2001; Joshi and Maharjan 2003; Shrestha No date). The problem is exacerbated by the fact that pipelines carrying drinking water run parallel to the city's sewerage system. During the monsoon, heavy rains cause the pipes to burst leading to contamination of the drinking water supply (Ono, Rai et al. 2001; Joshi and Maharjan 2003; Moffat 2003; Shrestha No date). However, as discussed in Chapter 2, many of the city's slum communities do not have access to government water supplies and instead collect drinking water from tube wells, deep wells and stone taps. These ground water sources are often even more heavily polluted than the NWSC water supplies. Ono et al. (2001) reported that of the 57 water samples they collected in Kathmandu, 75% were found to be bacterially contaminated, over half with *Escherichia coli*. Similarly, a study sampling ground water sources in Patan (an area in the south of the city) reported that 86% were contaminated with coliforms, with 69% containing faecal coliforms (Maharjan and Sharma 1999).

Most of the mothers interviewed during this project viewed water as a potential cause of sickness and diarrhoea in their children. 'Bad' or 'dirty' water was described as being cloudy and sediment-filled, with an unpleasant taste and colour. When consumed, such water could produce sickness, diarrhoea and fevers in young children. However, even apparently 'clean' water was capable of producing sickness due to its inherent 'coldness'. As described in Chapter 3, exposure to 'cold' (*chiso)* was felt to be responsible for many of the common symptoms experienced by young children, such as diarrhoea, fevers and colds. The majority of mothers said they gave their young children boiled water ('*umaleko paani'*) to drink; however, the primary motivation for this seemed to be an attempt to mitigate the inherent 'coldness' or *chiso* of the water, rather than a means of sterilizing the water (though some mothers did cite this as an additional reason.)

The boiling of water represents an example of a positive hygiene practice that can limit children's exposure to pathogenic organisms. However, as with self-reports of hand-washing

behaviour, there is reason to believe that statements regarding this practice represented the *ideal* rather than the *actual* behaviour and thus may not have offered significant protection against pathogens. Although about three-quarters of mothers said they gave their child boiled water to drink, it became clear as the interviews progressed that this was not always the case. It was apparent that age of the child was an important factor in determining whether s/he was given boiled water; the younger the child, the more likely s/he was to received boiled water. As children got older they were encouraged to drink the untreated (but often filtered) water that the rest of the family drank. As one woman explained to me, it is important to get the child 'used to' drinking un-boiled water. If her child only ever drank boiled water he could get very sick if he was given untreated water by someone else; by gradually 'weaning' him onto untreated water she hoped to make his stomach stronger.

The general expectation therefore was that children would progress onto untreated water at an appropriate age. (It should be noted, however, that not all families followed this pattern: about a fifth of mothers reported that the entire family only ever drank boiled water). An exception to this came when the child became sick. Children suffering from colds, diarrhoea or chesty coughs were often given boiled water to aid their recovery. Such illnesses were often said to be caused by *chiso* and thus the provision of hot water was motivated by a need to combat the coldness within the child, alongside a desire to provide clean, safe water.

Even when children were given boiled water to drink it still may not have resulted in safe drinking water. I was rarely in a position to witness whether or not the mothers boiled water for their children, nor was this a behaviour targeted for observation by fieldworkers. However, I suspect that where water was treated, it was often simply heated up to near boiling point and was rarely, if ever, boiled for a significant period of time. Guidelines on the boiling of drinking water vary, however current WHO guidelines state that drinking water should be brought to a rolling boil for at least five minutes and preferably up to 20 minutes to kill all pathogens, cysts, spore and worm eggs (Kayaga 2005).

It is highly unlikely that even the most dedicated mothers conformed to these strict guidelines for treating drinking water. As Mintz et al. (1995) note, such a strategy is environmentally and economically unsustainable: it takes a kilogram of firewood to bring a litre of water to boil for one minute and people require a minimum of two litres of drinking water per person per day.

In addition, as noted in Chapter 1, boiling all drinking water would put significant financial strain on a family (Gilman and Skillicorn 1985). At the time of this study, a prolonged strike in the Terai region had led to fuel shortages and considerable hikes in the price of kerosene and food. Boiling water for a sufficient amount of time to kill all coliforms is likely to have been seen as a 'waste' of fuel and thus, even where children were given boiled water, it may still have been bacterially contaminated.

A small minority of families (5%) used the SODIS method to treat their drinking water, whereby water is left in clear plastic or glass bottles in direct sunlight for several hours: UV-A rays and temperature increase in the water act to destroy any pathogenic micro-organisms (EAWAG 2009). However, this method was generally unpopular with families, either because they disliked the taste of the water or because bottles left on roofs to disinfect were often stolen.

7.1.2 Contaminated food

Bacterially contaminated food also poses a significant threat to child health. Promoting hand-washing with soap before preparing food or feeding a child can act as an effective means of preventing faecal contamination. However, food can easily become contaminated by other routes. As mentioned above, the bacteriological quality of the water in Kathmandu is very poor, yet raw fruits and vegetables were often washed in untreated water and given to children to eat. Similarly, though most food consumed is cooked, untreated water may be added to it after cooking, thus re-contaminating it.

The majority of women said that they cooked fresh *dal bhaat* in both the morning and the evening. Since this food was freshly cooked and immediately consumed the potential for contamination is limited. However, for the poorest families in the slums, this was often not an option, as one woman explained to me:

> *Freshly cooked food is best but I can't do this every day. I can't prepare fresh food every time because it is too expensive. The food is expensive but also the fuel is very expensive and we can't afford to keep using it for cooking each time. One litre of kerosene lasts for only about two days and it's so expensive nowadays. Usually I*

cook in the morning and my son eats then and goes to school. Then at five o'clock he comes back and if there is enough fuel I'll cook again but if not he has food left over from the morning. It's not good, not good for health to eat leftovers.

Purna Tamang, mother of Sona Tamang

For women in the poorest families, cooking only once a day was a financial necessity. However, the bacterial load in food that has been left out in high temperatures all day can be extraordinarily high; insufficient reheating can further exacerbate the problem (Motaryemi, Kaferstein et al. 1993). Although the mothers knew that this strategy potentially put their family at risk of disease, their choice in this matter was significantly constrained by the poverty in which they lived. Thus children from the poorest families were those who were most frequently exposed to risky, yet unavoidable, practices. Not surprisingly, children from the poorest families were also the ones with the poorest growth.

7.1.3 Contaminated environment

The general living environment of the slums also poses significant threats to child health. Firstly, the children often live in cramped, dark and damp houses. Over half (57%) of families in this study lived in just one room that served as kitchen, bedroom and living area for up to eight people. Wood-burning stoves are used daily for cooking by the poorest families in the community. These houses do not have chimneys and are poorly ventilated, exposing children to high levels of bio-fuel smoke which have been linked to increased risk of respiratory infections in both Nepal and elsewhere (Pandey, Smith et al. 1989; Smith, Samet et al. 2000). Risk of respiratory infections is also increased by damp living conditions that encourage the proliferation of spores and moulds (Peat and Dickerson 1998). In addition, high levels of overcrowding mean that infectious diseases can spread rapidly throughout the entire community, often disproportionately affecting the young.

Sanitation problems in the slums also mean that many of these children are growing up in an environment that is highly faecally contaminated. As discussed in Chapter 1, hand-washing with soap is an effective way of interrupting the faecal-oral route, once the environment has been contaminated. However, a far more effective intervention would be one that prevents

faecal matter from contaminating the environment in the first place. Through the action of NGOs and the local communities themselves, sanitation in the slum settlements in this study have improved greatly over the past few years; only one household I spoke to said they had no access to a toilet facility and had to defecate on the riverbanks[23]. However, though this represents a significant improvement in sanitary conditions, faecal matter was still commonly seen on the ground in the poorest areas of the slums. Several of the mothers commented that the area in which they lived was very dirty because people allowed their children to defecate on the ground and never cleaned it up. As one woman explained,

> *Most of the kids around here just crap on the floor and their mothers never bother to clean it up. It's usually the little kids that do this – the ones about four to seven years old. They don't like using the toilets, you see? They prefer to just go on the ground. Once they're about ten or eleven they start to use the toilets because they become shy and so don't want to go in public, in front of everyone. Some parents teach their children from a young age to use the toilets, but not very often. Most people don't bother. I've taught my children this – they all use the toilet, they have that habit now. But if I see a little kid crap on the floor I go and get their mother and tell them to clear it up…. The trouble is that often the kids get up before the parents are awake and so they don't see them do it and so they don't know to clean it up. But then the child keeps on doing it, because they aren't taught otherwise. And even if they are taught, they see other kids crapping on the floor and get taught that instead.*
>
> Purna Tamang, mother of Sona Tamang

As I spent time in the slums I frequently saw faeces on the ground and occasionally saw young children defecating on the floor. More commonly, however, I observed animal faeces on the ground. Large numbers of dogs roamed about the settlements and I never saw anyone attempt to dispose of their faeces from the pathways and communal areas. In addition, some families kept chickens, ducks, pigs and cows and their faeces were often observed on the ground where children frequently played.

---

[23] This family subsequently moved back to their natal village and so was excluded from the statistical analyses.

Once faecal matter enters the environment, the pathogens it contains are easily spread to other surfaces and people. It is possible therefore that in the face of persistent and significant faecal contamination of the environment, hand-washing may have done little to interrupt disease transmission. Young children learn by actively exploring and engaging with their environment. At this young age, the mouth is the most sensitive part of their body and thus every new object is investigated by putting it into their mouth. Whilst part of the natural learning process, in a highly contaminated environment such behaviour represents a very significant pathway of exposure to faecal matter and pathogenic organisms.

The mothers were aware of the threat that the unsanitary environment in which they lived posed to their children but felt powerless to do anything about it. As one woman explained:

> *To be a good mother you must be clean, you must not give dirty food or let your children play with dirty things. But here that's just impossible. I try to keep my children clean but they just go straight outside and get dirty again because it's dirty out there. What's the point? It's impossible.*
>
> Sarmila Pariyar, mother of Sujal Pariyar

This sense of pointlessness was echoed by another woman:

> *This place is so dirty. If they cleared it up it would be much better, it wouldn't smell as bad. But that will only work if everyone does this, if everyone helps to keep it clean. If I tried, it would just be pointless. Nobody will work together in this area. One person tries to clean it up and then someone else comes along and just messes it all up again.*
>
> Purna Tamang, mother of Sona Tamang

7.1.4 Implications of multiple pathways for intervention effectiveness

Recognition of these multiple pathways of transmission may be very important for interpreting the results from this current intervention study. As set out in Chapter 1, the F-diagram (Figure 1.8) is a useful pictorial representation of the multiple pathways by which enteric pathogens in faecal material can pass through the environment and enter a new host. Children are clearly

often exposed to several pathways of transmission at once, and yet rarely has there been any specific examination of the way in which these different pathways interact and modulate each other's effects (VanDerslice and Briscoe 1995). Whilst acknowledging these multiple routes of transmission, there has been a tendency among researchers to examine the effects of these routes *individually* and in turn, rather than attempting to model the dynamic and potentially synergistic interaction between them (Eisenberg, Scott et al. 2007).

Because of the existence of multiple pathways of transmission and their interaction, it is possible that a potentially very effective intervention (like hand-washing) may fail to achieve its expected impact. Indeed, in such a context, even tackling the dominant route of transmission – the one that accounts for the greatest proportion of pathogens transferred to the host – may be ineffective. Two very informative theoretical papers by Briscoe (1984; 1987) explain why this is the case. Consider the following example, outlined in Figure 7.1.

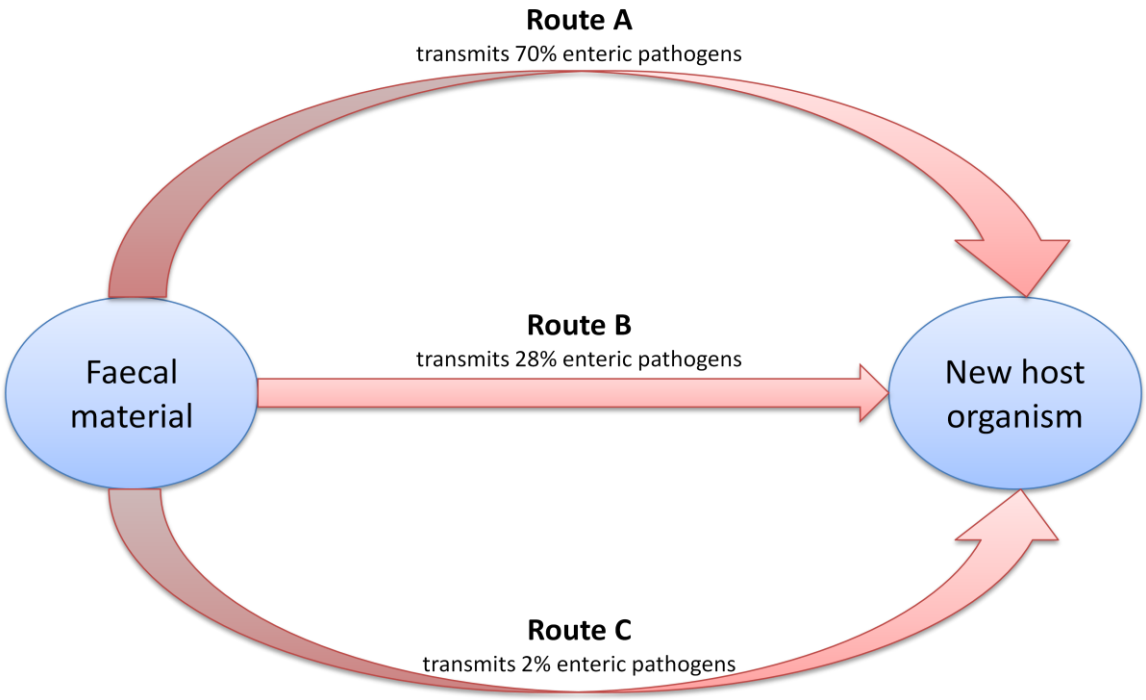


Figure 7.1 Routes of transmission of pathogenic organisms to a new host. Adapted from Briscoe (1987).

In this hypothetical example there are three pathways through which enteric pathogens can be transmitted from faecal material into a new host: Route A represents the dominant route of

transmission, accounting for 70% of the enteric pathogens potentially transmitted to the host; Routes B and C account for just 28% and 2%, respectively. In all three types of transmission, the most common form of dose-response relationship (log-linear) between exposure and resultant disease in the host is assumed (Briscoe 1984:452).

Let us now consider the effect of different types of interventions that tackle either single or combined routes of transmission (outlined in Table 7.1). The first group is our control group: no intervention has been implemented and all three routes of transmission remain active. Correspondingly there is no reduction in the number of organisms transmitted, or the number of cases of diarrhoea incurred.

	Intervention	Remaining exposure routes	Proportion of original no. of organisms still transmitted	Proportion of original no. of cases of disease still incurred[24]
1	No intervention	A + B + C	100	100
2	Eliminate Route A only	B + C	30	74
3	Eliminate Route B only	A + C	72	93
4	Eliminate Routes A and B	C	2	15

Table 7.1 An example demonstrating the effect of eliminating different transmission routes on disease incidence. Taken from Briscoe (1987:100).

In the second group, an intervention is implemented that eliminates the dominant route of transmission – Route A – whilst Routes B and C are left unaffected. In this case, 70% of the pathogens are prevented from reaching the host population, but 30% of pathogens are still transmitted via Routes B and C. Because of the log-linear dose-response relationship between exposure and resultant disease, eliminating Route A reduces disease incidence only by about a quarter – a far smaller proportion than one might have expected given its apparent importance in the transmission of pathogens to the new host. In the third group, Route B is eliminated while Routes A and C remain unaffected. In this case, 72% of the original number of organisms are still transmitted and results in only a 7% reduction in disease incidence. Finally, both Routes A *and* B are eliminated by an intervention programme, preventing the transmission of all but 2% of the faecal pathogens to the host population via Route C. As a

[24] For this example, probability of infection = 0.5 log10 (dose) (Briscoe 1984:449).

result, disease incidence drops dramatically to just 15% of the original proportion of expected cases.

Two important conclusions can be drawn from this hypothetical example. Firstly, it demonstrates that the effectiveness of an intervention (even one that eliminates the dominant route of transmission) can be significantly compromised by the environment in which it is implemented and the number of other transmission routes present in that environment. And secondly, that the combined effect of an intervention that tackles multiple, rather than single, routes of transmission, is far more effective than the effect seen from eliminating either route individually. As Briscoe points out, 'the importance of eliminating Route A is not [its] modest direct effect, but rather the fact that its elimination creates conditions that allow subsequent interventions to be much more effective' (Briscoe 1987:99).

Briscoe's argument has been backed up by a more recent study that used household-level stochastic models to investigate the interdependence of transmission pathways of enteric pathogens (Eisenberg, Scott et al. 2007). The authors found the potential efficacy of an intervention designed to improve drinking water quality depends very significantly on the pre-existing sanitation and hygiene conditions of the community in which it is implemented. Their models suggested that where community sanitary conditions are poor, improving quality of drinking water is likely to have minimal public health benefit in terms of reducing diarrhoeal disease. Where sanitary conditions are good, however, improving drinking water quality has the potential to effect a significant reduction in disease incidence. As they explain, 'under conditions in which each pathway alone is sufficient to maintain disease at high levels…single-pathway interventions will have minimal benefits and ultimately an intervention will be successful only if all sufficient pathways are eliminated' (Eisenberg, Scott et al. 2007:851). Their theoretical models therefore provide a compelling explanation for the highly variable results that have been observed from empirical water quality interventions: the efficacy of such an intervention is likely to depend greatly on the presence or absence of previous interventions to improve community sanitation, drainage and hygiene behaviours.

Empirical evidence for this modulating effect of community sanitation on other interventions is provided by Vanderslice and Briscoe's (1995) study on the Filipino island of Cebu. This study collected data from a random sample of over two thousand Filipino infants over their

first year of life. Forty-one homogenous neighbourhoods were identified from the 17 administrative districts surveyed and each one was assessed for levels of community hygiene by a sanitary engineer. Neighbourhoods with dense housing, poor drainage and readily observable faecal material were classified as having poor community sanitation (*ibid*:137).

In a main effects model that does not allow for any interaction between transmission pathways, the prevalence of diarrhoea was found to be significantly greater where: drinking water was contaminated; households did not have private excreta disposal facilities; excreta was observed in the yard; and where community sanitation was very poor. Subsequent models that allowed for interactions between environmental variables, however, revealed a more complex picture of exposure. A significant negative interaction was observed between drinking water quality and community sanitation, implying that the impact of improving water quality was modulated by the pre-existing level of hygiene and sanitation in the community. For children living in highly contaminated environments, quality of drinking water had very little effect on the risk of diarrhoea since children continued to be exposed to enteric pathogen through a variety of other routes. In areas with better community sanitation however, the bacteriological quality of water was very strongly associated with childhood diarrhoea, since the other potential routes of transmission had already been addressed through improved sanitation. Clearly the type and effectiveness of an intervention depends very much on the environmental context in which it is implemented.

What are the implications of these theoretical models and empirical studies for the results of this current hand-washing intervention? If the effectiveness of drinking water quality is modulated by environmental contamination, could a similar process be at work regarding this hand hygiene intervention? It may be that hand-washing was capable of having an effect on the more severe type of gut damage that results in diarrhoeal disease, but failed to impact on sub-clinical forms of gut damage which may be more subtly affected by other routes of transmission. In other words, hand-washing may be necessary *but not sufficient* to effect a reduction in sub-clinical infection in the context of Kathmandu's slums.

7.2 Nutrition and growth

As the above discussion demonstrates, the pathogenic nature of the slum environment is a major cause of ill-health and growth faltering in young children. Children are continually exposed to infectious organisms through a number of different pathways, many of which remain unaffected by improvements in hand-hygiene. The continued existence of these other pathways of infection may therefore explain why the hand-washing campaign failed to reduce sub-clinical infections and improve growth status in these children.

However, exposure to infectious disease is only one of the main causes of childhood growth faltering. As noted in Chapter 1, under-nutrition – in terms of both food quantity and quality – is another major cause of childhood growth faltering. An inadequate intake of calories and/or protein in a child results in weight loss and, under chronic conditions, a slowing or even cessation of linear growth. Similarly, the nutritional quality of the diet – in terms of micronutrients such as vitamin A, iron and zinc – is also important in maintaining proper growth. Both macro- and micro-nutrient deficiencies can lead to a weakening of the child's immune system and thus the impact of being exposed to infection on a child's health is modulated by his/her pre-existing nutritional state: a well-nourished child is better able to fight off infections.

Because of time and resource constraints during the study, I could not measure children's nutritional intake in order to determine its importance in causing growth faltering. However, since nutrition is such an important factor in determining health and growth status, I did question the mothers about their children's diet and feeding practices. Some of the important issues to come out of these conversations are discussed below.

Compromised nutritional status can start even before birth; birth weights are consistently related to maternal nutritional status and BMI (Neggers, Goldenberg et al. 1995). The mothers I interviewed were well aware of this and often explained their child's perceived small size and lack of growth in terms of their own nutritional status during pregnancy. As one woman commented,

> *I didn't eat much during my pregnancy as I still had to work so hard all day* [as a labourer]. *I couldn't take good care of myself. Whatever you eat during pregnancy*

makes the baby big or small. I didn't eat much so he was very small when he was born.

Kamala Rai, mother of Anamol Rai

And another woman explained,

[My daughter] *was so small when she was born. I think it is because I didn't eat nutritious foods when I was pregnant with her, like meat and beans. I didn't know I was pregnant for several months and so I was still working and was too busy to cook properly. We didn't have enough money for good food at that time either. That's why she's so small.*

Gita Karki, mother of Reshma Karki

The problems of nutrition in pregnancy were exacerbated by having to work hard, not having enough money for food, and also by the relative position of the woman in the household. One woman explained that her first child was much smaller than her second because of the difficult family circumstances she was living in at the time.

*During my first pregnancy I didn't get much to eat and so my eldest child was much small*er [than my second child] *when she was born. We were living with my Sasura* [father-in-law] *at the time and my husband had to give all his money to him so I didn't have any money of my own to spend on good, healthy, nutritious food. My Sasu* [mother-in-law] *was dead so I was the only woman in the household with my husband, my Sasura and my husband's brother. They didn't give me any special attention while I was pregnant and I wasn't given any good food to eat*.

Chhina Tamang, mother of Phulmaya Tamang

She went on to explain that shortly after her first daughter was born, she and her husband moved into their own rented house and she therefore had much more control over her diet and care during her second pregnancy.

The quantity and nutritional quality of the child's diet was also seen as very important in determining a child's health and growth. The majority of mothers interviewed felt that

generally their family had enough food to eat; most of the women explained that though they struggled on their income they always had enough money to be able to provide a nutritionally adequate (if very simple) diet for their family. However, for some of the poorest families – with the poorest growing children – this was not the case.

Take, for example, the case of 23-year-old Nim Dolma Sherpa, mother of Sonam Sherpa. She fell pregnant with Sonam just before her husband left the country to work as a carpenter in Malaysia. At the time of our interview Sonam was 13 months-old and was severely stunted and under-weight. Usually, families receiving remittances from abroad tended to have higher-than-average household incomes. However, as we sat in her tiny, cramped, dark room Nim Dolma explained to me that her husband had sent almost no money home since he left Nepal as he had been unable to find work. In fact, she has not received any money from him in almost a year. At first she survived on the little money he had sent at the start and from hand-outs from his family, but a few months ago she decided to take up a cleaning job which gave her just 1000Rs per month to provide for her and her daughter.

> *We haven't had enough money and it's been so difficult for us. I don't think I've been able to give Sonam enough food to eat. It's getting a bit easier now with my job but before it was so difficult. We had no money and so we could only afford to eat jaulo* [simple mixture of rice and lentils]. *Whenever I had some money I would make proper dal bhaat tarkari* [staple dish of rice, lentils and vegetables]. *But even now we still don't have enough money to survive… I try to give her meat, eggs, beans – nutritious food to help her grow – but I can't afford to do this much. I don't think Sonam gets enough food to eat compared to other children here. Sometimes she cries because she is so hungry… Before I got this job I used to eat less or skip meals altogether in order to give her enough food.*
>
> Nim Dolma Sherpa, mother of Sonam Sherpa

Cases such as Nim Dolma's were fairly exceptional; the majority of women I interviewed felt that their family usually had enough food to eat. Of more pressing concern for these women was the nutritional quality and diversity of the diet. Meat, eggs, yoghurt, fruit, green leafy vegetables (*sag*) and lentils (*dal*) were all mentioned by mothers as highly nutritious foods that

help a child become healthy and strong. However, with the exception of *sag*, these foods were also the most expensive. Access to these nutritious foods was therefore limited and children in the poorest families rarely, if ever, ate any animal protein.

What became apparent during these interviews was the fragility of the women's situations and their vulnerability to social and economic 'shocks'; with few resources (material or social) to buffer them against these shocks it would not have taken much to quickly push them into a situation where access to food was a pressing issue. Since many of these families were exclusively reliant on their husband's income and had little or no savings to fall back on, the sudden incapacitation of the main wage earner could rapidly push a family into extreme poverty and significant nutritional stress. Indeed, in all of the interviews where mothers expressed concerns about having enough food to eat, the husband was currently out of work.

An interview with Ranju Basnet, a mother from one of the intervention sites brought this fragility and vulnerability home to me. Like most of the men in this area, her husband worked as a labourer for a construction company. However, recently he had fallen sick, was unable to work and had been told he needed an operation on his stomach. Since this happened they had been living off the money that was owed to him from previous work he had done, but this money was rapidly running out. Unlike other families, however, Ranju had some gold jewellery that she was given at her wedding that she could pawn to tide them over until her husband recovered.

7.3 Vulnerability and resilience in the slums

Clearly, the slum settlements of Kathmandu offer a far from healthy environment in which to raise children: contaminated environments lead to frequent infections and poor quality diets put children at risk of malnutrition. However, it is also true that there is significant variation within the slum populations with regard to child health and growth status. Some children in this study experienced a high frequency of colds, fevers and episodes of diarrhoea, whilst other children reported very few symptoms. Whilst most children exhibited gut damage (as defined in Chapter 2) the majority of the time, about a sixth of children (17%) experienced gut damage only once or not at all. Similarly, the variation in levels of blood biomarkers was very

large, ranging from a two-fold difference between the highest and lowest values for haemoglobin, to a seven-fold difference for AGP.

Growth was also highly variable. About one-third of children in the current study were of normal height-for-age (>-1 z-scores), yet a quarter were moderately-to-severely stunted (<-2 z-scores). Weight-for-age varied dramatically between the children, whose WAZ scores ranged from -5.39 to 2.04 z-scores; overall, about a fifth of children were of normal weight-for-age, while almost a third were moderately-to-severely underweight. Though the majority of children (69%) were not wasted, there was still a large range of weight-for-height z-scores ranging from one child who was severely wasted (WHZ = -3.52) to another who was technically over-weight (WHZ = 2.38).

Whilst all of these children lived in slum conditions, it appeared that some families were better than others at mitigating the negative impact of the slum environment on their child's health and growth. Clearly, not all children living in the slums were equally at risk of infectious diseases or malnutrition – some families were more vulnerable to these problems, whilst others proved more resilient. I believe this variation in exposure may have been largely a function of the households' relative levels of poverty.

Though slum communities are generally characterised by poverty, it is important to note that they are not homogenous; significant variation in household socio-economic status exists even within these deprived communities. During my fieldwork in the slums, I became very aware of the heterogeneity of circumstances in which these families were living. It was easy to pick out both the poorest and the wealthiest (relatively speaking) households in the slums. These variations in socio-economic status – resulting in differences in resources, attitudes and behaviours – may account for some of the differences in health and growth status observed between children.

Working at a number of different levels, poverty can have a hugely detrimental impact on a child's health and growth. Children living in the poorest households are likely to have poorer access to high-quality, nutritious foods and be at much greater risk of exposure to pathogens than children living in better social and economic conditions. Any disease resulting from this exposure is also likely to have more severe consequences in poorer children because of, for

example, pre-existing differences in nutritional and immunological status or their access to health care facilities. However, poverty also constrains human agency such that these pre-existing problems are further exacerbated, for example, by preventing mothers from adopting health-promoting behaviours and practices.

In the following section I wish to address the impact of poverty on health, focusing on a number of risk and protective factors identified during interviews with the mothers. In doing so, I draw upon case studies of particular women in order to illustrate the ways in which poverty and disadvantage constrain mothers' behaviours and choices, often resulting in their children being at greater risk of infection or malnutrition.

7.3.1 Risk factors in the slums

Working mothers

Perhaps one of the most important detrimental consequences of poverty for child health was the need for mothers to start working again when their child was still very young. At the baseline survey conducted in May 2007, only about a sixth of mothers were engaged in any form of paid employment, though this proportion had increased by the end of the study. Because I did not have comprehensive data on the type of work each mother was engaged in, plus the fact that most women were not currently employed, it was not possible to conduct any statistical analysis regarding the relationship between maternal work and child health and growth. However, from interviews conducted with the mothers it became clear that there were numerous implications for child health when a mother had to return to work early.

The decision to return to work involves an important trade-off between two competing priorities: the need to generate extra income versus the reduction in time available for maternal care. For about a third of the mothers who worked, this conflict was less significant: they were engaged in activities such as weaving, sewing or shop-keeping – all of which were done from their own homes. It was therefore possible for them to look after their children whilst carrying out these economic activities, although the quality and quantity of care may still have been affected (Leslie 1989) . However, the majority of working mothers were engaged in labouring

or cleaning which required the mother to be away from home for extended periods during the day.

A few studies have found a positive effect of maternal work on children's health and nutritional status (Leslie 1989; Vial, Muchnik et al. 1989; Brown, Yohannes et al. 1994; Lamontagne, Engle et al. 1998), possibly because increased household income results in improvements in the nutritional quality and quantity of the child's diet. However, this beneficial effect may only be seen when adequate childcare arrangements are in place to compensate for the reduction in maternal attention. In families where there is no suitable alternative care-giver, mothers must either take their child to work with them, or leave the child at home (either unsupervised or in the care of an older sibling). Both of these practices have been identified as significant risk factors for poor child health (Engle 1991; Hernandez, Zettna et al. 1996; Lamontagne, Engle et al. 1998).

All of the mothers engaged in work outside the home that I interviewed stressed that they only did so out of financial necessity: in some cases the woman's spouse was currently unemployed leaving her wage as the only form of household income; in others, her husband's wage was simply too low to support the entire family. However, few of these women had relatives who were able to care for the child whilst they worked. Thus they were often forced to engage in practices that put their children at risk: either taking the child to work or leaving him/her at home without adequate supervision.

Take, for example, the case of Gita Karki – mother of 13 month-old Reshma. In our interview, she explained to me that her husband's income of 2000Rs per month was simply not enough to support them and their three children and so she had returned to work when Reshma was still quite young. Whilst her older children were enrolled at a local school, Gita had no relatives in the area that she could leave Reshma with while she worked; she was therefore forced to take Reshma with her, working all day with her daughter strapped to her back. Gita worked for about seven hours a day as a sweeper at the local bus station in Balaju. She described it as unpleasant work since she breathed in dust and traffic fumes from the buses all day long; she was also concerned about the effect that this was having on Reshma's health. She described how one day some *'bideshis'* (foreigners, presumably working for an NGO or international aid

agency) saw her working with Reshma on her back and told her that it was very bad for Reshma's health to be exposed to such air pollution. Gita's response to this advice:

What can I do though? I have to work.

Gita's frustration highlights the importance of poverty in constraining human agency with regard to health-promoting or health-protecting behaviour. Clearly, Gita was well aware this was not an ideal situation for either herself or her child. But for her family, survival was about balancing numerous risks; which was more pressing – protecting her daughter from breathing in dust or being able to provide her with food to eat that evening?

