Early life nutrition and lifelong health
February 2009
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Chair, Board of Science  Professor Sir Charles George
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Head of Science and Education/Project lead  Nicky Jayesinghe
Authors  Professor Mark Hanson
         Professor Caroline Fall
         Dr Sian Robinson
         Dr Janis Baird
Contributors  Luke Garland
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**Professor Mark Hanson**, Director – Institute of Developmental Sciences, Director – Developmental Origins of Health and Disease Division, British Heart Foundation Professor of Cardiovascular Science, University of Southampton

**Professor Caroline Fall**, Professor of International Paediatric Epidemiology, Medical Research Council (MRC) Epidemiology Resource Centre, University of Southampton

**Dr Sian Robinson**, Principal Research Fellow, MRC Epidemiology Resource Centre, University of Southampton

**Dr Janis Baird**, Senior Research Fellow and Honorary Consultant in Public Health, MRC Epidemiology Resource Centre, University of Southampton

**Professor Alan Jackson**, Director, Institute of Human Nutrition, Developmental Origins of Health and Disease Division, University of Southampton

**Professor Cyrus Cooper**, Director of the MRC Epidemiology Resource Centre and Professor of Rheumatology at the University of Southampton

**Professor Keith Godfrey**, MRC Epidemiology Resource Centre, University of Southampton and Centre for Developmental Origins of Health and Disease, University of Southampton

**Dr Tim Lobstein**, Director of the Childhood Obesity Programme International Association for the Study of Obesity.

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**Address for correspondence:** Professor Mark Hanson, Institute of Developmental Sciences, Mailpoint 887, Southampton General Hospital, Tremona Road, Southampton, SO16 6YD
Tel: 02380 798421 Fax: 02380 795255 Email: m.hanson@soton.ac.uk
About the authors

Mark Hanson  MA DPHI Cert Ed FRCOG is Director of the Division of Developmental Origins of Health and Disease and British Heart Foundation Professor of Cardiovascular Science at the University of Southampton School of Medicine. He is founding Director of the Institute of Developmental Sciences at Southampton. His research concerns the ways in which the developmental environment, and especially nutrition, affects cardiovascular, metabolic and behavioural characteristics of the individual, influencing how well they respond to later challenges and the resulting risks of disease. He is much involved in promoting the public understanding of this area of biomedical science.

Caroline Fall  MBChB, DM, FRCP, FRCPCH is a consultant paediatrician and Professor of International Paediatric Epidemiology at the MRC Epidemiology Resource Centre, University of Southampton. Her research focuses on the effects of maternal and infant nutrition on obesity, diabetes and cardiovascular disease in later life.

Sian Robinson  PhD R Nutr is a Registered Nutritionist and a Principal Research Fellow at the MRC Epidemiology Resource Centre at the University of Southampton. She is responsible for the nutritional components of two large UK cohorts run by the Epidemiology Resource Centre, the Southampton Women’s Survey and the Hertfordshire Cohort Study. Her principal research interests are in maternal and infant nutrition.

Janis Baird  MB BCh, PhD, FFPH is a Senior Research Fellow and Honorary Consultant in Public Health at the MRC Epidemiology Resource Centre, University of Southampton. Her research focuses on translating evidence of the developmental origins of health and disease into public health policy.
Abbreviations

AA  Arachidonic acid
AGA  Appropriate-for-gestational-age
ALSPAC  Avon Longitudinal Study of Parents and Children
ARM  Annual representative meeting
BMA  British Medical Association
BMC  Bone mineral content
BMI  Body mass index
CHD  Coronary heart disease
CI  Confidence intervals
COMA  Committee on Medical Aspects of Food and Nutrition
CSE  Certificate of secondary education
DALY  Disability adjusted life year
DDT  Dichlorodiphenyl trichloroethane
DH  Department of Health
DHA  Docosahexanoic acid
DNA  Deoxyribonucleic acid
Dnmt  DNA N-methyl transferase
DXA  Dual-energy x-ray absorptiometry
EFNEP  Expanded Food and Nutrition Education Programme
FSA  Food Standards Agency
GP  General practitioner
HDL  High-density lipoprotein
HIV  Human Immunodeficiency Virus
IBFAN  International Baby Food Action Network
IFS  Infant feeding survey
Ig  Immunoglobulin
IGF  Insulin-like growth factors
IGT  Impaired glucose tolerance
INCAP  Institute of Nutrition of Central American and Panama
IQ  Intelligence quotient
IUGR  Intrauterine growth restriction
LCPUFA  Long-chain polyunsaturated fatty acids
LDL  Low-density lipoprotein
LGA  Large-for-gestational-age
LIDNS  Low Income Diet and Nutrition Survey
MRC  Medical Research Council
NDNS  National Diet and Nutrition Survey
NICE  National Institute for Health and Clinical Excellence
PCB  Polychlorinated biphenyls
PSA  Public service agreement
RB  Representative body
RCPCH  Royal College of Paediatrics and Child Heath
RCT  Randomised controlled trial
SACN  Scientific Advisory Committee on Nutrition
SBP  Systolic blood pressure
SD  Standard deviation
SGA  Small-for-gestational-age
UK  United Kingdom
UNICEF  United Nations Children's Fund
USA  United States of America
WHO  World Health Organisation
WIC  US Special Supplemental Nutrition Programme for Women, Infants and Children
Adiposity – relating to, or composed of adipose tissue containing fat cells

Anaemia – a condition in which the number of active red blood cells and/or levels of the oxygen carrying pigment haemoglobin in the blood is reduced

Atherogenic – conducive to or causing atherogenesis, or the build up of fatty deposits in the arteries, which leads to cardiovascular disease

Body mass index – an indicator of whether a person is under- or overweight, derived from the weight of a person (in kilograms) divided by the square of their height (in metres)

Cardiomyocytes – cardiac muscle cells

Cardiovascular disease – all conditions that affect the heart and blood vessels

Coeliac disease – a condition in which the small intestine fails to digest and absorb food, due to a permanent sensitivity to the protein gliadin, contained in gluten

Coronary heart disease – heart disease caused by narrowing of the arteries that supply blood to the heart

Diastolic blood pressure – the pressure of the blood against the walls of the main arteries when the ventricles are relaxed and refilling

Disability adjusted life year (DALY) – a health gap measure that extends the concept of potential years of life lost due to premature death to include equivalent years of ‘healthy’ life lost by virtue of being in states of poor health or disability

Dual-energy X-ray absorptiometry – a means of measuring bone mineral density

Dyslipidaemia – a disturbance in the amount of lipid in the blood

Epigenetic – please refer to Appendix 1

FTO gene – a gene on chromosome 16 in humans, certain variants of which appear to correlate with obesity in humans

Galactosaemia – the inability to utilise the sugar galactose, leading to its accumulation within the blood

Gastroenteritis – the inflammation of the stomach and intestine, usually due to infection or food-poisoning

Gastro-intestinal illness – illness relating to the stomach and the intestine

Glycaemic index – a dietary index that is used to rank carbohydrate-based foods. The index indicates the rate at which the food we eat will increase blood sugar levels

Goitre – swelling of the neck due to enlargement of the thyroid gland. This may be due to a lack of dietary iodine

Gonadotrophin – any of several hormones synthesised and released by the pituitary gland that act on the testes or ovaries to promote the production of sex hormones and either sperm or ova

Hodgkin's lymphoma – a malignant disease of lymphatic tissue, usually characterised by painless enlargement of one or more groups of lymph nodes

Homeostasis – the physiological process by which the internal systems of the body are maintained at equilibrium, despite variations in external conditions

Hyperinsulinaemia – excessive insulin secretion

Hyperphagia – abnormally large intake of food

Hypertension – high blood pressure, above the normal range expected for a particular age group and sex

In utero – a Latin term meaning inside the womb, and used with reference to the fetus during pregnancy

Insulin-like growth factors – polypeptides with high sequence similarity to insulin, they are part of a complex system that cells use to communicate with their physiological environment and to regulate their growth and metabolism

Intrauterine growth restriction – a reduction in the growth of the fetus, most commonly resulting from placental dysfunction, poor maternal nutrition, or maternal smoking, and resulting in a baby that is small for gestational age.
Ischaemic heart disease – heart disease resulting from an inadequate flow of blood to the heart, caused by narrowing or blockage of the blood vessels supplying it

Lymphoblastic leukaemia – a type of leukaemia characterised by abnormal cells with a large nucleus and little cytoplasm present in the blood and blood forming organs

Mastitis – inflammation of the breast, usually caused by bacterial infection via damaged nipples

Meiosis – a type of cell division, consisting of two successive separations, resulting in four daughter cells, each with half the original number of chromosomes of the original cell; the process used in gamete (sperm and ova) formation

Meiotic – pertaining to meiosis

Mitosis – a type of cell division resulting in two daughter cells, genetically identical to the original cell; the process used in growth

Mitotic – pertaining to mitosis

Neuroblastoma – a malignant tumour usually of childhood, composed of embryonic nerve cells. It may originate in any part of the sympathetic nervous system

Non-genomic inheritance – any pattern of inheritance in which traits do not segregate in accordance with genomic factors

Normoglycaemic – a condition of the blood in which the blood sugar level is within normal limits

Obesity – refer to Appendix 2

Obesogenic – factors tending to cause or produce obesity in an individual

Osteoporosis – the loss of bone tissue, resulting in bones which are brittle and liable to fracture

Otitis media – inflammation of the middle ear associated with perforations of the eardrum

Overweight – refer to Appendix 2

Palaeolithic – pertaining to a prehistoric era distinguished by the development of the first stone tools, extending from approximately 2.5 million to around 10,000 years ago

Perinatal care – care relating to the period starting a few weeks before birth, including birth and a few weeks after birth

Phenotype – the observable characteristics of an individual resulting from the interaction between their genes and the environment

Plasminogen activator inhibitor – an inhibitor of enzymes that convert inactive plasminogen into the active enzyme plasmin, which digests blood clots

Polymorphism – a condition in which a chromosome or a genetic character occurs in more than one form, resulting in the coexistence of more than one morphological type within a population

Pro-inflammatory cytokines – proteins produced predominantly by activated immune cells, involved in the amplification of inflammatory reactions

Rickets – a disease in which bones do not harden due to a deficiency of vitamin D

Statin – any one of a class of pharmaceuticals that inhibit the action of an enzyme (hydroxymethylglutaryl coenzyme A reductase) involved in the production of cholesterol within the liver

Stroke – a sudden attack of weakness affecting one side of the body or other loss of neural function lasting at least 24 hours. It is the consequence of an interruption to the flow of blood to the brain

Systolic blood pressure – the pressure of the blood against the walls of the main arteries when the ventricles are contracting

Teratogenic – the effect of a substance, agent or process that induces the formation of developmental abnormalities in a fetus

Type 1 diabetes – an autoimmune disease that results in the permanent destruction of insulin producing beta cells of the pancreas

Type 2 diabetes – a metabolic disorder, primarily characterised by insulin resistance, relative insulin deficiency and hyperglycaemia

Undernutrition – inadequate nutrition from any cause

von Willebrand factor – a glycoprotein necessary for platelet function, abnormalities of which can result in an inherited disorder of the blood which is characterised by episodes of spontaneous bleeding similar to haemophilia
For 175 years the BMA has promoted medicine and the allied sciences as part of its founding principle to maintain the honour and interests of the medical profession. One element of this work is to be advocates for the health of the public. In doing so we listen first to our members’ concerns. At the annual representative meeting (ARM) members tell us of the problems they see in the communities and patients they serve. Motions are proposed by grass roots doctors and debated at the ARM. Approximately 600 doctors form the representative body (RB), which is the democratically elected, policy-making body of the BMA. Motions passed at the ARM are usually adopted as BMA policy. A list of all BMA polices can be found at www.bma.org.uk

This report was commissioned after an ARM debate in June 2007 that called on the BMA’s Board of Science to recognise and promote the importance of fetal and early life nutrition and its relationship to lifelong health. The Board of Science has previously produced three reports that broadly cover childhood nutrition and exercise. Growing up in Britain: ensuring a healthy future for our children (1999) discusses child health, with a focus on nutrition rather than exercise, from conception to the age of five. Adolescent health (2003) reviews nutrition, exercise and obesity in teenagers (13-19 year olds). It serves in part to develop the 1999 report in order to cover children up to the age of 12 years. It highlights the main aspects of childhood nutrition and exercise, draws attention to the role of the clinician, and provides links to sources of further information. It also makes recommendations for tackling the obesity epidemic in the UK. Preventing childhood obesity (2005) highlights the situation with regard to childhood obesity and the impact this can have on children’s current and future health. It examines the role of healthcare professionals and the environmental barriers to change that need to be overcome or removed.

This report concerns early life nutrition – predominantly fetal and infant nutrition – providing useful reference information and ‘key messages’ for healthcare professionals. It discusses the evidence-base and draws conclusions about the ways in which the patterns of early life nutrition can be improved, and the likely consequences of such improvements. This is now of critical importance in addressing the rapid increase in the incidence of so-called lifestyle diseases such as cardiovascular disease and type 2 diabetes, which are linked to overweight and obesity. In addition there is now compelling evidence for a role of early life nutrition in setting the risk of other conditions including osteoporosis, asthma, lung disease and some forms of cancer. Evidence is growing that early life nutrition can play a role in behavioural and cognitive problems in children and adolescents, and possibly even in cognitive decline and other aspects of ageing.

This report from the BMA Board of Science is intended to be a useful point of reference for a wide audience, including health professionals, policy makers and members of the public. The approach of the BMA Board of Science is to provide a clear synthesis of the available research, and to develop evidence-based conclusions and recommendations for policy.

Professor Sir Charles George
Chair, Board of Science
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Studies of human populations show clear links between variability in early life environment and risk of later ill health and chronic disease. A critical aspect of environment is the interaction of nutrition with other stressors. Nutrition is important in terms of its quantity and also in the balance of foods and their components, both macronutrients and micronutrients such as minerals and vitamins. An inadequate or unbalanced dietary intake can lead to ‘malnutrition’: the consequences of which embrace the range from under-weight, through the normal range to overweight. The health and other consequences of malnutrition across the entire spectrum are cause for considerable concern in all societies, especially among the poor and most economically deprived. The risk for this burden of ill health is set in train from very early life, providing opportunities for effective interventions at all ages. Future research will help identify further opportunities for effective interventions through better definition of biomarkers of risk, and underlying mechanisms.

In developed societies many women consume poor quality diets, which may result in nutritional deficiencies on the one hand and overweight and obesity on the other. In developing societies maternal under-nutrition remains a major problem but maternal overweight is also of concern. Effective interventions are therefore needed to improve the nutrition of young women of childbearing age in many societies. Less healthy diets are more common among women of low educational attainment, and among women who have low incomes and who are food insecure. Both nutrition education and counselling have been shown to improve women's nutrition knowledge and behaviour. These approaches need to be increased to be effective at a population level and other interventions developed to improve the diets of young women as a matter of urgency.

Our understanding of the specific mechanisms through which maternal nutrition leads to long-term effects is limited in humans, but there is strong evidence relevant to human disease from studies in animals that changing nutrition in pregnancy can produce effects on the offspring. Underlying mechanisms use developmental plasticity to produce adaptive responses to nutritional cues crossing the placenta from the mother. Some cues may even operate in the periconceptional period, before the woman knows she is pregnant. Positive steps therefore need to be taken to ensure that young people understand the importance of health and well-being before pregnancy – giving attention to their diet, maintaining a healthy body weight, stopping smoking and limiting alcohol consumption.

Infants are particularly vulnerable because of their immaturity and the nutritional demands of rapid growth and development. Exclusive breastfeeding is recommended for at least the first six months of life and is associated with reduced susceptibility to infection. The preferred growth associated with breastfeeding enables normal cognitive and physical development and may protect against obesity and chronic disease in adult life. Breastfeeding rates are unacceptably low in the UK and especially so among disadvantaged women. Mothers may need personal support to establish successful breastfeeding and wider society should support parents in the initiation and prolongation of the duration of breastfeeding. Interventions which educate women about the benefits and practice of breastfeeding, and which promote baby friendly policy and practice, are effective at promoting the initiation and prolonging the duration of breastfeeding. The ten steps to successful breastfeeding and the Baby Friendly Hospital Initiative guidelines should become a minimum standard of care. Action is also needed to improve the opportunities for women to breastfeed in public places and to support women who return to work to continue breastfeeding.

There are wide variations in complementary feeding (weaning) practice in the UK. The influence of the timing and nutritional content of complementary feeding and the specific effects of variations in quality of complementary feeding on current and later health in countries such as the UK are still to be determined. There are wide variations in infant size, weight gain, linear growth and body...
composition. There is increasing evidence that these influence the risk of developing obesity, diabetes, cardiovascular disease and other health outcomes in later life. The optimal pattern(s) of infant growth to minimise the risk of obesity, cardiovascular disease and diabetes need(s) to be determined. There is strong evidence that undernutrition (stunting or wasting) during the first two years of life leads to impaired adult cognitive, physical and economic capacity, which cannot be repaired even if nutrition improves later in childhood.

Dietary patterns run in families and the diet of infants tends to be similar to that of mothers. It is likely that interventions which improve the diets of young women will also lead to improved diets for their children. The Healthy Start scheme provides support for eligible women and children through provision of food vouchers and vitamin supplements, and needs to be promoted widely. Its impact on the nutrition of mothers and young children should be evaluated at a national level. Development of future interventions highlights the need for biomarkers of poor developmental nutrition in infants and children.

While there are gaps in the evidence about the long-term consequences of poor maternal and infant nutrition, and we do not as yet understand the mechanisms fully, it is clear that steps need to be taken to promote healthy diets in young women and their families, to encourage breastfeeding and the use of appropriate complementary foods.
Report conclusions

Chapter 1
• Balanced nutrition during human development is of critical importance for later health and wellbeing and for reducing the risks of many chronic diseases.
• Unbalanced nutrition can result from diets which have either excessive or inadequate nutrient intakes. Energy dense diets can none the less be poor in micronutrients.
• The consequences of unbalanced nutrition at both ends of the dietary range are associated with increased risks of adult chronic disease.
• Humans evolved to consume a diet very different from that consumed by many people today. This makes our physiology potentially mismatched to our contemporary lifestyles, increasing the risks of ill health.
• During development, humans like other animals attempt to match the structure and functions of their organs and tissues to the world in which they expect to live. The prediction is based on cues from the mother’s environment via the placenta and her milk.
• Inaccurate predictions, for example through socio-economic change leading to a nutritional transition between generations, increase mismatch and risk of disease.
• In addition to maternal undernutrition, the rising incidence of maternal obesity and diabetes in pregnancy will exacerbate the epidemic of chronic disease in developed societies.
• As low income countries develop, the cycle of diseases such as diabetes and obesity triggered by nutritional mismatch may be followed by further cycles arising from relative overnutrition during fetal and infant development.

Chapter 2
• Studies of human populations show clear links between early life environment and later health and disease.
• While our knowledge of the specific components of maternal nutrition which produce such effects is limited in humans, experimental animal studies provide strong evidence that changing nutrition in the periconceptional period or in pregnancy can produce effects on the offspring which mimic human chronic disease.
• In developed societies, we know that many women consume poor quality diets, which result on the one hand in nutritional deficiencies and on the other in overweight and obesity.
• In developing societies, while maternal undernutrition remains a major problem, maternal overweight is also of concern.
• Development of future interventions will require the identification of biomarkers of poor developmental nutrition in infants and children.
• There is a need for better understanding of the implications of current variations in diet and nutrition for fetal development and long-term health.
• Positive steps need to be taken to ensure that young people understand the importance of health and wellbeing before pregnancy – giving attention to their diet, optimal body weight, to stopping smoking and to limiting alcohol consumption.

Chapter 3
• Infants have special nutritional requirements because of their rapid growth and development and vulnerability to infection. Optimal nutrition in infancy is essential for normal cognitive and physical development and may protect against obesity and chronic disease in adult life. Many parents need guidance to provide adequate nutrition for their children at this stage of life.
• Breast milk is the ideal food for babies in their first few months. Mothers need support in order to breastfeed successfully. There is consistent evidence of better childhood cognitive development, and a lower risk of several disease outcomes including obesity and diabetes, in children and adults who were breastfed rather than formula-fed. It is not known whether these effects are causal or reflect the generally healthier lifestyles of the families whose mothers choose to breastfeed.
The effects of the quality of complementary feeding on current and later health in countries such as the UK are largely unknown. There are wide variations in infant size, weight gain, linear growth and body composition. There is increasing evidence that these influence the risk of developing obesity, diabetes, cardiovascular disease and other health outcomes in later life. The optimal pattern(s) of infant weight gain in order to minimise the risk of obesity, cardiovascular disease and diabetes is, however, not yet known. There is strong evidence that undernutrition (stunting or wasting) during infancy leads to impaired adult cognitive, physical and economic capacity, even if nutrition improves later in childhood.

