

Low Birthweight – Nutrition policy discussion paper No. 18

Table of Contents

<u>Low Birthweight – Nutrition policy discussion paper No. 18</u>	1
<u>Preface</u>	1
<u>Acknowledgements</u>	2
<u>Foreword</u>	2
<u>Executive Summary</u>	3
<u>I. Introduction</u>	7
<u>Opening Remarks</u>	7
<u>Structure of the Report</u>	7
<u>Statement of the Problem</u>	8
<u>II. Epidemiology, Causes and Consequences of Low Birthweight</u>	10
<u>Levels, Patterns and Determinants of LBW in Developing Countries</u>	10
<u>Morbidity and Mortality Consequences of LBW in Neonates and Infants</u>	13
<u>Growth in LBW Children</u>	13
<u>Long-term Consequences of LBW: The Foetal Origins of Disease Hypothesis</u>	14
<u>III. Interventions to Reduce Low Birthweight: Global Experiences</u>	16
<u>History</u>	16
<u>Examples of LBW Prevention Programmes</u>	17
<u>Dietary Supplements During Pregnancy</u>	21
<u>The Role of Micronutrients in Improving Birthweight</u>	24
<u>IV. Nutritional Interventions to Reduce Low Birthweight: Critical Issues</u>	32
<u>Women's Body Composition</u>	32
<u>Pregnancy Weight Gain</u>	33
<u>Critical Moments for Intervention</u>	34
<u>Cultural Beliefs, Social Practices and Behavioural Change</u>	35
<u>V. Improving Outcomes of Low Birthweight Infants: Potential Interventions</u>	39
<u>VI. Conclusions and Future Research</u>	42
<u>Symposium Recommendations</u>	42
<u>Workshop Programme Recommendations</u>	43
<u>References</u>	45
<u>Appendix: Symposium Agenda</u>	56
<u>List of Participants</u>	57
<u>Publications – July 2000</u>	61
<u>Publications Order Form</u>	65

Low Birthweight – Nutrition policy discussion paper No. 18

Written and edited by

Judith Pojda and Laura Kelley

September 2000

United Nations Administrative Committee on Coordination
Sub-Committee on Nutrition
Nutrition Policy Paper No. 18. September 2000

Support from the Bangladesh Integrated Nutrition Project (BINP), Government of Bangladesh; UNICEF; collaborating members of USAID's Child Health Research Project: Harvard's Applied Research on Child Health Project, ICDDR,B: Centre for Health and Population Research, International Clinical Epidemiology Network, Johns Hopkins Family Health and Child Survival, World Health Organization Department of Child and Adolescent Health; and the World Bank (Nutrition Centre of Excellence, ICDDR,B) for sponsoring this symposium and workshop is gratefully acknowledged.

Preface

Administrative Committee on Coordination

Sub-Committee on Nutrition

(ACC/SCN) THE UN SYSTEM'S FORUM FOR NUTRITION

The Administrative Committee on Coordination (ACC), which is comprised of the heads of the UN Agencies, recommended the establishment of the Sub-Committee on Nutrition (SCN) in 1977, following the World Food Conference. This was approved by the Economic and Social Council of the UN (ECOSOC). The UN members of the SCN are the ADB, FAO, IAEA, IFAD, ILO, UN, UNAIDS, UNDP, UNEP, UNESCO, UNFPA, UNHCHR, UNHCR, UNICEF, UNRISD, UNU, WFP, WHO and the World Bank. From the outset, representatives of bilateral donor agencies have participated actively in SCN activities. The Secretariat is hosted by WHO in Geneva.

The **mandate of the ACC/SCN** is to serve as the UN focal point for harmonizing nutrition policies and strategies throughout the UN system, and to strengthen collaboration with partners for accelerated and more effective action against malnutrition. The **aim of the SCN** is to raise awareness of and concern for nutrition problems at global, regional and national levels; to refine the direction, increase the scale and strengthen the coherence and impact of actions against malnutrition worldwide; and to promote cooperation among UN agencies and partner organizations. The SCN's annual meetings have representation from UN Agencies, donor agencies and NGOs; these meetings begin with symposia on subjects of current importance for policy. The SCN brings such matters to the attention of the ACC and convenes working groups on specialized areas of nutrition. Initiatives are taken to promote coordinated activities – inter-agency programmes, meetings, publications – aimed at reducing malnutrition, primarily in developing countries. The SCN compiles and disseminates information on nutrition, reflecting the shared views of the agencies concerned. Regular reports on the world nutrition situation are issued. Nutrition Policy Papers are produced to summarize current knowledge on selected topics. *SCN News* is published twice a year, and the *RNIS* (Refugee Nutrition Information System report) is published quarterly.

Cover Design: Marie Arnaud Snackers

Photos: Laura Kelley

Suggested citation form for this report:

ACC/SCN (2000) *Low Birthweight: Report of a Meeting in Dhaka, Bangladesh on 14–17 June 1999*. Eds. Pojda J and Kelley L. Nutrition Policy Paper #18. Geneva: ACC/SCN in collaboration with ICDDR,B.

Readers are encouraged to review, abstract, reproduce or translate this document in part or in whole, but as copyright holder, the UN ACC/SCN Secretariat requests due acknowledgement. Use other than for educational or non-commercial purposes requires explicit prior authorization in writing from the ACC/SCN Secretariat. Original illustrations are the sole property of the ACC/SCN. The designations employed and the presentation of material in this publication does not imply the expression of any opinion whatsoever on the part of the ACC/SCN or its UN member agencies concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries.

To obtain further information on the Sub-Committee on Nutrition and its publications, please visit our website at <www.acc.unsystem.org/scn> or address all inquiries to:

ACC/SCN
c/o World Health Organization
20, Avenue Appia
CH-1211 Geneva 27
Switzerland
Email: accscn@who.int
Telephone: +41 22 791 0456
Fax: +41 22 798 8891

We gratefully acknowledge funding assistance from the Government of the Netherlands and USAID for the preparation and production of this Nutrition Policy Paper.

Acknowledgements

The report of the International Low Birth Weight (LBW) Symposium and Workshop, held at the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR,B): Centre for Health and Population Research on 14–17 June 1999, was prepared by Judith A Pojda, and Laura M Kelley with research assistance from Elizabeth Johnston. It was written from the lecture notes of all the presenters: Sultana Khanum, Robert Black, Fernando Barros, Caroline Fall and Shams El Arifeen on the epidemiology, causes, and consequences of LBW; Roger Shrimpton, Jane Kusin, Andrew Prentice and Usha Ramakrishnan on global experiences of interventions to reduce LBW; and Caroline Fall, Kathy Krasovec, Fernando Barros, Rae Galloway, Andrew Prentice, George Fuchs and Gretel Pelto on critical issues related to interventions to reduce LBW. We thank all of the chairs and rapporteurs of the sessions. We are especially grateful to the following reviewers for their comments and suggested revisions: Kathleen Kurz, H el ene Delisle and George Fuchs. Special thanks is also extended to George Fuchs and Shams el Arifeen for organizing the symposium and workshop.

The symposium and workshop were organized by ICDDR,B. Sponsors were: the Bangladesh Integrated Nutrition Project (BINP); UNICEF; the collaborating members of USAID's Child Health Research Project: Harvard's Applied Research on Child Health Project, ICDDR,B: Centre for Health and Population Research, the International Clinical Epidemiology Network (INCLIN), Johns Hopkins Family Health and Child Survival Cooperative Agreement, and the World Health Organization Department of Child and Adolescent Health; and the World Bank (Nutrition Centre of Excellence, ICDDR,B).

Foreword

Birthweight is a powerful predictor of infant growth and survival. Infants born with a low birthweight begin life immediately disadvantaged and face extremely poor survival rates. Approximately every ten seconds, an infant from a developing country dies from a disease or infection that can be attributed to low birthweight.

It is therefore encouraging that the international public health community has begun to increase its attention toward these four million infants who die each year and the many more who survive with a diminished quality of life. Low birthweight is a reasonably well-defined problem caused by factors that are potentially modifiable. Health care professionals can expect further research to lead to the development of an intervention or package of interventions that will impact on the rates of low birthweight infants and/or the sequelae of low

birthweight. This Low Birthweight Symposium and Workshop conducted in Dhaka, along with increased global efforts by other agencies, are helping to position low birthweight as an issue that merits attention and adequate resources.

Each year approximately 17 million infants are born with low birthweight in developing countries. Many of those infants who survive suffer cognitive and neurological impairment. Moreover, a child born with low birthweight has, in later life, a greater risk of illness and premature death from cardiovascular disease, hypertension, and diabetes compared to others with adequate birthweights. In fact, the great majority of low birthweight is believed to be directly linked to abnormalities that extend throughout the lifecycle. Low birthweight is an intergenerational problem in which low birthweight infants grow up to be undernourished and stunted children and adolescents and, ultimately, undernourished women of childbearing age, and undernourished pregnant women who themselves deliver low birthweight infants.

The costs of preventing many causes of low birthweight are well within reach, even in poor countries. However, due to the intergenerational characteristics of low birthweight, some successful interventions may require substantial programme and donor commitment over a sustained, extended period. This recognition, as well as a new sense of urgency, has underlined the need for greater attention to the problem of low birthweight. And new solutions are forthcoming from well-designed research on low birthweight.

This Nutrition Policy Paper was produced by the ACC/SCN in collaboration with the International Centre for Diarrhoeal Disease Research, Bangladesh: Centre for Health and Population Research. It summarizes the proceedings of the Low Birthweight Symposium and Workshop held in Dhaka in June 1999. Evidence regarding interventions to prevent low birthweight and to improve the health of infants affected with low birthweight is presented in this Nutrition Policy Paper. We hope this spurs policy-makers, donors and programme planners to take immediate action regarding this important public health problem.

*Richard Jolly
Chairman
ACC/SCN*

*George Fuchs
Associate Director
ICDDR,B: Centre for Health and Population*

Executive Summary

Low birthweight – the problem

Infants born with low birthweight (less than 2500 grams) suffer from extremely high rates of morbidity and mortality from infectious disease, and are underweight, stunted or wasted beginning in the neonatal period through childhood. Infants weighing 2000–2499 g at birth are 4 times more likely to die during their first 28 days of life than infants who weigh 2500–2999 g, and 10 times more likely to die than infants weighing 3000–3499 g. Low birthweight is associated with impaired immune function, poor cognitive development, and high risks of developing acute diarrhoea or pneumonia. It is estimated that in Bangladesh, almost half of the infant deaths from pneumonia or diarrhoea could be prevented if low birthweight were eliminated. Those low birthweight infants who survive have little chance of fully reaching their growth potential. Moreover, evidence now shows that adults born with low birthweight face an increased risk of chronic diseases including high blood pressure, non-insulin dependent diabetes mellitus, coronary heart disease and stroke in adulthood.

Low birthweight – the causes

Prematurity (born before 37 weeks of gestation) and intrauterine growth retardation (a condition where foetal growth has been constrained) are the two main causes of low birthweight. The majority of low birthweight in developing countries is due to intrauterine growth retardation, while most low birthweight in industrialized countries is due to preterm birth. In many cases, the causes of prematurity are unknown; they may include high maternal blood pressure, acute infections, hard physical work, multiple births, stress, anxiety, and other psychological factors.

Causes of intrauterine growth retardation are complex and multiple, but center on the foetus, the placenta, the mother, and combinations of all three. The maternal environment is the most important determinant of

birthweight, and factors that prevent normal circulation across the placenta cause poor nutrient and oxygen supply to the foetus, restricting growth. These factors may include maternal undernutrition, malaria (where it is endemic), anaemia, and acute and chronic infections (such as sexually transmitted diseases and urinary tract infections). Foetal, genetic or chromosomal anomalies; first-time births, multiple births, as well as maternal disorders such as renal diseases and hypertension are also associated with intrauterine growth retardation. Cigarette smoking and pre-eclampsia cause the highest relative risks for intrauterine growth retardation in industrialized countries, while alcohol and drug use may also restrict foetal growth.

Major determinants for low birthweight in developing countries, however, are poor maternal nutritional status at conception, low gestational weight gain due to inadequate dietary intake, and short maternal stature due to the mother's own childhood undernutrition and/or infection. Because maternal undernutrition is a major determinant of low birthweight in developing countries, high rates of low birthweight should be interpreted not merely as an indicator of undernutrition, morbidity and mortality for the newborn, but as an urgent public health warning that women of childbearing age are undernourished as well. Countries with higher percentages of low birthweight infants generally have a higher percentage of women with low body mass index and a higher percentage of underweight children. To address these issues successfully, the underlying and basic causes of low birthweight in developing countries such as household food security, maternal and child care, access to and quality of antenatal and other health services, sanitation and hygiene, education, gender discrimination and poverty must be included in any long-term strategies for prevention.

Premature infants and intrauterine growth retarded infants should be studied as separate groups because they show different patterns of growth, morbidity and mortality. From a programmatic viewpoint these differences have enormous implications on intervention strategies and limitations of the approach of nutritional recovery in early childhood.

Low birthweight – problem great enough to trigger public health action

At least 17 million infants are born every year with low birthweight, representing about 16% of all newborns in developing countries. Nearly 80% of all intrauterine growth retarded newborns who are low birthweight and full term are born in Asia (mainly south-central Asia, with Bangladesh having the highest rate in the world (50%)). About 15% and 11% are born full term with low birthweight and intrauterine growth retardation in middle and western Africa respectively, and approximately 7% in the Latin American and Caribbean region. Although there are many qualitative and quantitative limitations to available data, it is clear that many developing countries exceed the internationally recommended cut-off levels which should trigger public health action. Incident rates of >15% for low birthweight and >20% for intrauterine growth retardation indicate a major public health problem. Population-wide interventions aimed at prevention and improving infant health are therefore urgently required. One of the nutritional goals of the 1990 World Summit for Children was to reduce the prevalence of low birthweight to less than 10% by the year 2000 – needless to say, this remains a formidable challenge for the 21st century.

Low birthweight – the lifecycle

The causes and effects of low birthweight are complex and best considered within the lifecycle conceptual framework. Poor nutrition often begins in the intrauterine environment and extends throughout the lifecycle. Low birthweight is an intergenerational problem where low birthweight infants grow up to be undernourished and stunted children and adolescents and, ultimately, undernourished women of childbearing age, and undernourished pregnant women who deliver low birthweight infants. This amplifies risks to the individual's health and perpetuates the cycle of poverty, undernutrition and disease. This is especially so when adolescents become pregnant before their own growth is completed, leaving little to fulfil their own or their infant's nutritional requirements.

Undernutrition, evident by decreased maternal height (stunting), and below-normal prepregnancy weight and pregnancy weight gain, are among the strongest predictors of delivery of a low birthweight infant. There are few well-defined and proven effective nutritional interventions directed at adolescent girls and women of childbearing age and during pregnancy. For those interventions proven to be scientifically efficacious (e.g., dietary supplementation during pregnancy), programme demands (cost, logistics of implementation) are great.

The lifecycle is further taken into account by the foetal origins of disease hypothesis. This hypothesis (also known as the Barker hypothesis) states that foetal undernutrition at critical periods of development in the intrauterine environment and during infancy leads to permanent changes in body structure and metabolism. These changes result in increased adult susceptibility to coronary heart disease and non-insulin dependent diabetes mellitus. There is also growing evidence that those adults born with low birthweight suffer an

increased risk of high blood pressure, obstructive lung disease, high cholesterol and renal damage. Thus, a poorly growing foetus is an undernourished foetus prone to reduced growth, altered body proportions, and a number of metabolic and cardiovascular changes.

It has been suggested that these changes are adaptations for foetal survival in an inadequate nutritional environment, and that these changes persist post-natally, contributing to adult chronic disease when nutrients are plentiful. Thus, due to the intergenerational characteristics of low birthweight, which persist throughout the lifecycle, successful interventions will likely require substantial programme and donor commitment over a sustained, extended period of time.

Low birthweight – the role of nutrition

Intervention at any point has the potential to break the cycle of intergenerational undernutrition and low birthweight. Where resources are scarce, programmes designed to increase prepregnant weight and weight gain during pregnancy should be given priority. In many populations, food supply and birthweights are seasonal, so it is especially important to target interventions during hungry or wet seasons. During the last two trimesters of pregnancy, a woman should gain at least 1 kg per month for a total of 6 kg. Lack of weight gain during these last two trimesters, or weight loss is very detrimental to the foetus and/or mother and requires immediate action. Good nutrition throughout the lifecycle is critical. Breastfeeding, appropriate complementary feeding, and adequate micronutrient status are especially important during infancy and early childhood. It is less clear what can be done during the adolescent period to promote growth, particularly in height. Adolescents, however, should receive dietary counselling, especially if they are pregnant.

Evidence now exists to suggest that infant outcomes can be improved by improving maternal nutritional status. The most recent five year randomized controlled trial from the Gambia has reported that a high energy, antenatal dietary supplement can increase maternal weight gain, reduce LBW by 35%, and significantly reduce stillbirth and neonatal deaths by 55% and 40% respectively. Overall there was a 49% reduction in perinatal deaths and 40% reduction in early neonatal deaths but no effect on post-neonatal mortality. The 1999 meta-analysis by the Cochrane Review included a special note about the high energy supplement provided in the Gambian trial, which gave considerably larger effects regarding improved foetal growth and important statistically significant reductions in stillbirths and neonatal deaths than the other fourteen trials of balanced protein/energy supplements which were analyzed. Given the success of the Gambian trial's high energy supplement in reducing low birthweight and neonatal mortality, other trials of this sort are urgently needed.

Low birthweight – the role of micronutrient supplementation

Good micronutrient status is important to birth outcome. Micronutrient supplementation may cause an increase in maternal appetite, which may lead to increased food intake and/or reduced morbidity. Deficiency in one or more micronutrients may be due to inadequate food intake, poor dietary quality, or when micronutrients are not readily released from foods, not absorbed efficiently, or a combination of these factors. Thus, quite often in developing countries where low birthweight is prevalent, multiple micronutrient deficiencies co-exist and are likely to be of great public health concern. Over 100 studies have examined the role of micronutrients during pregnancy. Many of the earlier supplementation trials were conducted in industrialized countries among women who were not deficient and therefore less likely to benefit from the interventions. Some nutrients, namely vitamin A, calcium, iron and zinc, have been studied extensively, but much less is known about others, such as vitamin B complex. There is considerable variation in the methodologies used in the studies, ranging from cross-sectional and prospective studies to randomized, double-blind, placebo-controlled trials. Although multi-vitamin mineral supplements are prescribed and consumed regularly by women in many industrialized countries, there are few studies that have examined the benefits of these supplements on birthweight.

Unlike industrialized countries where zinc supplementation during pregnancy in certain populations has improved birthweight, no benefit has been observed from recent well designed trials in developing countries. Similarly, vitamin A supplementation does not appear to have an important role in improving birthweight while, in contrast, there is some evidence that iodine supplementation might be important. Folic acid can prevent neural tube defects, but evidence as to whether iron and/or folic acid supplements reduce the prevalence of low birthweight, prematurity and maternal mortality is limited. And while anaemia during pregnancy is associated with low birthweight, the benefit to birthweight of iron supplementation is less clear. Recent data also suggest benefits from multiple vitamin-mineral supplements, particularly with regard to decreasing adverse pregnancy outcomes. Although there is evidence of interactions among several micronutrients at the metabolic level, very little is known about the significance of these interactions for pregnancy outcomes.

The need for randomised, controlled studies are especially urgent in developing countries, i.e., among populations with high rates of low birthweight due to intrauterine growth retardation and where nutrient deficiencies do not occur in isolation and multiple micronutrient deficiencies are common. If multiple micronutrient supplementation is proven to be safe and efficacious in developing country settings, the practical programmatic implications need to be addressed such as supplement composition, cost and bioavailability, the mode and timing of delivery, and compliance. Fortification of common staples would also need to be considered as an alternative to supplements.

Low birthweight – behaviour change

It is widely recognized that cultural beliefs and practices play a role in the successful delivery of nutrition messages to the community. Communication is a central aspect of directed behaviour change, and communication, by definition, involves multiple parties. An important question is, *Whose cultural beliefs and practices will constrain or facilitate the implementation of interventions?* The multiple parties involve not only the recipients of the interventions – the women in the communities where low birthweight is endemic and their husbands, mothers, mothers-in-law, neighbours and community leaders – but the cultural beliefs of public health and nutrition professionals at different levels of the system must also be examined and understood. Some general principles as to how information on cultural beliefs and practices can be used in the design of interventions to prevent low birthweight include: (1) Work with local people to adapt dietary recommendations for locally available foods, identify appropriate analogies (an analogy that links a new idea with one that is already present in the culture helps to make this new information understandable and at least potentially actionable), and develop educational messages built on local concepts. (2) Avoid making recommendations that are *head-on collisions* with local cultural beliefs. Frame recommendations in terms of the cultural beliefs of the community. In addition to the challenges of obtaining information on local beliefs quickly and inexpensively, the concept of *non-shared assumptions* must be considered. This means health professionals engaging in cross-cultural communication which may run counter to their own beliefs and can present a major hurdle.

Low birthweight – the ‘packages’

Little has been implemented to date that translates existing information and recommendations into effective programmes for improving maternal nutrition and preventing low birthweight. Programmes to prevent low birthweight call for “packages” of interventions at various stages of the lifecycle. At minimum, low birthweight prevention programmes should have three components: (1) case management which increases the coverage of antenatal care and helps prevent repeated pregnancies in quick succession, (2) behavioural change communication strategies including those that defer pregnancy until after adolescence and to improve nutrition, and (3) linkages between existing health care facilities and the community. It is crucial to develop and implement intervention packages for low birthweight prevention and particularly for safe motherhood and reproductive health programmes, and for Integrated Management of Childhood Illness (IMCI) programmes. The workshop participants reflected the role of the lifecycle in their recommendations to improve health outcomes for low birthweight infants.

Low birthweight – conclusions

Low birthweight has multiple etiologies, however, intrauterine growth retardation accounts for the majority of low birthweight in developing countries. Many questions remain unanswered about intrauterine growth retardation, such as the appropriate timing, amount and characteristics of nutritional supplementation to improve growth; the role of micronutrient supplements; the impact of infection control on low birthweight prevention; and the full magnitude of health problems faced in adulthood by children born with low birthweight and intrauterine growth retardation. This ICDDR,B Symposium and Workshop in Dhaka highlighted the point that there is an urgent need to find answers on sustainable practices to improve women’s nutritional status prior to pregnancy, and their weight gain during pregnancy. These practices require behavioural change within households, and behavioural change and communication strategies have been a weak link in many programmes trying to address this issue. The lifecycle was used to view low birthweight determinants with respect to the different age and physiologic subgroups of the population to give priority to target groups and interventions. It was concluded that low birthweight solutions require packages of interventions, and that these need to be incorporated into all antenatal health care programmes and that both the number and coverage of these existing programmes be expanded. Care for women should be the foundation upon which all low birthweight prevention/reduction programmes are based. The symposium participants acknowledged the limitations of applying results of randomized controlled trials in industrialized countries to developing countries, and recommended that findings from observational studies not be discounted in order to determine programme effectiveness, if programme interventions are to move forward.

There is an urgent need to prevent low birthweight and its devastating sequelae. The identification of effective and practical interventions to prevent low birthweight and to improve the outcome of infants born with low birthweight, including those with a strong behavioural change component, would have an enormous impact on the health and productivity of individuals and society. Low birthweight is a problem that merits attention and adequate resources.



I. Introduction

Opening Remarks

Professor George Fuchs, Interim Director of ICDDR,B, welcomed participants and special guests: Honourable Salahuddin Yusuf, Minister of Health and Family Welfare of the People's Republic of Bangladesh; Mohammed Abu Hafiz, Director of the Bangladesh Integrated Nutrition Project; Milla McLachlan of the World Bank; and Roger Shrimpton of UNICEF. Professor Fuchs noted that the symposium presented an opportunity to review and address urgent, fundamental questions regarding prevention of LBW and its devastating sequelae. The Honourable Salahuddin Yusuf then expressed his appreciation for the symposium speakers and participants who have joined the Government of Bangladesh in positioning low birthweight as a high priority health issue. He stated, "The Government of Bangladesh is committed to better health for its people and will ensure all possible help to alleviate low birthweight." Dr McLachlan reported that four out of every ten infants born in Bangladesh will have low birthweight, and the social cost to Bangladesh of this preventable problem is enormous. According to a 1993 World Bank estimate, stunting alone will cost Bangladesh more than US\$10 billion in lost productivity over a ten year period.¹ Dr Shrimpton told the audience that UNICEF programme priorities include the intention to establish low birthweight reduction programmes in eleven countries, and made a plea that these deliberations yield pragmatic and constructive recommendations to improve low birthweight prevention programmes.

Structure of the Report

This report, which incorporates references to symposium presentations and discussion points made by participants, is a record of the meeting and is intended to be used as a reference for programme managers. It is also intended to serve as an information document for health professionals to influence policy and research funding. The meeting was held in two parts (see Appendix). The symposium agenda consisted of presentations by experts who reviewed state-of-the-art scientific evidence on a defined aspect of the multifaceted problem of low birthweight, concluding with a two-hour discussion session on future research and programme priorities. The workshop objective was to use this research to improve the Bangladesh Integrated Nutrition Programme (BINP). A small group, selected from the symposium participants, attended this two-day workshop which included a field trip to a BINP site.

Statement of the Problem

The period of intrauterine growth and development is one of the most vulnerable in the human lifecycle.² The weight of an infant at birth is an important indicator of maternal health and nutrition prior to, and during pregnancy, and a powerful predictor of infant growth and survival (Box 1). Infants born with low birthweight (LBW) suffer from extremely high rates of morbidity and mortality from infectious disease, and underweight, stunting or wasting beginning in the neonatal period through childhood. Every year approximately 17 million infants in developing countries are born with LBW³ and those infants who survive have little chance of fully reaching their growth potential. Moreover, evidence now shows that adults born with LBW face an increased risk of chronic diseases including high blood pressure, non–insulin dependent diabetes mellitus, coronary heart disease and stroke in adulthood.⁴

BOX 1. DEFINITIONS

Low Birthweight (LBW) – Low birthweight is defined as a body weight at birth of less than 2500 grams (g). There are two main causes for LBW: prematurity and intrauterine growth retardation (IUGR). LBW is often used as a proxy indicator to quantify the magnitude of IUGR in developing countries because valid assessment of gestational age is generally not available.

Premature – The term *premature* is used for infants born before 37 weeks gestation. Most, but not all premature infants weigh less than 2500 g. There are many reasons for premature delivery, however, in many cases the cause is unknown. Reasons include high maternal blood pressure, acute infections, multiple births, hard physical work, or stress. The word *preterm* may also be used to describe these infants.

Intrauterine Growth Retardation (IUGR) – Intrauterine growth retardation is a subtype of LBW of extraordinary importance to developing countries. IUGR is a condition where foetal growth has been constrained. An inadequate nutritional environment *in utero* can be one reason for this constrained growth. IUGR is usually assessed clinically when the foetus is born by relating the size of the newborn to the duration of the pregnancy using the 10th percentile of a reference population. A small size for gestational age indicates IUGR, or the inability of the foetus to reach its growth potential. Infants diagnosed with IUGR may be:

- (1) LBW at term (?37 weeks gestation and <2500 g);
- (2) preterm (<37 weeks gestation and weight less than the 10th percentile); or
- (3) IUGR at ?37 weeks gestation and weight less than the 10th percentile with a birthweight ?2500 g. (de Onis et al., 1998 *Eur J Cl Nutr* 52(S1))

Thus, because not all preterm infants are IUGR, LBW among preterm infants overestimates poor growth due to nutritional causes; and because some IUGR infants weigh more than 2500 g (the third classification), LBW at term underestimates the overall magnitude of the IUGR problem.

IUGR–LBW – In developing countries IUGR affects about two–thirds of infants born with LBW; the remaining one–third of these LBW infants are born preterm, some of whom are also affected with IUGR. (Arifeen, 1997) IUGR–LBW is used in some publications to refer only to IUGR infants who are LBW at term. IUGR infants born at term (?37 weeks gestation) with LBW (< 2500 g) are referred to in this publication as *LBW at term*.

Small for Gestational Age (SGA) – SGA infants have birthweights below a given low percentile cut–off for gestational age. SGA and IUGR are not strictly synonymous: some SGA infants (e.g., those born to short mothers) may represent merely the lower extreme of the “normal” foetal growth distribution, while other infants who meet the criteria for “appropriate for gestational age” may have actually been exposed to one or more growth–inhibiting factors. In individual cases, however, it is usually very difficult to ascertain whether or not the observed birthweight is the result of restricted *in utero* growth; classification of an infant as IUGR is thus based, *de facto*, on the established cut–off for SGA. (WHO, 1995, Report No. 854)

Undernutrition – In this report the term undernutrition refers collectively to stunting, underweight, wasting, low body mass index, and foetal growth retardation – conditions of inadequate nutrition.

Malnutrition – In this report the term malnutrition refers to both undernutrition and overnutrition – conditions of both deprivation and excess.

Chronic Energy Deficiency (CED) – “A steady state at which a person is in an energy balance although at a cost either in terms of increased risk to health or as an impairment of functions and health.” A BMI <18.5 kg/m² in adults indicates CED. (James et al., 1988 *Eur J Cl Nutr* 42(12))

Symposium participants recognized that the causes and effects of LBW are complex and best considered within the lifecycle conceptual framework (Figure 1). Poor nutrition often begins *in utero* and extends throughout the lifecycle. This amplifies risks to the individual’s health and increases the likelihood of damage to future generations through further foetal undernutrition. Undernutrition, manifested by decreased maternal height (stunting), and below-normal prepregnancy weight and pregnancy weight gain, are among the strongest predictors of delivery of a LBW infant. There are few well-defined and proven effective nutritional interventions directed at adolescent girls and women of childbearing age and during pregnancy. For those interventions proven to be scientifically efficacious (e.g., dietary supplementation during pregnancy), programme demands (cost, logistics of implementation) are great. Also, due to the intergenerational characteristics of the LBW problem, successful interventions will likely require substantial programme and donor commitment over a sustained, extended period of time.