Bishnu-Maya Gurung – mother of nine month-old Pratik – was another woman who intermittently took up labouring work to supplement her husband's income. She explained that whenever she had to do this, it was very hard to ensure that her child was fed properly. Starting work early, she did not have time to cook a proper meal of *dal bhaat* and often they would eat nothing at all in the mornings. Taking Pratik to work with her, she would feed him some biscuits or other dry foods during the day, but it would not be until the evening that she would be able to cook a proper meal of rice and vegetables. She was aware that having just one proper meal a day was not enough for her son, but there were times when it was a financial necessity that she go and earn some money.

Alcoholism

Not all of the mothers who worked took their child with them. In some cases they were able to leave the child with relatives who would take care of him/her during the day. However, this did not necessarily mean that the child was any better off, as highlighted by the case of Bhumika Limbu and her daughter Sujata.

Of all the children enrolled in this study, Sujata was by far the child I was most concerned about. At the start of the study she was almost a year old and yet weighed little over five kilograms and measured just 62.5 cm. She was severely stunted and underweight (-3.5 and -5.52 for HAZ and WAZ, respectively) and her growth trajectory did not improve at all over the period of the intervention. Interviewing her mother, towards the end of the study, I started to build up a picture of why Sujata's health and growth had been so compromised.

Sujata was a premature baby and was delivered at home by her father as Bhumika felt too scared to go to the hospital. Bhumika could not tell me exactly how many weeks premature she was, but recalls that she was very small when she was born. Despite this, she did not take her to be seen by a doctor once she was born as '*she seemed to be fine'*. Just three weeks after giving birth, Bhumika returned to her job working as a labourer. I was surprised at first as to why she returned to work so quickly, but Bhumika told me that she simply had no choice; Bhumika's husband was an alcoholic and provided very little money for Bhumika and her two children. She was therefore the main wage-earner in their family. As she explained,

> *Sometimes he will get a few days of work doing labouring, but then he usually goes and spends most of the money he earns on drink. His mother lives nearby and she has a shop where she makes and sells rakshi* [distilled rice spirit] *and he just goes and spends all day there getting drunk…. Sometimes he will work about 15 days a month, but at least half of what he gets paid he spends on alcohol.*

Alcoholism (in both men and women) is not uncommon in the slum communities and it was often cited by the mothers as one of the worst problems of living in the slums. It is, of course, both a symptom *and* a significant cause of poverty. As one man remarked to me,

> *Lots of people here drink alcohol, too much alcohol. It's a big problem…These families, they spend their money on alcohol rather than food for their own children.*

In Bhumika's case, her husband's drinking certainly had a very detrimental impact her daughter's nutrition and growth (as well as affecting her own health and well-being in terms of being subject to high levels of stress and domestic violence). As she explained,

> *I went back to work about 20 days after she was born. I couldn't take such a young baby with me when I was doing this physical work so I left her with my Sasu* [mother-in-law] *during the day. I was breastfeeding her in the mornings, evenings and during my lunch break, but obviously that wasn't enough. It wasn't enough food for her so I told my Sasu to give her some rice mashed up with some milk while I was working… I know you shouldn't give such young children food so early. The doctor told me I should give food much later, but what could I do? I have to*

work. That's the reason why she's so small – she didn't get enough milk from me when she was born and so she's never grown properly.

For Bhumika therefore, poverty forced her into actions and behaviours that certainly would have had a negative impact on her daughter's health by limiting her access to breast milk and exposing her to pathogenic organisms in weaning foods at a very early age. Yet, as she rightly points out, her choices in this situation were severely constrained.

Poverty and disadvantage

The powerful ways in which poverty and disadvantage constrain behaviour and agency are exemplified by the case of Sarmila Pariyar, a 25-year old *dalit* (low-caste) mother living in one of the intervention areas. Sarmila and her husband had four children (aged eight, five and three years, and 13-months) and lived in a small, single room on the banks of a highly polluted river. Sarmila's youngest son – Sujal – was enrolled in the study and at baseline was moderately under-weight but severely stunted. Sujal's family was one of the poorest in this study – a fact that was abundantly obvious to me as we started our interview and which I commented on in my field notes:

We sit outside in the bright sunshine for this interview. Sarmila looks unkempt in old, worn clothes – such a contrast from most of the other mothers who always manage to look so clean and tidy despite the squalor they live in. Her husband is asleep inside and her children play outside with us. Sujal sits in his mother's lap. Sarmila's older children – a little girl aged five and a boy aged three – are absolutely filthy. The little girl is covered in dust and mud all over her arms, legs and face. The little boy wears only a t-shirt and has a nasty cut on his face from when he fell the other day. He too is absolutely caked in mud. Both the children's fingernails are black. They constantly fight with each other and often one child or the other is crying loudly. Sarmila usually reacts to this by shouting and hitting them even more. The little three year-old child comes running over to us, falls and cracks his head on a brick quite badly. Instead of comforting him, Sarmila, obviously exasperated, shouts at him and hits him which makes him cry even more….. It is clear that this is a really poor family and they are not doing well at all.

Field notes dated 04.12.07

Sarmila told us that her husband was diagnosed with epilepsy about six months ago and could no longer work, though he apparently stopped working long before this. Though Sarmila never told us directly, we gathered from conversations with the local Community Motivator and Sarmila's neighbours that, in fact, her husband is an alcoholic and this is the real reason why he no longer works. The family is therefore entirely dependent on the small wage Sarmila earns washing clothes and dishes for richer families in houses nearby. She works for a few hours in both the morning and afternoon and gets paid just 2000Rs per month. She admits financially it is extremely hard for them and they often don't get enough food to eat:

> *When I'm working it's so hard because I don't have the time to make sure they're fed properly. I give them dal bhaat in the evening when I get home from working… Meat is good for you, but my children only get this about once a month. It's so expensive, we can't afford it. I think maybe they often go hungry. I'm usually OK because I get to have tea and khaajaa* [snacks] *at the houses I work at, but I worry that they don't get enough to eat.*

When she first moved to this area she had just two children and her husband was still working. At that time her mother-in-law was also living with them, and she was able to look after the children if Sarmila needed to work. However, her mother-in-law died two years ago and since her husband stopped working the family has fallen further and further into poverty. She was forced to go back to work just 15 days after giving birth to her youngest child, returning throughout the day to breastfeed him whenever possible. None of her older children went to school as they could not afford the school fees; while she worked they were ostensibly left in the care of their father. However, Sarmila admitted that he spent much of the day asleep and mostly the children took care of themselves. Sujal was usually left on a rug outside the house where a neighbour or his older siblings kept an eye on him.

Given the difficult circumstances in this household, Sujal was especially vulnerable to malnutrition and infectious disease. Sujal's access to the nutritional and immunological benefits of his mother's breast milk was curtailed because of his mother's need to work. As he got older, Sarmila would leave food for her children while she worked, relying on her five-

year-old daughter to feed Sujal whenever he cried. However, it is increasingly recognised that caregiver-child interactions during feeding can be extremely important in determining nutrient intake (Ruel, Brown et al. 2003). Young children often need encouragement to eat and it has been suggested that behaviours such as 'physically helping the young child to eat, verbal encouragement and prompting, role playing, persistence and patience, offering additional spoonfuls or bites, monitoring of child cues of appetite and satiation, and a variety of other strategies' are important in ensuring the child consumes a nutritionally adequate diet (*ibid*: 22). It seems unlikely that a five-year-old would be capable of taking enough care to ensure her younger sibling ate enough food during the day and thus Sujal's dietary intake was probably inadequate, even when there was food was available for him to eat.

In addition, without proper adult supervision, Sujal was almost certainly at much greater risk of being exposed to pathogenic material or physical injury. Again, his older siblings were unlikely to understand the importance of hygiene (particularly with regard to hand-washing), nor would they necessarily have been able to identify risks to safety and protect him from these. As a result, Sujal experienced a higher-than-average level of gut damage over the study period, exhibiting significant damage on five of the seven months of study. Indeed, as far as I am aware, Sujal was the only child who suffered from a severe illness during the course of this study: a wound on his head became infected resulting in a very high fever and massive swelling on the left side of his skull; consequently, he was hospitalised for ten days while the pus was drained from his head and the infection treated by intravenous antibiotics.

Sarmila was also one of the mothers that the local Community Motivator identified as not being receptive to the hand-washing message. In our fortnightly team meetings, the Community Motivator for Sarmila's area said that she felt Sarmila was uninterested in the programme and it did not seem to be making any difference to her behaviour at all. Given the particularly difficult circumstances in which she lived it is understandable that hand-washing with soap was not her first priority. In circumstances of significant poverty, survival is about balancing out risks; the potential threat of sickness in her child at some point in the future was less pressing than earning enough money to survive on for the next week, especially as she was never at home to be able to instil this new hygiene behaviour anyway.

Fatalism

As the above examples demonstrate, poverty can powerfully constrain people's agency, limiting their ability to choose behaviours that could optimise their health and well-being. The association between poor health and poverty, therefore, is not caused by a deficit of knowledge; Sarmila, for example, knew that leaving her son while she worked was not good for him. Quite simply, for the poorest people, it is rarely a matter of simply choosing to give better food or pay better attention to hygiene; often the only choices they have are between a bad situation and a dreadful one.

The lack of choice and control experienced by the poorest women in this study has important implications for health, not only on a practical level (in terms of enabling healthy choices) but on a psychological level too (Bolam, Hodgetts et al. 2003). Perceptions of self-efficacy and control over one's life are recognised to be important factors in many of the behavioural models outlined in Chapter 3. The implication is that objectively having a choice in any situation is not enough; one must also *believe oneself to have that choice*.

Clearly there is a mutually reinforcing relationship between psychological beliefs and consequent behaviours. A lack of agency can result in the development of fatalistic attitudes; these attitudes can then further restrict people's choices by undermining their confidence in these actions to make a difference to their lives. This mutually-reinforcing relationship between behaviour and fatalism has been noted by Powe (1996) in African-American populations with regard to cancer. African-Americans have very high rates of cancer that cannot be fully explained by factors such as a lack of education, poverty or poor access to health services. There is a tendency for this group, therefore, to view cancer as inevitable and unavoidable (1996:18). Consequently, African-Americans are significantly less likely to participate in cancer screening services and thus cancers are rarely detected until they are in the advanced stages. Death often follows rapidly, reinforcing the view that there is nothing that can be done.

A similar sense of fatalism was often expressed by women in this study, particularly those living in the poorest conditions. However, their views were rarely as definite and fixed as Powe (1996) suggested in her paper. Rather these women often seemed to engage in form of 'double-think' whereby they simultaneously believed in quite contradictory things. Thus, it

was very common in interviews for mothers to tell me all the things one could do to prevent sickness – give nutritious food, keep the baby and the home clean, protect the child from cold – whilst simultaneously professing a belief that there was virtually nothing that could be done to prevent sickness in a child. Billig (1996) suggests that this form of thought process is 'dilemmatic,' referring to the ways in which people work to make sense of the dilemma of holding competing and contradictory standpoints. Thus a person's perception of control cannot be regarded as a 'single, unitary and unified… internal status, but an inherently discursive phenomenon' (Bolam, Hodgetts et al. 2003:18).

Nonetheless, mothers did often profess a very strong sense of fatalism and resignation with regard to their ability to protect their child from harm. For some this sense of fatalism stemmed from a belief that their current living environment would negate any positive behaviours they engaged in to protect health.

> *There's nothing you can do to prevent diarrhoea in children. It's just what happens. Diarrhoea is caused by dirt and we live in such a dirty area. We have to live next to this dirty, smelly stream and there's nothing we can do. That's what causes all the diseases round here. No matter what you do, you can't keep yourself or your children clean and healthy if you have to live in a place like this.*
>
> Sarala Karki, mother of Alok Karki

In this quote, Sarala confirms the argument I made at the start of the chapter: that focusing on a single pathway of exposure is unlikely to be effective in the face of such contaminated living environments.

For other mothers, however, the belief that they were largely powerless to prevent sickness in their children stemmed from a more supernatural set of beliefs regarding the concept of 'fate' and the belief that one's life course is determined by the gods. The notion of 'fate' (*bhagya*) is very important in Nepal and was the subject of the first comprehensive portrait of Nepal produced by an indigenous anthropologist. In his book *Fatalism and Development,* Dor Bahadur Bista (1991) argued that the pervasive belief in fate – leading to a systematic evasion of responsibility - was one of the key reasons why Nepal had failed to modernise and develop. Written almost 20 years ago, Bista's analysis now seems somewhat dated, but the role of

fatalism in reproducing social inequalities and their negative consequences remains apposite. Indeed, Morrison et al. (2005) also pointed to the cultural phenomenon of fatalism as encapsulated by the ubiquitous Nepali phrase *'ke garne?'* (what to do?) as an important factor influencing the process of establishing women's groups in rural areas. As they explain, '[o]ur study experience was that fatalism affected both the way people viewed themselves in relation to a problem, and also the power and capacity they believed themselves to have in overcoming it (2005: paragraph 38).

Certainly, a strong belief in fate was expressed by many of the mothers I interviewed and provided insight into how poverty not only restricts choices, but may simultaneously restrict belief in the efficacy of such choices in the first place. Take for example, this quote from one of the mothers in the study:

> *There's nothing I can do that makes a difference to my child's health. It doesn't matter how clean I keep this house, he still might get sick – maybe it's just his fate? If it has to happen, then it will and there is nothing I can do to stop it…. On the day you are born, your whole life is written on that day and there is nothing you can do to change it. If it is written, then it will happen. It's already decided.*
>
> Laxmi Bhujel, mother of Chandra Bhujel

Given such beliefs, how convincing would an intervention programme emphasising the importance of washing hands to prevent disease actually be for these women? Indeed, their own experiences seem to undermine this message on a daily basis. As noted in the Chapter 2, many of the women expressed frustration that, despite their best effort, their children often fell ill, whilst the children of '*bad*' and inattentive parents never became sick. The seemingly capricious nature of illness could even be seen within a single family:

> *I took so much special care of my first child. I made sure I kept him warm by the fire, kept everything so clean, put oil on his head every day, but he was still always sick. Now with this one* [her youngest daughter] *I don't try as hard and she's never*

been sick. I don't think it makes any difference what I do. If they're going to get sick, they'll get sick.

Gunga Thapa, mother of Nani Thapa

It is possible therefore that 'risky' behaviours continue to be practiced by the mothers firstly, because they require less effort (than, for example, taking particular care to encourage their child to eat or to remember to wash hands with soap) and secondly, because they remain unconvinced that their own actions in this matter are likely to make any significant difference. It may also be that the development of fatalistic attitudes is a coping mechanism that allows them deal with their own powerlessness over their children's health (Bolam et al. 2003). In a sense it relieves them of the responsibility for their children's health, by placing the responsibility onto the child's 'fate' or the 'will of the gods'. This sentiment was expressed most clearly to me during an interview with Saraswoti Sunuwar, mother of 12 month-old Kalpana.

When Kalpana was very little she was quite sick and so I took her to the dhami jhankri [shaman]. *He told me that she was going to be very small and sickly until she reaches about three years-old. After that she will be fine again. This is just her fate. There is nothing I can do about it.*

At first Saraswoti was not sure if she believed the *dhami jhankri*, but Kalpana continued to get sick and she concluded that the shaman must have been right after all. Kalpana was amongst the poorest growing of all the children in the study and, at the time of our interview, she was severely stunted and under-weight and mildly wasted. For Saraswoti, therefore, the fact that her child failed to grow was confirmation of her child's 'fate', absolving her of responsibility for it; it was not the fact that they were poor and lived in a slum that accounted for Kalpana's failure to thrive, but simply that this was her fate. She explained how sad she felt whenever her daughter got sick, but knew that after she reached three years of age, her health would improve. Given the absolute powerlessness of Saraswoti to improve the conditions in which her family lived, one can imagine the comfort that this belief might have afforded her.

7.3.2 Forms of capital

As the case studies described above demonstrate, the poorest families in this study were living in extremely difficult circumstances that severely constrained their ability to ensure their own health and well-being. Whilst few families were as desperately poor as Bhumika and Sarmila, many households seemed to be living in highly vulnerable and precarious circumstances; in their current situation they were managing to cope, but few seemed to have any resources to fall back on should catastrophe strike.

The availability of resources within a household is key in understanding vulnerability and resilience. In order to survive households need to draw on a number of different forms of capital and a lack of any particular type of capital can put the family at risk. This principle is at the core of the Sustainable Livelihoods Framework, adopted as a planning and assessment tool by international agencies such as the UK's Department for International Development (DFID 1999). This model identifies five different forms of 'capital' that are needed to ensure survival and well-being (Figure 7.2). Although originally developed for use in rural areas, this model can also be applied within an urban context and is a useful tool for understanding the creation of vulnerability and resilience in the slums.

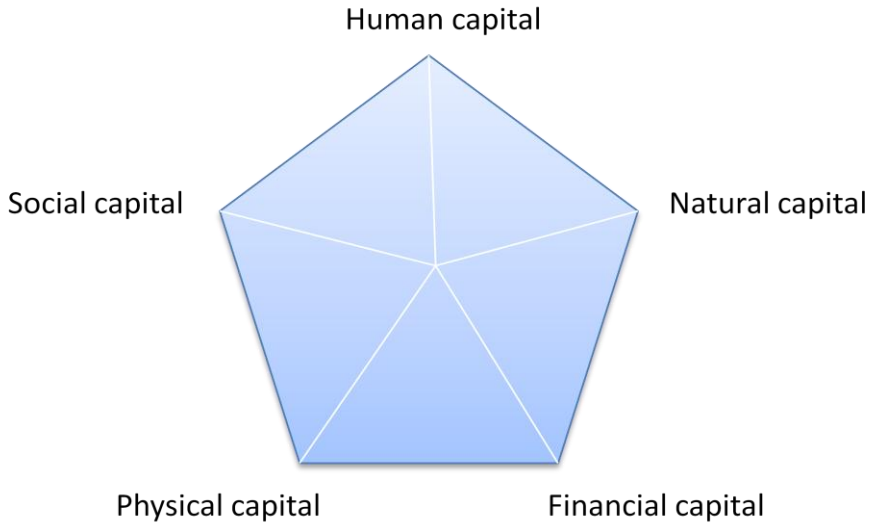


Figure 7.2 The asset pentagon, taken from the Sustainable Livelihoods Framework, DFID (1999).

Within this model, natural capital refers to the natural resource stocks on which livelihoods may be based (DFID 1999). In rural areas this might refer to land and water resources, or access to trees, wildlife and wild foods. Obviously within an urban context where few people derive their livelihood directly from the natural environment (through fishing or farming, for example) a lack of these forms of natural capital does not necessarily result in significant hardship. Physical capital refers to the basic infrastructure and physical environment that provides for people's basic needs and thus includes access to adequate shelter, clean water and sanitation facilities (*ibid*). The amount and quality of labour available to a household is its human capital and is directly linked to the skills, knowledge and good health within a family. Social capital – a much contested concept – is taken here to mean the social resources people can draw upon in order to meet their needs. These social resources include informal networks, membership of formalised groups and relationships of trust that facilitate co-operation and mutual aid (*ibid*). Finally, financial capital refers to the economic resources available to a family in terms of income from employment or trade, remittances, savings and credit.

As discussed in Chapter 2, the physical capital in slum settlements is often very low, with most residents living in dilapidated housing with restricted access to water and sanitation. In addition, slum dwellers suffer from a lack of natural capital in terms of their access to land: most houses are built on extremely small plots of land which, for the squatter residents at least, is illegally occupied. Being largely determined on a community-level, access to these assets did not vary dramatically between households in the slums. There did, however, appear to be significant variations between families with regard to human, social and financial capital. For example, as mentioned above, all of the women who went out to work did so as a result of low financial capital – the family simply could not survive without this additional income. In the case of Sarmila, her husband's drinking meant that he was unable to work, leading to a loss of human capital. The problems associated with maternal working were also often exacerbated a lack of social capital in the form of restricted social connections: in most cases there was no suitable care-giver available to look after the child while the mother worked. Having moved from their natal villages, most women lacked the support network of their wider family who could have helped them with childcare responsibilities.

I have talked at some length about the difficult circumstances of some of the poorest families in the study. However, as already noted, there was significant variation in household socio-economic status and access to these various forms of 'capital' within the slums. I wish to conclude this chapter by considering the experience of some of the richer families in the study in order to show how access to financial and social resources in particular can act as 'buffers' against the negative consequences of living in the slums.

Financial resources

The case of Anjala Dahal and her family exemplifies the way in which economic resources can reduce the vulnerability of a family and prevent an unexpected crisis from pushing the family into severe poverty and destitution. Living in one of the intervention areas, Anjala's family consisted of her husband – Rajendra - and their two children - Salina and Rohit, aged six years and 13 months, respectively. Anjala's household was quite clearly one of the wealthiest families in the sample, and were proof that not everyone in the slums is poor.

> *We walk into her room and it is clear this is a much richer household than many of the others. The room we go into is fairly large and very light, neat and clean. Unlike most of the other houses we've been to this house has proper, shop-bought furniture – in this room two beds and a large wardrobe. The floor is covered with linoleum rather than bare concrete and both beds have fancy bed-linen on them.*
>
> Field notes 03.12.07

Anjala's family were high-caste Brahmins and both she and her husband had been educated to secondary-school level. Her husband worked as a taxi driver and earned a considerable wage – approximately 18-20,000Rs per month. In addition they owned their own house which consisted of six rooms. Anjala and her family used three of these rooms while the remaining rooms were rented out to other families. From the rental of these rooms they earned an extra 2200Rs per month. In comparison to the median wage of 4500Rs per month for families in this study, Anjala's family were very well-off and this wealth translated into good health for her son Rohit. Rohit was one of the best growing children in the study, with his height-for-age, weight-for-age and weight-for-height z-scores all falling well within the 'normal' range. In comparison to other children in the study, he experienced low levels of gut damage and IgG

and high levels of haemoglobin. He also experienced well-below average days of sickness with diarrhoea and fevers (9 days vs. 20 days for diarrhoea; 5 days vs. 18 days for fevers), although he did report an above-average number of days with cold-like symptoms (78 days vs. 56 days).

After providing us with tea, Anjala started to tell us about the terrible experiences her family had just lived through over the recent Dasain festival period. Dasain is one of the most important Nepali festivals and is characterised by huge feasting and gifts being exchanged between brothers and sisters; as such it is often a very expensive festival and where possible families save up money in the preceding months in order to pay for the festivities. Anjala had managed to save about 15,000Rs but in fact none of this money was spent on festivities because of the catastrophe that hit her family just weeks before Dasain.

Anjala's husband, Rajendra, had accepted a fare to take someone out to Manakamana – a holy temple a little over 100km to the west of Kathmandu. On the return journey, however, Rajendra was involved in a head-on collision with a truck on the Kathmandu-Manakamana Highway. Miraculously, neither Rajendra nor the truck driver was badly hurt, but a pedestrian was killed in the collision. Anjala insisted that the accident had not been Rajendra's fault, but both he and the truck driver were jailed for one month in Dhading in the West of Nepal.

Anjala was therefore faced with the prospect of being without both her husband and his income for a month. Both of her children were extremely distressed by their father's absence and Anjala recalled how Rohit experienced terrible diarrhoea during this period. She suggested this sudden sickness in her usually very healthy son was a result of the trauma Rohit felt at being separated from his father.

Though clearly a very distressing experience for the entire family, the actual consequences of this accident were very minimal. Anjala managed to provide for her family relatively easily during this period using the money she had saved for the Dasain festival. Clearly there was no celebration or feasting in their house that year, but they still had money to buy food and meet all their other needs without having to borrow money or sell any of their assets. By contrast, had this event occurred in a poorer family living on the knife-edge of poverty, its consequences for the health and well-being of the entire family could have been catastrophic.

Social resources

Another important 'buffer' against poverty and its impact on health was having a strong family and kin support network – i.e. what the Sustainable Livelihoods Model refers to as social capital. As mentioned in Chapter 4, children in larger households had better height-for-age and weight-for-age z-scores. This finding is perhaps not surprising since larger families potentially mean a) greater earning potential for the family and b) more support for the mother and supervision for the child; both of these could translate into healthy growth by facilitating better nutrition and reducing exposure to infectious diseases.

I have already discussed above the difficulties women faced when they were forced to return to work but did not have anyone to leave their child with. This was not an uncommon situation in the slums since these communities were often fairly transitory in nature and did not consist of extensive kin networks. Very few of the women in the study were native to the Kathmandu valley and often had only moved into the slums upon marriage. Most women had lived in their current home for about four years, though over a fifth had only settled in the slums within the past year. I did not systematically collect data for each household documenting the whether they had family living near-by. However, it was apparent from interviews with both the mothers and local community leaders that the many of the slum dwellers had moved into Kathmandu from rural areas to seek work, and thus had very few family or kinship ties within the city. Two-thirds of the families in this study consisted of just the husband and wife and their children; extended families that included grandparents, uncles and aunts were fairly uncommon.

However, both the statistical analyses and my interviews suggested that extended families offered a significant advantage to young children in terms of promoting healthy growth. Of the 88 children in this study, four children in particular grew exceptionally well with their mean WAZ score being above +1 z-scores. (The next best-growing child had a mean WAZ of just .34, so these children did seem to be particularly exceptional). I therefore interviewed the mothers of each of these children (amongst others) in order to elucidate why their children grew so much better than the others in the study.

All four of these children came from households about double the size of the median household of just four people. They were also relatively well-off, scoring well above average

on the SES scale. As mentioned above, a larger household may result in a higher monthly income since there are more potential wage-earners. Even after taking into account the money spent on food for these extra family members, it is likely that larger households are still better off because they can benefit from economies of scale that are not available to smaller families. It is possible therefore that it was these families' relative wealth, rather than their size, that accounted for their children's excellent growth. I am sure that this is partly the case; any family that gained its income from a variety of sources would be much better able to withstand economic shocks and catastrophes and avoid extreme poverty. However, as shown in Chapter 4, statistical analysis suggest that the size of household had some effect on child growth, over and above that of SES: though the coefficient for household size decreased after controlling for SES, it remained a significant predictor of a child's weight-for-age.

I believe that it is the additional support given to the mother and the shared responsibility and investment in each child that might account for why children in larger families tended to grow better. Take, for example, the case of Dilkumari Lama and her daughters Bimala and Reshma. At the time of our interview, Bimala was just over a year old and Reshma was two-and-half. Being in the target age range, Bimala was recruited into the study and was consistently a very healthy height and weight. She experienced only one episode of diarrhoea during the study period, had below average levels of L:C, IgG and AGP and above average levels of albumin and haemoglobin. She was generally a very healthy, happy child.

Bimala's sister – Reshma – also appeared to be growing well and was clearly well-cared for, despite the fact that she was profoundly disabled. When Reshma was 15 days old she contracted an extremely high fever and her parents immediately took her to the local children's hospital. Diagnosed with cerebral meningitis, Reshma spent 17 days in hospital but it was clear that the illness had left her permanently brain damaged. At two-and-half years of age Reshma was unable to speak or sit up unsupported and needed constant supervision. Caring for two young children is challenging in the best of circumstances, but caring for a child with significant disabilities is even more so. Dilkumari said she worried a great deal about her daughter Reshma.

> *It's very hard with Reshma. I worry about her a lot... She needs constant attention. I have to watch her every second. And it's so hard and frustrating for me because I*

can't always tell whether she is hungry or not or whether she needs anything. She can't tell us what she needs so we just have to guess.

However, despite these difficulties, Reshma was very well cared for by her family. She was well-nourished, clean and tidy. During the interview I observed all the family members interacting and playing with Reshma and it was clear that she was loved and supported by them. Dilkumari explained that though she was obviously the main carer for her two daughters, she had a great deal of support from her parents-in-law and her husband, brother and cousin who also lived in the house. The responsibility for caring for these children was therefore spread between the six adult members of the family and Dilkumari admitted that this made it very easy for her to cope. In addition, her husband's family had lived in this area for over 15 years and thus were well integrated into the local community and had a broad network of friends and contacts (*aphno maanchhe*, literally translated as 'one's own people') who they could call on for support. In contrast to some of the poorest families in this study who had had only recently moved into the city and thus had no contacts, support network or social capital, Dilkumari's family were well supported and less vulnerable to poverty and unforeseen calamitous events.

Summary

This chapter has reviewed some of the reasons why the hand-washing intervention may have failed to have an impact on child health and growth. It started by suggesting that in a highly contaminated environment, where multiple routes of pathogenic transmission exist, tackling a single behaviour is unlikely to have a significant impact on health. It then described in more detail the ways in which poverty and deprivation in the slums can make a child more vulnerable to malnutrition and exposure to infectious organisms. Hand-washing, whilst important, can do little to affect these wider issues of poverty and vulnerability. This point is discussed in more detail in the following chapter.

CHAPTER 8
Conclusions and Implications: Priorities for the 21st Century

Introduction

This chapter concludes the thesis by reflecting on the wider implications of this study for public health policy and practice. It suggests that, while individual behaviour is important for health, the impact of behavioural change is modulated by environmental conditions. Thus, interventions to improve health must focus on creating social, economic, political and physical environments that are conducive to health. Some broad recommendations for global public health are suggested. It ends with a summary of the study's findings and its contribution to the public health literature.

8.1 Why behavioural health interventions fail

Facilitating behavioural change is clearly an important focus of public health practice. As noted in Chapter 3, many health issues could be prevented or alleviated through promoting changes in individual behaviour; for example, improving hand-washing practice to prevent diarrhoea, using bed-nets to prevent malaria, increasing exercise to prevent obesity, stopping smoking to prevent lung cancer. Yet, there remain remarkably few examples of truly successful and sustainable behavioural health interventions (Higginbotham, Briceno-Leon et al. 2001; Merzel and D'Afflitti 2003; Panter-Brick, Clarke et al. 2006). This study sheds light on why this might be the case by emphasising the importance of wider environmental conditions in determining the success or failure of interventions that target human behaviour. (I use the term 'environment' here and throughout this chapter in its widest sense, referring to both the natural physical environment and the wider social, economic, political and historical circumstances). As discussed in the previous chapter, this study suggests that prevailing

environmental conditions can dramatically affect the success of a behavioural intervention: firstly, in terms of enabling or preventing a healthy behaviour to be adopted; and secondly, in terms of the potential impact that behavioural change will have on health.

For the poorest women in this study, their social and economic position was such that they were least likely to be able to truly effect a change in their hand-washing practice. Those mothers living in extreme poverty were forced to spend much of their time working in order to feed their family and often had to leave their children in the care of older siblings. To a large extent therefore, they were not able to take control of their family's hygiene practices. In addition, it is possible (or indeed likely) that a hygiene intervention message was wholly irrelevant for women who are on the edge of survival; hand-hygiene was likely the least pressing issue they had to worry about.

The theoretical model outlined in Chapter 3 suggests that in order to be effective, interventions need to change people's attitudes, social norms and perceptions of self efficacy. For the majority of mothers in the intervention areas the hand-washing programme appeared to be effective in doing this, resulting in significant behavioural change. However, those women whom the Community Motivators identified as not increasing their hand-washing practice were uniformly from the poorest and most disadvantaged families in the study. Lacking social and economic resources, these women faced enormous barriers to behavioural change that this intervention was simply unable to remove. Their inability to increase hand-washing practice therefore represents a failure on the part of the intervention in adequately addressing the wider constraints on their behaviour. An important conclusion from this is that interventions that fail to adequately address these wider constraints on human agency run the risk of exacerbating pre-existing health inequities, since the better-off will always be more able to 'choose' the healthy behaviours than the poorest people (Woodward and Kawachi 2000).

However, these women whom the intervention failed were in a minority: the majority of mothers in this study were able to successfully initiate and sustain behavioural change in terms of their hand-washing practice. Yet despite this increase in hygiene practice, the expected benefits to health (in terms of a reduction in gut damage and immune stimulation and an improvement in growth) failed to materialise. I have suggested that the squalid nature of their

environment meant that the impact and effectiveness of any positive behavioural change was severely compromised by conditions outside their control.

