Influences in Childhood on the Development of Cardiovascular Disease and Type 2 Diabetes in Adulthood

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Foreword

This paper provides a comprehensive review of the medical literature around the childhood determinants of adult diabetes and cardiovascular disease. The study was undertaken by Dr Nicola Nelson, as part of her advanced training in paediatrics, during her six-month attachment to the Ministry of Health. The context for the research is that it is a building block for the Ministry's 'Leading for Outcomes' workstream of the Clinical Services Directorate. This paper does not necessarily define formal Ministry of Health policy in this area, but is intended to inform policy decisions and programme implementation in the health sector.

It is an important background document for health researchers, funders and planners and clinicians who are attempting to stem the growth of chronic disease in adulthood. It clearly identifies the precursors of the major chronic conditions that have the potential to cripple not only individuals, but also the health system. In addressing root causes, the paper also picks up the first 'Future direction' of the New Zealand Child Health Strategy 1998, which supports 'a greater focus on health promotion, prevention and early intervention'.

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Acknowledgements

This study is based on the framework of levels of causation from *Looking Upstream: Causes of death cross-classified by risk and condition New Zealand 1997* (Tobias 2004a). I thank Dr Martin Tobias (Ministry of Health) for allowing me to use this and for assisting me with my understanding of health determinants. I acknowledge the work of Dr Hiran Thabrew (formerly of the Ministry of Health), whose previous research on childhood obesity was extremely useful to me. I am indebted to a large number of people, both within the Ministry of Health and externally, who assisted me with this project. In particular I thank Dr Pat Tuohy (Ministry of Health), my supervisor, and Professor Barry Taylor (University of Otago), who peer reviewed my work. I gratefully acknowledge the invaluable information support service provided by the library staff at the Ministry of Health, especially Emma Roache, Stuart Cretney and Melissa Toohey. I also thank Dr Stuart Dalziel (University of Auckland) for his technical assistance.

Nicola Nelson
February 2005

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Introduction

Currently there is a global epidemic of chronic diseases, such as cardiovascular disease and type 2 diabetes, that are intimately related to dietary and lifestyle factors. Type 2 diabetes, once thought of as a disease of adulthood, is emerging in children and youth. Childhood obesity, which is both a precursor for type 2 diabetes and a risk factor for cardiovascular disease, is escalating rapidly. New Zealand is no exception to this pandemic. In addition, there are significant ethnic disparities in the distribution of disease in this country. Underpinning this, children, and particularly Māori and Pacific children, are overrepresented in the most socioeconomically disadvantaged sector of our population.

Lifestyle choices for children are made, and habits formed, within the context of their family or whānau, and also within the broader context and constraints of the structural features of society, economy and environment. A family's lifestyle is governed by many factors, including their income, which is a key determinant of health.[1] There is also evidence that in New Zealand, and abroad, the negative effects of low socioeconomic status in childhood persist into adulthood, particularly in regard to cardiovascular disease.[2–6]

A lifecourse perspective on non-communicable disease prevention and control is critical, as taste, dietary and physical activity habits are established early in life.

> Maternal health and nutrition before and during pregnancy, and early infant nutrition are important in the prevention of non-communicable diseases throughout the lifecourse. Exclusive breastfeeding for six months and appropriate complementary feeding after that, contribute to optimal physical growth, mental development and the prevention of non-communicable diseases. Infants who suffer growth restriction in utero, are of low birth weight, and/or are not breastfed, or are stunted as a result of micronutrient deficiencies, are at increased risk for non-communicable disease in later life.[7]

According to the World Health Organization (WHO), unhealthy diets and physical inactivity are the leading causes of the major non-communicable diseases, including cardiovascular disease and type 2 diabetes.[7] The Public Health Intelligence unit of the Ministry of Health has analysed causes of death in New Zealand by risk and found that diet is by far the largest contributor to mortality. Diet, tobacco, deprivation, cholesterol, blood pressure, body mass index (BMI) and insufficient physical activity are the predominant risk factors contributing to death in New Zealand (see appendix).[8] These factors are all potentially modifiable.

Objective

The objective of this review is to draw together current evidence relating to the factors in childhood that influence the development of cardiovascular disease and type 2 diabetes in adulthood, with the intention of identifying the most important points of intervention.

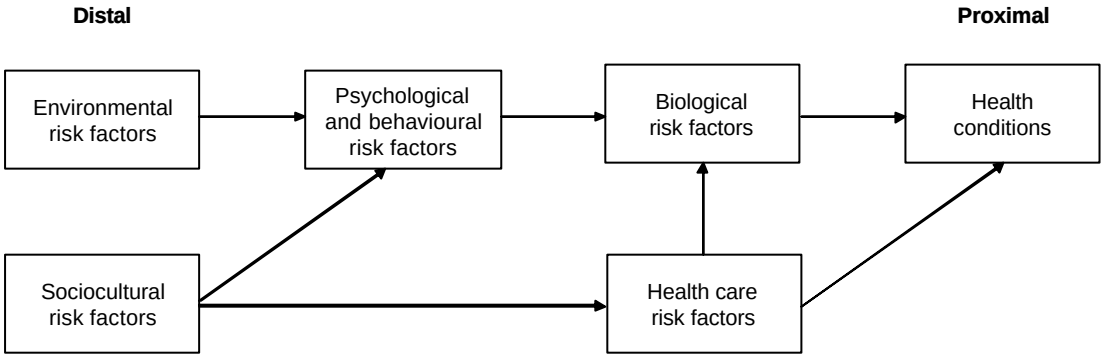
Methodology

The information for this review was obtained from multiple sources. Much of it comes from relevant Ministry of Health publications, along with research reports published in academic and scientific journals and books. Several strategies were used to find documents, with a focus on information relevant to New Zealand. This included repeated Medline searches using key words, surfing Internet websites, searching bibliographies from key texts and use of a bibliographic database search carried out by the Ministry's information services staff. Further studies were identified from citations from retrieved articles and discussion with experts.

Levels of causation and joint effects

Risk factors operate at multiple levels of causation.

Figure 1: Levels of causation



Note: Only the major causal pathways are shown.

Source: Tobias M. 2004. *Looking Upstream: Causes of death cross-classified by risk and condition New Zealand 1997*. Public Health Intelligence Occasional Bulletin Number 20. Wellington: Ministry of Health.

Proximal biological and behavioural influences are in turn shaped by more distal sociocultural and environmental factors.

> This means that risk factors may not act independently of each other. Instead, the effect of one risk factor may be (partly) mediated by another, or its effect may depend on the level of another. Also, clustering of risk factors in the same individual may lead to synergistic or antagonistic rather than the expected multiplicative effects.[8]

More proximal causes are often easier to identify. Biological causes are usually measurable and therefore quantifiable and able to be examined scientifically. The priority that is often given to these types of causes is due to the greater scientific certainty and universality with which causal attributions can be made.[9] However, the importance of the more distal causes to which whole populations are exposed has been expounded by Geoffrey Rose. As he states, these population characteristics are the determinants of incidence rate. Therefore, despite the greater uncertainty that might surround more distal causes of disease the potential gain from preventative strategies that prioritise distal

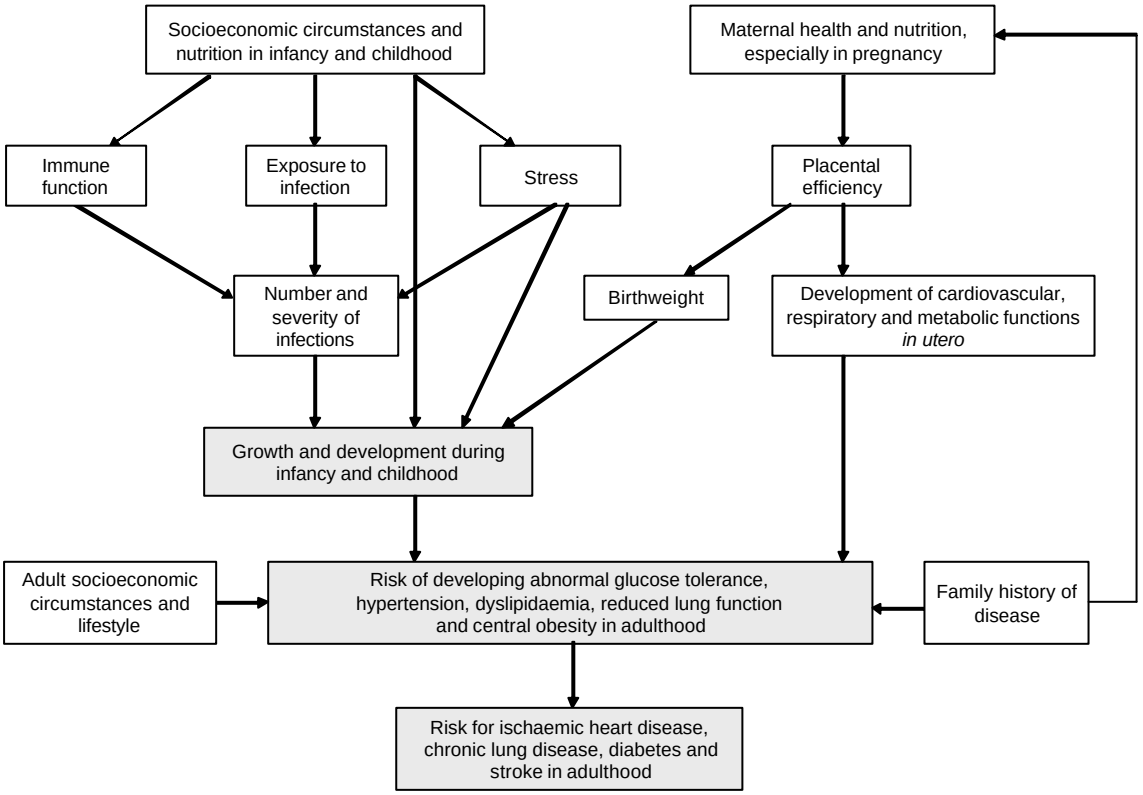
causes is far greater. These population strategies involve mass environmental control methods or attempts to alter some of society's norms of behaviour. However, Rose also cautions that for population strategies the risk:benefit ratio for individuals may be small.[10] This emphasises the importance of evaluation and monitoring for such strategies.

Risk factors are cumulative over the lifecourse. People who have experienced adverse conditions in the past are at greatest risk at any stage of the lifecourse. Therefore children who experience disadvantage early in life are launched on a potential trajectory of poor outcomes. Policies need to provide springboards to offset this.[11]

Observations from autopsy studies by the Bogalusa Heart Study and the multicentre Pathobiological Determinants of Atherosclerosis in Youth study document a strong correlation between coronary atherosclerosis and cardiovascular risk factors in young people. The extent of atherosclerotic lesions in the coronary vessels increases markedly in young people with multiple risk factors, supporting the concept of a synergistic effect.[12]

Lamont et al have developed a model that hypothesises the relationships between early life and later experience and adult disease.[13]

Figure 2: Relationships between early life and later experience and adult disease



Source: Spencer N. 2003. *Weighing the Evidence, How is Birth Weight Determined?* Oxon: Radcliffe Medical Press.