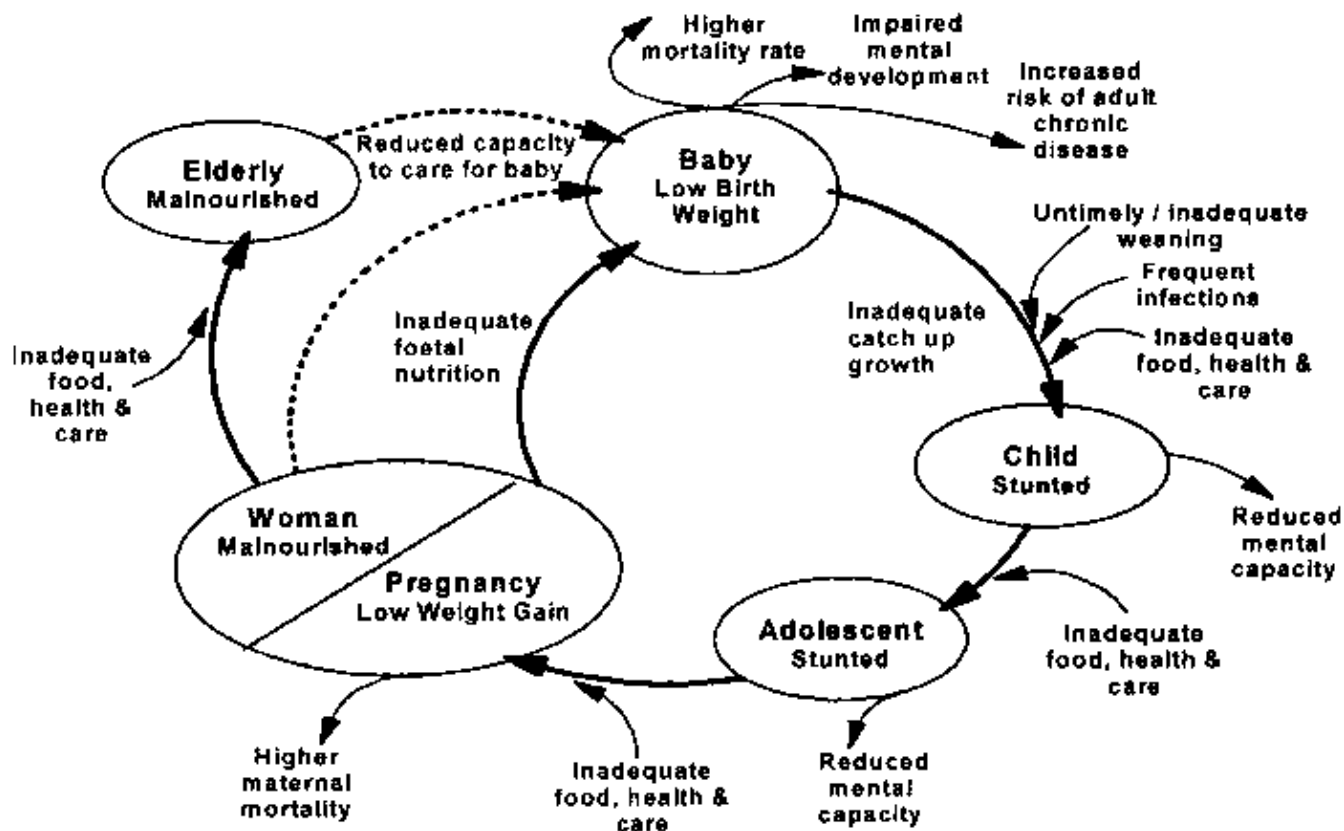


Figure 1. Nutrition throughout the lifecycle

Source: Commission on the Nutrition Challenges of the 21st Century (2000) Final report to the ACC/SCN.

There is an urgent need to answer basic and fundamental questions about the prevention of LBW and its devastating sequelae. The appropriate timing, amount and characteristics of nutritional supplementation; the role of micronutrients; the impact of infection control on LBW prevention; and the full magnitude of health problems faced by adults born with LBW are all poorly understood. Answers to these questions are fundamental to understanding the potential effect of various optional biological inputs. They are, however, only a prelude to investigating the sustainable ways in which these biological effects will be promoted and actually occur through the behaviours of people in households, service settings and communities. The identification of effective and practical interventions to prevent LBW and to improve the outcome of infants born with LBW, including those with a strong behavioural change component, would have an enormous impact on the health and productivity of individuals and society, particularly in those regions where the prevalence of LBW is high.

LBW perpetuates the intergenerational cycle of poverty, undernutrition and disease. This is especially so when adolescents become pregnant before their own growth is completed, leaving little to fulfil their own or their infant’s nutritional requirements. One of the nutritional goals of the 1990 World Summit for Children was

to reduce the prevalence of LBW to less than 10% by the year 2000 – needless to say, LBW remains a formidable challenge for the 21st century.

II. Epidemiology, Causes and Consequences of Low Birthweight

Levels, Patterns and Determinants of LBW in Developing Countries

At least 17 million infants are born every year with LBW, representing about 16% of all newborns in developing countries. Nearly 80% of all affected newborns with LBW *at term* are born in Asia (mainly south–central Asia, with Bangladesh having the highest incident rate in the world⁵); about 15% and 11% are born LBW *at term* in middle and western Africa respectively, and approximately 7% in the Latin American and Caribbean region.³ The geographical incidence of LBW *at term* in selected Asian and African countries (Figures 2 and 3 respectively) confirm that many developing countries exceed the internationally recommended cut–off levels which should trigger public health action. Incident rates of >15% for LBW and >20% for IUGR indicate that LBW *at term* is a major public health problem. Population–wide interventions aimed at preventing LBW *at term* are therefore urgently required.⁶

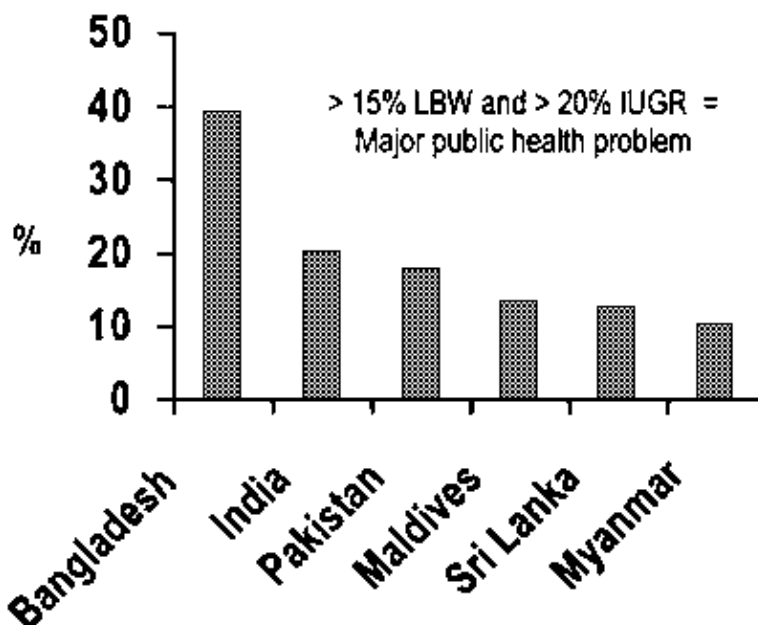


Figure 2. Incidence of LBW *at term* in selected Asian countries

Source: de Onis et al. (1998) *Eur J Cl Nutr* 52(S1):S5.

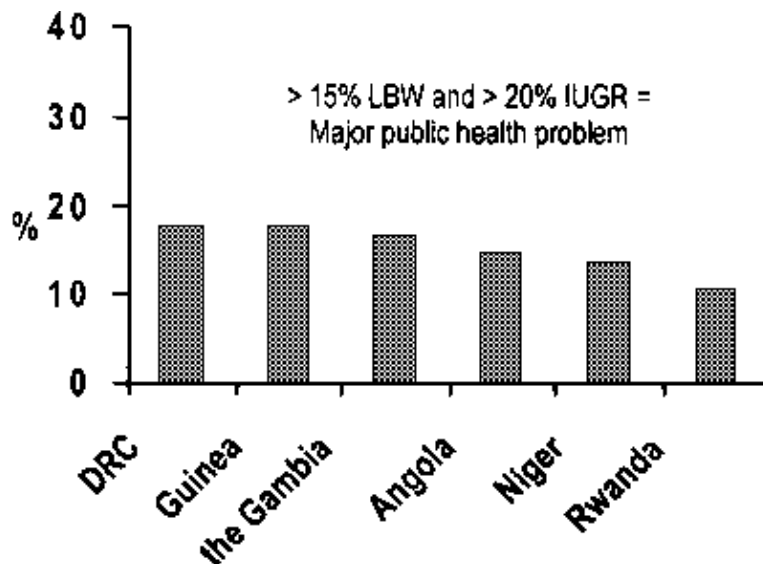


Figure 3. Incidence of LBW *at term* in selected African countries

Source: de Onis et al. (1998) *Eur J Cl Nutr* 52(S1):S5.

Historically, because valid assessment of gestational age is often not available in developing countries, evidence of LBW has often been used as a proxy to quantify the magnitude of IUGR. The incident rates for LBW *at term* conservatively estimate IUGR because when all infants below the 10th percentile of the birthweight-for-gestational-age reference are considered, approximately 24% or 30 million newborns in developing countries would be affected each year. Major constraints to deriving this estimate include both the quantitative and qualitative limitations of the available birthweight data.⁶ Most of the data available from different parts of the world are from clinic or hospital deliveries, whereas, in some regions of Africa and south-east Asia most infants are born at home and are not measured. There is a need to determine whether data from hospital-born infants in developing countries are representative of the large population born at home.⁶

Prematurity and IUGR are the two main causes of LBW. The majority of LBW in developing countries is due to IUGR, while most LBW in industrialized countries is due to preterm birth.⁷ In many cases, the causes of prematurity are unknown; they may include high maternal blood pressure, acute infections, hard physical work, multiple births, stress, anxiety, and other psychological factors.^{8,9} Causes of IUGR are complex and multiple, but center on the foetus, the placenta, the mother, and combinations of all three. For instance, growth will be retarded *in utero* if the placenta is abnormally small or blocked causing insufficient nutrients to reach the foetus. The maternal environment is the most important determinant of birthweight, and factors that prevent normal circulation across the placenta cause poor nutrient and oxygen supply to the foetus, restricting growth. These factors may include maternal undernutrition, malaria (where it is endemic), anaemia, and acute and chronic infections (such as sexually transmitted diseases and urinary tract infections).⁹ Also associated with IUGR are primiparity; multiple gestation; foetal, genetic or chromosomal anomalies; as well as maternal disorders such as renal diseases and hypertension.¹⁰ Cigarette smoking and pre-eclampsia cause the highest relative risks for IUGR in industrialized countries, while alcohol and drug use may also restrict foetal growth.^{8, 11}

Major determinants for LBW in developing countries, however, are poor maternal nutritional status at conception, low gestational weight gain due to inadequate dietary intake, and short maternal stature due to the mother's own childhood undernutrition and/or infection (Box 2).¹² Because maternal undernutrition is a major determinant of LBW in developing countries, high rates of LBW should be interpreted not merely as an indicator of undernutrition, morbidity and mortality for the newborn, but as an urgent public health warning that women of childbearing age are undernourished as well. Countries with higher percentages of LBW infants generally have a higher percentage of women with low body mass index (BMI) and a higher percentage of underweight children.^{13,14} To address these issues successfully, the underlying and basic causes of LBW in developing countries such as household food security, maternal and child care, access to and quality of antenatal and other health services, sanitation and hygiene, education, gender discrimination and poverty must be included in any long-term strategies for prevention (Figure 4).

Box 2. Determinants of LBW in Developing Countries

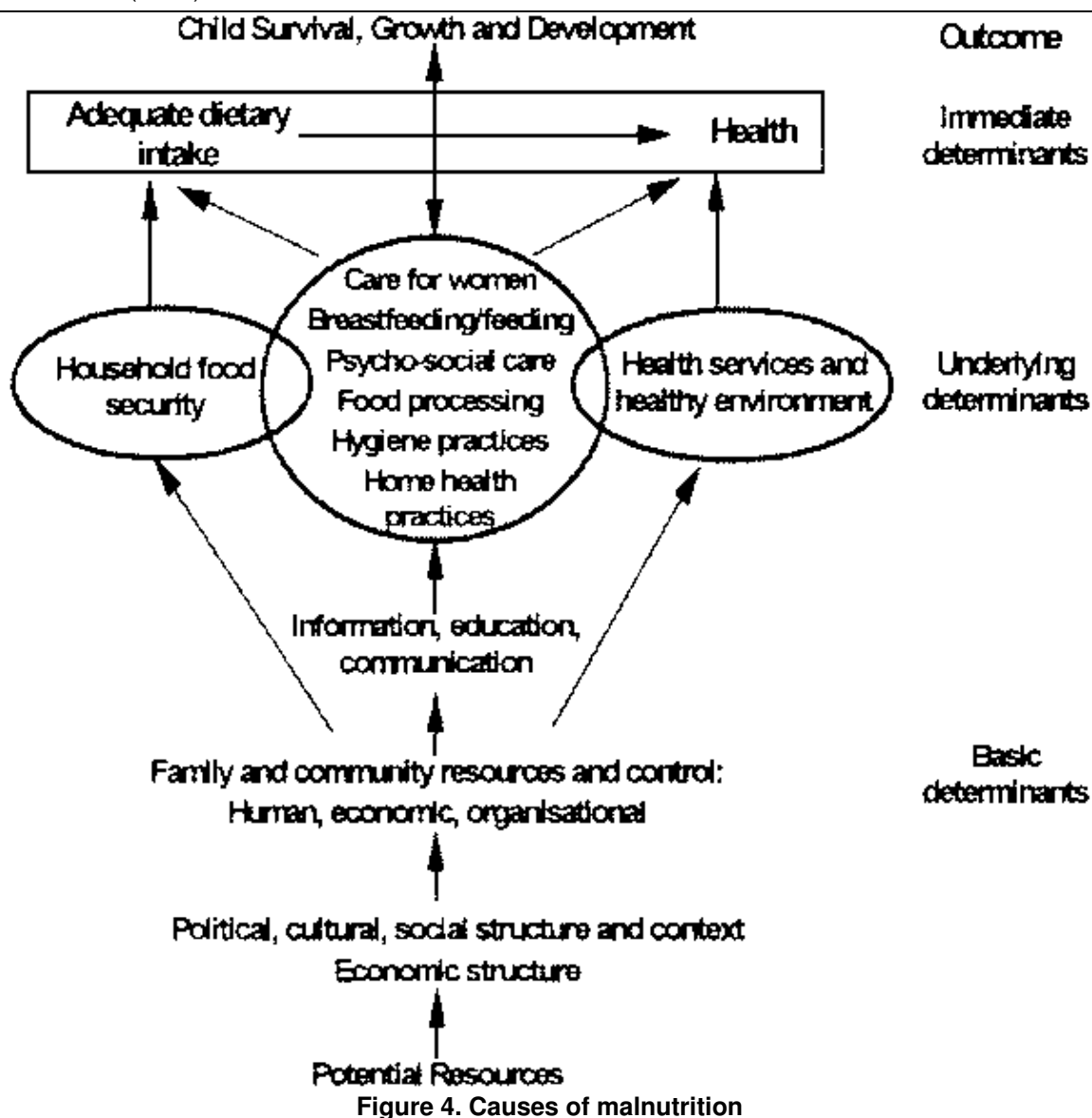
Maternal undernutrition – a major determinant of LBW in developing countries as evidenced by the following nutritional deficiencies:

- ? Low gestational weight gain
- ? Low pre-pregnancy body mass index (BMI)
- ? Short maternal stature
- ? Micronutrient deficiencies

Other etiologic determinants include:

- ? Young maternal age (adolescent)
- ? Malaria during pregnancy
- ? Gastro-intestinal, respiratory intestinal parasitosis, and/or other infections
- ? Cigarette smoking

Source: Kramer (1987) *Bull WHO* 65:663.



Source: UNICEF (1997) *The Care Initiative: Assessment, Analysis and Action to Improve Care for Nutrition*, UNICEF: NY.

Morbidity and Mortality Consequences of LBW in Neonates and Infants

LBW is generally associated with increased morbidity and mortality, impaired immune function, and poor cognitive development for neonates (newborns 1–28 days of age) and infants. Infants born LBW are at risk to develop acute diarrhoea or to be hospitalized for diarrhoeal episodes at a rate almost two to four times greater than their normal birthweight counterparts.^{15–18} Infants who are LBW risk contracting pneumonia or acute lower respiratory infections (ALRI) at a rate almost twice that of infants with normal birthweight; and more than three times greater if their weight is less than 2000 g.^{17–20} LBW is also implicated as a contributor to impaired immune function which may be sustained throughout childhood.^{21–23}

The risk of neonatal death for infants who are LBW weighing 2000–2499 g at birth is estimated to be four times higher than for infants weighing 2500–2999 g, and ten times higher than for infants weighing 3000–3499 g.²⁴ In Brazil, 67% of all infants dying during their first week of life are LBW infants; in Indonesia the rate is 40%; and in the Sudan the rate is 35%. Infant mortality (less than one year of age) due to LBW was slightly lower: 47% in Brazil and 19% in Indonesia.^{25–27} LBW infants during the post–neonatal period (>28 days of age) also have high mortality rates –and in some cases their risk may be greater than those for LBW infants during the neonatal period.^{5,28} LBW accounted for 69% of the ALRI deaths in India, and it is estimated that in Bangladesh, almost half of the infant deaths from pneumonia or ALRI and diarrhoea could be prevented if LBW were eliminated.^{5,29}

Growth in LBW Children

Do LBW infants grow normally? What are the consequences of LBW on body size, composition, strength and cognitive development? Attaining full growth potential is especially important for women and girls in order to break the intergenerational cycle of LBW and have fewer delivery complications. Maternal height is not only a reflection of genetic make–up, but also reflects her dietary history. From societal, community and individual standpoints, adolescents and adults born with LBW generally have less strength and lower lean body mass resulting in decreased work capacity and lost productivity, which may cost nations billions of dollars.^{1,30}

When growth restriction *in utero* occurs early in pregnancy, infants exhibit symmetrical (or proportional) growth with length, weight, head and abdominal circumference all below the 10th percentile reference for a given gestational age (stunting). When growth restriction *in utero* occurs late in pregnancy, the infant exhibits asymmetrical (or disproportionate) growth with a normal length and head circumference, but low weight due mainly to a lower proportion of visceral and fat tissue (wasting).^{7,31} Neonatal mortality rates are reported to be higher among asymmetrical IUGR infants, but if they survive, they have a better prognosis for long–term growth and development than that for symmetrical IUGR infants. IUGR infants catch–up partially in growth relative to their appropriate birthweight counterparts during their first one or two years of life. Thereafter, IUGR children maintain their place in the distribution and neither catch–up nor fall further behind. They remain about 5 cm shorter and 5 kg lighter as adults. Premature infants (who are usually asymmetric LBW), who survive their first year, have a much better prognosis in terms of future growth than IUGR infants. Despite their earlier disadvantage, preterm children gradually catch–up with their appropriate birthweight, term counterparts. Premature infants and IUGR infants should be studied as separate groups because they show different patterns of growth, morbidity and mortality. From a programmatic viewpoint these differences have enormous implications for intervention strategies and limitations of the approach of nutritional recovery of IUGR infants in early childhood.^{10,25,30,32–36}

Neurological dysfunction is often associated with attention deficit disorders, hyperactivity, clumsiness, and poor school performance. Neurologic dysfunction, when present, seems to affect IUGR boys more than girls, and children of lower socioeconomic circumstances. If IUGR infants are symmetrical and head growth is affected, there seems to be more of an impact on neurological function and it is not clear whether interventions directed toward these infants will improve their outcome. For asymmetric IUGR infants, preventing asphyxia should reduce the prevalence of major and minor handicaps, especially cerebral palsy and mental impairment frequently seen in these infants.^{37,38} IUGR is a much larger public health problem in developing countries than in industrialized countries and the outcomes are more likely to be aggravated by obstetric complications and perinatal problems, and later by poor health and nutrition as well as psycho–social deprivation.³⁷

In developing countries children are exposed to poor nutrition, high levels of infections, and other conditions of poverty, thus, their long term development is dependent to a large extent on the quality of their environment. It is difficult to isolate the effects of IUGR from these factors in relation to cognitive development. Cognitive

deficits appear to change over time. For instance, when IUGR infants were examined, no differences were found during the first year of life, but differences emerged during two and three years of age; and then differences disappeared at four to five years. Deficits have been found in children with very low birthweights, the smallest size, or with early IUGR (growth restriction prior to 26 weeks gestation). Since LBW occurs more often in deprived environments, it can serve as a marker for the associated poor outcomes throughout life. A length deficit at an early age (stunting) would be the best predictor of motor and mental development deficits.^{39,40}

Long-term Consequences of LBW: The Foetal Origins of Disease Hypothesis

The foetal origins of disease hypothesis states that foetal undernutrition at critical periods of development *in utero* and during infancy leads to permanent changes in body structure and metabolism. These changes result in increased adult susceptibility to coronary heart disease (CHD) and non-insulin dependent diabetes mellitus (NIDDM). There is also growing evidence that those adults born with LBW suffer an increased risk of high blood pressure, obstructive lung disease, high blood cholesterol and renal damage. Thus, a poorly growing foetus is an undernourished foetus prone to reduced growth, altered body proportions, and a number of metabolic and cardiovascular changes. It has been suggested that these changes are adaptations for foetal survival in an inadequate nutritional environment, and that these changes persist post-natally, contributing to adult chronic disease when nutrients are plentiful.⁴

The foetal origins of disease hypothesis, also known as the Barker hypothesis, was generated by David JP Barker and colleagues of the MRC Environmental Epidemiology Unit of the University of Southampton. Barker's group was puzzled that CHD was the most common cause of death among certain men who otherwise had low risk characteristics, i.e., they were slim, non-smokers, and had low blood cholesterol. This suggested that the etiology of CHD needed further exploration. The group speculated that foetal undernutrition during the first trimester may result in a proportionately small (symmetrical or stunted) infant prone to haemorrhagic stroke. Foetal undernutrition during the latter stages of pregnancy may result in a disproportionate (asymmetrical or thin) infant prone to CHD and an increased risk of insulin resistance, or a short infant prone to CHD and thrombotic stroke.⁴¹

The foetal origins hypothesis originated in the 1980's when Dr. Barker replicated a study from Norway which demonstrated a strong correlation between infant mortality rates (IMR) at the beginning of the century with current death rates from CHD. The author of the Norwegian study suggested that because infant mortality is a sensitive indicator of the quality of the immediate post-natal environment, perhaps growing up in poverty causes "some sort of deficit" which results in a "lifelong vulnerability" to aspects of an affluent adult lifestyle such as a high fat diet.⁴² Dr. Barker found a similar correlation between IMR and death from CHD in England and Wales but suggested, however, that since CHD was more closely correlated with neonatal mortality than with post-neonatal mortality, CHD may find some of its roots in IUGR as reflected by LBW.⁴³ The historical evolution of the Southampton group's research included a move from geographical associations, to associations related to individuals, then to biological risk factors. Birth records from Hertfordshire, UK, were first used to study mortality in relation to birthweight. The study showed that the highest death rates were in men and women who had the lowest birthweights, and death rates fell as birthweight increased. This pattern was specific for CHD and chronic obstructive lung disease.⁴⁴ A similar pattern was found for biological risk factors (hypertension and impaired glucose tolerance (IGT) and diabetes) for CHD in men: the highest rates were in men who had been small infants. More than 20% of men whose birthweights were lower than 2500 g had abnormal glucose tolerance, compared with under 10% of those weighing more than 4000 g at birth.⁴⁵ These study results have now been replicated by several groups in many different countries including the USA, Sweden, Finland, India and China.^{41,46-53}

The Barker research group also proposed a foetal programming hypothesis in which there is a "brain-sparing" reflex that, in an undernourished foetus, diverts or conserves the blood flow to the head, while simultaneously reducing the blood flow to the liver, pancreas and kidneys. This results in a reduced secretion of growth hormones, insulin and other endocrine changes which leads to CHD and NIDDM in adulthood (Figure 5). The Barker theory remains hypothetical since no causal relationships have yet been established, only associations. Two other explanations for the association between LBW and adult disease include the 'confounding' explanation (Figure 6) and a genetic explanation (Figure 7). The 'confounding explanation' suggests that LBW is a marker for poor socioeconomic status: poor people have smaller infants who are more likely to smoke, be exposed to stress, grow up with inadequate nutrition and become obese – all factors which cause CHD.⁵⁴ The genetic explanation, on the other hand, suggests that if an individual has a gene for insulin resistance, this would lead to LBW, and the same genetic pre-disposition would lead to an increased risk of

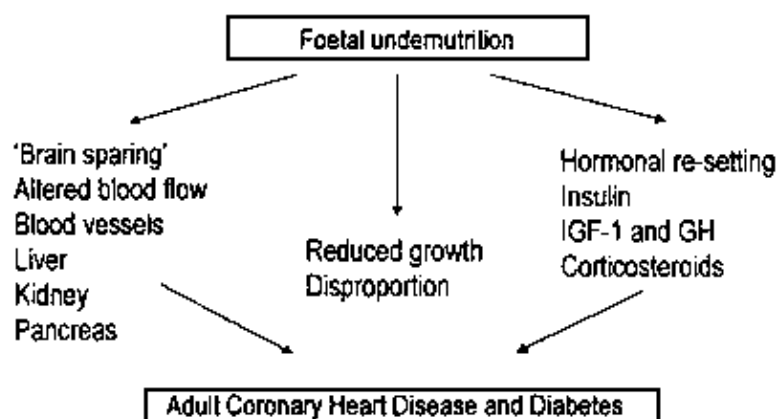


Figure 5. LBW and adult disease 'Foetal Programming' Hypothesis

Source: Fall (1999) Personal communication.

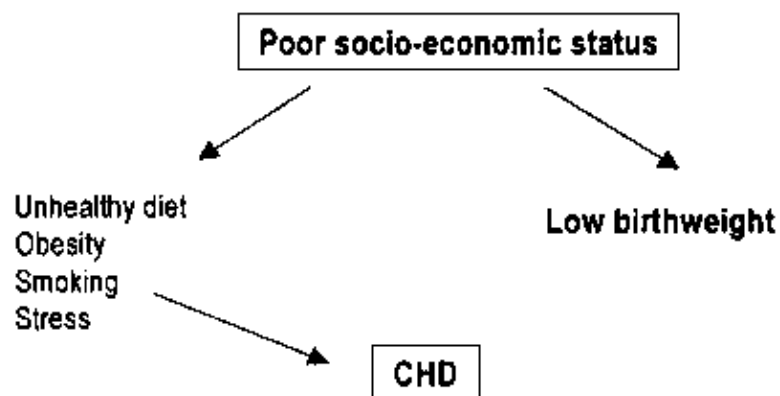


Figure 6. LBW and adult disease 'confounding' explanation

Source: Whincup (1997) *Diab* 40:319.

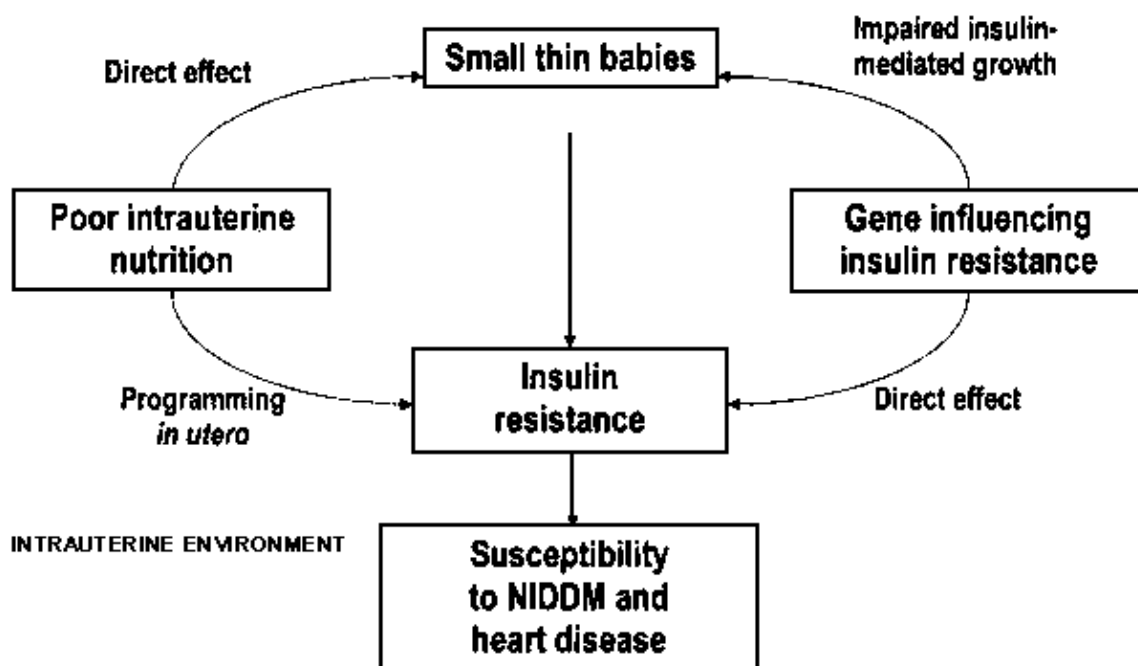


Figure 7. LBW and adult disease 'genetic' explanation

Source: Hattersley (1999) *Lancet* 353:1789.