Chapter 4

- Many young women have diets of poor quality and inadequate nutritional status. Less healthy diets are more common among women of low educational attainment, and among women who have low incomes and who are food insecure. Although evidence of effective interventions to improve the diets of young women is limited, this does not mean that the goal should not be pursued vigorously. Both nutrition education and counselling have been shown to improve women’s nutrition knowledge and behaviour.
- Breastfeeding rates are low in the UK, and it is less common among disadvantaged women. Interventions that educate women about the benefits and practice of breastfeeding, and that promote baby friendly policies and practice, are effective at promoting the initiation and prolonging the duration of breastfeeding.
- Current studies of complementary feeding [weaning] show wide variations in practice in the UK. There are few studies of interventions to influence the timing and nutritional content of complementary feeding in developed countries.
- Infant diet is strongly linked to mother’s diet – suggesting that interventions to improve the diets of young women will also have direct consequences for children’s diets.
- Effective interventions are needed to improve the nutrition of young women of childbearing age. Such interventions may influence the way in which mothers feed their children as well as influencing the diets of women themselves. They will therefore have beneficial health effects across generations.
- Efforts to encourage the initiation and to prolong the duration of breastfeeding need to continue and be extended. The ten steps to successful breastfeeding and the Baby Friendly Hospital Initiative guidelines should become a minimum standard of care. Action is also needed to improve the opportunities for women to breastfeed in public places and to support continued breastfeeding in women who return to work.
- The Healthy Start scheme provides support for eligible women and children through provision of food vouchers and vitamin supplements, and needs to be promoted widely. Its impact on the nutrition of mothers and young children should be evaluated at a national level.
- Other gaps in the evidence should be addressed, including interventions to support breastfeeding mothers in the workplace and interventions to optimise the timing and content of complementary feeding.
Chapter 1: The importance of fetal and infant nutrition

Our current level of interest in the link between nutrition and health is unprecedented. Over the past century nutritional science has grown from the recognition of the properties of nutrients and their ability to prevent deficiency diseases to an understanding of the benefits of good nutrition which include the promotion and maintenance of good health and protection from disease. The definition of an optimal diet – which provides a balanced supply of nutrients in sufficient amounts to support optimal metabolic function – is not necessarily clear. What constitutes an appropriate balance of foods and nutrients in the diet may differ between individuals, based on their genetic makeup and previous experience.

A poor, unbalanced diet has been shown in recent studies to be common in the general population even in developed societies. Poor micronutrient and vitamin status, such as folate and vitamin D, occurs in many people but particularly in those of reproductive age even though their energy intakes may be sufficient. In developing countries maternal and child undernutrition is the cause of 3.5 million deaths each year, constituting 35 per cent of the burden of disease in children under five years. Sub-optimal breastfeeding, especially non-exclusive breastfeeding in the first six months of life, results in 1.4 million deaths and 10 per cent of the disease burden in children under five years. At the same time, there are over 300 million people around the world who are obese. The effects of overnutrition are not only a concern in developed societies, living a ‘Western’ lifestyle. Populations which are emerging from poverty are also at risk of the adverse effects on health caused by excesses in the diet, even in people who are not obese by Western standards. Diets which lead to over-nutrition (eg excess calories) are often micronutrient poor. Thus the possible effects of poor, unbalanced and excessive nutrition on human development need to be considered across a broad spectrum.

Key message

Unbalanced nutrition can result from both inadequate and excessive dietary intakes, and both can exist at the same time in many populations. Moreover diets which lead to over-nutrition (eg excess calories) are often micronutrient poor.

Nutritional transitions and patterns of chronic disease

Human diets have changed substantially during the course of our evolution and history. Compared with current diets, the pre-agricultural hunter-gatherer diets of our Palaeolithic ancestors were based on wild animal and plant foods and were much higher in protein and lower in carbohydrate (see Figure 1).
Figure 1: The human dietary transition

Estimated dietary components in Palaeolithic human ancestors, based in part on hunter-gatherer diets, compared with those in contemporary United States of America (USA). Note the relatively high protein and low carbohydrate content of the hunter-gatherer diet.


The introduction of agriculture 10,000 years ago permanently changed the nature of our food supply, as plants and animals were domesticated for the first time. Further dietary change followed the Industrial Revolution, which created opportunities to process foods such as grains and cereals; and the onset of global trading introduced new foods into the UK diet. Our dietary patterns have continued to evolve to the present day – as new food products have become available and we are influenced by dietary trends occurring in other parts of the world.

Although it is argued that this rate of change in diet from Palaeolithic times has been too fast to allow the human genome to adapt, and is linked to the rising incidence of chronic diseases, many of the improvements in nutrition in the UK over the last century have had an enormous beneficial impact on mortality and public health. Diseases such as goitre or rickets, which were historically associated with social deprivation and malnutrition, are now rarely seen. Improved nutrition has therefore played a role in the dramatic increase in life expectancy. Some recent trends, however, are a cause for concern, such as the increase in the sugar and salt content of the diet. These recent dietary changes have also been accompanied by reductions in physical activity, and there is considerable concern about the consequences of the combined effects of these changes on the incidence and patterns of obesity and associated diseases.

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A typical hunter-gatherer diet versus a typical modern USA diet.
What is good nutrition?

Good nutrition implies that the body’s needs for energy and nutrients, from the macronutrients protein, carbohydrate and fats to the micronutrients such as vitamins and trace elements, are being met by the diet. The body’s needs for nutrients arise from a wide range of processes. The most immediately obvious are linked to basal metabolism – such as the need for dietary fat and carbohydrate to produce energy, and thiamine (vitamin B1) in its active pyrophosphate form as a cofactor in oxidative decarboxylation of pyruvic acid and α-ketoglutaric acid. Although physical activity and work require energy, the greater part of our need for dietary energy is to support basal metabolism. Nutrient intake needs to be sufficient to maintain body reserves and to replace the turnover of components such as muscle proteins that are broken down during normal function. An adequate nutrient intake is essential for immunity; nutritional status and the intake of nutrients such as n-3 polyunsaturated fatty acids, antioxidant nutrients and zinc affect immune function – and the activities of the immune system may themselves have negative effects on nutritional status. Finally, nutrient requirements are increased during pregnancy, and during postnatal growth and development. The many functions of nutrients means that adequate nutrition is central to metabolic integrity, and of key importance to growth, reproductive function and the maintenance of good health.

Key message

The many functions of nutrients mean that adequate nutrition is central to metabolic integrity, and of key importance to growth, reproductive function and the maintenance of good health.

Malnutrition implies that the body’s needs for nutrients are not being met; it also describes an imbalance of these constituents that can be associated with either inadequate or excessive food intake. During short periods of insufficient energy supply the body can use reserves, such as fat and glycogen in liver and muscle, and degrade some tissues to preserve its function. In the longer term, an insufficient supply of nutrients will become limiting to function. In early life an insufficient supply of nutrients will result in reduced growth and impaired development. It is noteworthy that during development these effects may be due to deficiencies in dietary constituents that are not usually essential in adult life. For example, because the fetal requirements for the non-essential amino acid glycine are large, it becomes conditionally essential in late gestation and poor nutrition can produce effects of glycine insufficiency.

The developmental consequences of inappropriate nutrition

The focus of this report is on the long-term consequences for health arising from impaired growth and development in early life – both in prenatal life and in early childhood. Every individual has a ‘blue-print’ that is genetically determined, and that sets their growth potential; but realisation of this potential is only possible if the nutrient supply during growth is adequate. Among babies born to chronically malnourished women, low birth weight is relatively common, and stunting in infancy is prevalent in communities where food supplies are insufficient. Beyond these extreme examples though, our understanding of the importance of variations in diet and nutritional status, such as those seen in the UK population, and how these impact on early development, is limited. Babies continue to be born and to survive in populations that are chronically malnourished, and nutrient supplementation studies carried out in these communities often result in small changes in birth size. In the past it might have been concluded that prenatal growth is largely protected from variations in maternal diet and nutritional status. We now know that variations in birth size, that we describe as being within the normal range, are predictive of disease incidence much later
in life – and that this is evident even in developed communities such as the UK. Within this range, lower birth weight and impaired early growth are linked to an increased occurrence of chronic conditions such as cardiovascular disease and type 2 diabetes in adult life.

**The importance of development for later health**

The discovery that adult chronic disease is linked to low birth weight and poor weight gain in infancy led Hales and Barker, in 1992, to put forward the ‘thrifty phenotype’ hypothesis (see Figure 2). The central element of the hypothesis is that fetal and infant malnutrition are important drivers of the processes leading to disease in later life. Fetal malnutrition could arise through maternal malnutrition or failure of the ‘fetal supply line’ such as placental insufficiency. It was proposed that malnutrition during these periods of rapid development forced the fetus or infant to become ‘thrifty’ and to prioritise limited resources to some tissues at the expense of others. Thus, brain growth would be supported, but truncal growth, the growth of skeletal muscle, the development of abdominal organs (liver, pancreas, kidneys) and some parts of the vascular tree would be deprived. It was also proposed that this altered growth led to permanent changes in the structure and physiology of many tissues, leading to reduced functional capacity in later life. This reduced capacity may not be important to individuals who continued to be poorly nourished, but disease would be triggered more readily by ‘stressors’, such as obesity, in later life. Hence if fetal and/or infant undernutrition resulted in a reduced pancreatic beta cell mass, this would be more likely to result in type 2 diabetes and the metabolic syndrome if the individual became obese in childhood or adult life.

**Key message**

It has been known for many years that unbalanced nutrition during development can cause long-term consequences leading to permanent changes in the structure and physiology of many tissues, leading to reduced functional capacity in later life. The consequences of this for human chronic disease are now increasingly appreciated.
It is now thought that the responses to fetal malnutrition not only alter key organs to ensure survival at that time, but also set in train metabolic adaptations that could improve postnatal survival. The mother in effect gives the fetus and infant a ‘forecast’ of the nutrition it can expect after birth, so that its metabolism can develop to match the environment into which it is born. These adaptations only become detrimental when the postnatal environment differs from that predicted.\textsuperscript{12}

This is the reasoning behind the ‘mismatch’ model of increased risk of chronic disease (see Figure 3).\textsuperscript{13} The use of such terms as ‘forecast’ or ‘predicted’ in this context does not imply that it is a conscious process. It involves fundamental processes of developmental plasticity common to many other species. The cues for such predictions come from the developmental environment and nutritional cues are of paramount importance. Development comprises critical periods in which plasticity operates over a defined period of time, and after this critical period for an organ or system has elapsed, the characteristics of the organism (their phenotype) is fixed (see Box 1). For example, the maturation of the heart muscle cells in humans is completed before birth; there is limited capacity to increase the number of cardiomyocytes postnatally and increases in cardiac muscle mass can only be achieved by hypertrophy of existing cells. This can place them at additional strain and predispose to later heart failure.
Box 1: The impact of phenotypic adaptation on human health

The period of human development is one when the individual attempts to match the structure and function of their organs and tissues to the world they expect to inhabit, particularly the future nutritional environment. These processes are unique to development and may be irreversible. It is for this reason that fetal and infant nutrition is particularly important to future health. Early interventions are not only more likely to be more efficacious in preventing disease than later interventions – it is possible that later interventions may be ineffective. This is because some phenotypic attributes which confer risk of later disease are established in development. Clearly intervention to prevent them can only be made during development itself. This is why understanding the developmental processes involved – and how they may be optimised during human development – is an urgent priority for promoting human health.

According to the mismatch model, the risk of chronic disease will be greatest when there are extreme transitions from one nutritional environment to another, either within an individual’s own lifetime or between generations. This is borne out by the high risk of disease in migrants from poor environments in developing countries to the richer environments of the West. It also emphasises that the risk will be high in populations that start from a low nutritional plane, even if the transition does not place them at a nutritional level that would be considered rich by Western standards. Hence the speed of change in developing countries experiencing economic progress is producing disease in individuals who are younger, and relatively less obese, than in the developed world.

Figure 3: Mismatch model of chronic disease

Over the course of our evolution, humans have adapted to a range of environments, including nutritional and physical activity levels. Within this range (grey zone) we should meet environmental challenges and remain healthy. Outside this range, the risk of disease increases both from inadequate and excessive nutrient/energy level (red lines). Cues from the developmental environment before birth and in infancy alter the phenotype of the person, using epigenetic processes, and hence set their adaptive range. Thus an impaired developmental environment increases the risk of disease due to a phenotype not being well matched to the environment.

**Key message**

Ill-health can result when a person cannot respond adequately to the challenges their lifestyle presents. The settings of these responses are established in part during fetal and infant life, based on nutrition during these periods.

**Obesity**

Obesity is one of the most important problems of our time. The incidence is increasing rapidly all over the world. The rising levels of obesity in the UK, particularly among children, and the significant impact that this will have on the future health of the population are of concern to politicians and healthcare professionals alike. The prevalence of obesity is now so high that it is commonly referred to as an epidemic (see Box 2).

**Box 2: Prevalence of overweight and obesity in the UK**

- In England in 2003, 22 per cent of men and 23 per cent of women were obese and a further 43 per cent of men and 33 per cent of women were overweight.\(^{14}\)
- In England in 2003, more than 2.5 million children aged two to 15 were either overweight or obese.\(^{14}\)
- In Scotland in 2003, one in five (22%) of children aged 11-12 were obese.\(^{15}\)
- The 2005/06 Welsh health survey reported 56 per cent of adults were classified as overweight or obese; 61 per cent of men compared with 51 per cent of women.\(^{16}\)
- The Northern Ireland health and social wellbeing survey 2005/06 reported 59 per cent of adults were classified as overweight or obese; 64 per cent of men compared with 54 per cent of women.\(^{17}\)
- The Scottish health survey 2003 reported 65 per cent of men and 60 per cent women were classified as overweight or obese.\(^{15}\)
- Recent estimates suggest that without action, by 2050, 60 per cent of men, 50 per cent of women and 25 per cent of all children under-16 could be obese. The financial impact of this to the NHS alone could be an additional £5.5 billion per year at current prices.\(^{18}\)
Overweight and obesity in developing countries

Economic development and improvements in agricultural production are transforming the nutritional situation for children and adults in developing countries, although rising food prices and continued food insecurity are a concern. Undernutrition, wasting, stunting and micronutrient deficiencies are still major problems in developing countries, and occurring in early life, they cause long-term damage to human capital and health. Improved availability of energy-rich foods has however, enabled large numbers of people to escape from hunger. This has brought considerable benefits, but is already giving rise to obesity and obesity-related disease. Developing countries are reporting high rates of coronary heart disease (CHD) and type 2 diabetes that have appeared in one or two generations to become leading causes of morbidity and mortality. These epidemics are expected to intensify. By the year 2030, the prevalence of diabetes is predicted to rise by over 100 per cent in India, China, sub-Saharan Africa, Latin America, the Caribbean and the Middle East; an increase far exceeding that in high-income countries (54%). The highest number of people with diabetes will be of working age (45-64 years) unlike developed countries, where the greatest numbers are in older age groups, and in addition diabetes in pregnancy will become a common problem. There are similar increases anticipated in hypertension, CHD and stroke. By 2015, an estimated 20 million people will die from cardiovascular disease every year.

In the context of this report on early life nutrition, obesity is important in a number of ways. Firstly, as described, obesity arising during childhood or adult life, can ‘unmask’ the effects of adaptations induced by undernutrition during fetal life or infancy. Secondly, there is good evidence that increased adiposity and/or an adverse distribution of body fat is itself an outcome of malnutrition (both undernutrition and excessive nutrition) in early life. Adults who had a lower birth weight have been shown to have a higher waist circumference. Low birth weight babies born to underweight women in India are excessively adipose relative to their body weight. In experimental animals, maternal undernutrition during pregnancy is associated with increased adiposity in the adult offspring. The mechanisms for this phenomenon are unknown, but may arise when the correct balance of nutrients required for tissue synthesis is not available, leading to energy storage as adipose tissue. Maternal obesity, another form of maternal malnutrition, also increases the adiposity of the fetus and newborn baby. This phenomenon is exacerbated further if maternal obesity is complicated by gestational diabetes, an example of fetal overnutrition — in which maternal glucose, freely crossing the placenta, stimulates fetal insulin secretion and adipose tissue deposition. It is known that maternal gestational diabetes is a risk factor for early-onset type 2 diabetes in the offspring, and recent data suggest that maternal obesity alone, even in the absence of gestational diabetes, increase the risks of metabolic syndrome in the offspring. A recent follow-up study of more than 9,000 women with normal glucose tolerance at initial screening, found that increasing maternal glycaemia was associated with a greater risk of obesity in the children, even amongst children of normal birth weight. Importantly, among those who fulfilled criteria for gestational diabetes, the relationship between maternal glycaemia and offspring obesity was lost if the mother received treatment.

Maternal obesity is an increasing problem worldwide, and it seems likely that this will contribute increasingly to the burden of diabetes in the future. Gestational diabetes is an example of how susceptibility to diabetes can be transmitted non-genetically from one generation to the next. Women who were themselves of low birth weight are at increased risk of developing gestational diabetes, especially if they become obese in adult life.
Nutrition during infancy also determines later risk of obesity. Rates of overweight and obesity are lower in people who were breastfed, although there is debate as to whether this is a causal relationship. Rapid weight gain in infancy also predicts an increased risk of obesity.

**Key message**

The increasing incidence of obesity will have adverse consequences for the development and health of future generations.

**Previous work by the BMA**

The BMA recognises the importance of nutrition during early life. Before and during pregnancy it is vital that women eat healthily in order to maintain their health and for the pregnancy to develop successfully. A child’s diet during infancy can also impact significantly on their health, growth and development, and on their health in adulthood. Evidence suggests that early life nutrition influences an individual’s risk of adult CHD, diabetes, stroke, asthma and cancer; and has long-term effects on bone health, muscle function, immune and cognitive function and rates of ageing. These relationships may result from either under- or overnutrition in early life.

The BMA publication *Growing up in Britain* (1999) reviews the nutritional needs of children from birth to age five and the health consequences of poor nutrition, including the effects on growth and cognitive development. It highlights the importance of breastfeeding and raises concerns about the need to increase breastfeeding rates in the UK – including addressing the inequalities in breastfeeding between socio-economic groupings. This report also discusses the evidence that ‘influences which act in fetal and infant life permanently set structures and metabolic processes’ in the body. In particular it highlights evidence of a link between undernutrition in utero, low birth weight and later risk of CHD. *Growing up in Britain* makes a number of recommendations about ways in which inequalities in child health should be tackled. In relation to nutrition it states that:

- ensuring good standards of nutrition and advice on healthy eating for young women and their babies must be a priority for Government action with the provision of a safety net for children at nutritional risk, for example, by providing free nursery and school milk, fruit and a balanced meal.
- Government-led strategy should be developed to improve diets of infants and young children and help prevent anaemia, dental caries and obesity. Particular efforts should be focused on increasing breastfeeding rates and improving the quality of weaning diets; breastfeeding should be actively promoted by Government and health professionals, to employers as well as parents, and the benefits monitored.

Many of the themes from the 1999 report are revisited here to establish current thinking about the relationship between early life nutrition and later health; to what degree inequalities in nutrition still exist; and what factors are influential in determining consumption patterns. Concerns about the future health of children have grown further since its publication. Public health initiatives are now focusing attention on the importance of maternal and infant nutrition, both in relation to poor and unbalanced diet and also to overnutrition. This is true in both developed counties such as the UK and also in the developing world. Further research, dissemination of the evidence and discussion of the health promotion policy necessary are urgently needed.
Conclusions

- Balanced nutrition during human development is of critical importance for later health and wellbeing and for reducing the risks of many chronic diseases.

- Unbalanced nutrition can result from diets which have either excessive or inadequate nutrient intakes. Energy dense diets can nonetheless be poor in micronutrients.

- The consequences of unbalanced nutrition at both ends of the dietary range are associated with increased risks of adult chronic disease.

- Humans evolved to consume a diet very different from that consumed by many people today. This makes our physiology potentially mismatched to our contemporary lifestyles, increasing the risks of ill-health.

- During development, humans like other animals attempt to match the structure and functions of their organs and tissues to the world in which they expect to live. The prediction is based on cues from the mother's environment via the placenta and her milk.

- Inaccurate predictions, for example, through socio-economic change leading to a nutritional transition between generations, increase mismatch and risk of disease.

- In addition to maternal undernutrition, the rising incidence of maternal obesity and diabetes in pregnancy will exacerbate the epidemic of chronic disease in developed societies.

- As low income countries develop, the cycle of diseases such as diabetes and obesity triggered by nutritional mismatch may be followed by further cycles arising from relative overnutrition during fetal and infant development.
Chapter 2: Fetal nutrition: impact on development and later life

Having a nutritious diet during pregnancy helps to protect the mother’s health and to control her level of weight gain. Current UK recommendations for diet before and during pregnancy are given in Appendix 3. The embryo and fetus receive all their nutrients directly from the mother; good maternal nutrition is therefore imperative for optimal prenatal development. Unbalanced nutrition will also cause metabolic and hormonal changes in the mother. In animal models this can affect the allocation of stem cells, embryonic and placental lineages, and have long-term effects on offspring growth and health. In humans, maternal diet and body composition affect the growth of the early embryo making a focus on diet before pregnancy as important as that during pregnancy. Later in gestation, when fetal growth is maximal, undernutrition leads to a range of adaptive responses such as redistribution of blood flow in the fetal body and changes in the production of fetal and placental hormones which control growth.\(^\text{30}\) These responses may include changes in placental transport function, an area of research about which we currently know relatively little.\(^\text{31}\) Even without changes in overall fetal body size, the growth of certain organs such as the heart and kidney can be altered. Thus even fetuses of normal birth size may have mounted adaptive responses to unbalanced nutrition and are therefore phenotypically altered. If the nutritional challenge is too great or too prolonged for these adaptive responses to cope, eventually slowing in overall fetal body growth must result, leading to low birth weight. In late gestation this growth restriction is likely to be asymmetrical, with the head being less affected than the body.