Although this model fails to acknowledge intergenerational effects of socioeconomic status, it does demonstrate plausible pathways by which factors in childhood may combine to influence adult health outcomes.[13]

Environmental factors

Cardiovascular disease, type 2 diabetes and obesity result from an interaction between genetic and environmental factors. However, the rapid rise in obesity and the parallel surge in type 2 diabetes in recent decades indicates the relative importance of environmental influences in contributing to this epidemic.

Modern children live in an 'obesogenic' environment. Mechanisation has resulted in many manual tasks becoming redundant and hence physical activity as a part of daily life has been reduced. The ease and speed of modern transport promotes sedentary behaviour. Walking and cycling to school has declined in New Zealand.[14] Evidence suggests that modern inactive lifestyles are at least as, if not more, important than diet in the aetiology of obesity.[15]

Town planning and transportation, the physical environment and safety issues, availability, affordability and accessibility of facilities and healthy food – all influence individual behaviour, yet are largely outside an individual's control. Collaboration between government sectors, local government, non-governmental organisations and industry is required to create environments and infrastructures that support healthy food choices and physical activity.[7, 16]

Media

The media urges us to consume more. The food industry spends millions of dollars on advertising and much of it is targeted at children.[17, 18] The media is effective in influencing children's eating patterns and food choices. Even brief exposure to food commercials can influence children's preferences. Among children as young as three years of age, the amount of weekly television viewing is significantly related to their caloric intake, as well as their requests and parent purchases of specific advertised foods.[19] Television viewing is associated with increased consumption of fast foods and soft drinks and reduced consumption of fruit and vegetables in children and youth.[19, 20]

Most, but not all, large national cross-sectional studies in the US, and several longitudinal studies, indicate that children who spend more time watching television are more likely to be overweight than children who do not. Experimental interventions indicate that reducing the time children spend watching television may be an effective intervention for childhood obesity. The body of evidence indicates that media-related policies can contribute to a comprehensive effort to prevent and reduce childhood obesity.[19] The WHO recommends that governments take a collaborative approach with consumer groups and the food industry to develop appropriate approaches to deal with the marketing of food to children, and that media literacy skills be included in school curriculum.[7]

Fast food

In the US, consumption of fast food or restaurant food trebled between 1977 and 1996.[21]

> The impact of fast food on obesity is difficult to determine precisely, but no doubt increased consumption of take-away foods represents a major dietary change in society. Americans spend more on fast food than on movies, books, magazines, papers, videos, and music combined. This has occurred in conjunction with a deliberate policy by advertisers to market products directly to children.[22]

On average, New Zealand households spend 23 percent of weekly food expenditure on meals away from home or ready to eat foods.[23]

A large fast food meal (double cheeseburger, fries, soft drink, dessert) could contain 2200 kcal,[17] which is within the range of the total recommended daily intake of calories for a 12–15-year-old.[24] At 85 kcal per mile it would require a full marathon to metabolise this.[17]

Results of several studies have shown an association between fast-food consumption and total energy intake or body weight in adolescents and adults.[17] A nationally representative study in over 6000 American children and adolescents has recently confirmed this. The findings of Bowman et al suggest nearly one-third of children in the US eat fast food on a given day and this is associated with an increased caloric intake of 187 kcal.[25] Fast food consumption in children is also associated with reduced intake of fruit, vegetables and milk.[25, 26]

Portion sizes

Data from the US shows that in recent decades food portion sizes have increased markedly, both for food consumed at fast food outlets and in the home.[27] Bigger portions equals more calories. Several studies have shown that food portion sizes are positively related to energy intake in children and adults.[28]

Evidence suggests that during infancy and toddlerhood eating is primarily in response to hunger and satiety cues. However as children develop their food intake is increasingly influenced by environmental and sociocultural factors. Rolls et al investigated this idea in a study of preschool children and found that portion size influenced food intake of five-year-olds but not three-year-olds.[29]

Sociocultural factors

Family

Families have a strong influence on children and young people. Other environmental effects on children are mediated through the family.[30] Parents and other family members role model health-related behaviours such as physical activity, dietary habits and smoking to their children. The home environment and family lifestyle affects behaviours related to the risk of obesity. A bedroom television increases viewing by an average of 38 minutes per day.[17] There is a positive correlation between hours of viewing and overweight.[31] Television viewing promotes weight gain not only by reducing physical activity, but also by increasing energy intake. Social support from parents and others correlates strongly with participation in physical activity.[17]

Children consume more energy when meals are eaten in restaurants rather than at home, but constructive behaviours like eating a family dinner can reduce television viewing and improve diet quality.[17] Evidence from observational and case-control studies suggests a powerful role for child-feeding practices in shaping how much children eat and the extent to which children are responsive to the energy density of the diet in controlling their food intake. Children's responsiveness to energy density is diminished when adults use control strategies that focus children on external cues to encourage consumption.[32]

Parental obesity is a risk factor for future obesity. Parental obesity more than doubles the risk of adult obesity for children under the age of 10, whether or not they themselves are obese. In children under three years of age, the primary predictor of obesity in adulthood is the obesity status of their parents. The child's obesity status at this age is not an indicator of the risk of adult obesity. Among older children, childhood obesity is an increasingly important predictor of adult obesity. After the age of 10 years, the child's obesity status is the main predictor of adult obesity.[33]

> Results of observational studies show a direct relation between maternal obesity, birthweight, and obesity later in life; however, the relative contributions of shared maternal genes versus intrauterine factors are difficult to differentiate.[17]

Findings of studies in animals indicate the potential long-term consequences of maternal obesity per se – implying the obesity epidemic could accelerate through successive generations independent of further genetic or environmental factors.[17]

It is important that obesity in children and adolescents be treated within a family context. There is even some evidence to support the involvement of parents alone in interventions for obesity in children of primary school age.[31]

Schools

Schools are in a key position to influence behaviour. The New Zealand health and physical education curriculum aims for students to enhance their personal health and physical development, develop motor skills and positive attitudes towards physical activity, enhance their relationships with other people, and participate in creating healthy communities and environments by taking responsible and critical action.[34] The WHO advocates for not only health, nutrition, and physical activity education, but also media literacy skills to be taught in schools.[7]

School-based interventions have had varying and often limited success in improving health-related behaviours or reducing the prevalence of obesity.[17] However, some programmes have achieved significant results. One such example is the Singaporean TAF (Trim and Fit) Program launched in 1992.

> It involved all primary and secondary schools, with the aim of developing strategies to reduce the prevalence of obesity and improve the physical fitness in the entire student population. Overweight children went into a remediation program, and those who were severely overweight received additional management through the School Health Service. The prevalence of overweight dropped from 14 percent in 1992 to 9.9 percent in 1998.[31]

The WHO recommends daily physical activity as part of school curriculum.[7] It has been noted in New Zealand that the amount of physical education taught in schools is declining.[14] In the National Children's Nutrition Survey (NCNS) one in five 5–10-year-olds, and one in ten 1–14-year-olds, reported having no physical education class in the previous seven days.[35]

Schools can influence students' food choices through the affordability and availability of food and drinks that are offered or sold at school. The American Academy of Pediatrics has recently issued a policy statement aimed at eliminating the vending and advertising of soft drinks in schools.[36]

Many schools in New Zealand have been concerned about the numbers of children coming to school hungry and have taken steps to address this.[37] In Manukau City, Te Ora o Manukau – Manukau the Healthy City initiative, a collaboration of local agencies, provides more than 1300 meals per day in 41 schools.[38] The Ministry of Health is currently undertaking a pilot project in low decile (1 and 2) schools in greater Auckland (20 schools) and Northland (five schools), providing fresh fruit daily. This trial's evaluation report will be available later this year, but current feedback is positive.[39]

Schools can also be effective in reducing smoking. A US report of the Surgeon General, *Preventing Tobacco Use among Young People*, states that school-based smoking prevention programmes that identify social influences to smoke and teach skills to resist those influences have demonstrated consistent and significant reductions in adolescent smoking prevalence.[40]

Health Promoting Schools (HPS) are an example of collaboration between the Ministries Health and Education to pursue shared objectives. Health Promoting Schools were developed under the principles of the Ottawa Charter for Health Promotion.

> A health promoting school is one that works in a way which demonstrates a whole school commitment to improving and protecting the health and well-being of the school community.[41]

The concept of HPS was first introduced in New Zealand in 1997, through pilot schemes. Evaluations of these pilot schemes found that HPS were being implemented in very different ways with a wide variation in levels of achievement. Lack of leadership from the Ministry of Education was identified as a major barrier in determining the success of HPS. The evaluators believed that links with the Health and Physical Education curriculum could be made more explicit and that the role of the Ministry of Health in communicating and collaborating with the Ministry of Education was very important in this context.[42]

Currently it is estimated that there are at least 350 HPS in New Zealand, but the exact number is not known. There is a lack of national resources and tools, and no national co-ordination or infrastructure. Auckland Central is the only area in which the concept has been extended to preschools.[43] There is great potential for further development here, as early childhood education centres could also be instrumental in health promotion.

Ethnicity and culture

We cannot underestimate the pervasive effect of social structure on health. 'In particular, the role of discrimination in underpinning deprivation and directly contributing to the excess mortality experienced by Mäori and Pacific peoples warrants further attention'.[8]

Forty percent of the babies born in New Zealand in 2001 were of Mäori or Pacific ethnicity.[44] The changing demographics of New Zealand society have significant implications for the nation's future wellbeing and prosperity, as these children currently experience worse health and educational outcomes than their counterparts.

The NCNS data show much higher rates of obesity and larger girths for Pacific and Mäori children compared with New Zealand European and other ethnicities (NZEO).[35] In this survey the Cole et al (2000) reference cut-off values were used for determining the prevalence of obesity and overweight.[45] One of the recognised limitations of using these internationally recognised cut-off values is the possible inappropriateness for the different ethnic groups in New Zealand. There have been two notable studies in this area in New Zealand. Tyrrell et al found no clinically significant differences in the relationship between body mass index (BMI) and body composition in different ethnicities.[46] They recommend for New Zealand children that the same BMI value be used to identify obesity in all ethnicities. However, Rush et al have suggested that BMI thresholds need to be raised by 3–4 units for Mäori and Pacific Island girls aged 5–14 years, and they have developed an equation for estimation of free fat mass (FFM) that they think is more suitable for the determination of body fatness in New Zealand children.[47]

Type 2 diabetes in children and youth in New Zealand is escalating and largely confined to obese Mäori and Pacific peoples.[48, 49] Complication rates for type 2 diabetes are higher and complications are more severe in Mäori and Pacific peoples compared with New Zealand Europeans.[50]

In New Zealand in 1996–99, cardiovascular mortality rates were three times higher for Mäori males and 2.5 times higher for Pacific males compared with non-Mäori non-Pacific males. Among females the corresponding relative risk was 4.2 for Mäori and 3.1 for Pacific people.[51] The relative risk for Mäori for cardiovascular death is higher than non-Mäori even after adjusting for socioeconomic position.[52] It is interesting to note that the relative risk for cardiovascular death for Pacific people is lower than Mäori even though their rates for obesity are higher and proportionally more live in relative poverty.