As discussed in the previous chapter, the empirical and theoretical papers by Vanderslice & Briscoe (1995) and Eisenberg et al. (2007) show how the potential effect of a health intervention can be enhanced or diminished by the pre-existing conditions in a community; water quality improvements are a boon when community sanitary conditions are high but are largely irrelevant where sanitation remains a significant issue. This study applied similar reasoning to the effect of a behavioural intervention and suggests that, just as improving water quality is insufficient to improve health in conditions of poor sanitation, focusing on behavioural change may be irrelevant in the face of massive environmental contamination and poor living conditions. I therefore suggest that, in certain circumstances, behavioural change is necessary *but not sufficient* in itself to effect a significant and sustainable impact on health.

The finding that behavioural change does not necessarily translate into an appreciable health impact echoes the conclusions of another behavioural health intervention conducted in The Gambia (Panter-Brick, Clarke et al. 2006). This intervention encouraged women living in rural Gambia to repair holes and tears in bednets to prevent malarial infections from disease-carrying mosquitoes. The intervention – which made use of posters and locally-composed songs – was successful in effecting behavioural change: the mean percentage of repaired holes in bednets rose from 27% in August to 41% by November. However, despite this impressive increase in repairing activities, the majority of nets remained badly torn and there was no decrease in the number of mosquitoes counted inside the nets by the end of the intervention (Panter-Brick, Clarke et al. 2006). In evaluating the impact of this intervention, local people identified several reasons for this lack of success. The most important factor was the lack of access to good quality nets: the quality of netting could vary considerably, with second-handing netting (which was extremely vulnerable to damage) often being used in these poor areas. Secondly, the cost of repairing large holes (requiring patching by a tailor) was prohibitively expensive for many families. Thirdly, women found it difficult to keep up with the repairs to the bed nets, especially as the malarial season coincided with the busiest agricultural period of the year. As one villager commented ‘It is a constant battle to keep a bednet free from holes’ (Panter-Brick, Clarke et al. 2006:2820).

Although taking place in a different country, requiring a different form of behavioural change and aimed at tackling a different type of disease, Panter-Brick et al.'s (2006) study has important parallels with the conclusions from this hand-washing intervention. In both cases, the intervention was largely successful in changing behaviour but this behavioural change failed to translate into the expected impact on health. In both cases, the impact of behavioural change was undermined by the prevailing environmental conditions. In The Gambia, poor quality netting negated the positive impact of repairing holes, as women simply could not keep up with the rate of repair needed. In Nepal, the highly contaminated environment and poor conditions of the slums meant children remained exposed to infectious diseases through multiple pathways.

8.2 Domains of responsibility

This study in the slums of Kathmandu thus adds to a growing body of literature demonstrating the modulating effect of wider environmental conditions for health interventions. This modulating effect has significant implications for public health practice since it raises questions regarding who is responsible for improving health and the types and timing of interventions that should be implemented.

As noted by Cairncross et al. (1996) transmission of disease can occur in both public and domestic domains. Consequently, there are also two domains of *responsibility* for health and well-being: the public domain that creates and sustains an environment (in its widest sense) that either promotes or damages health; and the individual domain where each person exerts a level of control and influence over their health through their own behaviours. The health of every person, whether rich or poor, is subject to the influence of both of these domains; however, the *degree* of influence each domain has on an individual's health is by no means equal across all sections of society. For those at the poorest end of the scale, the level of control they have over their health is often relatively minimal – they are often powerless to combat the wider environmental influences that can damage their health. As Woodward and Kawachi note, 'there is no doubt that health is more than a matter of personal choice: the decisions that people make about health are shaped by the environment in which they are

conceived, raised and live their adult lives. There are many instances in which personal responsibility plays a very small part' (Woodward and Kawachi 2000:924).

The excessive burden of morbidity and mortality suffered by the poor is therefore largely socially produced – the product of poverty and inequality that translates itself into poor housing, lack of basic services, infection and malnutrition (CSDH 2008). Indeed, a recent study commissioned by the WHO estimated that a quarter of the global burden of disease, and over a third of the burden among children, is due to modifiable environmental such a un-safe drinking water and inadequate sanitation (Prüss-Üstün and Corvalán 2006). Given the disproportionate influence of this 'public' domain on health, it becomes clear that any desire to alleviate ill-health must start here. Basic conditions for health and well-being must be equitably provided to all members of the population: clean water supplies, adequate sanitation, a nutritionally adequate diet, decent housing and access to good quality health care services are essential. Undoubtedly, health is also influenced by the actions of individuals and thus interventions that focus on behavioural change are also very necessary. However, only once a positive public domain of health has been created will these behavioural interventions be able to be truly effective.

8.3 Implications for public health practice

There are two important implications for public health to come out of this discussion. Firstly, it has important political and ideological ramifications. In order to effect the greatest improvements to health and prevent a widening of the health gap between the richest and the poorest, interventions must focus on helping the poor to adopt healthier behaviours by removing the socio-economic and political barriers that restrict their agency and participation. The most effective type of public health interventions, therefore, would be ones that tackle these underlying issues of poverty and inequity. Consequently, the eradication of poverty and the more equitable distribution of money, power and resources must be a fundamental and core aim of public health. This conclusion is one of three recommendations put forward by the WHO's Commission on the Social Determinants of Health (CSDH 2008).

Secondly, it has implications for the types of interventions to be implemented and the timing of their delivery. In the context of the slums, there are broadly two types of interventions that could be implemented to improve health: those that focus on improving environmental conditions (such as providing clean water and adequate sanitation) and those that focus on behavioural change (such as promoting hand-washing). Clearly both types of intervention are necessary to improve health. However, because of the ways in which these types of interventions interact, it is likely that neither is sufficient in itself to effect significant improvements in health. For example, environmental improvements – such as improved water and sanitation – are of course important, but in the absence of accompanying behavioural change they will have only a moderate impact on health: if people do not ensure the water supply is kept free from contamination or properly maintain the sanitary facilities, there will be little net benefit to health. In other words, behavioural change must accompany environmental improvements. Equally, if behavioural change is targeted without addressing environmental considerations (as was the case in this study) little effect can be expected. Behavioural change is essential, but in the face of multiple pathways of infection (and other issues related to general poverty), it cannot alone create the desired health impact.

Clearly both types of interventions are crucial. However, the timing of these interventions is important. As Briscoe (1987) points out, certain types of interventions (e.g. improvements to environment) are required to create the necessary conditions that later (behavioural) interventions need in order to be maximally effective. Behavioural interventions are important and we must continue to investigate the best ways to help people change their behaviour. However, behavioural change is largely the end point of a significant amount of preparative work; only when behavioural interventions build upon significant environmental and social improvements will we see their full effect. In others words, the responsibility of improving health must necessarily remain within the public domain, until such a time that the choices people make in relation to their health are truly free and capable of being fully effective. The urgent priority of public health in the 21st Century therefore must be to create environmental and social conditions that a) enable people to freely choose health-promoting behaviours and b) maximise the impact of these behavioural changes.

8.4 Moving forward: the social and political debates surrounding public health in the 21st Century

This study was about promoting hand-washing to improve the health and growth of children living in Kathmandu's slums. However, it has highlighted the need to consider the wider social and political debates that must be engaged in if public health interventions focusing on behavioural changes are to be effective. As a result of my experience of conducting this research in Nepal, I therefore wish to present here a number of broad recommendations that relate to public health practice and research on a more global level.

8.4.1 Getting our priorities right

The international public health community (alongside politicians, policy-makers and other relevant stakeholders) must urgently re-think their funding and development priorities to better reflect the true burden of morbidity and mortality. The principle aims of public health are the prevention of disease and the promotion of health and well-being in both individuals and communities. Currently our way of determining priorities does not seem to fit with an equitable or even epidemiologically-informed approach to such a mission.

Two diseases that are currently benefitting from massive amounts of national and international funding and support are malaria and HIV/AIDS. The sixth Millennium Development Goal aims to reduce the global mortality burden from major preventable diseases, with these two diseases (along with tuberculosis) being the only ones specifically mentioned (United Nations 2008). Undoubtedly, these two diseases cause millions of unnecessary deaths every year and should indeed be a significant concern to public health. But is the current attention focused on these diseases is wholly justifiable in terms of promoting health on a global scale?

Malaria and HIV/AIDS account for about 8% and 3%, respectively, of all childhood deaths (Rudan, El Arifeen et al. 2007). Thus, every year, just over one in ten of the children who will die before they reach their fifth birthday, dies from one or other of these diseases. Yet, *one in three* will die as a result of diarrhoea or acute respiratory infections such as pneumonia (*ibid*). These two diseases are the *leading killers* of young children, accounting for 17% and 19% of

childhood mortality, respectively, yet neither of these diseases is mentioned specifically in the targets set for the sixth MDG.

It appears that there is a misinterpretation of the relative significance of these diseases for the global burden of morbidity and mortality by those in charge of setting our global health development priorities and distributing the funding needed to meet them. For example, acute respiratory infections account for 26% of the global communicable disease burden, yet receives just 2.5% of the available direct funding. By contrast, while HIV/AIDS accounts for 31% of this disease burden, it receives almost half (46%) of all direct funding (Shiffman 2006). Similarly, as Morris et al. (2008) point out, between 2000 and 2002 HIV/AIDS programmes received $2.2 billion of foreign aid per year, compared to just $250-300 million for programmes tackling the far more ubiquitous problem of under-nutrition. Surely these figures suggest a severe mismatch in our priority setting? Undoubtedly, HIV/AIDS and malaria are important and research should continue into their control and eradication, but this should not be at the expense of programmes that aim to tackle the diseases that cause the greatest number of deaths.

Rudan et al. (Rudan, El Arifeen et al. 2007) lay the blame for this situation on the dominant research model employed by major global funding bodies. The current approach favours (often explicitly) basic research that generates 'new knowledge', rather than research into how to more effectively and efficiently apply and scale-up our current knowledge. As they note, 'the development and proof of effective interventions has been seen in the past as the legitimate endpoint of research' (2007:56), with no consideration given to how these interventions can be rolled out in the poorest countries with few resources and little existing infrastructure. For example, a recent analysis of the funding policies of the National Institute for Health and the Gates Foundation found that 97% of grants were given for the development of 'new' technologies to improve health; just 3% was spent on research on improving delivery and use of existing interventions (Leroy, Habicht et al. 2007). Thus we have been left with a situation where we know about some very simple and very effective interventions, but we do not know how to deliver these interventions at scale to ensure that those who are most in need receive them (Victora, Hanson et al. 2004; Costello, Filippi et al. 2007; Rudan, El Arifeen et al. 2007).

My first recommendation, therefore, (following Rudan et al. 2007 and Victora et al. 2004) is a reform of the current funding models employed by international donor agencies. Whilst basic research into new areas of science is necessary, donors should also actively encourage research into the successful and cost-effective application of health interventions at scale in poorly-resourced countries. Such investment could result in millions of lives saved: it has been estimated that 'about two-thirds of child deaths could be prevented by interventions that are available today and are feasible for implementation in low-income countries at high levels of population coverage' (Jones, Steketee et al. 2003:69). Such intervention programmes should be subject to comprehensive evaluation not only regarding their impact on health and well-being outcomes, but also in terms of the way in which they are implemented. As Victora et al. (2004) suggest, it is necessary to evaluate the implementation strategies of interventions as rigorously as the interventions themselves in order to be able to determine critical elements of success and apply this learning elsewhere. In conjunction, there must be recognition of the longer-time scale needed to properly design, implement and evaluate intervention programmes. Funding tranches of three-to-five years are simply too short if we are truly to implement, evaluate and learn from such interventions.

8.4.2 Water and sanitation for all

Following the recommendation to focus on the application and scaling up of successful interventions, my second urgent recommendation is for unwavering and universal commitment to the provision of clean water and adequate sanitation *for all*.

One child dies every 17 seconds from diarrhoeal disease (WHO 2005). Millions more experience numerous non-fatal episodes of diarrhoea per year and it is likely that the majority of children living in the poorest countries experience sub-clinical damage to the intestinal mucosa on a more-or-less chronic basis. These infections are strongly associated with childhood growth faltering, which in turn has been associated with over half of all childhood deaths. In addition, inadequate water and sanitation systems are associated with numerous other diseases such as intestinal helminth infections, dracunculiasis, schistosomiasis and trachoma. Almost half of those living in the developing world have one or more of these serious diseases and over half of the hospital beds in the world are occupied by people with

these complaints (Bartram, Lewis et al. 2005). The lack of two basic and fundamental pre-requisites for health – water and sanitation – is a public health issue of epic proportions.

We have known the best way to prevent these diseases ever since Sir Edwin Chadwick's campaign to improve sanitary conditions in Victorian London. Indeed, sanitation was recently voted as *the* most important medical advance in the past 150 years by readers of the British Medical Journal (ahead of the development of antibiotics, vaccines and the contraceptive pill) (Mackenbach 2007). Yet, millions of people throughout the world still have no access to this basic service. Current estimates suggest that over a billion people in the world do not have access to a safe water supply and 2.6 billion lack sanitation facilities (UNICEF 2006). There are compelling reasons, however, to suspect that the actual figures may be much higher (Satterthwaite 2003). Increasing pressure on the already-stretched infrastructure of the poorest nations that will be caused by rapid urbanisation in the coming decades is likely to worsen this situation dramatically. In addition, climate change models predict that by the end of the century many more populations will experience significant water-stress. This will make the provision of adequate water and sanitation even more complicated and challenging (Costello, Abbas et al. 2009). Yet, if we are serious about tackling the global burden of disease, this is one of the most fundamental and effective interventions to choose.

One of the targets of the Millennium Development Goals is to 'halve, by 2015, the proportion of the population without sustainable access to safe drinking water and basic sanitation' (United Nations 2008). Good progress has been made on reaching the drinking water target. In 1990, 71% of the world's population had access to safe drinking water; by 2006 this had increased to 84% just two percentage points short of the 86% target set for 2015 (United Nations 2008). Progress regarding sanitation has been much less impressive and unless dramatic action is taken in the next few years, this target is most unlikely to be met (Mara, 2003). In 1990, 41% of the world was using improved sanitation facilities; by 2006 this had only increased to 53%, well short of the 2015 target of 71%. The regional situations in South Asia and sub-Saharan Africa present a much bleaker picture: as of 2006, only a third of people in these regions had access to adequate sanitation facilities. It should also be noted that even if the 2015 target is met in full, this will still leave almost a third of people in the world without sanitation and one-in-six without safe water supplies.

Since water and sanitation are essential for a healthy life, the current situation represents a blatant violation of the human rights of millions of people throughout the developing world. On moral grounds alone, therefore, concerted international effort should be given to this critical issue. However, unfortunately moral arguments tend to carry much less weight than economic ones. Yet, even from an economic point of view, provision of basic water and sanitation services for all people makes good sense (Bartram, Lewis et al. 2005).

Hutton and Haller (2004) recently undertook a comprehensive and extensive cost-benefit analysis on water and sanitation improvements at the global level. Their analysis considered five different intervention scenarios, ranging from minimal improvements to current water and sanitation facilities to the ideal situation whereby all households have access to a piped water supply and sewerage connections. Costs for each of these different intervention were estimated using data from a variety of sources and included both start-up costs (in terms of planning and supervision, hardware and construction costs) and recurrent costs (operational and maintenance costs). The potential benefits considered included: value of deaths avoided, value of productive days of work or school attendance gained, value of time saved due to improved access to water and sanitation, and savings to both the health sector and patients due to avoided illness. The results showed that for all five intervention scenarios, the potential benefits out-weighed the costs: the return on each $1 of investment was in the range of $5 to $28. Even under the most pessimistic scenarios (where costs data were given their upper-bound limit and benefits data were given their lowest) the potential benefits generally continued to out-weigh the costs.

It seems unlikely that the MDG target for sanitation will be met by 2015. But providing basic water and sanitation is wholly achievable as a medium-term aim. As Bartram et al. note, ‘expanding safe drinking water and sanitation coverage is not complex: it requires neither colossal sums of money nor scientific breakthroughs and technological advances’ (Bartram, Lewis et al. 2005:811). What it does require, however, is political will and deliberate commitment by donors to not only increase their funding levels, but also to refocus their spending priorities onto the provision of these basic services.

8.4.3 Urban health and the slums

My third recommendation is for special attention to be paid to the creation and management of healthy urban environments, in particular focusing on how we can manage the dual problems of the increasing global population and the rapid urbanisation of humanity.

The previous 250 years have witnessed a massive and unprecedented increase in the global human population (Figure 8.1). In 1750, at the start of the Agricultural and Industrial Revolutions, the world's total population stood at about just under 800 million people (United Nations Population Division 1999). By 1900 the global population had more than doubled to 1.6 billion people. Fifty years later it had increased to 2.5 billion and in 1999, just a few months shy of the new millennium, the human population of the world passed the 6 billion mark (*ibid*). As of October 2009, the current estimated population stands at about 6.8 billion, and this figure is expected to continue to increase until about 2050, when it is finally expected to stabilise somewhere between 9-10 billion (UNFPA 2001).

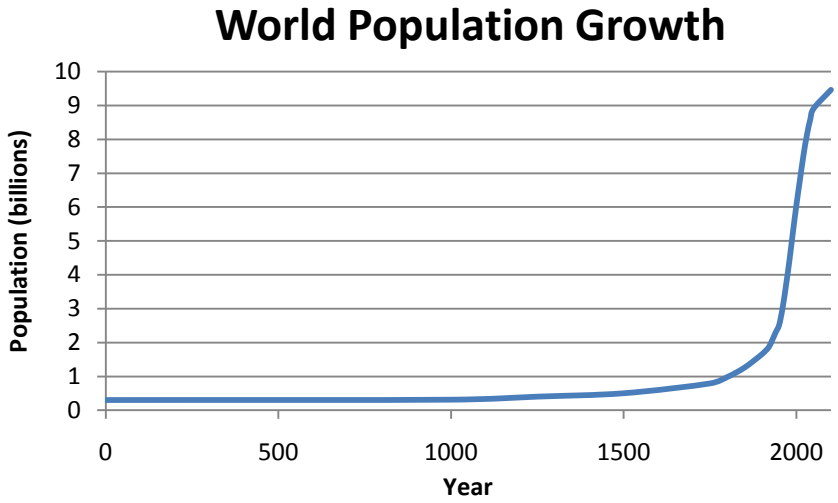


Figure 8.1 World population growth (in billions) from 0-2050 AD. Data taken from United National Population Division (1999).

This explosion in the global population has precipitated the massive urbanisation of huge parts of the world. In 1950, just 29% of the world lived in urban areas, increasing to 37% by 1975 (UN-Habitat 2003). However, by 2006 over half of humanity lived in towns and cities. This

dramatic shift in human ecology is expected to continue, with almost two-thirds of the global population living in urban areas by 2030 (*ibid*).

Over 90% of this increase in urban populations over the next 30 years is expected to take place in the less developed regions of the world (Sclar et al., 2005); in these regions the urban population is increasing by 2.3% per annum, versus just 0.4% in the developed world (UN-Habitat 2003). The poorest countries of the world will therefore have to bear the greatest burden of this population growth, yet almost no planning or development effort has thus far gone into how and where these people are going to be accommodated (*ibid*). Even where planning has occurred, often the rate of urban population growth has massively outstripped the ability of local authorities to provide affordable housing, basic services and health infrastructure (Ooi and Phua 2007). In such circumstances, the poorest migrants to the cities are usually forced to live in squalid and informal settlements and the number of people living in the slums is therefore expected to double by 2030, possibly rising as high as three billion by 2050 if urgent action is not taken to tackle this serious issue (Vlahov, Fruedenberg et al. 2007).

Accompanying these changes in the last few decades has been a significant increase in levels of poverty and inequity in certain parts of the world, and the rapid urbanisation of poverty. In the past, living in an urban centre usually offered significant health benefits. Urban populations generally have better access to water and sanitation, more secure food supplies, higher levels of parental education and better access to health services. However, more recent data has questioned this supposed 'urban advantage'. Recent studies that disaggregate urban morbidity and mortality data indicate that for the poorest urban residents there is no urban advantage and children living in the poorest urban environments are at least as disadvantaged in terms of health as their rural counterparts. Timaeus & Lush (1995) found that the mortality rate for children living in urban environments in Ghana, Brazil and Thailand was at least as high as that seen in rural populations. Brockerhoff & Brennan (1998) reported a much slower decline in early mortality in residents living in big cities than those living in smaller towns and villages, and an actual increase in urban infant mortality rates in sub-Saharan Africa since the 1970s. Haddad et al. (1999) found that in 12 of the 16 counties they studied, absolute levels of underweight in urban children were increasing, and at a much faster rate than in rural area, suggesting that the locus of malnutrition may be shifting from the rural to the urban population.

The health problems associated with urbanisation and increasing poverty are likely to be greatly exacerbated by the potentially devastating effects of global climate change. Increases in global temperatures will adversely affect food production and could leave billions of people facing severe food shortages by the end of the century (Costello, Abbas et al. 2009). Changing patterns of rainfall and increased global temperatures will result in significant challenges to the provision of basic needs for health such as clean drinking water and adequate sanitation. As parts of the world become too hot, too dry or too prone to natural disasters, massive migration of human populations will occur, primarily to urban centres of the developing world (*ibid*). As described above, these towns and cities are woefully under-prepared for such a massive influx of displaced people.

Creating healthy urban environments is therefore likely to be one of the greatest public health challenges ever faced. Immediate attention must be given to how we can deal with these issues and provide for the basic needs of all people. Particular attention must be paid to the rapid increase in slum populations. As pointed out by Payne (2005), we need a twin-track approach to this issue that involves a) improving the lives of the current slum dweller population by developing innovative approaches to tenure security, upgrading existing buildings and improving access to public services, transport, education and employment and b) concerted efforts to address the need for low-cost urban housing in order to prevent a dramatic increase in slum areas in the coming decades.

With regard to this second point, special attention should be focused on cities such as Kathmandu in an attempt to avert a potential humanitarian crisis. As mentioned in Chapter 2, Kathmandu currently has a relatively small urban slum population. However, it is also one of the fastest growing cities in South Asia (Pradhan 2004); unless urgent action is taken, the slum population will burgeon dramatically in the coming decades. The majority of urban growth that will take place in the first half of this century will not take place in the mega-cities like Mumbai or Dhaka, but in more medium-sized cities and towns like Kathmandu (UN-Habitat 2003; Vlahov, Fruedenberg et al. 2007). It is crucial that national and local governments urgently consider how they will accommodate and meet the basic needs of this expected influx of people to such cities. The development of low-cost, sustainable housing, accompanied by development of existing transport, health and education infrastructure is crucial if we are to

avoid a significant worsening of health outcomes in the 21st Century. Addressing these issues now in a timely and proactive manner would be a means of preventing a human disaster in the making. Innovative and creative ideas on how to deal with these issues must be learned from the experiences of other cities; local people must be involved at every stage of the planning and implementation of solutions to this potential problem.

8.4.4 Tackling root causes of ill-health: poverty and inequality

My fourth and final recommendation is a call for those working in the field of public health (both practitioners and academic researchers) to tackle the root causes of ill-health – poverty and inequality – by speaking out against the policies that create them. This inevitably means moving beyond a narrow definition of the role of public health and actively engaging with those working in public and political spheres. Some have argued strongly against public health practitioners and researchers getting involved in political and policy-related debates (Rothman and Poole 1985; Rothman, Adami et al. 1998; Savitz, Poole et al. 1999). Yet, the creation of both health and scientific knowledge is *always* a profoundly political issue (Singer 1995); it is therefore disingenuous to suggest that public health workers have no place engaging in political debates. As Freedman explains, although often presented as a form an objective scientific inquiry, public health research is inherently a value-laden activity and as such it is always, and inevitably, a highly political endeavour (1995:314). It is appropriate therefore that public health practitioners and researchers question whether the social, political and economic systems that are currently in place (and the values that under pin them) are conducive to the aim of promoting the health and well-being of all people (Krieger 1999).

'Poverty is the single most important determinant of poor health' (Katz 2004:752). The excessive burden of morbidity and mortality in the poor, whether in rich or poor countries, is the result of inequitable access to power, income, good and services. It is not in any sense a natural or inevitable phenomenon, but, as the Commission on the Social Determinants of Health puts it, the product of 'a toxic combination of poor social policies and programmes, unfair economic arrangements and bad politics (CSDH 2008:1).

Since the 1980s, neo-liberal economic policies have been aggressively promoted across the globe resulting in a removal of barriers to international trade, the liberalisation of capital flows and the creation of strong patent regimes that regulate the use and transfer of new technologies and intellectual property rights (Cornia 2004). Proponents of the neo-liberal position argue that free-trade and liberalization promotes economic growth and increases a country's GDP, consequently raising the standards of living of all its citizens. However, this has largely failed to happen. It is now widely recognised that very few of the benefits from economic growth 'trickle down' to the poor unless governments make specific efforts to equitably redistribute economic gains (Katz 2004). Indeed, the wide-scale adoption of neo-liberal policies since the 1980s have generally been very successful in slowing, halting or even reversing the social and economic gains made in the preceding era.

A study commissioned by the Center for Economic and Policy Research recently sought to evaluate the impact of globalization on a number of social and economic outcomes by comparing data from the pre-globalization era (1960-1980), generally characterised by greater government control of public resources and redistribution of wealth, with data from 1980-2000, representing the period of globalization and neo-liberal reform (Weisbrot, Baker et al. 2002). The outcome measures they assessed comprised of: per capita GDP, life expectancy, infant mortality rate, adult mortality rate, public spending on education, literacy rate and gross primary school enrolment. Their results make disturbing reading and point overwhelmingly in one direction: 'in every category, the comparisons show diminished progress overall in the period of globalization compared with the prior two decades' (2002:249). Although the authors note that these data cannot be taken as proof that the policies associated with globalization are directly responsible for these declines in performance, they do present a strong *prima facie* case to suggest that globalization has largely failed to deliver on its promises of economic and social development.

The irrationality of our current economic system is typified in its approach to international aid. The Commission on Macroeconomics and Health, led by economist Geoffrey Sachs, has recently suggested that increased international aid, aimed at implementing a basic set of medical interventions in the developing world, would result in approximately $360 billion dollars in economic gains per year (WHO 2001). Yet $700 billion are lost annually through

unfair trade, $382 billion through debt repayments, £160 billion through capital flight and tax havens and $250 billion through untaxed and uncontrolled financial flows (Katz 2008). Macro-economic reforms to address such issues would therefore potentially release almost $1.5 trillion per year, as opposed to Sachs' estimate of $360 billion from increased international aid. As Katz wryly notes, 'This is not a difficult choice, were it to be presented transparently to the people of the developing world' (2008:3).

Given these disturbing facts, I believe it is the responsibility of public health practitioners and researchers to continue to question and speak out against the hegemony of an economic system that puts the pursuit of profit above the welfare of individuals, communities, entire nations and indeed the planet as a whole. If the aim of public health is the prevention of disease and the promotion of health in individuals and communities, then the eradication of poverty and inequality must be a fundamental core principle and public health workers must continue to engage with and influence public policy and political debate on these issues.

8.5 Summary of this study and its contribution

The aim of this study was to investigate ways of reducing levels of childhood growth faltering. Growth faltering is a dangerous and insidious problem affecting millions of children through the developing world, but particularly in South Asia. Framed within the bio-cultural research paradigm, this study used a mixed-methods approach in order to design, implement and evaluate a community-based intervention that sought to improve the growth of young Nepali children living in the slums of Kathmandu.

There has been growing interest in recent years in the role of sub-clinical infections in causing childhood growth faltering. Clinically symptomatic infections that cause diarrhoeal disease are known to depress growth in young children (Black, Brown et al. 1984a; Bairagi, Chowdhury et al. 1987; Walker, Grantham-McGregor et al. 1992; Torres, Peterson et al. 2000; Moore, Lima et al. 2001). However, most childhood diarrhoeal episodes are infrequent and short-lived and cannot account for the very high levels of growth faltering seen in children from the poorest parts of the world. It has been suggested, therefore, that sub-clinical, yet chronic, infections may be an important causal factor in childhood growth faltering (Checkley, Gilman

et al. 1997). Such infections cause damage to the mucosal lining of the small intestine resulting in maldigestion and malabsorption of nutrients (Lunn 2000). In addition, damage to the mucosa stimulates an immune and inflammatory response in the child, thus diverting energy away from processes such as growth. These infections may not be severe enough to produce physically visible symptoms (such as diarrhoea) in the child, yet because such infections are often *chronic*, over time they can significantly depress a child's growth.

Reducing sub-clinical levels of gut damage and immune stimulation may therefore be crucial in alleviating the problem of growth faltering in young children. Various suggestions have been made as to how this could be done, though most of these have focused on nutritional supplementation (Thurnham, Northrop-Clewes et al. 2000; van de Merwe 2006) or pharmacological interventions aimed at eliminating intestinal parasites (Northrop-Clewes, Rousham et al. 2001; Goto, Mascie-Taylor et al. 2009). This current study was the first to investigate a behavioural intervention aimed at reducing gut damage and immune stimulation.

This study was based on the hypothesis that reducing a child's exposure to enteric pathogens through improved hygiene would result in a reduction in sub-clinical gut damage and immune stimulation, and therefore potentially reduce growth faltering. Hand-washing with soap has been shown to be one of the simplest, cheapest and most effective means of preventing diarrhoeal disease in young children, resulting in a risk reduction of 42-47% (Curtis and Cairncross 2003). However, the impact of this simple intervention on rates of sub-clinical infection had not yet been documented. This study therefore sought to fill this gap in the literature by investigating whether hand-washing with soap not only reduced diarrhoeal disease, but also had an impact on sub-clinical gut damage, immune stimulation and growth faltering.

To this end, a culturally compelling hand-washing intervention was designed, informed by data collected during structured observations, interviews and focus groups with local women from the slums of Kathmandu. This intervention was implemented with mothers living in the intervention areas for six months, whilst control mothers continued with their normal hygiene practices.

The intervention appeared to be successful in improving hand-washing rates amongst most of the mothers in the intervention areas: reported hand-washing at the five key junctures improved significantly over the period of study for mothers in the intervention areas and were significantly higher than those reported by control mothers by the end of the intervention. As expected, this improvement in hand-hygiene resulted in a reduction in diarrhoeal disease: children from the intervention areas experienced 31% fewer diarrhoeal episodes than the control children, resulting in a 41% reduction in the total number of days with diarrhoeal symptoms over the period of study.

However, this reduction in clinical morbidity appeared to have no effect on levels of sub-clinical gut damage and immune stimulation, nor did the intervention reduce rates of growth faltering in these children. There were no significant differences between intervention and control groups for levels of gut damage (L:C) or immune stimulation (IgG, AGP) in children over the period of study. Similarly, there was no improvement in biochemical nutritional status (haemoglobin, albumin) nor in growth status (HAZ, WAZ, WHZ).

Following consideration of the different possible reasons for these negative findings, I have suggested that behavioural interventions, such as those focusing on improving hand-hygiene, may be ineffective when children live in a slum environment that continually exposes them to infectious diseases through numerous other pathways. In addition, the ability of a mother to 'choose' to improve her hand-hygiene may be severely constrained by the general conditions of poverty and insecurity in which she lives.

What this study shows, therefore, is that concentrating on modifying a single behaviour, whilst ignoring the wider environmental and socio-economic context in which that behaviour takes place, will have limited results. Behavioural change can only be truly effective when it builds upon wider environmental improvements. I argue, therefore, that future public health interventions must consider the need to a) improve environmental conditions to allow behavioural change to be effective and b) alleviate poverty in order to increase self-efficacy and people's ability to practice healthy behaviours. In others words, there is a need for a return to a basic needs approach to the diseases of poverty, in line with a public health philosophy based on the principles of equity and social justice.

References

Adair, L. (1999). "Filipino children exhibit catch-up growth from age 2-12 years." Journal of Nutrition **129**: 1140-1148.

Adelekan, D. A., C. A. Northrop-Clewes, et al. (2003). "Use of biomarkers of sub-clinical infection, nutrition and neonatal maturity to interpret plasma retinol in Nigerian neonates." British Journal of Nutrition **90**(2): 353-361.