Impact of fetal nutrition on health in later life: the developmental origins concept

The developmental origins of disease concept has a long history. The seminal studies of Dorner and colleagues linked pre- and postnatal nutrition to later risks of obesity,\(^\text{32}\) arteriosclerosis\(^\text{33}\) and diabetes\(^\text{34, 35}\) and Gennser et al showed that blood pressure was higher in men of low birth weight.\(^\text{36}\) Forsdahl’s study of Norwegian populations showed that a poor childhood environment was associated with increased chronic adult disease, even if the person’s circumstances improved in later years.\(^\text{37}\) Similarly, Lucas had proposed that detrimental influences in early life may increase risk for later disease.\(^\text{38}\) The most well-known reports, however, are those from Barker and his colleagues in the 1980s, showing an association between low birth weight and risk of later cardiovascular disease in middle-aged men and women in the UK for whom detailed birth records were available.\(^\text{39}\) These early papers from Barker and colleagues were followed by others showing that low birth weight was not only associated with increased risk of death from cardiovascular disease but of also hypertension, insulin resistance, type 2 diabetes, dyslipidaemia and central obesity; in fact each of the criteria which define the metabolic syndrome.\(^\text{40}\)

**Key message**

Low birth weight is associated with increased risk of death from cardiovascular disease, hypertension, insulin resistance, type 2 diabetes, dyslipidaemia and central obesity.
Box 3: Fetal programming

In this context the term ‘fetal programming’ is widely used. It is somewhat misleading, as it implies deterministic mechanisms, similar to its original use for the genetic programme for development. It suggests analogy with a computer programme which, once running, cannot be altered. There is now more use of the terms programming and reprogramming to refer to the processes by which stem cells become pluripotent during embryonic life. This is in line with the original use, as it suggests switches between predetermined developmental pathways, enabling embryonic stem cells to differentiate to lung, liver, lymphocytes, etc. The use of ‘fetal programming’ or ‘programming of disease’ is at odds with the plastic nature of adaptive responses by the embryo, fetus and infant and also underplays the role of later environment in exacerbating the risk of disease. Developmental origins of disease, or induction of risk of disease are more accurate terms for this process.

The validity of the concept of the developmental origins of disease has been questioned because of the wide variability reported among associations between birth weight and proxy markers of disease such as later blood pressure, however associations are much stronger when the outcome measure is clinical disease. Interpretation is also influenced by the recognition that pregnancies in the historical cohorts occurred when nutrition and healthcare were very different from those of contemporary developed societies. The lower birth weight individuals in these cohorts, for example, would be unlikely to include very pre-term or intrauterine growth restriction (IUGR) fetuses, or the higher birth weight group to include fetuses of diabetic mothers. Another criticism has concerned the size of the associations between indices of the developmental environment such as birth weight and later markers of disease. A meta-analysis suggests for example that, even if causal, a large (1kg) increase in birth weight would be associated with only a 10 to 20 per cent reduction in adult ischaemic heart disease across the population. This assumes, however, that birth weight is the exposure of interest. The hypotheses relating fetal nutrition and growth to later disease suggest that birth weight, a crude summary of fetal development, is likely to be a distant reflection of the cellular and molecular processes affected by intra-uterine environment. For disease such as type 2 diabetes, childhood obesity has a large exacerbating effect.

Correction for adult BMI is another area which has proved controversial. There is an association between larger size at birth and increased adult BMI itself a strong risk factor for type 2 diabetes and cardiovascular disease). Since the associations between size at birth and adult diseases are in the opposite direction, when adjusting these associations for adult BMI the effect of small size at birth appears stronger. The interpretation of this effect is controversial; it could indicate that an adverse effect of small size at birth is revealed more clearly by removing the influence of adult BMI, or it could indicate that the key insult is the transition from being a small baby to being an obese adult as in the mismatch concept (see Figure 3). It is impossible to distinguish between these two possibilities using observational data. The evidence actually suggests that both of these scenarios apply. Many studies have shown adverse effects of small size at birth on adult disease without adjustment for BMI (see Figure 4).
Figure 4: Patterns of infant and childhood growth associated with later heart disease

Growth (shown as Z score, i.e., shift in standard deviation from the group mean shown as 0) in 357 boys who developed CHD from a cohort of 4,630 born in Helsinki between 1934 and 1944. Their childhood growth pattern is characterised by small size at birth, slow growth in years 0-2, then relatively greater increase in weight and BMI.


There is also clear evidence of adverse effects of excessive weight gain in childhood on adult disease risk (see Figure 5) but controversy about the effect of weight gain during infancy (see Chapter 3).

Given the similarities in responses between humans and other animals in terms of adaptive responses to environmental challenges during development, it may be helpful to consider them in terms of the spectrum of effects which might be produced on theoretical grounds (see Figure 5). This is important because it shows how even a modest nutritional challenge during development might be expected to have long-term consequences, even without an effect on birth weight.
Figure 5: Graded effects of nutrition in development on later function

Link between the nutritional plane in development and effect on later health and fitness. Different types of effect are produced, depending on the severity of the challenge (and also its timing and duration, not shown). They can be grouped into:

- those where an environmental challenge is so great that it causes a disruption of development, akin to a teratogenic effect. This is rare for unbalanced nutrition
- a less severe challenge but one which nonetheless reduces overall fetal growth and hence birth weight. The offspring has to cope with the detrimental effects of this in later life
- responses to a less severe challenge, which alter the phenotype of the offspring in prediction of its later environment. Such responses are in the majority across the range of human development, and produce risk of disease when the induced phenotype is not matched to the adult environment
- responses to overnutrition in development; this category is becoming increasingly common.


Many studies, including those from countries other than the UK, have replicated the original association between lower birth weight and death from ischaemic heart disease and also with non-fatal coronary artery disease and stroke. A recent study of 13,830 men and women from the Finnish National Death Register showed an association between low birth weight and all cause mortality among women. A study of 10,803 children born in Aberdeen in the 1950s has confirmed an inverse association between
birth weight and later coronary artery disease and stroke. This is important because the 1950s was a time when environmental influences were relatively favourable for infants compared with earlier studies, and so the results may have contemporary relevance. Large cohort studies, such as that of almost 90,000 Swedish army recruits born between 1973 and 1981, also show that lower birth weight is linked to higher adulthood blood pressure and, although small in magnitude, the difference was independent of socio-economic factors or familial effects. Even a small rise in blood pressure is important when viewed from the perspective of risk of later disease. A recent meta-analysis of data from 198,000 individuals from 20 Nordic studies (cohorts of people born between 1910 and 1987) shows unequivocally an inverse and linear association between birth weight in males, but a "U" shaped relationship with females; those of a birth weight higher than 4kg having a higher systolic blood pressure (SBP) in adult life. Similar confirmation of the association with low birth weight and raised SBP has been derived from a British birth cohort study of 3,157 men and women born in 1946.

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Mechanisms by which fetal nutrition affects risk of later disease

The concept of developmental origins of health and disease has stimulated wide ranging investigations into the underlying mechanisms. These investigations have now revealed a great deal of information on the processes involved, and their operation during critical periods of development during fetal and infant life. The fetus is at the end of a long supply line for nutrient delivery from its mother (see Figure 6) so there are several points at which its nutrition might be impaired.

Figure 6: The fetal supply line for nutrients

Many processes intervene between the food consumed by the mother and the delivery of nutrients to the fetal tissues.
Most of the mechanisms have been investigated using animal models. Studies in rats, mice, sheep, guinea pigs and in non-human primates all lend strong support to the developmental origins of health and disease hypothesis. Most notably, several characteristics of the offspring phenotypes arising from a range of maternal nutritional interventions, both by undernutrition and overnutrition, are shared across species. It has proved remarkably easy to demonstrate characteristics equivalent to human disease, particularly features of the metabolic syndrome, through experimental manipulation of the diet of the pregnant animal or her offspring. The effects include cardiovascular and renal dysfunction, insulin resistance, glucose intolerance, hyperphagia, altered stress responses and even preference for food high in fat and sugar in the offspring.

Adulthood adiposity has also been induced. The behavioural effects on the offspring include reduced exploratory behaviour, activity levels and even impaired learning behaviour. These animal models offer the potential for investigation of mechanisms, leading to suggestions for intervention. To date the most effective interventions reported have been supplementation of a low protein diet during pregnancy in the rat with glycine or folate, or treatment of the pups neonatally with leptin. The adverse effects on the offspring of feeding a high fat diet in pregnancy can be reduced by giving a statin to the pregnant animal in late gestation. Animal investigations reveal that the transmission of risk factors can be passed to more than just the next generation. After a nutritional challenge to animals during pregnancy effects were seen in their grand-offspring, even without a further nutritional challenge.

**Key message**

Unbalanced nutrition during development can lead to greater risks of chronic disease in successive generations.

One of the most striking perceptions to arise from recent experimental work in this area is that the effects of relatively modest dietary restriction or imbalance can affect offspring development and have long-term consequences for health even when they are administered to the pregnant animal in either the periconceptional period, or in early pregnancy. At this time they do not invariably affect fetal growth or birth weight. They operate via effects on embryonic stem cell allocation or yolk sac function, and have long-term effects on cardiovascular, endocrine and metabolic function. Embryo transfer experiments also reveal long-term effects on these systems, and this raises questions about the long-term consequences of assisted reproductive technologies. These observations are of general relevance because a high proportion of pregnancies are unplanned, and thus dietary and other effects can be exerted well before the woman realises that she is pregnant.

**Epigenetic mechanisms**

The environmental effects on the embryo referred to above suggest in particular that epigenetic processes may be involved. The processes of phenotypic induction through developmental plasticity produce integrated changes in a range of organs via epigenetic processes. The term ‘epigenetic’ was coined by Waddington in about 1942 to refer to the ways in which the developmental environment can influence the mature phenotype. His work and that of others on developmental plasticity stemmed from observations that environmental influences during development could induce alternative phenotypes from a genotype. They establish a life-course strategy for meeting the demands of the predicted later environment. This explains why impaired early nutrition produces a range of effects. These include alterations in cardiovascular and metabolic homeostasis, growth and body composition, cognitive and behavioural development, reproductive function, repair processes and longevity – some of which are associated with increased risk of chronic
diseases including cardiovascular and metabolic disease, ‘precocious’ puberty, osteoporosis and some forms of cancer. Understanding the epigenetic processes thus holds the key to understanding the underlying pathophysiology and to developing approaches to early diagnosis, prevention and treatment of these diseases (see Appendix 1).

**Maternal undernutrition and long-term outcomes in the offspring**

Newborn size is related to maternal energy balance, increasing with the mother’s BMI and adiposity. Her height, and independently leg length, head circumference and birth weight, predict the baby’s size, suggesting that her own early nutritional biology influences how she nourishes the fetus. The ‘fetal origins hypothesis’ proposed that maternal ill health, poverty and undernutrition impair fetal and infant growth and were root causes of adult chronic disease. If this is so, indices of poor maternal nutrition would be associated with cardiovascular disease or its risk factors in the offspring. So far, unfortunately, there are only crude data available to test this.

There is very little evidence concerning the effects of maternal undernutrition and nutrition in the developing world on the longer term health of the offspring. Studies in developing or historically poor countries have shown that low maternal weight, BMI or skinfold thickness in pregnancy are associated with higher offspring blood pressure, insulin resistance and risk of CHD. Follow-up of people exposed to the 1944-45 Dutch Hunger Winter showed that exposure of the mother to acute famine was associated with increased cardiovascular risk in the adult offspring. Outcomes varied according to the timing of famine exposure; late gestation exposure was associated with greater risk of glucose intolerance and early gestation exposure with obesity, atherogenic lipid profiles and CHD. Famine effects were independent of birth weight, suggesting that maternal undernutrition may impair adult health without reducing size at birth.

The Institute of Nutrition of Central America and Panama (INCAP) trial in Guatemala (1969-77) is the only randomised trial of a nutritional intervention in which cardiovascular risk factors have been measured in the adult offspring. In the original trial, pregnant women and children received one of two supplements: a high-protein, high-energy drink or a lower nutrient drink. There was no difference in birth weight between these two groups but the children of mothers who received a high-protein, high-energy drink had lower blood triglyceride and higher high-density lipid (HDL) cholesterol concentrations in adult life (profiles usually associated with better cardiovascular health). The Pune Maternal Nutrition Study was set up specifically to investigate the relationship of maternal nutrition to the children’s future risk of diabetes and heart disease. It collected prospective information on diet, workload and micronutrient status in pregnant women in rural Indian villages. At six years of age the children were thin by international standards but had a higher percentage body fat than European children. Maternal folate concentrations predicted higher insulin resistance in the child, and children born to mothers with the lowest vitamin B12 concentrations and highest folate concentrations were the most insulin resistant. Thus an imbalance in the mother in two vitamins important in methyl group provision predicted higher adiposity and insulin resistance in the child, suggesting an important role for 1-carbon metabolism in the developmental origins of type 2 diabetes.
Associations between maternal obesity, diabetes, raised birth weight and adulthood disease

The rising birth weight in developed countries (with the possible exception of Japan)\(^9\) associated with the increasing incidence of obesity in pregnancy and related gestational diabetes, also has potential for detrimental influences on the developing child.\(^9\) There is a U shaped relationship between birth weight and later insulin resistance and obesity,\(^5\)\(^9\)\(^,\)\(^10\)\(^1\) and there is now good evidence that children of women who are diabetic in pregnancy are themselves more likely to develop insulin resistance in later life and to become overweight.\(^9\) Initially shown in the Pima Indians, a population with a very high incidence of diabetes, this seems to occur in all populations. School children in Taiwan showed a similar phenomenon\(^1\)\(^2\) and Indian children of gestationally diabetic mothers had increased adiposity and hyperinsulinaemia.\(^1\)\(^3\) While this may represent in part a genetically inherited disorder, studies of sibling pairs discordant for maternal diabetes show that the effect occurs only in the offspring prenatally exposed to diabetes, strongly suggesting a diabetic trait acquired during development.\(^1\)\(^4\)\(^,\)\(^1\)\(^5\)\(^\)\(^1\)\(^6\)\(^\)\(^1\)\(^7\) Infants of mothers with gestational diabetes have a greater neonatal fat mass independent of birth weight\(^1\)\(^8\) and it is reported that a high rate of childhood metabolic syndrome (central adiposity, insulin resistance and hypertension) occurs in large-for-gestational-age (LGA) babies born to mothers with gestational diabetes\(^1\) compared with appropriate-for-gestational-age (AGA) babies. Importantly, the risk of metabolic syndrome is also present, but to a lesser extent, in LGA babies from normoglycaemic mothers. In a recent trial of two diets, normoglycaemic mothers on a high glycaemic index diet gave birth to more LGA babies.\(^1\)\(^9\) Children of mothers with higher glucose concentrations within the normal range are more adipose.\(^2\)\(^7\) Higher birth weight in normoglycaemic pregnancies is generally associated with raised adulthood BMI, and larger babies tend to become heavier adults. Although detailed investigations of the relative contributions of lean and fat mass have sometimes suggested that this association reflects increased lean body mass rather that fat mass, a recent study of infants from obese normoglycaemic mothers shows clear evidence of increased adiposity.\(^1\)\(^1\)\(^0\) To date there have been few attempts to define accurately the relationship between maternal, childhood and adulthood body composition over any range of maternal BMI. Ongoing prospective longitudinal studies in contemporary cohorts such as the Avon Longitudinal Study of Parents and Children (ALSPAC) cohort in Avon UK\(^1\)\(^1\) and the Southampton Women's Survey (SWS)\(^1\)\(^1\) will be very informative in this regard. In the latter it is already clear that mother’s body composition and dietary balance before pregnancy can affect fetal cardiovascular development in late gestation.\(^1\)\(^1\)\(^3\)

**Key message**

Extremes of maternal body composition, either excessive thinness or obesity, are associated with adverse patterns of fetal and infant development leading to poorer long-term health.

**Transgenerational effects**

There is concern that the detrimental effects of the nutritional transition in many populations around the world will have an effect on the risk of chronic disease which will persist for several generations. Animal studies show that a dietary or glucocorticoid challenge administered in pregnancy can have effects on the metabolism, cardiovascular function and gene expression in the grand-offspring, even without any additional challenge in the second generation.\(^7\)\(^4\)\(^,\)\(^1\)\(^1\)\(^4\) The effects can involve epigenetic changes.\(^7\)\(^5\) Similar changes have been reported in humans exposed to poor nutrition during childhood development\(^1\)\(^1\)\(^5\) including the intriguing possibility that the effects can be transmitted through the paternal as well as the maternal line. There is now much interest in the concept that non-genomic inheritance can affect more than one successive generation, both from scientific, social and medical perspectives.\(^7\)
Taking a multigenerational perspective also focuses attention on the ways in which disease risk can be transmitted during periods of nutritional transition (see Figure 7). Obesity, type 2 diabetes and altered metabolism in women resulting from poor nutrition during development can result in greater glucose levels or gestational diabetes in pregnancy. This is likely to induce similar metabolic changes in their offspring.

**Figure 7: Cycles of disease risk**

Cycles of disease risk can involve more than one generation. Women who are malnourished or who have sub-optimal body composition and weight gain in pregnancy are more likely to have offspring with stunting and premature death and morbidity (left part of diagram). This becomes a self-perpetuating cycle. Their offspring are at higher risk of cardiovascular and metabolic disease, especially if socio-economic transition or migration gives access to high fat and glycaemic index food and reduced physical activity. Women of this generation are more likely to develop gestational diabetes and to have offspring which in turn are also at risk of obesity and diabetes. This process, which may involve different mechanisms from the first, nonetheless produces another cycle of disease.

Conclusions

- Studies of human populations show clear links between early life environment and later health and disease.

- While our knowledge of the specific components of maternal nutrition which produce such effects is limited in humans, experimental animal studies provide strong evidence that changing nutrition in the periconceptional period or in pregnancy can produce effects on the offspring which mimic human chronic disease.

- In developed societies, we know that many women consume poor quality diets, which results on the one hand in nutritional deficiencies and on the other in overweight and obesity.

- In developing societies, while maternal undernutrition remains a major problem, maternal overweight is also of concern.

- Development of future interventions will require the identification of biomarkers of poor developmental nutrition in infants and children.

- There is a need for better understanding of the implications of current variations in diet and nutrition for fetal development and long-term health.

- Positive steps need to be taken to ensure that young people understand the importance of health and wellbeing before pregnancy – giving attention to their diet, optimal body weight, to stopping smoking and to limiting alcohol consumption.
Chapter 3: Infant nutrition

Introduction
Infancy refers to the first one to two years after birth. It is a period of rapid growth in weight, length and brain size and rapid social, cognitive and motor development. These changes carry high nutritional needs at a time of total dependence on carers who may have little knowledge about nutrition or the special requirements of infancy. During the first 12 months of life, the infant must make a transition from a milk-only diet to one containing the diversity of foods eaten by the rest of the family. Good nutrition is essential for the infant to maintain and develop its immature immune defences as it meets pathogens for the first time. Patterns of weight gain and growth, and nutritional experience, in infancy can have long-term effects on health (see page 40).

Key Message
Infancy is a period of high nutrient requirements, to support rapid growth (including brain growth) and critical social, cognitive and motor development.

Breast milk is the ideal food for newborn babies and currently in the UK, exclusive breastfeeding is recommended until the age of six months. Other (often termed ‘complementary’) foods are then introduced and gradually increased in variety and quantity. It is not known what constitutes the ideal diet during this transition but there are well-established guidelines on nutrient requirements and on foods and feeding practice. In the UK, there are wide variations in infant feeding practices and in how closely parents follow these guidelines (see page 31). For discussion on the contraindications to breastfeeding see page 35.

In all populations infancy is a period of relatively high mortality. The infant mortality rate (IMR) in England and Wales (five per 1,000 live births in 2006) has fallen continuously for the past 100 years (14.3 in 1976; 105 in 1910). Just over half the deaths are neonatal and related to congenital abnormalities, low birth weight and pre-term birth. There are marked regional differences in IMR. In 2006, it was highest, 6.4, in the West Midlands and lowest, 4.0, in South West England. There are also strong social class differentials. It is not known to what extent these inequalities have a nutritional basis.

Developing country perspective on infant nutrition
Infant undernutrition is a huge problem in developing countries. It is estimated that 178 million (32%) children under five years are stunted, 55 million are wasted, and micronutrient deficiencies are widespread and frequently multiple. Undernutrition causes three million deaths among children under the age of five in developing countries every year. Infancy is the period when growth commonly starts to falter and it is difficult to reverse stunting after the age of two years. The main causes are inadequate food (energy, animal protein and micronutrients) and high rates of infection. Sub-optimal breastfeeding and poor quality complementary foods are key factors. Efforts to improve nutrition in developing countries should focus on pregnant women and infants to get the maximum benefits for growth, development, health and later economic productivity.
The case for breastfeeding

All current guidelines, including those from the Department of Health (DH), recommend exclusive breastfeeding for newborns and for the first six months of infancy. Breast milk provides all the nutrients required at this age in a form that is hygienic and easy to digest. The protein, carbohydrate and fat profiles are unique to breast milk and differ in many ways from other animal milks. Breast milk also contains a range of bioactive components, including anti-microbial and anti-inflammatory factors, digestive enzymes, hormones and growth factors. Anti-microbial agents include leucocytes, secretory immunoglobulin (Ig)A, IgM and IgG antibodies, oligosaccharides, lysozyme, lactoferrin, lipids, fatty acids and mucins. Growth factors are thought to be important for gut maturation. Lactoferrin is one of several specific binders in human milk that greatly increase the bioavailability of micronutrients.