As Blakely and Dew (2004) have noted, Pacific culture seems to have protected Pacific people from the full impact of lower socioeconomic position. Research and understanding of the beneficial effects of culture on health is required in New Zealand, because it may identify positive policy options for inequalities in health.[54]

Food is a fundamental commodity in all cultures, but it can have more than just nutritional value. Food can be used as a means to show love and respect, to express hospitality and to bring people together. However, whether one culture places more importance on food than another is questionable. Traditionally among Pacific peoples, as in many cultures, obesity has been associated with prosperity. However, prior to the 1960s obesity was very rare in Pacific communities.[55] Any perception that obesity is regarded as desirable in Pacific culture is unjustified.[56]

Culture is a pervasive influence on an individual's expectations and behaviour. In order to promote wellbeing, health workers need to understand the cultural paradigms in which people live. Healthy food choices and ways of being physically active which are culturally acceptable need to be identified and promoted. Even the method of communication and delivery of services need to be verified as culturally appropriate if they are to be effective. For this purpose, Professor Thomas, from the Division of Community Health at the University of Auckland, has developed a framework for assessing the cultural appropriateness of health programs or services in New Zealand.[57]

The potential success of culturally appropriate interventions is demonstrated by Kids In Action – Pasifika Challenge. This is a community driven, multidisciplinary, family-centred programme in South Auckland. The idea for Kids In Action grew out of a direct request from parents of children in the Samoan immersion unit at Finlayson Park School to the South Seas Public Health nurses. An obesity intervention programme was developed using AAP guidelines that would meet the needs of local children. Initial results are encouraging. In 2003, of the 63 children who attended the programme for between 4–28 weeks, 70 percent either lost or maintained their weight over that time. However, those who lost weight did tend to gain over the holiday period when the programme was not running. The motivation of the child and family, and family involvement and attendance were identified as important factors in weight loss success.[58] The long-term sustainability of this success remains to be seen.

Socioeconomic status

The link between socioeconomic status and health is well established.[1, 6, 11, 59] Psychosocial influences relate to the social distribution of behavioural risks, but there is also evidence of direct connections between the psychological characteristics of social position and biological functioning.[60] Nor are the effects confined to the poor. The social gradient in health runs right across society.[11] However, the strength of the relationship may differ across different populations.

Social rank is determined by many factors, including:

- wealth and income
- gender roles
- ethnicity – an aspect of identity that partly determines social class but also determines access to opportunities, power and privileges independent of social class due to personal or institutional discrimination
- age – children are in a position of relative powerlessness
- disability.

Society is a finely graded hierarchy along these interacting axes. This hierarchical structure of society determines opportunities and risks, thereby determining health outcomes. Children, because of their inherent age-related disadvantage, fall to the bottom of the pecking order. In addition, a disproportionate number of New Zealand children live in relative poverty.

Poverty is a contested concept, especially in New Zealand, which is often thought of as a land of plenty. The New Zealand Poverty Measurement Project (NZPMP) has done extensive work analysing the incidence and severity of poverty in New Zealand since 1991.* The NZPMP's definition of poverty is a relative one.

> Poverty is a lack of access to sufficient economic and social resources that would allow a minimum adequate standard of living and participation in that society.

The NZPMP's measure of poverty is income based, but rather than set a poverty line at an arbitrary fraction of the mean or median household income, they have sought to address the issue of assessing adequacy.

> The approach involves the use of focus groups to draw on the knowledge and practical experience of low-income householders (ie, on their judgement) to estimate minimum adequate household expenditure in a full range of household expenditure categories.

* The NZPMP was undertaken by three organisations: Business Economic Research Limited (BERL), the Public Policy Group at Victoria University of Wellington (VUW) and the Family Centre Social Policy Research Unit (FCSPRU). The research leaders of the project were Paul Frater (BERL), Robert Stephens (VUW) and Charles Waldegrave (FCSPRU). Currently the project continues under the leadership of Robert Stephens and Charles Waldegrave.[61]

The NZPMP's preferred relative measure of poverty threshold is 60 percent of median, equivalent, disposable, household income after adjusting for housing costs. Using this threshold between 1993–98, around 19 percent of households were below the poverty line. A third of children and over 70 percent of the single-parent households were in poverty. Mäori were more than twice as likely, and Pacific people more than three times as likely, as New Zealand Europeans to be in poverty.[61]

These figures are similar to those from the Ministry of Social Development. In the year to June 2001, 29.1 percent of dependent children in New Zealand were living in relative poverty (economic family units below the 60 percent of 1998 median equivalent net-of-housing line). This is an increase from 27.5 percent in 1998 and is almost twice the proportion in 1988 (14.6 percent), but below the peak of 36.4 percent in 1994.[53]

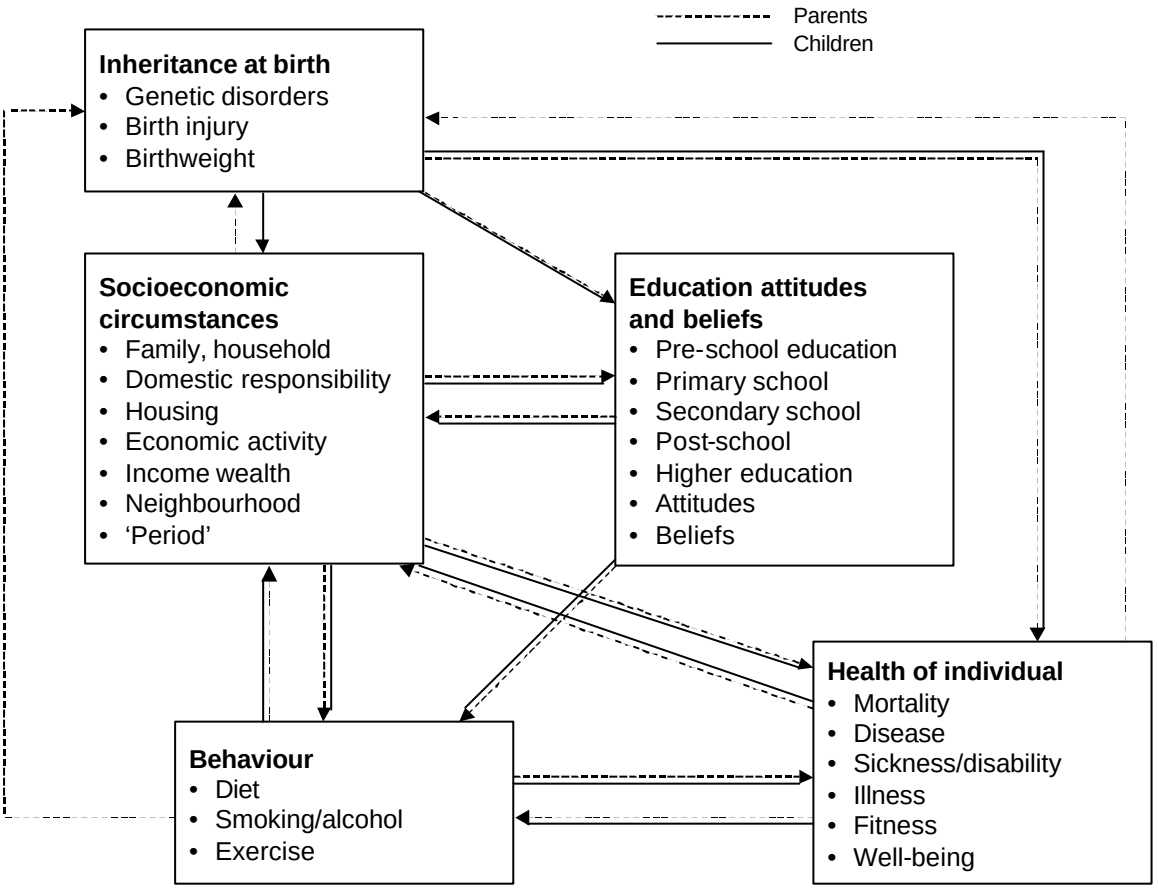
The Ministry of Social Development has developed the Economic Living Standard Index (ELSI) – 'an indicator of how people are living in terms of their possessions, activities and how they get by financially'. In 2000, 20 percent of the New Zealand population had low living standards according to this index. There are clear disparities, with 29 percent of children having low living standards according to this index, 39 percent of all Mäori, and 42 percent of all Pacific people.[53] Supporting this is evidence from Crampton et al who, by using census data, have produced an atlas of socioeconomic difference for New Zealand. This shows 56 percent of Mäori and 67 percent of Pacific peoples live in the most socioeconomically deprived areas (NZDEP 96 deciles 8, 9, and 10), compared with only 22 percent of NZEO ethnic groups.[62]

High housing costs relative to income contributes to financial difficulty and can leave low-income households with insufficient income to meet other basic needs.[16, 53] In 2001, the proportion of households spending more than 30 percent of their income on housing was 24 percent of all households, and 42 percent of households in the lowest quintile of household income distribution. Both of these figures have substantially increased since 1988, when they were 11 percent and 16 percent respectively.[53]

Income is used as an indicator of socioeconomic status, but may be a better predictor of health status than any other socioeconomic status indicator.[59] The National Health Committee, in a report on the social, cultural and economic determinants of health in New Zealand, states that 'income is the single most important modifiable determinant of health'.[1] Adequate income is a prerequisite for many other determinants of health, such as a nutritious diet and educational opportunities.[1] Disadvantages tend to concentrate among the same people, and their effects on health accumulate throughout life. People at the lower end of the social ranking usually run at least twice the risk of serious illness and premature death as those near the top, but the social gradient in health runs right across society.[11]

Socioeconomic position influences health through a number of pathways. Primarily this is either via material resources or psychosocial factors, which in turn are partly mediated by health behaviours. A model developed by Power et al depicts the complex interrelationship of social, behavioural, genetic and illness factors on health outcomes for parents and their children. From this it can be seen how poor socioeconomic environment leads to poor adult health and how this cycle is perpetuated.[13]

Figure 3: Inter- and intragenerational relationships between health and circumstance



Source: Spencer N. 2003. *Weighing the Evidence, How is Birth Weight Determined?* Oxon: Radcliffe Medical Press Ltd.