Ahmed, N., M. Zeitlin, et al. (1993). "A longitudinal study of the impact of behavioural change intervention on cleanliness, diarrhoeal morbidity and growth of children in rural Bangladesh." Social Science & Medicine **37**(2): 159-171.

Aiello, A., E. Larson, et al. (2007). "Consumer antibacterial aoaps: Effective or just risky?" Clinical Infectious Diseases **45**(s2): S137-S147.

Ajzen, I. (1991). "The theory of planned behaviour." Organizational Behaviour and Human Decision Processes **50**: 179-211.

Ajzen, I. and M. Fishbein (1980). Understanding Attitudes and Predicting Social Behaviour. Englewood-Cliff, NJ, Prentice-Hall.

Akre, J. (1989). "Infant feeding: the phsyiological basis." Bulletin of the World Health Organisation **67**(supplement): 1-108.

Alam, D., G. Marks, et al. (2000). "Association between clinical type of diarrhoea and growth of children under 5 years in rural Bangladesh." International Journal of Epidemiology **29**: 916-921.

Alam, N., B. Wojtyniak, et al. (1989). "Mothers' personal and domestic hygiene and diarrhoea incidence in young children in rural Bangladesh." International Journal of Epidemiology **18**(1): 242-247.

Asian Development Bank (2001). Cities data book: Urban indicators for managing cities. Manilla, Asian Development Bank.

Bairagi, R., M. Chowdhury, et al. (1987). "The association between malnutrition and diarrhoea in rural Bangladesh." International Journal of Epidemiology **16**(3): 477-481.

Bandura, A. (1977). "Self-efficacy: Towards a unifying theory of behavioural change." Psychological Review **84**: 191-125.

Bandura, A. (1986). Social Foundations of Thought and Action: A Social Cognitive Theory. Englewood-Cliff, NJ, Prentice-Hall.

Bandura, A. (1997). Self-efficacy: The exercise of control. New York, Freeman.

Barker, D. J. (1998). "In utero programming of chronic disease." Clinical Science **95**(2): 115-128.

Barrell, R. and M. G. M. Rowland (1979). "Infant foods as a potential source of diarrhoeal illness in rural West Africa." Transactions of the Royal Society of Tropical Medicine **73**: 85-90.

Bartram, J., K. Lewis, et al. (2005). "Focusing on improved water and sanitation for health." The Lancet **365**(9461): 810-812.

Beasley, B. (2003). Lactose and creatinine excretion as an alternative to the Lactose/Mannitol permeability test. Department of Biological Anthropology. Cambridge, University of Cambridge. MPhil in Biological Anthropological Science.

Beasley, B. and P. Lunn (2004). "Lactose and creatinine excretion as an alternative to the lactose-mannitol intestinal permeability test." Annals of Human Biology **31**(5): 605.

Becker, M., D. Haefner, et al. (1977). "The health belief model in the prediction of dietary compliance: A field experiment." Journal of health and social behaviour **18**: 348-366.

Becker, S., R. Black, et al. (1991). "Relative effects of diarrhea, fever, and dietary energy intake on weight gain in rural Bangladeshi children." American Journal of Clinical Nutrition **53**(6): 1499-1503.

Berkman, D. S., A. G. Lescano, et al. (2002). "Effects of stunting, diarrhoeal disease, and parasitic infection during infancy on cognition in late childhood: a follow-up study." The Lancet **359**(9306): 564-571.

Berrant, M., M. Khourie, et al. (1992). "Effect of iron deficiency on small intestinal permeability in infants and young children." Journal of Pediatric Gastroenterology and Nutrition **14**: 17-20.

Bhandari, N., M. Bhan, et al. (1989). "Association of antecedent malnutrition with persistent diarrhoea: a case-control study." British Medical Journal **13**(298(6683)): 1284-1287.

Bhutta, Z. (2000). "Why has so little changed in maternal and child health in south Asia." British Medical Journal **321**: 809-812.

Billig, M. (1996). Arguing and Thinking: Approaches to Social Psychology. Cambridge, Cambridge University Press.

Binkin, N., R. Yip, et al. (1988). "Birth weight and childhood growth." Pediatrics **82**: 828-834.

Biran, A., et al. (2005). "Formative research for hygiene promotion in Kyrgyzstan." Health Policy and Planning **20**(4): 213-221.

Bista, D. (1991). Fatalism and Development: Nepal's Struggle for Modernisation. India, Orient Longman Ltd.

Black, R., K. Brown, et al. (1984a). "Malnutrition is a determining factor in diarrheal duration, but not incidence, among young children in a longitudinal study in rural Bangladesh." American Journal of Clinical Nutrition **39**(1): 87-94.

Black, R. E., K. H. Brown, et al. (1984b). "Effects of Diarrhea Associated with Specific Enteropathogens on the Growth of Children in Rural Bangladesh." Pediatrics **73**(6): 799-805.

Bogin, B. (1999). Patterns of human growth. Cambridge, Cambridge University Press.

Bolam, B., D. Hodgetts, et al. (2003). "'Just do it': An analysis of accounts of control over health amongst lower socioeconomic status groups." Critical Public Health **13**(1): 15-31.

Borsook, H. and J. W. Dubnoff (1947). "The hydrolysis of phosphocreatine and the origin of urinary creatinine." Journal of Biological Chemistry **168**(2): 493-510.

Briend, A., K. Hasan, et al. (1989). "Are diarrhoea control programmes likely to reduce childhood malnutrition? Observations from rural Bangladesh." The Lancet **ii**: 319-322.

Briscoe, J. (1984). "Intervention studies and the definition of dominant transmission routes." American Journal of Epidemiology **120**(3): 449-456.

Briscoe, J. (1987). "A role for water supply and sanitation in the child survival revolution." Pan American Health Organization **21**(2): 93-105.

Brockerhoff, M. and E. Brennan (1998). "The poverty of cities in developing regions." Population and Development Review **24**(1): 75-114.
Brown, K. H. (2003). "Diarrhea and Malnutrition." Journal of Nutrition **133**(1): 328S-332.
Brown, L. R., Y. Yohannes, et al. (1994). "Rural Labor-Intensive Public Works: Impacts of Participation on Preschooler Nutrition: Evidence from Niger." American Journal of Agricultural Economics **76**(5): 1213-1218.
Cairncross, S. (2003). "Editorial: Handwashing with soap; a new way to prevent ARIs?" Tropical Medicine & International Health **8**(8): 677-679.
Cairncross, S., U. Blumenthal, et al. (1996). "The public and domestic domains in the transmission of disease." Tropical Medicine and International Health **1**: 27-34.
Calder, P. and A. Jackson (2000). "Undernutrition, infection and immune function." Nutrition Research Reviews **13**: 3-19.
Calvin, J., G. Neale, et al. (1988). "The relative merits of acute phase proteins in the recognition of inflammatory conditions." Annals of Clinical Biochemistry.
Campbell, D. I., M. Elia, et al. (2003). "Growth faltering in rural Gambian infants is associated with impaired small intestinal barrier function, leading to endotoxemia and systemic inflammation." Journal of Nutrition **133**(5): 1332-1338.
Campbell, D. I., P. G. Lunn, et al. (2002). "Age-related association of small intestinal mucosal enteropathy with nutritional status in rural Gambian children." British Journal of Nutrition **88**(5): 499-505.
Campbell, M., R. Fitzpatrick, et al. (2000). "Framework for design and evaluation of complex interventions to improve health." British Medical Journal **321**: 694-696.
Cartwright, M. A. Lauritsen, et al. (1946). "The anemia of infection. I. Hypoferremia, hypercupremia, and alterations in porphyrin metabolism in patients." Journal of Clinical Investigation **25**(1): 65-80.
Casewell, M. and J. Phillips (1977). "Hands as a route for transmission for *Klebsiella species*." British Medical Journal **2**: 1315-1317.
Castillo-Duran, C., P. Vial, et al. (1988). "Trace mineral balance during acute diarrhea in infants." The Journal of Pediatrics **113**(3): 452-457.
Cave, B. and V. Curtis (1999). Effectiveness of promotional techniques in environmental health. WELL Study Task No. 165.
Central Intelligence Agency. (2009). "The World Factbook." Retrieved 3rd April, 2009, from https://www.cia.gov/library/publications/the-world-factbook/.
Checkley, W., L. D. Epstein, et al. (2003). "Effects of acute diarrhoea on linear growth in Peruvian children." American Journal of Epidemiology **157**(2): 166-175.
Checkley, W., R. H. Gilman, et al. (1997). "Asymptomatic and symptomatic Cryptosporidiosis: Their acute effect on weight gain in Peruvian children." American Journal of Epidemiology **145**(2): 156-163.
Chen, L. C., E. Huq, et al. (1981). "A prospective study of the risk of diarrheal diseases according to the nutritional status of childrenA " American Journal of Epidemiology **114**(2): 284-292.
Condon-Paoloni, D., J. Cravioto, et al. (1977). "Morbidity and growth of infants and young children in a rural Mexican village." American Journal of Public Health **67**(7): 651-656.
Conner, M. and P. Norman, Eds. (2005). Predicting health behaviour : Research and practice with social cognition models. Maidenhead, Open University Press.

Cornia, G. (2004). Inequity, growth and poverty: An overview of changes over the last two decades. Inequity, growth and poverty in an era of liberalization and globalization. G. Cornia. New York, Oxford University Press**:** 3-25.

Cornwall, A. and R. Jewkes (1995). "What is participatory research?" Social Science & Medicine **41**(12): 1667-1676.

Costello, A., M. Abbas, et al. (2009). "Managing the health effects of climate change: Lancet and University College London Institute for Global Health Commission." The Lancet **373**(9676): 1693-1733.

Costello, A., V. Filippi, et al. (2007). "Research challenges to improve maternal and child survival." The Lancet **369**(9569): 1240-1243.

Cousens, S., B. Kanki, et al. (1996). "Reactivity and repeatability of hygiene behaviour: structured observations from Burkina Faso." Social Science & Medicine **43**(9): 1299-1308.

CSDH (2008). Glosing the gap in a generation: health equity through action on the social determinants of health. Final report of the Commission on Social Determinants of Health. Geneva, World Health Organisation.

Curtis, V. (2001). "Hygiene: How myths, monsters and mothers-in-law can promote behaviour change." Journal of Infection **43**: 75-79.

Curtis, V., A. Biran, et al. (2003). "Hygiene in the home: relating bugs and behaviour." Social Science & Medicine **57**(4): 657-672.

Curtis, V. and S. Cairncross (2003). "Effect of washing hands with soap on diarrhoea risk in the community: a systematic review." The Lancet Infectious Diseases **3**(5): 275-281.

Curtis, V., S. Cairncross, et al. (2000). "Domestic Hygiene and Diarrhoea - Pinpointing the Problem." Tropical Medicine and International Health **5**(1): 23-32.

Curtis, V., S. Cousens, et al. (1993). "Structured observations of hygiene behaviours in Burkina Faso: validity, variability and utility." Bulletin of the World Health Organization **71**(1): 23-32.

Curtis, V., B. Kanki, et al. (2001). "Evidence of behaviour change following a hygiene promotion programme in Burkina Faso." Bulletin of the World Health Organization **79**(6): 518--527.

Curtis, V., B. Scott, et al. (2005). The Hand-washing Handbook. Washington, The World Bank.

Darboe, M. K., D. I. Thurnham, et al. (2007). "Effectiveness of an early supplementation scheme of high-dose vitamin A versus standard WHO protocol in Gambian mothers and infants: a randomised controlled trial." The Lancet **369**(9579): 2088-2096.

Davidson, K., M. Goldstein, et al. (2003). "Evidence-based medicine: What is it and how do we achieve it?" Annals of Behavioural Medicine **26**(3): 161-171.

de Koning, K. and M. Martin, Eds. (1996). Participatory research in health : Issues and experiences. London; Atlantic Highlands, N.J., Zed Books.

de Onis, M., E. Frongillo, et al. (2000). "Is malnutrition declining? An analysis of changes in levels of child malnutrition since 1980." Bulletin of the World Health Organisation **78**: 1222-1233.

de Zoysa, I., M. Rea, et al. (1991). "Why promote breastfeeding in diarrhoeal disease control programmes?" Health Policy and Planning **6**(4): 371-379.

DFID. (1999). "Sustainable livelihoods guidance sheets." Retrieved 12 August, 2009, from http://www.eldis.org/vfile/upload/1/document/0901/section2.pdf.

Duggan, M. B., J. Alwar, et al. (1986). "The nutritional cost of measles in Africa." Archives of Diseases in Childhood **61**(1): 61-66.
EAWAG. (2009). "SODIS: Solar water disinfection." Retrieved 4th Sep, 2009, from http://www.sodis.ch/index.htm.
Eisenberg, J. N. S., J. C. Scott, et al. (2007). "Integrating Disease Control Strategies: Balancing Water Sanitation and Hygiene Interventions to Reduce Diarrheal Disease Burden." American Journal of Public Health **97**(5): 846-852.
Engle, P. (1991). "Maternal work and child care strategies: Nutritional effects." Child Development **62**: 954-965.
Esrey, S. (1996). "Water, Waste and Well-Being: A Multicountry Study." American Journal of Epidemiology **143**(6): 602-623.
Esrey, S. and R. Feachem (1989). Interventions for the control of diarrhoeal diseases among young children: promotion of food hygiene. Geneva, WHO.
Esrey, S., J. Potash, et al. (1991). "Effects of improved water supply and sanitation on ascariasis, diarrhoea, dracunculiasis, hookworm infection, schistosomiasis and trachoma." Bulletin of the World Health Organisation **69**(5): 609-615.
Farthing, M. (1984). Giardiasis: Pathogenesis of chronic diarrhea and impact on child growth and development. Chronic diarrhea in children. E. Lebenthal. New York, USA, Vevy/Rasven Press**:** 253-265.
Feachem, R. (1984). "Interventions for the control of diarrhoeal diseases among young children: promotion of personal and domestic hygiene." Bulletin of the World Health Organisation **61**(3): 4767-4776.
Feachem, R. and M. Koblinsky (1984). "Interventions for the control of diarroeal diseases among young children: promotion of breastfeeding." Bulletin of the World Health Organisation **62**: 271-291.
Fewtrell, L., R. Rachel B Kaufmann, et al. (2005). "Water, sanitation, and hygiene interventions to reduce diarrhoea in less developed countries: a systematic review and meta-analysis." Lancet Infectious Diseases **5**: 42-52.
Field, A. (2005). Discovering statistics using SPSS. London, Sage Publishing.
Fishbein, M. (1996). "Great expectations, or do we ask too much from community-level interventions?" American Journal of Public Health **86**(8): 1075-1076.
Fishbein, M. (2000). "The role of theory in HIV prevention." AIDS Care **12**(3): 273-278.
Fishbein, M. and I. Ajzen (1975). Belief, Attitude, Intention and Behaviour: An Introduction to Theory and Research. Reading, MA, Addison-Wesley.
Fishbein, M. and M. Yzer (2003). "Using Theory to Design Effective Health Behavior Interventions." Communication Theory **13**(2): 164-183.
Fleck, A. (1989). "Clinical and nutritional aspects of changes in acute-phase proteins during inflammation." Proceedings of the Nutrition Society **48**(03): 347-354.
Fournier, T., N. Medjoubi-N, et al. (2000). "Alpha-1-acid glycoprotein." Biochimica et Biophysica Acta (BBA) - Protein Structure and Molecular Enzymology **1482**(1-2): 157-171.
Freedman, L. (1995). "Reflections on emerging frameworks of health and human rights." Health and Human Rights **1**(4): 314-348.
Fry, S., B. Cousens, et al. (2002). Health of children living in urban slums in Asia and the Near East, Environmental Health Project Activity Report #109.

Fuhrman, M. (2002). "The albumin-nutrition connection: Separating myth from fact." Nutrition **18**: 199-200.

Galavotti, C., K. Pappas-DeLuca, et al. (2001). "Modeling and reinforcement to combat HIV: The MARCH approach to behavior change." American Journal of Public Health **91**(10): 1601-1607.

Gallagher, K. (1992). "Squatting in the Kathmandu Valley: A historical perspective." Contributions to Nepalese Studies **19**(2): 249-259.

Gallant, M. and E. Maticka-Tyndale (2004). "School-based HIV prevention programmes for African youth." Social Science & Medicine **58**: 1337-1351.

Gilman, R. H. and P. Skillicorn (1985). "Boiling of drinking-water: Can a fuel-scarce community afford it?" Bulletin of the World Health Organisation **63**: 157-163.

Girou, E., S. Loyeau, et al. (2002). "Efficacy of handrubbing with alcohol based solution versus standard handwashing with antiseptic soap: randomised clinical trial." British Medical Journal **325**(7360): 362-.

Glasgow, R., L. Klesges, et al. (2004). "The future of health behavior change research: What is needed to improve translation of research into health promotion practice?" Annals of Behavioral Medicine **27**(1): 3-12.

Golden, M. (1994). "Is complete catch-up growth possible for stunted malnourished children?" European Journal of Clinical Nutrition **48**(Suppl.1): S58-71.

Goodman, A. and T. Leatherman, Eds. (1998). Building a new biocultural synthesis : political-economic perspectives on human biology. Ann Arbor University of Michigan Press.

Gordon, J., M. Gúzman, et al. (1964). "Acute diarrhoeal disease in less developed countries." Bulletin of the World Health Organisation **31**: 9-20.

Goto, K., F. Chew, et al. (1999). "Epidemiology of altered intestinal permeability to lactulose and mannitol in Guatemalan infants." Journal of Pediatric Gastroenterology and Nutrition **28**(3): 282-290.

Goto, R. (2006). Effects of anti-*Giardia* and antihelminthic treatment on infant nutritional and biochemical status and inestinal permeability in rural Bangladesh. Department of Biological Anthropology. Cambridge, University of Cambridge. **PhD thesis**.

Goto, R., C. Mascie-Taylor, et al. (2009). "Impact of anti-Giardia and antihelminthic treatment on infant growth and intestinal permeability in rural Bangladesh: a randomised double-blind controlled study." Transactions of the Royal Society of Tropical Medicine and Hygiene **103**: 520-529.

Goto, R., C. Panter-Brick, et al. (2002). "Poor intestinal permeability in mildly stunted Nepali children: associations with weaning practices and Giardia lamblia infection." British Journal of Nutrition **88**(2): 141-149.

Grantham-McGregor, S. (1995). "A Review of Studies of the Effect of Severe Malnutrition on Mental Development." Journal of Nutrition **125**(8 Supplement): 22380-22385.

Graystone, J. (1968). Creatinine excretion during growth. Human Growth: Body composition cell growth, energy and intelligence. D. Cheek. Philadelphia, Lea and Febiger**:** 182-197.

Habicht, J.-P., J. DaVanzo, et al. (1988). "Mother's milk and sewage: Their interactive effects on infant mortality." Pediatrics **81**(3): 456-461.

Haddad, L., M. T. Ruel, et al. (1999). "Are urban poverty and undernutrition growing? Some newly assembled evidence." World Development **27**(11): 1891-1904.

Haggerty, P., K. Muladi, et al. (1994). "Community-based hygiene education to reduce diarrhoeal disease in rural Zaire: impact of the intervention on diarrhoeal morbidity." International Journal of Epidemiology **23**(5): 1050-1059.

Han, A. and T. Hlaing (1989). "Prevention of diarrhoea and dysentery by hand-washing." Transactions of the Royal Society of Tropical Medicine **83**(1): 128-131.

Han, A., K. Oo, et al. (1986). "Personal toilet after defecation and the degree of hand contamination according to different methods used." Journal of Tropical Medicine and Hygiene **89**: 237-241.

Hautvast, J., J. Tolboom, et al. (2000). "Consequences of infections for three-month length increment in young children in rural Zambia." Acta Paediatrica **89**(3): 296-301.

Hawe, P. (2000). "How much trial and error should we tolerate in community trials." British Medical Journal **320**(7227): 119.

Henry, F., N. Alam, et al. (1987). "Dysentery, not watery diarrhoea, is associated with stunting in Bangladeshi children." Human Nutrition, Clinical Nutrition **41**(4): 243-249.

Hernandez, P., A. Zettna, et al. (1996). "Childcare needs of female street vendors in Mexico City." Health Policy and Planning **11**(2): 169-178.

Hicks, J. and R. Boeckx (1984). Pediatric Clinical Chemistry. Philadelphia, WB Saunders Company.

Higginbotham, N., R. Briceno-Leon, et al. (2001). Applying Health Social Science: Best Practice in the Developing World. London, New York, Zed Books.

Hoare, S., S. D. Poppitt, et al. (1996). "Dietary supplementation and rapid catch-up growth after acute diarrhoea in childhood." British Journal of Nutrition **76**(04): 479-490.

Hudelson, P., T. Huanca, et al. (1995). "Ethnographic studies of ARI in Bolivia and their use by the national ARI programme." Social Science & Medicine **41**(12): 1677-1683.

Hutchinson, R. (1956). "Some observations on the method of spread of Sonne dysentery." Monthly Bulletin of the Ministry of Health and the Public Health Laboratory Service **15**: 110-118.

Huttly, S., S. Morris, et al. (1997). "Prevention of diarrhoea in young children in developing countries." Bulletin of the World Health Organisation **75**(2): 163-174.

Hutton, G. and L. Haller (2004). Evaluation of the costs and benefits of water and sanitation improvements at the global level. Geneva, WHO.

Internal Displacement Monitoring Centre (2008). Nepal: Sustainability of IDP returns undermined by lack of assistance Geneva, Norwegian Refugee Council.

Isaacs, D., H. Dickson, et al. (1991). "Handwashing and cohorting in prevention of hospital acquired infections with respiratory syncytial virus." Archives of Diseases in Childhood **66**(2): 227-231.

James, J. W. (1972). "Longitudinal study of the morbidity of diarrheal and respiratory infections in malnourished children." American Journal of Clinical Nutrition **25**(7): 690-694.

Jones, G., R. W. Steketee, et al. (2003). "How many child deaths can we prevent this year?" The Lancet **362**(9377): 65-71.

Jongpiputvanich, S., S. Veeravongs, et al. (1998). "Difficulties in conducting participatory action research to prevent diarrhoea in a slum area of Bankok." Journal of Diarrhoeal Disease Research **16**(3): 187-193.

Joshi, D. and M. Maharjan (2003). Urban ecosystems and health in Kathmandu: Community-based biological assessment of drinking water sources. Third annual conference on

environment and health, Chennai, India, Department of Geography, University of Madras and Faculty of Environmental Studies, York University.

Kac, G., I. Podglajen, et al. (2005). "Microbiological evaluation of two hand hygiene procedures achieved by healthcare workers during routine patient care: a randomized study." Journal of Hospital Infection **60**(1): 32-39.

Kaltenthaler, E. and J. Pinfold (1995). "Microbiological methods for assessing handwashing practice in hygiene behaviour studies." Journal of Tropical Medicine and Hygiene **98**: 101-106.

Kapur, D., et al (2005). "Dietry Intake and Growth Pattern of Children 9-36 Months of Age in an Urban Slum in Delhi." Indian Pediatrics **42**: 351-356.

Katz, A. (2004). "The Sachs Report: *Investing in Health for Economic Development* - or increasing the size of the crumbs from the rich man's table? Part I." International Journal of Health Services **34**(4): 751-773.

Katz, A. (2008). "'New Global Health': A reversal of logic, history and principles." Social Medicine **3**(1): 1-3.

Kayaga, S. (2005). Emergency treatment of drinking water at point of use.Technical Notes for Emergencies #5. Prepared by Water Engineering and Development Centre - Loughborough University. Geneva, WHO.

Khan, M. (1982). "Interruption of shigellosis by hand washing." Transactions of the Royal Society of Tropical Medicine and Hygiene **76**(2): 164-168.

Kirkwood, B., S. Cousens, et al. (1997). "Issues in the design and interpretation of studies to evaluate the impact of community-based interventions." Tropical Medicine and International Health **2**(11): 1022-1029.

Kosek, M., C. Bern, et al. (2003). "The global burden of diarrhoeal disease, as estimated from studies published between 1992 and 2000." Bulletin of the World Health Organisation **81**: 197-204.

Kossmann, J., P. Nestel, et al. (2000). "Undernutrition and childhood infections: a prospective study of childhood infections in relation to growth in the Sudan." Acta Paediatrica **89**(9): 1122-1128.

Krieger, N. (1999). "Questioning epidemiology: objectivity, advocacy, and socially responsible science." American Journal of Public Health **89**(8): 1151-1153.

Kukuruzovic, R. H., A. Haase, et al. (1999). "Intestinal permeability and diarrhoeal disease in Aboriginal Australians." Archives of Diseases in Childhood **81**(4): 304-308.

Lambert, H., E. J. Gordon, et al. (2006). "Introduction: Gift horse or Trojan horse? Social science perspectives on evidence-based health care." Social Science & Medicine **62**(11): 2613-2620.

Lamontagne, J. F., P. L. Engle, et al. (1998). "Maternal employment, child care, and nutritional status of 12-18 month-old children in Managua, Nicaragua." Social Science & Medicine **46**(3): 403-414.

Larson, E. L., S. X. Lin, et al. (2004). "Effect of Antibacterial Home Cleaning and Handwashing Products on Infectious Disease Symptoms: A Randomized, Double-Blind Trial." Annals of Internal Medicine **140**(5): 321-329.

Laurell, C. (1985). Acute phase proteins - a group of protective proteins. Recent Advances in Clinical Biochemistry. C. Price and K. Alberti. New York, Churchill Livingstone**:** 103-124.

Leroy, J. L., J.-P. Habicht, et al. (2007). "Current priorities in health research funding and lack of impact on the number of child deaths per year." American Journal of Public Health **97**(2): 219-223.

Leslie, J. (1989). Women's work and child nutrition in the Third World. Women, Work and Child Welfare in the Third World. J. Leslie and M. Paolisso. Boulder, Colorado, Westview Press.

Lifschitz, C. (1985). "Intestinal Permeability." Journal of Pediatric Gastroenterology and Nutrition **4**(4): 520-522.

Loevinsohn, B. (1990). "Health education Interventions in Developing Countries: A Methodological Review of Published Articles." International Journal of Epidemiology **19**(4): 788-794.

Lohman, T., A. Roche, et al. (1988). Anthropometric Standardisation Reference Manual. Champaign, Illinois, Human Kinetics Books.

Luby, S., M. Agboatwalla, et al. (2002). "The effect of antibacterial soap on impetigo incidence, Karachi, Pakistan." American Journal of Tropical Medicine & Hygiene **67**(4): 430-435.

Luby, S. P., M. Agboatwalla, et al. (2005). "Effect of handwashing on child health: a randomised controlled trial." The Lancet **366**(9481): 225-233.

Luby, S. P., M. Agboatwalla, et al. (2004). "Effect of Intensive Handwashing Promotion on Childhood Diarrhea in High-Risk Communities in Pakistan: A Randomized Controlled Trial." Journal of the American Medical Association **291**(21): 2547-2554.

Lumanti (2001). A situation analysis or urban poor communities in Kathmandu and Lalitpur. Kathmandu, Lumanti Support Group for Shelter.

Lunn, P. (2000). "The impact of infection and nutrition on gut function and growth in childhood." Proceedings of the Nutrition Society **59**: 147-154.

Lunn, P. G., C. A. Northrop-Clewes, et al. (1991). "2. Chronic diarrhoea and malnutrition in The Gambia: studies on intestinal permeability." Transactions of the Royal Society of Tropical Medicine and Hygiene **85**(1): 8-11.

Lutter, C., J. Mora, et al. (1989). "Nutritional supplementation: effects on child stunting because of diarrhea." American Journal of Clinical Nutrition **50**(1): 1-8.

Mackenbach, J. P. (2007). "Sanitation: pragmatism works." British Medical Journal **334**(suppl_1): s17-.

Maharjan, M. and A. Sharma (1999). "Quality of groundwater and its impact on the public health in urban Patan." Tribhuvan University Journal **xxii**(2): 1-9.

Maharjan, M. and A. Sharma (2000). "Bacteriological quality of groundwater in urban Patan and antibiotic sensitivity against isolated enteric bacteria." Journal of Nepal Medical Association **39**(268-274).

Makris, A., L. Morgan, et al. (2000). "Effect of a comprehensive infection control program on the incidence of infections in long-term care facilities." American Journal of Infection Control **28**(1): 3-7.

Martorell, R., J. P. Habicht, et al. (1975). "Acute morbidity and physical growth in rural Guatemalan children." American Journal of Diseases in Childhood **129**(11): 1296-1301.

Martorell, R. and T. Ho (1984). "Malnutrition, morbidity and mortality." Population and Development review **10**(Supplement): 49-68.

Martorell, R., J. Rivera, et al. (1990). "Consequences of stunting in early childhood for adult body size in rural Guatemala." Ann. Nestle **48**: 85-92.

Martorell, R., J. Rivera, et al. (1992). Long-term consequences of growth retardation during early childhood. Human Growth: Basic and Clinical Aspects. M. Hernandez and J. Argente. Amsterdam, Elsevier Science Publishers.

Mata, L., R. Kromal, et al. (1977). "Effect of infection on food intake and the nutritional state: perspectives as viewed from the village." Am J Clin Nutr **30**(8): 1215-1227.

McCullough, F. S. W., C. A. Northrop-Clewes, et al. (1999). "The effect of vitamin A on epithelial integrity." Proceedings of the Nutrition Society **58**(02): 289-293.

McLennan, J. D. (2000). "To boil or not to boil: drinking water for children in a periuban barrio." Social Science & Medicine **51**: 1211-1220.

Meites, S. (1989). Pedicatric clinical chemistry: reference (normal) values. Washington, AACC Press.

Mendez, M. (1999). "Severity and timing of stunting in the first two years of life affect performance in cognitive tests in late childhood." Journal of Nutrition **129**: 1555-1562.

Merzel, C. and D'Afflitti (2003). "Reconsidering community-based health promotion: promise, performance, and potential." Public Health Matters **93**(4): 557-574.

Mills, J., S. PH, et al. (1986). "Early growth predicts timing of puberty in boys: results of a 14-year nutrition and growth study." Journal of Pediatrics **109**: 543-547.

Mintz, E., F. Reiff, et al. (1995). "Safe water treatment and storage in the home: A practical new strategy to prevent waterborne disease." Journal of the American Medical Association **273**: 948-953.

Moffat, T. (2001). Growth stunting among children, aged birth to five years, in peri-urban Kathmandu, Nepal. Perspectives in Human Growth, Development and Maturation. P. Dasgupta and R. Hauspie. Dordrecht, Kluwer Academic Publishers**:** 251-268.

Moffat, T. (2003). "Diarrhea, Respiratory Infections, Protozoan Gastrointestinal Parasites, and Child Growth in Kathmandu, Nepal." American Journal of Physical Anthropology **122**: 85-97.

MOHP, Ministry of Health and Population (MOHP) [Nepal], et al. (2007). *Nepal Demographic and Health Survey 2006*. Kathmandu, Nepal, Ministry of Health and Population, New ERA, and Macro International Inc.

Molla, A., A. Molla, et al. (1983). Effects of acute diarhoea on absorption of macronutrients during disease and recovery. Diarrhea and Malnutrition: Interactions, Mechanisms and Interventions. L. Chen and N. Scrimshaw. New York, Plenum Press**:** 143-154.

Molla, A., A. Molla, et al. (1983). Food intake during and after recovery from diarrhoea in children. Diarrhea and Malnutrition: Interactions, Mechanisms and Interventions. L. Chen and N. Scrimshaw. New York, Plenum Press**:** 113-123.

Moore, S., A. Lima, et al. (2001). "Early childhood diarrhoea and helminthiases associate with long-term linear growth faltering." International Journal of Epidemiology **30**(6): 1457-1464.

Morrison, J., S. Tamang, et al. (2005). "Women's health groups to improve perinatal care in rural Nepal." BMC Pregnancy and Childbirth **5**(1): 12.

Motaryemi, Y., F. Kaferstein, et al. (1993). "Contaminated weaning food: a major risk factor for diarrhoea and associated malnutrition." Bulletin of the World Health Organization **71**(1): 79-92.