Key message

All current guidelines, including those from DH, recommend exclusive breastfeeding for the first six months after birth.

The volume and composition of breast milk vary with the stage of lactation, within each individual feed, and with maternal nutritional status. Recent research in animals has shown that reduced fetal growth caused by placental insufficiency can impair the quality and quantity of breast milk. Colostrum, produced during the first few days is low in volume and nutrient content but high in anti-microbial factors. Volume and nutrient content reach a peak, in mature breast milk, several weeks after birth. The mother’s macronutrient intakes do not have much influence on milk composition, but her diet does affect the long-chain fatty acid and vitamin content of her milk.

Breastfed infants have more control over the flow of milk than bottle-fed infants. Breastfeeding is therefore often described as an ideal ‘supply and demand’ regulation system. The feeding behaviour of the baby and the quality of the breast milk change with time in a way that may prevent overfeeding, teach the infant how to recognise satiety signals, and regulate energy intake differently from formula-fed infants.

The role of leptin in breast milk may be of particular importance in the early development of both adipose tissue and appetite regulatory systems in the infant, and ultimately on propensity to obesity in later life. A recent study showed that administration of physiological levels of leptin to suckling rats caused a significantly lower body weight in adulthood. Observational studies have shown that breastfeeding is associated with lower rates of childhood obesity. Bearing in mind the absence of leptin in formula milk, this may have important implications for the prevention of obesity in children and in adults.
Current infant feeding practices
The national Infant feeding survey (IFS) is conducted every five years; the latest data are from 2005 (see Box 4). These reports provide a wealth of information about variations in feeding practice, factors that influence the type of milk and duration of milk feeding. They also detail trends in feeding over time. Exclusive breastfeeding is recommended up to the age of six months in the UK. The IFS shows, however, that less than 1 per cent of mothers are exclusively breastfeeding at six months. Current guidelines are to introduce solid foods from six months, to provide a varied diet that includes starchy foods, fruit and vegetables and meat and fish, and to encourage the use of home-prepared rather than commercial baby foods (see Appendix 4).

Box 4: The UK Infant feeding survey 2005 findings on breastfeeding

- Initial breastfeeding rates in 2005 had increased since 2000 (78% in England, 70% in Scotland, 67% in Wales, and 63% in Northern Ireland).
- Only 48 per cent of mothers were breastfeeding at six weeks and 25 per cent at six months. 45 per cent were breastfeeding exclusively at one week, 21 per cent at six weeks, 7 per cent at four months and <1 per cent at six months.
- Three-quarters of mothers had given their baby milk other than breast milk by six weeks, and 92 per cent by six months.
- Just under half of all mothers who had prepared powdered infant formula in the last seven days had not followed the instructions properly, for example by not using cooled boiled water.

Breastfeeding rates in the UK are much lower than in many European countries – for example, more than 90 per cent of babies in a nationally representative sample studied in Norway were breastfed from birth, and high rates were still present at one, four and six months.

The IFS shows that there are strong relationships between the mother’s socio-economic status and educational attainment and breastfeeding prevalence – these factors are associated with both initiation rates and breastfeeding duration (see Figure 8). In the UK Gateshead Millennium Baby Study (1999-2000) 84 per cent of mothers with higher education initiated breastfeeding compared with 25 per cent of mothers with no educational qualifications. Percentages still breastfeeding at four months were 49 per cent and seven per cent. There were also strong negative associations with Townsend score, a measure of deprivation based on residential postcode.

Key message
Breastfeeding rates in the UK are much lower than in many European countries. Less than 1 per cent of mothers in the UK are exclusively breastfeeding at six months.
Box 5: Reasons why mothers do not breastfeed or cease breastfeeding early

A recent focus group study in the UK\(^3\) suggested further detail of the reasons women may not breastfeed or why they stop breastfeeding early. These were as follows.

- **The attitude of other people** – women felt that breastfeeding in public was unacceptable and embarrassing, while bottle-feeding was accepted by everybody and in all places. A lack of places to breastfeed out of sight restricted women’s ability to get out of the house. This may be a bigger issue for low-income women, who may not have the option of breastfeeding in the car. Some women reported breastfeeding in public toilets as the only option. Women wished that cafés and shops could provide places to breastfeed with some privacy.

- **Attitudes of family and friends** – some women said that even family and friends found it ‘repulsive’ to be in the same room when they were breastfeeding. Some grandparents thought it excluded them from having the chance to feed the new baby. It was clear that the opinion of family and friends was a stronger influence than that of health practitioners.

- **Lack of knowledge** – women vaguely knew that breastfeeding was supposed to be beneficial, but they could not name any benefits, and were not convinced about them. In the study only one woman had learnt at school about benefits of breastfeeding; most did not hear about it until they were pregnant. Feeding was not well covered in antenatal classes.

- **Lack of professional support** – women experienced difficulty in trying to establish breastfeeding but were unwilling ‘to bother the midwife’. Bottle feeding seemed easier.

- **Experience** – breastfeeding seemed difficult and painful, and many women experienced problems ranging from getting the baby latched on, sore nipples, and disturbed sleep. Women, especially adolescents, complained of a lack of freedom to travel/socialise/work.

- **Worry about baby’s weight gain** – women said that health visitors were ‘always worried about weight gain’.

Although some women in this study mentioned the benefits of breastfeeding – including feelings of wellbeing and relaxation during feeds, convenience (less washing up), and less expense, it is clear that there are significant barriers for women in the UK which impact on their choice to breastfeed.

### Box 6: Reasons given by mothers for stopping breastfeeding by duration of breastfeeding

<table>
<thead>
<tr>
<th>Why stopped breastfeeding</th>
<th>Less than 1 week</th>
<th>1 week, less than 2 weeks</th>
<th>2 weeks, less than 6 weeks</th>
<th>6 weeks, less than 4 months</th>
<th>4 months, less than 6 months</th>
<th>6 months, less than 9 months</th>
<th>All mothers giving up†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insufficient milk</td>
<td>26</td>
<td>41</td>
<td>53</td>
<td>48</td>
<td>37</td>
<td>24</td>
<td>39</td>
</tr>
<tr>
<td>Baby rejected breast</td>
<td>34</td>
<td>21</td>
<td>17</td>
<td>13</td>
<td>11</td>
<td>16</td>
<td>20</td>
</tr>
<tr>
<td>Painful breasts/nipples</td>
<td>24</td>
<td>29</td>
<td>17</td>
<td>9</td>
<td>1</td>
<td>2</td>
<td>14</td>
</tr>
<tr>
<td>Took too long/tiring</td>
<td>11</td>
<td>17</td>
<td>19</td>
<td>19</td>
<td>5</td>
<td>5</td>
<td>14</td>
</tr>
<tr>
<td>Mother was ill</td>
<td>8</td>
<td>11</td>
<td>9</td>
<td>8</td>
<td>5</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Domestic reasons</td>
<td>4</td>
<td>7</td>
<td>9</td>
<td>10</td>
<td>3</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Returned to work/college</td>
<td>1</td>
<td>–</td>
<td>1</td>
<td>8</td>
<td>15</td>
<td>22</td>
<td>7</td>
</tr>
<tr>
<td>Too stressful</td>
<td>7</td>
<td>7</td>
<td>6</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Baby ill</td>
<td>6</td>
<td>7</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Baby could not be fed by others</td>
<td>1</td>
<td>5</td>
<td>4</td>
<td>6</td>
<td>4</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Did not like breastfeeding</td>
<td>2</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>*</td>
<td>3</td>
</tr>
<tr>
<td>Breastfed for as long as intended</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>10</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>More settled on formula</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>8</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Baby not gaining weight</td>
<td>*</td>
<td>1</td>
<td>2</td>
<td>6</td>
<td>4</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Teething/biting††</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All Stage 3 mothers who stopped breastfeeding during survey period</td>
<td>1143</td>
<td>351</td>
<td>1087</td>
<td>1284</td>
<td>195</td>
<td>727</td>
<td>4827</td>
</tr>
</tbody>
</table>

† Includes some cases where mother’s breastfeeding duration not known
†† Code introduced at later Stages
* Percentage less than 0.5%
Percentages do not add to 100% as some mothers gave more than one answer

Developing country perspective on breastfeeding
As in the UK, breastfeeding rates are sub-optimal in most developing countries. Although more than 95 per cent of babies overall receive some breastfeeding, rates of exclusive breastfeeding are only 50 per cent on average up to the age of two months, and 20 to 30 per cent from two to five months.

Complementary feeding practices in the UK
In comparison with milk feeding in UK infants, current variations in complementary feeding practice and the weaning diet are less well documented. Some information on weaning practice is collected in the IFS (see Box 7).
Box 7: The UK Infant feeding survey 2005 findings on complementary feeding

- In 2005, mothers were introducing solid foods later than in the 2000 survey (51% by four months compared with 85% by four months respectively).
- Very few women (2%) followed DH guidance to delay weaning onto solid foods until six months.
- Babies given solid foods between four and six months were much more likely to be fed commercially-prepared foods (85%) than home-prepared foods (51%).
- Most mothers (92%) avoided the use of salt completely in the diets of their babies.
- The proportion of mothers avoiding the use of salt increased between 2000 and 2005 (33% to 51%), rises were also seen in the proportion avoiding nuts (30% to 48%) and honey (6% to 13%); a greater awareness of food allergies was a key reason for this change.

The IFS showed that in 2005, although there was a decline in prevalence of very early introduction of solid foods since 2000, most infants had been introduced to solid foods before the recommended age of six months. The main reasons given for the introduction of solid foods before three months were that the baby was not satisfied with milk alone, or the mother’s experience with a previous baby. Early introduction of solid foods was more common in younger mothers, and among mothers of lower socio-economic status and lower educational attainment (see Figure 9).

Figure 9: Age by which solids had been introduced according to mother’s socio-economic classification: Infant Feeding Survey 2005

Among working mothers, delaying return to work until at least six months was associated with later introduction of solid foods. 137

The nature of the weaning diet varied by mother’s socio-economic status. When compared with other women, mothers in managerial/professional occupations were more likely to feed their infants cooked vegetables and fruit, rice and pasta, and meat and fish, and less likely to provide regular servings of sweets, biscuits and savoury snacks. 131

There are two large cohort studies of UK infants in which variations in complementary feeding and weaning practice and dietary intakes have been assessed: ALSPAC and the Southampton Women's Survey (SWS). The ALSPAC study collected dietary data using three-day diet diaries for over 1,000 infants born in 1991-92. 135, 136 At eight months, 34 per cent of the infants had not eaten any fruit and 25 per cent had not eaten vegetables in the three-day study period, while 26 per cent of the infants had eaten chocolate. At 18 months, 16 per cent of the children had not eaten fruit and 8 per cent had not eaten vegetables in the three-day period but 63 per cent of the children had eaten chocolate and 66 per cent had eaten savoury snacks. Although intakes of most nutrients were above the reference nutrient intakes (considered adequate for 97% of children), vitamin D intakes were low at both ages, and iron intakes were low at 18 months.

The ALSPAC study also showed that at 18 months, the most common container for drinks was a trainer cup. Eighty-six per cent of children had at least one drink from a trainer cup over a 24-hour period, 64 per cent of children were given at least one drink in a bottle, and 10 per cent solely used a bottle. Use of a bottle was more common among children who had not been breastfed, who had older siblings, and who had mothers who smoked. 137 Children being fed milk in a bottle consumed greater volumes when compared with children given milk in a cup; this was particularly marked for the children only fed by bottle. A wide variety of drinks were given at this age, including low calorie squashes (48%), tea and coffee (17%), and fizzy drinks (7%). Maternal education was the most important factor associated with the types of drinks consumed at this age – more highly educated mothers gave their children fruit juices more often, whereas women with low levels of educational attainment were more likely to give their children tea, coffee and soft drinks. Seventeen per cent of children whose mothers had Certificate of Secondary Education (CSE) qualifications or less were given fizzy drinks over a 24-hour period.

More recently the dietary patterns of 1,434 infants born to women in the SWS between 1999-2003 were reported. 138 The most important pattern of diet identified at both six months and 12 months of age was consistent with current infant feeding guidance, and was characterised by high consumption of vegetables, fruit, meat and fish, and other home-prepared foods. This dietary pattern was labelled the 'infant guidelines' pattern. Infants who had high scores for this pattern of feeding were also more likely to have been breastfed. A number of factors, including a range of maternal and family characteristics were considered as influences on the infants' pattern scores. At both ages the key determinant of the nature of the infant diet was the quality of the mother's diet. Infants whose mothers had high prudent diet scores and 'healthier' dietary patterns, characterised by high intakes of fruits and vegetables, and wholemeal bread, 139 were more likely to have a comparable diet, and were fed in accordance with infant feeding guidelines. Conversely, mothers who had low prudent diet scores and whose diets were characterised by high intakes of white bread and chips, added sugar and full-fat dairy products, were less likely to provide a diet for their infant that conformed with infant feeding guidance (see Figure 10). The influence of mother's dietary pattern on her infant's diet was independent of a range of other factors, including her educational attainment and smoking status.
Key message

In comparison with milk feeding, less attention has been given to the introduction of nutritious weaning foods. It is known that the quality of the weaning diet for UK infants is strongly influenced by the socio-economic status and educational attainment of the mother.

Figure 10: 12-month ‘infant guidelines’ pattern score according to prudent diet score of the mother


Children’s diets and food preferences have been shown to be influenced by their food environment, and by the eating behaviours of their parents. Children’s diets and food preferences have been shown to be influenced by their food environment, and by the eating behaviours of their parents. The SWS provided evidence that influences of the food environment on children’s diet operate very early – as soon as infants start to eat solid food. The findings also suggest that interventions to change mothers’ diets will have direct consequences for the diets of their children.

Key message

Influences of the food environment – including maternal diet – operate very early on children’s diets, from weaning onwards. Interventions to change mothers’ diets will have direct consequences for the diets of their children.
Developing country perspective on complementary feeding

Poor-quality complementary feeds are the norm for infants in disadvantaged families and a major cause of infant stunting. Programmes to support breastfeeding, promote better complementary feeding and supply micronutrient supplements, are some of the most effective measures that could be taken to reduce undernutrition in these countries.123

Guidelines on infant feeding

Breastfeeding guidelines

In 2003, the World Health Organisation (WHO) recommended that all babies should be exclusively breastfed for the first six months of life (the previous recommendation was four to six months). Thereafter complementary feeding should be introduced, with breastfeeding continuing as long as possible up to two years. The evidence underlying the advice to extend exclusive breastfeeding from four to six months is persuasive in developing countries, but less so for high-income countries.143-145 Breastfed babies, even in high-income countries, experience significantly fewer episodes of gastro-intestinal and respiratory infection.146 Benefits to the mother of breastfeeding include convenience, less expense, a faster return to pre-pregnant weight, delayed onset of ovulation, and possibly long-term protection against breast and ovarian cancer.

Key message

Breastfed babies, even in high-income countries, experience significantly fewer episodes of gastro-intestinal and respiratory infection compared with formula-fed babies.

The National Service Framework for Children, Young People and Maternity Services highlights the importance of breastfeeding, and recognises that it is a skill that needs to be taught and supported. The DH produces a booklet for health professionals on how best to do this entitled Good practice and innovation in breastfeeding.147

More recently NICE148 have produced guidance on improving the nutrition of pregnant and lactating mothers from lower socio-economic groups as a way of targeting those most in need of advice and support.

Maternal diet during lactation guidelines

For most women in the UK, a normal healthy diet is adequate to support breastfeeding. The Food Standards Agency (FSA) provides specific dietary advice for lactating mothers.150 Vitamin D supplements (10 micrograms daily) are recommended. Mothers are advised that fish is good for their own health and for the development of the baby, but to limit the intake of oily fish to two portions per week because of mercury levels. If there is a family history of peanut allergy, women are advised to avoid eating peanuts during lactation.151
Developing country perspective on maternal diet during lactation

Maternal micronutrient deficiency during lactation (especially for the B vitamins, vitamin A and iodine) can lead to low levels in breast milk. Even undernourished mothers in developing countries can, however, usually breastfeed successfully, and milk quality appears to be largely unimpaired.

Box 8: Contraindications for breastfeeding

There are some circumstances in which breast milk can contain harmful agents, such as HIV, environmental pollutants (e.g. dichlorodiphenyl trichloroethane (DDT), polychlorinated biphenyls (PCBs), dioxins, radiation), stimulants (caffeine, nicotine), drugs and food allergens. The few contraindications to breastfeeding include galactosaemia in the infant, psychotropic drug abuse by the mother and active maternal tuberculosis. In the UK, HIV-positive women are advised not to breastfeed because of a risk of transmission to the baby. This advice is not appropriate in countries where the risks of bottle-feeding are high, especially because of the lack of clean safe water, and outweigh the risk of HIV infection. Some therapeutic drugs are contraindicated during lactation or require monitoring: antineoplastic drugs and radioactive substances, retinoids, iodides, lithium, phenindione, tetracyclines, chloramphenicol and amiodarone. Drugs commonly used by UK mothers during lactation include analgesics, antibiotics and anti-depressants and for most of these, concentrations in breast milk are too low to cause problems. The British National Formulary (BNF) has a section on safety of drugs during breastfeeding. Several reviews have also been published concerning this topic.

Formula feeding

If mothers cannot or choose not to breastfeed, there is a range of commercially available formula milks designed as total substitutes for breast milk during the baby’s first year. They are the only safe alternatives to breast milk and infants can grow and develop normally on these feeds. The content of infant formula is tightly regulated. There is an international code, legally binding in the UK, governing its marketing, to prevent mothers from being encouraged to formula-feed (see Appendix 5).

Formula milk has changed considerably over the past 70 years and there are frequently new compositional changes. Standard formula is based on skimmed cow’s milk, with whey protein, vegetable oil and extra vitamins and minerals added to bring the composition closer to human milk. Formula does not contain most of the bioactive factors present in human milk, the protein content is higher and different in quality, and the concentrations of most vitamins are higher (see Appendix 6).

Several specific additives, which are present in breast milk but low in cow’s milk, have been introduced to some formula milks recently and may be unfamiliar to many professionals. These include amino acids such as taurine; long-chain polyunsaturated fatty acids (LCPUFAs) such as docosahexaenoic acid (DHA); oligosaccharides; and pre-formed nucleotides. Docosahexaenoic acid is a major constituent of neuronal cell membranes and there is some evidence that newborns, especially pre-term babies, cannot synthesise adequate quantities for optimal brain development, although this theory is controversial. Oligosaccharides are analogous to cell surface receptors for pathogens and are thought to inhibit the pathogenicity of bacteria. In infant formula they are
known as prebiotics (distinct from ‘probiotics’ or so-called ‘friendly’ bacteria, which are not currently added to formula). Pre-formed nucleotides are thought to be required for the growth of rapidly dividing cells in bone marrow and intestinal epithelium, and also to enhance beneficial intestinal bacterial growth. There is currently no strong evidence that these additives have health benefits.

**Complementary feeding**

Complementary feeding is the term used for the introduction of foods other than milk (sometimes called weaning foods or ‘solids’). It is recommended that complementary feeds are introduced from the age of six months onwards, and that infants progress onto the same foods as the rest of the family by around one year. The optimal diet for infants at this stage is not known, and is certainly not ‘innate knowledge’ for parents. Detailed guidelines are available on suitable foods at every stage (see Appendix 4). Key recommendations are a varied diet, adequate energy density, high-quality (preferably animal) protein sources, and fresh fruit and vegetables.

**Vitamin supplements for infants**

Advice in the UK is that healthy breastfed infants under six months do not require supplements provided the mother has adequate vitamin status. If there is any doubt about this, supplements should start at one month. From six months, infants receiving breast milk as their main drink should have vitamin A and D supplements, but these are not required by infants on formula milk, which is fortified with vitamins. Pre-term infants are a special case and require supplements from birth. Between one and five years, it is recommended that all children have vitamin A and D supplements unless their diet is diverse and plentiful and there is good exposure to sunlight. There are many preparations available. Supplementary vitamins are available free for families receiving certain benefits under the Healthy Start scheme, and all families can buy these preparations.

**Developing country perspective on complementary feeding**

Infants in developing countries are at increased risk of undernutrition after six months, when breast milk alone is no longer nutritionally adequate but complementary foods are frequently of very low nutritional quality. The WHO has developed guidelines for infant feeding which are suitable for use in all countries (see Appendix 4). Educational interventions and supplementation programmes, designed to improve the quality of complementary feeding, have been shown to reduce or prevent stunting in vulnerable infants.