There is a strong consistent relationship between low socioeconomic status in early life and obesity in adulthood.[63] Both current socioeconomic status and that experienced during upbringing are significant predictors of adult health.[64]

Children living in areas of socioeconomic deprivation in New Zealand are more likely to be obese. The relative risk of obesity for children in the highest NZDep01 quintile (most deprived) compared with those in the lowest quintile is 3.2 for males and 4.5 for females.[35] And there is a two-fold increase in the risk of cardiovascular death for those people living in the areas with the lowest socioeconomic rating as compared with the highest, as measured by the NZDep score.[52] There is a body of evidence from prospective cohort studies that low socioeconomic status in childhood is associated with poorer health in adulthood independent of adult socioeconomic status.[2–5] In examining the influence of socioeconomic position over a lifetime on morbidity and mortality, Smith et al have concluded that the risk of premature death from cardiovascular disease is particularly sensitive to socioeconomic influences acting early in life.[4]

A longitudinal cohort study in Dunedin of 1000 children (born in New Zealand during 1972–73) showed that children who grew up in low socioeconomic status families had poorer cardiovascular health at age 26 years, compared with those from high socioeconomic status backgrounds. The longitudinal associations could not be attributed to lifecourse continuity of low socioeconomic status, and upward mobility did not mitigate or reverse the adverse effects of low childhood socioeconomic status on adult health.[2]

The relationship between low socioeconomic status in childhood and poor health in adulthood seems clear, but the mechanism by which it occurs is less well understood. Poverty is a root cause of overcrowding, increased infectious diseases, food insecurity and poor nutrition. All of these factors can adversely impact on a child's health, which may have enduring effects. A relevant New Zealand example of infectious disease associated with poverty and overcrowding with a clear causal pathway to cardiovascular disease in adulthood is the link between streptococcal infection and rheumatic fever and heart disease. Furthermore, chronic stresses associated with social position may alter neuroendocrine and immune functioning, with later consequences for susceptibility to disease.[60]

The biological explanation of how poverty in childhood can predispose children to obesity relates to altered function of the hypothalamic-pituitary-adrenal axis. Children living in low socioeconomic settings produce more cortisol, compared with children from high socioeconomic settings.[65] Cortisol promotes central fat deposition and it can also trigger depression, which further increases the risk of obesity.[60, 66] Additionally, sympathoadrenal and autonomic activity may mediate the link between psychosocial exposures and cardiovascular disease. This is supported by a case control study of adults with metabolic syndrome, a precursor of coronary heart disease. Brunner et al found that psychosocial factors explained a substantial part of the increased normetanephrine output associated with metabolic syndrome.[67] There is some evidence that exercise has a protective effect which limits the adverse effects of stress. Endogenous opioids, linked with vigorous exercise and psychological wellbeing, may be important in the neuroendocrine counterregulatory system by inhibiting pituitary release of adrenocorticotropic hormone.[60]

Furthermore, elevated circulating levels of inflammatory markers, and antibodies to common infective agents, have been shown to predict the development of diabetes and coronary heart disease. Adipose tissue is involved in the synthesis and release of cytokines. Yajnik, in a small pilot study in India, have found that the circulating levels of leptin and cytokines were elevated many times over in the urban subjects compared with the rural subjects, being highest in urban slum dwellers. Yajnik postulates that adipose-derived cytokines may mediate insulin resistance and that the higher adipose tissue mass in urban dwellers, possibly stimulated by the infected and polluted environment, may contribute substantially to these.[68]

In New Zealand nutritional deficiency is associated with socioeconomic disadvantage.[69] The NCNS shows that many New Zealand households cannot afford to eat properly. Mäori and Pacific families are affected to a much greater extent, with over one-third of Mäori and over one-half of Pacific households with children not always being able to afford to eat properly. Adequate nutrition is a prerequisite for health across the lifecourse. This is discussed in greater detail below.

A recent comparison of child benefit packages in 22 countries shows New Zealand to be a laggard in this area.[70] The issue for low income New Zealanders is that income-related benefits have been the only source of assistance, and families have been out-of-pocket in respect of accommodation, schooling and health care, which are subsidised in many other countries.[71] Primary Health Organisations are a step forward for the health sector in addressing this disparity.

Food security

Food security is defined as 'access by all people at all times to the food needed for a healthy life'.[72] It encompasses the ready availability of nutritionally adequate and safe foods and the ability to acquire personally acceptable foods in a socially acceptable way.

Hunger in a primary agriculture producing nation such as our own is a paradox, but not one that is unique to New Zealand. First world hunger is a reality in many developed countries.[73]

If people are to consider lifestyle changes for long-term health gains, then their basic needs must be met first. Achieving food security is fundamental to improving nutrition and pertinent to the other priority population health objectives of the New Zealand Health Strategy, reducing obesity and increasing the level of physical activity.[74]

Household food security has only become a public health concern in New Zealand within the last decade. *Food for Health – The Report of the Nutrition Taskforce to the Department of Health* (1991) makes no reference to food security. Consideration is given to the potential economic constraints of a food and nutrition policy. At that time, for a household with two adults and two school-aged children, a basic nutritious diet was estimated to cost $84.26 per week. The average weekly household expenditure on food (1998/89) was $92.31, and for a couple with two children it was $124.61. From this it was concluded that 'the cost of a nutritious diet should not be prohibitive, even for low income groups'.[75]

However three years later food security had emerged as a public health concern in New Zealand. In 1994 the Public Health Commission (PHC) published the Nation Plan of Action for Nutrition (NPAN), one of the overriding themes of which was 'improving household food security' (Public Health Commission 1996). This was in response to an increasing number of reports suggesting that socioeconomically disadvantaged New Zealanders were unable to afford sufficient quantities and quality of food to meet their nutritional needs, and the growth in the number of food banks. There was a particular concern about the effect of low income on the nutritional status of children.[76]

In 1995, a PHC report estimated that over 20,000 New Zealand schoolchildren were perceived to be inadequately fed and there was a greater problem in regions with a higher proportion of Mäori and Pacific peoples.[37]

A national randomised survey of 400 low income households throughout New Zealand found that 64 percent of respondents had been unable to purchase essential foods during the previous three months.[77] New Zealand research suggests that food quality is a discretionary budget item for many low income households after other essential bills have been paid.[77, 78] National case studies from Australia, Canada, the US and New Zealand concur that when household income is inadequate, it is the food budget, being the most elastic, which suffers, and individuals and families, including many children, go hungry.[73]

Otago University's average estimated weekly food costs in 2003 for a basic nutritious diet based on the New Zealand Food and Nutrition Guidelines were $49 for a man, $48 for a woman and for children between $24 and $64, depending on age and gender. These costs are calculated assuming home preparation of meals and using the lowest priced items within each food category purchased from supermarkets.[79] From the Household Economic Survey (2001), the average weekly household income before tax for the lowest quintile of the population was $321. The average weekly expenditure on food for those households was $72. The average number of persons in those households was 1.82.[23] This equates to $40 per person per week. By comparing this with Otago University's estimated food costs, these figures suggest that the cost of a nutritious diet has become prohibitive for many New Zealand households.

The 2002 National Children's Nutrition Survey revealed the extent of food insecurity affecting New Zealand children, with huge disparities between ethnicities and socioeconomic areas. About one-half of Pacific and one-third of Māori households, and one-tenth of NZEO households reported that food ran out 'often or sometimes'.[35] This is consistent with the findings from the 1997 National Nutrition Survey.[80]

The prevalence of food insecurity identified in the NCNS is paralleled by alarming rates of nutrient deficiencies.

- Vitamin A deficiency was prevalent in 37.4% of Pacific females.
- Iron deficiency. The highest prevalence occurred in Māori and Pacific females aged 11–14 years (11.2%; 9.6%), compared with New Zealand European and Others (NZEO) females (3.2%).
- Inadequate folate intake. Females aged 11–14 years estimated prevalence Māori 22.8%; Pacific 30%; NZEO 15%.
- Inadequate calcium intake. Overall prevalence was 15.1% with the highest prevalence among Pacific children (males 40.5%; females 45.2%).[35]

These nutrients are all vital to healthy growth and development. For example, vitamin A is necessary for maintaining eye health and vision, growth, immune function and survival. Children under five years of age and women of reproductive age are at highest risk of this nutritional deficiency and its adverse health consequences which include foetal loss, low birth weight, pre-term birth, infant mortality and increased susceptibility to infectious diseases. The incidence of vitamin A deficiency in Pacific Island female children in New Zealand is similar to rates in Africa and parts of Asia.[81] Iron is needed for basic cellular functions in all tissues of the body, and is critically important in muscle, brain and red blood cells. Young children, pregnant and postpartum women are the most commonly and severely affected because of the high iron requirements of infant growth and pregnancy. There is a growing body of evidence that iron deficiency anaemia in early childhood reduces later intelligence.[81] This may contribute to perpetuating a cycle of low socioeconomic status and poor health outcomes. There is also evidence that iron deficiency decreases fitness and aerobic work capacity, thereby impacting on cardiovascular health.[81] (Folate and calcium are discussed later.)

The available evidence on the diets of low income New Zealand households indicates that they tend to be not only too high in fat, salt, and sugar, but also too low in nutritious foods such as fruit, vegetables, lean red meat and dairy products (82). The evidence suggests this is due to cost rather than ignorance. New Zealand mothers, including those on very low incomes, have a broadly accurate understanding of children's food needs. However food is the flexible item in the household budget, and high energy, low nutrient food tends to be cheaper than healthier alternatives.[82, 83]

In 2003 the Ministry of Health produced a strategic framework for improving nutrition, increasing physical activity and reducing obesity, *Healthy Eating – Healthy Action* (HEHA). This document builds on the directions and recommendations of NPAN and supercedes it. The strategy acknowledges that for a number of New Zealanders healthy food choice is significantly reduced by lack of money and that food security is an important issue. HEHA identifies five key priorities including lower socioeconomic groups and children, young people and their families. Nutrition plays a key role in maintaining health and preventing disease. Policies and services that assist in making the healthy choice an easy and accessible option for people with very limited money are key to improving the health and wellbeing of the nation.[16]

Adequate nutrition is a prerequisite for health and wellbeing at any age. However, there are critical periods of development when the potential for micro-nutrient, vitamin and mineral deficiencies to impact on adult health is far greater. The lifelong nutritional status of a mother, from her own conception up until the birth of her baby, impacts on the health of that baby. The particular importance of nutrition in the months leading up to conception is now recognised. It is during early pregnancy that the foetus is most vulnerable to nutrient deficiency.[84] However the critical period of neurodevelopment, during which the developing to brain is particularly susceptible to irreversible damage from undernourishment, extends into early childhood. Therefore, given that a high proportion of

pregnancies are unplanned,* it is particularly important to ensure food security for all young women and also young children.

Ensuring food security at the household level requires:

- accessibility to food – to conventional food sources and transportation
- availability of food that is safe, and nutritionally and culturally acceptable
- affordability of food.[78]

* The Dunedin Health and Development Study reported that 60 percent of pregnancies to participants under the age of 25 years were unintended.[85]

Health care factors

Health care risk factors are primarily to do with access to and the quality of care. These in turn are influenced by more distal factors such as socioeconomic status and geographic location. However, unlike many other influences, health care risk factors are under the direct control of the health sector. Discrimination, both consciously and unconsciously, can contribute to health disparities. It is important that any health service or intervention is closely monitored, and evaluated and viewed through an equity lens, to ensure that it is contributing to reducing inequalities rather than creating or maintaining them. For this purpose the Ministry of Health, in conjunction with Public Health Consultancy, have produced a Health Equity Assessment Tool which comprises a set of questions to prompt consideration of how particular inequalities have come about and where effective intervention points to tackle them lie.[86]

Evaluation and monitoring are also crucial to ensuring quality of care. We cannot always predict the possible adverse effects a particular intervention might produce, so careful monitoring is required to ensure the *primum non nocere* principle is upheld and health care policies and services altered accordingly as new information becomes available. This is especially important because there is currently a paucity of reports on effective interventions in childhood, particularly in regard to demonstrating long-term success in reducing cardiovascular disease and type 2 diabetes in adulthood.