Lack of information on possible confounding lifestyle and environmental factors, limitation of the initial Barker studies to two populations in the UK, the retrospective nature of the observations, and differences in study

methodologies all underscore the need to establish a core research protocol to investigate a longitudinal relationship between LBW due to poor foetal growth and disease in later life.⁵⁶ The foetal origins theory appears to be of greatest relevance to developing countries where mean birthweights remain low and rates of LBW are high. Many of these countries are experiencing a nutrition transition which includes changes in dietary intake, physical activity and body composition. The nutrition transition refers to a shift to diets high in total fat, sugar, and refined grains; it includes a more sedentary lifestyle; and increased use of tobacco products. Simultaneously an epidemiological transition is occurring in these countries. This is evident by a shift away from the high prevalence of infectious disease and undernutrition as causes of mortality to a high prevalence of chronic and degenerative diseases – conditions made worse by the nutrition transition. This raises urgent concerns regarding prevention of the already burdensome and growing ‘epidemic’ of CHD in these countries (Figure 8) because LBW, especially in association with increased body fat, either as an adult or as a child, leads to insulin resistance, and an increased risk of CHD. Regardless of the controversy over the foetal origins theory, the fact remains that “the effects of malnourishment at different stages of gestation are poorly understood”.⁴¹ ***“The foetal origins theory leaves the scientific community with unanswered questions, although waiting for these and other answers should not delay the programme implementation of those interventions that have already been shown to be, or are likely to be, efficacious against low birthweight.”***⁵⁷ The role of adequate pre-pregnancy weight has been established as a determinant of LBW in developing countries, so improvement in nutrition of young girls and women is very probably one important step toward the prevention of LBW and its accompanying disease burden.

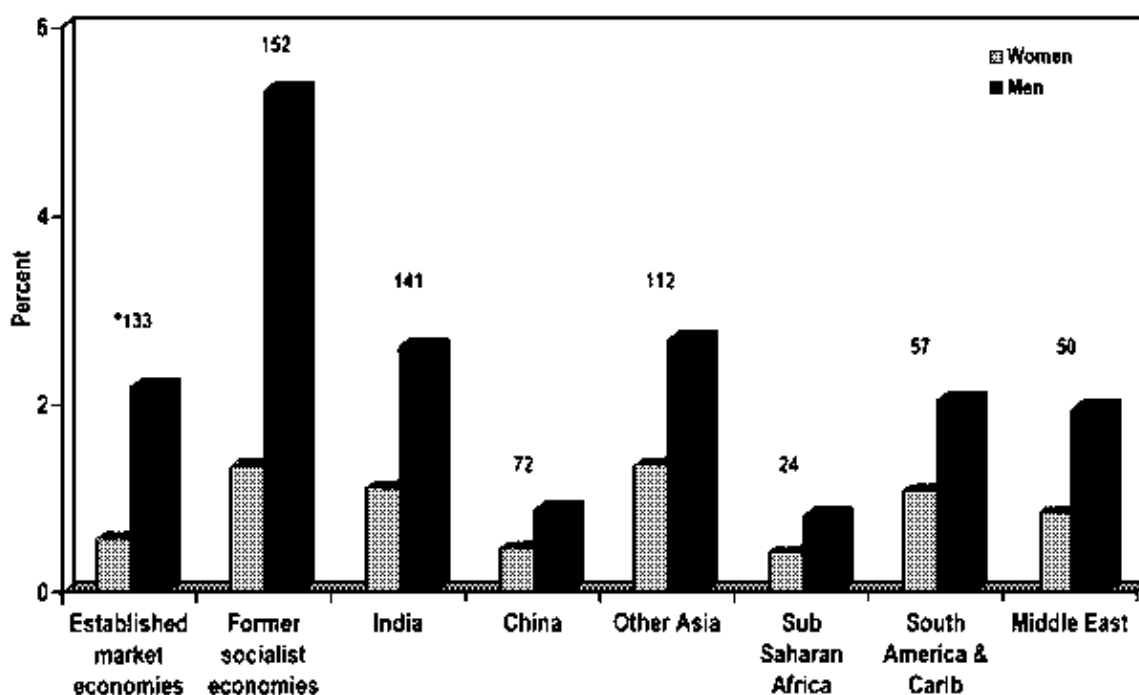


Figure 8. Coronary heart disease probability of death ages 15–60 years

Source: Murray & Lopez (1994) *Bull WHO* 72:447.

* estimated numbers of deaths in thousands (1990 – sexes combined)

III. Interventions to Reduce Low Birthweight: Global Experiences

History

Early textbooks made only tangential reference to LBW as a problem^{58,59} and it is only in the last decade that a body of literature has emerged which better elucidates the problem.

Prepregnancy weight and pregnancy weight gain are independent and completely additive (and subtractive) in their effect on birthweight, together accounting for a difference of up to 1000 g in birthweight.⁶⁰ (Krasovec and Anderson 1991, p 53)

In 1996 the International Dietary Energy Consultative Group (IDECG) held a workshop on the causes and consequences of being born with LBW in socioeconomically deprived populations.⁶¹ Most of the trials under consideration at the IDECG workshop were randomized control trials (RCT) carried out in industrialized countries and/or in non–nutritionally stressed populations. Participants at the symposium in Bangladesh acknowledged the limitations of RCTs and recommended that findings from observational studies not be discounted when determining programme effectiveness.

The first meeting of the ACC/SCN Working Group on the Prevention of Foetal and Infant Malnutrition (formerly the Working Group on the Lifecycle Consequences of Foetal and Infant Malnutrition) held in Geneva in April 1999 suggested that the perceived requirement for numerous RCTs may be a barrier to the development of LBW prevention programmes. The Working Group identified a need to document best practices for LBW treatment and prevention, suggest ways of improving programme design, and develop inter–agency proposals for LBW prevention programmes.⁵⁶

Given the historical aspects of the science of LBW reduction programmes, little has been implemented to date which translates existing information and recommendations into effective programmes to improve maternal nutrition and prevent LBW. The importance for programmes to be able to improve and advance through a “plausibility” approach for measuring programme performance and impact needs to be urgently considered.⁶² While RCTs are essential to advance scientific knowledge, programme interventions need additional mechanisms because reducing LBW will require packages of interventions. These packages cannot be evaluated by RCTs because it would be almost impossible to isolate the effects of each element of the package. It is of crucial importance to implement these intervention packages for LBW prevention, particularly for safe motherhood programmes and for Integrated Management of Childhood Illness (IMCI) programmes.

Some health and development administrators say it is too expensive to implement programmes to reduce LBW – expensive compared to what? This argument does not make ethical or economic sense. The cost of attrition and repetition rates in schools, in childhood illness, and in maternal mortality would far exceed the cost of any interventions we might implement... The cost to an individual family of losing a mother or a child is simply not measurable. (Shrimpton 1999)

Examples of LBW Prevention Programmes

This section describes a mix of government programmes, small scale interventions and operational research trials which claim to have successfully reduced or prevented LBW. *Information for several of these programmes has not been peer–reviewed and was supplied by official progress reports or the programmes’ annual reports to donors.*

Women, Infants and Children (WIC) Supplemental Food Program – USA

WIC is a nutritional supplementation programme which is part of a comprehensive strategy to reduce the incidence of LBW among high risk women in the USA. It is felt that this type of programme must be linked closely to antenatal services. Studies have suggested the WIC programme was responsible for a 20–30% reduction in LBW incidence during 1992 among women who had received WIC services compared with similar women who did not receive prenatal WIC services.^{63–65} An evaluation of the programme indicated that WIC had a significant positive effect on duration of pregnancy, birthweight, head growth and foetal mortality.⁶⁶ Savings from a reduction in estimated Medicaid expenditures in the first year postpartum more than offset the costs of the antenatal programme.^{67,68} In 1988 the programme reached 80% of eligible pregnant women (almost a half million).⁶⁹ The programme provides:

- ? vouchers to purchase nutritious foods (approximately 1000 kcal/day and 50 g of protein in the form of milk, eggs, cheese, juice, cereals and pulses)
- ? growth monitoring and hemoglobin level
- ? nutrition education
- ? referrals to antenatal and other social services.

Higgins Nutrition Intervention Programme: Montreal Diet Dispensary – Quebec, Canada

The outcome of pregnancy was evaluated in 1,544 women of low socioeconomic status between 1963 and 1970. Dietary needs for energy and protein were calculated for each woman based on BMI with adjustments for protein deficiency (as determined by a comparison of usual intake as quantified by a diet history), underweight and other conditions. The study found a direct relation between weight gain and birthweight which in turn were related to the length of time the women had received Diet Dispensary services.^{70,71} The incidence of LBW in this high risk group was brought down to the Canadian national rate and lower than the rate for the province of Quebec. Features of the programme included:

- ? food supplements of milk, eggs and oranges
- ? counselling on food selection for all clients
- ? visits to clients in their homes at least once during their pregnancy
- ? emotional support for the mothers as well as a maternity outfit and an infant layette.

Narangwal Nutrition Study – Punjab, India

The Narangwal Nutrition Study is best described as operational research carried out in ten villages of the Punjab in India during the period 1968 to 1973. Villages were selected as clusters and provided one of the following patterns of services: (1) nutrition care (anthropometric surveillance, food supplements and nutrition education); (2) medical care (immunization, health education, morbidity surveillance, early diagnosis and treatment of infections and other childhood illnesses); (3) a combination of nutrition care and medical care; and (4) control (no services other than minimal symptomatic treatment and emergency care). Perinatal mortality was reduced almost 40% in the nutrition care and combined nutrition/medical care groups, most likely a reflection of improved foetal growth. It was concluded that: "Prenatal nutrition care to pregnant women was the most cost-effective in preventing perinatal deaths."^{72,73} The study provided:

- ? growth monitoring for infants and pregnant women
- ? special emphasis on prolonged breastfeeding
- ? twice-daily food supplementation for all, but especially for those children with undernutrition or inadequate growth, consisting of a calorie-enriched milk in the morning, and a porridge-like gruel of crushed wheat, raw sugar, oil and milk powder in the afternoon (standard servings provided approximately 400 kcal and 11 g of protein, and children were fed *ad libitum*)
- ? prenatal nutrition care in the form of iron folate tablets for all mothers
- ? a food supplement for underweight mothers
- ? immunizations and/or medical care as required.

Child in Need Institute (CINI) – Calcutta, West Bengal, India

The Child In Need Institute began as a small clinic in 1974 in Thakurpukur, on the southern outskirts of Calcutta City. At that time, the clinic served the needs of mothers and their children suffering from moderate to severe undernutrition and diarrhoea. The programme directors were convinced that simple, low cost interventions (immunization, treating minor illnesses, and improving the capacity of mothers to take better care of their children) held promise of providing a better future for these children. Acknowledging that repeated bouts of preventable diseases left these children stunted in mind and body, and in many cases with permanent damage to their physical and mental potential, the intervention has moved from a more simplistic medical model to a primary health care approach. Since LBW is associated primarily with young maternal age, repeated pregnancies in quick succession, poor access to antenatal care, and a heavy workload during the third trimester, CINI has adopted a holistic lifecycle approach to maternal and child health. This approach includes organizing women's groups and providing support to poverty stricken families to improve their income and functional literacy. CINI now promotes sustainable health and nutrition development in 70 villages reaching a total of at least one million people.⁷⁴ The internal reporting systems of the project suggest that in CINI villages, LBW has been reduced by one-third, more women have been identified early in their pregnancy and have been registered at the clinic, and attended deliveries have increased. The proportion of pregnant women eating extra food, and the proportion of pregnant women resting during the last trimester of pregnancy have both reportedly been increased.^{74,75} The CINI programme has three components:

? case management which increases the coverage of antenatal care and helps prevent repeated pregnancies in quick succession

? behaviour change communication strategies which aim to increase awareness and improve interpersonal communication

? 'linkage' which aims to strengthen existing health care facilities and improve linkages within the community.⁷⁵

Bangladesh Integrated Nutrition Programme (BINP)

The Bangladesh Integrated Nutrition Project, with support from the Government of Bangladesh, the World Bank, UNICEF, and several other organizations, was implemented in 1995. The largest component of this project, the Community-Based Nutrition Component (CBNC), has become one of the most promising large scale community-based projects in the world aimed at reducing childhood and maternal undernutrition. This comprehensive project now targets over one million households in 23 project areas called *thanas*. It is too early to measure the project's impact on its objective of reducing the incidence of LBW by half; however, the findings of a study performed under the ICDDR,B BINP-Operations Research Project indicate that improvement in birthweight is achieved, but only when supplementation is initiated early in pregnancy and continued for at least 120–150 days up to delivery.¹⁸⁹ The report found that during the project's two and a half years of field implementation, severe stunting in children 6–23 months of age declined from 36% to 21%; severe wasting in the same age group declined from 12% to 2%, and severe underweight declined from 30% to 18%. During the same period, BINP has made a substantial improvement (2.3 percentage point reduction) in the prevalence of low BMI (<18.5 kg/m²) among women of childbearing age (15–49 years). Although BINP indicated improvements in knowledge of some health and nutrition-related behaviours between the project *thanas* and control *thanas* (Table 1), there has been relatively little effect on the practice of exclusive breastfeeding, or on the nutritional status of pregnant and lactating women. Success in the Information, Education and Communication (IEC) component, which is fundamental to the success of BINP, has remained a weak link in the project as a whole.^{76,77}

Table 1. Differences between women in project and control *thanas* regarding health and nutrition related behaviours

Behaviour	Project (%)	Control (%)
Initiated breastfeeding immediately after birth	60	31
Fed colostrum to newborns	94	76
Ate additional food during pregnancy	56	22
Visited by health worker for antenatal advice	94	75
Received tetanus toxoid immunization (women 14–49 years)	84	69
Use of sanitary latrines	19	09

Source: BINP Mid-term Evaluation (1999) INFS; Dhaka University.

The Government of Bangladesh is now extending the BINP into a National Nutrition Program (NNP), to give effect to the principles of the National Food and Nutrition Policy and the strategies articulated in their National Plan of Action for Nutrition (2000–2010). Nationwide implementation will require long-term commitment, a programmatic approach, and effective management of the large number of partners. Sustainable success at the national level will require substantial capacity building and coordination with other health and community development services.

In addition to delivering BINP's successful community-based nutritional services for children, NNP plans to develop, test and implement effective strategies to improve adolescent girls' and women's nutritional status. National nutrition institutions will be strengthened to facilitate and support programme design and implementation. Additional links to food security programmes, such as the government's Vulnerable Group Development Programme, will be provided to poor beneficiaries. Eventually the NNP will develop a model by which the Government's primary health care system can incorporate focused nutrition activities which will lay the foundation for sustainable, long-term solutions to micronutrient undernutrition problems.¹

The new National Nutrition Program (NNP) will consist of a large array of community based services with the aim of reducing LBW as one of its key mandates. The programme will provide:

- ? energy supplements of 600 kcal/day plus micronutrients
- ? “packages” of nutrition education messages to be delivered to mothers throughout pregnancy
- ? utilization of a “nutritional negotiation” process whereby women participate in decisions about their health
- ? programmes on immunization, birth spacing and postpartum contraception.

Integrating Food and Nutrition into Development – Thailand

Thailand’s achievements in health and social development since its First National Economic development plan (1961) and those of its National Food and Nutrition Plans (NFNP) have received worldwide acclaim. During the 1980s the nation experienced dramatic reductions in their number of underweight children, including the virtual elimination of severe underweight. The impact of programmes carried out under the Second NFNP were impressive. By the end of the programme in 1986, only 3–5% of children under five were moderately or severely underweight and only 18–20% were mildly underweight. In 1991 the incidence of LBW was 9.0% for the entire country.⁷⁸ The northern area of Thailand has the highest rate of LBW, possibly because it is an iodine deficient area and smoking among women is common. The Third NFNP (1987–91) utilized a more holistic “basic minimum needs” approach which included a national campaign to enhance the quality of life. Nutrition plans included:

- ? a nutrition education campaign aimed at producing behavioural changes at the household level
- ? development of more sensitive indicators for nutrition surveillance and growth monitoring
- ? integrating nutrition into rural community development plans and involving social scientists
- ? specific programs to control micronutrient deficiencies.

Nutrition and Health Transition – Costa Rica

Within the decade of the 1970s, Costa Rica demonstrated a remarkable improvement in its health and nutrition statistics, realized in part by the commitment of its government to devote its resources to health and education by abolishing its armed forces. Infant mortality rates dropped from 68 per 1000 in 1970 to 19 per 1000 in 1979; and by 1990 the rate was 13 per 1000.⁷⁹ The incidence of LBW *at term* in Costa Rica is considered to be about 3%.⁶ By 1990, 550 Integrated Centers for Child Health Care (CINAI) had active nutrition programmes supported by CARE, UNICEF, and the Government of Costa Rica’s Ministries of Health, Education and Agriculture.⁸⁰ The programme provided:

- ? warm meals targeted at low–income pregnant and breastfeeding women and preschool children who are able to come to CINAI
- ? whole powdered milk distribution to the children and pregnant and nursing mothers who are unable to come to CINAI
- ? food rations and nutrition education targeted to families showing nutritional vulnerability
- ? a holistic approach: providing vaccinations, family planning, environmental sanitation, pregnancy monitoring, growth and development monitoring in children as well as health education
- ? the expansion of school cafeterias
- ? the fortification of salt with iodine and fluoride; and sugar with vitamin A.

Dietary Supplements During Pregnancy

This section describes dietary supplementation trials which have successfully reduced or prevented LBW. Supplementation is associated with increases in maternal weight gain and mean birthweight, and a decrease in the number of IUGR infants.⁸¹ In the past, the overall methodological quality of many supplementation trials has been variable, and the degree of compliance and substitution of the normal diet may also have been quite variable across several of the trials.⁸¹ The potential public health benefit of reducing LBW and neonatal mortality through high energy dietary supplementation during pregnancy however, is great; and definitive RCTs must now be replicated elsewhere.

Institute of Nutrition of Central America and Panama (INCAP) Supplement Studies –Guatemala

A food supplementation programme, as well as preventive and curative outpatient care services, was provided to the inhabitants of four villages in Guatemala from 1969 to 1977.^{82–84} This was one of the most comprehensive studies on the relationship of maternal nutrition to the outcome of pregnancy ever undertaken. Individuals in two of the villages received an energy and protein supplement called *Atole* while inhabitants of the other two villages received an energy-only beverage called *Fresco*. Both supplements had similar amounts of vitamins and minerals. Since there was a range of supplement intakes throughout the course of pregnancy, investigators divided the women into a low supplement group (those who consumed less than 20,000 additional kcal) and a high supplement group (those who consumed more than 20,000 additional kcal). Data were reported on 405 infants born in four villages and demonstrated that:

? Birthweight increased by 50 g for each 10,000 kcal ingested by the mother during pregnancy.

? The rate of LBW was reduced by approximately 50% when mothers consumed >20,000 supplemented kcals throughout pregnancy versus those mothers who consumed <20,000 supplemented kcals throughout pregnancy.

? Placental weight was approximately 11% less for the group with the low supplementation.

? There were no significant differences based on the type of supplement, i.e., the inclusion of protein did not seem to confer any additional benefits. This led investigators to conclude that in a population experiencing chronic but moderate undernutrition, the limiting factor in the diet is energy, not protein.

? Subsequent supplementation of 169 mothers enrolled during two consecutive pregnancies and the intervening lactation period resulted in an average increased birthweight of 301 g in the high supplemented group (approximately an extra 180 kcal/day during pregnancy and 245 kcal/day during lactation).

Milk-based Food Supplements – Chile

A study was conducted among low-income pregnant women who attended antenatal clinics in the southeast area of Santiago, Chile during 1983–84. The women in the study were over 18 years of age, nonsmokers, non-consumers of alcohol, and were underweight. (Initial weight-for-height at first prenatal visit was below that recommended for women who weigh <95% of the reference at 12 weeks gestation). These women were given either a supplement of powdered milk or a milk-based fortified product.⁸⁵ These take-home products were distributed during regular monthly clinic visits. In addition to the monthly clinic appointments, all women were visited three times at their homes by trained nutritionists to assess food intake and supplement consumption. Women in the powdered milk group consumed an additional 174 kcal and 9.7 g of protein daily; women in the fortified group consumed an additional 173 kcal, 5.3 g of protein and 1.7 mg of bioavailable iron. The results of the study indicated that:

? Mean birthweights were significantly higher in the fortified group than the powdered milk group (3178 g vs 3105 g).

? The percentage of IUGR infants was significantly reduced in the fortified group as was the percentage of infants with birthweights <3001 g.

? Women in the fortified group had significantly greater weight gains during pregnancy.

? Although both groups had similar energy and protein levels, the additional micronutrient levels in the fortified group had a beneficial effect for both mother and infant.

Indonesia Supplementation Trial – East Java

In a rural community of East Java the effect of energy supplementation on birthweight was assessed. It was found that young women with a short birth interval and a prepregnancy weight of less than 41 kg or a weight of less than 45 kg in the second half of pregnancy were likely to deliver a LBW infant.²⁶ Food supplements were given to women during their last trimester of pregnancy and the impact of these high (465 kcal) and low (52 kcal) energy supplements was examined in relation to the birthweight of the infants. Supplementation at either level was associated with an increase in birthweight of 100 g and a reduction in LBW from 12.2% to 9.5%.⁸⁶ Results suggested:

- ? an increase in average birthweight of about 50 g per 10,000 additional kcal
- ? the minimum prepregnancy weight associated with a decreased the risk of LBW was 41 kg
- ? targeting supplementation to the lean seasons or to women with low prepregnancy weight may be the most cost-effective strategy for preventing LBW.

The Gambia

Evidence now exists to suggest that infant outcomes can be improved by improving maternal nutritional status. The most recent five year RCT trial from the Gambia has reported that a high energy, antenatal dietary supplement can increase maternal weight gain, reduce LBW by 35%, and significantly reduce stillbirth and neonatal deaths by 55% and 40% respectively (Figures 9 and 10). Overall there was a 49% reduction in perinatal deaths and 40% reduction in early neonatal deaths but no effect on post-neonatal mortality.^{87,88} This study confirms the positive results of the first Gambian study, which was an unmatched case-control study.^{89,90} The results of both trials show that supplementation increased weight gain during pregnancy and significantly increased birthweight during the nutritionally debilitating hungry season – from June to October – by 201 g (Figure 11). Head circumference was only increased by about 3 mm with no evidence of increased complications during birth. These findings are reinforced by other retrospective trials and observational studies from the past 13 years in the Gambia.⁸⁸

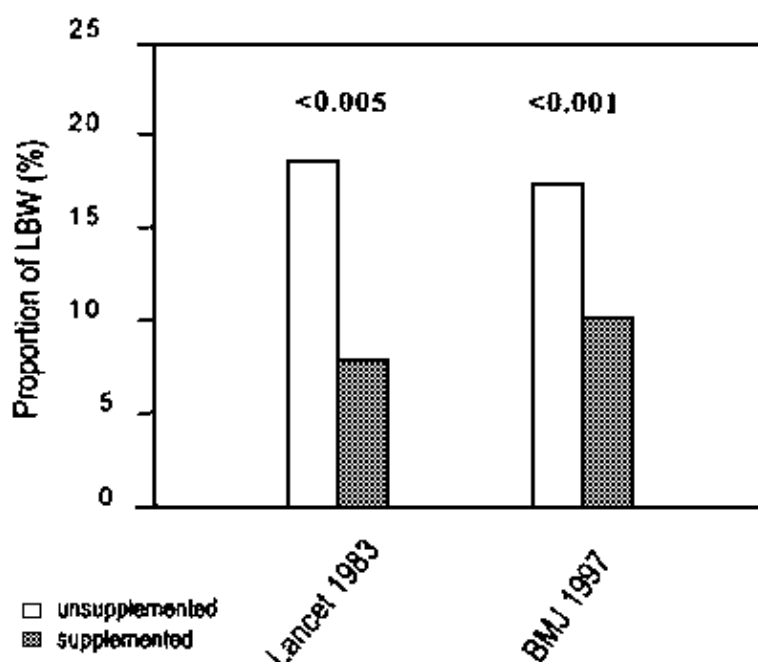


Figure 9. The effect of a high energy food supplement on proportion of LBW – the Gambia

Source: Cessay et al. (1997) *BMJ* 315:786; Prentice (1983) *Lancet* 1:489.

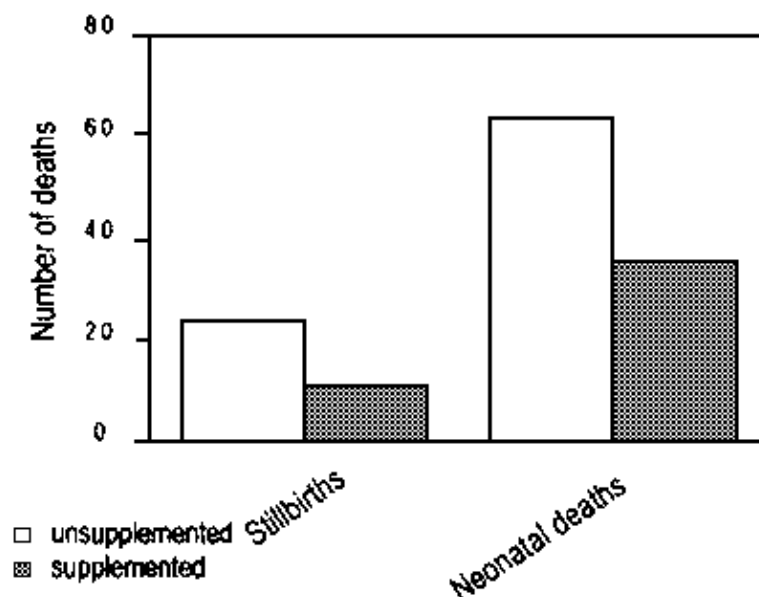


Figure 10. The effect of a high energy food supplement on stillbirths and perinatal mortality – West Kiang Trial only

Source: Ceesay et al. (1997) *BMJ* 315:786.

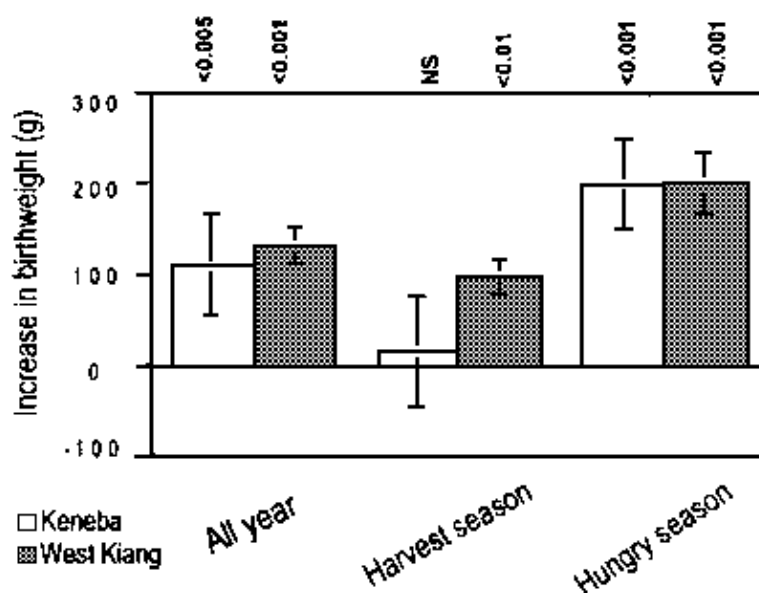


Figure 11. The effect of a high energy food supplement on birthweight in the Gambia – Keneba and West Kiang

Source: Ceesay et al. (1997) *BMJ* 315:786.

The food supplement consumed by the Gambian mothers for approximately 20 weeks during gestation was two high energy biscuits made from roasted groundnuts, rice flour, sugar, dried skim milk and groundnut oil. The supplement provided about 900 kcals, 22 g protein, 56 g fat, 47 mg calcium, and 1.8 mg iron per day. The biscuits were prepared with local ingredients by the community and were regularly distributed by the birth attendants who delivered health education messages while mothers consumed the supplement. This emphasizes the feasibility of community participation when incorporating a supplementary feeding programme into the healthcare system. All of the women in this study also received antenatal care and counselling, an iron and folate supplement, a tetanus toxoid injection if necessary, and malarial prophylaxis during the wet/rainy/hungry season.

Follow-up in the Gambia is now taking place to determine the health and nutritional status of ten year olds from the study-site villages in order to better understand the complexities of the timing of supplements as well as measuring deficits that may have occurred *in utero*. It has already been demonstrated that infants born during January through June of the Gambian harvest season have much higher survival rates in young adulthood compared to those born during July through November of the hungry or wet season – members of

the latter group frequently die of infectious diseases, suggesting that LBW may impact upon adult immunity.⁹¹ This somewhat links with the 'foetal origins of disease' hypothesis, i.e., that foetal programming may lead to sustained impairment of the immune system of LBW infants as they become children and adults.^{21,22} Early organ growth and development deficits *in utero* may be "more serious and long-lasting than those caused by later undernutrition".⁹²

The ability of dietary supplementation to reverse foetal growth retardation in the hungry season when provided for an average of only 82 days in the second half of pregnancy is consistent with findings from the Dutch "hunger winter" of 1944–5,⁹³ and illustrates that foetal growth is most sensitive to nutritional deprivation in the last trimester of pregnancy. Models used to analyze the effect of the Dutch hunger winter suggest that under famine conditions there may be a shift in the role of the placenta in the passage of energy and nutrients from mother to foetus, to the advantage of the mother.⁹⁴ Studies from East Java, Pakistan, Guatemala, Egypt, Mexico and Kenya have reported that women with BMI <18 kg/m² gained more weight and fat during pregnancy and lost more weight and fat during lactation; while women who were not chronically energy deficient and had BMI >22.5 kg/m² lost weight during pregnancy.^{95–97} Apparently, chronically energy deficient (CED) mothers have been found to replenish their energy stores at the expense of foetal growth but this process is poorly understood.