Moy, R. J. D., T. F. Marshall, et al. (1994). "Diarrhoea and growth faltering in rural Zimbabwe." European Journal of Clinical Nutrition **48**(11): 810-821.

Naikoba, S. and A. Hayward (2001). "The effectiveness of interventions aimed at increasing handwashing in healthcare workers - a systematic review." Journal of Hospital Infection **47**(3): 173-180.

Neggers, Y., R. L. Goldenberg, et al. (1995). "The Relationship Between Maternal and Neonatal Anthropometric Measurements in Term Newborns." Obstetrics & Gynecology **85**(2): 192-196.

Nichter, M. (1996). Drink boiled cooled water: A cultural analysis of a health education message. Anthropology and International Health: Asian Case Studies. M. Nichter and M. Nichter. Amsterdam, Gordon & Breach**:** 393-400.

Niffenegger, J. P. (1997). "Proper handwashing promotes wellness in child care." Journal of Pediatric Health Care **11**(1): 26-31.

Norgan, N. (2000). "Long-term physiological and economic consequences of growth retardation in children and adolescents." Proceedings of the Nutrition Society **59**: 245-265.

Northrop-Clewes, C., P. Lunn, et al. (1997). " Lactose maldigestion in breast-feeding Gambian infants." Journal of Pediatric Gastroenterology and Nutrition **24**: 257-263.

Northrop-Clewes, C. A., E. K. Rousham, et al. (2001). "Anthelmintic treatment of rural Bangladeshi children: effect on host physiology, growth, and biochemical status." American Journal of Clinical Nutrition **73**(1): 53-60.

Northrop, C., P. Lunn, et al. (1990). "Automated enzymatic assays for the determination of intestinal permeability probes in urine. 1. Lactulose and lactose." Clinica Chimica Acta **187**: 79-88.

Oberhelman, R., et al. (1998). "Correlations between intestinal parasitosis, physical growth, and psychomotor development among infants and children from rural Nicaragua." American Journal of Tropical Medicine & Hygiene **58**(4): 470-475.

Ono, K., S. Rai, et al. (2001). "Seasonal distribution of enteropathogens detected from diarrheal stool and water samples collected in Kathmandu, Nepal." Southeast Asian Journal of Tropical Medicine & Public Health **32**(3): 520-526.

Ooi, G. and K. Phua (2007). "Urbanization and slum formation." Journal of Urban Health **84**(1): i27-i34.

Pandey, M., K. Smith, et al. (1989). "Indoor air pollution in developing countries and acute respiratory infection in children." The Lancet **Feb 25 1**(8635): 427-428.

Panter-Brick, C., S. E. Clarke, et al. (2006). "Culturally compelling strategies for behaviour change: A social ecology model and case study in malaria prevention." **62**(11): 2810-2825.

Panter-Brick, C., P. Lunn, et al. (2001). "Elevated acute-phase protein in stunted Nepali children reporting low morbidity: different rural and urban profiles." British Journal of Nutrition **85**: 125-131.

Panter-Brick, C., P. G. Lunn, et al. (2009). "Pathways leading to early growth faltering: an investigation into the importance of mucosal damage and immunostimulation in different socio-economic groups in Nepal." British Journal of Nutrition **101**(04): 558-567.

Payne, G. (2005). "Getting ahead of the game: A twin-track approach to improving existing slums and reducing the need for future slums." Environment and Urbanization **17**(1): 135-146.

Peat, J. and J. Dickerson (1998). "Effects of damp and mould in the home on respiratory health: a review of the literature." Allergy **53**(2): 120-128.

Peters, W. and H. Gilles (1995). Tropical Medicine and Parasitology. London, Mosby-Wolfe.

Peterson, F. A., L. Roberts, et al. (1998). "The effect of soap distribution on diarrhoea: Nyamithuthu Refugee Camp." International Journal of Epidemiology **27**(3): 520-524.

Pether, J. and R. Gilbert (1971). "The survival of salmonellas on finger-tips and transfer of the organisms to food." Journal of Hygiene **69**: 673-681.

Picheansathian, W. (2004). "A systematic review on the effectiveness of alcohol-based solutions for hand hygiene " International Journal of Nursing Practice **10**(1): 3-9(7).

Pinfold, J. and N. Horan (1996). "Measuring the effect of a hygiene behaviour intervention by indicators of behaviour and diarrhoeal disease." Transactions of the Royal Society of Tropical Medicine and Hygiene **90**: 366-371.

Pokhrel, D. and T. Viraraghavan (2004). "Diarrhoeal diseases in Nepal *vis-a-vis* water supply and sanitation status." Journal of Water and Health **2**(71-81).

Poskitt, E., et al. (1999). "Less diarrhoea but no change in growth: 15 years' data from three Gambian villages." Archives of Diseases in Childhood **80**: 115-120.

Powe, B. (1996). "Cancer fatalism among African-Americans: A review of the literature." Nursing Outlook **44**(1): 18-21.

Pradhan, P. (2004). Population growth, migration and urbanisation. Environmental Change and its Implications for Population Migration. J. Unruh, M. Krol and N. Kliot. Dordrecht, Boston, London, Kluwer Academic Publishers.

Prentice, A. M. (1993). "Nutrient requirements for growth, pregnancy and lactation: The Keneba experience." South Africa Journal of Clinical Nutrition **6**(3): 33-38.

Price, P. (1938). "The bacteriology of normal skin: a new quantitative test applied to a study of the bacterial flora and the disinfectant action of mechanical cleansing." Journal of Infectious Diseases **63**: 301-318.

Prochaska, J. and C. DiClemente (1983). "Stages and processes of self-change of smoking: Towards an integrative model of change." Journal of Consulting and Clinical Psychology **51**: 390-395.

Prochaska, J., C. DiClemente, et al. (1992). "In search of how people change: Applications to addictive behaviours." American Psychologist **47**: 1102-1114.

Prochaska, J. and W. Velicer (1997). "The transtheoretical model of health behaviour change." American Journal of Health Promotion **12**: 38-48.

Proos, L., Y. Hofvander, et al. (1991). "Menarcheal age and growth pattern of Indian girls adopted in Sweden II: Catch-up growth and final height." Indian Journal of Pediatrics **58**(1): 105-114.

Prüss-Üstün, A. and C. Corvalán (2006). Preventing disease through healthy environments: Towards an estimate of the environmental burden of disease. Geneva, WHO.

Rabie, T. and V. Curtis (2006). "Handwashing and risk of respiratory infections: a quantitative systematic review." Tropical Medicine & International Health **11**(3): 258-267.

Ramalingaswami, V., U. Jonsson, et al. (1996). The Asian Engima. The Progress of Nations. New York, UNICEF.

Rivera, J. A., C. Hotz, et al. (2003). "The Effect of Micronutrient Deficiencies on Child Growth: A Review of Results from Community-Based Supplementation Trials." Journal of Nutrition **133**(11): 4010S-4020.

Rogers, E. (1983). Diffusion of Innovations. New York, Free Press.

Rogers, R. (1975). "A protection motivation theory of fear appeals and attitude change." Journal of Psychology **91**: 93-114.

Rogers, R. (1983). Cognitive and physiological processes in fear appeals and attitude change: A revised theory of protection motivation. Social Psychophysiology: A Source Book. J. Cacioppo and R. Petty. New York, Guildford Press**:** 153-176.

Rosenstock, I. (1966). "Why people use health services." Milbank Memorial Fund Quarterly **44**: 94-124.

Rosenstock, I. (1974). "Historical origins of the health belief model." Health Education Monographs **2**: 1-8.

Ross, D. and P. Vaughan (1986). "Health interview surveys in developing countries: a methodological review." Studies in Family Planning **17**(2): 78-94.

Rothman, K., H. Adami, et al. (1998). "Should the mission of epidemiology include the eradication of poverty?" The Lancet **352**: 810-813.

Rothman, K. J. and C. Poole (1985). "Science and policy making." American Journal of Public Health **75**(4): 340-341.

Rousham, E. K., C. A. Northrop-Clewes, et al. (1998). "Maternal reports of child illness and the biochemical status of the child: the use of morbidity interviews in rural Bangladesh." British Journal of Nutrition **80**(5): 451-456.

Rowland, M. G. M., T. J. Cole, et al. (1977). "A quantitative study into the role of infection in determining nutritional status in Gambian village children." British Journal of Nutrition **37**(03): 441-450.

Rowland, M. G. M., S. Rowland, et al. (1988). "Impact of infection on the growth of children from 0 to 2 years in an urban West African community." American Journal of Clinical Nutrition **47**: 134-138.

Roy, S., R. Behrens, et al. (1992). "Impact of zinc supplementation on intestinal permeability in Bangladeshi children with acute diarrhoea and persistent diarrhoea syndrome." Journal of Pediatric Gastroenterology and Nutrition **15**: 289-296.

Rudan, I., S. El Arifeen, et al. (2007). "Childhood pneumonia and diarrhoea: setting our priorities right." The Lancet Infectious Diseases **7**(1): 56-61.

Ruel, M., K. Brown, et al. (2003). Moving forward with complementaru feeding: Indicators and research priorities. Food Consumption and Nutrition Division. Discussion Paper # 146. Washington, International Food Policy Research Institute.

Ryan, A. (1997). "Iron-Deficiency Anemia in Infant Development: Implications for Growth, Cognitive Development, resistance to Infection, and Iron Supplementation." Yearbook of Physical Anthropology **40**: 25-62.

Ryan, M., R. Christian, et al. (2001). "Handwashing and rerspiratory illness among young adults in military training." American Journal of Preventive Medicine **21**(2): 79-83.

Rychetnik, L., M. Frommer, et al. (2002). "Criteria for evaluating evidence on public health interventions." Journal of Epidemiology and Community Health **56**: 119-127.

Satterthwaite, D. (2003). "The Millennium Development Goals and urban poverty reduction: great expectations and nonsense statistics." Environment and Urbanization **15**(2): 179-190.

Satyanarayana, K., K. Prasanna, et al. (1986). "Effect of early childhood undernutrition and child labour on growth and adult nutritional status of rural Indian boys around Hyderabad." Human Nutrition, Clinical Nutrition **40**: 131-139.

Savitz, D. A., C. Poole, et al. (1999). "Reassessing the role of epidemiology in public health." American Journal of Public Health **89**(8): 1158-1161.

Schilling, P. (1990). "Supplementary feeding programs: a critical analysis " Rev. Saude Publica **24**(5): 412-419.

Schwartz, C., M. Chesney, et al. (1997). "The control group dilemma in clinical research: applications for psychosocial and behavioral medicine trials." Psychosomatic Medicine **59**(4): 362-371.

Sclar, E. D., P. Garau, et al. (2005). "The 21st century health challenge of slums and cities." The Lancet **365**(9462): 901-903.

Scrimshaw, N. and J. SanGiovanni (1997). "Synergism of nutrition, infection, and immunity: an overview." American Journal of Clinical Nutrition **66**(2): 464S-477.

Scrimshaw, N., C. Taylor, et al. (1968). Interactions of nutrition and infection. Geneva, World Health Organization.

Sengupta, U. and S. Sharma (2006). "The challenge of squatter settlements in Kathmandu: Addressing a policy vacuum." International Development Planning Review **28**(1): 105-126.

Sepúlveda, A., W. Willet, et al. (1988). "Malnutrition and diarrhea: A longitudinal study among urban Mexican children." American Journal of Epidemiology **127**(2): 365-376.

Shahid, N. S., W. Greenough, et al. (1996). "Hand washing with soap reduces diarrhoea and spread of bacterial pathogens in a Bangladesh village." Journal of Diarrhoeal Disease Research **14**(2): 85-89.

Sharma, P. (2002). "Nepal paediatric society guidelines for childhood immunization." Journal of Nepal Medical Association **41**(143): 438-440.

Shiffman, J. (2006). "Donor funding priorities for communicable disease control in the developing world." Health Policy and Planning **21**(6): 411-420.

Shrestha, B. and K. Shrestha (2005). Study on Water Accessibility of Slums and Squatters of Kathmandu Valley. Kathmandu, NGO Forum.

Shrestha, R. (No date). Point-of-use drinking water treatment initiatives in Nepal. Kathmandu, Nepal, Environment and Public Health Office.

Shrimpton, R., C. Victora, et al. (2001). "Worldwide timing of growth faltering: implications for nutritional interventions." Pediatrics **107**(5): e75.

Singer, M. (1995). "Beyond the ivory tower: Critical praxis in Medical Anthropology." Medical Anthropology Quarterly **9**(1): 80-106.

Singer, M. and H. Baer, Eds. (1995). Critical Medical Anthropology. Amityville, New York, Baywood Publishing Company.

Sircar, B., P. Sengupta, et al. (1987). "Effect of handwashing on the incidence of diarrhoea in a Calcutta slum." Journal of Diarrhoeal Disease Research` **5**(2): 112-114.

Sivakumar, B. and V. Reddy (1975). "Absorption of vitamin A in children with ascariasis." Journal of Tropical Medicine and Hygiene **78**(5): 114-115.

Smith, K. R., J. M. Samet, et al. (2000). "Indoor air pollution in developing countries and acute lower respiratory infections in children." Thorax **55**(6): 518-532.

Smith, P., M. Moffat, et al. (1997). "Are community health interventions evaluated appropriately? A review of six journals." Journal of Clinical Epidemiology **50**(2): 137-146.

Sprunt, K., W. Redman, et al. (1973). "Antibacterial effectiveness of routine hand washing." Pediatrics **52**(2): 264-271.

St Sauver, J., M. Khurana, et al. (1998). "Hygienic practices and acute respiratory illness in family and group day care homes." Public Health Reports **113**: 554-551.

Stanton, B., J. Clemens, et al. (1987). "Twenty-four house recall, knowledge-attitude-practice questionnaires, and direct observations of sanutary practices: a comparative study." Bulletin of the World Health Organization **65**(2): 217-222.

Stanton, B. F. and J. D. Clemens (1987). "An educational intervention for altering water-sanitation behaviors to reduce childhood diarrhea in urban bangladesh: II. A randomized trial to assess the impact of the intervention on hygienic behaviors and rates of diarrhea." Am. J. Epidemiol. **125**(2): 292-301.

Stephenson, C. (1999). "Burden of infection on growth failure." Journal of Nutrition **129**(2S): 534S-538S.

Stephenson, J. and J. Imrie (1998). "Why do we need randomised controlled trials to assess behavioural interventions?" British Medical Journal **316**: 611-613.

Sullivan, P., P. G. Lunn, et al. (1992). "Persistent diarrhea and malnutrition - the impact of treatment on small bowel structure and permeability." Journal of Pediatric Gastroenterology and Nutrition **14**: 208-215.

Tabachnick, B. and L. Fidell (1996). Using Multivariate Statistics. Oxford, Harper Collins College Publishers.

Tanaka, M. (2009). "From confrontation to collaboration: a decade in the work of the squatters' movement in Nepal." Environment and Urbanization **21**(1): 143-159.

Thurnham, D. I., C. Northrop-Clewes, et al. (2000). "Innate immunity, gut integrity, and vitamin A in Gambian and Indian infants." The Journal of Infectious Diseases **182**(Supplement 1): S23-28.

Timaeus, I. and L. Lush (1995). "Intra-urban differentials in child health." Health Transition Review **5**: 163-190.

Tomkins, A. (1981). "Nutritional status and severity of diarrhoea among pre-school children in rural Nigeria." The Lancet **1**(8225): 860-862.

Tomkins, A. (1988). The risk of morbidity in a stunted child. Linear growth retardation in less developed countries. J. Waterlow. New York, Raven Press. **14:** 185-195

Torres, A., K. Peterson, et al. (2000). "Association of diarrhoea and upper respiratory infections with weight and height gains in Bangladeshi children aged 5 to 11 years." Bulletin of the World Health Organisation **78**(11): 1316-1323.

Ulijaszek, S. J. and D. A. Kerr (1999). "Anthropometric measurement error and the assessment of nutritional status." British Journal of Nutrition **82**(03): 165-177.

UN-Habitat (2003). The challenge of slums: Global report on human settlements 2003. London and Sterling, VA.

UNESCO Institute for Statistics. (2006). "Nepal General Profile." Retrieved 9th January, 2009, from http://stats.uis.unesco.org/unesco/TableViewer/document.aspx?ReportId=124&IF_Language=eng&BR_Country=5240&BR_Region=40535.

UNFPA (2001). The state of the world's population 2001. New York, UNFPA.

UNICEF. (2006). "Water and Sanitation Factsheet." Retrieved 19th March, 2009, from http://www.unicef.org.uk/campaigns/publications/pdf/watersanitation_06.pdf.
UNICEF. (2009, Jan 2009). "UNICEF Global database on child malnutrition. Monitoring the situation of Women and Children." from www.childinfo.org.
UNICEF/WHO (2006). Pneumonia: The Forgotten Killer. New York/Geneva, UNICEF/WHO.
United Nations (2008). The Millennium Development Goals Report 2008. New York, United Nations.
United Nations. (2008). "Millennium Development Goals website." Retrieved 20 August, 2009, from http://www.un.org/millenniumgoals/.
United Nations Population Division (1999). The World at Six Billion. New York, Department of Economic and Social Affairs.
United Nations Statistics Division (2008). Statistical Annex: Millennium Development Goals, Targets and Indicators, 2008. New York, United Nations Statistics Division.
van de Merwe, L. (2006). Long-chain n-3 polyunsaturated fatty acids in relation to gut integrity and growth of rural African children. MPhil/PhD Upgrading Document., Department of Epidemiology and Population Health. Nutrition & Population Health Intervention Research Unit. LSHTM.
VanDerslice, J. and J. Briscoe (1995). "Environmental Interventions in Developing Countries: Interactions and Their Implications." Am. J. Epidemiol. **141**(2): 135-144.
Vial, I., E. Muchnik, et al. (1989). Women's market work, infant feeding practices and infant nutrition among low-income women in Santiago, Chile. Women, Work and Child Welfare in the Third World. J. Leslie and M. Paolisso. Boulder, Colorado, Westview Press.
Victora, C., F. Barros, et al. (1990). "Pneumonia, diarrhoea and growth in the first 4y of life: a longitudinal study of 5914 urban Brazilian children." American Journal of Clinical Nutrition **52**: 391-396.
Victora, C. G., K. Hanson, et al. (2004). "Achieving universal coverage with health interventions." The Lancet **364**(9444): 1541-1548.
Vlahov, D., N. Fruedenberg, et al. (2007). "Urban as a determinant of health." Journal of Urban Health **84**(1): i16 - i26.
Wagner, E. and J. Lanoix (1958). Excreta disposal for rural areas and small communities. WHO Monograph Series No. 39. Geneva, WHO.
Walker, S., S. Grantham-McGregor, et al. (1992). "Morbidity and the growth of stunted and nonstunted children, and the effect of supplementation." American Journal of Clinical Nutrition **56**(3): 504-510.
Wallstone, K. and B. Wallstone (1978). "Development of multi-dimensional health locus of control (MHLC) scales." Health Education Monographs **6**: 160-170.
Wallstone, K. and B. Wallstone (1981). Health locus of control scales. Research with the Locus of Control Construct, Vol. 1. H. Lefcourt. New York, Academic Press.
Warner, N., J. Levy, et al. (2008). "Drinking water quality in Nepal's Kathmandu Valley: a survey and assessment of selected controlling site characteristics." Hydrogeology Journal **16**(2): 321-334.
Watcher, D., M. Joshi, et al. (1999). "Antibiotic dispensing by drug retailers in Kathmandu, Nepal. ." Tropical Medicine & International Health **4**(11): 782-788.

Weisbrot, M., D. Baker, et al. (2002). "The scorecard on globalization 1980-2000: Its consequences for economic and social well-being." International Journal of Health Services **32**(2): 229-253.

White, C. G., F. S. Shinder, et al. (2001). "Reduction of Illness Absenteeism in Elementary Schools Using an Alcohol-free Instant Hand Sanitizer." The Journal of School Nursing **17**(5): 258-265.

WHO (2001). Macroeconomics and health: Investing in health for economic development. Geneva, WHO.

WHO (2005). A Billion Voices: Listening and responding to the health needs of slum dwellers and informal settlers in new urban settings. Kobe, WHO Kobe Centre.

WHO (2005). The Treatment of Diarrhoea: A Manual for Physicians and Other Senior Health Workers. Geneva, WHO Press.

WHO (2006). World Health Organization releases new child growth standards. Geneva, WHO. Press Release (April 27, 2006).

WHO (2008). Worldwide Prevalence of Anaemia 1993-2005. Geneva, WHO.

Wilson, J., G. Chandler, et al. (1991). "Hand-washing reduced diarrhoea episodes: a study in Lombok, Indonesia." Transactions of the Royal Society of Tropical Medicine **85**: 819-821.

Woodward, A. and I. Kawachi (2000). "Why reduce health inequalities?" Journal of Epidemiology and Community Health **54**(12): 923-929.

World Bank (2008). Poverty data: A supplement to the World Development Indicators 2008. Washington DC, The World Bank.

Yoon, P., R. Black, et al. (1997). "The effect of malnutrition on the risk of diarrheal and respiratory mortality in children < 2 y of age in Cebu, Philippines." American Journal of Clinical Nutrition **65**(4): 1070-1077.

Zaman, K., A. Baqui, et al. (1997). "Malnutrition, cell-mediated immune deficiency and acute upper respiratory infections in rural Bangladeshi children." Acta Paediatrica **86**(9): 923-927.

Zumrawi, F., H. Dimond, et al. (1987). "Effects of infection on growth in Sudanese children." Human Nutrition, Clinical Nutrition **41**(6): 453-461.

Appendix 1 - Structured Observations Schedule

Namaste. My name is _____ and I am working for Beki Langford who is from the UK doing research into Nepali family life and women's work. Today I am doing a survey on Nepali family life and women's work and I would like to spend some time with you and your family this morning. I would like to watch how you spend time with your family and what work you do in the mornings. I would like to come into your house and sit quietly for about three hours. You should carry on as normal as if I wasn't here. Would you be willing to take part in this observation?

Consent given? Yes

No (Thank respondent and leave for next house on list)

Section 1: Identification

1.1	Name of observer	
1.2	Area name	
1.3	Child's name	
1.4	Child's ID	
1.5	Mother's name	
1.6	Date of visit	
1.7	Arrival time	
1.8	Observation start time	
1.9	Observation complete time	

Section 2: Index child defecation

2.1	Did the index child defecate while you were present?	
	No, s/he didn't *(Go to section 3.1)*	0
	Yes, I saw	1
	Yes, I think so	2
	ONLY FILL IN NEXT SECTION IF THE INDEX CHILD DEFECATED	
2.2	What time did the child defecate?	
2.3	Where did the child defecate (first time)?	
	In a nappy/clothing	1
	On floor of yard/house	2
	In potty	3
	In toilet	4
	Other _____	5
2.4.1	Did someone clean the child's bottom?	
	Nobody *(Go to section 2.5.1)*	1
	Mother	2
	Sister	3
	Grandmother	4
	Other _____	5
2.4.2	Immediately after completing stool contact, did the person…	
	Hands not washed *(Go to section 2.5.1)*	1
	Rinse one hand with water	2
	Rinse both hands with water	3
	Wash one hand with soap	4
	Wash both hands with soap	5

	Rinse hands in soapy water	6
	Unable to see	7
2.4.3	Where did the water for the hand-washing (first person) come from?	
	From container in house/yard	1
	Laundry water	2
	A tap	3
	Rower pump/tube well	4
	Unable to see	5
2.4.4	If soap was used, where did the soap for hand-washing come from?	
	Soap not use	1
	Soap kept near to water source	2
	Soap distant to water source	3
	Unable to see	4
2.5.1	Did someone clear up the child's stools straight away?	
	Nobody *(Go to section 2.6.1)*	1
	Mother	2
	Sister	3
	Grandmother	4
	Other _____	5
2.5.2	Immediately after cleaning up the stools, did the person…	
	Hands not washed *(Go to section 3.1)*	1
	Rinse one hand with water	2
	Rinse both hands with water	3
	Wash one hand with soap	4
	Wash both hands with soap	5

	Rinse hands in soapy water	6
	Unable to see	7
2.5.3	Where did the water for the hand-washing come from?	
	From container in house/yard	1
	Laundry water	2
	A tap	3
	Rower pump/tube well	4
	Unable to see	5
2.5.4	If soap was used, where did the soap for hand-washing come from?	
	Soap not use	1
	Soap kept near to water source	2
	Soap distant to water source	3
	Unable to see	4
2.6.1	If no one cleared up stool straight away, did someone clear up the child's stools later on?	
	Nobody *(Go to section 3.1)*	1
	Mother	2
	Sister	3
	Grandmother	4
	Other _____	5
2.6.2	How long after defecation did this happen?	_____minutes
2.6.3	Immediately after cleaning up the stools, did the person…	
	Hands not washed *(Go to section 3.1)*	1
	Rinse one hand with water	2
	Rinse both hands with water	3
	Wash one hand with soap	4

	Wash both hands with soap	5
	Rinse hands in soapy water	6
	Unable to see	7
2.6.4	Where did the water for the hand-washing come from?	
	From container in house/yard	1
	Laundry water	2
	A tap	3
	Rower pump/tube well	4
	Unable to see	5
2.6.5	If soap was used, where did the soap for hand-washing come from?	
	Soap not use	1
	Soap kept near to water source	2
	Soap distant to water source	3
	Unable to see	4

Section 3: Feeding index child

3.1	Did the index child eat anything during the observation period (not breast milk)?	
	No, nothing *(go to section 4.1)*	1
	Child was fed by a carer	2
	Child fed him/herself	3
	ONLY FILL IN NEXT SECTION IF INDEX CHILD WAS FED BY A CARER	
3.2	For the first item of food or meal, who fed the child?	
	Mother	1
	Sister	2

	Grandmother	3
	Other _____	4
3.3	What was the food and how was it served?	
	Dal bhaat served with a spoon	1
	Dal bhaat served with hands	2
	Khajaa served with a spoon	3
	Khajaa served with hands	4
3.4	Immediately before feeding did the person….	
	Hands not washed *(Go to section 3.1)*	1
	Rinse one hand with water	2
	Rinse both hands with water	3
	Wash one hand with soap	4
	Wash both hands with soap	5
	Rinse hands in soapy water	6
	Unable to see	7
3.5	Where did the water for the hand-washing come from?	
	From container in house/yard	1
	Laundry water	2
	A tap	3
	Rower pump/tube well	4
	Unable to see	5
3.6	If soap was used, where did the soap for hand-washing come from?	
	Soap not use	1

	Soap kept near to water source	2
	Soap distant to water source	3

Section 4: Mother's defecation

4.1	Did the mother go for defecation/toilet while you were present?	
	No, she didn't *(Go to section 5.1)*	0
	Yes, I saw	1
	Yes, I think so	2
	ONLY FILL IN NEXT SECTION IF THE MOTHER WENT FOR DEFECATION	
4.2	Where did the mother go to toilet?	
	Public toilet	1
	Toilet in her house	2
	Toilet in neighbour's house	3
	To the riverbanks	4
	Unable to see	5
4.3	Immediately after completion, did she…	
	Hands not washed *(Go to section 6.1)*	1
	Rinse one hand with water	2
	Rinse both hands with water	3
	Wash one hand with soap	4
	Wash both hands with soap	5
	Rinse hands in soapy water	6
	Unable to see	7

4.4	Where did the water for the hand-washing come from?	
	From container in house/yard	1
	Laundry water	2
	A tap	3
	Rower pump/tube well	4
	Unable to see	5
4.5	If soap was used, where did the soap for hand-washing come from?	
	Soap not use	1
	Soap kept near to water source	2
	Soap distant to water source	3

Section 5: Other person's defecation

5.1	Did anyone else go for defecation/toilet while you were present?	
	No, *(Go to section 6.1)*	0
	Yes, I saw. Who? _____	1
	Yes, I think so. Who? _____	2
	ONLY FILL IN NEXT SECTION IF SOMEONE ELSE WENT FOR DEFECATION	
5.2	Where did the person go to toilet?	
	Public toilet	1
	Toilet in her house	2
	Toilet in neighbour's house	3
	To the riverbanks	4
	Unable to see	5
5.3	Immediately after completion, did the person…	

	Hands not washed *(Go to section 6.1)*	1
	Rinse one hand with water	2
	Rinse both hands with water	3
	Wash one hand with soap	4
	Wash both hands with soap	5
	Rinse hands in soapy water	6
	Unable to see	7
5.4	Where did the water for the hand-washing come from?	
	From container in house/yard	1
	Laundry water	2
	A tap	3
	Rower pump/tube well	4
	Unable to see	5
5.5	If soap was used, where did the soap for hand-washing come from?	
	Soap not use	1
	Soap kept near to water source	2
	Soap distant to water source	3

Section 6: Cooking of food

6.1	Did anyone prepare or cook food while you were present? (NOT TEA)	
	Yes. Who? _____	1
	No	2
	ONLY FILL IN NEXT SECTION IF SOMEONE PREPARED FOOD	
6.2	Immediately before handling the food did the person…	
	Hands not washed *(Go to section 6.1)*	1
	Rinse one hand with water	2
	Rinse both hands with water	3
	Wash one hand with soap	4
	Wash both hands with soap	5
	Rinse hands in soapy water	6
	Unable to see	7
6.3	Where did the water for the hand-washing come from?	
	From container in house/yard	1
	Laundry water	2
	A tap	3
	Rower pump/tube well	4
	Unable to see	5
6.4	If soap was used, where did the soap for hand-washing come from?	
	Soap not use	1
	Soap kept near to water source	2
	Soap distant to water source	3

Appendix 2 - Socioeconomic & Demographic Questionnaire

Index Child			
Name of child		ID No.	
Nepali DOB		English DOB	
Sex	M(1) F(2)	Birth Order	
Mother			
Mother's name		Mother's age	
Where born?		Age at marriage	
How long lived in this area?			
Age at first birth		Age at birth of index child	
Mother's height		Mother's weight	
Can read and write Nepali?	Y(1) N(0)		
Highest completed level of education:	No education (0) Primary (1) Lower Secondary (2)		Secondary (3) Plus 2 (4) Bachelors (5)
Any paid employment?	Y (1) N(0)	What?	
Father			
Father's name		Father's age	
Where born?		Age at marriage	
How long lived in area?			
Can read and write Nepali	Y(1) N(0)		
Highest completed level of education:	No education (0) Primary (1) Lower Secondary (2)		Secondary (3) Plus 2 (4) Bachelors (5)

Any paid employment?	Y (1) N(0)	What?	
Household			
Total number of people in household			
Total number of adults (>16yrs)			
Total number of children aged 5-15 yrs			
Total number of children ages <5 yrs			
Ethnicity	Dalit Baishaya Bahun-Chhetri		
Religion	Hindu Buddhist Other _____		
House tenure	Own(1) Rent(0)		
No. rooms in house used by the family			
Separate kitchen area	Y(1) N(0)		
Fuel type	Firewood Kerosene Gas		
Toilet type	Public toilet Shared with other families Own toilet		
Valuable items possessions	TV Y(1) N(0) Radio Y(1) N(0) Telephone Y(1) N(0) Mobile phone Y(1) N(0) Bicycle Y(1) N(0) Motorbike Y(1) N(0) Fridge Y(1) N(0)		

Appendix 3 - Pregnancy, Feeding and Childcare Practices Questionnaire

Child Name		Child ID	
Maternal age at last birth		<18yrs	(1)
		18-34 yrs	(0)
		>35yrs	(1)
During your last pregnancy did you:			
Smoke		Yes (1)	No(0)
Drink		Yes (1)	No(0)
Take iron tablets		Yes (0)	No(1)
How many times did you see a healthcare professional during your last pregnancy?		Less than 4 times (1)	4 or more times (0)
Was your last child born prematurely?		Yes (1)	No(0)
Where did you give birth to your last child?		Home(1)	Hospital(1)
When did you first breastfeed your child?		Less than 60 mins after birth (0)	More than 60 mins after birth (1)
Did you give your child anything else to eat/drink before breastfeeding him/her for the first time?		Yes(1) What? _____	No(0)
Did you feed your child colostrum?		Yes (0)	No(1)
At what age did you first give your child something to eat other than breast milk (not just ceremonial rice feeding)		Before 6 months (1)	
		At 6 months (0)	
		At 7 months or more (1)	
What type of water to you give your baby to drink?		Boiled (0)	
		Filtered (1)	
		SODIS (0)	
		Untreated (1)	
Had your child had all his/her vaccinations to date?		Yes(0)	No(1)

When a child is sick do they need	Less liquid than normal (1)
	The same amount of liquid as normal(1)
	More liquid than normal (0)
When a child is sick do they need	Less food than normal (1)
	The same amount of food as normal(1)
	More food than normal (0)

Appendix 4 - Hygiene Questionnaire

When do you usually wash your hands? *Do not suggest answers but keep prompting the mother about when she washes her hands. For each answer ask what she uses to wash her hands. If the mother has not spontaneously mentioned all five junctures outlined below, you should then ask her specifically about the remaining junctures and record her answers below.*			
After going to toilet	Water	Mud/Ash	Soap
After cleaning baby's bottom	Water	Mud/Ash	Soap
Before cooking food	Water	Mud/Ash	Soap
Before feeding baby	Water	Mud/Ash	Soap
Before eating food	Water	Mud/Ash	Soap
Any other junctures mentioned by the mother…			
	Water	Mud/Ash	Soap
	Water	Mud/Ash	Soap
	Water	Mud/Ash	Soap
	Water	Mud/Ash	Soap
	Water	Mud/Ash	Soap
What type of soap do you normally use for hand-washing?	Body soap	Dish soap	Laundry soap
What brand of soap do you normally buy?	Body soap _____		
	Dish soap _____		
	Laundry soap _____		
Do you have any soap in the house now? Can you show me?	Soap seen	Soap not seen	

Appendix 5 – Morbidity Report

Child Name		**Child ID**	
Area		**Date**	
Diarrhoea			
During the last seven days since my last visit, what have your child's stools been like?		Completely normal (0) *Go to Q7*	Looser than normal (1)
If looser than normal, what were the stools like?		Like yoghurt (1)	Like water (2)
On the worst day, did the child pass three or more stools in one day?		Yes (1)	No (0)
Was there any blood present in the stools?		Yes (1)	No (0)
How long did the symptoms last for?		_____days	
Is the child still having loose stools today?		Yes (1)	No (0)
Cough/Cold			
Has the child has a cough/cold in the last seven days since I last saw you?		Yes (1)	No (0) *Go to Q11*
What symptoms did the child have? (Circle all those mentioned by the mother)			
	Cough (1)	Runny nose (2)	Wheeziness (3)
	Sneezing (4)	Blocked nose (5)	Sore throat (6)
	Headache (7)	Runny/red eyes (8)	
	Other _____		
How long did these symptoms last for?		_____days	
Is the child still sick with cough/cold today?		Yes (1)	No (0)
Fever			
Has the child had a fever in the last seven days?		Yes (1)	No (0) *Go to Q14*
How long did the fever last?		_____days	
Does the child still have a fever today?		Yes (1)	No (0)

Other symptoms				
Has the child experienced any other symptoms not mentioned above in the last seven days?		Yes (1)		No (0)
For each symptoms mentioned, record the number of days of sickness and if the child still has symptoms today				
Soap Usage				
How many NEW bars of soap have you STARTED this week?				
	Laundry ____	Body ____	Dish ____	TOTAL ____

Appendix 6 – Consent form

Nepal Child Health Project 2007

Information Sheet & Consent Form

Introduction

This project aims to learn about Nepali family life and the health of young Nepali children.