Health care providers, especially for primary care health care, can play an important part in prevention and the identification of high-risk groups. The promotion of healthy practices and behaviours by health care providers, taking a lifecourse approach, can reach a large part of the population and provide a cost-effective intervention. Routine enquiries as to dietary habits and physical activity and family history, combined with appropriate measurement of biological risk factors (such as weight, height and assessment of growth; blood pressure and serum cholesterol, if indicated), can identify high-risk individuals, and when done in an educative and supportive manner helps promote behavioural changes. Training of health personnel, dissemination of appropriate guidelines, and the availability of incentives are key underlying factors in implementing interventions.[7]

Behavioural factors

In examining behavioural risk factors it is important to recognise that individual autonomy and choice is constrained by social position and the physical environment.[9] Also individual behaviour is largely dictated by societal norms. Once a behaviour has become accepted as a social norm and once the supply industries (and local governments, town planners, transport authorities and architects) have adapted themselves to the new pattern, then maintenance of that situation no longer requires effort from individuals.[10]

Diet

Prevention of cardiovascular disease through intervention in early childhood is supported by the fact that dietary habits and food preferences are formed in early life and that diets consumed by families tend to persist in the new generation.[87]

Children's eating is influenced by exposure to and accessibility of foods, modelling behaviour of peers and family, the physiologic consequences of ingestion, and child-feeding practices.[32] The rate of weaning and the establishment of satiety levels has been an issue of some contention. It has been postulated that toddlers have a natural satiety mechanism that leads them to refuse food when they are adequately nourished. Parents may perceive the lack of regular food intake that they have become accustomed to in the child's first year of life to be of serious concern. Overriding the innate control mechanisms at this age by overfeeding toddlers is thought to result in the re-establishment of the threshold for satiety at a higher level, thereby encouraging overfeeding and increasing the risk of obesity in later life.[88] In the US, formula-fed infants show more rapid growth than breast-fed infants. It is speculated that the difference may be due to the heightened maternal control over the infant's intake in formula-fed infants. Research suggests that when given the opportunity, infants are capable of being responsive to the energy density of formula and adjusting intake accordingly; however, maternal control can override the infant's regulatory ability. Child-feeding practices that encourage or restrict children's consumption of foods may decrease the extent to which children use internal signals of hunger and satiety as a basis for adjusting energy intake.[32]

The general pattern in New Zealand children is for dietary quality and nutrient intake to decline with age. This is paralleled by an increase in overweight and obesity with age. Younger children are more likely to have healthier food intake and be more physically active, compared with older children.[35] Intervention strategies need to be directed at maintaining the healthy habits of young children into later childhood and adulthood.

Breastfeeding

There is difficulty in obtaining robust evidence for the effect of breastfeeding on obesity because we are dependent on observational studies with differing methodologies of varying rigour. Many studies are retrospective and subject to greater bias than prospective studies. Also there is a problem with definition – exclusive versus non-exclusive breastfeeding. Other reasons for discordant findings among studies include the large sample size needed to adjust for confounders, different studies measure confounders using different criteria and adjust for these to varying degrees, and the selection of disparate end points for measurement of obesity.[89]

The majority of cohort studies suggest that breastfeeding confers a small protective effect against subsequent obesity.[31]

In a systematic review of infant feeding and blood cholesterol, Owen et al found a consistent association between breastfeeding in infancy and increased mean serum total cholesterol levels and low-density lipoprotein in infancy, but lower levels in adulthood. They have projected that this modest (0.2 mmol/L) reduction in adult serum total cholesterol would result in a 10 percent reduction in cardiovascular disease. Although the causality of this relationship is yet to be established, they suggest that the mechanism may be via nutritional programming in early life resulting in long-term changes in cholesterol metabolism. Breast milk has a relatively high cholesterol content and high cholesterol intake in infancy reduces endogenous synthesis of cholesterol. However, whether this effect persists into adult life remains to be established.[90]

Known advantages of breastfeeding include reduced risk of infectious disease and reduced risk of food allergy. Also, breast milk contains optimal ratios of polyunsaturated fatty acids required for retinal and brain development.[91] This may improve neurodevelopment and even mitigate against the negative effects of maternal smoking during pregnancy on the foetus.[92] Breastfeeding is associated with lower blood pressure in later life.[93] Das attributes this beneficial effect to the significant amounts of long-chain polyunsaturated fatty acids (LCPUFAs) in breast milk. Breast-fed infants have been found to have significantly more LCPUFAs in their tissues, compared with formula-fed infants. Das proposes that the availability of adequate amounts of LCPUFAs during critical periods of growth prevents development of hypertension in adulthood. He suggests that perinatal supplementation of LCPUFAs may prevent hypertension in adult life.[94] This notion requires further investigation.

There are questions surrounding the optimum duration of breastfeeding, especially in regards to its possible impact on cardiovascular disease. This is due to an observed association between prolonged breastfeeding and reduced arterial distensibility in early adult life and increased rates of ischaemic heart disease in later life. An observational study by Leeson et al of 331 adults in Cambridge, United Kingdom, which links duration of breast feeding with reduced arterial distensibility in adult life may contain significant selection bias, as only 22 percent of the initial sample of 1526 agreed to participate and were investigated.[95] Further work is required to clarify if there is any causal association, and to establish the optimum duration of breastfeeding in relation to cardiovascular disease.

The WHO recommends exclusive breastfeeding for the first six months of life.[7] While New Zealand has relatively good initiation rates of breastfeeding, the rates drop off substantially by six weeks and again at three months.[96] There has been little improvement in New Zealand's breastfeeding rates for the past 10 years, and for Mäori and Pacific babies the rate has remained consistently lower.[97] Breastfeeding rates for 2002/03 at six weeks were NZEO 69%, Mäori 60% and Pacific 61%, and at three months were NZEO 55%, Mäori 47%, Pacific 50%. At six months the overall rate was 24% (Mäori 17%, Pacific 20%) for full or exclusive breastfeeding. There are significant regional variations.[98]

Fibre

There is debate over the recommended levels of dietary fibre intake for children. High fibre diets are not recommended for young children, but a gradual increase in dietary fibre is recommended from the age of two years.[99] However, studies in adults indicate that fibre may be as important as fat intake in the prevention or treatment of obesity. Under conditions of fixed energy intake, the majority of studies in adults indicate that an increase in dietary fibre increases satiety, reduces energy intake and is associated with weight loss. Howarth et al have conducted a review of studies examining the effects of dietary fibre on hunger and satiety. When energy intake is ad libitum, mean values indicate that consumption of an additional 14 g of fibre per day for more than two days is associated with a 10 percent decrease in energy intake. When increased fibre intake is sustained over several months, this is associated with weight loss (mean 1.9 kg over 3.8 months). This is modest, but similar in magnitude to results obtained in studies comparing high-fat and low-fat diets. More pronounced effects are seen in obese individuals.[100] More research in this area is required in children.

Fruit and vegetables

Consumption of fruit and vegetables is protective against cardiovascular disease and some common cancers. Possible mechanisms for the protective effect of fruit and vegetables include: antioxidant vitamins and minerals; folate, which lowers serum homocysteine; soluble fibre, which improves serum lipid profiles, and a blood pressure lowering effect via potassium.[101]

Fruit and vegetables form part of a healthy balanced diet for children, providing important vitamins and minerals necessary for optimal growth and development. In addition, eating habits established in childhood are an important influence on fruit and vegetable consumption in adulthood.[102]

The NCNS results show only two out of five New Zealand children eat fruit at least twice a day and only about three out of five eat vegetables the recommended three or more times a day.[35]

It is not known what level of fruit and vegetable intake is associated with the lowest risk of cardiovascular disease. The current 'gold standard' is the Mediterranean diet, for which the estimated mean population intake of fruit and vegetables is 600 g per day. However it is possible that maximal benefit may be obtained from even higher intakes of fruit and vegetables.[101] Inadequate vegetable and fruit consumption was estimated to have contributed to 1600 deaths alone in New Zealand in 1997.[8] Turley et al have estimated that only a modest increase (40 g per day, ie, half a serving) in mean population intake now could prevent 334 deaths per year from 2011.[101]

Epstein et al have shown in a family-based intervention to prevent childhood obesity that promoting increasing fruit and vegetable intake can be more effective in achieving behavioural change and parental weight loss than trying to reduce fat and sugar intake.[103]

Fat

Population, clinical, and experimental studies of adults have shown a strong significant positive association between dietary fat (particularly saturated fat) and serum total cholesterol levels.[101] Saturated fat may have adverse cardiovascular effects, in addition to increasing cholesterol. Saturated fats interfere with the formation of vasodilator prostaglandins, elevate blood pressure and exacerbate spontaneous hypertension.[94] Conversely, polyunsaturated fats are cardioprotective. Fish oil, a rich source of omega-3 polyunsaturated fat, reduces blood viscosity and lowers blood pressure.[104] Long-chain polyunsaturated fatty acids (LCPUFAs) have multiple actions similar to statins that reduce the risk of major vascular events.[94]

There are concerns that low-fat diets in young children may cause growth failure because of inadequate energy supply. A Finnish study of children aged 7–36 months of age (n=540 intervention children) showed that repeated, individualised counselling in early childhood aimed at reducing consumption of saturated fat and cholesterol was effective and did not restrict growth.[87]

A six-centre, randomised controlled clinical trial in 663 American children aged 8–10 years showed dietary changes are effective in achieving modest lowering of LDL cholesterol over three years while maintaining adequate growth, iron stores, nutritional adequacy and psychological wellbeing during the critical growth period of adolescence.[105]

New Zealand recommendations are that after two years of age children should gradually adopt a diet that by about five years of age has the same fat recommendations as adults (ie, total fat intake to provide 30–35 percent of total energy).[99] Results from the 2002 National Children's Nutrition Survey (NCNS) showed the mean contribution to daily energy from total fat was lower for NZEO children (males 32.6%; females 32.3%) than for Mäori (34.2%; 43%) and Pacific children (35%; 34.3%) (Parnell et al 2003).