Data from the world literature have been analyzed in order to test whether low BMI is a useful indicator of functional impairment of lactation performance.⁹⁸ No detectable relationship between maternal BMI and the volume of milk produced by mothers has been found, even when BMI is <18.5 kg/m². Remarkably, this work showed that very high milk volumes were produced by very thin mothers.^{98,99} It is accepted that the composition of breastmilk is relatively unaffected by general undernutrition of the type that would be indicated by a low BMI with the possible exception of milk fat levels and hence the energy content.⁹⁸ Lactation performance must become compromised when undernutrition is sufficiently severe and probably occurs only in famine or near famine conditions. Human lactation performance is extremely robust; BMI does not provide a useful indicator of function at the levels studied to date.⁹⁸

Another factor which requires further investigation is the effect of supplementation trials on the interrelation between maternal and infant outcomes over one or more reproductive cycles. A study of the Guatemalan experimental trial of 1969–1977 found that generally, the birthweight of the second infant was an average of 118 g less than that of the first newborn in the undernourished group of mothers, and 114 g and 140 g higher in the marginally nourished and well nourished groups respectively. It was concluded that energy supplements improved maternal weight in the undernourished group over one reproductive cycle, but could not prevent impaired foetal growth unless an extra 117 kcals per day were ingested by the mother during pregnancy. The study emphasized that long-term supplementation with adequate amounts of energy is required in order to result in positive pregnancy outcomes for mothers and infants.^{97,100}

The 1999 meta-analysis by the Cochrane Review included a special note about the high energy supplement provided in the Gambian trial, which gave considerably larger effects regarding improved foetal growth and important statistically significant reductions in stillbirths and neonatal deaths than the other fourteen RCTs of balanced protein/energy supplements which were analyzed. The main conclusions of the meta-analysis were that balanced protein/energy supplementation (where protein accounts for <25% of the total energy content) results in modest increases in maternal weight gain and foetal growth. The report also noted that neither balanced, isoenergetic protein supplementation nor high-protein supplementation appeared beneficial to either mother or infant and could even impair foetal growth.⁸¹ Given the success of the Gambian trial's high energy supplement in reducing LBW and neonatal mortality, other trials of this sort are urgently needed.

The Role of Micronutrients in Improving Birthweight

Micronutrients can affect birthweight directly and/or indirectly by their interactions with each other, i.e., micronutrient supplementation may cause an increase in maternal appetite, which may lead to increased food intake and/or reduced morbidity. Deficiency in one or more micronutrients is due to inadequate food intake, poor dietary quality, poor bioavailability (where micronutrients are either not readily released from foods or are not absorbed efficiently), or a combination of these factors. Thus, quite often in developing countries where LBW is prevalent, multiple micronutrient deficiencies co-exist and are likely to be of great public health concern. Over 100 studies have examined the role of micronutrients during pregnancy. Many of the earlier supplementation trials were conducted in industrialized countries among women who were not deficient and therefore less likely to benefit from the interventions. Some nutrients, namely vitamin A, calcium, iron and zinc, have been studied extensively, but much less is known about others, such as vitamin B complex. There

is considerable variation in the methodologies used in the studies, ranging from cross-sectional and prospective studies to randomized, double-blind, placebo-controlled trials.

Vitamin A

Whether vitamin A supplementation alone can increase birthweight has not been clearly demonstrated (Figure 12). In two studies conducted in South India^{101,102} where the prevalence of LBW is high, birthweights in the supplemented groups were about 130 g higher than birthweights in the non-supplemented groups. These differences were not statistically significant, primarily due to the limited sample size (<50 subjects per group). Another trial from England was conducted among South Asian immigrants who had a higher risk of LBW than their British counterparts, but no differences in birthweight were detected.¹⁰³ A randomized, double-blind trial that was recently conducted among HIV-positive women in Tanzania also failed to detect any significant differences in mean birthweight. The prevalence of LBW was, however, slightly lower in the vitamin A group at 14.5% compared to 17% in the placebo group who received an iron-folate supplement.¹⁰⁴ A trial in Nepal showed that vitamin A supplementation can reduce maternal mortality up to 40%, however, results regarding birthweight have not yet been published.¹⁰⁵

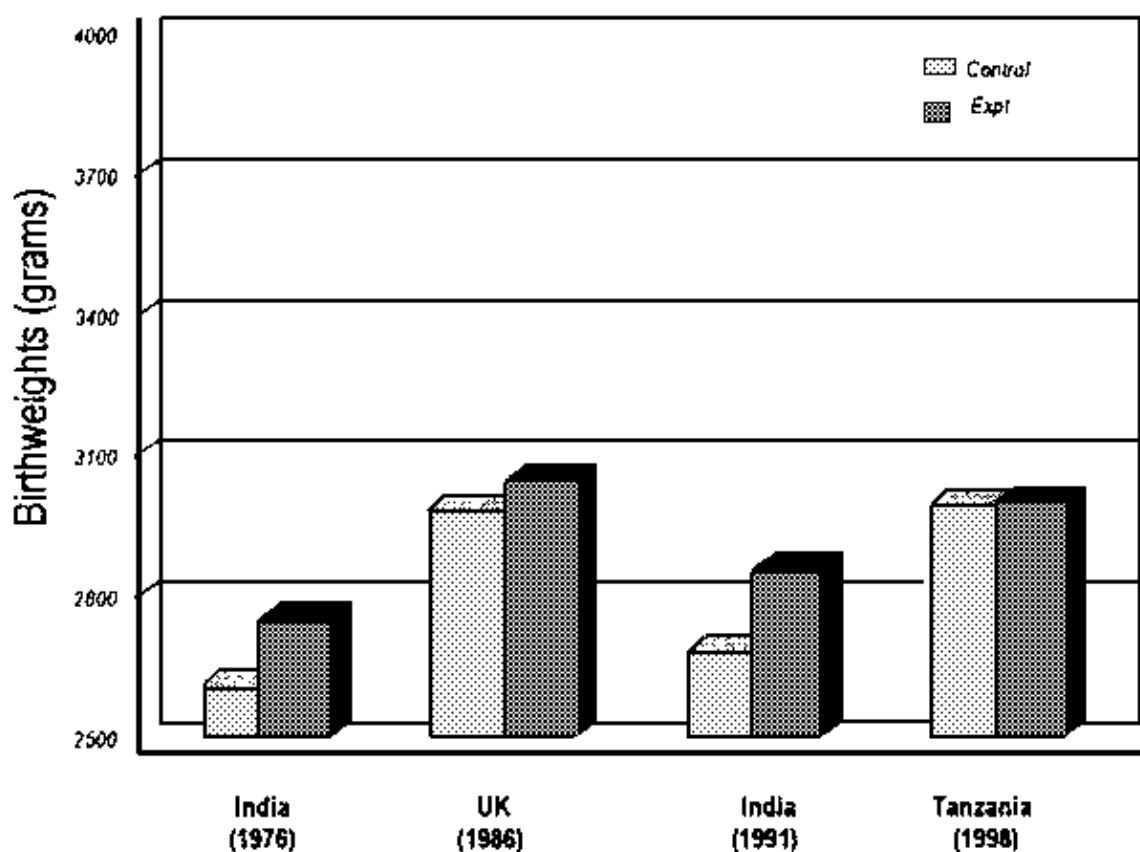


Figure 12. The effect of vitamin A on birthweight: Experimental trials

Source: Rao et al. (1976) *Ind J Med Res* 64:1261; Howell et al. (1986) *Hum Nutr Clin Nutr* 40C:43; Panth et al. (1991) *Inter J Vit Nutr* 61:17; Fawzi et al. (1998) *Lancet* 351:1477.

Folic Acid

Several prospective studies, mainly from industrialized countries, have reported a positive association between folate and birthweight. For example, it was found that both low dietary intakes of folate ($\approx 240 \mu\text{g}/\text{day}$) and lower concentrations of serum folate measured at 28 weeks of pregnancy were associated with a two-fold increased risk of preterm delivery and LBW even after controlling for several maternal characteristics.¹⁰⁶ Earlier experimental studies, however, were less equivocal about the effects of folate on birthweight (Figure 13). One study conducted in India reported a 50% decrease in SGA infants and increased birthweight using a design which compared iron to iron-folate supplementation.¹⁰⁷ Another trial in France used a similar supplementation regimen, and gestation was increased by an average of one week in the folate-supplemented women, who gave birth to longer and heavier infants.¹⁰⁸ In another randomized, controlled trial conducted in Denmark, the infants in the folic acid group were 13% heavier than those in the control group.¹⁰⁹ In contrast, three studies that were conducted in Ireland and Hungary showed no benefits of

folic acid supplements on foetal growth.^{110–112} Although the benefits of folic acid in reducing the risk for neural tube defects is well established, the results for birthweight are less clear and there is a need for well–designed experimental trials in settings where folic acid deficiency and LBW are common.

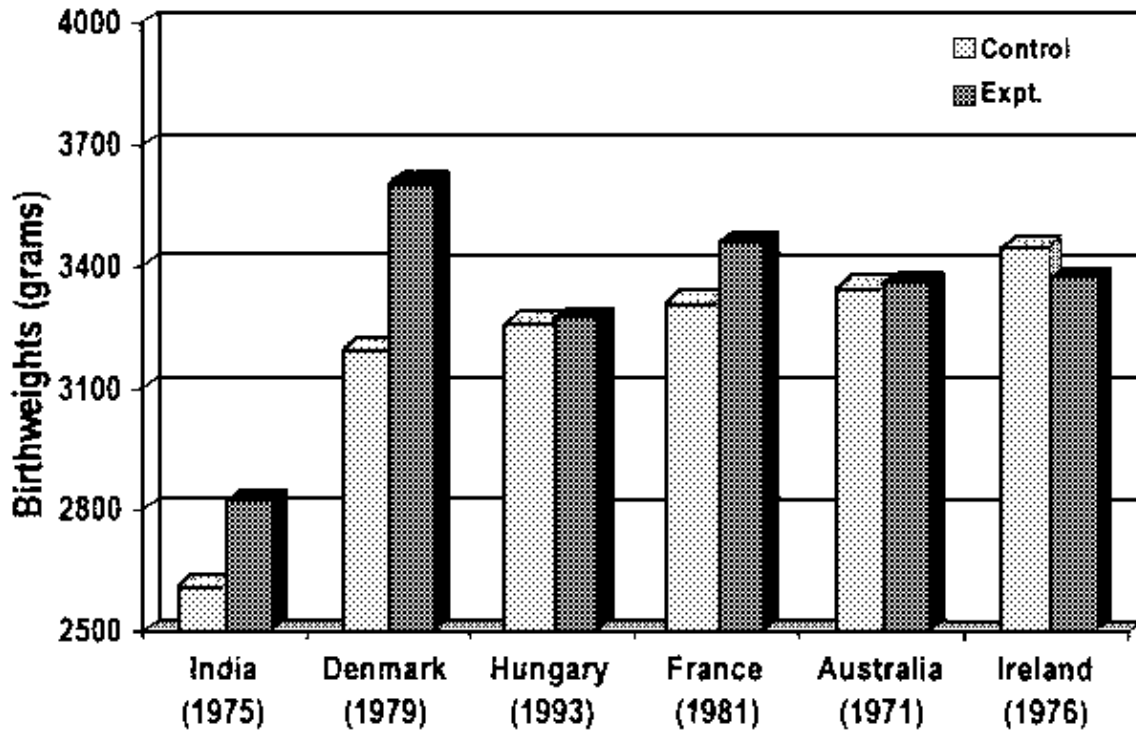


Figure 13. The effect of folic acid on birthweight: Experimental trials

Source: Iyengar et al. (1975) *Am J Ob Gyn* 122(3):332; Rolschau et al. (1979) *Acta Ob Gyn Scand* 58(4):343; Czeizel (1993) *Ann NY Acad Sc* 678:266; Blot et al. (1981) *Gyn Ob Inves* 12(6):294; Giles et al. (1971) *Med J of Aus* 2(1):17; Kirke et al. (1992) *Arch Dis Child* 67(12):1442.

Iron

Maternal anaemia diagnosed *prior to mid–pregnancy* has been associated with an increased risk of preterm delivery. Maternal anaemia detected *during the later stages of pregnancy* often reflects the expected (and necessary) expansion of maternal plasma volume and is usually not associated with increased risk of preterm delivery. *High hemoglobin concentration, elevated hematocrit and increased levels of serum ferritin late in pregnancy have been associated with increased risk of preterm delivery.* This risk may reflect in part the failure to expand maternal plasma volume adequately, thus diminishing appropriate placental perfusion. Although controlled trials of iron supplementation during pregnancy have consistently demonstrated positive effects on maternal iron status at delivery, they have not demonstrated reductions in risk of preterm delivery or LBW.¹¹³

Iron deficiency is the most widespread micronutrient deficiency in the world, and its associated anaemia has been linked with a three–fold increased risk of LBW and a two–fold increased risk of preterm birth in a prospective study of African–American women.¹¹⁴ To date however, there are very few well–designed experimental trials examining the effect of iron on birthweight (Figure 14). Conducting such experimental trials are unethical, however, as they would deny standard treatment to the control group. Many countries provide routine iron or iron/folate supplementation as part of their antenatal care programmes. Two trials with iron supplementation from Finland and Denmark failed to detect any significant differences in mean birthweight and the prevalence of LBW, but these studies had methodology problems which included lack of blinding, suitability of the control group, and small sample size.^{115,116}

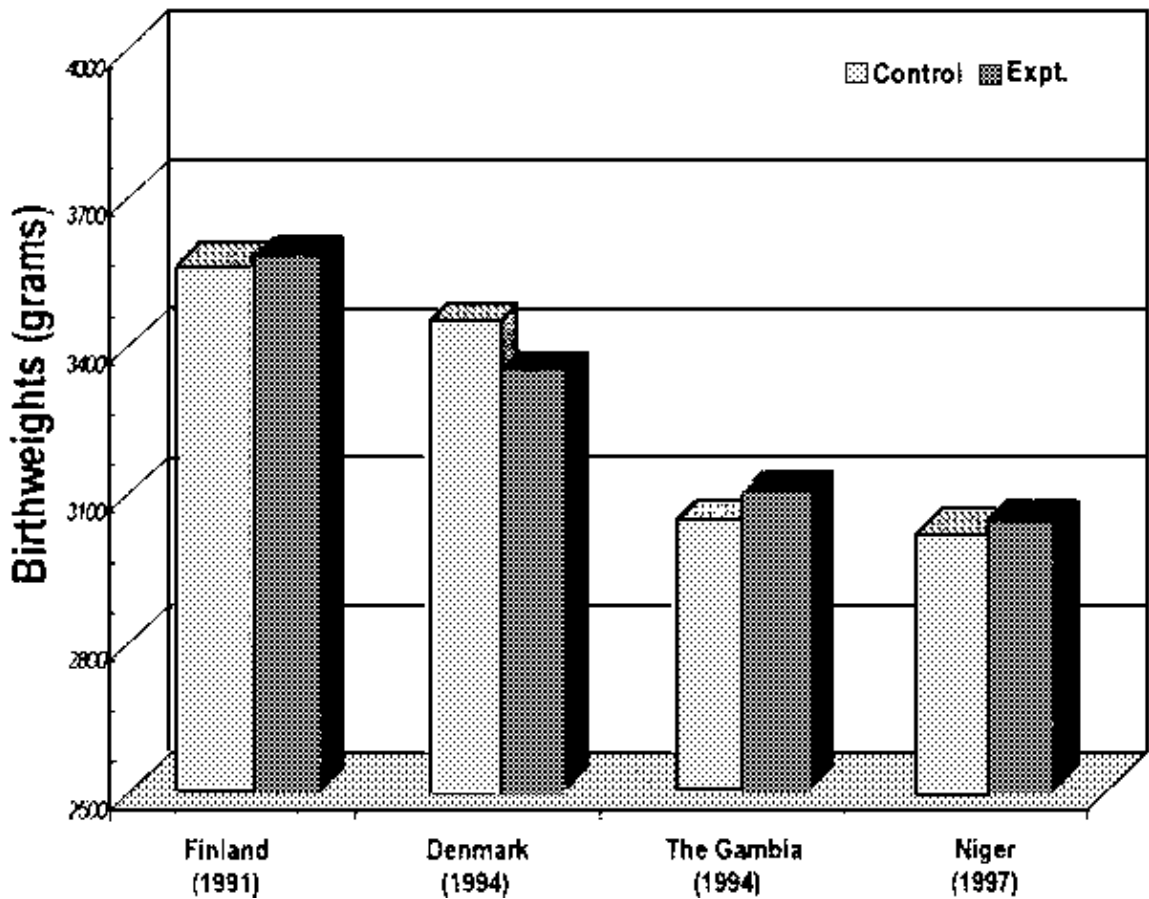


Figure 14. The effect of iron on birthweight: Experimental trials

Source: Hemminki et al. (1991) *J Am Col Nutr* 10(1):3; Milman et al. (1994) *Acta Ob Gyn Scand* 73(3):200; Menendez et al. (1994) *Trans R Soc Trop Med Hyg* 88(5):590; Preziosi et al. (1997) *Am J Cl Nutr* 66(5):1178.

Two trials of iron supplementation conducted in developing countries did result in higher birthweights in the iron supplemented groups. In a trial of pregnant women in the Gambia, the mean birthweight of infants born to women who received daily supplements containing 200 mg of ferrous sulfate and 500 µg of folic acid during pregnancy was 56 g greater than that of infants born to the controls who received only folic acid. Although this difference in birthweight was not statistically significant, there were statistically significant reductions in the prevalence of anaemia and iron deficiency among mothers. Further, there was evidence of a positive dose-response relationship between birthweight and the degree of iron deficiency. For example, among women who took at least 80 tablets (a three month supply), a significant difference of nearly 100 g was observed between the two groups.¹¹⁷ A study, which was possible due to the lack of routine administration of iron or folic acid supplements to pregnant women in a clinic in Niamey, Niger, randomly allocated 197 women to receive either 100 mg iron per day or a placebo during the last trimester of pregnancy. As expected, the iron supplement contributed significantly to reductions in the prevalence of anaemia and higher serum ferritin values in both mothers and their infants. Another important result was that mean length and heart rate, respiratory effort, muscle tone, reflex irritability and colour (Apgar score) were significantly higher in infants who were born to mothers in the iron group compared to those in the placebo group. Although mean birthweight was 30 g higher in the iron supplemented group these differences were not statistically significant.¹¹⁸

No adverse effects of iron supplementation on pregnancy outcome have been demonstrated to date. Pregnant women living in malarial endemic areas of developing countries and having the haemoglobin genotype AS, however, may not benefit from iron supplementation as this has been associated with reduced birthweight.¹¹⁹ Questions about the efficacy of iron supplementation during pregnancy for reducing adverse outcomes such as preterm delivery and side effects from iron supplementation, including the potential for oxidation of lipids and DNA¹²⁰ require further research.¹¹³ It is ethically unjustifiable to conduct more randomized, double-blind, placebo controlled experimental trials in light of the WHO/UNICEF international recommendations for routine iron/folate supplementation during pregnancy. Well designed prospective studies in developing country settings, however, with data on pregnancy outcomes as well as biochemical indicators, supplement use, and dietary intakes during pregnancy will be useful in order to examine the benefits of

improved iron nutriture on pregnancy outcomes.

Zinc

For children in developing countries, zinc deficiency may be common and associated with immune impairment and increased risk of serious infectious diseases such as diarrhoea, pneumonia, and malaria. RCTs have shown a therapeutic effect of zinc supplementation on diarrhoea duration and severity, and have a preventive effect on the incidence of diarrhoea, ALRI and malaria.^{121–123} In a recently completed trial in Bangladesh, no effect was observed on infant birthweight, LBW, or gestational age when over 600 pregnant women were supplemented with zinc or a placebo daily during the second and third trimester. Although LBW rates were unaffected, infants of the women who received antenatal zinc had a reduced incidence of dysentery and reduced severity of acute watery diarrhoeal illness during the first six months of life compared to infants of mothers receiving the placebo.¹²⁴ Similar results were observed in a similar study of antenatal zinc supplementation in Peru.¹³¹

Yet to be published results from Bangladesh of zinc supplementation RCTs in two LBW cohorts (antenatal supplementation only and postnatal infant supplementation from 4–24 weeks of age) have recently become available. Psychomotor development assessed at 12 months of age showed no benefit with zinc as compared to the placebo in either of the trials. Unexpectedly, performance scores on certain measurements were actually lower in the zinc groups of children of both the antenatal and postnatal studies, although the significance of these is not known.⁵⁷ It is possible that in this population of poor and undernourished infants, zinc as a single micronutrient supplementation has limitations and/or any potential benefits of zinc or other similar interventions are overwhelmed by significant levels of undernutrition.

The physiological role of zinc during periods of rapid growth and development emphasizes the importance of zinc during foetal life and gestation.¹⁹⁰ Results of cross-sectional studies have associated low dietary zinc intake or low maternal plasma zinc with increased risks of LBW and preterm delivery.^{191–193} Low plasma zinc has also been reported to correlate with pregnancy complications such as prolonged labour, hypertension, postpartum hemorrhage, spontaneous abortion, and congenital malformation.¹⁹⁴ Despite these associations, the evidence from zinc supplementation trials in pregnant women to improve pregnancy outcome has been less convincing.

Results of experimental trials of the effects of zinc on birthweight are summarized in Figure 15. The 1993 study in India demonstrated significant increases in birthweight and gestational age, a reduced incidence of prematurity and of IUGR, and a higher Apgar score in the group supplemented with 45 mg of elemental zinc as compared to the untreated group.¹²⁶ However, caution is warranted in interpreting these results because of methodological limitations. Zinc supplementation has also been associated with reductions in the incidence of IUGR and in delivery complications for pregnant women who were at high risk of delivering SGA infants. The inclusion criteria for this 1991 study were low prepregnant weight, birth of a previous SGA infant and smoking. Results showed a three to four-fold reduction in the prevalence of IUGR (27% controls; 7% treatment). Although the mean birthweight was 170 g greater in the zinc supplemented group, these differences were not statistically significant, probably due to the small sample size.¹²⁷ Evidence of an interaction between maternal weight and response to zinc supplements was demonstrated in a trial among low income pregnant adolescents in the USA. The prematurity rates were reduced in the group of normal-weight women who received zinc supplements, and underweight mothers given zinc supplements had longer gestational lengths than the control group.¹²⁸

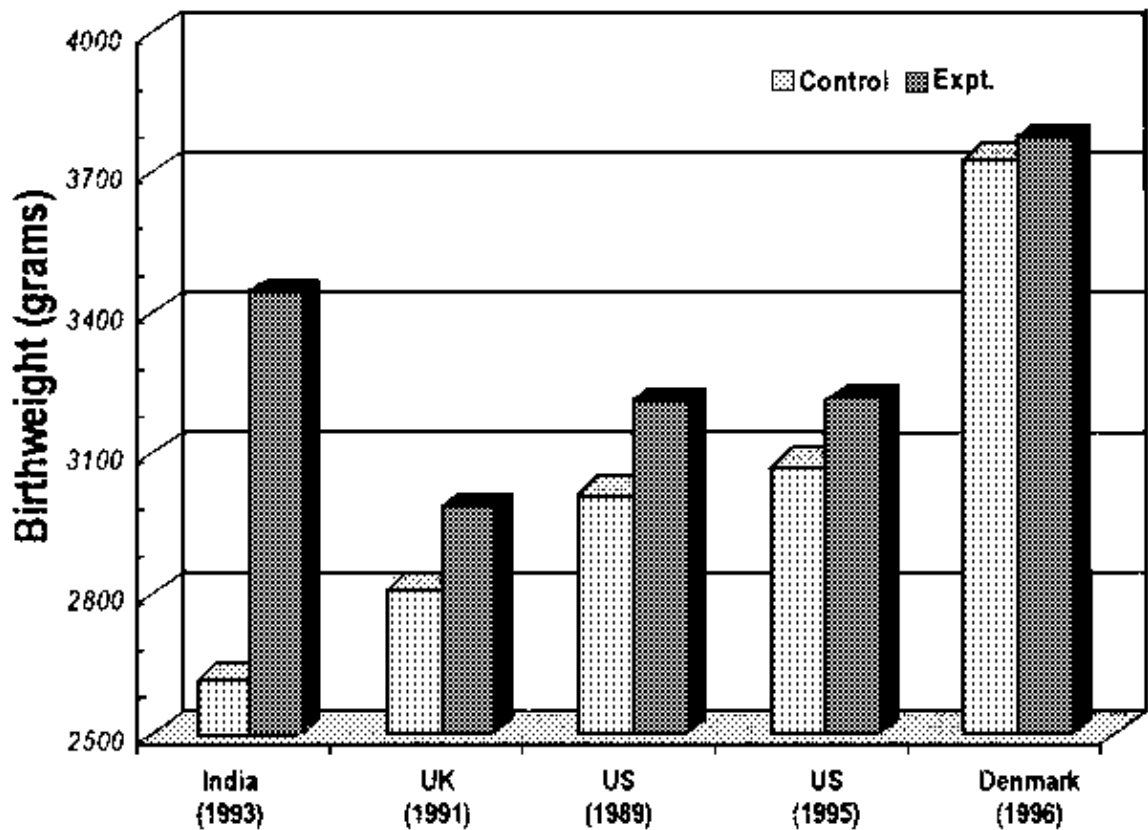


Figure 15. The effect of zinc on birthweight: Experimental trials

Source: Garg et al. (1993) *Ind J Phy Pharm* 37(4):276; Simmer et al. (1991) *Eur J Cl Nutr* 45:139; Cherry et al. (1989) *Am J Cl Nutr* 50:945; Goldenberg et al. (1995) *JAMA* 274(6):463; Jonsson et al. (1996) *Acta Ob Gyn Scand* 75(8):725.

In 1995 a trial was carried out in a poor, urban area of the USA and has provided evidence on the benefits of providing zinc supplements during pregnancy. Pregnant women with low serum levels of zinc were randomly assigned one of two supplements options: a multivitamin compound with 25 mg of zinc or a multivitamin compound without zinc. The infants born to the zinc-supplemented women were an average of 126 g heavier and had larger head circumference values (0.4 cm). The rate of prematurity was also lower. Women with BMI less than 26.0 kg/m² (BMI \geq 25.0 kg/m² is considered overweight) showed the greatest benefits.¹²⁹ Another trial in Denmark randomly assigned 1206 women who were less than 20 weeks pregnant to receive either 44 mg of zinc or a placebo. The outcome measures studied were large for gestation age, SGA, premature rupture of foetal membranes, preterm labour, pre-eclampsia and bleeding in second or third trimester. No differences concerning any of the outcomes were observed in the two groups. These women were from a healthy, middle class population however, and may have had adequate zinc through their diets. These findings suggest that the benefits of zinc supplementation may be limited to certain subgroups with marginal micronutrient status.¹³⁰

Unfortunately, very few trials have been performed in developing countries among populations at risk of zinc deficiency. Investigators of two recent trials from Peru and Bangladesh did not observe any effect of maternal zinc supplementation in infant birthweight. In Peru, 1295 women between 10–24 weeks gestation received supplements containing 60 mg iron and 250 μ g folate with or without 15 mg zinc. The infants were followed to one year of age.¹⁹⁵ In Bangladesh, 559 women were supplemented daily with 30 mg elemental zinc or placebo from 12–16 weeks gestation until delivery. The infants were followed to six months of age to assess growth, morbidity and immune response.¹²⁵ The findings of these two trials were remarkably comparable and showed that maternal zinc supplementation during pregnancy did not result in improved birthweights or gestational age even though it increased maternal serum zinc and infants' cord blood zinc.^{125,196} Results from both studies showed reduced morbidity from dysentery and watery diarrhoeal diseases, skin infections and febrile episodes in infants during the first six months.^{197,198} Thus, studies to date do provide evidence that good antenatal nutrition and micronutrient supplementation have the potential to improve outcomes of LBW infants if not LBW itself.

A beneficial effect of zinc administered to SGA infants was also observed in India. Preliminary results of a recent RCT indicate that mortality was significantly reduced by 67% in SGA infants 1–8 months old who were

supplemented daily for the first year of life with zinc only (Figure 16).¹³² Because of the public health impact of these findings, large scale trials of this type need to be replicated and results, if shown to be universal, would have obvious major implications for the management of LBW or SGA infants. Prevention of LBW is important, but after-care interventions must be simultaneously implemented.