**Determinants of infant weight gain and growth**

A characteristic of the healthy infant is rapid, though also rapidly decelerating, weight gain and linear growth, but the factors which determine these processes and the relative accrual of lean and fat body mass are still poorly understood. Given adequate postnatal nutrition, babies who experienced intra-uterine growth restriction due to placental insufficiency, small maternal size, maternal undernutrition or smoking tend to show ‘catch-up’ weight gain and growth, while macrosomic babies of mothers with gestational diabetes ‘catch-down’. Early postnatal growth rates therefore compensate for intra-uterine restriction or enhancement of growth. Boys gain weight and grow slightly faster than girls in early infancy. Weight gain and growth are modified by the type of feed used. Formula-fed babies gain weight and length faster than breastfed babies after the age of three to four months (see Figure 11). Small-for-gestational-age (SGA) babies grow faster on enriched than on standard formula. Infant feeding is largely demand-driven in that mothers tend to offer feeds when the baby appears to want them, and appetite is likely to be one of the factors influencing weight gain. Recurrent infections are a major cause of poor infant weight gain and growth in developing countries.
Figure 11: Mean weight and length of breastfed and formula-fed infants from birth to 18 months

Karlberg described postnatal growth in three distinct phases – infancy, childhood and puberty. During infancy the fetal growth hormones, such as insulin and the insulin-like growth factors (IGFs) play a more important role than growth hormone. From six to 12 months, growth hormone starts to have a significant effect on growth and becomes the main endocrine growth regulator during childhood. Nutritional status, especially during infancy, interacts with these hormones, and both circulating IGF-1 concentrations and receptors are reduced in undernutrition. Thyroid hormone is required for normal infant growth. In early infancy, gonadotrophin concentrations are high, and testosterone concentrations in boys are raised; they fall over the first six postnatal months to very low levels that persist until the onset of puberty. There may be mild elevation of oestrogen concentrations in girls in the neonatal period.

The reasons for differences in weight gain and growth between breastfed and formula-fed babies are not known. Formula milk contains more protein than breast milk, which may alter growth factor concentrations. The greater control that the infant exerts over the flow of milk, and the ‘supply and demand’ interaction between infant and mother, may influence appetite, satiety and energy intake differently from formula-fed babies. ‘Inadequate growth’ is often a reason for mothers deciding that they have insufficient milk, and changing to formula feeding (see pages 28 and 29). There are also numerous social factors influencing parents’ infant feeding choices. This complex interplay between biological and societal influences makes it difficult to disentangle the factors that control infant growth.

Key message

‘Inadequate growth’ is often a reason for mothers mistakenly deciding that they have insufficient milk, and changing to formula feeding.

Recommendations about size and growth during infancy

Growth monitoring is one of the prime activities of health visitors and doctors looking after infants in the UK. The aims are to detect inadequate weight gain or growth (‘failure to thrive’), which may indicate illness or problems with feeding or diet, or excessive weight gain. Research suggests that it is important to parents that their child is ‘normal’ in size or growth and a belief that an infant is not gaining weight normally is one of the commonest reasons mothers give for stopping breastfeeding. The growth charts currently in general use in the UK are based on the 1990 UK reference data. These came from a study of fetal growth in white Caucasian pregnancies in London, and Cambridge infants taking part in a study of the effects of infant feeding practices on growth.

In 2006, the WHO launched a new growth reference, which is now recommended for use worldwide. There were a number of reasons for developing the new reference. Formula-fed infants gain weight more rapidly after the first four to six months than breastfed infants and studies had shown that the weight gain pattern of breastfed infants deviated considerably from most current reference data. An optimal reference was considered to be that based exclusively on breastfed infants. Infants from all ethnic groups can grow equally well, given an optimal environment and adequate nutrition, and a unified standard would have benefits for comparing data from different populations. The WHO has defined the new reference as a standard rather than a reference, ie a description of how children should grow. The infants from which it was derived lived in conditions ‘favourable to child growth’: they were singletons born at full-term without major neonatal morbidity; had no known health or environmental constraints to growth;
the mothers were willing to breastfeed exclusively for at least four months continuing until 12 months, and to introduce complementary feeds by six months; and none were smokers. The data were collected using standardised techniques in the USA, Oman, Norway, Brazil, Ghana and India in 1997-2003. One concern about the generalisability of these standards is the wide international variation in birth weight. Mean maternal height and birth weight in Oman and India were much lower than in Norway and the USA, indicating maternal constraint on fetal growth in these developing countries, which is also likely to be associated with effects on postnatal growth, inducing more rapid catch-up growth.

**Box 9: Comment on the WHO infant growth reference standard**

‘The most interesting thing about the study that gave rise to these standards is that despite being based on very diverse populations around the globe, they produced very similar growth curves, indicating that genetics plays a very small part in determining growth rates compared with environmental factors which were optimised for this study – healthy pregnancy, good mother and child nutrition, no smoking, full breastfeeding etc.’

Tim Lobstein, Director Childhood Obesity Research Programme

A joint expert group from SACN and the Royal College of Paediatrics and Child Heath (RCPCH) met to consider the routine use of the WHO standards in the UK. They concluded that UK infants would appear large at birth and show apparent marked catch-down growth during the first few weeks, which could discourage mothers from breastfeeding. Using the WHO standards would, however, reduce the number of UK infants showing weight faltering, and thus being classified as underweight, after four months. They found little difference in length between the UK 1990 and WHO references at any age, and there was convergence of weight in the two references from around two years onwards. The group advised adoption of the WHO Growth Standard for routine use from two weeks to 24 months, while retaining the UK 1990 reference from 24 months onwards. Since the WHO reference population did not include pre-term infants, the UK 1990 reference would also need to be retained for these infants. They recommended piloting of the new charts, training for health professionals, and a well-planned communications strategy preceding implementation. It is not clear when all this will happen, but it is unlikely that the standard will be in general use before 2009.

**Key message**

Infant growth-charts currently in general use in the UK (based on 1990 UK reference data) are now considered inadequate because weight gain patterns of breastfed infants deviate considerably from such data. Old growth charts should be replaced by the 2006 WHO new growth reference – based exclusively on breastfed babies – for assessment all infants aged between two weeks and 24 months.
Breastfeeding and health: short- and long-term outcomes

Even in developed countries, with modern breast milk substitutes and easy access to clean water for preparation, breastfeeding is associated with fewer infections, especially gastroenteritis and respiratory infection, and possibly otitis media. In one UK study, babies who were fully or partially breastfed for 13 weeks or more were seven to 17 per cent less likely to have a gastro-intestinal illness and were 70 per cent less likely to be admitted to hospital with gastro-intestinal illness. Significant reductions in gastro-intestinal illness in this group were found up to one year of age.

McCance and Widdowson demonstrated in animals over 40 years ago that early postnatal nutrition has long-term effects. They showed that by manipulating litter size at birth, rat pups could be transiently overfed or underfed during lactation; reducing the litter size resulted in more rapid early growth and larger adult size. Later overfeeding did not have this effect, suggesting that there is a critical period within which postnatal nutrition permanently influences growth and body composition.

Most research into long-term effects of infant feeding in humans has compared rates of various health outcomes between children or adults who were breastfed or formula-fed. Despite a wealth of literature, there is still much that we do not know. The ability to combine infant feeding data with adult outcomes means that a dataset must be quite old, dating back to when bottle feeds were very different from those in use today. Information about feeding is usually sparse, recording whether or not a baby was initially breastfed, and sometimes the duration of breastfeeding and/or the age at which solids were introduced. Collating evidence across studies can be difficult because of the different ways of describing infant feeding. There is no information in these studies on the quality and quantity of breast or bottle milk. A major problem in interpreting these studies is that of confounding. A mother’s choice to breastfeed is related to many other factors that could influence the later health of the child, especially her socio-economic status and education (see page 30). These influences change with time. Adjusting for these factors, to isolate the ‘biological’ effects of early feeding, is an imperfect science.

Randomised trials would be helpful, but it is clearly impossible to randomise healthy babies to be breastfed or formula-fed. A recent trial in Belarus in which mothers attending different health clinics were randomised to receive or not receive intensive breastfeeding education and support, leading to greatly increased rates of breastfeeding in the former group, is likely to yield useful data. Lucas and colleagues have carried out randomised trials of different infant feeds among babies born pre-term or SGA (see page 41). The ongoing European Childhood Obesity Project is carrying out a series of randomised trials in babies whose mothers have decided to bottle-feed, testing the effects of different protein intakes from formula milk.

Developing country perspective on breastfeeding and health

Breastfeeding is a lifesaver in developing countries, where formula feeds are often by necessity made with unsterile water and fed from unsterile bottles and teats. A recent review estimated relative risks for diarrhoea incidence and mortality under six months at 3.0 (95% confidence interval (CI) 1.3 to 7.0) and 4.6 (95% CI 1.8 to 11.8) for partially breastfed infants and 3.7 (95% CI 1.7 to 7.9) and 10.5 (95% CI 2.8 to 39.6) for non-breastfed infants, compared with exclusively breastfed babies. Estimates for pneumonia incidence and mortality were 2.5 (95% CI 0.2 to 27.2) and 2.5 (95% CI 1.0 to 6.0) in partially breastfed infants and 2.1 (95% CI 0.2 to 22.6) and 15.1 (95% CI 0.6 to 373.8) in non-breastfed infants. The same review estimated that sub-optimal breastfeeding causes 1.4 million preventable deaths (12% of under-five deaths in developing countries) and 43 million disability adjusted life years (DALYs).
Breastfeeding and later cognitive function and neurodevelopment
A large number of studies have compared neurodevelopmental scores and visual function between children who were breastfed and those who were formula fed. A consistent message is that at all ages children who were breastfed score an average two to eight developmental quotient points higher and/or have better visual function. Several studies report dose-response effects with duration of breastfeeding. The size of these effects is small, and unlikely to make a difference to an individual child, although on a population scale they could be important. The issue of confounding is especially important for cognitive outcomes, because mothers who breastfeed tend to be more affluent and better educated than women who formula-feed. In a large recent study, it was shown that the mother’s intelligence quotient (IQ) predicted her likelihood of breastfeeding even more strongly than these factors. A one standard deviation advantage in maternal IQ more than doubled the odds of breastfeeding. Before adjustment, breastfeeding was associated with an increase of around four points in childhood cognitive ability, but when adjusted for a range of confounders including maternal IQ, the effect was non-significant (+0.5 points, 95% confidence interval -0.2 to +1.2).

There are however, plausible biological reasons for better cognitive development in breastfed babies. The close contact and interaction between mother and baby may enable more visual, social and motor stimulation in early infancy. Constituents of breast milk could favour brain development. The LCPUFAs, DHA and arachidonic acid (AA) are required in large quantities in the developing brain and retina. They have not been added to formula milk until recently (~2002 onwards) and are not present in all formulas. DHA can however, be synthesised from precursors in formula, and there are only small differences in brain DHA content between breastfed and formula-fed infants. It is not yet known how important these differences are in functional terms. They may be more important in pre-term babies, since accumulation of DHA in the fetal brain occurs mainly in the last trimester of pregnancy. Randomised studies in pre-term infants, comparing banked breast milk with pre-term formula (both given by nasogastric tube) showed that breastfed infants had an eight point IQ advantage at eight years, even after adjustment for confounding factors.

Key message
There is consistent evidence of better cognitive function in children who were breastfed. There is controversy as to whether this is a direct effect, or the result of ‘confounding’ resulting from the fact that mothers who are more intelligent and/or better educated are more likely to breastfeed.

Breastfeeding and later body composition and obesity
There are several recent systematic reviews of studies comparing indices of obesity between children and adults who were breastfed or formula-fed. BMI, which does not distinguish between fat and lean body mass, was the main outcome in most studies, but some used specific measures of adiposity such as skinfold thickness or dual-energy x-ray absorptiometry (DXA) measurements.

Breastfeeding is associated with a lower mean BMI in later life in most studies. The difference is small (-0.04 kg/m^2 [95% CI -0.05 to -0.02]) but the lower incidence of obesity (BMI >30 kg/m^2) is more impressive (odds ratio [OR] 0.87 [95% CI 0.85 to 0.89]). Several studies have shown a dose-response relationship, with longer duration of breastfeeding associated with lower rates of obesity. Again, there are major confounding issues, because the choice to breastfeed is associated
with less ‘obesogenic’ family characteristics.\textsuperscript{191, 192} A recent study showed however, that fat mass assessed using DXA at age nine to 10 years was inversely related to the duration of breastfeeding and this association, although attenuated, was robust to adjustment for a range of confounding factors.\textsuperscript{192} On the other hand, there were no differences in skinfold thickness at six years of age between children in the two arms of the Belarus trial.\textsuperscript{180}

Mechanisms that could protect breastfed babies from later obesity include aspects of the feeding process or particular constituents of breast milk. As already mentioned, breastfed infants may learn to regulate their energy intake more effectively. Breastfed babies grow more slowly in infancy than formula-fed babies, which may be protective. Constituents of milk, such as protein, may influence body composition through effects on hormones such as insulin and IGF-1.\textsuperscript{127, 193}

**Key message**

Breastfeeding is associated with a lower mean BMI in later life, and a longer duration of breastfeeding is associated with lower rates of obesity. Breastfed babies grow more slowly in infancy than formula-fed babies, which may be protective against the later development of obesity. There is also evidence that breastfeeding is associated with a lower risk of both type 1 and type 2 diabetes and some forms of cancer, and lower LDL-cholesterol concentrations in childhood and adulthood.

**Diabetes**

Type 1 diabetes is an auto-immune disease in which antibodies are formed against components of the pancreatic beta cells, leading to insulin secretory failure. It usually presents in childhood or adolescence. Case-control studies have shown a 30 to 50 per cent reduced risk of type 1 diabetes in children who were breastfed, but more evidence is required in this area. It has been suggested that the avoidance of cow’s milk formula is important rather than breastfeeding per se, because there is structural similarity between bovine albumin and beta cell surface proteins, and children presenting with type 1 diabetes have elevated antibodies to bovine albumin.\textsuperscript{194}

Type 2 diabetes is a common disease and, until recently, usually presents in middle-to-old age. It is strongly associated with obesity, and the underlying cause is insulin resistance, which imposes an increased demand for insulin that eventually ‘exhausts’ the pancreatic beta cells. The obesity epidemic in Western countries has led to increasing numbers of children presenting with type 2 diabetes. It has been estimated that there could be 1,400 children with type 2 diabetes and 20,000 with pre-diabetes (impaired glucose tolerance (IGT)) undetected in the UK.\textsuperscript{195} There is some evidence that breastfeeding protects against the risk of developing type 2 diabetes in later life, possibly through effects on adiposity. A meta-analysis of seven studies showed a reduced risk in six (overall OR 0.61; 95% CI 0.44 to 0.85; p=0.003).\textsuperscript{196} This was little changed after adjusting for confounding factors (maternal size, birth weight, parental diabetes, socio-economic status and adult body size).
Blood pressure and cardiovascular disease

There were no differences in blood pressure at six years of age between children in the two arms of the Belarus trial. Two recent systematic reviews found that breastfeeding was associated with a small reduction in blood pressure of 1-1.5 mmHg (systolic) and 0.5 mmHg (diastolic) in observational studies. Effect estimates were lower after adjusting for confounding. Although small in individual terms, an effect of this size at a population level could translate into significant reductions in hypertension and CHD. A meta-analysis of the few studies that have related infant feeding to cardiovascular disease found no evidence of a lower incidence in men and women who were breastfed. There was some evidence that prolonged breastfeeding (for more than a year) was associated with a small increase in adult cardiovascular disease mortality in this analysis, and with increased arterial stiffness in one other cohort study.

Other cardiovascular risk factors

There are few data for other risk factors such as serum lipid concentrations and inflammatory markers. A meta-analysis found that breastfed babies have higher serum cholesterol and low-density lipid (LDL) cholesterol concentrations in infancy itself (probably because of the higher cholesterol content in breast milk) but concentrations were lower in adult life by -0.18 mmol/l (CI -0.3 to -0.06). This difference would be associated with an approximate 10 per cent reduction in CHD risk. Consistent with these findings, in a follow up study of pre-term infants randomly assigned to banked breast milk or pre-term formula, children in the former group had lower LDL cholesterol and apolipoprotein B concentrations. A recent study of 8,000 members of the 1958 UK birth cohort, whose infant feeding was recalled by the mother at seven years, and in which it was possible to adjust for confounding factors, showed that a longer duration of breastfeeding was associated with lower plasma fibrinogen, plasminogen activator inhibitor and von Willebrand factor, although the effects were small.

Cancer

Higher birth weight and taller adult height are associated with an increased risk of some cancers, suggesting effects of nutrition in early life. This may reflect exposure to hormones promoting early growth. A recent meta-analysis suggested reduced rates of pre-menopausal breast cancer among women who were breastfed in infancy, but found no evidence of an effect on overall cancer incidence or other specific adult cancers. There may be small reductions in some childhood cancers, including acute lymphoblastic leukaemia, Hodgkin's lymphoma, and neuroblastoma, but data are limited.

Allergic/atopic disease

The incidence of allergic disease in infancy and childhood (wheezy bronchitis, asthma and atopic dermatitis or eczema) has increased recently in many Western countries including the UK. Several studies have suggested a protective effect of breastfeeding, but systematic reviews show that the findings are inconsistent. Infants at high risk because of a family history of allergic disease do seem to benefit from exclusive breastfeeding for three to four months. Soya-based formula does not protect against atopic disease, but there is some evidence that hydrolysed formulas are protective compared with formula containing intact bovine protein.

Mental health

One of the proposed benefits of breastfeeding is increased bonding between mother and baby, and an increased sense of security that could promote psychosocial resilience and reduce mental illness. This is a remarkably poorly studied outcome. Fergusson found no evidence of better psychosocial adjustment in adolescents who were breastfed, while another small study suggested lower anxiety levels in response to parental separation in breastfed children.
**Other outcomes: bone health, coeliac disease, inflammatory bowel disease**

Breastfed infants, consistent with their smaller size, have lower bone mineral content (BMC) than formula-fed infants. The calcium and phosphate content of formula has been shown to influence BMC during infancy in randomised intervention studies. There are, however, no data on long-term outcomes such as adult bone density or fracture risk.

A systematic review found consistent evidence from six case-control studies that longer duration of breastfeeding, and breastfeeding at the time of introduction of gluten containing foods, protected against the later development of coeliac disease. There is inconclusive evidence of protection against inflammatory bowel disease.

**Other aspects of infant diet and long-term outcomes**

There are very few studies relating long-term outcomes to other aspects of infant diet, for example effects of different formulas, the age at which solids were started, the type and quality of complementary feeds, and the use of micronutrient supplements.

Lucas and Singhal have followed up children who took part in a series of randomised trials of different formula feeds as babies born pre-term or SGA. Among pre-terms those who received banked breast milk had the lowest blood pressures and 32-33 split proinsulin concentrations (an indicator of insulin resistance) at 13-16 years. Those fed enriched pre-term formula had the highest, and those fed standard formula had intermediate levels. Term SGA babies fed standard formula had lower diastolic blood pressures at six to eight years than those fed enriched formula.

The macronutrient balance of the infant diet may be important. Higher protein intakes in late infancy have been associated with greater adiposity in childhood. Another study suggested that animal protein (especially dairy protein), but not vegetable protein, was linked to later adiposity. The INCAP trial in Guatemala, randomised villages to receive one of two supplements for mothers and children: high-protein, high-energy drink (protein 6.4 g/100ml, energy 900 kcal/l), or a lower nutrient drink (energy 330 kcal/l, no protein). Both contained multiple micronutrients. People who received high-nutrient drink in utero (through their mother) and/or infancy (either through their mother’s breast milk or by direct intake) had lower adult triglyceride concentrations and higher HDL cholesterol concentrations.

Evidence linking the early introduction of solid feeds to allergic disease (eczema, asthma, and others) and obesity in childhood is inconclusive.

**The timing of introduction of solids and later health**

It has been suggested that introducing certain foods too early or too late during infancy increases the risk of developing food allergies and atopy (eczema and wheezing). The evidence base is not strong. In the UK it is recommended that certain foods are never introduced before six months (see Appendix 4).

Symptoms of food allergy and intolerance in infancy can be obvious (facial swelling, urticaria) or non-specific (conyza, wheezing, vomiting, diarrhoea, colic, blood in the stools). The commonest allergies are to cow’s milk protein, egg, soya, wheat, nuts and shellfish. Diagnosis is based on history, skin prick testing, the measurement of food-specific IgE antibodies, patch tests, and food challenge testing. The latter should be carried out using a blinded format, and placebo controls.
There is little evidence that adding probiotics (live non-pathogenic bacteria that colonise the gut) or prebiotics (nondigestible food components such as oligosaccharides that stimulate the growth of non-pathogenic bacteria in the gut) to infant feeds reduces later food allergy/intolerance.

**Infant growth and long-term outcomes**

There is strong and consistent evidence that undernutrition in infancy is associated with permanent adverse effects on growth and development. In a recent analysis of young adults from five developing countries, stunting and/or underweight at the age of two years were associated with reduced adult height, fewer years of attained schooling and lower adult income or assets. Among women, undernutrition in infancy was associated with lower birth weight in the next generation. These relationships remained after adjustment for childhood socio-economic status and parental education, suggesting that they were related to infant undernutrition, rather than simply a reflection of continuing disadvantage in later life. A literature review also found consistent evidence of impaired cognitive ability in children who had been undernourished (stunted or wasted) in infancy.

**Key message**

There is strong and consistent evidence that poor weight gain and growth during infancy are associated with permanent stunting and cognitive impairment, resulting in fewer years of attained education, and lower adult productivity and earning capacity.