The target set by the New Zealand Department of Health in 1991 for saturated fat intake in 2000 was 8–12 percent of energy.[101] Results from the NCNS suggest we are well off that mark, with saturated fat contributing 14.5 percent to daily energy intake in 5–14-year-olds. The main sources of saturated fat in the diets of New Zealand children are milk, potatoes, kumara and taro, biscuits, pies and pasties, dairy products and processed meats.[35]

Carbohydrate

Dietary carbohydrates are digested and converted into glucose. Historically carbohydrates have been classified according to saccharide chain length, and hence referred to as 'complex carbohydrates' or 'simple sugars'. The glycemic index is a relatively recent alternative classification system for foods containing carbohydrate based on blood glucose response. In general, refined starchy foods have a high glycemic index, whereas non-starchy fruit and vegetables have a low glycemic index.[106]

Ingestion of meals with a high glycemic index produces a high blood glucose that stimulates insulin secretion. This is often followed by reactive hypoglycaemia. Thus, as Ludwig proposes, 'the habitual consumption of high-glycemic index foods may increase risk for obesity, type 2 diabetes, and heart disease, a hypothesis that derives considerable support from laboratory studies, clinical trials, and epidemiological analyses'.[106] Warren et al have shown in a three-way crossover study of 37 children aged 9–12 years, that low glycemic breakfasts can reduce subsequent intake. Consumption of a low glycemic breakfast, compared with a high glycemic breakfast, reduced lunchtime intake by 145 ± 54 kcal.[107] There is a body of evidence supporting the use of glycemic index as a dietary guide. As Ludwig suggests, the public health message can be simple: increase consumption of fruits and vegetables, choose whole grain products, and limit intake of potatoes, white bread and sugar. These recommendations would tend to promote diets high in fibre, micronutrients and antioxidants, and low in energy density.[106]

Among school-aged children, total energy intake is positively associated with soft-drink consumption. It has been shown that children drinking an average of 265 ml or more of soft drinks daily consume about 835 more kilojoules than those not drinking soft drinks.[108] An observational study by Ludwig et al (2001) followed 548 children with an average age of 11.7 years over a 19-month period and found a weak but statistically significant association between sugar-containing beverage consumption at baseline and BMI at the end of the study (0.18 kg/m^2 per daily serving, 95% CI 0.09–0.27), adjusted for body size at baseline and demographic variables including physical activity, television viewing and total energy intake. Intake of sugar-sweetened drinks increased from baseline to follow-up. The change in consumption of sugar-containing beverages was associated with both BMI at the end of the study (0.24 kg/m^2 per daily serving, 95% CI 0.1–0.39) and with the incidence of obesity in 398 children who were non-obese at baseline (OR 1.6 (1.14–2.24)).[108]

One-quarter of the total sugar intake for New Zealand children comes from beverages.[35] In addition to increasing energy consumption, soft drinks may affect diet quality by displacing milk consumption and lowering calcium intake.[109] In the US, children's consumption of sugar-sweetened beverages, sugars and sweets and sweetened grains is associated with increased saturated fat intake and decreased fibre intake. It also decreases the likelihood of children meeting the recommended intakes for calcium, folate and iron,[110] and reduces mean intakes of vitamin A, vitamin C, riboflavin and phosphorous.[109]

Sodium

Dietary sodium is an important determinant of blood pressure.[101] The main source of sodium in the diet is salt. Population studies have generally found a positive association with habitual high sodium intake and increased blood pressure. In populations in which there is a lifetime exposure to high salt intake, blood pressure levels, especially systolic, rise dramatically with each successive decade. This is in contrast to populations with a very low intake of sodium, who do not experience an increase in blood pressure with ageing.[111]

Data from observational studies and dietary sodium restriction trials in adults, generally suggest that a decrease in dietary sodium of 100 mmol per day could lower systolic blood pressure by up to 6 mmHg, depending on age and baseline blood pressure.[101]

Over one-third of New Zealand children usually have salt added to their meals during food preparation, and about a half add further salt to their meals at the table. NZEO children are significantly less likely to have salt added during food preparation or at the table compared with Pacific and Mäori children.[35]*

Calcium

A large US study (CARDIA) of young adults (aged 18–30 years) has shown a strong inverse association of dairy consumption with the insulin resistance syndrome (obesity, glucose intolerance, hypertension and dyslipidaemia) among those who are overweight. Pereira et al conclude that increased dairy consumption may reduce the risk of type 2 diabetes and cardiovascular disease.[112] This has significant implications for New Zealand and not only for our dairy industry. Pacific children, followed by Mäori, are most likely to have inadequate calcium intake, which reflects consumption of milk and dairy products.[35] This is the same population that is most at risk for obesity and type 2 diabetes. The cultural appropriateness of increasing dairy products in Mäori and Pacific children may be questionable, and perhaps increasing the intake of more traditional foods such as fish (which is a source of calcium and cardioprotective omega-3 oils) may be more appropriate. This area warrants further research.

Calcium intake is also relevant to cardiovascular disease, as an adequate amount of dietary calcium is associated with lower blood pressure. There is no significant difference between calcium from dairy and calcium from non-dairy sources with respect to the effect on reducing blood pressure.[104] Also, calcium may be a factor in regulation of body weight. A prospective study in preschool children found that calcium intake was negatively related to percentage body fat and total body fat.[31]

* The iodine status of New Zealand children is low and indicative of mild iodine deficiency.[35] As iodised table salt has been a key method of preventing iodine deficiency, public health strategies to reduce salt intake also need to consider alternative methods of improving iodine intake in our population.

Vitamin D

Vitamin D is a steroid hormone that is involved in regulation of calcium metabolism. Vitamin D3, cholecalciferol, is either produced in the skin by the action of UV B light on 7-dehydrocholestrol or it is obtained from dietary sources (fatty fish, cod liver oil, butter and liver). It is then activated by hydroxylation, first in the liver, then in the kidney. Dark-skinned people produce less vitamin D because melanin absorbs UV light. Inadequate calcium intake aggravates vitamin D insufficiency. Also, glucocorticoids (eg, cortisol) inhibit vitamin D-dependent intestinal calcium absorption.[113]

Some, but not all, studies in adults have shown that supplementation with vitamin D or exposure to UV B light can reduce blood pressure in hypertensive patients.[114] The mechanism by which this occurs is not entirely clear, but it has been observed in a mouse model that vitamin D can down-regulate rennin and angiotensin and thereby decrease blood pressure.[115] There is also a suggestion that vitamin D has a cardioprotective effect through its modulation of inflammation. Vitamin D deficiency has also been linked to diabetes. Experimental studies have shown that decreased vitamin D activity can result in both increased insulin resistance and reduced insulin secretion.[114]

If vitamin D is as important in preventive medicine as Zittermann and Holick propound, there are significant implications for child health. Because breast milk is a poor source of vitamin D, exclusively breast-fed infants are at risk of vitamin D deficiency, especially if they live at extreme latitudes and therefore have inadequate exposure to sunlight.[116] Dark-skinned children face an additional risk. Obese individuals also have an increased risk for low circulating vitamin D due to fat storage of vitamin D and its precursors.[114] Vitamin D supplementation is recommended for breast-fed infants in Alaska.[116] Holick advocates for annual screening for vitamin D deficiency and makes recommendations regarding sun exposure.[115] Because New Zealand is in a unique geographical location, with relatively high UV exposure and concomitant high rates of skin cancer, specific recommendations appropriate to our population and the environment we live in are required.

Physical activity

The benefits of physical activity in adulthood are well documented – it is important for reducing the risk of cardiovascular disease, type 2 diabetes, and obesity. Regular physical activity is associated with reduced risk of cardiovascular disease morbidity and mortality. More intense and frequent activity confers greater protection; that is, there is a dose-related effect. In adults the protective effect of physical activity is greatest in individuals at higher risk of cardiovascular disease.[52, 117] Results in obese and hypertensive children are consistent with this, indicating that those in the high percentile for fatness and blood pressure can benefit the most from an increase in physical activity.[118]

Studies of the relation of physical activity or energy expenditure to BMI or body fatness in children have had inconsistent findings, but are generally suggestive of a protective effect of activity against developing obesity.[63] A recent Australian study of 5–10-year-olds has shown a significant inverse correlation between body fat and BMI with physical activity levels (r=-.43, p=0.002 and r=-0.45, p=.001), which is consistent with findings in other

studies of both younger children and adolescents which have shown correlations with physical activity and percentage body fat of -0.52 and -0.53 respectively.[119]

Regular moderate-intensity physical activity reduces the risk of developing type 2 diabetes.[120] The most compelling evidence has come from population-based cohort studies, where the risk of developing diabetes is lowest in those who are physically active, even after adjusting for BMI. Between a third and a half of new cases of type 2 diabetes could be prevented by regular, moderate, physical activity and this effect appears to be strongest in individuals at highest risk. Secondary prevention of diabetes is also importantly associated with physical activity. For those who have diabetes, physical activity may improve glucose metabolism, increase insulin sensitivity and prevent an increase in atherosclerosis.[117]

A recent British study indicates that modern children establish a sedentary lifestyle at a young age.[121] A New Zealand study of trends in health-related physical fitness of 10–14-year-old children suggests that both health and physical fitness among this age group have deteriorated over the last decade.[14] This is consistent with statistics from Sport and Recreation New Zealand (SPARC) that suggest that activity levels for young people may be declining overall, with a change from 69% active in 1997–98 to 66% in 2000–01.[122] Data from SPARC indicate that for 5–17-year-old New Zealanders 9% are sedentary (no activity in last two weeks) and 22% relatively inactive (less than 2.5 hours physical activity in the last week). This indicates that one-third of New Zealand young people are inactive. The SPARC survey shows inactivity rates for Mäori are similar, but Pacific young people are much less active, with 19% sedentary and a total of 48% inactive. However this is not consistent with the NCNS survey, which found Pacific children had the lowest proportion in the least active group.[35] This may reflect the fact that the SPARC survey includes an older age range with much lower levels of physical activity. There are more Mäori young people (50%) who are highly active (five or more hours of physical activity per week), compared with NZEO (45%) and Pacific (33%) young people. For young people participation in sport or active leisure noticeably declines at school leaving age.[122]

Cross-sectional national data from the US suggests that youth who participate in sport teams or exercise programmes are less likely to be overweight.[123] The strongest positive predictors of whether New Zealand youth will be involved in sport are the perceived importance of sports to them, and parental participation.[124]

The following benefits arising from childhood physical activity are postulated by Boreham and Riddoch:

1. Direct improvement in child health status; evidence is accumulating that more active children generally display healthier cardiovascular profiles, are leaner and develop higher peak bone masses than their less active counterparts.
2. A biological carry-over effect into adulthood, whereby improved adult health status results from childhood physical activity.
3. A behavioural carry-over into adulthood, whereby active children are more likely to become more active (healthy) adults.[125]

There is some evidence that reducing sedentary behaviours in obese children is as effective for weight management as increased activity.[31] Television viewing is one measure of physical inactivity. National Children's Nutrition Survey data suggest that 27 percent of New Zealand schoolchildren watch on average more than two hours of television a day on school days and in the weekend this figure rises to over 40 percent.[35] The American Academy of Pediatrics recommends that television viewing for children is limited to 1–2 hours per day.[126] This recommendation could be actively promoted to all New Zealanders.

There is some limited evidence that increasing physical activity improves weight-loss outcomes in children or adolescents and may be effective by itself if vigorous.[31] However, there is need for research into the optimal physical activity prescriptions for effective weight loss in obese children and adolescents.[31] Future developments recommended by Boreham and Riddoch include the implementation of large-scale, longitudinal studies spanning childhood and young adulthood, the further refinement of tools for measuring physical activity accurately in young people, and research into the relative strength of association between fitness, as well as activity, and health in children.[125]

The fact that New Zealand children have high rates of participation in sports and leisure activities up until school-leaving age[122] suggests that this transition period is a key area for strategies to improve physical activity levels to focus on. Young people need to be encouraged to participate in physical activities that are enjoyable and will be sustainable over their lifetime. Perhaps less emphasis on high level competitive team sports, and more exploration and encouragement of alternative physical activities that can more easily be continued once young people enter the workforce, would improve the sustainability of physical activity levels. The decline in physical activity at school leaving age is probably also a reflection on the employment environment, which could be encouraged to be more supportive of its employees in maintaining physical activity.