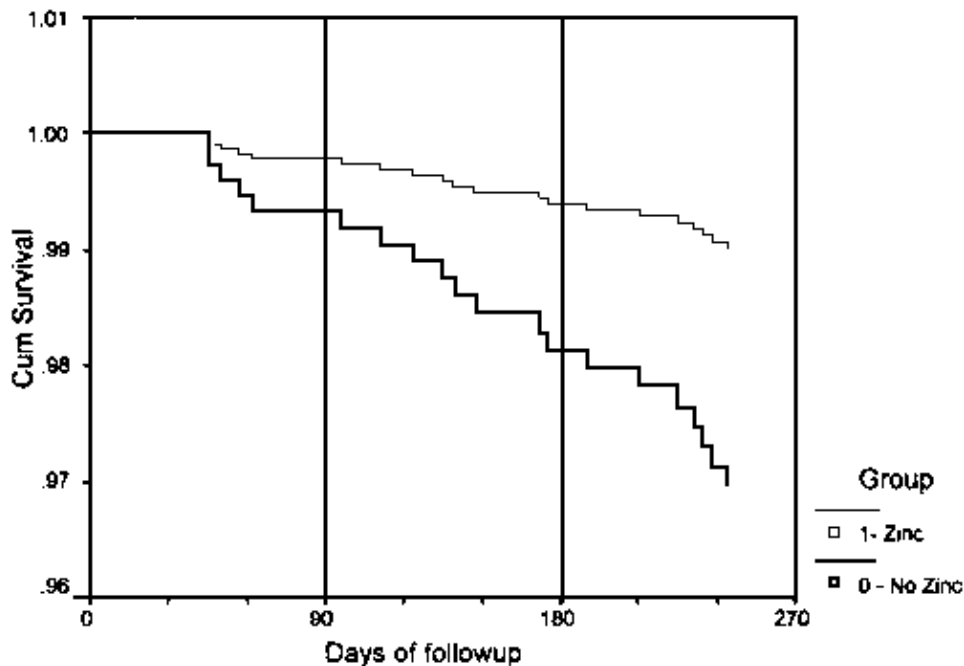


Figure 16. Impact of zinc supplementation on mortality in SGA children

Source: Black (1999) Personal communication.

Iodine

The importance of iodine in preventing mental impairment and cretinism is well established, but the evidence linking it to other outcomes such as LBW and prematurity is weaker, especially in the case of mild iodine deficiency. In a recent review of the published evidence, it was concluded that “the administration of iodized oil before or during pregnancy prevents endemic cretinism and brain damage by correcting iodine deficiency and thyroid function in pregnant women, fetuses, neonates, infants and children”.¹³³ A study in Peru provided evidence that iodine deficient mothers had a consistent tendency to deliver infants with lower birthweights, lengths and cephalic circumferences when compared to those mothers who were treated antenatally with iodized oil.¹³⁴ The most detailed studies of iodized oil given during pregnancy have been conducted in the Democratic Republic of the Congo (DRC, formerly Zaire), Algeria and Malawi. These are areas of severe iodine deficiency where endemic goiter is complicated by endemic cretinism.^{135–137} In the double-blind, randomized, controlled trial in DRC, almost 1000 pregnant women were recruited at their first antenatal clinic visit and were assigned to receive either iodized oil or a placebo. The intervention had a positive impact on important neonatal outcomes such as birthweight, infant mortality and psychomotor development. Although the increase in mean birthweight was not statistically significant for the overall group, birthweight was significantly greater for the children born to the subset of mothers who were iodine deficient at the beginning of the study. Significant improvements in infant mortality rates (167 versus 250/1000) were seen for the overall group, with greater benefits for those who were severely iodine deficient.¹³⁷ A more recent study from Algeria compared the benefits of oral administration of 0.5 ml of Lipiodol at various stages, i.e., one-to-three months prior to conception, during the first month of pregnancy, and during the third month of pregnancy within the context of an iodized oil prevention programme. Untreated mothers served as controls. This study demonstrated significant reductions in the rates of prematurity, stillbirths and spontaneous abortions in the treated groups. The mean birthweight was similar in all the treated groups (3400 g), but was significantly greater than the untreated controls (3200 g).¹³⁵ Finally, the placebo RCT in Malawi in which 0.5 ml of iodized oil was given during the last trimester of pregnancy also demonstrated similar benefits, however, these results are preliminary.¹³⁸

Calcium

Calcium supplementation has been shown to reduce the incidence of hypertension and prematurity, but its role in reducing LBW is less clear. Most of the studies to date have been conducted in industrialized countries,

with the exception of a recent trial of calcium supplementation in India. The results of this trial were significant. Infants born to first-time mothers who received 2 g of calcium daily from 20 weeks of gestation were heavier by about 100 g at birth (2731 ± 278 g) compared to the placebo group (2626 ± 309 g).¹³⁹

Magnesium

Magnesium deficiency has been suggested as a predisposing factor to preterm labour, premature rupture of foetal membranes and IUGR.¹⁴⁰ These findings were confirmed in a trial of nearly 1000 women at risk for pregnancy-induced hypertension: there were fewer premature births, and a smaller proportion of SGA and IUGR infants in the group who received 15 mmol magnesium sulphate supplementation compared to those who received a placebo.¹⁴¹ Similarly in a double blind study in a Swiss population, 568 women supplemented with 15 mmol of magnesium aspartame hydrochloride from the 16th week of gestation had significantly fewer maternal hospitalizations, a reduction in preterm delivery and less frequent referral of the newborn to the neonatal intensive care unit. There were no significant differences, however, between the two groups with respect to placental weight, birthweight, infant length and head circumference. The authors concluded that for women at risk of pregnancy-induced hypertension, magnesium supplementation during pregnancy has a significant influence on foetal and maternal morbidity both before and after delivery.¹⁴² The role of magnesium in reducing LBW is still controversial, and there have been no such studies in developing countries where the problem of pregnancy-induced hypertension may be greater.

Multiple Micronutrient Supplements

Although multi-vitamin mineral supplements are prescribed and consumed regularly by women in many industrialized countries, there are few studies that have examined the benefits of these supplements on birthweight. Relevant findings from observational studies are summarized below as well as two experimental studies on multiple micronutrient supplements.

The prevalence of LBW was significantly lower among Swedish women who regularly consumed iron and/or a multivitamin-mineral supplement, although confounding variables such as smoking or prenatal care were not assessed in the study.¹⁴³ Another study in the USA found that antenatal multivitamin-mineral supplement use during the first and second trimester resulted in a two-fold reduction in LBW, but was primarily due to reductions in premature births rather than reductions in IUGR among term infants.¹⁴⁴ Associations between dietary intakes of multiple mineral and several B vitamins and birthweight were positive in a 1991 UK study, but no association was found with this combination in a study among African American women in the USA.^{145,146} Dietary intakes of riboflavin and niacin (vitamins B₂ and B₃) were associated with an increase in birthweight in a study in Ecuador;¹⁴⁷ and in Mexico there were no reported associations between birthweight and iron, zinc and vitamin C supplements given to pregnant women who were deficient in these micronutrients.¹⁴⁸

In 1993 the Hungarian Family Planning Program supplemented over 4000 women from pre-conception until week 12 of their pregnancy with either a multivitamin-mineral or a trace element supplement. There were no differences in birthweights and mean birthweights were over 3000 g. The birthweights of both groups, however, were significantly higher by approximately 100 g than the birthweights of the general population. The LBW prevalence was nearly twice as high in the general population, and although there was a higher proportion of first-time mothers and highly educated women in the treatment groups, the role of multiple micronutrient supplementation can not be ignored (Figure 17).¹¹²

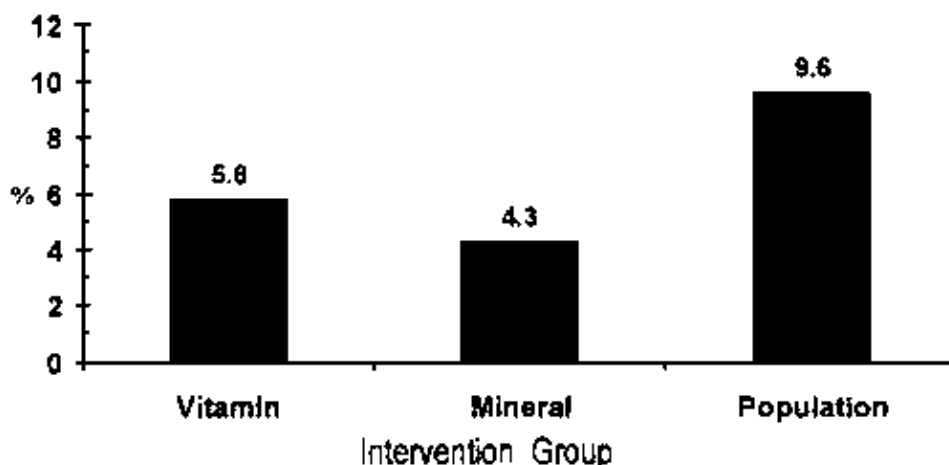


Figure 17. Prevalence of LBW with multiple micronutrient supplementation – Hungary

Source: Czeizel et al. (1993) *Ann NY Acad Sc* 678:266.

In another study of multiple micronutrients, HIV+ Tanzanian women were supplemented from 12–27 weeks of pregnancy until the time of delivery with either a multivitamin–mineral (MVTM), a MVTM without vitamin A, vitamin A, or a placebo. There were significant differences of approximately 120 g in the mean birthweights for mothers in both groups who consumed the MVTM. The LBW prevalence was approximately twice as high in the vitamin A and placebo group (Figure 18). Overall the multivitamin supplementation decreased the risk of LBW by 44%, severe preterm birth (<34 weeks gestation) by 39%, and SGA by 43%.¹⁰⁴

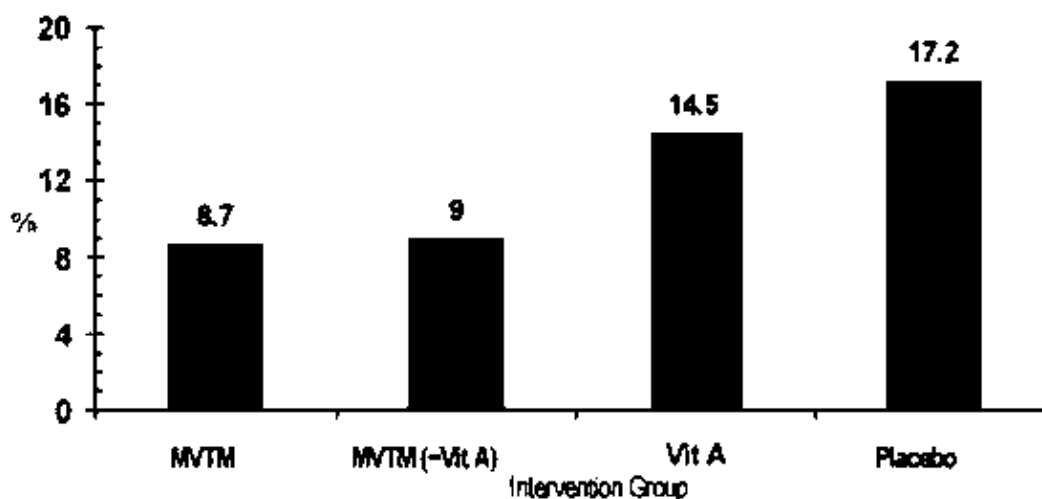


Figure 18. Prevalence of LBW with multiple micronutrient supplementation* – Tanzania

Source: Fawzi et al. (1998) *Lancet* 351:1477. *Intervention groups were given one of the following: MVTM vitamins A, B1, B2, niacin, B6, B12, C, E, and 120 mg ferrous iron (400 mg ferrous sulfate), 500 µg folic acid; MVTM without vitamin A; Vit A: 5,000 IU vitamin A, 120 mg ferrous iron, 500 µg folic acid; Placebo: 120 mg ferrous iron and 500 µg folic acid.

An ongoing RCT is examining the daily ingestion of MVTM supplements compared to daily ingestion of iron supplements in pregnant, rural Mexican women. Preliminary results from this trial are not yet available.¹³⁸

IV. Nutritional Interventions to Reduce Low Birthweight: Critical Issues

Women's Body Composition

Improvements in the growth of girls and in the nutritional status of young women and mothers may be essential elements to reducing LBW. The Pune Maternal Nutritional Study in the Maharashtra, India detailed the relationship between the anthropometry of 2500 women living in six rural villages (measured every three

months) and the anthropometry of the 633 full-term infants born to these women over a three year period. Mothers were short and underweight (mean height 1.52 m; weight 42 kg; and BMI 18 kg/m²) and their infants' birthweight averaged 2648 g.¹⁴⁹ When compared to measurements of mothers and infants born at Southampton, the Pune mothers and their infants were smaller in all dimensions. The height and weight of mothers from Pune were lower by almost two standard deviations, as was birthweight and placental weight. There was considerable variation, however, in the degree of deficit seen in the various components of the infants' weight. Mid-arm and abdominal circumferences were dramatically lower by almost three standard deviations. In contrast, the skinfold thickness was only slightly reduced, and therefore subcutaneous fat was relatively spared. Head and length growth were also relatively preserved, although not to the same degree as fat. Thus, although these underweight babies were very thin in the sense that they had reduced soft-tissue mass, they were actually relatively 'fat'. It may be that fat is preserved as a neonatal survival mechanism. This neonatal phenotype, with reduced muscle mass and relatively increased fat, resembled the phenotype of the adult Indian patient with insulin resistance and diabetes – possibly that phenotype is laid down *in utero*.¹⁵⁰

Heavier mothers had infants who were larger in all these dimensions. From the prepregnant anthropometric measurements four maternal components were derived: height, head circumference, fat mass (calculated from skinfolds) and muscle mass (calculated from arm muscle area). Maternal height predicted neonatal length. Maternal fat mass predicted neonatal skinfold thickness. Maternal muscle mass was unrelated to neonatal size, possibly due to the indirect nature of the measurement. The maternal measurement most strongly related to overall foetal growth was head circumference. Mothers with larger head circumference measurements delivered infants who were larger in all these dimensions, independent of the other maternal measurements. This relationship supports the concept that a mother's head measurement reflects her own growth in early life and that a woman needs good nutrition and growth at all stages of her own life if she is to be able to nourish her foetus in all its dimensions and components.¹⁵¹

A study in Mysore, India, led to the development of a theory about how body composition is related to LBW, intergenerational undernutrition and chronic disease. In a population which has been chronically undernourished over generations, such as some rural populations of India, LBW, childhood stunting, and adult chronic energy deficiency mean that the mothers are short and thin. They have short thin infants. As adults, if these infants are less physically active or even mildly obese, insulin resistance increases as well as the risk of cardiovascular heart disease (CHD). As nutrition improves, women who were LBW and stunted in early life become overweight or obese. Due to their own LBW, they are insulin resistant. During pregnancy they develop hyperglycaemia and diabetes which leads to pancreatic overstimulation in the foetus, and macrosomic changes in the form of increased fat deposition. These are changes which pre-dispose that foetus to pancreatic failure in later life, further increased insulin resistance, and diabetes in adult life. The situation may resolve itself when improvements in nutrition continue and persist such that improved nutrition filters down to the foetus and filters down to girls and young women.¹⁵⁰ Overall, these studies from India add to the evidence that maternal nutrition throughout the lifecycle is important for optimal foetal growth and may provide clues to the high prevalence of LBW infants in India.

Pregnancy Weight Gain

A woman's prepregnancy weight and nutritional status, coupled with the amount of weight gained during pregnancy, are extremely useful indicators for interventions to reduce LBW.¹² WHO recommends that women in developing countries gain at least 1 kg per month during the last two trimesters of pregnancy, resulting in a weight gain of at least 6 kg.⁶⁰ Although attainable and realistic, most women in developing countries still do not gain the recommended amount of weight. Many of these mothers practice *eating down* (eating less) during pregnancy due to their fears of the possibility of cephalo-pelvic disproportion (CPD) which may lead to obstructed labour. Scientific studies have revealed that increases in head circumference due to food supplements have been too small (about 1–3 mm) to effect CPD.¹⁵² In fact, food supplementation during pregnancy is not only highly unlikely to cause CPD, convincing evidence exists that food supplements actually reduce perinatal mortality.⁸⁸ The primary objective of food supplementation during pregnancy is to *prevent* low birthweight and not to promote foetal overgrowth.

The timing of a woman's pregnancy weight gain is also important. Many investigators agree that weight gain in the second and third trimester is of greater importance for ensuring foetal growth than weight gain during the first trimester. Examination of the components of a woman's weight gain during pregnancy supports the importance of later weight gain (Figure 19). Weight gained earlier in pregnancy primarily contributes to maternal reserves, and is secondarily due to the growth of the placenta, breasts, uterus and increased amniotic and extracellular fluids. Not until after twenty weeks does the foetus begin to increase dramatically in

size.

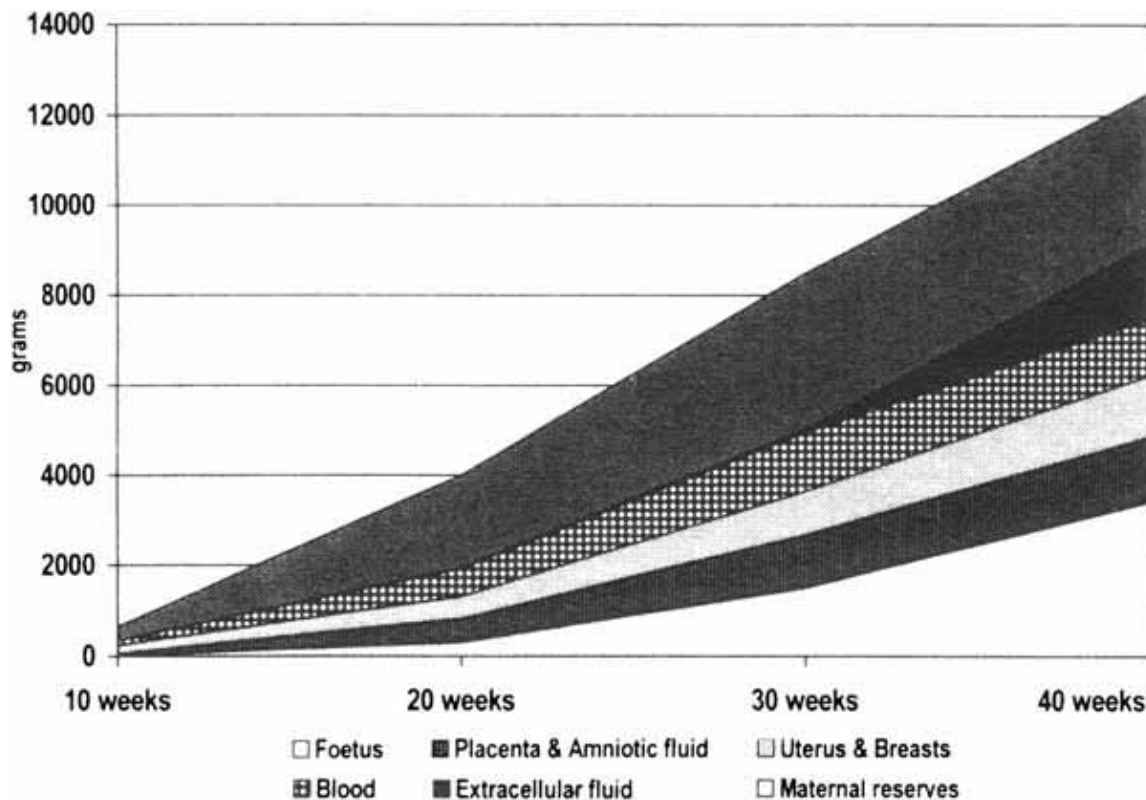


Figure 19. Components of weight gain in pregnancy: later weight gain ensures healthier birthweight

Source: Hytten (1970) *Maternal physiological adjustments*. NAS, Wash. DC

Monthly weight-gain monitoring in pregnancy may not be feasible for many developing country settings, however, usually a minimum of two measurements can be taken at least one month apart anytime during the second or third trimester. Lack of weight gain of approximately 1 kg per month or weight loss between two consecutive measurements is very detrimental to the foetus and/or mother and requires immediate action.⁶⁰ Where weight-gain monitoring is not feasible at all, screening with measurements that require only one contact with a woman, such as prepregnancy weight (or weight-for-height or arm circumference) is still predictive of pregnancy outcome. There is an urgent need for research to assist in the development of pregnancy weight-gain charts which establish appropriate weight-gain curves for women in developing countries. These charts should be clear about the outcomes they are intended to predict and prevent, such as LBW or mortality.⁶⁰

Critical Moments for Intervention

Intervention at any point has the potential to break the cycle of intergenerational undernutrition and LBW. Where resources are scarce, programmes designed to increase prepregnancy weight and weight gain during pregnancy should be given priority. LBW has long-term physiological consequences, and a woman born as a LBW infant herself may have difficulty developing a placenta that will provide adequate nutrition to her own foetus. Studies that have examined the relationship between prepregnancy weight and weight gain during pregnancy have shown that heavier women and women who gain more weight during pregnancy give birth to heavier infants. One study in the Central African Republic of 1,477 women and their children found that maternal prepregnancy weight, representing a woman's long-term nutritional status, was the most important determinant of birthweight, and accounted for 13% of the variance in birthweight. Weight gain during pregnancy, representing a woman's short-term nutritional situation, explained only 5.6% of the variance.¹⁵³ Thus, the case is made to increase the prepregnancy weight of women first, because women who are well-nourished prior to pregnancy will be most likely to deliver heavier infants.

Because the rate of LBW decreases as pregnancy weight gain increases in undernourished women, it is crucial to target this group. In many populations, food supply and birthweights are seasonal, so it is especially important to target interventions during hungry seasons, or the times of lowest resources (Figure 11). In many

regions of the world, women breastfeed one child while pregnant with the next. A study among rural Guatemalan women participating in a nutrition supplementation trial found that lactation overlapped with pregnancy in more than 50% of the pregnancies.¹⁵⁴ For cases where overlap occurred, 41% continued to breastfeed into the second trimester and 3% in the third trimester. Women who breastfeed while pregnant increased their intake of the supplements which were offered. The women also increased their supplement intake if they had short recuperative periods (less than six months when a woman is both non-pregnant and non-lactating) – a time when maternal fat stores are reduced. Women with no overlap in breastfeeding and pregnancy and those with overlap had children with different birthweights. The longest overlap group had children with the lowest birthweights and women with long recuperation periods had infants with the highest birthweights.¹⁵⁵ Family planning programmes to extend the birth interval to a minimum of two years would, therefore, help to decrease LBW.

A child's nutritional status was associated with the BMI of the mother, the socioeconomic status of the family, and the child's own breastfeeding status in a study in Bangladesh. Thus, maternal nutritional status is a proximate determinant of the child's nutritional status and should be considered in programmes aimed at improving child health.^{156,157} SGA infants will experience catch-up growth if given adequate nutrition, especially if breastfed.¹⁵⁸ Micronutrient supplementation has been shown to increase children's appetite, energy and growth. An iron supplementation trial in Kenya found that provision of iron supplements resulted in improved growth and improved appetite in children 6–11 years of age.¹⁵⁹ The critical times to target food and micronutrient interventions in children are those during the highest growth velocity, before the age of three¹⁶⁰ – at which times it may also be possible to reverse earlier growth retardation. Thus, interventions during infancy and childhood can result in some growth recovery. It is less clear what can be done during the adolescent period to promote growth, particularly in height, and more research is needed in this area.¹⁶¹ Adolescents, however, should receive dietary counselling especially if they are pregnant.

In summary, improving prepregnancy weight and weight gain during pregnancy are effective strategies which reduce and prevent LBW. Good nutrition throughout the lifecycle, however, is critical. Breastfeeding, appropriate complementary feeding, and adequate micronutrient status are especially important during infancy and early childhood. Improved dietary intake should be promoted among adolescents, particularly among pregnant adolescents. Food supplements need to reach undernourished pregnant women. Reducing and preventing LBW requires a commitment to implement long-term strategies.

Cultural Beliefs, Social Practices and Behavioural Change

What are the important cultural beliefs and social practices related to food behaviour during pregnancy, and beliefs related to birthweight, that could *affect* interventions aimed at improving maternal diet? To date, public health professionals have focused mainly on the role of beliefs and practices as *determinants of food intake* during pregnancy, but it is also necessary to understand the effects of pre-existing beliefs and practices on the ways in which individuals and groups respond to interventions. In other words, attention must be given to the role of cultural beliefs and social practices as *determinants of responses to directed behaviour change*. When the focus shifts from determinants of intake to determinants of responses to interventions, it becomes critical to understand the beliefs and practices that are relevant for each specific type of intervention. In the basic question: "What are the cultural beliefs and social practices that could constrain or facilitate people's responses to _____?", the blank space could be micronutrients delivered through capsules, micronutrients delivered in the form of a powder to add to a drink, the provision of additional food, food subsidies, nutrition education, counseling, or various combinations of these. Communication is a central aspect of directed behaviour change, and communication, by definition, involves multiple parties. Whose cultural beliefs and practices will constrain or facilitate the implementation of interventions? The multiple parties not only involve the recipients of the interventions – the women in the communities where LBW is endemic and their husbands, mothers, mothers-in-law, neighbours and community leaders – but the cultural beliefs of public health and nutrition professionals at different levels of the system must also be examined and understood.

It is widely recognized that cultural beliefs and practices play a role in the successful delivery of nutrition messages to the community. Although there is not a body of experimental studies to document this concept, there are various types of evidence to validate this statement. The largest body of relevant data about the importance of cultural beliefs and practices comes from case studies in which social scientists use ethnographic and survey data to show how people in a community or region have interpreted a health or nutrition intervention in ways that were not intended by the public health professionals and that this constrained or reduced the effectiveness of the intervention. Another kind of evidence consists of reports about successful interventions that used culturally sensitive and appropriate materials. Commonly, with a

culturally sensitive approach, investigators describe population responses that are more positive than is typically the case with public health and nutrition interventions.

Other types of inferential evidence come from observations of beliefs and practices that reflect population interpretations of common public health interventions. One example, which has been repeatedly documented in many parts of the world, concerns the use of oral rehydration solutions (ORS) for childhood diarrhoea. In many populations, families give ORS in teaspoon-size quantities instead of in the larger quantities needed to combat diarrhoea. This misunderstanding may occur because the context in which ORS is being promoted leads families to conclude that the solution is a kind of medicine and must be given in small doses. Similarly, many families will use ORS for some episodes of diarrhoea but not for others. An investigation of why this occurs reveals that in most cultures people believe there are many different types of diarrhoea, each having its own descriptive word. Because educational messages about ORS typically only use the word 'diarrhoea', and not other common descriptive terms, the recipients of these messages conclude that ORS is useful only for the one specific type of 'diarrhoea'.

Finally, another line of argument to support the need for culturally appropriate interventions is the increasing demand from ethnic groups within large national entities for locally appropriate health and education. This demand is particularly evident within organized minorities in industrialized countries, where articulate spokesmen and women have put forward arguments, under the general rubric of autonomy, that include a perceived need for culturally appropriate education and services.

As would be expected of an experience that is as central and as difficult as pregnancy and childbirth, virtually all cultures have beliefs about appropriate behaviours during pregnancy. In most cultures, *bad* outcomes for either mother or infant are often attributed to a failure to engage in appropriate behaviours during pregnancy. In many cultures these behaviours often relate to food, particularly the avoidance of certain foods. Other beliefs involve more general recommendations such as the idea that eating a lot or *too much* during pregnancy will result in a large baby and a difficult delivery.

The following example from Gujarat, India, is typical of many cultures, particularly in Asia. In an ethnically diverse community, a non-governmental organization research team, SEWA-Rural, found that the local cultural beliefs about how women should eat during pregnancy were heavily slanted towards foods to be avoided.¹⁶² These avoidances were directly related to local concepts of health and illness (Table 2). The investigators also found two opposing views about why eating a large quantity of food during pregnancy could result in a *bad* outcome either for the foetus or the mother. Some members of the community assume that food shares the same space as the foetus in the mother's abdomen, thus limiting the space for the foetus to grow and eventually causing it to *waste away*. For other people, eating a lot of food is thought to make the foetus grow fat thus, causing problems for the mother at delivery. Either scenario produces a fear of eating too much during pregnancy.

The extent to which individual women believed in these guidelines varied. Some women felt that these beliefs were simply wrong. There was also variation in the extent to which women actually engaged in restricted intake or avoided particular foods. But there was also evidence that women who believed in following these guidelines did, indeed, eat less. Many of the respondents said that they ate what their mothers-in-law gave them, and, in general, the older women were more strongly committed to these ideas.

How can information on cultural beliefs and practices be used in the design of interventions to prevent LBW? Some general principles can be suggested.

1. Work with local people to:

- ? adapt dietary recommendations for locally available foods

- ? identify appropriate analogies; an analogy that links a new idea with one that is already present in the culture helps to make this new information understandable and at least potentially actionable

- ? develop educational messages built on local concepts.

2. Avoid making recommendations that are *head-on collisions* with local cultural beliefs. For example, one strategy in designing a LBW prevention package for the community in Gujarat, would be to encourage weight gain during pregnancy. Advising women to "eat more" runs contrary to local customs. Alternatively recommendations could be framed in terms of eating

to help prevent *wasting* in the foetus, as women are concerned with this. Positive eating behaviours can be related to mother's health and this, in turn, to the importance of maternal health in preventing and overcoming delivery problems. Eating recommendations could thus be linked to the concept of achieving *balance* during pregnancy, which can be associated with cultural concepts of *hot* and *cold*.

In addition to the challenges of obtaining information on local beliefs quickly and inexpensively, paying attention to cultural beliefs and practices in the design and implementation of interventions presents other challenges. One important challenge is how to overcome the problem of *non-shared assumptions*. Health professionals may not be comfortable talking about *hot* and *cold* foods, *ratevo* or *chonte* (Table 2). Engaging in cross-cultural communication involving ideas which run counter to their own beliefs can present a major hurdle for many health professionals.