The relationship of infant size and growth to later health outcomes in well-nourished populations is currently unclear and controversial. This is partly because of a paucity of data. Cohort studies old enough to hold data on both infant size and adult disease are few. In a recent systematic review relating size in infancy to the 12 diseases accounting for the highest burden of disease (ischaemic heart disease, stroke, depression, lung cancer, road traffic accidents, alcohol abuse, dementia, chronic obstructive lung disease, suicide, breast cancer and diabetes (split into type 1 and type 2)) only 19 good quality studies, relating to 10 of the disease outcomes, were found. The highest number of studies for a single disease was seven for type 1 diabetes, a disease with its onset mainly in childhood. Three of these seven studies showed a positive association between infant weight or BMI and type 1 diabetes risk. This is consistent with the data described previously showing a lower risk of type 1 diabetes in children who were breastfed.

Several studies of cardiovascular risk factors in children have shown higher blood pressure in those who gained more weight during infancy. In non-randomised analyses of Lucas’s infant feeding trials, SGA babies who gained more weight between birth and nine months had higher diastolic blood pressure in childhood and pre-term babies who gained more weight in the first two weeks had higher 32-33 split proinsulin concentrations and LDL/HDL ratios at 13-16 years. Greater weight gain in infancy is consistently associated with higher BMI, and in some but not all studies with greater adiposity, in later childhood and in adult life. These studies have led to the Growth Acceleration Hypothesis, which suggests that rapid weight gain in infancy, as well as in later childhood, is a risk factor for later cardiovascular disease.

In marked contrast to these data, retrospective studies of adult cohorts suggest benefits of infant weight gain. In the Hertfordshire cohort, men with a higher weight at one year had lower cardiovascular disease mortality (see Figure 12) and were less likely to have impaired glucose tolerance or type 2 diabetes. Men and women in Finland who developed CHD or type 2
diabetes were significantly lighter, thinner and shorter than average in infancy. In the New Delhi birth cohort in India, lower weight at one year predicted an increased risk of IGT or type 2 diabetes.

Figure 12: Cardiovascular disease mortality according to weight at one year in Hertfordshire men

![Graph showing cardiovascular disease mortality according to weight at one year in Hertfordshire men.](image)


Two of these three adult cohort studies had data on weight and BMI gain in childhood and adolescence as well as in infancy. Men and women with CHD or diabetes were light and thin as infants but gained weight more rapidly than average in later childhood (see Figure 13a and b) suggesting that infant weight gain and childhood weight gain may have completely different effects on later disease risk. There was no evidence that increased height at any stage of childhood was associated with adverse outcomes in adult life. The reasons for the different findings in the adult and children's studies are not known. One possibility is that the rapid infant weight gain seen in the Hertfordshire, Finland and Delhi babies reflected healthy weight gain in predominantly breastfed babies, whereas the rapid weight gain seen in recent studies of children reflected the effects of predominant formula feeding. Recent data suggest that breastfeeding attenuates the effect of rapid infant growth on adiposity at the age of two years.

Key message

While the long-term effects of rapid weight gain in infancy require further research, there is clear evidence that excessive weight gain in childhood and adolescence is associated with an increased risk of adult obesity, type 2 diabetes and cardiovascular disease.
Mean standard deviation (SD) scores for height, weight and body mass index in infancy (up to two years) and from two to 11 years after birth among boys and girls who had CHD as adults. The dotted line at zero represents the average for the whole cohort.

There is another paradox in the data from the older cohorts, which needs to be investigated. High BMI is a risk factor for cardiovascular disease and diabetes, but the relationships of infant weight gain to adult BMI and to adult disease are in opposite directions. The key to understanding this may be to obtain better information about body composition in infancy. The Delhi study showed that infant BMI gain was more strongly related to adult lean mass than to adult adiposity, while BMI gain in later childhood was strongly related to adult adiposity. Infant weight gain in Hertfordshire was associated with taller adult height and bone mineral content, which would also be associated with greater lean mass.

Key message

Several studies of cardiovascular risk factors in children have shown higher blood pressure in those who gained more weight during infancy. On the other hand, studies of adults have shown a lower risk of cardiovascular disease and type 2 diabetes in those who gained more weight during infancy. These apparently paradoxical findings need to be investigated further.

There remain many unanswered questions. Most people would agree that normal infant weight gain should be protected, but should it be actively promoted or should catch-up or rapid weight gain be actively prevented? Is the answer to this question the same in all populations? And how modifiable, in any case, is the weight gain of a healthy well-nourished and demanding infant?

These are important questions, because infancy is an ‘accessible’ stage of life when optimal nutrition could have lifelong benefits. In developing countries, paediatricians generally try to promote catch-up weight gain and linear growth in small babies. There is good evidence that this reduces infant mortality, improves growth and development and has lasting benefits on education and work attainment. In more affluent countries such as the UK, getting infant nutrition right is likely to be an important element of efforts to prevent obesity and common causes of death and disability such as cardiovascular disease and diabetes.

Key message

Getting infant nutrition right has lasting benefits for growth and development, and is important in the prevention of obesity and chronic disease in adult life.
Conclusions

- Infants have special nutritional requirements because of their rapid growth and development and vulnerability to infection. Optimal nutrition in infancy is essential for normal cognitive and physical development and may protect against obesity and chronic disease in adult life. Many parents need guidance to provide adequate nutrition for their children at this stage of life.

- Breast milk is the ideal food for babies in their first few months. Mothers need support in order to breastfeed successfully. There is consistent evidence of better childhood cognitive development, and a lower risk of several disease outcomes, including obesity and diabetes, in children and adults who were breastfed rather than formula-fed. It is not known whether these effects are causal or reflect the generally healthier lifestyles of the families whose mothers choose to breastfeed.

- The effects of the quality of complementary feeding on current and later health in countries such as the UK are largely unknown. There are wide variations in infant size, weight gain, linear growth and body composition. There is increasing evidence that these influence the risk of developing obesity, diabetes, cardiovascular disease and other health outcomes in later life.

- The optimal pattern(s) of infant weight gain in order to minimise the risk of obesity, cardiovascular disease and diabetes is however, not yet known. There is strong evidence that undernutrition (stunting or wasting) during infancy leads to impaired adult cognitive, physical and economic capacity, even if nutrition improves later in childhood.
Chapter 4: Influences on maternal and infant nutrition and opportunities for intervention

Interventions in maternal and infant nutrition – a global perspective

In May 2004, WHO published a report, *Global strategy on diet, physical activity and health*, which stated that ‘A life-course perspective is essential for the prevention and control of non-communicable diseases. This approach starts with maternal health and prenatal nutrition, pregnancy outcomes, exclusive breastfeeding for six months, and child and adolescent health. It reaches children at schools, adults at worksites and other settings, and the elderly and encourages a healthy diet and regular physical activity from youth into old age’. This was followed by the WHO report *Promoting optimal fetal development: report of a technical consultation*. Its recommendations included initiatives to:

- prolong the interval between menarche and first pregnancy (> four years)
- improve maternal nutrition prior to conception (BMI and micronutrient status)
- optimise weight gain and nutrition during pregnancy
- reduce malaria, HIV and smoking
- promote breastfeeding
- develop strategies to avoid gross mismatch between the developmental environment and childhood nutritional environment.

In March 2006, the World Bank produced a report entitled *Repositioning nutrition as central to development*, which stated ‘The emphasis of any programmes to combat poor nutrition should target pregnant women and children under two years of age’. This formed part of the World Bank’s attempt to calculate the cost of a poor start to life. This was based on the savings gained from moving an infant from a low birth weight (<2500g) to a normal (>2500g) category, under the headings of:

- reduced infant mortality
- reduced neonatal care
- reduced costs of infant/child illness
- productivity gain from reduced stunting
- productivity gain from increased ability
- reduction in costs of chronic diseases
- intergenerational benefits.

The total saving was $510 (US) for each low birth weight prevented. This is recognised to be an under-estimate and work is now ongoing to amend it to take into account newer perceptions. Additional factors that may increase the estimate include the fact that the risk of non-communicable disease is graded across the normal birth weight range and is not just a consequence of birth weight below 2500g, and that disease risk is also associated with high birthweight. Chronic non-communicable diseases now start earlier in life in many populations and the effects can be passed across more than one generation. The ‘discount’ model, which assesses the relative saving on later poor health by investing in preventative measures, may be too simple for calculating the real cost-benefit relation. Notwithstanding the need to obtain new estimates of the cost, influential economists agree that human development offers a very important time over which to intervene to promote later health.

Interventions designed to improve short-term outcomes

Many trials have tested the effect of nutritional supplementation in the mother (usually started in mid-late pregnancy) on birth weight. Few have assessed the effect of pre-conceptional supplementation on fetal growth but there are ongoing trials aimed at doing this. In an early trial in the Gambia, a high-energy biscuit for severely undernourished pregnant women increased birth weight by more than 130g, but most trials of energy and protein supplementation have shown only small increases in birth weight (around 50g overall). The same is true of multiple
micronutrient supplementation trials. Birth weight is a crude measure of fetal development, and may under-estimate effects on specific fetal tissues and organs. The Gambia high-energy biscuit trial, and a recent trial of multiple micronutrient supplementation in Indonesian mothers, reported reduced infant mortality but opposing results were found in two earlier trials in Nepal. More measurements of functional outcomes such as growth and risk factors for later disease are required in such trials.

**Developing country perspective**

The BMA report *Improving the health of the world’s poor* (2007), highlights the persisting problems of poverty and undernutrition in many developing countries and its effects on maternal, fetal and infant health. Young women often have little control over family finances and food purchases, and come last in the family hierarchy in terms of access to food. Babies born to undernourished women are more likely to be born pre-term and/or growth restricted. Sub-optimal breastfeeding practices and poor-quality complementary feeding frequently follow and set the infant on course to becoming a stunted child, with impaired physical development and cognitive ability. An especially adverse situation, in terms of adult health, comes from poor nutrition in early life followed by obesity in later life. This combination is increasingly common in developing countries and carries a high risk of adult hypertension, diabetes and cardiovascular disease. Of the eight United Nations Millennium Development Goals, Numbers 1 (Eradicate extreme poverty and hunger), 4 (Reduce child mortality) and 5 (Improve maternal health) directly relate to the issues discussed in this report. In addition Number 2 (Achieve universal primary education), and 3 (promote gender equality and empower women) are related goals. While progress towards these is being made, it is likely that many of them will not be met by the target year of 2015.

**Guidelines for improving the diets of young women in the UK**

There have been consistent dietary messages to adults over many years to encourage consumption of fruit and vegetables, starchy foods and oily fish, and to limit consumption of dietary fat, salt and added sugar. The recommendations for a ‘healthy’ diet were based on evidence of the role of diet in the aetiology of cardiovascular disease and some cancers, and recently specific advice on oily fish consumption, salt and vitamin D intakes has been updated. Dietary guidelines are comparable across the developed world. Current UK advice is depicted in the *Eatwell plate* model. While this advice may be widely understood, the most recent National Diet and Nutrition Survey (NDNS) shows that many adults do not comply with these dietary guidelines, and that the diets of young adults may be a particular cause for concern (see Box 10).
Box 10: The diets of young people: findings from the National diet and nutrition survey

- When compared with older adults, young men and women in the NDNS were more likely to consume ‘fast foods’, savoury snacks, and carbonated soft drinks, and less likely to eat wholemeal bread, whole grain cereals and oily fish.
- Although fruit and vegetable consumption was below the target of five daily servings for many adults, consumption was particularly low among young adults (1.3 and 1.6 servings per day in men and women aged 19 to 24 years respectively).
- In comparison with older age groups in the NDNS, younger men and women obtained the largest proportion of food energy from non-milk extrinsic sugars and had the lowest intakes of dietary fibre (non-starch polysaccharides), vitamin A, riboflavin, folate, vitamin D, iron, calcium, magnesium and zinc.

These findings show that poor quality diets are common among young people in the UK – raising the possibility that, for many young women, current patterns of diet and nutritional status could impact on their ability to meet the nutrient needs of future pregnancies.

There are currently few specific dietary recommendations for pregnancy (see Appendix 3). The most consistent dietary message to pregnant women in the UK is to increase folate intake before conception, and recent data show widespread awareness of the need for adequate folate status in pregnancy. Among mothers studied in the Health survey for England in 2002 who had planned their pregnancy, more than half (55%) reported taking dietary supplements or changing their diet prior to pregnancy to increase folate intake. Of mothers who increased their folate intake before conception, 61 per cent reported that they had done so at least three months before becoming pregnant. Use of folic acid supplements was uneven, however, and was less common among women of lower social status. The value of recommendations for preconception supplementation with folic acid may be limited due to the high rates of unplanned pregnancies in the UK.

Key message

Poor quality diets are common among young people in the UK – raising the possibility that, for many young women, current patterns of diet and nutritional status could impact on their ability to meet the nutrient needs of future pregnancies.

Apart from these specific recommendations for pregnancy, young women are encouraged to comply with general healthy eating advice before and during pregnancy. Given that so many young women do not follow current guidance, it is clear that it is not sufficient just to popularise dietary messages. To achieve change in dietary behaviours, the influences on food choice need to be considered and understood.
Influences on food choices in women

A vast array of factors influence food choice. They include cultural, social and environmental factors, as well as knowledge and understanding of the role of food in health and disease. In a recent study of 6,125 women aged 20 to 34 years living in Southampton, compliance with current healthy eating recommendations was described using a score for a ‘prudent’ dietary pattern. Women with high scores had diets characterised by a high consumption of fruits, vegetables and wholemeal bread while women with low scores had diets characterised by a high consumption of white bread and chips, added sugar and full-fat dairy products. The most important influence on the prudent diet score was the educational attainment of the woman, such that lower scores were much more common among women with few educational qualifications (see Figure 14). This influence was far more important than any other factor considered, including the woman’s social class, the deprivation score of her neighbourhood, or whether she was in receipt of financial benefits.

Figure 14: Percentage of women with ‘prudent’ diet scores in lowest quarter of distribution, according to their educational attainment: 6,125 women in the Southampton Women’s Survey


Although cost is a recognised barrier to eating healthily the less prudent diets observed in this study did not seem to be simply a result of lower income.

The relationship between income and diet is complex. The low income diet and nutrition survey (LIDNS) published in 2007 yielded a positive view of the diets of people living on low incomes in the UK, since in comparison with the general population, the types and quantities of many foods eaten and patterns of nutrient intake were similar. Consumption of fruit and vegetables by low-income participants however, was lower, and consumption of soft drinks, processed meats, whole milk and sugar was greater. Food security was also assessed in this study; 39 per cent of the low-income population reported that they had been worried that their food would run out before they got money to buy more in the past year and 36 per cent said that they could not afford to eat...
balanced meals. Although the diets of children in food insecure households were comparable with those of children in food secure households, this was not the case for women. Women in food insecure households had typically less healthy diets, with lower consumption of fruit, wholemeal bread, meat and meat dishes and fish and fish dishes. Drewnowski and Specter describe an inverse relationship between the energy density of foods and energy cost – so that high-fat energy-dense diets may be more affordable than prudent diets based on lean meats, fish and fresh fruit and vegetables. This may partly explain the link between food insecurity and greater levels of obesity observed in some studies, particularly among women.

Key message

Educational attainment is a key influence on the quality of young women’s diets.

While a number of studies have examined the associations between diet and socio-demographic factors, there are important environmental and contextual influences on food choice that are less commonly considered. These include:

- the effects of the physical environment, such as the distance between home and shops that may determine the accessibility and affordability of foods, and the ability to store and cook food at home
- the nature of the contexts within which food is bought and eaten that may impact directly on purchasing patterns and foods consumed
- structural differences in families and households that define the nature of shared meals
- the wider influences of food advertising and the promotion of foods.

Increases in fruit and vegetable consumption have been observed following the building of new superstores in areas of poor retail provision, and purchasing patterns of confectionery can be changed by altering its location in a cafeteria. Interventions to change aspects of the food environment, so that consumers are encouraged to choose healthier foods, may offer important opportunities to achieve change in eating habits.

The choice of foods eaten is influenced by individual factors. Some of these have been extensively researched with a view to predicting and understanding food choices. They include variations in personal taste preferences, differences in knowledge, attitudes and beliefs as well as psychological factors such as matters of control and self-esteem.

There are complex interactions between socio-demographic, environmental and individual influences that act to determine food choice – and these influences must also underlie the reasons why current dietary recommendations are not met. To address the dietary and nutritional inequalities that exist across the general population we need to identify the primary influences on food choice. With this understanding, effective interventions can be defined to achieve dietary change.
Interventions to improve maternal nutrition

Box 11: Comment on nutritional intervention in women living in poverty

‘In the UK the health and wellbeing of millions of women are influenced by living in poverty. Food choices, dietary intake and feeding behaviour are far from optimal among poorer women, and this situation is reflected in higher rates of diet-related morbidity and mortality well beyond maternal and child health considerations. Interventions to change diet need to take account of the factors that influence household economic status and should be integrated at policy, community and individual level. There is a dearth of research on effective interventions in this subgroup of the population from within the UK, although there are some encouraging United States’ (US) examples to draw on in terms of possible intervention approaches.

Lack of evidence does not mean that policy work should be delayed. It is recognised that engaging with ‘hard to reach’ groups of women is a challenge for intervention implementation and evaluation, and it is essential that policy work should be evaluated for its ability to engage with target groups as well as for behavioural change and health outcomes.’

Professor Annie Anderson, Professor of Food Choice University of Dundee (2007)

A systematic review of interventions that aimed to improve diet in women who were pregnant or of childbearing age suggested that interventions that included elements of education or counselling, support and empowerment can improve nutrition knowledge and behaviour. Much of the existing evidence on interventions to improve maternal nutrition comes from evaluation of the federally funded US health programmes. These include the US Special Supplemental Nutrition Programme for Women, Infants and Children (WIC) and the Expanded Food and Nutrition Education Programme (EFNEP) both of which focus on people from disadvantaged backgrounds. Evaluation suggests that the WIC programme which combines food supplementation, nutrition counselling and referral to health and social services, can lead to improvement in maternal diet during pregnancy, increased maternal weight gain and increased breastfeeding rates. WIC programmes have also been effective at increasing fruit and vegetable consumption. EFNEP, which aims to give low-income families food knowledge and skills and to bring about dietary behaviour change through practical sessions delivered by peers, has been shown to improve food practices among recipients. Evaluation of smaller-scale intervention programmes within the UK has also suggested that imparting food knowledge and skills to women from disadvantaged backgrounds has the potential to improve diet in this group. The Cookwell project, set in eight urban communities in Scotland aims to improve diet in areas of social deprivation by delivering food skills sessions each week over a seven-week period: in a study of 113 adults living in areas of social deprivation, 88 per cent of whom were women, the intervention led to early increases in fruit consumption and at six months follow-up there was a significant increase in the proportion of subjects who reported confidence in following recipes and cooking from basic ingredients.'
Key messages

US programmes which combine food supplementation, nutrition counselling, cookery skills and referral to health and social services, can lead to an improvement in maternal diet during pregnancy and to increased breastfeeding rates.

Two recent systematic reviews have considered the effectiveness of interventions which aim to increase fruit and vegetable consumption in adults and the findings of both have relevance for women of childbearing age. The first focused on community interventions that were effective in increasing fruit and vegetable consumption in people aged four years and over. Fifteen studies, all of which were based in the USA, were included in the review. Four trials were specifically targeted at parents with young children. The most effective interventions gave clear messages about the benefits of fruit and vegetables, were intensive and used multiple strategies delivered over a prolonged period of time, and involved families as a group in the intervention. The second, and more recent, systematic review collated evidence from the published and grey literature relating to programmes that promote fruit and vegetable consumption in adults. Forty-four studies were reviewed, with over 70 per cent set in the USA and around 16 per cent in Europe. Consistent positive effects were seen in studies involving face-to-face education or counselling. A more recent US intervention study of 269 adults from low-income backgrounds evaluated the impact of the Sisters in health nutrition education programme which aimed to increase fruit and vegetable consumption among low-income groups. The intervention, which involved peer-led small group sessions to teach cooking skills and provide social support, led to significant improvements in fruit and vegetable intake at the end of the intervention programme.

Key message

There is limited evidence of interventions that are effective in improving maternal nutrition. There is, however, some evidence that interventions that include elements of education or counselling can bring about improvements in nutrition knowledge and behaviour.

Improving breastfeeding initiation and increasing duration of breastfeeding

Women surveyed in the most recent IFS were asked about their experience of breastfeeding; 73 per cent of the women who initiated breastfeeding, but who gave up in the survey period, said that they would have preferred to have continued to breastfeed for longer. The most common reason given for stopping breastfeeding was insufficient milk. Stopping breastfeeding within two weeks of birth was associated with rejection of the breast, or painful breasts; in later months, returning to work or college was also associated with stopping breastfeeding.

Interventions that educate women about the benefits and practice of breastfeeding are effective at increasing breastfeeding initiation. A recent Cochrane systematic review of seven randomised controlled trials (RCTs) of 1,388 women demonstrated that interventions based on one-to-one health education and support are effective at increasing initiation rates. Overall, the relative likelihood of breastfeeding initiation was 1.59 (95% CI 1.25 to 1.88) in the group who received educational interventions compared with those who received routine care. A further systematic review based on non-randomised controlled trials as well as RCTs has suggested that peer support
may be effective in improving breastfeeding initiation rates in women from disadvantaged backgrounds.\textsuperscript{261} One community-based controlled trial, considered in the review, was carried out over a two-year period in two socially deprived communities in Glasgow, Scotland. One of the communities received peer counselling from trained lay breastfeeding counsellors who were resident within the community. A second trial set in Chicago, USA evaluated the effectiveness of a hospital-based breastfeeding peer counsellor programme delivered to women who expressed an interest in breastfeeding. Both studies demonstrated improved initiation rates in women who had expressed an interest in breastfeeding, suggesting that peer-counselling programmes can be effective at encouraging women who would like to breastfeed to follow through with their decision.