Smoking

Risk of coronary heart disease is two-three times higher in smokers. The relationship between smoking and cardiovascular disease is continuous.[52] Cigarette smoking is associated with increased cholesterol and selected lipoproteins and lower blood pressure and weight. This corresponds with an increased risk of coronary heart disease due to smoking in individuals who initiate the habit at a young age. Smoking among girls increases health risks further when associated with oral contraceptive use.[127]

Although the length of exposure to smoking may be relatively short in young people, the adverse cardiovascular effects of smoking are already evident in pathology studies of youth. The extent of fatty-streak lesions in the coronary vessels of children and young people at autopsy was significantly higher in cigarette smokers than in non smokers in both the Bogalusa Heart Study and the PADY study.[12]

Smoking is highly addictive and the risk of cardiovascular disease associated with it increases with length of exposure. Therefore prevention needs to be directed at young people before they develop the habit.

Children and young people can be exposed to smoking either passively or actively. There are intergenerational effects of smoking through in utero exposure of the foetus and passive smoking by infants and children whose caregivers smoke. Data from the Dunedin Multidisciplinary Health and Development Study suggests about 40 percent of children are exposed to second-hand smoke.[128]

Maternal smoking is an important determinant of birth weight. There is a consistent dose-response relationship with increased smoking associated with decreased birth weight. A large Swedish study has estimated that maternal smoking of more than 10 cigarettes per day more than doubles the risk of an infant's birth weight being less than 2500 g.[13] Birth weight is a major determinant of mortality and morbidity in infancy and childhood.[13] If the association between low birth weight and increased cardiovascular mortality is causal, then this provides further support for efforts aimed at preventing girls from starting to smoke and encouraging young women smokers to quit.

Adoption of smoking during childhood is a determining factor in the establishment of smoking behaviour for most individuals.[127] In New Zealand, as in other developed countries, most adult smokers started by 18 years of age. Smoking prevalence increases rapidly during the late teens so that smoking prevalence rates among 18-year-olds resembles adult rates.[126] Smoking among adolescents in New Zealand is increasing. Self-reported rates of daily smoking for 14–15-year-old school students in 2000 were 14 percent for males and 16 percent for females. About twice this number reported smoking at least monthly. Smoking rates for Mäori youth were double for males and treble for females compared with NZEO.[128]

A Health Sponsorship Council survey of 14–17-year-olds found that the home was the most common place for smoking to occur.[128] This suggests that a family-based approach to curbing smoking in youth is warranted.

The US Surgeon General's report, *Preventing Tobacco Use among Young People*, concludes that 'community-wide efforts that include tobacco tax increase, enforcement of minors' access laws, youth- oriented mass media campaigns, and school-based tobacco-use prevention programs are successful in reducing adolescent use of tobacco'.[40]

Biological factors

Birth weight

The relationship between birth weight and obesity is not clear. In their review of the literature, Parsons et al found good evidence for a relationship between higher birth weight and increased fatness, but in studies which attempted to address potential confounding by gestational age, parental fatness or social group, the relationship was less consistent. They concluded that the extent to which the observed relationship is due to a direct effect of the intrauterine environment, or to genetic or other factors, is unclear.[63]

However, there is a consistent J-shaped relationship between birth weight and cardiovascular mortality, with a decline in risk with increasing birth weight, but those babies in the heaviest birth weight having a slightly increased risk.[13] Multiple observational studies have linked low birth weight with adult morbidity and mortality, including hypertension, type 2 diabetes and coronary heart disease.

Birth weight is a proxy indicator for the foetal environment. Barker's hypothesis is that coronary heart disease originates in utero through the persistence of physiological adaptations that the foetus makes when it is undernourished.[161]

Barker's hypothesis has been extended to encompass 'catch-up' growth, after a study in Finland of 3641 men showed that the highest death rates from coronary heart disease occurred in males who were thin at birth but were average or above average body mass from the age of seven years, suggesting that prenatal undernutrition followed by improved postnatal nutrition places children at increased risk of coronary heart disease.[129] Similar observations have been made for the increased incidence of type 2 diabetes in babies who were thin in infancy and subsequently have accelerated weight gain.[130] This phenomenon has been described as the 'thrifty phenotype'. The theory is that nutritional deprivation in the foetus during critical periods of development results in metabolic and physiological programming that then becomes maladaptive if the individual is exposed to a contrasting high level of nutrition.[131] This theory is supported by the observation that in a cohort of men born around the time of the Dutch famine (1944–45), exposure to famine during the first half of pregnancy resulted in significantly higher rates of obesity. However, although birth weights are not reported in this study, it is noted that famine exposure during the third trimester of pregnancy retarded foetal growth, yet exposure to famine during this time and the first months of life produced significantly lower obesity rates.[132]

Barker's hypothesis is contested by some. Lucas et al have pointed out that when size in early life is related to later health outcomes only after adjustment for current size, it is probably the enhanced postnatal growth (centile crossing), rather than foetal biology, that is implicated.[133] This is consistent with the findings of Williams and Poulton (2002) that later blood pressure is related to postnatal growth rather than birth weight.[134] It is the disharmony between foetal growth and later growth rates that seems to be the best predictor of later pathology.[131]

Even if the association between restricted foetal growth and adult chronic disease is causal, the evidence is consistent in suggesting that restricted foetal growth plays a minor role in terms of public health impact. As Kramer states, 'a disparity ('ecologic dissonance') between observed geographic and temporal trends and those expected under the programming hypothesis undermines the public health importance of the effects suggested by the hypothesis'. For example, the rising coronary heart disease mortality in Eastern Europe was not preceded by a corresponding downward trend in birth weight.[135] Similarly, for Indian people the cardiovascular epidemic is a recent phenomenon, but the birth weight of Indian babies has been low for centuries.[68] Kramer suggests that any effect of foetal growth on subsequent coronary heart disease is dwarfed by lifestyle factors, such as diet, physical activity, and smoking.[135]

Obesity

The WHO defines overweight in adulthood as a BMI of at least 25 kg/m^2 and obesity as a BMI of at least 30 kg/m^2. However, risk of disease in all populations increases progressively from adult BMI levels of 20–22 kg/m^2.[81] Body mass index in childhood changes substantially with age. Cole et al have developed a definition of overweight and obesity in childhood, based on pooled international data for BMI and linked to the adult cut-off points.[45] This is the definition used in the NCNS.[35] In clinical practice BMI percentiles are used, with a BMI above the 85th percentile suggesting overweight and a BMI above the 95th percentile suggesting obesity. BMI is significantly associated with body fatness in children and adolescents and has been validated against more direct measures of obesity.[31]

The NCNS identified that a third of New Zealand children are either overweight (21.3%) or obese (9.8%).[35] There is an increasing proportion with increasing age, which is more significant for females. Obesity is more prevalent in Mäori (males 15.7%, females 16.7%) and Pacific (males 26.1%, females 31%) children, compared with New Zealand European and other. The NCNS 2000 was the first national survey of this kind for children, so there are no previous national data to compare the findings with, but it is generally recognised in New Zealand that obesity in childhood is escalating. This is confirmed by a study in the Hawkes Bay of 11–12-year-old children that showed that the proportion of obese children almost quadrupled between 1989 and 2000. The mean BMI increased from 18.1 to 19.8 over this time, a relative increase of 9.2 percent.[136]

Obesity is the result of chronic excess energy intake over expenditure. Cardiovascular risk factors and obesity cluster in childhood and track through to adolescence and adulthood.[12, 31, 137] There is a high incidence of additional cardiovascular risk factors, such as elevated blood pressure, hyperlipidaemia and insulin resistance, in obese children and adolescents.[138–140] Childhood obesity is also associated with physical and psychological morbidity, which impairs current wellbeing. Obesity in childhood is associated with orthopaedic problems, obstructive sleep apnoea, asthma, hepatic steatosis, gastro-oesophageal reflux, cholelithiasis, and low self-esteem.[31] The obstructive sleep apnoea associated with obesity independently contributes to increased insulin resistance and hypertension.[141, 142] Obese children are also more likely to suffer from low bone mass, leading to more fractures.[14]

Obesity in childhood is associated with increased adult cardiovascular morbidity and mortality. In a 40-yearar follow-up of a cohort of children who had been admitted to hospital in Stockholm between 1921–47 for obesity, excessive overweight in puberty (standard deviation weight score >+3) was associated with higher than expected morbidity and mortality in adult life. Interestingly in this selected historic cohort of Swedish children, excluding those with secondary obesity, there was a tendency to revert to a more normal weight in adulthood. The mean weight for height standard deviation score (SDS) fell from +3.4 before puberty to +1 SDS in adult age-groups. However, 47 percent were still overweight in adulthood (SDS >+1), with 20 percent having a SDS >+2.[143]

Obesity is a hallmark of type 2 diabetes. Of the young people attending the Auckland Regional Adolescent Diabetes Clinic in 2002, those with type 2 diabetes were all markedly overweight, with a mean BMI of 35 (range 28–42).[48] Although there is a strong hereditary component to the disease, the recent escalation in type 2 diabetes prevalence has occurred too quickly to be the result of alterations in the gene pool, emphasising the importance of environmental factors.[144]

Adverse effects of obesity on glucose metabolism are evident early in childhood. Total adiposity has been shown to account for about 55 percent of the variance in insulin sensitivity in healthy white children. Visceral fat in obese adolescents is directly correlated with hyperinsulinaemia and inversely correlated with insulin sensitivity. Both insulin resistance and inadequate insulin secretion are necessary for hyperglycaemia to develop. There is debate about which is the primary defect in the development of type 2 diabetes, but the clinical characteristics in children with type 2 diabetes suggests that the initial abnormality is impaired insulin action, compounded later with pancreatic β-cell failure. It has been proposed that hyperglycaemia may worsen both insulin resistance and insulin secretory abnormalities, thus promoting the transition from impaired glucose tolerance to diabetes.[144]

Obesity tracks from childhood to adulthood, and the predictive power of this association increases with age.[31] Although the studies on tracking of body weight in childhood are in general agreement, not all the studies use the same definition of obesity and there have been variable predictions. The persistence of overweight is a universal phenomenon – what varies from study to study is the degree of effect and the age from which the prediction is valid.[31] The fact that tracking is not complete gives hope that obesity in adulthood is not inevitable and lends support to intervention in childhood.