Table 2. Common food avoidances in an Indian community

<i>Reason for Avoiding</i>	<i>Descriptive Term</i>	<i>Examples of Foods</i>
Cold or sour. Causing problems in labour	<i>Thanda Khata</i>	Milk, Curds, Banana
Hot, causing pus, rotting and miscarriage	<i>Garam</i>	Meat, Fish
Causes <i>ratevo</i> , anemic illness category that includes a variety of perinatal illnesses	<i>Ratevo thay</i>	Cooking oil, spices, salt, meat, fish
Difficult, delayed childbirth because baby "sticks" to the uterus	<i>Chonte</i>	Milk, curds, buttermilk groundnuts.

Source: SEWA (1994) in Gittelsohn J et al. *Listening to Women Talk About Their Health: Issues and Evidence from India*. New Delhi: Har-Anand Publications

The cultural beliefs, values, social behaviours of all these parties are involved in the actions we call "nutrition interventions." Therefore, it is essential to understand and address the beliefs of the professionals, the implementers and the community in the design of the interventions. (Pelto, 1999)

Cultural sensitivity may be enhanced by the use of Community Nutrition Promoters (CPNs) who have a key role in the implementation of the community based nutrition services. This is done in the Bangladesh Integrated Nutrition Programme (BINP). CPNs are recruited from women residing in the village who have a minimum of eight years of education, and have the willingness and time to serve the community in return for a modest honorarium. On-the-job training and the development of problem-solving skills are provided when necessary as these women attempt to encourage behavioural change in their clients.⁷⁷

Nutrition programmes in Bangladesh, Costa Rica and Thailand have included behavioural change components with varying success. In Bangladesh, success of the *information, education and communication* (IEC) component is considered fundamental to the BINP given the project's focus on caring practices to promote growth in young women and children, and on mobilizing communities to take action against undernutrition. Evaluation of the programme indicates that there is improvement in a limited number of behaviours but much of the communication remains didactic and general with inadequate attention to addressing key resistance points identified through initial BINP formative research. Additionally, the lack of funds have limited mass communications activities. The IEC component is viewed as a weak link in the project as a whole but steps have been taken to further develop the training manuals from a behaviour change perspective.

As the BINP evolves into the broader National Nutrition Program (NNP) the behaviour change component and counselling for mothers will be strengthened. Again, behaviours and messages to be conveyed will be based on formative research conducted during the preparation and implementation of BINP (Figure 20). Where necessary, additional research will be conducted to identify barriers to the adoption of key behaviours. Customized "packages" of messages will be prepared and conveyed during the planned 30 contact sessions throughout pregnancy and the first 24 months of life.¹

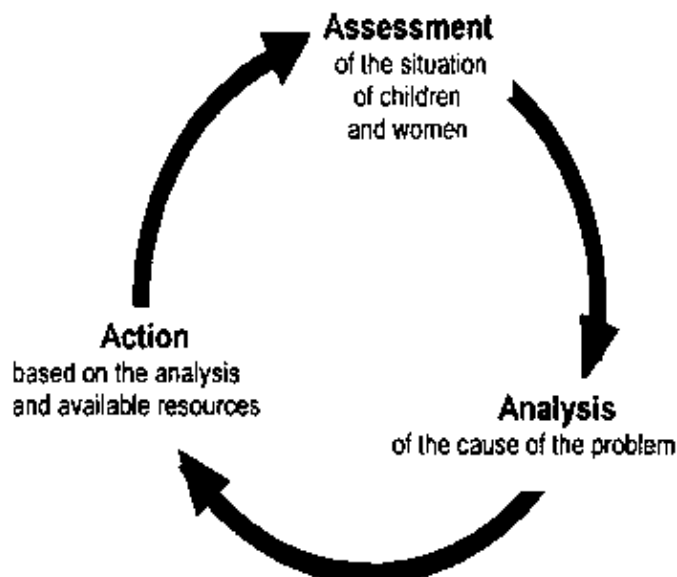


Figure 20. Triple A Cycle; Assessment–Analysis–Action

Source: UNICEF (1997) *The Care Initiative*. UNICEF, NY.

The reduction of LBW is a key outcome measure for the new NNP and women at risk will receive supplements providing 600 kcal/day, which are to be consumed in addition to the normal daily diet. A multiple micronutrient supplement will also be provided. Additionally, health and family planning services will be strengthened at both the primary and secondary levels and will be monitored for their effectiveness. There will also be outreach centres and clinics for immunization, birth spacing and postpartum contraception, pregnancy-related care and treatment of pneumonia and diarrhoea. Household food security issues will also be addressed with income-generating activities such as poultry rearing and “nutritious” gardening, and a micro-credit facility will be available. Since the Bangladesh public already receives a large number of behavioural change messages from the health and family welfare programme, nutrition messages will be integrated into this programme’s communication strategy.¹

Costa Rica is one of the few developing countries to achieve impressive health statistics despite a low per capita income. Because of a highly motivated and committed government with the foresight to divert some of its funds from curative to preventive medicine, Costa Rica made remarkable achievements in reducing its LBW and infant mortality during the 1970s. The complementary food programme was associated with a series of educational activities targeting programme recipients as well as the population in general. Activities include educational talks; demonstrations on how to prepare different meals; nutritional information implemented by other members of the health team including physicians, nurses and rural health assistants; and the use of radio programmes and printed materials to deliver messages. This is an excellent example of how limited resources invested wisely can result in improvements in health that rival many industrialized countries.⁸⁰

Thailand is another example of a country that has achieved substantial success by integrating nutrition into rural community development activities and the primary health care framework. Their *quality of life* campaign indicated that good nutrition was not a goal but rather a means of promoting development. The nutrition programme has not been implemented as a single programme but is part of consecutive broader health and nutrition plans. In implementing their programme to modify food habits, field researchers lived in villages for four to eight months to gain an in-depth understanding of the target populations. Their food habits and beliefs, as well as their food selection and processing practices, and maternal and child nutritional behaviours were studied. Several food practices were found to affect general nutrition among the targeted population. These included inappropriate traditional beliefs about food, pregnancy and child development; food taboos and restrictions during pregnancy, lactation and child illness; inadequate awareness of nutrition; and the role of external influences, such as the elderly or media advertisements, on food selection and consumption. Field researchers then worked with community members to develop a *top-down support and coordination* and a *bottom-up planning and implementation* model to facilitate change in nutrition-related practices. Nutrition communication took many forms depending on what was best suited for the individual community. Media were selected based on their appropriateness in terms of local socio-cultural conditions, accessibility and frequency of exposure to the target population. These media promoted new food habits and educated target group members through a process of creating new knowledge; initiating new attitudes, perceptions and beliefs; and introducing new or modified food and nutrition practices. Supportive activities included the development of new recipes or the adaptation of existing ones; food production and preservation

demonstrations; school lunch program improvements; literacy campaigns; environmental sanitation campaigns; and basic health services.

Evaluation of the programme showed improvements in outcome parameters in a number of areas. Attitudes towards proper food habits, food preservation, the dangers of eating potentially harmful foods all improved significantly. In northeastern Thai villages where undernutrition was high, mothers who participated in the programme intervention delivered infants with an average weight of 2,913 g compared to a birthweight of 2,846 g for non-participating mothers. Additionally, the prevalence of LBW was 12% in the programme areas compared to 14% in the nearby villages without the programme. Nutritional status and dietary intake results showed an improvement after programme implementation, although this improvement was not statistically significant.⁷⁸

There are many lessons to be learned from this programme. The investigators concluded that the changing of food habits requires an accurate, early analysis of the pragmatics of community life. This means consideration of socio-cultural, economic, political, psychological and physical environmental suitability and the roles these play in shaping existing food practices. A clear understanding is also needed about the target groups' behaviour, attitudes and environmental constraints at a personal and family level. Communities, local development organizers and project personnel must share activities and responsibilities. Finally the intervention must develop a variety of supportive nutrition communication messages and activities that fit the practicalities of village life and its members' interests. **“This is crucial because, ultimately, community members are the ones who make the real difference. They are the ones who must change themselves.”**⁷⁸

V. Improving Outcomes of Low Birthweight Infants: Potential Interventions

Effective and large-scale practical interventions to prevent LBW will have enormous impact on the health and productivity of individuals and society, particularly in those regions where the prevalence of LBW is high. The causes and impact of LBW are complex and best considered within the proposed lifecycle illustration (Figure 1). A primary public and individual health strategy, as well as a prevention strategy, must be accorded to interventions directed at mitigating the detrimental effects of LBW. With the exception of prematurity, it is probably more accurate to view IUGR manifested as LBW as a marker or risk factor rather than a direct cause of poor outcome. There are surprisingly few well-designed childhood interventions to improve outcomes of LBW infants. LBW infants that are thin for length (disproportionate) exhibit catch-up weight gain if provided with adequate early nutrition. The catch-up growth potential of infants who are short with a normal weight relative to their length (proportionate) is limited with accelerated length accrual generally restricted to the first few months with optimal nutrition.¹⁶³ Yet even with maximal early nutrition, these infants as a group do not attain normal length and are destined to be stunted compared to their non-LBW counterparts.³

Measuring Size at Birth – The early identification of the LBW infant is essential for any comprehensive initiative to improve its chances of survival.^{25,164} In developing countries where most of the world's LBW infants are born, however, a large proportion of births take place at home and birthweight, statistics are not available.⁶ Alternative anthropometric measurements to identify LBW have been proposed.¹⁶⁵⁻¹⁶⁸ Chest circumference has been recommended as a simple and accurate surrogate for birthweight measurements, but no cut-off point has yet been determined for use internationally.¹⁶⁵ Chest and head circumference were used to identify LBW preterm infants in Ethiopia, and cut-off points with the best sensitivity and specificity were 30 cm for chest and 31 cm for head circumference.¹⁶⁹ Studies from India also showed a chest circumference of ≥ 30 cm and a mid-arm circumference of ≥ 8.7 cm had the best sensitivity and specificity for identifying LBW infants. Mid-arm and chest circumferences are simple, practical, quick, and reliable indicators for predicting LBW and neonatal outcome in the community.^{168,170} Whether measuring chest circumference can be easily taught to community health workers or traditional birth attendants (because of the rapid chest movements accompanying breathing) was a subject of debate among the conference participants in Dhaka.

More recently, head and chest circumferences in more than 5,000 newborns in Pelotas, Brazil were used to identify infants at risk for morbidity and mortality.¹⁷¹ A head circumference of 33 cm was sensitive (91.6%) and somewhat specific (85.5%) in identifying LBW preterm births. The same measure was less sensitive in predicting neonatal deaths (67.6% sensitivity; 81.9% specificity) but still useful. A chest circumference of 31 cm identified 92.8% of LBW preterm and 76.5% of neonatal deaths. No cut-off has been established for the number of days after birth during which the chest circumference can be considered a reliable surrogate for birthweight measurement. When the head and chest circumferences are used together, they have an increased capability to identify infants at risk of morbidity and mortality.¹⁷¹

Breastfeeding and Complementary Feeding – Although catch-up growth of the LBW infant is difficult to achieve, further growth faltering may be averted by optimum breastfeeding and complementary feeding practices. Exclusive breastfeeding for about six months can be recommended even for LBW term infants in developing countries.¹⁶³ Exclusive breastfeeding not only provides immunity and protection against illnesses, especially ALRI and diarrhoea,^{17,18,172} it lengthens the period of postpartum amenorrhoea and hence, lengthens the birth interval, which is strongly related to infant and child survival.¹⁷³ Immediate and exclusive breastfeeding is especially important as suckling increases the infant's body temperature, and the proximity to the mother that breastfeeding provides is beneficial in reducing hypothermia. The *Kangaroo Care Method* is also effective in reducing hypothermia, and has been shown to reduce perinatal and neonatal mortality and help these infants grow faster.^{174,175} In the Kangaroo Care method, a well preterm or LBW infant, wearing only a diaper, is placed between the mother's breasts with skin-to-skin contact, instead of being placed in an incubator. Infants should be put to the breast within one hour after birth and should not go without breastfeeding for more than three hour intervals since fasting is associated with a fall in blood glucose. Practices that restrict the early initiation and frequency of breastfeeding are likely to increase the incidence of hypoglycemia.^{176,177} There is evidence to link having been breastfed as a child with improved cognitive performance,^{178,179} and that breastfeeding may have a metabolic programming effect in preventing obesity¹⁸⁰ and reducing the risk of several chronic diseases.^{181,182}

The increased rates of growth failure and undernutrition in LBW infants observed after six months of age are probably related, perhaps in a major way, to poor complimentary feeding practices. While appropriate complementary feeding is important for all infants, it might be of even greater importance for LBW infants because of their poorer growth and increased nutritional vulnerability upon entering this stage of dietary transition. Research in Bangladesh indicates that the timely introduction of appropriate nutrient-rich complementary foods, and regular feeding in addition to ongoing breastfeeding after six months requires special attention.¹ The NNP intends to provide mothers with regular counseling on the use of colostrum and exclusive breastfeeding during the first six months, and the continuation of breastfeeding until the age of two years. Mothers, as well as fathers, mothers-in-law, and other caregivers will be encouraged to introduce locally appropriate, nutrient-dense complementary foods in sufficient quantity and quality from the age of six months. This focus on complementary feeding at the individual level will be supported by a similar emphasis in the national behaviour change communication activities.¹

Care – The two most important elements of a *Care* strategy include a method of assessment, analysis and action (Figure 20), and a conceptual framework which indicates that inadequate maternal and child care is an underlying cause of undernutrition (Figure 4), and thus perpetuates LBW. The UNICEF *Nutrition Strategy* for LBW prevention generically emphasizes care for women (especially antenatal care), breastfeeding/complementary feeding, psychosocial care, food processing, hygiene practices, and home health practices as programme elements. This *Care for Women and Children* component (*Care Initiative*) additionally includes practices and/or behaviours that the family, community and/or government should provide for women (Box 3) as part of their human right to health and their right to adequate food.¹⁸³ The UNICEF *Nutrition Strategy 1990* has been exploring mechanisms to mobilize and support existing local skills and resources while simultaneously implementing technical interventions. Early establishment of community-based monitoring systems and involvement of communities, particularly of women in planning, implementing and monitoring nutrition programmes, are criteria for success.⁷⁵

Box 3: The UNICEF Care Initiative "Care for Women"	
Practice/Behaviour	Comment
Education	? Ensure that girls have equal access to school
	? Ensure that women have access to essential health information (i.e., distribute <i>Facts for Life</i> published by UNICEF, WHO and UNESCO)
Workload and Time	? Help to reduce domestic chore workload (collecting water and fuel, planing and tending crops, preparing food) to ensure more time for child care

Autonomy/ Respect in the family	?	Ensure that mothers have adequate decision-making power, access to family income, assets and credit
	?	Lack of the above may undermine a woman's self-esteem, self-confidence, and her ability to care for herself and her children
Mental Health/Stress Self-confidence	?	Often compromised by pressures of poverty, low status in family and community, and lack of control over basic life decisions such as reproduction and child care
Physical health	?	View from a lifecycle perspective
Nutritional status	?	Ensure a fair share of family food and resources at all ages
	?	Identify and resolve gender gaps in nutritional status and health seeking behaviours for all age groups
Reproductive health	?	Delay age of first pregnancy
	?	Provide support for birth spacing
Pregnancy Lactation	?	Protect/support health and nutrition of mother for + pregnancy outcome
	?	Provide extra amounts of family foods
	?	Reduce workload
	?	Facilitate prenatal care and safe birthing
	?	Post-partum rest

Source: UNICEF (1997) *The Care Initiative: Assessment, Analysis and Action to Improve Care for Nutrition*, UNICEF: New York.

Behavioural and Cognitive Development – There is substantial evidence that reduced breastfeeding, SGA birthweight, iron and iodine deficiency, underweight and stunting are associated with long-term deficits in cognition and school achievement.¹⁸⁴ Physical stunting is closely linked to impaired mental and psychomotor development. The only supplementation study aimed specifically at stunted children was conducted in Jamaican children aged 9 to 24 months.¹⁸⁵ The stunted children received nutritional supplementation for two years with or without psychosocial stimulation. Supplementation and stimulation produced independent benefits to the children's mental and motor development. The benefits from a combination of supplementation and stimulation were additive, and only the children receiving both treatments caught up to the non-stunted control group in developmental levels. The implications of these findings are that at least part of the deficit in the development of stunted children is due to poor nutrition.¹⁸⁶ Interventions which promote catch-up growth also improve long-term mental ability, provided they are timely and accompanied by mental stimulation such as maternal involvement in the stimulation activity (child's play). The value of play, taught and encouraged by the mother, is an example of the importance of maternal care for a child's development and well-being.¹⁸⁷ In countries where stunting is highly prevalent, there is an urgent need to institute programmes to improve children's nutritional status. Such programmes are probably most effective if instituted with children within the first three years of life, and are integrated with child care and stimulation (play) interventions.¹⁸⁶

Micronutrient Interventions – Unlike developed countries where zinc supplementation during pregnancy in certain populations has improved birthweight, no benefit has been observed from recent well designed trials in developing countries. Similarly, vitamin A supplementation does not appear to have an important role in improving birthweight while, in contrast, there is some evidence that iodine supplementation might be important. Folic acid can prevent neural tube defects, but evidence as to whether iron and/or folic acid

supplements reduce the prevalence of LBW, prematurity and maternal mortality is limited. And while anaemia during pregnancy is associated with LBW, the benefit to birthweight of iron supplementation is less clear. Recent data also suggest benefits from multiple vitamin–mineral supplements, particularly with regard to decreasing adverse pregnancy outcomes. Although there is evidence of interactions among several micronutrients at the metabolic level, very little is known about the significance of these interactions for pregnancy outcomes.¹⁴⁴

There is a need for well designed RCTs that assess the effect of multiple micronutrients on maternal outcomes such as morbidity and mortality, anaemia, and pregnancy complications. These trials should focus on infant outcomes such as morbidity and mortality, growth and development, and micronutrient status, in addition to LBW and especially IUGR. It is essential that these trials be conducted with sufficient sample sizes if biologically relevant differences in birthweight (approximately 100 g) are to be detected. The need for RCTs are especially urgent in developing countries, i.e., among populations with high rates of LBW due to IUGR and where nutrient deficiencies do not occur in isolation and multiple micronutrient deficiencies are common.

VI. Conclusions and Future Research

Symposium Recommendations

LBW has multiple etiologies, however, IUGR accounts for the great majority of LBW in developing countries. Many questions remain unanswered about IUGR, such as the appropriate timing, amount and characteristics of nutritional supplementation; whether or not micronutrient supplements will have an impact; the impact of infection control on LBW prevention; and the full magnitude of health problems faced in adulthood by children born with LBW due to poor foetal growth. This symposium and workshop highlighted the point that there is an urgent need to find answers on sustainable practices to improve women's nutritional status prior to pregnancy, and their weight gain during pregnancy. These practices require behavioural change within households, and behavioural change and communication strategies have been a weak link in many programmes trying to address this issue. The symposium participants acknowledged the limitations of applying results of RCTs in industrialized countries to developed countries, and strongly suggested that findings from observational studies also be considered in order to determine programme effectiveness, if programme interventions are to move forward.

The participants identified important knowledge gaps and stressed the need to:

- ? build a body of evaluation research on behaviour change related to LBW interventions
- ? encourage ethnographic studies related to care, marriage, pregnancy (especially among adolescent girls), birth, and household behaviour (especially decision–making)
- ? strengthen the information, education and communication component of all LBW intervention programmes
- ? support well–designed RCTs which will examine the role of selected nutrient interactions and multivitamin–mineral supplements in improving pregnancy outcomes (especially in developing countries among undernourished pregnant women)
- ? further investigate the effect of food supplementation trials and its influence on the interrelation between maternal and infant outcomes over one or more reproductive cycle
- ? further research the relationship between LBW due to IUGR and chronic diseases in adulthood
- ? improve IEC messages and strategies to increase exclusive breastfeeding rates (rates for partial breastfeeding practices have been improving, however, many women do not exclusively breastfeed)
- ? create culturally appropriate growth curves for pregnant women (and pregnant adolescents) which identify cut–offs for weight and weight gain which will give good birthweight and

maternal fat store outcomes

? identify cut-offs for the simplest, most accurate and practical surrogate for measuring size at birth for infants delivered at home

? develop community worker training manuals which contain modules on diagnosis and guidance for care of the LBW infant.

Workshop Programme Recommendations

LBW Programme “Package” Elements

The lifecycle (Figure 1) was used to view LBW determinants with respect to the different age and physiologic subgroups of the population to give priority to target groups and interventions. It was concluded that LBW solutions require packages of interventions, and that these need to be incorporated into all antenatal health care programmes including safe motherhood programmes, reproductive health programmes, and Integrated Management of Childhood Illness programmes – and that both the number and coverage of these existing programmes be expanded. If multiple micronutrient supplementation is proven to be safe and efficacious in developing country settings, then practical programmatic implications need to be addressed such as supplement composition, cost and bioavailability, the mode and timing of delivery, and compliance. Fortification of common staples would also need to be considered as an alternative to supplements.

Packages of interventions for LBW prevention programmes should have three components:

? case management which increases the coverage of antenatal care and helps prevent repeated pregnancies in quick succession

? behavioural change communication strategies (including those that defer pregnancy until after adolescence and to improve nutrition)

? linkages between existing health care facilities and the community.

LBW prevention/reduction programmes should incorporate elements of UNICEF's *Care Initiative* (Box 3) including a mix of interventions:

? antimicrobial treatments

? antiparasitic treatments

? insecticide-treated bednets

? maternal health records to track gestational weight gain

? probable use of food supplements

? possible introduction of multiple micronutrients during pregnancy.

Recognition of the causes of malnutrition (Figure 4) and the role of the lifecycle conceptual framework (Figure 1) to address LBW was reflected in the structure of the two-day workshop held at ICDDR,B's Matlab training centre. Participants were assigned to one of four working groups: interventions for the management and treatment of infants born with LBW; interventions for reducing and/or preventing low birthweight in the long term (10–13 year plan) (childhood interventions); interventions during adolescence for both girls and boys; and interventions before and during pregnancy. Each group, apropos to their assigned topic, proceeded to define objectives and outline interventions and operational research activities. The working groups suggested that basic indicators should emphasize programme performance monitoring rather than biological measures. Community, private sector and/or international agency resources and intersectoral actors must be identified and involved in the implementation of LBW prevention strategies if the basic causes of LBW are to be addressed. The working groups' recommendations follow.

Management and treatment of LBW infants:

? identify LBW infants

? asphyxia management

? knowledge of crucial warning signs: inability to suck, fever, hypothermia, rapid breathing, umbilical discharge, incessant crying and convulsions

? hospitals to be baby-friendly (the “Ten Steps” which includes skin-to-skin contact and initiation of breastfeeding within one hour of delivery, and frequent breastfeeding) with adequate referrals to lactation consultants and other medical personnel

? Kangaroo Care

? protection of adequate micronutrient status for the infant

? family counselling for special care needs

? exclusive breastfeeding for about 6 months

? breastfeeding counselling (e.g., frequently waking sleepy infants to feed)

? attention to appropriate complementary feeding

? psychosocial stimulation for infant.



Childhood Interventions:

? teach families/mothers how to provide adequate nutrition and care

? school attendance throughout adolescence

? promote good nutrition

? provide micronutrient supplementation when needed.

Adolescent Interventions:

? define adolescence based on distribution curve of age of first marriage

? “Healthy Bride Campaign” to delay marital age and pregnancy

? promote good nutrition, especially among non-pregnant girls

? encourage sustained school attendance

? strengthen school health systems to provide counselling on nutrition and health education

? involve health and education sectors in coordination with community members to explicitly address the particular needs and vulnerabilities of pregnant adolescents

? include boys in all interventions.

Interventions for Pregnant and Postpartum Women:

? provide malaria prophylaxis if required

? reduce intestinal parasite load (mebendazole in 2nd or 3rd trimester)

? correct maternal anaemia (60 mg elemental iron plus 400 µg folic acid per day¹⁸⁸)

? increase to 100% the proportion of women gaining a minimum of 6 kg

? identify and treat abnormal vaginal discharges and other infections

? educate women on warning signs of a problem pregnancy

? improve coverage and quality of antenatal care programmes (encourage visits at least once per month for routine clinical assessments, especially for blood pressure, weight gain and breastfeeding counseling)

? provide food supplements to all pregnant and lactating women (if resources are scarce distribute food supplements during the hungry season, or target the most undernourished pregnant and lactating women)

? improve familial food provision if possible

? facilitate and strengthen Triple A processes and other participatory methods at community level in discussing causes of LBW; include reducing women's workload, increasing food intake, rest, and other underlying and basic causes

? initiate of breastfeeding within one hour after delivery

? increase colostrum feeding

? provide ongoing breastfeeding counseling to increase the proportion of mothers exclusively breastfeeding at one month postpartum, and to manage breastfeeding problems

? introduce appropriate complementary foods at 6 months with continued breastfeeding until at least two years.



References

1. McLachlan M (1999) National Nutrition Project Washington, DC: World Bank.

2. Gülmezoglu M, de Onis M, Villar J (1997) Effectiveness of interventions to prevent or treat impaired fetal growth. *Obstetrical and Gynecological Survey* 52:139–149.
3. ACC/SCN (2000) *Fourth Report on the World Nutrition Situation*. Geneva: ACC/SCN in collaboration with IFPRI.
4. Barker DJP (1998) *Mothers, Babies and Health in Adult Life*. Edinburgh: Churchill Livingstone.
5. Arifeen SE (1997) Birth weight, intrauterine growth retardation and prematurity: a prospective study of infant growth and survival in the slums of Dhaka, Bangladesh Doctor of Public Health dissertation, Johns Hopkins University, Baltimore MD.
6. de Onis M, Blössner M, Villar J (1998) Levels and patterns of intrauterine growth retardation in developing countries. *European Journal of Clinical Nutrition* 52(Suppl. 1):S5–S15.
7. Villar J, Belizán JM (1982) The relative contribution of prematurity and fetal growth retardation to low birth weight in developing and developed societies. *American Journal of Obstetrics and Gynecology* 143:793–798.
8. Kramer M (1998) Socioeconomic determinants of intrauterine growth retardation. *European Journal of Clinical Nutrition* 52(S1):S29–S33.
9. Cameron M, Hofvander Y (1983) *Manual on Feeding Infants and Young Children*, ed. Series, Third ed. Oxford: Oxford University Press.
10. Prada J, Tsang R (1998) Biological mechanisms of environmentally induced causes in IUGR. *European Journal of Clinical Nutrition* 52(S1):S21–S28.
11. Henriksen T (1999) Foetal nutrition, foetal growth restriction and health later in life. *Acta Paediatrica* 429S:4–8.
12. Kramer M (1987) Determinants of low birth weight: methodological assessment and meta-analysis. *Bulletin of the World Health Organization* 65:663–737.
13. WHO (1997) National reports on the third evaluation of the implementation of “Health for All” strategies. New Delhi: WHO Global Database.
14. de Onis M, Blössner M (1997) *WHO Global Database on Child Growth and Malnutrition*. Geneva: WHO.
15. Bukenya G, Barnes T, Nwokolo N (1991) Low birthweight and acute childhood diarrhoea: evidence of their association in an urban settlement of Papua New Guinea. *Annals of Tropical Paediatrics* 11(4):357–362.
16. Ittiravivongs A, Songchitratna K, Rattapalo S, Pattara-Arechachai J (1991) Effect of low birthweight on severe childhood diarrhea. *Southeast Asian Journal of Tropical Medicine and Public Health* 22(4):557–562.
17. Victora CG, Smith PG, Vaughan JP, Nobre LC, Lombardi C, Teixeira AM et al. (1989) Infant feeding and deaths due to diarrhea. *American Journal of Epidemiology* 129(5):1032–1041.
18. Victora CG, Barros FC, Kirkwood BR, Vaughan JP (1990) Pneumonia, diarrhea, and growth in the first 4 y of life: a longitudinal study of 5914 urban Brazilian children. *American Journal of Clinical Nutrition* 52:391–396.
19. Cerqueiro M, Murtagh P, Halac A, Avila M, Weissenbacher M (1990) Epidemiologic risk factors for children with acute lower respiratory tract infections in Buenos Aires, Argentina: a matched case-control study. *Reviews of Infectious Diseases* 12(S8):S1021–1028.
20. Fonseca W, Kirkwood BR, Victoria CG, Fuchs SR, Flores JA, Misago C (1996) Risk factors for childhood pneumonia among the urban poor in Fortaleza, Brazil: a case-control study. *Bulletin of the World Health Organization* 74:199–208.
21. Chandra RK (1999) Nutrition and immunology: from the clinic to cellular biology and back again. *Proceedings of the Nutrition Society* 58(3):681–683.