There is also good evidence that appropriate support for breastfeeding mothers can prolong the duration of breastfeeding. A Cochrane systematic review updated in November 2006, based on 34 trials of 29,385 mother-infant pairs, showed that all forms of extra support together were associated with an increase in duration of ‘any’ (includes partial and exclusive) breastfeeding – the relative likelihood of stopping breastfeeding before six months was 0.91 (95% CI 0.86 to 0.96).\textsuperscript{269} Additional professional support (over and above standard care) was effective in prolonging ‘any’ breastfeeding but its effect on exclusive breastfeeding was unclear. Additional support from trained lay people was effective in prolonging exclusive breastfeeding while its effect on duration of any breastfeeding was unclear. Exclusive breastfeeding was significantly prolonged with use of WHO/United Nations Children’s Fund (UNICEF) Baby Friendly Hospital Initiative training for professionals. Overall, the relative likelihood of stopping exclusive breastfeeding during the study period considered was 0.69 (95% CI 0.52 to 0.91) in the six RCTs using the WHO/UNICEF training.

### Key message

| Interventions that educate women about the benefits and practice of breastfeeding are effective at increasing breastfeeding initiation. Appropriate support for breastfeeding mothers can prolong the duration of breastfeeding. |

**The WHO/UNICEF Baby Friendly Hospital Initiative**

The Baby Friendly Hospital Initiative is a worldwide programme of the WHO and UNICEF. It was launched in 1992 to encourage maternity hospitals to implement the Ten steps to successful breastfeeding (see Box 12) and to practise in accordance with the International Code of Marketing of Breast Milk Substitutes (Appendix 5).

Hospitals are encouraged to introduce a breastfeeding policy, educate staff to implement the policy according to role and introduce practices which promote, protect and support breastfeeding. External assessment can then lead to accreditation for those facilities implementing the standards effectively. In the UK, the Initiative has been expanded to include community healthcare facilities. Accreditation of universities which introduce approved education for breastfeeding into their pre-registration courses for midwives and health visitors has also been introduced.
Key message

Baby Friendly accredited programmes worldwide encourage hospitals and community healthcare facilities to promote breastfeeding and provide the training for healthcare practitioners. They have been shown to prolong exclusive breastfeeding. NICE guidance recommends that the WHO/UNICEF Baby Friendly Hospital Initiative should be implemented as routine practice.

Data from the Millennium Cohort Study of 18,819 children born in the year 2000 found that mothers delivering in Baby Friendly accredited hospitals are 10 per cent more likely to initiate breastfeeding (after adjustment for confounding variables) than those who deliver in non-accredited units. The study found that Baby Friendly hospital accreditation is not associated with an increase in breastfeeding at one month of age.270

Data on all babies (n=464,246) born between 1995 and 2002 in Scottish maternity units with more than 50 births per year show that babies born in Baby Friendly hospitals are 28 per cent more likely to be exclusively breastfed at seven days postnatal age than babies born in other hospitals. Breastfeeding rates increased faster in the Baby Friendly hospitals: an 11.39 per cent (95% 10.35 to 12.43) rise compared with 7.97 per cent (95% 7.21 to 8.73) in other units.271

A cluster randomised controlled trial among 17,046 mother-infant pairs in Belarus found that implementing the Baby Friendly Hospital Initiative significantly increased the duration and exclusivity of breastfeeding and decreased the risk of gastro-intestinal tract infection and of atopic eczema in the first year of life. Infants from Baby Friendly hospitals were significantly more likely than control infants to be breastfed to any degree at 12 months (19.7% vs 11.4%; adjusted OR 0.47, 95% CI 0.32, 0.69), were more likely to be exclusively breastfed at three months (43.3% vs 6.4%; P<.001) and at six months (7.9% vs 0.6%; P =.01), and had a significant reduction in the risk of one or more gastro-intestinal tract infections (OR, 0.60; 95% CI, 0.40 to 0.91) and of atopic eczema (OR, 0.54; 95% CI, 0.31 to 0.95).272
Box 12: Ten steps to successful breastfeeding

1. Have a written breastfeeding policy that is routinely communicated to all healthcare staff
2. Train all health care staff in the skills necessary to implement this policy
3. Inform all pregnant women about the benefits and management of breastfeeding
4. Help mothers initiate breastfeeding soon after birth
5. Show mothers how to breastfeed and how to maintain lactation if they are separated from their infants
6. Give newborn infants no food or drink other than breast milk unless medically indicated
7. Practice rooming-in and allow mothers and infants to stay together 24 hours a day
8. Encourage breastfeeding on demand
9. Give no artificial teats or pacifiers (also called dummies or soothers) to breastfeeding infants
10. Foster the establishment of breastfeeding support groups and refer others to them on discharge from the hospital or clinic.

Source: www.babyfriendly.org.uk

Doctors’ role in supporting breastfeeding
In common with all other health professional groups in the UK, doctors require education to enable them to implement the WHO/UNICEF standards effectively. Paediatricians and obstetricians require an understanding of the role of breastfeeding in improving the health of mothers and babies. Paediatricians also require an understanding of the normal neonatal adaptation to intermittent feeding after delivery, how poor practice can lead to excessive weight loss in breastfed babies and how this can be avoided, and how to protect safety and breastfeeding when caring for babies who are reluctant to feed after delivery or who are jaundiced or at risk of hypoglycaemia. It is recognised that in the UK hospital doctors are not the primary caregivers for breastfeeding and therefore it is not considered necessary for their education to include clinical skills in supporting breastfeeding.

The requirement to educate general practitioners (GPs) to implement the breastfeeding policy has been a source of confusion ever since the initiative was launched. GPs in the UK have a limited role in supporting breastfeeding. Their role requires them to have a basic understanding of how breastfeeding works, a knowledge of how to treat common breast conditions (mastitis, thrush etc), access a reliable reference source for prescribing drugs for breastfeeding mothers and refer mothers to the most appropriate professional for breastfeeding help and support (particularly when a baby is failing to thrive).

Key message

GPs can have a significant effect on whether or not mothers give up breastfeeding when experiencing health problems. Baby Friendly programmes show GPs how they can support the continuation of breastfeeding in such circumstances.
Teaching packs for Paediatricians and GPs are available from the UNICEF UK Baby Friendly Initiative www.babyfriendly.org.uk

**NICE Guidance on Breastfeeding**

Recent guidance from the NICE gives evidence-based recommendations relating to the promotion of breastfeeding initiation and duration. The guidance was formulated through the integration of published scientific literature with practitioner expertise and experience obtained during a national consultation exercise with professionals working in areas that could have an impact on maternal and child nutrition. These included hospitals, primary care trusts and social services. The consultation exercise focused on areas of deprivation. The guidance recommends that the WHO/UNICEF UK Baby Friendly Initiative should be implemented as routine practice. An appropriate range of educational and support programmes should be routinely delivered by health professionals and peer supporters. These include antenatal breastfeeding education combined with peer support programmes to increase breastfeeding initiation and duration among women from disadvantaged backgrounds. Other recommendations include avoidance of routine hospital practices that might lead to separation of mother and infant since these have been shown to be harmful to breastfeeding rates. The guidance also states the importance of encouraging mothers to breastfeed on demand in order to encourage breast milk production and prevent breast engorgement and mastitis. Early skin-to-skin contact has also been shown to be effective in increasing the initiation and duration of breastfeeding. A Cochrane review updated in 2007, considered the findings of 30 studies of 1,925 mother-infant pairs. Early skin-to-skin contact had positive effects on breastfeeding initiation (odds ratio of initiation was 1.82, 95% CI 1.08 to 3.07) and breastfeeding duration (weighted mean difference in duration was 42.55 days, 95% CI 1.69 to 86.79).

**Support for breastfeeding mothers returning to work**

In the UK the proportion of women in employment has increased considerably in recent years and about 50 per cent of women with pre-school children are in paid work outside the home. Working mothers often return to work early after having a baby and can have difficulty maintaining breastfeeding if not supported by their employers. This may discourage working mothers from breastfeeding at all. Recent findings from the Millennium cohort study demonstrated that women who were employed full-time were less likely to initiate breastfeeding than mothers who were not employed – rate ratio was 0.92 (95% CI 0.89 to 0.96) after adjustment for confounding factors. Strategies to support working mothers to continue with breastfeeding are needed. A recent Cochrane review to identify workplace interventions to promote breastfeeding, found no published RCTs of relevance. This represents an important gap in the evidence given the rapid increase in maternal employment in recent years.

**Key message**

Strategies are needed to support working mothers to continue with breastfeeding.

Interventions that educate women about the benefits and practice of breastfeeding and that promote baby friendly policy and practice are effective at promoting the initiation and prolonging the duration of breastfeeding. There are still some important gaps in the evidence, however, and studies are needed to look for effective ways of supporting breastfeeding in the workplace.
Interventions in complementary feeding

There is little evidence relating to interventions that aim to influence the timing and nutritional content of complementary feeding in developed country settings. One study of WIC participants suggested that peer-delivered educational interventions can be effective in reducing inappropriate early introduction of solid foods. The study was based on 181 adolescent mothers who were shown a videotape made by a peer-group of black adolescent mothers which challenged the barriers to following infant feeding guidance. The intervention was delivered as part of a home-visiting programme and assessed in a RCT. Mothers who were part of the intervention group were significantly more likely to introduce their babies to solid foods later and were more likely to report accurate knowledge of the optimal timing for the introduction of complementary food. A recent RCT set in Germany assessed the effect on infant diet of dietary counselling derived from food-based guidelines for infant nutrition. The study found that more intensive telephone counselling was more effective than less intensive counselling or written information alone in improving adherence to infant feeding guidance (assessed by food-based and meal-based dietary scores). The authors suggested that face-to-face counselling may have even greater positive effects on infant feeding practices. In developing countries where tackling infant undernutrition is the primary focus of intervention, there is evidence that educational interventions, combined with food support in countries that are food insecure are effective in improving infant nutrient intake and growth.

Mechanisms to address nutritional inequalities in the UK

Inequalities in the nutrition of young women and their children in the UK (see Box 13) can have long-term health consequences. Although evidence of successful interventions to address these inequalities may be limited, we already have a sufficiently clear understanding of the effects of early disadvantage to make the nutritional needs of mothers and their children a priority.

Box 13: Inequalities in maternal and infant nutrition

- Women of lower educational attainment are more likely to have diets of poor quality than other women.
- Consumption of fruit and vegetables is lower in low income groups in the UK while consumption of soft drinks, processed meats, whole milk and sugar is higher.
- Many people with low incomes in the UK are food insecure, and food insecurity in women is associated with poorer diets.
- Breastfeeding rates are lower in mothers from disadvantaged backgrounds.
- Early introduction of solids is more common in young mothers and mothers of lower socio-economic status.
- Compliance with infant feeding guidance to provide a diet based on fruits, vegetables and home-prepared foods is strongly associated with the quality of the mother’s own diet.
**NICE guidance**

National policy will influence the nutrition of women of childbearing age and their children. Recent public health programme guidance from NICE on maternal and child nutrition endorses existing policy on nutritional recommendations for pregnant and pre-pregnant women and for infants. The guidance strongly reinforces and builds on the previous evidence-based guidance from NICE on the promotion of breastfeeding, and recommends that all staff involved in the care of pregnant or breastfeeding women have the appropriate knowledge and skills to support and advise women about breastfeeding. Recent cross-government policy to tackle obesity also highlights the importance of breastfeeding and improved childhood nutrition in the prevention of obesity.

**Healthy Start schemes**

National programmes introduced within the UK to address inequalities in the health of young women and children may have an important impact on nutrition. The Healthy Start scheme was introduced in 2005, following a review of the Welfare Foods Scheme in 2000 by the Committee on Medical Aspects of Food and Nutrition (COMA). The review recommended that pregnant women should be given vouchers for a wider range of foods, and identified the need for health professionals to give general dietary advice during pregnancy, emphasising the importance of breastfeeding. The aim of Healthy Start is to reduce inequalities in nutrition for women and children. The scheme provides food support, professional advice and support to pregnant women and mothers of young babies and children from disadvantaged backgrounds. Like the Welfare Foods Scheme before it, food support comes in the form of vouchers to buy milk and infant formula and provision of vitamin supplements. As recommended by COMA, however, there is greater flexibility within the Healthy Start scheme allowing the vouchers to be used to buy fruit and vegetables. Early evaluation of the scheme in Devon and Cornwall suggested that women who were recipients of Healthy Start were buying more fruit and vegetables than they were before the scheme began. Further follow-up will be needed to confirm whether these beneficial effects on fruit and vegetable uptake are observed on a wider scale and maintained in the longer term. The evaluation did not examine overall uptake of the scheme nor were any nutritional outcomes measured. A survey of health professionals was also carried out: interviews with a small sample of health visitors and midwives suggested that most professionals interviewed were aware of the scheme and how to apply it. Knowledge about some elements of the scheme remained low, however, and the authors of the evaluation concluded that coverage of training to prepare for Healthy Start had been somewhat patchy. Evaluation at national level is needed to assess uptake of the scheme and to examine its impact on the nutrition of women and young children. Other public health programmes that are directed at families with young children including Sure Start Children’s Centres, also have the potential to bring about improvements in maternal nutrition and infant feeding practices.

Although national policy may influence maternal and child nutrition, the central importance of the family should also be recognised. A woman’s diet before and during pregnancy, and her views on breastfeeding and weaning will be influenced by her partner, friends and family. Clear and consistent guidance for families on the benefits of healthy eating is needed, particularly to explain the long-term consequences for their children of early diet and nutrition. In the future it needs to be ensured that all professionals who care for pregnant women and their families have appropriate knowledge and skills to provide this guidance.
Tackling the problem of obesity
The DH has set targets to address obesity. The public service agreement (PSA), established in 2007 has the aim ‘by 2020 we will have reduced the proportion of overweight and obese children to 2000 levels’. The Government Office for Science through the Foresight programme has developed a strategic approach to address the problem.

In terms of chronic disease prevention, it appears that addressing infant nutrition could impact on these relatively short-term targets, and it would be very important in influencing the incidence of chronic disease in the longer term. Some research shows that weight gain even in the first few weeks of life is related to childhood obesity.

The BMA policy report Preventing childhood obesity (2005) raises concerns at the rising levels of childhood obesity and poor nutritional quality of diets of children and adolescents.\textsuperscript{282} The report discusses those factors contributing to the rise in obesity and highlights the responsibilities of individuals in addressing obesity, and of policy makers to provide an environment which is less obesogenic. The importance of infant feeding is noted and the protective effect of breastfeeding on childhood obesity is discussed. A primary theme of this work focuses on enabling parents and carers, as well as children, to make healthy choices through effective education and information provision which is consistent and free from adverse influences which encourage unhealthy food choices. Recent follow up work to this report has concentrated on:

- improving school food and food education in school
- lobbying for restrictions on the advertising of foods high in fat, salt and sugar to children
- supporting and encouraging adoption of ‘traffic light’ front-of-pack signposting.

The Foresight project Tackling obesities: future choices was announced in 2005. The BMA was a key stakeholder and had an input particularly focused on systems mapping and scenario building. The outcomes report\textsuperscript{283} was published in October 2007 and examines the scientific evidence base from across a wide range of disciplines in order to understand the relationships and importance of key factors influencing obesity. Using this evidence it aims to identify effective interventions, predict how obesity might change in the future and identify the most effective responses. As well as the final report, this project produced a series of reviews of the current science and models for future trends of obesity and policy options and the impacts on health.

The Foresight report argued for a life-course approach to prevention and emphasised that society would have to be prepared to measure success over longer timeframes than currently. A specific recommendation was the promotion and implementation of a programme of early interventions at birth or in infancy.

Key message

Improving infant nutrition could be very important in influencing the incidence of chronic disease in the longer term.
Box 14: Government’s obesity strategy

In January 2008, the Cross-Government Obesity Unit published its strategy for England, to address the rising prevalence of obesity. This strategy recognises the importance of promoting children’s health and wellbeing. It encompasses a range of recommendations that include promotion of breastfeeding, further investment in the Healthy Schools Programme, and the promotion of a culture of healthy eating. Through the development of a Healthy Food Code of Good Practice, the Government aims to work with industry and other stakeholders to develop a single approach to food labelling, to rebalance the advertising and marketing of foods that are high in fat, salt or sugar to children, and to promote the consumption of healthy foods, particularly fruit and vegetables. As part of the Code, all food businesses will work with the Foods Standards Agency (FSA), DH and other stakeholders to deliver a single set of key healthy eating messages.

In November 2007, the BMA hosted a stakeholder event on food labelling. Invited representatives from health-related organisations and the food industry came together to discuss ways in which healthy eating messages can be reinforced through advice about nutrition and food labelling. The group agreed a position statement, which is available on the BMA website, (see Box 15).

Box 15: A BMA food labelling statement

January 2008

The British Medical Association (BMA) has a long-term interest in the health of the public and believes that the increase in the frequency of obesity is a cause for great concern. While tackling obesity requires commitment to a multi-faceted approach, there is a particular need to change dietary behaviour. The BMA considers improved and consistent food labelling to be an important mechanism for enabling consumers to make informed dietary choices.

The BMA believes that the food and drink industry should implement a standardised, consistent approach to food labelling. This approach should be based upon the traffic light front of pack labelling recommended by the FSA. In order to increase the nutritional information available to consumers we propose that the labelling should also include Guideline Daily Amount (GDA) information.

The BMA also recognises that public awareness should be raised about the health benefits of micronutrients. This could be done in the form of in-store leaflets explaining the benefits of healthy food choices and stating which foods are rich in which micronutrients.
Conclusions

- Many young women have diets of poor quality and inadequate nutritional status. Less healthy diets are more common among women of low educational attainment, and among women who have low income and who are food insecure. Although evidence of effective interventions to improve the diets of young women is limited, this does not mean that the goal should not be pursued vigorously. Both nutrition education and counselling have been shown to improve women's nutrition knowledge and behaviour.

- Breastfeeding rates are low in the UK, and it is less common among disadvantaged women. Interventions that educate women about the benefits and practice of breastfeeding, and that promote baby friendly policies and practice, are effective at promoting the initiation and prolonging the duration of breastfeeding.

- Current studies of complementary feeding show wide variations in practice in the UK. There are few studies of interventions to influence the timing and nutritional content of complementary feeding in developed countries.

- Infant diet is strongly linked to mother’s diet – suggesting that interventions to improve the diets of young women will also have direct consequences for children's diets.

- Effective interventions are needed to improve the nutrition of young women of childbearing age. Such interventions may influence the way in which mothers feed their children as well as influencing the diets of women themselves. They will therefore have beneficial health effects across generations.

- Efforts to encourage the initiation and to prolong the duration of breastfeeding need to continue and be extended. The ten steps to successful breastfeeding and the Baby Friendly Hospital Initiative guidelines should become a minimum standard of care. Action is also needed to improve the opportunities for women to breastfeed in public places and to support continued breastfeeding in women who return to work.

- The Healthy Start scheme provides support for eligible women and children through provision of food vouchers and vitamin supplements, and needs to be promoted widely. Its impact on the nutrition of mothers and young children should be evaluated at a national level.

- Other gaps in the evidence should be addressed including interventions to support breastfeeding mothers in the workplace and interventions to optimise the timing and content of complementary feeding.
Appendix 1: Epigenetic processes

In addition to the sequence of nucleotide bases which comprise the genetic code, and which are inherited from the parents (the genotype) the characteristics of an individual (their phenotype) are affected by epigenetic processes, which alter the ways in which genes are switched on and off. Epigenetic processes do not affect the DNA sequence, but do alter gene expression. Epigenetic changes occur during development and can be affected by the environment before and after birth.

Figure 15: Components of the epigenetic code

Diagram of chromosome being unravelled to show deoxyribonucleic acid (DNA) wrapped around histone protein cores to form nucleosomes. Protruding histone ‘tails’ are subject to a range of chemical modification which can alter structure and make genomic DNA more accessible to transcription. CG base pairs of the DNA can also be modified by methylation, which silences transcription. Thus these processes can affect gene expression, and hence development and phenotypic characteristics, without altering the genomic DNA code itself.

Epigenetic inheritance is defined as biological processes that regulate mitotically or meiotically heritable changes in gene expression without altering the DNA sequence. Of particular relevance is methylation of specific CpG dinucleotides in gene promoters and alterations in DNA packaging in nucleosomes arising from chemical modifications of the chromatin histone core around which DNA wraps. The histone modifications include acetylation, methylation, ubiquitination, and phosphorylation. Other changes may involve other DNA associated proteins. Recently microRNAs which affect DNA transcription and mRNA stability and translation into proteins have been implicated as a further mechanism for inherited epigenetic effects, including those transmitted via the paternal line.