Observations

We would like to find out about the work of Nepali mothers living in Kathmandu. We are very interested in how you spend your time and what activities you do.

To find out about this we would like to come to your home one morning from 6-9am and watch what you and other members of your family do during this time. If you agree, a trained field-worker will come to your house and will make observations and record them on a form. You and your family should carry on as normal, as if she was not there.

Health Checks

During this project we also wish to measure the height and the weight of your child, and also take a small amount of urine for check-up. We will also take a few drops of blood by pricking the finger of your child. Most of the results of the check-up cannot be given immediately, as the samples will be sent back to UK for analysis. We can however tell you straight away about normal growth performance and iron levels in the blood.

We will come to your community every month from June in order to do this. We will tell you when and where you should bring your baby.

You can ask us more questions, after we have demonstrated these measures to you. You do not have to join this part of the project if you do not wish to. If you join, you can withdraw from the project whenever you wish and you do not have to give us a reason why.

I have explained the study to the person named below in a language that she understands well. I believe she has understood and is participating out of her own free will.

I agree to participate in the observations	Yes	No
I agree to participate in the monthly health checks	Yes	No

Name of child	……	ID No.	…….
Signature/ Mark of Parent	…….	Date	…….
Name of person obtaining consent	…….	Position	……

Thank you for your participation in this study.

Appendix 7 - Attrition Data

Index Child		**Attrition (n=11)**		**Study (n=88)**		**Test**	**P**
Age	mean (SD)	7.69	(2.19)	7.60	(2.38)	t-test	0.91
Sex %	Male	54.50		47.70		Chi-square	0.46
	Female	45.50		52.30			
Birth order	median (IQ range)	2.00	(1-2)	2.00	(1-3)	Mann-Whitney U	0.47
Place of birth %	Home	36.40		53.40		Chi-square	0.29
	Hospital	63.60		46.60			
Mother							
Age	mean (SD)	23.18	(3.16)	24.42	(4.60)	t-test	0.39
Age at marriage	mean (SD)	19.82	(2.04)	18.69	(3.48)	t-test	0.30
Yrs residency	median (IQ range)	3.00	(2-3)	4.00	(1.63-7)	Mann-Whitney U	0.30
Place of birth %	inside KTM	27.30		6.80		Chi-square	0.06
	outside KTM	72.70		93.20			
Literacy %	Literate	72.70		46.60		Chi-square	0.09
Education %	None	27.30		53.40		Cell count too low	
	Primary	45.50		18.20			
	secondary+	27.30		28.40			
Employment %	Employed	9.10		17.00		Chi-square	0.44

Father		**Attrition (n=11)**		**Study (n=88)**		**Test**	**P**
Age	mean (SD)	27.73	(5.42)	28.14	(5.54)	t-test	0.82
Age at marriage	mean (SD)	23.36	(2.38)	22.74	(4.37)	t-test	0.64
Yrs residency	Median	3.00	(1.5-5)	6.00	(3-15)	Mann-Whitney U	0.08
Place of birth %	inside KTM	0.00		13.60		Chi-square	0.22
	outside KTM	100.00		86.40			
Literacy %	Literate	90.90		72.70		Chi-square	0.18
Education %	None	9.10		27.30		Cell count too low	
	Primary	27.30		20.50			
	secondary+	63.70		52.20			
Employment %	Employed	100.00		95.50		Chi-square	0.62
Household							
Household size	median (IQ range)	4.00	(3-6)	4.00	(4-5)	Mann-Whitney U	0.45
Adults in house	median (IQ range)	2.00	(2-3)	2.00	(2-3)	Mann-Whitney U	0.65
Children 5-15yrs	median (IQ range)	0.00	(0-2)	1.00	(0-1.75)	Mann-Whitney U	0.63
Children <5 yrs	median (IQ range)	1.00	(1-2)	1.00	(1-2)	Mann-Whitney U	0.51
Ethnicity %	Dalit	9.10		11.4		Cell count too low	
	Hill Tribe	45.40		55.7			
	Newar	9.10		9.1			
	Bahun-Chhetri	36.40		36.4			
Religion %	Hindu	81.80		76.10		Cell count too low	
	Buddhist	18.20		18.20			
	Other	0.00		5.60			

Socio-economic status		Attrition (n=11)		Study (n=88)		Test	P
Own house %		63.60		54.50		Chi-square	0.41
Land outside KTM %		90.90		64.80		Chi-square	0.07
Toilet %	Own	0.00		18.40		Cell count too low	
	Shared	63.60		56.30			
	Public	36.40		25.30			
Rooms in house %	1 room	45.50		56.80		Chi-square	0.35
	2+ rooms	54.50		43.20			
Fuel type %	Firewood/kerosene	54.50		70.10		Chi-square	0.24
	Gas	45.50		29.90			
Income per month %	median (IQ range)	5000	(3.5-5K)	5000	(4-7K)	Mann-Whitney U	0.11
	<1000Rs	36.40		35.20		Chi-square	0.59
	>1000Rs	63.60		64.80			
Possessions %	0-1	63.60		45.50		Chi-square	0.21
	2+	36.40		54.50			
SES Score	mean (SD)	7.36	(2.54)	6.76	(3.34)	t-test	0.57

Growth, Biochemistry and Morbidity		Attrition (n=11)		Study (n=88)		Test	P
HAZ	mean (SD)	-1.03	(0.71)	-1.27	(0.97	t-test	0.44
WAZ	mean (SD)	-0.56	(0.99)	-1.07	(1.19)	t-test	0.18
WHZ	mean (SD)	0.53	(1.09)	0.13	(0.97)	t-test	0.21
Gut Damage Log10	mean (SD)	-0.98	(0.23)	-0.88	(0.28)	t-test	0.23
Albumin	mean (SD)	30.89	(10.01)	33.11	(7.19)	t-test	0.36
IgG	mean (SD)	6.32	(3.43)	6.71	(2.30)	t-test	0.61
AGP	mean (SD)	0.99	(0.54)	0.85	(0.33)	t-test	0.21
HB	mean (SD)	105.45	(9.23)	104.74	(8.78)	t-test	0.80
Diarrhoea	median (IQ range)	1.00	(0-2)	1.00	(1-2)	Mann-Whitney U	0.92
Cough/Cold	median (IQ range)	2.00	(1-3)	2.00	(1-3)	Mann-Whitney U	0.44
Fever	median (IQ range)	1.00	(0-1)	1.00	(0-1)	Mann-Whitney U	0.76

Appendix 8 - Differences in reported morbidity between control and intervention groups

		Control			Intervention				
		Median	25th centile	75th centile	Median	25th centile	75th centile	U	P
Diarrhoea	May	1.33	0.67	2.00	0.67	0.67	1.80	877.50	0.443
	June	1.00	0.00	2.00	1.00	0.00	1.50	945.00	0.841
	July	0.00	0.00	1.00	0.00	0.00	1.00	950.50	0.874
	Aug	0.00	0.00	1.00	0.00	0.00	1.00	905.50	0.527
	Sep	0.00	0.00	1.00	0.00	0.00	0.50	818.50	0.124
	Oct	0.00	0.00	0.00	0.00	0.00	0.00	917.50	0.534
	Nov	0.00	0.00	1.00	0.00	0.00	0.50	896.50	0.448
Cold	May	2.67	1.33	3.33	2.00	0.80	3.27	874.00	0.430
	June	2.00	1.00	3.00	1.00	1.00	2.83	767.00	0.087
	July	2.00	1.00	3.00	2.00	1.00	3.00	967.50	1.000
	Aug	2.00	1.00	2.00	1.00	0.00	2.00	815.50	0.188
	Sep	1.00	0.00	1.33	1.00	0.00	2.00	846.50	0.290
	Oct	1.33	0.00	2.67	0.00	0.00	1.33	765.50	0.075
	Nov	1.33	0.00	3.00	1.00	0.00	3.00	949.50	0.877
Fever	May	0.67	0.67	1.33	0.67	0.00	1.33	771.00	0.091
	June	1.00	0.00	2.00	0.00	0.00	1.00	801.50	0.129
	July	1.00	0.00	2.00	1.00	0.00	1.50	956.50	0.923
	Aug	0.00	0.00	1.00	0.00	0.00	1.00	919.00	0.653
	Sep	1.00	0.00	1.00	0.00	0.00	1.00	755.00	0.044
	Oct	0.00	0.00	1.33	0.00	0.00	0.67	802.50	0.090
	Nov	0.00	0.00	1.00	0.00	0.00	1.00	962.00	0.957

	Control			Intervention				
	Median	25th centile	75th centile	Median	25th centile	75th centile	U	*P*
Total Diarrhoea Score	4.33	3.00	6.53	3.00	1.67	5.63	732.00	0.049
Total Cold Score	10.00	8.00	15.33	8.00	5.67	14.00	767.00	0.094
Total Fever Score	4.67	3.00	8.67	3.67	2.33	5.83	774.00	0.106

Appendix 9 - Differences in days of sickness between control and intervention groups

		Control			Intervention				
		Median	25th centile	75th centile	Median	25th centile	75th centile	U	P
Diarrhoea	May	5.33	1.33	11.20	2.67	0.00	5.67	768.50	0.094
	June	3.00	0.00	10.00	2.00	0.00	6.00	901.00	0.556
	July	0.00	0.00	4.00	0.00	0.00	4.50	966.00	0.989
	Aug	0.00	0.00	4.00	0.00	0.00	2.00	898.50	0.486
	Sep	0.00	0.00	4.00	0.00	0.00	0.50	832.50	0.167
	Oct	0.00	0.00	0.00	0.00	0.00	0.00	920.00	0.555
	Nov	0.00	0.00	3.00	0.00	0.00	1.50	901.50	0.482
Cold	May	11.33	5.33	17.60	9.33	4.00	15.67	842.50	0.296
	June	8.00	4.00	14.00	4.00	0.00	10.50	724.50	0.041
	July	7.00	3.00	16.00	8.00	3.00	14.00	904.00	0.595
	Aug	7.00	3.00	10.00	5.00	0.00	12.50	878.00	0.451
	Sep	4.00	0.00	7.00	3.00	0.00	6.50	784.50	0.117
	Oct	8.00	0.00	13.33	0.00	0.00	6.67	733.00	0.041
	Nov	7.00	0.00	14.00	6.00	0.00	12.17	892.50	0.523
Fever	May	2.67	0.80	4.67	1.33	0.00	5.07	785.50	0.123
	June	2.00	0.00	6.00	0.00	0.00	3.00	787.00	0.103
	July	3.00	0.00	7.00	3.00	0.00	6.50	919.50	0.682
	Aug	0.00	0.00	4.00	0.00	0.00	3.50	945.50	0.841
	Sep	2.00	0.00	4.00	0.00	0.00	1.50	755.00	0.049
	Oct	0.00	0.00	4.00	0.00	0.00	0.67	783.00	0.059
	Nov	0.00	0.00	3.00	0.00	0.00	3.50	946.00	0.835

	Control			Intervention				
	Median	25th centile	75th centile	Median	25th centile	75th centile	U	*P*
Total days of Diarrhoea	16.33	12.67	30.33	9.67	4.83	25.50	695.00	0.023
Total days of Cold	50.00	35.33	78.00	40.00	19.23	65.50	744.00	0.062
Total days of Fever	16.33	7.67	27.00	11.00	7.10	19.67	783.50	0.125

Appendix 10 - Relationship between biomarkers on a month-by-month basis

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	0.392	0.096	4.070	0.000	0.200	0.583	
IgG1	L:C1	2.633	10.862	0.240	0.809	-18.964	24.229	0.144
(May)	constant	2.856	3.790	0.750	0.453	-4.680	10.391	
	age2	0.453	0.079	5.740	0.000	0.296	0.609	
IgG2	L:C2	-1.338	5.543	-0.240	0.810	-12.358	9.682	0.263
(June)	constant	2.289	1.934	1.180	0.240	-1.557	6.135	
	age3	0.283	0.094	3.010	0.003	0.096	0.470	
IgG3	L:C3	9.364	11.797	0.790	0.430	-14.092	32.820	0.078
(July)	constant	0.270	4.089	0.070	0.947	-7.860	8.401	
	age4	0.267	0.084	3.180	0.002	0.100	0.434	
IgG4	L:C4	16.810	8.771	1.920	0.059	-0.630	34.249	0.109
(Aug)	constant	-1.627	3.120	-0.520	0.603	-7.831	4.577	
	age5	0.242	0.098	2.480	0.015	0.048	0.436	
IgG5	L:C5	6.314	15.528	0.410	0.685	-24.559	37.188	0.046
(Sep)	constant	2.434	5.344	0.460	0.650	-8.191	13.060	
	age6	0.013	0.126	0.100	0.918	-0.237	0.263	
IgG6	L:C6	11.303	9.588	1.180	0.242	-7.761	30.367	0.000
(Oct)	constant	5.260	3.369	1.560	0.122	-1.437	11.958	
	age7	0.415	0.137	3.020	0.003	0.142	0.688	
IgG7	L:C7	13.356	14.044	0.950	0.344	-14.567	41.279	0.080
(Nov)	constant	-1.076	5.111	-0.210	0.834	-11.238	9.086	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	0.306	0.090	3.400	0.001	0.127	0.486	
IgG1	AGP1	2.602	0.654	3.980	0.000	1.302	3.903	0.278
(May)	constant	2.179	0.804	2.710	0.008	0.582	3.777	
	age2	0.419	0.077	5.450	0.000	0.266	0.572	
IgG2	AGP2	1.148	0.457	2.510	0.014	0.240	2.056	0.314
(June)	constant	1.107	0.734	1.510	0.135	-0.352	2.566	
	age3	0.283	0.089	3.190	0.002	0.107	0.459	
IgG3	AGP3	2.321	0.703	3.300	0.001	0.922	3.719	0.177
(July)	constant	1.469	1.049	1.400	0.165	-0.617	3.555	
	age4	0.256	0.079	3.250	0.002	0.100	0.413	
IgG4	AGP4	3.120	0.816	3.820	0.000	1.497	4.742	0.207
(Aug)	constant	1.886	1.019	1.850	0.068	-0.140	3.912	
	age5	0.234	0.091	2.570	0.012	0.053	0.415	
IgG5	AGP5	3.197	0.983	3.250	0.002	1.243	5.152	0.150
(Sep)	constant	2.414	1.241	1.950	0.055	-0.052	4.881	
	age6	-0.011	0.120	-0.100	0.924	-0.250	0.227	
IgG6	AGP6	2.353	0.756	3.110	0.003	0.850	3.856	0.082
(Oct)	constant	7.238	1.553	4.660	0.000	4.150	10.325	
	age7	0.365	0.138	2.640	0.010	0.090	0.640	
IgG7	AGP7	1.369	0.896	1.530	0.130	-0.413	3.151	0.095
(Nov)	constant	2.774	1.852	1.500	0.138	-0.909	6.457	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	0.425	0.091	4.650	0.000	0.244	0.607	
IgG1	alb1	0.094	0.030	3.090	0.003	0.033	0.154	0.230
(May)	constant	0.378	1.309	0.290	0.773	-2.225	2.982	
	age2	0.469	0.071	6.580	0.000	0.327	0.610	
IgG2	alb2	0.121	0.028	4.320	0.000	0.065	0.176	0.395
(June)	constant	-2.128	1.116	-1.910	0.060	-4.347	0.092	
	age3	0.333	0.091	3.670	0.000	0.152	0.514	
IgG3	alb3	0.103	0.033	3.120	0.002	0.037	0.168	0.167
(July)	constant	-0.755	1.600	-0.470	0.638	-3.936	2.426	
	age4	0.295	0.074	3.970	0.000	0.148	0.443	
IgG4	alb4	0.119	0.022	5.340	0.000	0.075	0.163	0.304
(Aug)	constant	-0.268	1.135	-0.240	0.814	-2.524	1.989	
	age5	0.292	0.088	3.310	0.001	0.117	0.467	
IgG5	alb5	0.167	0.038	4.380	0.000	0.091	0.243	0.220
(Sep)	constant	-1.945	1.794	-1.080	0.282	-5.512	1.623	
	age6	0.222	0.124	1.800	0.076	-0.024	0.468	
IgG6	alb6	0.163	0.039	4.140	0.000	0.085	0.241	0.149
(Oct)	constant	0.347	2.484	0.140	0.889	-4.592	5.286	
	age7	0.350	0.130	2.700	0.008	0.092	0.608	
IgG7	alb7	0.163	0.047	3.490	0.001	0.070	0.255	0.187
(Nov)	constant	-1.237	2.171	-0.570	0.570	-5.554	3.081	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	0.388	0.095	4.090	0.000	0.199	0.576	
IgG1	Hb1	-0.029	0.026	-1.120	0.265	-0.080	0.022	0.156
(May)	constant	6.793	2.811	2.420	0.018	1.205	12.382	
	age2	0.451	0.079	5.740	0.000	0.295	0.607	
IgG2	Hb2	-0.005	0.018	-0.260	0.797	-0.040	0.031	0.263
(June)	constant	2.336	2.002	1.170	0.246	-1.643	6.316	
	age3	0.274	0.094	2.910	0.005	0.087	0.460	
IgG3	Hb3	0.016	0.023	0.690	0.495	-0.030	0.062	0.077
(July)	constant	1.801	2.552	0.710	0.482	-3.274	6.876	
	age4	0.247	0.085	2.920	0.004	0.079	0.416	
IgG4	Hb4	0.023	0.020	1.150	0.255	-0.017	0.062	0.085
(Aug)	constant	1.670	2.307	0.720	0.471	-2.917	6.257	
	age5	0.244	0.096	2.530	0.013	0.052	0.436	
IgG5	Hb5	0.035	0.021	1.700	0.093	-0.006	0.076	0.075
(Sep)	constant	0.774	2.483	0.310	0.756	-4.163	5.711	
	age6	0.013	0.124	0.110	0.914	-0.233	0.260	
IgG6	Hb6	0.048	0.027	1.780	0.078	-0.006	0.102	0.014
(Oct)	constant	3.739	3.220	1.160	0.249	-2.665	10.142	
	age7	0.407	0.139	2.920	0.004	0.130	0.684	
IgG7	Hb7	0.033	0.031	1.100	0.276	-0.027	0.094	0.088
(Nov)	constant	-0.233	3.663	-0.060	0.949	-7.517	7.051	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	0.030	0.015	2.070	0.042	0.001	0.059	
AGP1	L:C1	-1.745	1.643	-1.060	0.291	-5.012	1.522	0.044
(May)	constant	1.202	0.573	2.100	0.039	0.062	2.342	
	age2	0.026	0.018	1.470	0.146	-0.009	0.062	
AGP2	L:C2	1.528	1.259	1.210	0.228	-0.976	4.032	0.022
(June)	constant	0.152	0.440	0.350	0.730	-0.722	1.026	
	age3	-0.002	0.014	-0.180	0.860	-0.030	0.025	
AGP3	L:C3	-0.081	1.719	-0.050	0.963	-3.498	3.337	0.000
(July)	constant	0.874	0.596	1.470	0.146	-0.311	2.059	
	age4	-0.003	0.011	-0.250	0.806	-0.023	0.018	
AGP4	L:C4	-0.485	1.099	-0.440	0.660	-2.670	1.699	0.000
(Aug)	constant	0.877	0.391	2.240	0.027	0.100	1.654	
	age5	0.001	0.010	0.120	0.902	-0.019	0.021	
AGP5	L:C5	0.621	1.616	0.380	0.701	-2.591	3.834	0.000
(Sep)	constant	0.462	0.556	0.830	0.409	-0.644	1.567	
	age6	0.015	0.017	0.880	0.383	-0.019	0.049	
AGP6	L:C6	2.136	1.294	1.650	0.102	-0.436	4.708	0.022
(Oct)	constant	-0.007	0.454	-0.010	0.988	-0.910	0.897	
	age7	0.031	0.016	1.960	0.053	0.000	0.063	
AGP7	L:C7	3.727	1.636	2.280	0.025	0.474	6.980	0.068
(Nov)	constant	-0.760	0.595	-1.280	0.205	-1.944	0.423	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	0.035	0.014	2.450	0.016	0.007	0.064	
AGP1	alb1	0.009	0.005	1.870	0.065	-0.001	0.018	0.069
(May)	constant	0.285	0.206	1.380	0.171	-0.125	0.694	
	age2	0.029	0.018	1.630	0.108	-0.006	0.065	
AGP2	alb2	0.007	0.007	0.950	0.344	-0.007	0.021	0.016
(June)	constant	0.429	0.281	1.530	0.130	-0.130	0.988	
	age3	0.004	0.014	0.270	0.789	-0.023	0.031	
AGP3	alb3	0.011	0.005	2.250	0.027	0.001	0.021	0.034
(July)	constant	0.396	0.238	1.660	0.100	-0.077	0.870	
	age4	0.003	0.009	0.350	0.727	-0.015	0.022	
AGP4	alb4	0.014	0.003	5.040	0.000	0.008	0.020	0.212
(Aug)	constant	0.197	0.141	1.400	0.166	-0.083	0.478	
	age5	0.003	0.010	0.260	0.799	-0.017	0.023	
AGP5	alb5	0.006	0.004	1.300	0.198	-0.003	0.014	0.004
(Sep)	constant	0.451	0.205	2.200	0.030	0.044	0.858	
	age6	0.016	0.018	0.890	0.374	-0.020	0.053	
AGP6	alb6	-0.002	0.006	-0.300	0.763	-0.013	0.010	0.000
(Oct)	constant	0.753	0.370	2.030	0.045	0.017	1.488	
	age7	0.029	0.017	1.740	0.085	-0.004	0.062	
AGP7	alb7	-0.002	0.006	-0.260	0.799	-0.013	0.010	0.012
(Nov)	constant	0.552	0.277	1.990	0.050	0.001	1.103	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	0.032	0.015	2.190	0.031	0.003	0.061	
AGP1	Hb1	0.001	0.004	0.180	0.854	-0.007	0.009	0.031
(May)	constant	0.529	0.431	1.230	0.223	-0.328	1.386	
	age2	0.028	0.018	1.580	0.117	-0.007	0.064	
AGP2	Hb2	0.003	0.004	0.790	0.432	-0.005	0.011	0.012
(June)	constant	0.311	0.457	0.680	0.498	-0.597	1.220	
	age3	-0.003	0.014	-0.240	0.811	-0.030	0.024	
AGP3	Hb3	0.004	0.003	1.080	0.282	-0.003	0.010	0.000
(July)	constant	0.474	0.369	1.280	0.203	-0.260	1.208	
	age4	-0.002	0.010	-0.190	0.852	-0.023	0.019	
AGP4	Hb4	-0.001	0.002	-0.540	0.594	-0.006	0.004	0.000
(Aug)	constant	0.852	0.285	2.990	0.004	0.286	1.419	
	age5	0.000	0.010	-0.020	0.988	-0.020	0.020	
AGP5	Hb5	0.003	0.002	1.290	0.199	-0.001	0.007	0.000
(Sep)	constant	0.389	0.259	1.510	0.136	-0.125	0.904	
	age6	0.017	0.017	1.000	0.320	-0.017	0.051	
AGP6	Hb6	0.004	0.004	0.990	0.323	-0.004	0.011	0.002
(Oct)	constant	0.272	0.443	0.610	0.541	-0.609	1.154	
	age7	0.028	0.017	1.690	0.094	-0.005	0.061	
AGP7	Hb7	-0.005	0.004	-1.380	0.172	-0.012	0.002	0.027
(Nov)	constant	1.058	0.435	2.430	0.017	0.193	1.923	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	-0.405	0.326	-1.240	0.217	-1.053	0.243	
Alb1	L:C1	-18.605	36.813	-0.510	0.615	-91.798	54.589	0.000
(May)	constant	42.404	12.845	3.300	0.001	16.866	67.943	
	age2	-0.177	0.274	-0.650	0.520	-0.722	0.368	
Alb2	L:C2	25.366	19.270	1.320	0.192	-12.948	63.679	0.000
(June)	constant	24.684	6.725	3.670	0.000	11.312	38.056	
	age3	-0.544	0.294	-1.850	0.068	-1.129	0.041	
Alb3	L:C3	-0.512	36.957	-0.010	0.989	-73.993	72.969	0.016
(July)	constant	40.978	12.811	3.200	0.002	15.508	66.449	
	age4	-0.383	0.361	-1.060	0.291	-1.101	0.334	
Alb4	L:C4	-1.318	37.708	-0.030	0.972	-76.292	73.656	0.000
(Aug)	constant	37.204	13.414	2.770	0.007	10.533	63.875	
	age5	-0.347	0.250	-1.390	0.168	-0.844	0.150	
Alb5	L:C5	-17.381	39.811	-0.440	0.664	-96.535	61.774	0.000
(Sep)	constant	44.730	13.701	3.260	0.002	17.488	71.971	
	age6	-1.196	0.318	-3.760	0.000	-1.829	-0.563	
Alb6	L:C6	17.353	24.251	0.720	0.476	-30.863	65.570	0.123
(Oct)	constant	46.475	8.520	5.450	0.000	29.536	63.415	
	age7	0.348	0.300	1.160	0.250	-0.249	0.945	
Alb7	L:C7	17.844	30.689	0.580	0.562	-43.173	78.862	0.000
(Nov)	constant	22.876	11.169	2.050	0.044	0.669	45.083	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	-0.380	0.318	-1.200	0.235	-1.012	0.252	
Alb1	Hb1	0.163	0.086	1.890	0.063	-0.009	0.334	0.034
(May)	constant	18.945	9.413	2.010	0.047	0.230	37.659	
	age2	-0.135	0.265	-0.510	0.611	-0.662	0.392	
Alb2	Hb2	0.159	0.060	2.660	0.009	0.040	0.279	0.058
(June)	constant	16.112	6.755	2.390	0.019	2.681	29.543	
	age3	-0.600	0.276	-2.170	0.033	-1.150	-0.050	
Alb3	Hb3	0.227	0.068	3.360	0.001	0.093	0.362	0.131
(July)	constant	17.270	7.506	2.300	0.024	2.347	32.193	
	age4	-0.402	0.349	-1.150	0.253	-1.096	0.292	
Alb4	Hb4	0.180	0.081	2.210	0.030	0.018	0.342	0.045
(Aug)	constant	17.408	9.516	1.830	0.071	-1.513	36.330	
	age5	-0.250	0.236	-1.060	0.292	-0.719	0.219	
Alb5	Hb5	0.145	0.050	2.880	0.005	0.045	0.245	0.082
(Sep)	constant	22.516	6.069	3.710	0.000	10.447	34.585	
	age6	-1.204	0.314	-3.830	0.000	-1.829	-0.580	
Alb6	Hb6	0.096	0.068	1.410	0.162	-0.039	0.232	0.138
(Oct)	constant	41.749	8.159	5.120	0.000	25.528	57.970	
	age7	0.234	0.279	0.840	0.404	-0.321	0.788	
Alb7	Hb7	0.256	0.061	4.190	0.000	0.135	0.377	0.165
(Nov)	constant	2.431	7.328	0.330	0.741	-12.143	17.004	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	-0.026	0.402	-0.070	0.948	-0.825	0.772	
Hb1	L:C1	14.242	45.368	0.310	0.754	-75.962	104.446	0.000
(May)	constant	100.177	15.830	6.330	0.000	68.703	131.650	
	age2	-0.104	0.479	-0.220	0.828	-1.056	0.848	
Hb2	L:C2	31.205	33.670	0.930	0.357	-35.740	98.150	0.000
(June)	constant	95.503	11.751	8.130	0.000	72.138	118.867	
	age3	0.246	0.443	0.560	0.580	-0.635	1.127	
Hb3	L:C3	-2.387	55.614	-0.040	0.966	-112.962	108.188	0.000
(July)	constant	104.271	19.278	5.410	0.000	65.942	142.600	
	age4	0.169	0.464	0.360	0.716	-0.753	1.091	
Hb4	L:C4	58.331	48.447	1.200	0.232	-37.994	154.656	0.000
(Aug)	constant	87.746	17.234	5.090	0.000	53.479	122.012	
	age5	-0.134	0.513	-0.260	0.795	-1.153	0.886	
Hb5	L:C5	46.758	82.853	0.560	0.574	-118.005	211.521	0.000
(Sep)	constant	91.912	28.324	3.250	0.002	35.587	148.238	
	age6	0.262	0.489	0.540	0.594	-0.711	1.235	
Hb6	L:C6	75.396	37.276	2.020	0.046	1.281	149.511	0.031
(Oct)	constant	81.618	13.096	6.230	0.000	55.580	107.656	
	age7	0.423	0.496	0.850	0.396	-0.564	1.409	
Hb7	L:C7	50.634	50.058	1.010	0.315	-48.912	150.180	0.000
(Nov)	constant	86.531	18.307	4.730	0.000	50.125	122.937	