22. Chandra RK (1997) Nutrition and the immune system: an introduction. *American Journal of Clinical Nutrition* 66(2):460S–463S.
23. Victora C, Smith P, Vaughan J, Nobre L, Lombardi C, Teixeira A et al. (1988) Influence of birthweight on mortality from infectious diseases: A case–control study. *Pediatrics* 81(6):807–811.
24. Ashworth A (1998) Effects of intrauterine growth retardation on mortality and morbidity in infants and young children. *European Journal of Clinical Nutrition* 52(Supplement 1):S34–S42.
25. Barros FC, Huttly SRA, Victoria CG, Kirkwood BR, Vaughan JP (1992) Comparison of the causes and consequences of prematurity and intrauterine growth retardation: a longitudinal study in southern Brazil. *Pediatrics* 90:238–244.
26. Kusin JA, Kardjati S, de With C (1989) Infant mortality in Madura, Indonesia. Implications for action. *Journal of Tropical Pediatrics* 35:129–132.
27. Taha T, Gray R, Abdelwahab M (1993) Determinants of neonatal mortality in central Sudan. *Annals of Tropical Paediatrics* 13(4):359–364.
28. Ashworth A, Feachem RG (1985) Interventions for the control of diarrhoeal diseases among young children: prevention of low birth weight. *Bulletin of the World Health Organization* 63:165–184.
29. Datta N, Kumar V, Kumar L, Singhi S (1987) Application of case management to the control of acute respiratory infections in low–birth–weight infants: a feasibility study. *Bulletin of the World Health Organization* 65:77–82.
30. Martorell R, Ramakrishnan U, Schroeder DG, Melgar P, Neufeld L (1998) Intrauterine growth retardation, body size, body composition and physical performance in adolescence. *European Journal of Clinical Nutrition* 52(Supplement 1):S43–S53.
31. Bakketeig LS (1998) Current growth standards, definitions, diagnosis and classification of fetal growth retardation. *European Journal of Clinical Nutrition* 52(Supplement 1):S1–S4.
32. Villar J, Smeriglio V, Martorell R, Brown C, Klein R (1984) Heterogeneous growth and mental development of intrauterine growth–retarded infants during the first 3 years of life. *Pediatrics* 74:783–791.
33. Albertsson–Wikland K, Karlberg J (1994) Natural growth in children born small for gestational age with and without catch–up growth. *Acta Paediatrica* 399(suppl):64–70.
34. Fitzhardinge PM, Inwood S (1989) Long–term growth in small–for–date children. *Acta Paediatrica Scandinavia* 349(suppl):27–33.
35. Hoffman H, Bakketeig L (1984) Heterogeneity of intrauterine growth retardation and recurrence risks. *Seminars in Perinatology* 8:15–24.
36. Hass J, Balcazar H, Caulfield L (1987) Variations in early neonatal mortality for different types of fetal growth retardation. *American Journal of Physical Anthropology* 76:467–473.
37. Goldenberg R, Hoffman H, Cliver S (1998) Neurodevelopmental outcome of small–for–gestational age infants. *European Journal of Clinical Nutrition* 52(S1):S54–S58.
38. Harvey D, Price J, Bunton J, Parkinson C, Campbell S (1982) Abilities of children who were small–for–gestational age babies. *Pediatrics* 69:296–300.
39. Hack M (1998) Effects of intrauterine growth retardation on mental performance and behavior, outcomes during adolescence and adulthood. *European Journal of Clinical Nutrition* 52(Supplement 1):S65–S71.
40. Grantham–McGregor SM (1998) Small for gestational age, term babies, in the first six years of life. *European Journal of Clinical Nutrition* 52(Supplement 1):S59–S64.
41. Leon DA (1998) Fetal growth and adult disease. *European Journal of Clinical Nutrition* 52(Supplement 1):S72–S82.

42. Forsdahl A (1977) Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart disease? *British Journal of Preventive Social Medicine* 31:91–95.
43. Barker DJP, Osmond C (1986) Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet* i:1077–1081.
44. Barker DJP, Winter PD, Osmond C, Margells B (1989) Weight in infancy and death from ischaemic heart disease. *Lancet* ii:577–580.
45. Hales CN, Barker DJP, Clark PMS, Cox LJ, Fall G, Osmond C et al. (1991) Fetal and infant growth and impaired glucose tolerance at age 64. *British Medical Journal* 303:1019–1022.
46. Curhan GC, Chertow GM, Willett WC, Spiegelman D, Colditz GA, Manson JE et al. (1996) Birth weight and adult hypertension and obesity in women. *Circulation* 94(6):1310–1315.
47. Curhan GC, Willett WC, Rimm EG, Spiegelman D, Ascherio AL, Stampfer MJ (1996) Birth weight and adult hypertension, diabetes mellitus, and obesity in US men. *Circulation* 94:3246–3250.
48. Rich-Edwards J, Stampfer M, Hanson J, Rosner B, Hawkinson SL, Colditz GA et al. (1997) Birthweight and risk of coronary heart disease in a cohort of women followed up since 1976. *British Medical Journal* 315:396–400.
49. Rich-Edwards JW, Colditz GA, Stampfer MJ, Willett WC, Gillman MW, Hennekens CH et al. (1999) Birthweight and the risk for type 2 diabetes mellitus in adult women. *Annals of Internal Medicine* 130(4 Pt 1):278–284.
50. Lithell HO, McKeigue PM, Berglund L, Moshen K, Lithell U, Leon DA (1996) Relationship of birthweight and ponderal index to non-insulin dependent diabetes and insulin response to glucose challenge in men aged 50–60 years. *British Medical Journal* 312:406–410.
51. Forsen T, Eriksson JG, Tuomilehto J, Teramo K, Osmond C, Barker DJP (1997) Mother's weight in pregnancy and coronary heart disease in a cohort of Finnish men: follow up study. *British Medical Journal* 315(7112):837–840.
52. Stein CE, Fall CHD, Kumaran K, Osmond C, Cox V, Barker DJP (1996) Fetal growth and coronary heart disease in South India. *Lancet* 348:1269–1273.
53. Mi J, Law C, Zhang KL, Osmond C, Stein C, Barker DJP (2000) Effects of infant birthweight and maternal body mass index in pregnancy on components of the insulin resistance syndrome in China. *Annals of Internal Medicine* 132(4):253–260.
54. Whincup PH, Cook DG, Adshhead F, Taylor SJ, Walker M, Papacosta O et al. (1997) Childhood size is more strongly related than size at birth to glucose and insulin levels in 10–11-year-old children. *Diabetologia* 40(3):319–326.
55. Hattersley AT, Tooke JE (1999) The fetal insulin hypothesis: an alternative explanation of the association of low birthweight with diabetes and vascular disease. *Lancet* 353(9166):1789–1792.
56. ACC/SCN (1999) Prevention of Foetal and Infant Malnutrition. Geneva: UN ACC/SCN Working Group.
57. Fuchs G (1999) Personal communication.
58. Jelliffe D (1968) *Infant nutrition in the subtropics and tropics*, 2nd ed., Monograph series. Geneva: World Health Organization.
59. Beaton G, Bengoa J (1976) *Nutrition in preventive medicine*, Geneva: World Health Organization.
60. Krasovec K, Anderson MA, eds. (1991) *Maternal nutrition and pregnancy outcomes: Anthropometric assessment*, Scientific Publication No. 529 Washington, DC: Pan American Health Organization.
61. Scrimshaw N, Schürch B (1996) Causes and consequences of intrauterine growth retardation in *Proceedings of an IDECG Workshop*. Baton Rouge, Louisiana, USA: International Dietary Energy Consultative

Group *European Journal of Clinical Nutrition* 52(S1).

62. Habicht J, Victora C, Vaughan J (1999) Evaluation designs for adequacy, plausibility and probability of public health performance and impact. *International Journal of Epidemiology* 28(1):10–18.
63. Ahluwalia I, Hogan V, Grummer–Strawn L, Colville W, Peterson A (1998) The effect of WIC participation on small–for–gestational–age births: Michigan–1992. *American Journal of Public Health* 88(9):1374–1377.
64. Avruch S, Cackley A (1995) Savings achieved by giving WIC benefits to women prenatally. *Public Health Reports* 110(1):27–34.
65. Abrams B (1993) Preventing low birthweight: does WIC work? A review of evaluations of the special supplemental food program for women, infants, and children. *Annals of the New York Academy of Sciences* 678(Mar 15):306–316.
66. Rush D (1986) *The national WIC evaluation: an evaluation of the special supplemental food program for women, infants, and children*, vol. I & II New York: Research Triangle Institute and New York State Research Foundation for Mental Hygiene.
67. Buescher P, Larson L, Nelson M, Jr, Lenihan A (1993) Prenatal WIC participation can reduce low birthweight and newborn medical costs: a cost–benefit analysis of WIC participation in North Carolina. *Journal of the American Dietetic Association* 93(2):163–166.
68. Devaney B, Bilheimer L, Schore J (1992) Medicaid costs and birth outcomes: the effects of prenatal WIC participation and the use of prenatal care. *Journal of Policy Analysis and Management* 11(4):573–592.
69. Rush D, Leighton J, Sloan N, Alvir J, Horvitz D, Seaver W et al. (1988) The National WIC Evaluation: evaluation of the Special Supplemental Food Program for Women, Infants, and Children: VI. Study of infants and children. *American Journal of Clinical Nutrition* 1988 (48):2S.
70. Primrose T, Higgins A (1971) A study in human antepartum nutrition. *The Journal of Reproductive Medicine* 7(6):257–264.
71. Higgins AC, Moxley JE, Pencharz PB, Mikolainis D, Buboiss S (1989) Impact of the Higgins nutrition intervention program on birthweight: a within–mother analysis. *Journal of the American Dietetic Association* 89(8):1097–1103.
72. Kielmann A, Taylor C, Parker R (1978) The Narangwal Nutrition Study: a summary review, *American Journal of Clinical Nutrition* 31:2040–2052.
73. Katz M (1978) Resume of the discussion on “The Narangwal Study”. *American Journal of Clinical Nutrition* 31:2053–2057.
74. CINI (2000) <<http://www.cini-india.org>>.
75. Shrimpton R (1999) Personal communication.
76. Dhaka University (1999) Bangladesh Integrated Nutrition Project, Ministry of Health and Family Welfare, Government of the People’s Republic of Bangladesh Mid–Term Evaluation 1998 – Final Report: 155.
77. Government/UNICEF/IDA (1999) Bangladesh Integrated Nutrition Project Mid–term Review Dhaka, Bangladesh: Government/UNICEF/IDA, 33.
78. Winichagoon P, Kachondham Y, Attig GA, Tontisirin K, eds. (1992) *Integrating Food and Nutrition into Development: Thailand’s Experiences and Future Visions*, Bangkok: Institute of Nutrition, Mahidol University and UNICEF East Asia and the Pacific Regional Office.
79. PAHO (1994) *Health Conditions in the Americas. Volume I*. Washington, DC: Pan American Health Organization.
80. Munoz C, Scrimshaw NS (1995) *The Nutrition and Health Transition of Democratic Costa Rica*, Boston, Massachusetts: International Foundation for Developing Countries.

81. Kramer M (1999) Balanced protein/energy supplementation in pregnancy (Cochrane Review) in *The Cochrane Library*. Oxford: Update Software.
82. Lechtig A, Yarbrough C, Delgado H, Habicht JP, Martorell R, Klein RE (1975) Influence of maternal nutrition on birthweight. *American Journal of Clinical Nutrition* 28(11):1223–1233.
83. Lechtig A, Habicht JP, Delgado H, Klein RE, Yarbrough C, Martorell R (1975) Effect of food supplementation during pregnancy on birthweight. *Pediatrics* 56(4):508–520.
84. Lechtig A, Yarbrough C, Delgado H, Martorell R, Klein RE, Behar M (1975) Effect of moderate maternal malnutrition on the placenta. *American Journal of Obstetrics and Gynecology* 123(2):191–201.
85. Mardones–Santander F, Rosso P, Stekel A, Ahumada E, Llaguno S, Pizarro F et al. (1988) Effect of a milk–based food supplement on maternal nutritional status and fetal growth in underweight Chilean women. *American Journal of Clinical Nutrition* 47:413–419.
86. Kardjati S, Kusin JA, de With C (1988) Energy supplementation in the last trimester of pregnancy in East Java: I. Effect on birthweight. *British Journal of Obstetrics and Gynaecology* 95:783–794.
87. Ceesay et al. (1997) Effect on stillbirths and perinatal mortality: West Kiang Trial only. *British Medical Journal* 315:786–790.
88. Ceesay SM, Prentice AM, Cole TJ, Foord F, Weaver LT, Poskitt EM et al. (1997) Effects on birth weight and perinatal mortality of maternal dietary supplements in rural Gambia: 5 year randomised controlled trial. *British Medical Journal* 315(7111):786–790.
89. Prentice AM, Whitehead R, Watkinson M, Lamb W, Cole T (1983) Prenatal dietary supplementation of African women and birthweight. *Lancet* 1:489–492.
90. Prentice AM, Cole T, Foord F, Lamb W, Whitehead R (1987) Increased birthweight after prenatal dietary supplementation of rural African women. *American Journal of Clinical Nutrition* 46:912–925.
91. Moore S, Cole T, Poskitt E, Sonko B, Whitehead R, McGregor I et al. (1997) Season of birth predicts mortality in rural Gambia. *Nature* 388(31 July):434.
92. Winick M (1971) Cellular growth during early malnutrition. *Pediatrics* 47(6):969–978.
93. Susser M, Stein Z (1994) Timing in prenatal nutrition: a reprise of the Dutch Famine Study. *Nutrition Reviews* 52(3):84–94.
94. Susser M (1991) Maternal weight gain, infant birthweight and diet: causal sequences. *American Journal of Clinical Nutrition* 53:1384–1396.
95. Allen L, Lung'aho MS, Shaheen M et al. (1994) BMI and pregnancy outcomes in the Nutrition CRSP. *European Journal of Clinical Nutrition* 48(Suppl 3):S68–S77.
96. Kusin JA, Kardjati S, Renqvist UH (1994) Maternal body mass index: the functional significance during reproduction. *European Journal of Clinical Nutrition* 48(Suppl. 3):S56–S67.
97. Winkvist A (1992) Maternal depletion among Pakistani and Guatemalan women. Dissertation, Cornell University, Ithaca NY.
98. Prentice AM, Goldberg GR, Prentice A (1994) Body mass index and lactation performance. *European Journal of Clinical Nutrition* 48(Suppl 3):S78–86.
99. Barbosa L, Butte NF, Villalpando S, Wong WW, Smith EO (1997) Maternal energy balance and lactation performance of Mesoamerindians as a function of body mass index. *American Journal of Clinical Nutrition* 66(3):575–583.
100. Villar J, Rivera J (1988) Nutritional supplementation during two consecutive pregnancies and the interim lactation period: Effect on birthweight. *Pediatrics* 81:51–57.

101. Jaya Rao KA, Shatrugna V (1976) Effect of vitamin A supplementation on plasma progesterone levels in pregnancy. *Indian Journal of Medical Research* 64:1261–1266.
102. Panth M, Raman L, Ravinder P, Sivakumar B (1991) Effect of vitamin A supplementation on plasma progesterone and estradiol levels during pregnancy. *International Journal of Vitamins and Nutrition* 61:17–19.
103. Howell DW, Haste F, Rosenburg D, Brown IRF, Brooke OG (1986) Investigation of vitamin A nutrition in pregnant British Asians and their infants. *Human Nutrition. Clinical Nutrition* 40(C):43–50.
104. Fawzi WW, Msamanga GI, Spiegelman D, Urassa EJ, McGrath N, Mwakagile D et al. (1998) Randomised trial of effects of vitamin supplements on pregnancy outcomes and T cell counts in HIV-1-infected women in Tanzania. *Lancet* 351(9114): 1477–1482.
105. West KPJ, Katz J, Khattry SK, LeClerq SC, Pradhan EK, Shrestha SR et al. (1999) Double blind, cluster randomised trial of low dose supplementation with vitamin A or beta carotene on mortality related to pregnancy in Nepal. The NNIPS-2 Study Group. *British Medical Journal* 318(7183):570–575.
106. Scholl TO, Hediger ML, Schall JI, Khoo CS, Fischer RL (1996) Dietary and serum folate: their influence on the outcome of pregnancy. *American Journal of Clinical Nutrition* 63(4):529–525.
107. Iyengar L, Rajalakshmi K (1975) Effect of folic acid supplement on birth weights of infants. *American Journal of Obstetrics and Gynecology* 122(3):332–336.
108. Blot I, Papiernik E, Kaltwasser JP, Werner E, Tchernia G (1981) Influence of routine administration of folic acid and iron during pregnancy. *Gynecologic and Obstetric Investigation* 12(6):294–304.
109. Rolschau J, Date J, Kristoffersen K (1979) Folic acid supplement and intrauterine growth. *Acta Obstetricia et Gynecologica Scandinavica* 58(4):343–346.
110. Giles PF, Harcourt AG, Whiteside MG (1971) The effect of prescribing folic acid during pregnancy on birth-weight and duration of pregnancy. A double-blind trial. *Medical Journal of Australia* 2(1):17–21.
111. Kirke PN, Daly LE, Elwood JH (1992) A randomised trial of low dose folic acid to prevent neural tube defects. The Irish Vitamin Study Group. *Archive of Disease in Childhood* 67(12):1442–1446.
112. Czeizel AE (1993) Controlled studies of multivitamin supplementation on pregnancy outcomes. *Annals of the New York Academy of Sciences* 678:266–275.
113. Scholl TO, Reilly T (2000) Anemia, Iron and Pregnancy Outcome. *Journal of Nutrition* 130:443S–447S.
114. Scholl TO, Hediger ML, Fischer RL, Shearer JW (1992) Anemia vs iron deficiency: increased risk of preterm delivery in a prospective study. *American Journal of Clinical Nutrition* 55(5):985–988.
115. Hemminki E, Rimpela U (1991) A randomized comparison of routine versus selective iron supplementation during pregnancy. *Journal of the American College of Nutrition* 10(1):3–10.
116. Milman N, Agger AO, Nielsen OJ (1994) Iron status markers and serum erythropoietin in 120 mothers and newborn infants. Effect of iron supplementation in normal pregnancy. *Acta Obstetricia et Gynecologica Scandinavica* 73(3):200–204.
117. Menendez C, Todd J, Alonso PL, Francis N, Lulat S, Ceesay S et al. (1994) The effects of iron supplementation during pregnancy, given by traditional birth attendants, on the prevalence of anaemia and malaria. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 88(5):590–593.
118. Preziosi P, Prual A, Galan P, Daouda H, Boureima H, Hercberg S (1997) Effect of iron supplementation on the iron status of pregnant women: consequences for newborns. *American Journal of Clinical Nutrition* 66(5):1178–1182.
119. Menendez C, Todd J, Alonso PL, Francis N, Lulat S, Ceesay S et al. (1995) The response to iron supplementation of pregnant women with haemoglobin genotype AA or AS. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 89(3):289–292.

120. Gutteridge JMC, Halliwell B (1994) *Antioxidants in Nutrition, Health and Disease*, New York, NY: Oxford University Press.
121. Black RE (1998) Therapeutic and preventive effects of zinc on serious childhood infectious diseases in developing countries. *American Journal of Clinical Nutrition* 68(2S):476S–479S.
122. Bhutta ZA, Black RE, Brown KH, Gardner JM, Gore S, Hidayat A et al. (1999) Prevention of diarrhea and pneumonia by zinc supplementation in children in developing countries: pooled analysis of randomized controlled trials. *Journal of Pediatrics* 135(6):689–697.
123. Sazawal S, Black RE, Jalla S, Mazumdar S, Sinha A, Bhan MK (1998) Zinc supplementation reduces the incidence of acute lower respiratory infections in infants and preschool children: a double-blind, controlled trial. *Pediatrics* 102(1):1–5.
124. Roy SK, Tomkins AM, Haider R, Behren RH, Akramuzzaman SM, Mahalanabis D et al. (1999) Impact of zinc supplementation on subsequent growth and morbidity in Bangladeshi children with acute diarrhoea. *European Journal of Clinical Nutrition* 53(7):539–534.
125. Osendarp SJM, van Raaij JMA, Arifeen SE, Wahed MA, Baqui AH, Fuchs GJ (2000) A randomized, placebo-controlled trial on the effect of zinc supplementation during pregnancy on pregnancy outcome in Bangladeshi urban poor. *American Journal of Clinical Nutrition* 71(1):114–119.
126. Garg HK, Singhal KC, Arshad Z (1993) A study of the effect of oral zinc supplementation during pregnancy on pregnancy outcome. *Indian Journal of Physiology and Pharmacology* 37(4):276–284.
127. Simmer K, Lort-Phillips L, James C, Thompson RPH (1991) A double-blind trial of zinc supplementation in pregnancy. *European Journal of Clinical Nutrition* 45:139–144.
128. Cherry FF, Sandstead HH, Rojas P, Johnson LK, Batson HK, Wang XB (1989) Adolescent pregnancy: associations among body weight, zinc nutriture, and pregnancy outcome. *American Journal of Clinical Nutrition* 50:945–954.
129. Goldenberg RL, Tamura T, Neggers Y, Copper RL, Johnston KE, DuBard MB et al. (1995) The effect of zinc supplementation on pregnancy outcome. *Journal of the American Medical Association* 274(6):463–468.
130. Jonsson B, Hauge B, Larsen MF, Hald F (1996) Zinc supplementation during pregnancy: a double blind randomised controlled trial. *Acta Obstetrica et Gynecologica Scandinavica* 75(8):725–729.
131. Penny ME, Peerson JM, Marin RM, Duran A, Lanata CF, Lonnerdal B et al. (1999) Randomized, community-based trial of the effect of zinc supplementation, with and without other micronutrients, on the duration of persistent childhood diarrhea in Lima, Peru. *Journal of Pediatrics* 135(2):208–217.
132. Black RE (1999) Personal communication.
133. Delange F (1996) Administration of iodized oil during pregnancy: a summary of the published evidence. *Bulletin of the World Health Organization* 74(1):101–108.
134. Pretell E, Caceres A (1994) Impairment of mental development by iodine deficiency and its correction: a retrospective view of studies in Peru, in *The Damaged Brain of Iodine Deficiency*, ed. Stanbury JB. New York: Cognizant Communication, 287–292.
135. Chaouki ML, Benmiloud M (1994) Prevention of iodine deficiency disorders by oral administration of lipiodol during pregnancy. *European Journal of Endocrinology* 130(6):547–551.
136. Thilly C et al. (1994) Maternal, fetal and juvenile hypothyroidism, birthweight, and infant mortality in the etiopathogenesis of the IDD spectrum in Zaire and Malawi, in *The Damaged Brain of Iodine Deficiency*, ed. Stanbury JB. New York: Cognizant Communication, 241–250.
137. Vanderpass J, Thilly CH (1994) Endemic neonatal, infantile and juvenile hypothyroidism in Ubangi, Northern Zaire: clinical consequences and prevention, in *The Damaged Brain of Iodine Deficiency*, ed. Stanbury JB. New York: Cognizant Communication, 209–224.

138. Ramakrishnan U (1999) Personal communication.
139. Purwar M, Kulkarni H, Motghare V, Dhole S (1996) Calcium supplementation and prevention of pregnancy induced hypertension. *Journal of Obstetrics and Gynaecology Research* 22(5):425–430.
140. Conradt A, Weidinger H, Algayer H (1985) Magnesium therapy decreased the rate of intrauterine fetal retardation, premature rupture of membranes and premature delivery in risk pregnancies treated with betamimetics. *Magnesium* 4(1):20–28.
141. Kovacs L, Molnar BG, Huhn E, Bodis L (1988) Magnesium substitution in pregnancy. A prospective, randomized double-blind study. *Geburtshilfe Frauenheilkd* 48(8):595–600.
142. Spatling L, Spatling G (1988) Magnesium supplementation in pregnancy. A double-blind study. *British Journal of Obstetrics and Gynaecology* 95(2):120–125.
143. Kullander S, Kallen B (1976) A prospective study of drugs and pregnancy. *Acta Obstetrica et Gynecologica Scandinavica* 55:287.
144. Scholl TO, Hediger ML, Bendich A, Schall JI, Smith WK, Krueger PM (1997) Use of multivitamin/mineral prenatal supplements: influence on the outcome of pregnancy. *American Journal of Epidemiology* 146(2):134–141.
145. Haste FM, Brooke OG, Anderson HR, Bland JM (1991) The effect of nutritional intake on outcome of pregnancy in smokers and non-smokers. *British Journal of Nutrition* 65:347–354.
146. Johnson AA, Knight Em, Edwards CH, Oyemade UJ, Cole OJ, Westney OE et al. (1994) Dietary intakes, anthropometric measurements and pregnancy outcomes. *Journal of Nutrition* 124(6S):936S–942S.
147. Weigel MM, Narvaez WM, Lopez A, Feliz C, Lopez P (1991) Prenatal diet, nutrient intake and pregnancy outcome in urban Ecuadorian primiparas. *Archives of Latin American Nutrition* XLI(1):21–37.
148. Pfeffer F, Valdes-Ramos R, Avila-Rosas H, Meza C, Casanueva E (1996) Iron, zinc and vitamin C nutritional status is not related to weight gain in pregnant women. *Nutrition Research* 16:555–564.
149. Kinare A, Natekar A, Chinchwadkar M, Yajnik C, Coyaji K, Howe D (2000) Low midpregnancy placental volume in rural Indian women cause for low birthweight? *American Journal of Obstetrics and Gynecology* 182(2):443–448.
150. Fall C, Stein C, Kumaran K, Cox V, Osmond C, Barker D et al. (1998) Size at birth, maternal weight, and type 2 diabetes in South India. *Diabetic Medicine* 15(3):220–227.
151. Fall CHD, Yajnik CS, Rao S, Coyaju KJ, Shier RP (1999) The effects of maternal body composition before pregnancy on fetal growth; the Pune Maternal Nutrition and Fetal Growth Study in *Fetal Programming: influences on development and disease in later life*, eds. O'Brien PMS, Wheeler T, Barker DJP. London: RCOG Press.
152. Prentice AM et al. (1993) The counter viewpoint. *Lancet* 341:55–56.
153. Andersson R, Bergstrom S (1997) Maternal nutrition and socio-economic status as determinants of birthweight in chronically malnourished African women. *Tropical Medicine and International Health* 2(11):1080–1087.
154. Merchant K, Martorell R, Haas J (1990) Maternal and fetal responses to the stresses of lactation concurrent with pregnancy and of short recuperative intervals. *American Journal of Clinical Nutrition* 52(2):280–288.
155. Merchant K, Martorell R, Haas JD (1990) Consequences for maternal nutrition of reproductive stress across consecutive pregnancies. *American Journal of Clinical Nutrition* 52(4):616–620.
156. Islam MA, Rahman MM, Mahalanabis D (1994) Maternal and socioeconomic factors and the risk of severe malnutrition in a child: a case-control study. *European Journal of Clinical Nutrition* 48(6):416–424.

157. Rahman M, Roy SK, Ali M, Mitra AK, Alam AN, Akbar MS (1993) Maternal nutritional status as a determinant of child health. *Journal of Tropical Pediatrics* 39(2):86–88.
158. Lucas A, Fewtrell MS, Davies PS, Bishop NJ, dough H, Cole TJ (1997) Breastfeeding and catch-up growth in infants born small for gestational age. *Acta Paediatrica* 86(6):564–569.
159. Lawless JW, Latham MC, Stephenson LS, Kinoti SN, Pertet AM (1994) Iron supplementation improves appetite and growth in anemic Kenyan primary school children. *Journal of Nutrition* 124(5):645–654.
160. Tanner JM, Whitehouse RH, Marubini E, Resele LF (1976) The adolescent growth spurt of boys and girls of the Harpenden Growth Study. *Annals of Human Biology* 3(2):109–126.
161. Kurz K (1994) Improving the nutritional status of adolescent girls: lessons drawn from the ICRW multi-country projects. Anand, India: UNICEF Inter-regional Consultation on the Girl Child.
162. SEWA-Rural Research Team (1994) Beliefs and behaviour regarding diet in pregnancy in a rural area of Gujarat, western India, in *Listening to Women Talk About Their Health: Issues and Evidence from India*, ed. Gittelsohn J et al. New Delhi: Har-Anand Publications (Ford Foundation).
163. Dewey KG, Cohen RJ, Brown KH, Landa Rivera L (1999) Age of introduction of complementary foods and growth of term, low birthweight, breastfed infants: a randomized intervention study in Honduras. *American Journal of Clinical Nutrition* 69:679–686.
164. McCormick MC (1985) The contribution of low birthweight to infant mortality and childhood morbidity. *New England Journal of Medicine* 312:82–90.
165. WHO (1993) World Health Organization Collaborative Study of Birth Weight Surrogates. Use of a simple anthropometric measurement to predict birth weight. *Bulletin of the World Health Organization* 71:157–163.
166. Dusitsin N, Chompootaweep S, Poomsuwan P, Dusitsin K, Sentrakul P, Lumbiganond P (1991) Development and validation of a simple device to estimate birthweight and screen for low birthweight in developing countries. *American Journal of Public Health* 81:1201–1205.
167. Diamond ID, el-Aleem AM, Ali MY, Mostafa SA, el-Nashar SM, Guidotti RJ (1991) The relationship between birthweight, and arm and chest circumference in Egypt. *Journal of Tropical Pediatrics* 37:323–326.
168. Bhargava SK, Ramji S, Lumar A, Mohan M, Marwah J, Sachdev HP (1985) Mid-arm and chest circumference at birth as predictors of low birthweight and neonatal mortality in the community. *British Medical Journal* 291:1617–1619.
169. Raymond EG, Tafari N, Troendle JF, Clemens J (1994) Development of a practical screening tool to identify preterm, low birthweight neonates in Ethiopia. *Lancet* 344:524–527.
170. Bhargava SK, Sachdev HP, Iyer PU, Ramji S (1985) Current status of infant growth measurements in the perinatal period in India. *Acta Paediatrica Scandinavia* 319(S):103–110.
171. Barros FC (1999) Measuring size at birth: alternatives and implications for monitoring and evaluation. *in press*.
172. Cesar JA, Victora CG, Barros FC, Santos IS, Flores JA (1999) Impact of breastfeeding on admission for pneumonia during postneonatal period in Brazil: nested case-control study. *British Medical Journal* 318:1316–1320.
173. McNeilly AS, Glasier A, Howie PW (1985) Endocrine control of lactational infertility, in *Maternal nutrition and lactational infertility*, ed. Dobbing J. New York: Raven Press, 1–24.
174. Kambarami RA, Chidede O, Kowo DT (1998) Kangaroo care versus incubator care in the management of well preterm infants – a pilot study. *Annals of Tropical Paediatrics* 18(2):81–86.
175. Bergman JJ, Jurisoo LA (1994) The 'kangaroo method' for treating low birthweight babies in a developing country. *Tropical Doctor* 24(2):57–60.