Epigenetic mechanisms are widely implicated in cancer. In this situation the changes can be modified by alteration of methyl group metabolism. This suggests a mechanism by which folate and methyl donor status during development may operate. Gene promoter methylation is important for asymmetrical silencing of imprinted genes and retrotransposons. They also play a critical role in a range of developmental processes. With the exception of imprinted genes, widespread removal of epigenetic marks occurs following fertilisation when maternal and paternal genomes undergo extensive demethylation to ensure pluripotency of the developing embryonic cells. This is followed by de novo methylation just prior to implantation. About 70 per cent of CpGs are methylated, mainly in repressive heterochromatin regions and in repetitive sequences such as retrotransposable elements. DNA methylation also plays a key role in cell differentiation by silencing the expression of specific genes during the development and differentiation of individual tissues. For some genes there also appear to be gradations of promoter demethylation associated with developmental changes in the role of the gene product. Changes in methylation which are associated with cell differentiation and functional changes are established at different times during development of the embryo. The pattern of DNA methylation is copied during mitosis by DNA methyltransferase (Dnmt) activity. This provides an ‘epigenetic memory’ of patterns of gene regulation and hence cell function, which are established during development and which are passed to the adult. Environmental challenges at different times during development may induce stable changes in cell function, which persist in the adult organism. They may produce different phenotypic outcomes and in humans differential risk of disease.
Appendix 2: Definition of overweight

**World Health Organisation classifications of BMI**

BMI values are age-independent and the same for both sexes. It may not, however, correspond to the same degree of fatness in different populations due, in part, to different body proportions. The health risks associated with increasing BMI are continuous and the interpretation of BMI gradings in relation to risk may differ for different populations.

<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI (kg/m²)</th>
<th>Principal cut-off points</th>
<th>Additional cut-off points</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Underweight</strong></td>
<td></td>
<td>&lt;18.50</td>
<td>&lt;18.50</td>
</tr>
<tr>
<td>Severe thinness</td>
<td></td>
<td>&lt;16.00</td>
<td>&lt;16.00</td>
</tr>
<tr>
<td>Moderate thinness</td>
<td>16.00 - 16.99</td>
<td>16.00 - 16.99</td>
<td>17.00 - 18.49</td>
</tr>
<tr>
<td>Mild thinness</td>
<td>17.00 - 18.49</td>
<td>17.00 - 18.49</td>
<td></td>
</tr>
<tr>
<td><strong>Normal range</strong></td>
<td>18.50 - 24.99</td>
<td>18.50 - 24.99</td>
<td>23.00 - 24.99</td>
</tr>
<tr>
<td><strong>Overweight</strong></td>
<td>≥25.00</td>
<td>≥25.00</td>
<td>≥25.00</td>
</tr>
<tr>
<td>Pre-obese</td>
<td>25.00 - 29.99</td>
<td>25.00 - 27.49</td>
<td>27.50 - 29.99</td>
</tr>
<tr>
<td><strong>Obese</strong></td>
<td>≥30.00</td>
<td>≥30.00</td>
<td>≥30.00</td>
</tr>
<tr>
<td>Obese class I</td>
<td>30.00 - 34.99</td>
<td>30.00 - 32.49</td>
<td>32.50 - 34.99</td>
</tr>
<tr>
<td>Obese class II</td>
<td>35.00 - 39.99</td>
<td>35.00 - 37.49</td>
<td>37.50 - 39.99</td>
</tr>
<tr>
<td>Obese class III</td>
<td>≥40.00</td>
<td>≥40.00</td>
<td></td>
</tr>
</tbody>
</table>

Appendix 3: Current UK dietary recommendations for pregnancy

Before pregnancy
A healthy diet is important at any time but particularly when planning a pregnancy. The Eatwell plate makes healthy eating easier. Try to eat:
- plenty of fruit and vegetables – aim for at least five portions a day
- plenty of starchy foods
- some milk and dairy foods
- some meat, fish, eggs and other non-dairy sources of protein
- just a small amount of foods and drinks high in fat and/or sugar.

A 400μg folic acid supplement should be taken daily from the time contraception is stopped until the 12th week of pregnancy. Women who have previously had a baby with neural tube defects, or who are taking medication for diabetes, epilepsy or coeliac disease should take a 5mg supplement.

DH guidelines on the consumption of alcohol state that women trying to conceive should avoid drinking alcohol, but if they do choose to drink, to minimise the risk to the baby, they should not drink more than one to two units of alcohol once or twice a week and should not get drunk.

During pregnancy
A healthy diet is important in pregnancy. There is no need to eat for two – it’s the quality not the quantity of the diet that’s important. Try to eat a variety of foods including:
- plenty of fruit and vegetables – aim for at least five portions a day
- plenty of starchy foods
- plenty of iron-rich foods such as red meat, pulses, bread, green vegetables, and foods rich in vitamin C that will help the iron to be absorbed
- milk and dairy foods – these contain calcium
- foods rich in protein such as lean meat, chicken and fish (aim for at least two portions of fish each week including one of oily fish), eggs and pulses
- fibre-rich foods such as wholegrain bread and cereals.

Cut down on foods and drinks high in fat and/or sugar such as cakes and biscuits.

A 400μg folic acid supplement should be taken daily until the 12th week of pregnancy. Women who have previously had a baby with neural tube defects, or who are taking medication for diabetes, epilepsy or coeliac disease should take a 5mg supplement.

A 10μg vitamin D supplement should be taken throughout pregnancy.

Do not take dietary supplements that contain vitamin A such as fish liver oils, and avoid liver and liver-containing products.

Caffeine-containing drinks should be consumed in moderation to limit caffeine intake to less than 200mg a day (equivalent to two mugs of instant coffee, two mugs of tea or five cans of cola).

Avoid paté, certain cheeses and raw or partially cooked eggs, shellfish and meats, and unpasteurised milk.

DH guidelines on the consumption of alcohol state that pregnant women should avoid drinking alcohol but if they do choose to drink, to minimise the risk to the baby they should not drink more than one to two units of alcohol once or twice a week and should not get drunk. The BMA believe that the only sensible message for women who are pregnant must be complete abstinence from alcohol.
Vegetarianism and veganism

It is possible for vegetarians and vegans (people who eat no animal products at all, including dairy products) to be adequately nourished for successful pregnancy and lactation, but they need to be knowledgeable about nutrition and plan their diet carefully. For vegans, there is a high risk of deficient intakes of micronutrients such as vitamin B12, iodine, calcium, vitamin D and omega-3 fatty acids. There are reports of neurological deficits in children born to vitamin B12 deficient vegan mothers. Vegan mothers should therefore take vitamin B12 supplements, as this micronutrient is found only in foods of animal origin. The high levels of vitamin B12 found in some algae and seaweeds (eg spirulina) are not bio-active. For the other micronutrients listed above, supplements must be taken, or fortified foods (eg many soya milks are fortified with calcium) or specific foods sources included in the diet (eg kelp seaweed for iodine, or flaxseed for omega-3 fatty acids).

Good advice can be found on the websites of the Vegetarian Society and Vegan Society.
## Appendix 4: Infant feeding guidelines

<table>
<thead>
<tr>
<th>World Health Organisation</th>
<th>UK Food Standards Agency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breastfeeding: Practice exclusive breastfeeding from birth to six months, and introduce complementary foods at six months while continuing to breastfeed on-demand until two years of age or beyond.</td>
<td>Breastfeeding: Breastfeeding is best and provides all the nutrients a baby needs for the first six months. Continue exclusive breastfeeding for six months. The longer breastfeeding continues, the greater the benefits.</td>
</tr>
<tr>
<td><strong>Formula feeds:</strong> Not recommended – all babies should be breastfed. WHO has recently developed guidelines for the non-breastfed baby (partly for use by HIV-positive women).</td>
<td><strong>Formula feeds:</strong> Infant formula is the only alternative to breastfeeding until one year. Cow’s milk-based formula is best. Follow-on formula can be used from six months but is not essential. Other types of formula should be used only on medical advice: Hydrolysed protein formula may be useful in cases of cow’s milk allergy. Soya formula is an alternative but may also be allergenic. Goats’ milk formula is not suitable for babies.</td>
</tr>
<tr>
<td>Other drinks: Avoid giving drinks with low nutrient value, such as tea, coffee and sugary drinks such as soda. Limit the amount of juice offered so as to avoid displacing more nutrient-rich foods. Increase fluid intake during illness.</td>
<td>Other drinks: Water is the best alternative drink to milk. Breastfed babies don’t need any until they start solids. Under six months use tap water, boiled and cooled. Some bottled water has a mineral content too high for babies. Others are suitable (labelled accordingly). Bottled water should be boiled too. Fruit juices are a source of vitamin C but reduce the baby’s appetite for milk and can cause tooth decay; avoid before six months. After that use diluted (one in 10 with boiled water) in a feeding cup, at mealtimes only. Squashes, fizzy drinks, flavoured milk, juice drinks, tea and coffee are not suitable for infants.</td>
</tr>
<tr>
<td><strong>Starting complementary feeds:</strong> Start at six months with small amounts of food and increase as the child gets older while maintaining frequent breastfeeding. Energy needs from complementary foods in developing countries are ~200 kcal/day at six to eight months, 300 kcal/day at nine to 11 months, and 550 kcal/day at 12-23 months. In industrialised countries these estimates are 130, 310 and 580 kcal/day because of differences in average breast milk intake.</td>
<td><strong>Starting complementary feeds:</strong> Solids can be started from six months and gradually increased in amount and variety so that by twelve months, solid foods are the main part of the diet, with breast or formula milk making up the balance.</td>
</tr>
</tbody>
</table>
### World Health Organisation

**Progress with complementary feeds:**
Gradually increase food consistency and variety, adapting to the infant’s requirements and abilities. Infants can eat pureed, mashed and semi-solid foods beginning at six months, by eight months ‘finger foods’, by 12 months the same types of food as the rest of the family, keeping in mind the need for nutrient-dense foods. Avoid foods that may cause choking such as nuts, grapes, raw carrots.

Increase the number of times the child is fed complementary foods as he/she gets older. For the average infant complementary foods should be provided two to three times/day at six to eight months and three to four times/day at nine to 24 months. Additional nutritious snacks (pieces of fruit or bread or chapatti with nut paste) may be offered one to two times/day. If the energy density or amount of food per meal is low, or the child is no longer breastfed, more frequent meals may be required.

Feed a variety of foods to ensure that nutrient needs are met. Meat, poultry, fish, eggs and vitamin A-rich foods should be eaten daily or as often as possible. Provide diets with adequate fat content. After illness, encourage the child to eat more than usual.

Vegetarian diets cannot meet nutrient needs at this age unless nutrient supplements or fortified products are used.

### UK Food Standards Agency

**Progress with complementary feeds:**
Start with a teaspoon of smooth vegetable purée (carrot, parsnip, potato, yam) or fruit purée (banana, cooked apple, pear or mango) or cereal (not wheat-based) such as baby rice, sago, maize, corn meal or millet, given with the baby’s usual milk (breast or formula) at one feed in the day.

Gradually increase the amount within one feed, and then progress to two and three feeds per day. React to the baby’s appetite, giving more if wanted. Solids can include full-fat cows’ milk products (yoghurt, fromage frais, cheese sauce). Give cereals once a day. Use home-cooked foods mashed, sieved, or puréed. Introduce puréed red meat, poultry, fish or eggs, or puréed beans or pulses (lentils, hummus) at least once a day. Serve starchy foods (potatoes, yams, rice or bread) two to three times/day, and fruit and vegetables as finger foods at two or more meals/day.

As the baby continues to develop, use foods with a thicker and lumper texture to encourage chewing, even before teeth emerge. Give finger foods (toast, bread, breadsticks, pitta bread or chapatti, peeled apple, banana, carrot sticks, or cubes of cheese). Avoid sweet biscuits and rusks. Later, start minced or chopped meals and fruit between meals. When the baby is mobile (crawling and walking) increase the amount of food. Use full-fat dairy products; low fat is sensible for adults but not babies.

If the family is vegetarian, use pulses (such as red lentils, beans or chickpeas) or tofu as protein sources. Vitamin C in fruit and vegetables helps iron absorption, so include these at mealtimes.
<table>
<thead>
<tr>
<th>World Health Organisation</th>
<th>UK Food Standards Agency</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Do not force:</strong> Feed slowly and patiently; encourage but do not force. If children refuse foods, experiment with different foods, tastes, textures and methods of encouragement. Minimise distractions during meals. Feeding times are periods of learning and love – talk to children during feeding, with eye to eye contact.</td>
<td><strong>Do not force:</strong> Go at the baby’s pace. Allow plenty of time for feeding and to allow the baby to learn to swallow solids. Don’t rush or ‘force feed’. Most babies know when they’ve had enough to eat. Offer a wide variety of foods to avoid choosiness later on.</td>
</tr>
<tr>
<td><strong>Food safety:</strong> Practice good hygiene. Wash hands before food preparation. Store foods safely. Serving immediately after preparation, using clean utensils and serving dishes. Avoid using feeding bottles, which are difficult to keep clean.</td>
<td><strong>Food safety:</strong> Heat only what the baby will want. Do not reheat previously warmed food. Heat food thoroughly and allow it to cool. Do not refreeze food that’s been warmed or previously frozen. Everything for feeding the baby needs to be really clean.</td>
</tr>
<tr>
<td><strong>To be avoided:</strong> No specific guidelines</td>
<td><strong>To be avoided:</strong> Added salt: Babies under a year should have less than 1g salt per day. Don’t add salt to cooked foods. Limit high-salt foods (cheese, bacon, sausages, processed foods, pasta sauces, breakfast cereal). Avoid sugar and honey: Sweeten stewed sour fruit like rhubarb with mashed banana, breast or formula milk. Don’t use honey until after one year as it may contain harmful bacteria. Some foods can cause allergic reactions in some babies. If a family history of coeliac disease, consult a doctor before using wheat, rye or barley-based foods. Nuts and seeds – including peanuts, peanut butter and other nut spreads. Don’t give whole peanuts to children under five years old because they can cause choking.</td>
</tr>
<tr>
<td><strong>Vitamin-mineral supplements:</strong> Use fortified complementary foods or vitamin-mineral supplements as needed.</td>
<td><strong>Vitamin-mineral supplements:</strong> No specific guidelines. See DH guidelines in text.</td>
</tr>
<tr>
<td><strong>Starting ordinary cow’s milk:</strong> No specific guidelines.</td>
<td><strong>Starting ordinary cow’s milk:</strong> Full-fat cow’s milk not suitable as a drink until one year. Semi-skimmed milk not suitable as a drink until two years. Skimmed milk not suitable until five years.</td>
</tr>
</tbody>
</table>
Appendix 5: The International Code on the Marketing of Breast Milk Substitutes

The code was established by WHO in 1981 and has been modified by subsequent World Health Assembly resolutions. The essential elements are as follows.

- The Code forbids direct contacts between commercial representatives and medical personnel, mothers or pregnant women.

- Baby food companies may not distribute free or discounted samples of formula in hospitals and other health premises.

- Advertisements for baby foods must not target parents of infants younger than six months.

- There should be no promotional distribution of dummies (pacifiers), bottles or teats.

- Manufacturers of breast milk substitutes may not distribute promotional gifts to health workers, and information produced for health professionals should be strictly factual and based on scientific studies.

- Images of mothers and children are not allowed on packaging. Information on labels must be printed in easy to understand terms in the local language. Certain words, such as ‘motherly’, cannot be used. The labels must state that breastfeeding is the best way of feeding babies and that a substitute should only be used after consultation with health professionals.

The Code has no legal power unless it is incorporated into national legislatures (as in the UK). Many developing countries have no effective legislation. The Code was initiated in response to aggressive marketing of formula in developing countries in the 1970s and 80s. Code violations are monitored by the International Baby Food Action Network (IBFAN) (www.ibfan.org); according to IBFAN, violations are much less common but are still occurring.
## Appendix 6: Comparison of the nutrient composition of mature human breast milk and formula

<table>
<thead>
<tr>
<th></th>
<th>Mature breast milk</th>
<th>Breast milk substitutes or first milks*</th>
<th>Follow-on Formula*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Energy (kcal)</strong></td>
<td>670-700</td>
<td>670-700</td>
<td>670-700</td>
</tr>
<tr>
<td>(kJ)</td>
<td>2800-3000</td>
<td>2800-2950</td>
<td>2800-2950</td>
</tr>
<tr>
<td><strong>Protein (g/l)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Casein (%)</td>
<td>10-13</td>
<td>14-14.5</td>
<td>18-19</td>
</tr>
<tr>
<td>α-lactalbumin (%)</td>
<td>35-40</td>
<td>40-43</td>
<td>78-80</td>
</tr>
<tr>
<td>Lactoferrin (%)</td>
<td>22-33</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Secretory IgA (%)</td>
<td>10-15</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lysozyme</td>
<td>1-4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Fat (g/l)</strong></td>
<td>38-42</td>
<td>35-38</td>
<td>33-34</td>
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<tr>
<td>Saturated fat (%)</td>
<td>42</td>
<td>40-45</td>
<td>na</td>
</tr>
<tr>
<td>DHA (%)</td>
<td>0.07-1.0</td>
<td>0.2-0.5</td>
<td>varies</td>
</tr>
<tr>
<td><strong>Carbohydrate (g/l)</strong></td>
<td>69-74</td>
<td>70-75</td>
<td>74-81</td>
</tr>
<tr>
<td><strong>Vitamins</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A (µg Re/l)</td>
<td>600</td>
<td>540-820</td>
<td>650-800</td>
</tr>
<tr>
<td>β carotene (µg Re/l)</td>
<td>24</td>
<td>35-42</td>
<td>25-43</td>
</tr>
<tr>
<td>B1 (mg/l)</td>
<td>0.1-0.2</td>
<td>0.4-1.0</td>
<td>0.4-1.0</td>
</tr>
<tr>
<td>B2 (mg/l)</td>
<td>0.3-0.4</td>
<td>0.6-1.5</td>
<td>1.0-1.5</td>
</tr>
<tr>
<td>B3 (mg/l)</td>
<td>1.5-2.0</td>
<td>6.9-9.0</td>
<td>6.5-9.0</td>
</tr>
<tr>
<td>B6 (mg/l)</td>
<td>0.1</td>
<td>0.4-0.6</td>
<td>0.4-0.6</td>
</tr>
<tr>
<td>B12 (µg/l)</td>
<td>0.1-0.3</td>
<td>1.4-2.1</td>
<td>1.6-2.0</td>
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<tr>
<td>Folic acid (µg/l)</td>
<td>50-52</td>
<td>34-160</td>
<td>70-150</td>
</tr>
<tr>
<td>Biotin (µg/l)</td>
<td>7.6</td>
<td>10-20</td>
<td>20-30</td>
</tr>
<tr>
<td>Pantothenic acid (mg/l)</td>
<td>2.0-2.6</td>
<td>2.3-3.0</td>
<td>3.0-3.6</td>
</tr>
<tr>
<td>C (mg/l)</td>
<td>38-43</td>
<td>69-90</td>
<td>80-140</td>
</tr>
<tr>
<td>D (µg/l)</td>
<td>0.1-0.4</td>
<td>10-14</td>
<td>11-19</td>
</tr>
<tr>
<td>E (mg/l)</td>
<td>2.0-3.5</td>
<td>5-13</td>
<td>5-11</td>
</tr>
<tr>
<td>K (µg/l)</td>
<td>15</td>
<td>27-67</td>
<td>29-67</td>
</tr>
<tr>
<td><strong>Minerals and trace elements</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium (mg/l)</td>
<td>150-160</td>
<td>160-170</td>
<td>200-300</td>
</tr>
<tr>
<td>Potassium (mg/l)</td>
<td>550-600</td>
<td>570-740</td>
<td>850-900</td>
</tr>
<tr>
<td>Calcium (mg/l)</td>
<td>300-360</td>
<td>390-510</td>
<td>720-900</td>
</tr>
<tr>
<td>Phosphorus (mg/l)</td>
<td>140-155</td>
<td>240-290</td>
<td>480-530</td>
</tr>
<tr>
<td>Ca/P ratio</td>
<td>1.7-1.8</td>
<td>1.4-2.0</td>
<td>1.2-1.7</td>
</tr>
<tr>
<td>Iron (mg/l)</td>
<td>0.5-0.8</td>
<td>5-8</td>
<td>12-3</td>
</tr>
<tr>
<td>Zinc (mg/l)</td>
<td>3-4</td>
<td>3-6</td>
<td>4-9</td>
</tr>
<tr>
<td>Iodine (µg/l)</td>
<td>30-70</td>
<td>45-100</td>
<td>100-120</td>
</tr>
<tr>
<td>Copper (µg/l)</td>
<td>384-400</td>
<td>330-420</td>
<td>410-600</td>
</tr>
<tr>
<td>Selenium (µg/l)</td>
<td>10-30</td>
<td>10-15</td>
<td>15</td>
</tr>
<tr>
<td>Magnesium (mg/l)</td>
<td>29-40</td>
<td>45-52</td>
<td>65-70</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol (mmol/l)</td>
<td>163</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Taurine (mg/l)</td>
<td>40-54</td>
<td>47-67</td>
<td>50</td>
</tr>
</tbody>
</table>

* The composition of formula milk is presented as the range present in four leading brands each of cow’s milk-based first and follow-on preparations – information supplied by the manufacturers (date 31 January 2008). Breast milk varies in composition and therefore the values provided are approximate; information on the composition of breast milk was obtained from several sources. [126, 127, 298-301]

na in the table indicates that the authors of this study were unable to find the relevant data.

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