Appendix 11 – Relationship between mean biomarkers

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
mean IgG	Mean age	0.297	0.071	4.150	0.000	0.155	0.439	
	Mean L:C	23.894	11.757	2.030	0.045	0.518	47.269	0.178
	constant	-3.808	3.997	-0.950	0.343	-11.756	4.139	
	Mean age	0.247	0.072	3.440	0.001	0.104	0.390	
	Mean AGP	3.043	1.083	2.810	0.006	0.889	5.196	0.211
	constant	2.161	1.030	2.100	0.039	0.113	4.209	
	Mean age	0.327	0.075	4.390	0.000	0.179	0.475	
	Mean Alb	0.094	0.053	1.780	0.079	-0.011	0.198	0.169
	constant	0.598	2.146	0.280	0.781	-3.669	4.865	
	Mean age	0.292	0.073	3.990	0.000	0.147	0.438	
	Mean Hb	-0.001	0.022	-0.070	0.947	-0.044	0.041	0.138
	constant	4.319	2.404	1.800	0.076	-0.461	9.100	

		Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
mean AGP	Mean age	0.015	0.007	2.320	0.023	0.002	0.029	
	Mean L:C	3.236	1.098	2.950	0.004	1.052	5.420	0.118
	constant	-0.421	0.373	-1.130	0.263	-1.163	0.322	
	Mean age	0.016	0.007	2.190	0.032	0.001	0.030	
	Mean Alb	0.003	0.005	0.530	0.599	-0.007	0.013	0.031
	constant	0.557	0.209	2.660	0.009	0.141	0.972	
	Mean age	0.015	0.007	2.100	0.039	0.001	0.029	
	Mean Hb	0.002	0.002	0.730	0.466	-0.003	0.006	0.034
	constant	0.500	0.230	2.180	0.032	0.044	0.957	

		Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
mean Albumin	Mean age	-0.375	0.148	-2.540	0.013	-0.669	-0.081	
	Mean L:C	12.986	24.314	0.530	0.595	-35.357	61.328	0.053
	constant	33.777	8.267	4.090	0.000	17.341	50.213	
	Mean age	-0.389	0.143	-2.710	0.008	-0.674	-0.104	
	Mean Hb	0.100	0.042	2.360	0.020	0.016	0.184	0.108
	constant	27.569	4.712	5.850	0.000	18.201	36.937	

		Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
mean Hb	Mean age	0.137	0.362	0.380	0.706	-0.582	0.857	
	Mean L:C	108.220	59.537	1.820	0.073	-10.155	226.594	0.016
	constant	69.632	20.242	3.440	0.001	29.386	109.879	

MULTIVARIATE MODELS

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
mean IgG	Mean age	0.256	0.072	3.560	0.001	0.113	0.399	
	Mean L:C	15.481	12.044	1.290	0.202	-8.469	39.431	0.217
	Mean AGP	2.599	1.133	2.290	0.024	0.347	4.852	
	constant	-2.715	3.930	-0.690	0.492	-10.529	5.100	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
mean AGP	Mean age	0.008753	0.007147	1.22	0.224	-0.00546	0.022964	
	Mean L:C	2.694025	1.097636	2.45	0.016	0.511256	4.876795	0.160
	Mean IgG	0.022693	0.009889	2.29	0.024	0.003027	0.042359	
	constant	-0.33428	0.366382	-0.91	0.364	-1.06287	0.394313	

Appendix 12 – Relationship between biomarkers using Time Series Analysis

	Predictor	Coef.	Std. Err.	z	P		95% CI	Rho
IgG	age	0.492	0.038	12.820	0.000	0.417	0.567	
	L:C	1.310	3.731	0.350	0.725	-6.003	8.623	0.376
	constant	1.667	1.337	1.250	0.212	-0.952	4.287	
	age	0.493	0.037	13.390	0.000	0.421	0.565	
	AGP	1.977	0.243	8.140	0.000	1.501	2.453	0.398
	constant	0.483	0.461	1.050	0.295	-0.421	1.388	
	age	0.456	0.033	13.620	0.000	0.391	0.522	
	Alb	0.145	0.011	13.620	0.000	0.124	0.166	0.438
	constant	-2.484	0.507	-4.900	0.000	-3.478	-1.491	
	age	0.460	0.038	12.090	0.000	0.386	0.535	
	Hb	0.048	0.010	4.590	0.000	0.028	0.069	0.401
	constant	-2.718	1.139	-2.390	0.017	-4.951	-0.486	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Rho
AGP	age	0.007	0.005	1.390	0.164	-0.003	0.017	
	L:C	1.279	0.565	2.270	0.023	0.172	2.386	0.072
	constant	0.317	0.199	1.590	0.112	-0.074	0.708	
	age	0.005	0.005	1.060	0.290	-0.005	0.015	
	Alb	0.006	0.002	3.180	0.001	0.002	0.010	0.092
	constant	0.551	0.084	6.580	0.000	0.387	0.715	
	age	0.005	0.005	1.070	0.285	-0.005	0.015	
	Hb	0.001	0.001	0.610	0.539	-0.002	0.004	0.084
	constant	0.665	0.155	4.300	0.000	0.362	0.968	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Rho
Albumin	age	0.069	0.114	0.610	0.542	-0.153	0.292	
	L:C	5.200	11.912	0.440	0.662	-18.147	28.546	0.104
	constant	31.767	4.211	7.540	0.000	23.513	40.021	
	age	-0.022	0.109	-0.200	0.839	-0.235	0.191	
	Hb	0.213	0.031	6.970	0.000	0.153	0.272	0.121
	constant	11.696	3.395	3.450	0.001	5.042	18.350	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Rho
Hb	age	0.758	0.146	5.200	0.000	0.473	1.044	
	L:C	16.597	13.835	1.200	0.230	-10.518	43.713	0.550
	constant	93.619	5.024	18.630	0.000	83.773	103.466	

MULTIVARIATE MODELS

	Predictor	Coef.	Std. Err.	z	P		95% CI	Rho
IgG	age	0.448	0.033	13.760	0.000	0.384	0.512	
	AGP	1.598	0.215	7.450	0.000	1.177	2.018	
	Alb	0.130	0.011	12.140	0.000	0.109	0.151	0.459
	Hb	0.015	0.009	1.640	0.101	-0.003	0.034	
	constant	-4.839	0.979	-4.950	0.000	-6.757	-2.921	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Rho
AGP	age	-0.012	0.006	-2.040	0.041	-0.023	0.000	
	IgG	0.043	0.006	6.730	0.000	0.030	0.055	
	L:C	1.036	0.540	1.920	0.055	-0.023	2.095	0.092
	Alb	0.000	0.002	-0.050	0.964	-0.004	0.004	
	constant	0.287	0.201	1.430	0.153	-0.107	0.681	

Appendix 13 - Relationships between biomarkers and growth variables on a month-by-month basis

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	-0.085	0.043	-1.990	0.049	-0.171	0.000	
HAZ1	L:C1	6.705	4.839	1.390	0.169	-2.916	16.326	0.050
(May)	constant	-2.859	1.688	-1.690	0.094	-6.216	0.498	
	age2	-0.065	0.043	-1.530	0.129	-0.150	0.019	
HAZ2	L:C2	-7.455	2.990	-2.490	0.015	-13.399	-1.510	0.077
(June)	constant	1.771	1.044	1.700	0.093	-0.304	3.846	
	age3	-0.086	0.044	-1.960	0.053	-0.172	0.001	
HAZ3	L:C3	-11.346	5.477	-2.070	0.041	-22.236	-0.455	0.060
(July)	constant	3.239	1.899	1.710	0.092	-0.536	7.014	
	age4	-0.112	0.043	-2.620	0.010	-0.197	-0.027	
HAZ4	L:C4	-11.669	4.463	-2.610	0.011	-20.543	-2.795	0.107
(Aug)	constant	3.609	1.588	2.270	0.026	0.452	6.766	
	age5	-0.108	0.043	-2.530	0.013	-0.193	-0.023	
HAZ5	L:C5	-15.320	6.813	-2.250	0.027	-28.866	-1.775	0.085
(Sep)	constant	4.664	2.345	1.990	0.050	0.002	9.326	
	age6	-0.070	0.044	-1.580	0.117	-0.157	0.018	
HAZ6	L:C6	-7.356	3.345	-2.200	0.031	-14.006	-0.706	0.068
(Oct)	constant	1.604	1.175	1.370	0.176	-0.732	3.941	
	age7	-0.085	0.043	-1.970	0.053	-0.171	0.001	
HAZ7	L:C7	-6.491	4.417	-1.470	0.145	-15.273	2.290	0.039
(Nov)	constant	1.487	1.607	0.920	0.358	-1.709	4.683	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	-0.060	0.046	-1.300	0.196	-0.152	0.032	
HAZ1	IgG1	-0.081	0.048	-1.690	0.094	-0.177	0.014	0.061
(May)	constant	-0.262	0.382	-0.680	0.495	-1.022	0.499	
	age2	-0.056	0.052	-1.090	0.277	-0.159	0.046	
HAZ2	IgG2	-0.040	0.060	-0.660	0.513	-0.160	0.080	0.015
(June)	constant	-0.584	0.403	-1.450	0.152	-1.386	0.218	
	age3	-0.076	0.047	-1.610	0.110	-0.169	0.017	
HAZ3	IgG3	-0.013	0.051	-0.250	0.805	-0.115	0.089	0.014
(July)	constant	-0.551	0.469	-1.170	0.244	-1.484	0.382	
	age4	-0.097	0.046	-2.100	0.039	-0.189	-0.005	
HAZ4	IgG4	-0.013	0.056	-0.230	0.822	-0.124	0.099	0.036
(Aug)	constant	-0.318	0.523	-0.610	0.544	-1.358	0.721	
	age5	-0.083	0.045	-1.840	0.069	-0.172	0.006	
HAZ5	IgG5	-0.050	0.049	-1.020	0.309	-0.146	0.047	0.042
(Sep)	constant	-0.266	0.547	-0.490	0.627	-1.353	0.821	
	age6	-0.081	0.045	-1.820	0.072	-0.170	0.007	
HAZ6	IgG6	-0.019	0.039	-0.500	0.619	-0.096	0.057	0.018
(Oct)	constant	-0.525	0.647	-0.810	0.419	-1.812	0.761	
	age7	-0.070	0.046	-1.530	0.129	-0.161	0.021	
HAZ7	IgG7	-0.024	0.034	-0.690	0.493	-0.092	0.045	0.021
(Nov)	constant	-0.641	0.587	-1.090	0.278	-1.808	0.527	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	-0.091	0.044	-2.060	0.043	-0.179	-0.003	
HAZ1	AGP1	-0.030	0.321	-0.090	0.926	-0.668	0.608	0.029
(May)	constant	-0.550	0.394	-1.390	0.167	-1.333	0.234	
	age2	-0.072	0.044	-1.620	0.110	-0.160	0.017	
HAZ2	AGP2	-0.086	0.264	-0.320	0.746	-0.611	0.440	0.011
(June)	constant	-0.601	0.425	-1.420	0.160	-1.446	0.243	
	age3	-0.080	0.045	-1.780	0.078	-0.168	0.009	
HAZ3	AGP3	-0.165	0.354	-0.470	0.641	-0.869	0.538	0.016
(July)	constant	-0.454	0.528	-0.860	0.391	-1.504	0.595	
	age4	-0.098	0.043	-2.270	0.025	-0.185	-0.012	
HAZ4	AGP4	0.829	0.449	1.850	0.068	-0.063	1.721	0.072
(Aug)	constant	-0.960	0.560	-1.710	0.090	-2.074	0.154	
	age5	-0.094	0.043	-2.170	0.033	-0.181	-0.008	
HAZ5	AGP5	-0.281	0.469	-0.600	0.552	-1.214	0.653	0.034
(Sep)	constant	-0.305	0.592	-0.510	0.608	-1.482	0.873	
	age6	-0.069	0.043	-1.590	0.116	-0.155	0.017	
HAZ6	AGP6	-0.714	0.273	-2.610	0.011	-1.257	-0.171	0.089
(Oct)	constant	-0.223	0.561	-0.400	0.692	-1.338	0.893	
	age7	-0.089	0.044	-2.010	0.047	-0.176	-0.001	
HAZ7	AGP7	0.320	0.286	1.120	0.265	-0.248	0.888	0.029
(Nov)	constant	-0.885	0.590	-1.500	0.138	-2.059	0.289	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	-0.087	0.043	-2.020	0.046	-0.173	-0.002	
HAZ1	alb1	0.012	0.014	0.830	0.411	-0.017	0.040	0.037
(May)	constant	-0.995	0.619	-1.610	0.112	-2.227	0.237	
	age2	-0.078	0.044	-1.790	0.078	-0.164	0.009	
HAZ2	alb2	-0.023	0.017	-1.330	0.186	-0.057	0.011	0.030
(June)	constant	0.092	0.682	0.140	0.893	-1.263	1.447	
	age3	-0.078	0.045	-1.700	0.092	-0.168	0.013	
HAZ3	alb3	0.003	0.016	0.180	0.854	-0.030	0.036	0.013
(July)	constant	-0.718	0.801	-0.900	0.372	-2.310	0.873	
	age4	-0.089	0.043	-2.060	0.042	-0.175	-0.003	
HAZ4	alb4	0.030	0.013	2.300	0.024	0.004	0.055	0.091
(Aug)	constant	-1.463	0.659	-2.220	0.029	-2.773	-0.154	
	age5	-0.093	0.044	-2.120	0.037	-0.181	-0.006	
HAZ5	alb5	0.003	0.019	0.150	0.885	-0.035	0.041	0.030
(Sep)	constant	-0.601	0.896	-0.670	0.504	-2.382	1.181	
	age6	-0.076	0.048	-1.580	0.118	-0.172	0.020	
HAZ6	alb6	0.005	0.015	0.340	0.736	-0.025	0.036	0.017
(Oct)	constant	-0.964	0.968	-1.000	0.322	-2.888	0.961	
	age7	-0.082	0.044	-1.860	0.066	-0.169	0.006	
HAZ7	alb7	0.007	0.016	0.420	0.677	-0.025	0.038	0.017
(Nov)	constant	-0.913	0.735	-1.240	0.217	-2.374	0.548	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	-0.091	0.042	-2.150	0.034	-0.176	-0.007	
HAZ1	Hb1	0.018	0.012	1.520	0.133	-0.005	0.040	0.054
(May)	constant	-2.408	1.258	-1.910	0.059	-4.910	0.094	
	age2	-0.073	0.043	-1.690	0.094	-0.160	0.013	
HAZ2	Hb2	0.013	0.010	1.350	0.182	-0.006	0.033	0.031
(June)	constant	-2.055	1.107	-1.860	0.067	-4.255	0.146	
	age3	-0.084	0.044	-1.900	0.061	-0.171	0.004	
HAZ3	Hb3	0.018	0.011	1.670	0.099	-0.003	0.039	0.044
(July)	constant	-2.452	1.194	-2.050	0.043	-4.826	-0.077	
	age4	-0.101	0.044	-2.290	0.024	-0.189	-0.013	
HAZ4	Hb4	0.007	0.010	0.670	0.508	-0.014	0.027	0.040
(Aug)	constant	-1.106	1.201	-0.920	0.360	-3.493	1.281	
	age5	-0.088	0.044	-2.000	0.049	-0.175	0.000	
HAZ5	Hb5	0.005	0.009	0.510	0.614	-0.014	0.023	0.026
(Sep)	constant	-1.079	1.131	-0.950	0.343	-3.329	1.170	
	age6	-0.080	0.045	-1.790	0.077	-0.170	0.009	
HAZ6	Hb6	-0.004	0.010	-0.440	0.658	-0.024	0.015	0.018
(Oct)	constant	-0.238	1.165	-0.200	0.839	-2.555	2.080	
	age7	-0.083	0.044	-1.860	0.066	-0.171	0.006	
HAZ7	Hb7	0.009	0.010	0.930	0.353	-0.010	0.028	0.024
(Nov)	constant	-1.668	1.166	-1.430	0.156	-3.987	0.651	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	-0.269	0.046	-5.910	0.000	-0.360	-0.179	
WAZ1	L:C1	2.360	5.150	0.460	0.648	-7.881	12.600	0.282
(May)	constant	0.187	1.797	0.100	0.917	-3.386	3.760	
	age2	-0.276	0.045	-6.070	0.000	-0.366	-0.185	
WAZ2	L:C2	-10.910	3.193	-3.420	0.001	-17.258	-4.562	0.367
(June)	constant	4.785	1.114	4.290	0.000	2.570	7.001	
	age3	-0.296	0.043	-6.860	0.000	-0.382	-0.211	
WAZ3	L:C3	-17.330	5.421	-3.200	0.002	-28.108	-6.553	0.376
(July)	constant	7.155	1.879	3.810	0.000	3.419	10.891	
	age4	-0.279	0.044	-6.350	0.000	-0.366	-0.192	
WAZ4	L:C4	-15.599	4.592	-3.400	0.001	-24.728	-6.470	0.345
(Aug)	constant	6.476	1.633	3.960	0.000	3.229	9.724	
	age5	-0.268	0.045	-5.920	0.000	-0.358	-0.178	
WAZ5	L:C5	-27.217	7.204	-3.780	0.000	-41.539	-12.894	0.324
(Sep)	constant	10.269	2.479	4.140	0.000	5.340	15.198	
	age6	-0.228	0.046	-4.940	0.000	-0.320	-0.136	
WAZ6	L:C6	-12.916	3.515	-3.670	0.000	-19.904	-5.927	0.322
(Oct)	constant	5.279	1.235	4.280	0.000	2.824	7.735	
	age7	-0.251	0.048	-5.240	0.000	-0.347	-0.156	
WAZ7	L:C7	-10.296	4.901	-2.100	0.039	-20.039	-0.552	0.245
(Nov)	constant	4.759	1.784	2.670	0.009	1.213	8.305	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	-0.257	0.049	-5.200	0.000	-0.355	-0.159	
WAZ1	IgG1	-0.038	0.051	-0.740	0.459	-0.140	0.064	0.285
(May)	constant	1.137	0.408	2.780	0.007	0.325	1.949	
	age2	-0.292	0.057	-5.130	0.000	-0.405	-0.179	
WAZ2	IgG2	0.006	0.067	0.080	0.933	-0.127	0.138	0.280
(June)	constant	1.221	0.445	2.750	0.007	0.338	2.105	
	age3	-0.287	0.048	-5.990	0.000	-0.382	-0.192	
WAZ3	IgG3	0.001	0.053	0.020	0.985	-0.103	0.105	0.301
(July)	constant	1.296	0.480	2.700	0.008	0.342	2.250	
	age4	-0.260	0.049	-5.320	0.000	-0.357	-0.163	
WAZ4	IgG4	-0.014	0.059	-0.240	0.811	-0.132	0.104	0.257
(Aug)	constant	1.215	0.552	2.200	0.030	0.118	2.311	
	age5	-0.251	0.050	-5.040	0.000	-0.351	-0.152	
WAZ5	IgG5	0.036	0.054	0.670	0.507	-0.072	0.144	0.215
(Sep)	constant	0.943	0.609	1.550	0.125	-0.268	2.154	
	age6	-0.250	0.049	-5.070	0.000	-0.348	-0.152	
WAZ6	IgG6	-0.002	0.042	-0.050	0.963	-0.086	0.082	0.214
(Oct)	constant	1.261	0.713	1.770	0.081	-0.157	2.679	
	age7	-0.243	0.051	-4.730	0.000	-0.346	-0.141	
WAZ7	IgG7	0.001	0.039	0.030	0.974	-0.076	0.078	0.206
(Nov)	constant	1.251	0.662	1.890	0.062	-0.065	2.566	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	-0.268	0.047	-5.750	0.000	-0.361	-0.175	
WAZ1	AGP1	-0.114	0.338	-0.340	0.736	-0.786	0.557	0.281
(May)	constant	1.063	0.415	2.560	0.012	0.238	1.888	
	age2	-0.274	0.048	-5.720	0.000	-0.370	-0.179	
WAZ2	AGP2	-0.517	0.285	-1.810	0.074	-1.084	0.051	0.307
(June)	constant	1.568	0.458	3.420	0.001	0.657	2.479	
	age3	-0.287	0.045	-6.310	0.000	-0.378	-0.197	
WAZ3	AGP3	-0.227	0.361	-0.630	0.531	-0.945	0.491	0.305
(July)	constant	1.492	0.539	2.770	0.007	0.421	2.563	
	age4	-0.263	0.047	-5.650	0.000	-0.356	-0.170	
WAZ4	AGP4	0.174	0.482	0.360	0.720	-0.785	1.132	0.257
(Aug)	constant	1.033	0.602	1.720	0.090	-0.165	2.230	
	age5	-0.242	0.048	-5.060	0.000	-0.338	-0.147	
WAZ5	AGP5	-0.644	0.517	-1.240	0.217	-1.673	0.385	0.225
(Sep)	constant	1.540	0.653	2.360	0.021	0.241	2.838	
	age6	-0.232	0.047	-4.970	0.000	-0.324	-0.139	
WAZ6	AGP6	-0.985	0.293	-3.360	0.001	-1.569	-0.402	0.306
(Oct)	constant	1.894	0.603	3.140	0.002	0.695	3.093	
	age7	-0.244	0.050	-4.900	0.000	-0.343	-0.145	
WAZ7	AGP7	0.050	0.323	0.160	0.876	-0.593	0.694	0.206
(Nov)	constant	1.229	0.668	1.840	0.069	-0.099	2.558	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	-0.263	0.045	-5.820	0.000	-0.352	-0.173	
WAZ1	alb1	0.023	0.015	1.530	0.129	-0.007	0.053	0.300
(May)	constant	0.167	0.647	0.260	0.797	-1.119	1.452	
	age2	-0.292	0.048	-6.100	0.000	-0.388	-0.197	
WAZ2	alb2	-0.023	0.019	-1.230	0.222	-0.061	0.014	0.293
(June)	constant	1.994	0.750	2.660	0.009	0.502	3.486	
	age3	-0.290	0.046	-6.230	0.000	-0.382	-0.197	
WAZ3	alb3	-0.005	0.017	-0.320	0.748	-0.039	0.028	0.302
(July)	constant	1.521	0.818	1.860	0.066	-0.105	3.147	
	age4	-0.260	0.047	-5.550	0.000	-0.353	-0.167	
WAZ4	alb4	0.010	0.014	0.680	0.497	-0.018	0.037	0.260
(Aug)	constant	0.804	0.714	1.130	0.263	-0.616	2.224	
	age5	-0.240	0.049	-4.910	0.000	-0.337	-0.143	
WAZ5	alb5	0.009	0.021	0.430	0.667	-0.033	0.051	0.212
(Sep)	constant	0.753	0.994	0.760	0.451	-1.223	2.729	
	age6	-0.257	0.053	-4.850	0.000	-0.363	-0.152	
WAZ6	alb6	-0.006	0.017	-0.380	0.706	-0.040	0.027	0.216
(Oct)	constant	1.574	1.065	1.480	0.143	-0.543	3.691	
	age7	-0.242	0.049	-4.900	0.000	-0.340	-0.144	
WAZ7	alb7	-0.002	0.018	-0.110	0.909	-0.037	0.033	0.206
(Nov)	constant	1.314	0.827	1.590	0.116	-0.329	2.957	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	-0.271	0.045	-6.020	0.000	-0.361	-0.182	
WAZ1	Hb1	0.014	0.012	1.140	0.256	-0.010	0.038	0.291
(May)	constant	-0.474	1.334	-0.360	0.723	-3.126	2.177	
	age2	-0.288	0.048	-6.020	0.000	-0.383	-0.193	
WAZ2	Hb2	0.012	0.011	1.140	0.257	-0.009	0.034	0.291
(June)	constant	-0.073	1.220	-0.060	0.953	-2.500	2.354	
	age3	-0.289	0.045	-6.370	0.000	-0.380	-0.199	
WAZ3	Hb3	0.011	0.011	1.020	0.311	-0.011	0.033	0.310
(July)	constant	0.126	1.233	0.100	0.919	-2.325	2.576	
	age4	-0.265	0.046	-5.710	0.000	-0.357	-0.172	
WAZ4	Hb4	0.010	0.011	0.970	0.334	-0.011	0.032	0.264
(Aug)	constant	0.027	1.263	0.020	0.983	-2.484	2.538	
	age5	-0.241	0.049	-4.910	0.000	-0.339	-0.144	
WAZ5	Hb5	0.002	0.011	0.170	0.866	-0.019	0.023	0.206
(Sep)	constant	0.898	1.264	0.710	0.479	-1.615	3.412	
	age6	-0.247	0.049	-5.010	0.000	-0.345	-0.149	
WAZ6	Hb6	-0.007	0.011	-0.650	0.515	-0.028	0.014	0.218
(Oct)	constant	1.980	1.281	1.550	0.126	-0.566	4.527	
	age7	-0.243	0.050	-4.840	0.000	-0.342	-0.143	
WAZ7	Hb7	0.000	0.011	0.000	0.998	-0.022	0.022	0.201
(Nov)	constant	1.248	1.317	0.950	0.346	-1.371	3.868	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	-0.202	0.039	-5.230	0.000	-0.279	-0.125	
WHZ1	L:C1	-4.481	4.359	-1.030	0.307	-13.148	4.187	0.227
(May)	constant	3.159	1.521	2.080	0.041	0.135	6.184	
	age2	-0.241	0.039	-6.180	0.000	-0.318	-0.163	
WHZ2	L:C2	-4.764	2.741	-1.740	0.086	-10.215	0.686	0.322
(June)	constant	3.654	0.957	3.820	0.000	1.751	5.556	
	age3	-0.253	0.036	-7.030	0.000	-0.325	-0.181	
WHZ3	L:C3	-9.732	4.516	-2.160	0.034	-18.710	-0.754	0.366
(July)	constant	5.489	1.565	3.510	0.001	2.377	8.601	
	age4	-0.220	0.038	-5.790	0.000	-0.295	-0.144	
WHZ4	L:C4	-8.346	3.962	-2.110	0.038	-16.223	-0.468	0.280
(Aug)	constant	4.782	1.409	3.390	0.001	1.979	7.584	
	age5	-0.216	0.036	-6.010	0.000	-0.288	-0.145	
WHZ5	L:C5	-18.289	5.729	-3.190	0.002	-29.679	-6.899	0.313
(Sep)	constant	8.246	1.972	4.180	0.000	4.326	12.166	
	age6	-0.216	0.035	-6.240	0.000	-0.285	-0.147	
WHZ6	L:C6	-8.994	2.641	-3.410	0.001	-14.246	-3.742	0.387
(Oct)	constant	5.397	0.928	5.820	0.000	3.552	7.243	
	age7	-0.238	0.037	-6.450	0.000	-0.312	-0.165	
WHZ7	L:C7	-5.235	3.772	-1.390	0.169	-12.735	2.265	0.317
(Nov)	constant	4.518	1.373	3.290	0.001	1.789	7.248	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	-0.211	0.042	-5.010	0.000	-0.294	-0.127	
WHZ1	IgG1	0.033	0.044	0.760	0.447	-0.053	0.120	0.223
(May)	constant	1.503	0.347	4.330	0.000	0.812	2.193	
	age2	-0.269	0.046	-5.800	0.000	-0.361	-0.177	
WHZ2	IgG2	0.049	0.054	0.900	0.371	-0.059	0.157	0.305
(June)	constant	2.011	0.362	5.550	0.000	1.291	2.732	
	age3	-0.255	0.039	-6.620	0.000	-0.332	-0.179	
WHZ3	IgG3	0.029	0.042	0.670	0.502	-0.056	0.113	0.335
(July)	constant	2.103	0.387	5.440	0.000	1.334	2.872	
	age4	-0.211	0.041	-5.210	0.000	-0.292	-0.131	
WHZ4	IgG4	0.000	0.049	0.000	0.999	-0.098	0.098	0.243
(Aug)	constant	1.935	0.458	4.220	0.000	1.024	2.846	
	age5	-0.226	0.037	-6.030	0.000	-0.300	-0.151	
WHZ5	IgG5	0.110	0.041	2.700	0.008	0.029	0.190	0.292
(Sep)	constant	1.589	0.456	3.480	0.001	0.682	2.496	
	age6	-0.232	0.037	-6.340	0.000	-0.305	-0.159	
WHZ6	IgG6	0.016	0.032	0.510	0.609	-0.047	0.079	0.306
(Oct)	constant	2.444	0.530	4.610	0.000	1.391	3.498	
	age7	-0.250	0.039	-6.470	0.000	-0.327	-0.173	
WHZ7	IgG7	0.040	0.029	1.380	0.172	-0.018	0.098	0.316
(Nov)	constant	2.598	0.497	5.230	0.000	1.610	3.585	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	-0.195	0.040	-4.930	0.000	-0.274	-0.117	
WHZ1	AGP1	-0.070	0.288	-0.240	0.808	-0.642	0.502	0.218
(May)	constant	1.670	0.353	4.730	0.000	0.968	2.372	
	age2	-0.232	0.039	-5.960	0.000	-0.309	-0.154	
WHZ2	AGP2	-0.536	0.231	-2.320	0.023	-0.995	-0.077	0.340
(June)	constant	2.450	0.371	6.600	0.000	1.712	3.188	
	age3	-0.248	0.037	-6.720	0.000	-0.321	-0.174	
WHZ3	AGP3	-0.059	0.293	-0.200	0.840	-0.641	0.522	0.332
(July)	constant	2.251	0.436	5.160	0.000	1.383	3.119	
	age4	-0.213	0.038	-5.580	0.000	-0.288	-0.137	
WHZ4	AGP4	-0.645	0.395	-1.640	0.106	-1.430	0.139	0.266
(Aug)	constant	2.395	0.493	4.860	0.000	1.415	3.375	
	age5	-0.199	0.037	-5.350	0.000	-0.273	-0.125	
WHZ5	AGP5	-0.549	0.402	-1.370	0.176	-1.349	0.250	0.247
(Sep)	constant	2.458	0.508	4.840	0.000	1.449	3.467	
	age6	-0.223	0.036	-6.180	0.000	-0.295	-0.152	
WHZ6	AGP6	-0.436	0.228	-1.920	0.058	-0.889	0.016	0.332
(Oct)	constant	2.875	0.467	6.150	0.000	1.946	3.805	
	age7	-0.229	0.038	-6.060	0.000	-0.304	-0.154	
WHZ7	AGP7	-0.181	0.245	-0.740	0.462	-0.668	0.306	0.306
(Nov)	constant	2.828	0.506	5.590	0.000	1.823	3.834	