176. WHO (1997) *Hypoglycaemia of the newborn. A review of the literature*, WHO/CHD/97.1 Geneva: World Health Organization.
177. WHO (1989) *Protecting, promoting and supporting breastfeeding*. Geneva: World Health Organization.
178. Green LC, Lucas A, Livingstone MBE, Harland PA, Baker BA (1995) Relationship between early diet and subsequent cognitive performance during adolescence. *Biochemistry and Social Transaction* 23:376S.
179. Lucas A, Morley R, Cole TJ, Lister G, Lesson-Payne C (1992) Breast milk and subsequent intelligence quotient in children born preterm. *Lancet* 339:261–264.
180. von Kries R, Koletzko B, Sauerwald T, von Mutius E, Barnert D, Grunert V et al. (1999) Breastfeeding and obesity: a cross sectional study. *British Medical Journal* 319:147–150.
181. Koletzo A, Sherman P, Corey M, Griffiths A, Smith C (1989) role of infant feeding practices in development of Crohn's disease in childhood. *British Medical Journal* 298:1617–1618.
182. Saarinen UM, Kajosarri M (1995) Breastfeeding as prophylaxis against atopic disease: prospective follow-up study until 17 years old. *Lancet* 346:1065–1069.
183. ACC/SCN (1999) UNHCHR – Committee on Economic, Social and Cultural Rights: General Comment 12. *SCN News* 18:41–45.
184. Grantham-McGregor SM, Walker SP, Chang S (2000) Nutritional deficiencies and later behavioural development. *Proceedings of the Nutrition Society* 59(1):47–54.
185. Grantham-McGregor SM, Powell CA, Walker SP, Himes JH (1991) Nutritional supplementation, psychosocial stimulation, and mental development of stunted children: The Jamaica study. *Lancet* 338:1–5.
186. Grantham-McGregor SM, Fernald LC (1997) Stunting and Mental Development. A background paper provided to the Commission on the Nutrition Challenges of the 21st Century.
187. Commission on the Nutrition Challenges of the 21st Century (2000) *Ending Malnutrition by 2020: An Agenda for Change in the Millenium*, Final Report submitted to the ACC/SCN.
188. UNICEF/WHO (1999) *Report of the UNICEF/WHO Consultation: Prevention and Control of Iron Deficiency Anaemia in Women and Children 3–5 February 1999*, Canada: UNICEF.
189. Shaheen R, Arifeen SE, de Francisco A (2000) The optimal duration of nutritional supplementation for malnourished pregnant women. In: *Findings of studies performed under the BINP Operations Research Project (ORP)*. Vol 2. ed. Osendarp SMJ, Roy SK, Fuchs GJ. Dhaka: ICDDR,B, 8–9.
190. Walsh CT, Sandstead HH, Prasad AS, Newberne PM, Fraker PJ (1994) Zinc: health effects and research priorities for the 1990s. *Environmental Health Perspectives* 102(S12):5–46.
191. Swanson CA, King JC (1987) Zinc and pregnancy outcome. *American Journal of Clinical Nutrition* 46:763–771.
192. School TO, Hediger ML, Schall JI, Fischer RL, Khoo C (1993) Low zinc intake during pregnancy: its association with preterm and very preterm delivery. *American Journal of Epidemiology* 137:1115–1124.
193. Kirksey A, Wachs TD, Yunis F, et al. (1994) Relation of maternal zinc nutriture to pregnancy outcome and infant development in an Egyptian village. *American Journal of Clinical Nutrition* 60:782–792.
194. Caulfield LE, Zavaleta N, Shankar AH, Meriardi M (1998) Potential contribution of maternal zinc supplementation during pregnancy to maternal and child survival. *American Journal of Clinical Nutrition* 68(S):449S–508S.
195. Caulfield LE, Zavaleta N, Figueroa A, Leon Z (1999) Maternal zinc supplementation does not affect size at birth and pregnancy duration in Peru. *Journal of Nutrition* 129:1563–1568.

196. Caulfield LE, Zavaleta N, Figueroa A (1999) Adding zinc to prenatal iron and folate supplements improves maternal and neonatal zinc status in a Peruvian population. *American Journal of Clinical Nutrition* 69:1257–1263.

197. Osendarp SJM, van Raaij JMA, Darmstadt GL, Fuchs GJ (2000) Maternal zinc supplementation in Bangladeshi urban poor improved immunity and reduced morbidity in LBW infants. *Faseb Journal* 14(4):A560–406.2.

198. Shankar A, Gbakima A, Caulfield LE, Zavaleta N (1998) The influence of maternal zinc supplementation on immunological development of the neonate and perinatal morbidity. *Faseb Journal* 12(5):A818–4741.

Appendix: Symposium Agenda

INTERNATIONAL LOW BIRTH WEIGHT SYMPOSIUM AND WORKSHOP

**ICDDR,B: Centre for Health and Population Research
Mohakhali, Dhaka 1212, Bangladesh
14–17 June 1999**

14–15 June 1999

Welcome Address and Introduction to Symposium and Workshop: Prof. George Fuchs, Interim Director, ICDDR,B; Address by the Chief Guest: Mr. Salah Uddin Yusuf, Honourable Minister for Health and Family Welfare, Government of the People's Republic of Bangladesh; Address by Special Guests: Abu Hafiz, BINP Director; Milla McLachlan, World Bank, Washington; Roger Shrimpton, UNICEF, New York.

Session 1: Epidemiology, Causes, and Consequences of Low Birth Weight

Levels, patterns, and determinants of low birth weight in developing countries – Sultana Khanum

Mortality and morbidity consequences of LBW – Robert Black

Growth of LBW children – Fernando Barros

The Barker Hypothesis – Caroline Fall

The Bangladesh experience: epidemiology and consequences of LBW – Shams El Arifeen

Session 2: Interventions to Reduce Low Birth Weight: Global Experiences

LBW: Prevention and after-care in routine programmes – Jane Kusin

Effect of dietary supplements before and during pregnancy on birth weight – Andrew Prentice

The role of micronutrients in improving birth weight – Usha Ramakrishnan

Innovative global experiences on LBW: a review – Roger Shrimpton

Session 3: Interventions to Reduce Low Birth Weight: Critical issues (I)

Woman's body composition and implications for preventing LBW – Caroline Fall

Measurement of pregnancy weight gain and usefulness for targeting – Kathy Krasovec

Measuring size at birth: alternatives to and implications for monitoring and evaluation

– Fernando Barros

Session 4: Interventions to Reduce Low Birth Weight: Critical issues (II)

The critical moment for intervention: childhood, adolescence, pre–pregnancy, pregnancy – Rae Galloway

Does increased food intake during pregnancy result in increased obstetric complications? – Andrew Prentice

Improving outcome of LBW babies: interventions – George Fuchs

Cultural beliefs and social practices relating to maternal food intake and birth weight – Gretel Pelto

Session 5: Low Birth Weight Interventions in Bangladesh

Concluding Session: Research and programme priorities

Presentation by Rapporteurs: S. K. Roy, Chris Duggan, Shameem Ahmed, Rukhsana Haider

16–17 June 1999

Workshop: ICDDR,B/Matlab (Rapporteurs: Shams El Arifeen and Iqbal Kabir)

Field trip: BINP site in Shaharasti Thana

Session 1: BINP Policies and Activities on Reducing LBW: Review of Current Situation

Session 2: BINP Policies and Activities: Identification of Strategies for Reducing LBW

Session 3: BINP Policies and Activities Relating to LBW: Future Research Needs

Session 4: BINP Policies and Activities Relating to LBW: An Action Plan for Change

List of Participants

Shameem Ahmned
ICDDR,B
Dhaka 1212, Bangladesh
shameem@icddrb.org

Fernando Barros
Latin American Perinatology
WHO–PAHO
Casilla de Correo 627
11000 Montevideo, Uruguay
barrosfe@clap.ops–oms.org

Robert Black
Johns Hopkins SPH
615 North Wolfe Street
Baltimore, MD USA 21205
rblack@jhsph.edu

Zulfiqar Bhutta
Aga Khan University
Stadium Road
P O Box 3500

Karachi 74800, Pakistan
zulfiqar.bhutta@aku.edu

PO Bloomquist
UNICEF Nepal
Pob@mos.com.np

Karen Codling
UNICEF–EAPRO
PO Box 2–154
Bangkok 10200 Thailand
kcodling@unicef.org

Victor Cole
UNICEF Jakarta
PO Box 8318/JKSMP
Jakarta 1203 Indonesia
vcole@unicef.org

Chris Duggan
Harvard University/ARCH
Children's Hospital
300 Longwood Avenue
Boston, Mass USA 02115
duggan_c@al.tch.harvard.edu

Shams El Arifeen
ICDDR,B
Dhaka 1212, Bangladesh
shams@icddr.org

Caroline Fall
Environmental Epidemiology
Southampton General Hospital
Southampton, England
chdf@mrc.soton.ac.uk

George Fuchs
ICDDR,B
Dhaka 1212, Bangladesh
gfuchs@icddr.org

Rae Galloway
World Bank
1818 H Street, NW
Washington, DC USA 20433
rgalloway@worldbank.org

B Ganguli
Child in Need Institute (CINI)
P O Box 16742
Calcutta 700 027, India

Ellen Girerd–Barclay
UNICEF–ROSA Nepal
Kathmandu, Nepal
Egirerdbarclay@unicef.org

M Abu Hafiz
BINP, Dhaka
binp@bangla.net

Rukhsana Haider
ICDDR,B
Dhaka 1212, Bangladesh
rhaider@icddrb.org

Kamal Islam
UNICEF/New Delhi

AFM Iqbal Kabir
World Bank/Dhaka
akabir@worldbank.org

Iqbal Kabir
ICDDR,B
Dhaka 1212, Bangladesh
ikabir@icddrb.org

Manzur-ul-Karim
BINP, Dhaka
binp@bangla.net

Laura Kelly
Child Health Research Project
c/o Johns Hopkins University
Dept. International Health
lkelly@jhsph.edu

Sultana Khanum
WHO – I.P. Estate, Ring Road
New Delhi–110002, India
sultana@who.ernet.in

Kathy Krasovec
Abt Associates
Hampden Square, Ste 600
4800 Montgomery Lane
Bethesda, MD USA 20814–5341
Kathy_Krasovec@abtassoc.com

Jane Kusin
Royal Tropical Institute
Mauritskade 63
P O Box 95001
1090 HA Amsterdam
The Netherlands
jakusin@hetnet.nl

Thaneoke Kyaw–Myint
UNICEF/Dhaka
tkyaw–myint@unicef.org

Benjamin Loevisohn
Asian Development Bank
P O Box 789
Manila, The Philippines
bloevinsohn@mail.AsianDevBank.org

Zeba Mahmud
BRAC, Centre
75 Mohakhali
Dhaka–1212, Bangladesh

DS Manandhar
Director, MIRA
GPO Box 921
Kathmandu, Nepal
Davido@wlink.com.np

Milla Mclachlan
World Bank
1818 H Street, NW
Washington, DC USA 20433
mmclachlan@worldbank.org

David Osrin
MIRA
GPO Box 921
Kathmandu, Nepal
Davido@wlink.com.np

Dr. Pappu
Child in Need Institute (CINI)
P O Box 16742
Calcutta 700 027, India
cini@cal.vsnl.net.in

Gretal Pelto
Cornell University
Ithaca, NY USA 14853-6301
gp32@cornell.edu

Judith Pojda
UN ACC/SCN
c/o WHO, 20 Avenue Appia
CH-1211 Geneva 27
Switzerland
pojdamorrison@hotmail.com

Andrew Prentice
London School of Hygiene and Tropical Medicine
MRC 49-51 Bedford Square
London WC1B 3DP England
andrew.prentice@lshtm.ac.uk

Joop van Raaij
Human Nutrition & Epidemiology
Wageningen University
P O Box 8129
6700 EV Wageningen
The Netherlands
joop.vanraaij@staff.nutepi.wau.nl

Usha Ramakrishnan
Rollins School of Public Health
Emory University
Dept of International Health
1518 Clifton Road, NE
Atlanta, GA USA 30322
uramakr@sph.emory.edu

SK Roy
ICDDR,B
Dhaka 1212, Bangladesh
skroy@icddr.org

Yuki Shiroishi
UNICEF–BSL Office Complex 1
Minto Rd, Dhaka–1000
GPO Box 58, Bangladesh
yuki@mail.unicef.bangla.net

Roger Shrimpton
UNICEF/NY
3 UN Plaza TA24A
New York, NY 10017
rshrimpton@unicef.org

Eric Simoes
The Children's Hospital
Dept of Pediatrics
1056 E 19th Ave. B070
Denver, CO USA 80218–1088
eric.simoes@uchsc.edu

Patricia Stephenson
USAID/G/PHN/HN/CS
Ronald Reagan Building 3rd fl
Washington DC USA 20523–3700
mstanton@usaid.gov

Publications – July 2000

<http://acc.unsystem.org/scn/>

 *available for download from our website*

REPORTS ON THE WORLD NUTRITION SITUATION

Fourth Report on the World Nutrition Situation: Nutrition Throughout the Life Cycle, January 2000 

Third Report on the World Nutrition Situation, December 1997

Update on the Nutrition Situation, 1996: Summary of Results for the Third Report on the World Nutrition Situation, late 1996 (*out of stock – but photocopy available*)

Update on the Nutrition Situation, November 1994

Second Report on the World Nutrition Situation, Volume II, Country Data, March 1993

Second Report on the World Nutrition Situation, Volume I, Global and Regional Results, October 1992

Supplement on Methods and Statistics to the First Report on the World Nutrition Situation, December 1988 (*out of stock – but photocopy available*)

First Report on the World Nutrition Situation, November 1987 (*out of stock – but photocopy available*)

NUTRITION POLICY DISCUSSION PAPERS

Challenges for the 21st Century: A Gender Perspective on Nutrition Through the Life Cycle by Philip James, Suttalak Smitisiri, Per Pinstrup–Anderson, Rajul Pandya–Lorch, Christopher Murray, Alan Lopez & Isatou Semega–Jenneh. April 1998 (*NPP No. 17*)

Nutrition and Poverty by S. Gillespie, N. Hasan, S. Osmani, U. Jonsson, R. Islam, D. Chirmulay, V. Vyas & R. Gross. November 1997 (*NPP No. 16*)

How Nutrition Improves Report based on ACC/SCN Workshop held on 25–27 September 1993 at the 15th IUNS International Congress on Nutrition, Adelaide, Australia by S. Gillespie, J. Mason, R. Martorell. (SOA No. 15)

Controlling Vitamin A Deficiency Report based on ACC/SCN Consultative Group Meeting held in Ottawa July 1993. Prepared by Gillespie and Mason, January 1994. (SOA No. 14)

Effectiveness of Vitamin A Supplementation in the Control of Young Child Morbidity and Mortality in Developing Countries by G.H. Beaton, R. Martorell, K.J. Aronson, B. Edmonston, G. McCabe, A.C. Ross, B. Harvey. December 1993. (SOA No. 13)

Nutritional Issues in Food Aid Report of symposium held at the 19th Session of the ACC/SCN in Rome, February 1992. Includes papers on the support of public works by food aid as a nutrition intervention, which age groups should be targeted for supplementary feeding, effects of supplementary feeding in the growth of children with infection, experiences of feeding programmes, and protecting refugees' nutrition with food aid. August 1993. (SOA No. 12)

Nutrition and Population Links – Breastfeeding, Family Planning and Child Health Papers from the ACC/SCN 18th Session Symposium, held at UNFPA, New York, February 1991. Papers include “Nutrition and Family Planning Linkages: What More Can be Done?” by Sandra Huffman, “Reproductive Stress and Women’s Nutrition” by Reynaldo Martorell and Kathleen Merchant, “Breastfeeding, Fertility and Population Growth” by Roger Short, “Nutrition and its Influence on the Mother–Child Dyad” by Prema Ramachandran, and with final comments by Miriam Labbok, Barry Edmonston, and Beverly Winikoff. (SOA No. 11)

Nutrition–Relevant Actions – Some Experiences from the Eighties and Lessons for the Nineties Book developed from the original background paper for the ACC/SCN *ad hoc* group meeting held in London in November 1990. Proposes a framework for the analysis of policies and programmes affecting nutrition, before reviewing experiences during the 1980s in several countries, and moving on to consider options for improving nutrition in the 1990s. Complements and expands on Supplement to SCN News No. 7. Prepared by Stuart Gillespie and John Mason, October 1991. (SOA No. 10)

Controlling Iron Deficiency Report of ACC/SCN workshop held in Trinity College, Dublin, June 1990. Focuses on iron supplementation and practical means of improving large–scale programmes. Also introduces fortification and diet change. Gives information from six large–scale programmes. Prepared and edited by Gillespie, John Kevany, and John Mason, February 1991. (SOA No. 9)

Managing Successful Nutrition Programmes Report of ACC/SCN workshop held at IUNS meeting in Korea, August 1989. Includes reports on 16 large–scale nutrition programmes, and summary of discussions on targeting, staff issues, community participation, management information systems, sustainability and replicability. Edited by Joan Jennings, Stuart Gillespie, John Mason, Mahshid Lotfi and Tom Scialfa, October 1990. (SOA No. 8)

Appropriate Uses of Child Anthropometry Report based on workshop held by ACC/SCN, June 1989. Basic concepts, uses for screening, growth monitoring, population assessment, and surveillance. Prepared and edited by G. Beaton, A. Kelly, J. Kevany, R. Martorell, and J. Mason, December 1990. (SOA No. 7)

Women and Nutrition Background, and papers presented at SCN Symposium, held at UNICEF, New York, February 1989. Papers include “Beating the Zero Sum Game” by McGuire and Popkin, “Reflections from India and Pakistan” by Chatterjee and Lambert, “Grameen Bank Experience” by Quanine, “Improving the Nutrition of Women in Tanzania” by Kisanga, “Nutrition Security System at Household Level” by Bajaj, “Issues in Need of a Global Focus” by Ghassemi, October 1990. (SOA No. 6)

Malnutrition and Infection – A Review by A. Tomkins and F. Watson, October 1989, *reprinted June 1993* (SOA No. 5)

Women’s Role in Food Chain Activities and their Implications for Nutrition by Gerd Holmboe–Ottesen, Ophelia Mascarenhas and Margareta Wandel, May 1989. (SOA No. 4) (*out of stock – but photocopy available*)

The Prevention and Control of Iodine Deficiency Disorders by Basil S. Hetzel, March 1988, *reprinted June 1993*. (SOA No. 3)

Delivery of Oral Doses of Vitamin A to Prevent Vitamin A Deficiency and Nutritional Blindness by Keith P. West Jr and Alfred Sommer, June 1987, *reprinted June 1993. (SOA No. 2)*

**SCN NEWS – A periodic review of developments in international nutrition compiled from information available to the ACC/SCN, published twice yearly. Contains features, news and views, programme news, and reviews of publications
(Distributed free of charge)**

SCN NEWS No. 19 *December 1999* Nutrition and Healthy Ageing. 😊

SCN NEWS No. 18 *July 1999* Human Rights & the Right to Adequate Food: SCN's 26th Symposium Report. 😊

SCN NEWS No. 17 *December 1998* Nutrition and HIV/AIDS including HIV, Infant Feeding, Micronutrients in HIV Transmission. 😊

SCN NEWS No. 16 *July 1998* Nutrition of the School-aged Child: A summary of Working Group discussions, Oslo 1998, Abstracts from the Symposium on Challenges for the 21st Century: a Gender Perspective on Nutrition through the Life Cycle. 😊

SCN NEWS No. 15 *December 1997* Effective Programmes in Africa for Improving Nutrition, the 10th Annual Martin J. Forman Lecture: How are we doing in International Nutrition? 😊

SCN NEWS No. 14 *July 1997* The Nutrition Challenge in the 21st Century: What Role for the United Nations? Meeting the Nutrition Challenge: A Call to Arms; Update on the Nutrition Situation, 1996; Poor Nutrition and Chronic Disease; Effective Programmes in Africa for Improving Nutrition.

SCN NEWS No. 13 *late 1995* Interview with Dr A. Horwitz, SCN Chair, 1986–1995; Behavioural Change and Nutrition Programmes; Poor Nutrition and Chronic Disease Part I.

SCN NEWS No. 12 *early 1995* The Role of Care in Nutrition – A Neglected Essential Ingredient; Summary of findings from the recently published ACC/SCN “Update on the Nutrition Situation, 1994”; Specific Deficiencies Versus Growth Failure: Type I and Type II Nutrients; and Enrichment of Food Staples Through Plant Breeding. A New Strategy for Fighting Micronutrient Malnutrition. (*out of stock – but photocopy available*)

SCN NEWS No. 11 *mid 1994* Maternal and Child Nutrition: Adolescent Growth; Prepregnancy Nutritional Status and its Impact on Birthweight; Maternal Nutrition During Pregnancy as it Affects Infant Growth, Development and Health; The Consequences of Iron Deficiency and Anaemia in Pregnancy on Maternal Health, the Foetus and the Infant; Impact of Maternal Infection on Foetal Growth and Nutrition; Maternal Micronutrient Malnutrition: Effects on Breast Milk and Infant Nutrition, and Priorities for Intervention; Vitamin A Deficiency in the Mother–Infant Dyad; Maternal Protein–Energy Malnutrition and Breastfeeding; and Maternal Nutritional Depletion.

SCN NEWS No. 10 *late 1993* Nutrition and Food Aid, Nutrition and Human Rights, The Nutrition Transition.

SCN NEWS No. 9 *mid 1993* Focus on Micronutrients. Features: Addressing Micronutrient Malnutrition, Micronutrient Deficiency – The Global Situation, Effectiveness of Vitamin A Supplementation in the Control of Young Child Morbidity and Mortality in Developing Countries, Zinc Deficiency – Is It Widespread but Under–Recognized? (*out of stock – but photocopy available*)

SCN NEWS No. 8 *late 1992* Highlights of the World Nutrition Situation, Food Prices and Nutrition, Food Security and Nutrition 1971–91 – Lessons Learned and Future Priorities, Long–Term Effects of Improved Childhood Nutrition.

SCN NEWS No. 7 *mid 1991* Refugees' Nutrition Crisis, Breastfeeding, Birth Spacing and Nutrition, Community–Based Development – From a Programme Towards a Movement, Micronutrient Intakes, Incomes and Prices. Supplement: Some Options for Improving Nutrition in the 1990s – Reviews experience of policies and programmes, and grouping nutrition issues, leads to identifying options as building blocks for future action.

SCN NEWS No. 6 *late 1990* Preventing Anaemia, Policies to Improve Nutrition – What Was Done in the 80s, Weaning Foods – New Uses of Traditional Methods. (*out of stock – but photocopy available*)

SCN NEWS No. 5 *early 1990* Nutrition and School Performance, Uses of Anthropometry, Malnutrition and Infection (Part II), Flows of External Resources for Nutrition. (*out of stock – but photocopy available*)

SCN NEWS No. 4 *late 1989* Update on the Nutrition Situation, Women and Nutrition, Malnutrition and infection (Part 1), Targeted Food Subsidies. (*out of stock – but photocopy available*)

SCN NEWS No. 3 *early 1989* Does Cash Cropping Affect Nutrition?, Nutrition in Times of Disaster.

SCN NEWS Nos. 1 and 2 *March 1988* Vitamin A Deficiency, Urbanization, World Nutrition Situation, Economic adjustment (*out of stock – but photocopy available*)

REFUGEE NUTRITION INFORMATION SYSTEM



RNIS reports on the nutrition situation of refugee and displaced populations – issued every three months 😊 with an interim electronic mail update when warranted

Most recent issue:

RNIS 31 – July 2000 which includes supplements on the anthropometric assessment of the nutrition status in emergency-situations of adolescents and adults

COUNTRY CASE STUDIES

Brazil: The improvement in Child Nutritional Status in Brazil: How Did it Occur? by R. F. Iunes & C. A. Monteiro. September 1993.

Egypt: Review of Trends, Policies and Programmes Affecting Nutrition and Health in Egypt (1970–1990), by H. Nassar, W. Moussa, A. Kamel & A. Miniawi. January 1992 (*out of stock – but photocopy available*)

India: Nutrition in India, by V. Reddy, M. Shekar, P. Rao & S. Gillespie. December 1992 (*out of stock – but photocopy available*)

Indonesia: Economic Growth, Equity and Nutritional Improvement in Indonesia, by I. T. Soekirman, G. S. Idrus Jus'at & F. Jalal. December 1992. (*out of stock – but photocopy available*)

Tanzania: Nutrition–Relevant Actions in Tanzania, by F. P. Kavishe. April 1993 (*out of stock – photocopy available*)

Thailand: Nutrition and Health in Thailand: Trends and Actions, by Y. Kachondham, P. Winichagoon & K. Tontisirin. December 1992 (*out of stock – but photocopy available*)

Zimbabwe: Nutrition–Relevant Actions in Zimbabwe, by J. Tagwireyi, T. Jayne & N. Lenneye. December 1992 (*out of stock – but photocopy available*)

Sign up to our mailing list/order publications on line:

<http://acc.unsystem.org/scn/> **or by EMAIL:** accscn@who.int

or by mail (see publications order form)

ACC/SCN

c/o World Health Organization

20 Avenue Appia, CH 1211 Geneva 27, Switzerland

Fax: +41–22–798 88 91

Publications Order Form

Mailing List Subscription: (Note: Both these publications are distributed free of charge to all destinations)

- Check this box to be placed on the mailing list for **SCN NEWS**
- Check this box to be placed on the mailing list for **Refugee Nutrition Information System**

Please send the following ACC/SCN reports and nutrition policy papers:

If requesting from outside Australia, Europe, Japan, New Zealand and North America reports and NPP publications are free of charge. However, if requesting from within Australia, Europe, Japan, New Zealand and North America, checking this box means you undertake to remit the cost of the publication/s upon receipt of the request. We regret we do not have credit card facilities.

- Second Report on the World Nutrition Situation. Volume I, Global and Regional Results (Oct 1992) US\$10*
- Second Report on the World Nutrition Situation. Volume II, Country Data (March 1993) US\$10*
- Update on the Nutrition Situation (November 1994) US\$10*
- Update on the Nutrition Situation: Summary Results for the Third Report on the World Nutrition Situation (Late 1996)*
- Third Report on the World Nutrition Situation (December 1997) US\$15*
- Fourth Report on the World Nutrition Situation (January 2000) US\$15*
- SOA No. 2 Vitamin A (1987) (Reprinted 1993) US\$10*
- SOA No. 3 Iodine (1988) (Reprinted 1993) US\$10*
- SOA No. 4 Women's Role in the Food Chain (1990) US\$10*
- SOA No. 5 Malnutrition and Infection (1990) US\$15*
- SOA No. 6 Women and Nutrition (1990) US\$15*

- SOA No. 7 *Appropriate Uses of Child Anthropometry* (1990) US\$10
- SOA No. 8 *Managing Successful Nutrition Programmes* (1991) US\$15
- SOA No. 9 *Controlling Iron Deficiency* (1991) US\$15
- SOA No. 10 *Nutrition–Relevant Actions – Some Experiences from the Eighties and Lessons for the Nineties* (1991) US\$15
- SOA No. 11 *Nutrition and Population Links – Breastfeeding, Family Planning and Child Health* (1992) US\$10
- SOA No. 12 *Nutritional Issues in Food Aid* (1993) US\$15
- SOA No. 13 *Effectiveness of Vitamin A Supplementation in the Control of Young Child Morbidity and Mortality in Developing Countries* (1993) US\$10
- SOA No. 14 *Controlling Vitamin A Deficiency* (1994) US\$10
- SOA No. 15 *How Nutrition Improves* (1996) US\$15
- NPP No. 16 *Nutrition and Poverty* (1997) US\$15
- NPP No. 17 *Challenges for the 21st Century: A Gender Perspective* (1998) US\$15
- Report of a Workshop on the *Improvement of the Nutrition of Refugees and Displaced People in Africa, Machakos, Kenya* (December 1994) US\$10
- Brazil Country Case Study (September 1993) US\$10

Please send the following issues of **SCN NEWS**

- | | | | | |
|--------------------------|--------------------------|---------------------------------|--------------------------|---------------------------------|
| <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> No. 14 | <input type="checkbox"/> | <input type="checkbox"/> No. 20 |
| No. | No. | | No. | |
| 3 | 10 | | 17 | |
| <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> No. 15 | <input type="checkbox"/> | <input type="checkbox"/> No. |
| No. | No. | | No. | |
| 7 | 11 | | 18 | |
| <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> No. 16 | <input type="checkbox"/> | <input type="checkbox"/> No. |
| No. | No. | | No. | |
| 8 | 13 | | 19 | |

(Nos 1, 2, 4, 5, 6, 9 and 12 are out of print – a photocopy can be requested by circling the issues required)

PLEASE PRINT CLEARLY

Family Name: Given Name: Dr/Mr/Mrs/Ms

Organization:

Mailing Address:

Country:

Email: Telephone: Fax:



*ACC/SCN, c/o World Health Organization
20, Avenue Appia, CH 1211 Geneva 27, Switzerland
Telephone: [41 22] 791 0456, Fax: [41 22] 798 8891, EMail: ACCSCN@WHO.INT*

July 2000, News 20