	Predictor	Coef.	Std. Err.	t	P		95% CI	Adj. R-sq
	age1	-0.192	0.039	-4.970	0.000	-0.269	-0.115	
WHZ1	alb1	0.014	0.013	1.100	0.273	-0.011	0.040	0.229
(May)	constant	1.118	0.554	2.020	0.047	0.017	2.219	
	age2	-0.247	0.040	-6.240	0.000	-0.326	-0.168	
WHZ2	alb2	-0.002	0.016	-0.150	0.883	-0.033	0.029	0.299
(June)	constant	2.177	0.620	3.510	0.001	0.944	3.410	
	age3	-0.253	0.037	-6.750	0.000	-0.327	-0.178	
WHZ3	alb3	-0.010	0.014	-0.730	0.464	-0.037	0.017	0.336
(July)	constant	2.608	0.659	3.950	0.000	1.297	3.919	
	age4	-0.220	0.038	-5.750	0.000	-0.295	-0.144	
WHZ4	alb4	-0.022	0.011	-1.880	0.063	-0.044	0.001	0.273
(Aug)	constant	2.728	0.583	4.680	0.000	1.569	3.887	
	age5	-0.196	0.038	-5.160	0.000	-0.272	-0.121	
WHZ5	alb5	0.011	0.016	0.670	0.504	-0.022	0.044	0.235
(Sep)	constant	1.660	0.773	2.150	0.035	0.124	3.196	
	age6	-0.248	0.039	-6.320	0.000	-0.326	-0.170	
WHZ6	alb6	-0.014	0.012	-1.120	0.267	-0.039	0.011	0.314
(Oct)	constant	3.310	0.787	4.200	0.000	1.744	4.876	
	age7	-0.233	0.037	-6.210	0.000	-0.307	-0.158	
WHZ7	alb7	-0.004	0.013	-0.270	0.791	-0.030	0.023	0.302
(Nov)	constant	2.840	0.627	4.530	0.000	1.593	4.086	
	Predictor	**Coef.**	**Std. Err.**	**t**	**P**		**95% CI**	**Adj. R-sq**
	age1	-0.198	0.039	-5.120	0.000	-0.274	-0.121	
WHZ1	Hb1	0.000	0.010	0.010	0.992	-0.021	0.021	0.218
(May)	constant	1.616	1.143	1.410	0.161	-0.656	3.889	
	age2	-0.247	0.040	-6.240	0.000	-0.325	-0.168	
WHZ2	Hb2	0.002	0.009	0.170	0.865	-0.016	0.019	0.299
(June)	constant	1.941	1.007	1.930	0.057	-0.062	3.944	
	age3	-0.247	0.037	-6.690	0.000	-0.320	-0.173	
WHZ3	Hb3	-0.003	0.009	-0.310	0.761	-0.021	0.015	0.332
(July)	constant	2.486	1.002	2.480	0.015	0.494	4.478	
	age4	-0.212	0.038	-5.510	0.000	-0.289	-0.136	
WHZ4	Hb4	0.009	0.009	0.950	0.345	-0.009	0.026	0.250
(Aug)	constant	1.018	1.049	0.970	0.335	-1.068	3.104	
	age5	-0.204	0.038	-5.360	0.000	-0.280	-0.128	
WHZ5	Hb5	-0.001	0.008	-0.130	0.893	-0.017	0.015	0.237
(Sep)	constant	2.264	0.981	2.310	0.023	0.314	4.214	
	age6	-0.230	0.037	-6.260	0.000	-0.303	-0.157	
WHZ6	Hb6	-0.004	0.008	-0.520	0.607	-0.020	0.012	0.306
(Oct)	constant	3.021	0.954	3.170	0.002	1.124	4.917	
	age7	-0.232	0.038	-6.120	0.000	-0.307	-0.157	
WHZ7	Hb7	-0.006	0.008	-0.730	0.469	-0.023	0.010	0.300
(Nov)	constant	3.369	0.997	3.380	0.001	1.387	5.350	

Appendix 14 - Relationships between mean biomarker and growth variables

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
HAZ	mean age	-0.090	0.041	-2.170	0.033	-0.172	-0.007	
	mean L:C	-18.100	6.793	-2.660	0.009	-31.607	-4.594	0.098
	constant	5.457	2.310	2.360	0.020	0.865	10.050	
	mean age	-0.068	0.047	-1.470	0.146	-0.161	0.024	
	mean IgG	-0.060	0.063	-0.950	0.342	-0.186	0.066	0.034
	constant	-0.333	0.525	-0.630	0.528	-1.376	0.710	
	mean age	-0.081	0.044	-1.850	0.068	-0.169	0.006	
	mean AGP	-0.303	0.664	-0.460	0.650	-1.623	1.018	0.025
	constant	-0.385	0.632	-0.610	0.544	-1.641	0.871	
	mean age	-0.075	0.044	-1.680	0.096	-0.163	0.014	
	mean Alb	0.030	0.031	0.970	0.337	-0.032	0.093	0.033
	constant	-1.738	1.277	-1.360	0.177	-4.276	0.801	
	mean age	-0.088	0.043	-2.050	0.043	-0.172	-0.003	
	mean Hb	0.014	0.013	1.130	0.263	-0.011	0.039	0.037
	constant	-2.080	1.402	-1.480	0.141	-4.867	0.707	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
WAZ	mean age	-0.270	0.042	-6.490	0.000	-0.353	-0.187	
	mean L:C	-30.350	6.847	-4.430	0.000	-43.964	-16.737	0.400
	constant	11.345	2.328	4.870	0.000	6.716	15.974	
	mean age	-0.267	0.050	-5.320	0.000	-0.367	-0.167	
	mean IgG	0.012	0.068	0.170	0.862	-0.124	0.148	0.261
	constant	1.163	0.567	2.050	0.043	0.037	2.290	
	mean age	-0.252	0.047	-5.360	0.000	-0.345	-0.158	
	mean AGP	-0.825	0.709	-1.160	0.248	-2.235	0.585	0.272
	constant	1.757	0.674	2.610	0.011	0.417	3.098	
	mean age	-0.267	0.048	-5.570	0.000	-0.362	-0.172	
	mean Alb	-0.007	0.034	-0.220	0.828	-0.075	0.060	0.261
	constant	1.494	1.379	1.080	0.282	-1.247	4.236	
	mean age	-0.265	0.046	-5.760	0.000	-0.357	-0.174	
	mean Hb	0.010	0.014	0.750	0.454	-0.017	0.037	0.266
	constant	0.136	1.512	0.090	0.929	-2.871	3.143	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
WHZ	mean age	-0.228	0.033	-6.980	0.000	-0.293	-0.163	
	mean L:C	-18.596	5.373	-3.460	0.001	-29.279	-7.912	0.397
	constant	8.389	1.827	4.590	0.000	4.757	12.022	
	mean age	-0.252	0.037	-6.750	0.000	-0.326	-0.178	
	mean IgG	0.094	0.051	1.850	0.068	-0.007	0.194	0.338
	constant	1.791	0.420	4.270	0.000	0.957	2.626	
	mean age	-0.216	0.036	-6.080	0.000	-0.287	-0.146	
	mean AGP	-0.530	0.537	-0.990	0.327	-1.597	0.538	0.319
	constant	2.531	0.510	4.960	0.000	1.516	3.546	
	mean age	-0.239	0.036	-6.690	0.000	-0.310	-0.168	
	mean Alb	-0.038	0.025	-1.520	0.133	-0.088	0.012	0.330
	constant	3.640	1.028	3.540	0.001	1.596	5.684	
	mean age	-0.224	0.035	-6.430	0.000	-0.294	-0.155	
	mean Hb	0.001	0.010	0.070	0.946	-0.020	0.021	0.312
	constant	2.108	1.146	1.840	0.069	-0.171	4.387	

Appendix 15 – Relationship between biomarker and growth variables using Time Series Analysis

	Predictor	Coef.	Std. Err.	z	P		95% CI	Rho
HAZ	age	-0.099	0.005	-19.760	0.000	-0.109	-0.089	
	L:C	-1.158	0.454	-2.550	0.011	-2.049	-0.268	0.945
	constant	-0.065	0.192	-0.340	0.736	-0.441	0.312	
	age	-0.092	0.006	-16.020	0.000	-0.103	-0.081	
	IgG	-0.011	0.005	-2.150	0.031	-0.021	-0.001	0.945
	constant	-0.446	0.113	-3.950	0.000	-0.667	-0.225	
	age	-0.098	0.005	-19.540	0.000	-0.108	-0.088	
	AGP	-0.036	0.031	-1.180	0.239	-0.097	0.024	0.945
	constant	-0.431	0.116	-3.720	0.000	-0.658	-0.204	
	age	-0.098	0.005	-19.330	0.000	-0.108	-0.088	
	Alb	0.000	0.001	0.160	0.872	-0.003	0.003	0.945
	constant	-0.469	0.121	-3.880	0.000	-0.706	-0.232	
	age	-0.098	0.005	-19.050	0.000	-0.108	-0.088	
	Hb	0.000	0.001	-0.140	0.888	-0.003	0.003	0.945
	constant	-0.437	0.178	-2.460	0.014	-0.785	-0.089	
	MULTIVARIATE MODEL							
	Predictor	**Coef.**	**Std. Err.**	**z**	**P**		**95% CI**	**Rho**
	age	-0.093	0.006	-16.260	0.000	-0.104	-0.082	
	L:C	-1.162	0.452	-2.570	0.010	-2.049	-0.276	0.945
	IgG	-0.011	0.005	-2.180	0.029	-0.021	-0.001	
	constant	-0.047	0.191	-0.250	0.805	-0.422	0.328	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Rho
WAZ	age	-0.155	0.006	-25.800	0.000	-0.167	-0.143	
	L:C	-2.029	0.545	-3.720	0.000	-3.097	-0.961	0.935
	constant	0.762	0.225	3.380	0.001	0.320	1.204	
	age	-0.148	0.007	-21.340	0.000	-0.161	-0.134	
	IgG	-0.010	0.006	-1.570	0.116	-0.022	0.002	0.936
	constant	0.080	0.128	0.630	0.530	-0.171	0.331	
	age	-0.154	0.006	-26.110	0.000	-0.165	-0.142	
	AGP	-0.205	0.036	-5.670	0.000	-0.275	-0.134	0.938
	constant	0.239	0.130	1.840	0.066	-0.016	0.493	
	age	-0.155	0.006	-25.490	0.000	-0.167	-0.143	
	Alb	0.004	0.002	2.360	0.018	0.001	0.008	0.936
	constant	-0.057	0.137	-0.410	0.681	-0.326	0.213	
	age	-0.151	0.006	-24.260	0.000	-0.163	-0.139	
	Hb	-0.002	0.002	-1.460	0.143	-0.006	0.001	0.936
	constant	0.310	0.209	1.480	0.138	-0.100	0.719	
	MULTIVARIATE MODEL							
	Predictor	**Coef.**	**Std. Err.**	**z**	**P**		**95% CI**	**Rho**
	age	-0.159	0.006	-27.090	0.000	-0.170	-0.147	
	L:C	-1.932	0.525	-3.680	0.000	-2.960	-0.904	
	AGP	-0.219	0.036	-6.110	0.000	-0.289	-0.149	0.939
	Alb	0.006	0.002	3.450	0.001	0.003	0.009	
	constant	0.739	0.224	3.300	0.001	0.300	1.178	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Rho
WHZ	age	-0.078	0.008	-9.720	0.000	-0.093	-0.062	
	L:C	-1.616	0.725	-2.230	0.026	-3.036	-0.196	0.839
	constant	1.201	0.277	4.340	0.000	0.659	1.743	
	age	-0.075	0.009	-8.250	0.000	-0.093	-0.057	
	IgG	-0.001	0.008	-0.090	0.925	-0.017	0.015	0.841
	constant	0.648	0.122	5.300	0.000	0.408	0.888	
	age	-0.076	0.008	-9.780	0.000	-0.092	-0.061	
	AGP	-0.233	0.048	-4.840	0.000	-0.327	-0.139	0.844
	constant	0.841	0.127	6.640	0.000	0.593	1.090	
	age	-0.078	0.008	-9.760	0.000	-0.094	-0.062	
	Alb	0.005	0.002	2.170	0.030	0.000	0.010	0.842
	constant	0.497	0.140	3.560	0.000	0.223	0.770	
	age	-0.073	0.008	-8.880	0.000	-0.089	-0.057	
	Hb	-0.003	0.002	-1.510	0.131	-0.008	0.001	0.843
	constant	0.971	0.248	3.910	0.000	0.485	1.457	
	MULTIVARIATE MODEL							
	Predictor	**Coef.**	**Std. Err.**	**z**	**P**		**95% CI**	**Rho**
	age	-0.081	0.008	-10.360	0.000	-0.096	-0.066	
	L:C	-1.499	0.705	-2.130	0.034	-2.881	-0.117	
	AGP	-0.251	0.048	-5.210	0.000	-0.346	-0.157	0.844
	Alb	0.007	0.002	3.040	0.002	0.002	0.012	
	constant	1.163	0.277	4.190	0.000	0.619	1.707	

Appendix 16 – Differences between control and intervention groups for all biomarker and growth variables on a month-by-month basis

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
	age1	-0.001	0.001	-1.010	0.313	-0.003	0.001	
L:C1	group	-0.003	0.005	-0.600	0.551	-0.012	0.006	0.000
(May)	constant	0.343	0.008	43.700	0.000	0.327	0.359	
	age2	0.001	0.002	0.740	0.463	-0.002	0.004	
L:C2	group	0.014	0.007	1.960	0.054	0.000	0.028	0.028
(June)	constant	0.320	0.014	23.370	0.000	0.292	0.347	
	age3	-0.001	0.001	-0.700	0.486	-0.002	0.001	
L:C3	group	0.004	0.004	1.060	0.291	-0.004	0.012	0.000
(July)	constant	0.336	0.009	39.320	0.000	0.319	0.353	
	age4	-0.001	0.001	-1.080	0.283	-0.003	0.001	
L:C4	group	0.010	0.005	2.190	0.031	0.001	0.020	0.042
(Aug)	constant	0.337	0.011	30.960	0.000	0.315	0.358	
	age5	-0.001	0.001	-1.460	0.148	-0.002	0.000	
L:C5	group	0.006	0.003	1.840	0.069	0.000	0.012	0.036
(Sep)	constant	0.334	0.008	43.270	0.000	0.319	0.350	
	age6	0.002	0.001	1.200	0.235	-0.001	0.004	
L:C6	group	0.003	0.007	0.480	0.631	-0.010	0.016	0.000
(Oct)	constant	0.311	0.018	17.600	0.000	0.276	0.346	
	age7	-0.001	0.001	-0.810	0.422	-0.003	0.001	
L:C7	group	0.006	0.005	1.210	0.230	-0.004	0.016	0.001
(Nov)	constant	0.337	0.014	23.990	0.000	0.310	0.365	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
	age1	0.379	0.092	4.130	0.000	0.197	0.562	
IgG1	group	1.153	0.436	2.640	0.010	0.286	2.020	0.209
(May)	constant	3.238	0.757	4.270	0.000	1.732	4.744	
	igg1	0.409	0.082	4.960	0.000	0.245	0.572	
	age2	0.295	0.076	3.870	0.000	0.144	0.447	0.423
IgG2	group	-0.408	0.344	-1.190	0.239	-1.092	0.276	
(June)	constant	0.647	0.675	0.960	0.341	-0.696	1.990	
	igg1	0.180	0.104	1.730	0.087	-0.027	0.387	
	age3	0.201	0.097	2.080	0.041	0.009	0.393	0.183
IgG3	group	1.084	0.435	2.490	0.015	0.218	1.950	
(July)	constant	2.395	0.920	2.600	0.011	0.566	4.224	
	igg1	0.413	0.087	4.750	0.000	0.240	0.586	
	age4	0.085	0.081	1.060	0.293	-0.075	0.245	0.311
IgG4	group	0.510	0.363	1.410	0.164	-0.212	1.233	
(Aug)	constant	2.779	0.817	3.400	0.001	1.153	4.404	
	igg1	0.207	0.101	2.050	0.044	0.006	0.408	
	age5	0.139	0.094	1.480	0.141	-0.047	0.326	0.250
IgG5	group	1.587	0.423	3.760	0.000	0.747	2.428	
(Sep)	constant	3.454	1.017	3.390	0.001	1.431	5.477	
	igg1	0.608	0.133	4.570	0.000	0.344	0.873	
	age6	-0.202	0.124	-1.630	0.107	-0.449	0.044	0.176
IgG6	group	-0.298	0.557	-0.540	0.594	-1.407	0.810	
(Oct)	constant	7.697	1.429	5.390	0.000	4.855	10.538	
	igg1	0.428	0.134	3.200	0.002	0.162	0.695	
	age7	0.233	0.124	1.880	0.064	-0.014	0.479	0.374
IgG7	group	2.532	0.561	4.510	0.000	1.415	3.648	
(Nov)	constant	1.525	1.523	1.000	0.319	-1.503	4.553	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
	age1	0.030	0.014	2.160	0.034	0.002	0.059	
AGP1	group	0.159	0.067	2.370	0.020	0.026	0.291	0.091
(May)	constant	0.535	0.116	4.600	0.000	0.304	0.766	
	agp1	0.163	0.138	1.180	0.241	-0.112	0.438	
	age2	0.024	0.018	1.290	0.201	-0.013	0.060	0.013
AGP2	group	-0.074	0.088	-0.840	0.404	-0.249	0.101	
(June)	constant	0.587	0.178	3.310	0.001	0.234	0.941	
	agp1	0.102	0.104	0.990	0.327	-0.104	0.309	
	age3	-0.006	0.014	-0.430	0.667	-0.034	0.022	0.000
AGP3	group	0.068	0.066	1.020	0.311	-0.064	0.199	
(July)	constant	0.760	0.144	5.280	0.000	0.473	1.046	
	agp1	0.106	0.080	1.320	0.191	-0.054	0.266	
	age4	-0.005	0.011	-0.500	0.621	-0.027	0.016	0.000
AGP4	group	-0.012	0.051	-0.240	0.809	-0.114	0.089	
(Aug)	constant	0.662	0.119	5.580	0.000	0.426	0.897	
	agp1	0.011	0.074	0.140	0.888	-0.137	0.158	
	age5	-0.001	0.010	-0.100	0.921	-0.021	0.019	0.063
AGP5	group	0.134	0.047	2.840	0.006	0.040	0.227	
(Sep)	constant	0.613	0.117	5.250	0.000	0.380	0.845	
	agp1	0.189	0.130	1.450	0.151	-0.070	0.448	
	age6	0.013	0.017	0.740	0.461	-0.022	0.048	0.006
AGP6	group	-0.068	0.083	-0.820	0.413	-0.233	0.097	
(Oct)	constant	0.603	0.219	2.760	0.007	0.168	1.039	
	agp1	0.146	0.126	1.150	0.251	-0.105	0.398	
	age7	0.024	0.017	1.410	0.162	-0.010	0.057	0.020
AGP7	group	0.029	0.081	0.360	0.721	-0.131	0.189	
(Nov)	constant	0.430	0.224	1.920	0.059	-0.016	0.875	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
	age1	-0.415	0.315	-1.320	0.191	-1.041	0.211	
Alb1	group	3.441	1.493	2.300	0.024	0.472	6.410	0.053
(May)	constant	34.502	2.594	13.300	0.000	29.344	39.659	
	alb1	0.026	0.094	0.280	0.781	-0.160	0.212	
	age2	-0.158	0.274	-0.580	0.566	-0.701	0.386	0.019
Alb2	group	2.576	1.327	1.940	0.056	-0.063	5.216	
(June)	constant	30.862	4.089	7.550	0.000	22.731	38.993	
	alb1	0.076	0.099	0.770	0.443	-0.120	0.272	
	age3	-0.531	0.290	-1.830	0.070	-1.107	0.045	0.064
Alb3	group	2.692	1.399	1.920	0.058	-0.089	5.474	
(July)	constant	36.795	4.512	8.150	0.000	27.823	45.768	
	alb1	0.033	0.125	0.260	0.792	-0.215	0.281	
	age4	-0.373	0.365	-1.020	0.310	-1.098	0.353	0.000
Alb4	group	0.444	1.767	0.250	0.802	-3.070	3.958	
(Aug)	constant	35.343	5.916	5.970	0.000	23.580	47.107	
	alb1	0.116	0.083	1.410	0.162	-0.048	0.281	
	age5	-0.310	0.242	-1.280	0.204	-0.791	0.172	0.063
Alb5	group	2.134	1.173	1.820	0.072	-0.198	4.466	
(Sep)	constant	33.686	4.079	8.260	0.000	25.575	41.796	
	alb1	0.137	0.108	1.270	0.209	-0.078	0.353	
	age6	-1.109	0.319	-3.480	0.001	-1.743	-0.476	0.125
Alb6	group	-0.894	1.537	-0.580	0.562	-3.950	2.162	
(Oct)	constant	47.119	5.579	8.450	0.000	36.025	58.213	
	alb1	0.140	0.102	1.360	0.177	-0.064	0.343	
	age7	0.386	0.299	1.290	0.200	-0.208	0.981	0.017
Alb7	group	1.189	1.452	0.820	0.415	-1.698	4.076	
(Nov)	constant	23.030	5.472	4.210	0.000	12.147	33.912	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
	age1	-0.032	0.399	-0.080	0.937	-0.825	0.762	
Hb1	group	-1.051	1.892	-0.560	0.580	-4.812	2.710	0.000
(May)	constant	105.516	3.286	32.110	0.000	98.982	112.050	
	age2	-0.047	0.477	-0.100	0.922	-0.995	0.902	
Hb2	group	-2.292	2.269	-1.010	0.315	-6.804	2.219	0.000
(June)	constant	106.674	4.335	24.610	0.000	98.054	115.293	
	age3	0.245	0.442	0.550	0.581	-0.634	1.124	
Hb3	group	0.404	2.093	0.190	0.847	-3.758	4.566	0.000
(July)	constant	103.284	4.409	23.430	0.000	94.518	112.050	
	age4	0.092	0.462	0.200	0.843	-0.827	1.012	
Hb4	group	2.290	2.197	1.040	0.300	-2.078	6.657	0.000
(Aug)	constant	106.659	5.002	21.320	0.000	96.713	116.605	
	age5	-0.183	0.508	-0.360	0.720	-1.193	0.827	
Hb5	group	2.056	2.404	0.860	0.395	-2.724	6.836	0.000
(Sep)	constant	106.648	5.966	17.880	0.000	94.784	118.512	
	age6	0.383	0.495	0.770	0.441	-0.601	1.367	
Hb6	group	1.908	2.341	0.820	0.417	-2.747	6.563	0.000
(Oct)	constant	104.282	6.202	16.810	0.000	91.950	116.613	
	age7	0.385	0.496	0.780	0.440	-0.602	1.372	
Hb7	group	-1.268	2.345	-0.540	0.590	-5.932	3.396	0.000
(Nov)	constant	104.358	6.648	15.700	0.000	91.138	117.579	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
	age1	-0.091	0.043	-2.120	0.037	-0.176	-0.005	
	group	-0.139	0.204	-0.680	0.496	-0.544	0.266	0.034
HAZ1	constant	-0.505	0.354	-1.430	0.157	-1.209	0.198	
	age2	-0.072	0.043	-1.660	0.101	-0.158	0.014	
	group	-0.276	0.207	-1.340	0.185	-0.687	0.135	0.030
HAZ2	constant	-0.536	0.395	-1.360	0.179	-1.321	0.250	
	age3	-0.078	0.044	-1.750	0.083	-0.166	0.010	
	group	-0.210	0.210	-1.000	0.319	-0.628	0.207	0.025
HAZ3	constant	-0.500	0.443	-1.130	0.261	-1.380	0.379	
	age4	-0.097	0.043	-2.240	0.028	-0.183	-0.011	
	group	-0.382	0.206	-1.860	0.067	-0.791	0.027	0.073
HAZ4	constant	-0.207	0.469	-0.440	0.660	-1.139	0.725	
	age5	-0.090	0.043	-2.120	0.037	-0.175	-0.006	
	group	-0.387	0.203	-1.910	0.059	-0.791	0.016	0.070
HAZ5	constant	-0.339	0.499	-0.680	0.499	-1.330	0.653	
	age6	-0.081	0.044	-1.830	0.071	-0.170	0.007	
	group	-0.248	0.210	-1.180	0.241	-0.666	0.170	0.031
HAZ6	constant	-0.578	0.556	-1.040	0.302	-1.684	0.529	
	age7	-0.079	0.044	-1.820	0.072	-0.166	0.007	
	group	-0.132	0.207	-0.640	0.527	-0.544	0.280	0.020
HAZ7	constant	-0.660	0.584	-1.130	0.261	-1.820	0.500	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
	age1	-0.271	0.045	-5.970	0.000	-0.361	-0.181	
	group	-0.081	0.215	-0.380	0.709	-0.508	0.347	0.281
WAZ1	constant	1.030	0.374	2.760	0.007	0.287	1.773	
	age2	-0.288	0.048	-5.970	0.000	-0.384	-0.192	
	group	-0.091	0.229	-0.400	0.693	-0.547	0.365	0.282
WAZ2	constant	1.272	0.438	2.900	0.005	0.400	2.144	
	age3	-0.286	0.046	-6.280	0.000	-0.376	-0.195	
	group	-0.120	0.216	-0.550	0.581	-0.548	0.309	0.304
WAZ3	constant	1.353	0.454	2.980	0.004	0.450	2.256	
	age4	-0.262	0.046	-5.650	0.000	-0.354	-0.169	
	group	-0.232	0.220	-1.050	0.295	-0.669	0.205	0.266
WAZ4	constant	1.256	0.501	2.510	0.014	0.260	2.252	
	age5	-0.240	0.048	-5.010	0.000	-0.335	-0.145	
	group	-0.310	0.227	-1.360	0.176	-0.762	0.142	0.228
WAZ5	constant	1.231	0.559	2.200	0.030	0.119	2.344	
	age6	-0.249	0.049	-5.090	0.000	-0.346	-0.152	
	group	-0.235	0.232	-1.010	0.313	-0.696	0.225	0.224
WAZ6	constant	1.354	0.614	2.210	0.030	0.134	2.574	
	age7	-0.242	0.049	-4.970	0.000	-0.339	-0.145	
	group	-0.231	0.232	-0.990	0.323	-0.692	0.231	0.215
WAZ7	constant	1.365	0.654	2.090	0.040	0.065	2.664	

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
	age1	-0.198	0.039	-5.140	0.000	-0.275	-0.122	
	group	0.089	0.183	0.490	0.629	-0.275	0.452	0.220
WHZ1	constant	1.588	0.318	5.000	0.000	0.956	2.220	
	age2	-0.249	0.039	-6.370	0.000	-0.327	-0.171	
	group	0.261	0.186	1.400	0.164	-0.109	0.631	0.314
WHZ2	constant	1.987	0.355	5.590	0.000	1.280	2.694	
	age3	-0.248	0.037	-6.770	0.000	-0.321	-0.175	
	group	0.144	0.174	0.830	0.409	-0.201	0.490	0.337
WHZ3	constant	2.136	0.366	5.830	0.000	1.408	2.864	
	age4	-0.213	0.039	-5.520	0.000	-0.289	-0.136	
	group	0.173	0.183	0.950	0.346	-0.190	0.537	0.250
WHZ4	constant	1.861	0.417	4.470	0.000	1.033	2.690	
	age5	-0.200	0.038	-5.320	0.000	-0.275	-0.126	
	group	0.077	0.179	0.430	0.669	-0.279	0.432	0.233
WHZ5	constant	2.059	0.440	4.680	0.000	1.184	2.934	
	age6	-0.232	0.037	-6.320	0.000	-0.305	-0.159	
	group	0.033	0.173	0.190	0.847	-0.311	0.378	0.304
WHZ6	constant	2.571	0.459	5.600	0.000	1.658	3.484	
	age7	-0.234	0.037	-6.290	0.000	-0.307	-0.160	
	group	-0.093	0.177	-0.520	0.602	-0.444	0.259	0.303
WHZ7	constant	2.780	0.498	5.580	0.000	1.790	3.770	

Appendix 17 – Differences between control and intervention groups for mean biomarker and growth variables

	Predictor	Coef.	Std. Err.	z	P		95% CI	Adj. R-sq
L:C	mean age	0.000	0.001	-0.370	0.713	-0.002	0.001	
	group	0.006	0.003	1.910	0.059	0.000	0.012	0.020
	constant	0.331	0.007	47.490	0.000	0.317	0.345	
IgG	IgG (baseline)	0.464	0.062	7.490	0.000	0.341	0.587	
	mean age	0.107	0.057	1.860	0.066	-0.007	0.221	
	group	0.718	0.259	2.770	0.007	0.203	1.233	0.558
	constant	2.598	0.582	4.460	0.000	1.440	3.756	
AGP	AGP (baseline)	0.245	0.046	5.280	0.000	0.153	0.337	
	mean age	0.007	0.006	1.140	0.255	-0.005	0.019	
	group	0.010	0.030	0.350	0.724	-0.048	0.069	0.281
	constant	0.527	0.068	7.700	0.000	0.391	0.663	
Alb	Alb (baseline)	0.218	0.043	5.090	0.000	0.133	0.303	
	mean age	-0.300	0.126	-2.390	0.019	-0.550	-0.050	
	group	1.166	0.608	1.920	0.059	-0.044	2.376	0.323
	constant	29.493	2.036	14.490	0.000	25.444	33.541	
Hb	mean age	0.114	0.369	0.310	0.758	-0.620	0.847	
	group	0.292	1.750	0.170	0.868	-3.188	3.772	0.000
	constant	105.631	3.985	26.510	0.000	97.708	113.554	
HAZ	mean age	-0.084	0.043	-1.980	0.051	-0.169	0.000	
	group	-0.254	0.202	-1.260	0.213	-0.656	0.148	0.040
	constant	-0.473	0.460	-1.030	0.307	-1.388	0.442	
WAZ	mean age	-0.263	0.046	-5.710	0.000	-0.354	-0.171	0.267
	group	-0.186	0.218	-0.850	0.397	-0.620	0.248	
	constant	1.295	0.497	2.610	0.011	0.307	2.283	
WHZ	mean age	-0.225	0.035	-6.460	0.000	-0.294	-0.156	
	group	0.098	0.165	0.600	0.553	-0.230	0.427	0.314
	constant	2.138	0.376	5.680	0.000	1.390	2.886	

Appendix 18 – Differences between control and intervention groups for biomarker and growth variables using Time Series Analysis

	Predictor	Coef.	Std. Err.	z	P		95% CI	Rho
L:C	age	-0.001	0.000	-2.000	0.045	-0.002	0.000	0.226
	group	0.006	0.003	1.960	0.050	0.000	0.012	
	constant	0.337	0.005	74.780	0.000	0.328	0.346	
IgG	IgG (baseline)	0.463	0.060	7.670	0.000	0.345	0.582	0.178
	age (baseline)	0.108	0.056	1.930	0.054	-0.002	0.218	
	group	-0.235	0.397	-0.590	0.555	-1.013	0.544	
	time	0.384	0.055	7.020	0.000	0.277	0.491	
	time*group	0.237	0.077	3.100	0.002	0.087	0.387	
	constant	1.349	0.514	2.620	0.009	0.341	2.357	
AGP	AGP (baseline)	0.253	0.045	5.610	0.000	0.164	0.341	0.027
	age	0.003	0.005	0.560	0.577	-0.007	0.012	
	group	0.010	0.029	0.340	0.733	-0.047	0.067	
	constant	0.567	0.057	9.910	0.000	0.455	0.679	
Alb	Alb (baseline)	0.234	0.043	5.390	0.000	0.149	0.319	0.033
	age	0.033	0.103	0.320	0.747	-0.168	0.235	
	group	1.060	0.618	1.710	0.086	-0.152	2.272	
	constant	25.568	1.867	13.690	0.000	21.909	29.227	
Hb	age	0.743	0.145	5.110	0.000	0.458	1.028	0.552
	group	0.198	1.749	0.110	0.910	-3.231	3.627	
	constant	99.179	1.945	50.980	0.000	95.366	102.992	
HAZ	age	-0.098	0.005	-19.490	0.000	-0.108	-0.088	0.944
	group	-0.252	0.199	-1.270	0.205	-0.641	0.138	
	constant	-0.333	0.151	-2.210	0.027	-0.630	-0.037	
WAZ	age (baseline)	-0.263	0.045	-5.820	0.000	-0.352	-0.175	0.932
	group	-0.068	0.219	-0.310	0.755	-0.497	0.361	
	time	-0.122	0.008	-15.670	0.000	-0.137	-0.107	
	time*group	-0.027	0.011	-2.500	0.012	-0.049	-0.006	
	constant	1.065	0.374	2.850	0.004	0.333	1.798	
WHZ	age (baseline)	-0.225	0.034	-6.570	0.000	-0.292	-0.158	0.814
	group	0.241	0.172	1.400	0.162	-0.097	0.579	
	time	-0.045	0.010	-4.330	0.000	-0.065	-0.024	
	time*group	-0.034	0.014	-2.350	0.019	-0.062	-0.006	
	constant	1.699	0.285	5.960	0.000	1.140	2.258	