The Dunedin Multidisciplinary Health and Development Study of 1037 New Zealand children (born 1972–73), who were measured at 3, 5, 7, 9, 11 and 13 years, found that correlations between present and later BMI scores increased with age and were established by seven years of age. The correlation between weight at age seven years and weight at age 13 years was 0.71 ($p<0.002$). Subjects were divided into three groups according to BMI: light (<25th percentile), average (25th-75th percentile) and heavy (>75th percentile). Sixty percent of seven-year-olds and 65 percent of 11-year-olds in the heavy or light group remained in the same category by age 13 years.[145]

The Muscatine study of a cohort of 2631 school children aged 9–18 years in 1971, and followed into early adulthood, yielded similar results. From 48–75 percent of children in the upper quintile of BMI maintained this position as adults, with the degree of correlation highest for measurements obtained in older children.[146]

Fat distribution is more important, rather than obesity per se, as a predictor of coronary heart disease risk.[31, 52] Fat distribution in children and adolescents has a significant negative correlation with HDL cholesterol. Truncal obesity correlates with markers of metabolic syndrome. It has been shown that waist measurement can be used as a measure of fat distribution, as waist measurement alone has a strong negative correlation with HDL cholesterol.[31] A study in Crete of nearly 2000 school children (mean age 11.4 years) showed that waist circumference was a better predictor for cardiovascular disease risk factors (blood pressure; total, LDL and HDL cholesterol) than BMI.[147]

Dietz proposes three critical periods in childhood for the development of obesity: gestation and early infancy, the period of adiposity rebound that occurs between five and seven years of age, and adolescence. Obesity that begins at these periods appears to increase the risk of persistent obesity and its complications. However, the relative risks of the complications or persistence of obesity that originate at each of these 'critical' periods remain unclear. Age-specific therapeutic success rates have not been established.[138] Further research is required to identify the most cost-effective time and target for efforts to prevent and treat childhood obesity. However, these critical periods are linked. Maternal nutrition and weight impact on the health of the foetus. Underweight mothers are more likely to produce low birth weight (LBW) infants.[84] Undernutrition at important stages of foetal development may induce permanent physiological changes that predispose to obesity.[132] Maternal obesity increases the risk of gestational diabetes, hypertension and toxaemia during pregnancy and increases the risk of perinatal death and congenital malformations in the infant.[84] Given that parental obesity is a strong predictor of adult obesity for a child, reducing obesity in adolescents could reduce the risk of their offspring becoming obese adults. Therefore, as Ebbeling et al suggest, an opportune time to initiate obesity prevention might be before conception.[17]

Strategies aimed at the prevention of weight gain should be easier, less expensive and potentially more effective than those aimed at treating obesity after it has developed.[148]

Lipids

Plasma lipids circulate in association with proteins as lipoproteins. The largest and least dense of these are chylomicrons, followed in increasing order of size and density by very low density (VLDL), intermediate density (ID), low density (LDL), and high density (HDL) lipoproteins. The variations in density reflect the ratio of lipid to protein. HDL is considered to be antiatherogenic, in contrast to LDL, which is atherogenic.[149]

The protein components of lipoproteins are known as apoproteins. These protein components are possibly even better predictors of coronary artery disease than lipids or lipoproteins.[149] Apoprotein B is the major protein associated with LDL, VDL and IDL and is a good predictor of cardiovascular risk.[6]

Epidemiological observations clearly show an association between serum lipids and the probability of developing atherosclerosis.[146] Atherosclerosis begins in early childhood.[87, 150, 151] Progression of preatherosclerotic lesions depends on serum cholesterol concentrations during childhood.[87] Autopsy studies of children and young people have shown that the extent of arterial fatty streaks is positively associated with LDL-cholesterol and triglycerides (and also hypertension, impaired glucose tolerance and obesity), and negatively associated with HDL-cholesterol.[12, 150] Further evidential support for the relationship between childhood lipid levels and future coronary disease includes:

(a) lipid distributions of children in populations at low risk for adult coronary heart disease are much lower compared with high-risk populations [149, 152]

(b) children with elevated serum cholesterol levels often come from families in which there is a high incidence of coronary heart disease [149, 152]

(c) parent-child lipid levels correlate significantly[149, 152]

(d) children and adolescents with high cholesterol levels are more likely to have high levels as adults compared with the general population.[149]

Significant changes in lipid profiles occur during the developmental phase between birth and two years and again at puberty. The most dramatic changes in serum lipids and lipoproteins occur during the first year of life, with levels approaching those of school age children by two years of age.[149] Although the causality of the association between breastfeeding and lower total cholesterol and LDL in adults is yet to be established, Owen et al have hypothesised that high cholesterol intake in infancy, as occurs in breast-fed infants, may program long-term changes in cholesterol metabolism.[90]

Both genetic and environmental factors influence lipid levels. Adult serum cholesterol and LDL levels are influenced by adult adiposity, diet and other lifestyle factors. However, there is a high degree of tracking of lipid levels from childhood to adulthood.[90, 153] Cholesterol measurements obtained in childhood are predictive of adult levels of total and LDL cholesterol, with 25–50 percent of the adult cholesterol variability explained by childhood levels.[153]

The major dietary determinant of the difference in serum cholesterol levels between populations, for both children and adults, is the proportion of saturated fat in the diet.[152] A randomised control trial of 8–10-year-olds has shown that it is safe and efficacious to lower LDL-cholesterol in children of this age group by behavioural intervention directed at dietary manipulation.[105] Early intervention is supported by studies which show that the earlier serum lipids decrease in adults, the better the prognosis.[87]

Homocysteine

Homocysteine (a B vitamin) is an amino acid produced by the metabolism of methionine. There is a graded increased risk of cardiovascular disease with increased levels of homocysteine.[52] This is significant because serum homocysteine can be lowered by dietary folic acid.[52, 154] However, there is still contention over whether homocysteine is causative in the pathogenesis of atherosclerosis and whether normalising homocysteine reduces cardiovascular events.[52, 155] Evidence for cardiovascular risk reduction with folic acid supplementation comes from studies of patients with homocystinuria.[154] There is conflicting evidence from randomised controlled trials of vitamin B therapy in adults for secondary prevention of cardiovascular disease.[155]

The NCNS indicates that there is a high estimated prevalence of inadequate folate intake in New Zealand females aged 11–14 years (Mäori 22.8%; Pacific 30%; NZEO 15%) and Pacific males of the same age (25.5%).[35]

More work is required in this area in order to show a benefit for treating children with folate to prevent cardiovascular disease in adulthood. However, there is already good evidence for another benefit of folic acid supplementation. If taken during early pregnancy, folic acid reduces the risk of neural tube defects.[156] Mandated folate supplementation already occurs in North America. The high estimated prevalence of inadequate folate intake in our youth most at risk for teen pregnancy supports folate fortification in New Zealand.

Hypertension

Elevated blood pressure accelerates atherogenesis and increases the incidence of cardiovascular disease. The effect of hypertension is independent of the other risk factors and both systolic and diastolic blood pressure are related to cardiovascular risk.[157] The association between blood pressure and cardiovascular disease is continuous, with no lower threshold.[101]

Defining hypertension in children is controversial. Part of the difficulty arises because levels steadily increase from infancy through childhood and into adulthood. Also, accurate blood pressure recordings that are reliable are notoriously difficult to obtain in children. Serial measurements in relaxed conditions are required and these are compared with age-related percentile charts. Blood pressure levels above the 95th percentile on at least three separate occasions are considered abnormal. In Britain it is estimated that at most one percent of children have blood pressure consistently and appreciably higher than the 95th percentile.[158] Data for New Zealand is lacking, however rates are likely to be higher, particularly in Mäori and Pacific children, given the high incidence of renal disease in this population.[159]

There is disagreement over whether population screening, to identify children with hypertension should occur. It is recommended in America, but not in Britain.[156]

Results from the Bogalusa Heart Study showed that tracking to adulthood is just as high in five year olds as in older school-aged children. There was some persistence in extreme ranks. For the 208 children initially at or above the 90th percentile for systolic blood pressure, 35 percent remained at year 4 and 30 percent remained at year 6. At both subsequent examinations more than 50 percent remained in the top quintile.[160]

Results from the Dunedin Multidisciplinary Health and Development study show correlation coefficients for blood pressure at age seven years with blood pressure at age 18 years of 0.4 for girls and 0.5 for boys. However in the children and youth in this study group, although blood pressure rank was generally relatively stable overtime, high blood pressure tended to normalise. These results suggest children with blood pressure above the 95th percentile at one age are unlikely to remain in this group. The authors, St George and Williams, support the view of the British Hypertension Society that population screening to identify children with hypertension is not warranted.[158] The differences in the results from these two studies may represent ethno-cultural and socioeconomic differences between the two population groups. The Dunedin study group was significantly underrepresentative of Mäori and Pacific children, so the findings cannot be applied to these children. However, within the Dunedin study group there was a subgroup of children whose blood pressure showed some stability in the highest quintile. These children were heavier and taller than their peers and were more likely to have a parent with hypertension or symptomatic cardiovascular disease.[158] This suggests that targeted screening may be appropriate.

As noted above, in Westernised countries like New Zealand there is a continuous rise in blood pressure with increasing age, but this does not occur in hunter-gatherer societies.[101] Much of this difference in blood pressure can be attributed to differences in salt intake, but there may be other environmental causes related to living in modern society. Stress testing in the laboratory produces a brief rise in blood pressure, however there is still debate as to whether chronic stress causes sustained elevation of blood pressure.[158]

Environmental and genetic factors interact to produce hypertension and they are difficult to separate. Modifiable determinants of blood pressure include diet, body weight and physical activity.[101] As discussed previously, there is considerable epidemiological and experimental evidence to indicate that sodium chloride is a strong causative agent for hypertension. However other substances, such as calcium, potassium, magnesium, vitamin C[157] and vitamin D,[114] may be even more important. Optimum physiologic concentrations of these minerals and vitamins may maintain normal blood pressure by enhancing the synthesis and release of vasodilators and platelet antiaggregators. Vitamin C can reverse impaired endothelial vasodilation by blocking superoxide anion formation and enhancing vasodilator formation.[94] There is good evidence that increasing fruit and vegetable consumption (major sources of potassium and vitamin C) can lower blood pressure in the general population.[101]

Body mass index is correlated with salt intake and independently associated with blood pressure.[101] There is an interrelationship between obesity and elevated blood pressure during adolescence in the development of hypertension in adulthood. Positive correlations between weight gain in childhood and blood pressure have been observed in numerous populations.[160] There is some suggestion that abnormal carbohydrate metabolism contributes to the pathogenesis of hypertension. A study in rats has shown that chronic feeding with 10 percent glucose solution results in elevated systolic blood pressure, plasma insulin and glucose levels, and insulin resistance index.[94] In the Bogalusa Heart Study a positive correlation between insulin levels and blood pressure was found for white boys but not other race-sex groups.[160]

In adults increasing physical activity has been shown to lower blood pressure independently of its effects on body weight.[101] Aerobic training can lower blood pressure in hypertensive adolescents,[125] but there is little evidence that exercise training can decrease blood pressure in normotensive children.[118]

Summary

I have used Martin Tobias' model of levels of causation to examine the influences in childhood on the development of cardiovascular disease and type 2 diabetes in adulthood. These risk factors do not act independently of each other. Although more proximal causes are often easier to identify and examine scientifically, this should not detract from the importance of more distal influences for which the potential gain from preventative strategies that address these is far greater.

Atherosclerosis begins in early life. Cardiovascular disease and obesity cluster in childhood and track through to adulthood. Risk factors can be identified early in life and these factors are cumulative across the lifecourse.

A significant proportion of New Zealand children are at increased risk of developing cardiovascular disease and type 2 diabetes as adults. Some of these risk factors (socioeconomic disadvantage, ethnicity, parental obesity, maternal smoking) are present from prior to conception, so that a significant number of children already have the odds stacked against them from birth. Optimal foetal development requires a healthy in utero environment. This reflects maternal wellbeing, which includes physical and mental health, and social and financial security. Given that a substantial proportion of pregnancies, especially in youth, are unplanned, population strategies to maximise the health and wellbeing of all woman of child bearing age, and especially young women, have the greatest potential to improve the wellbeing of the developing foetus.

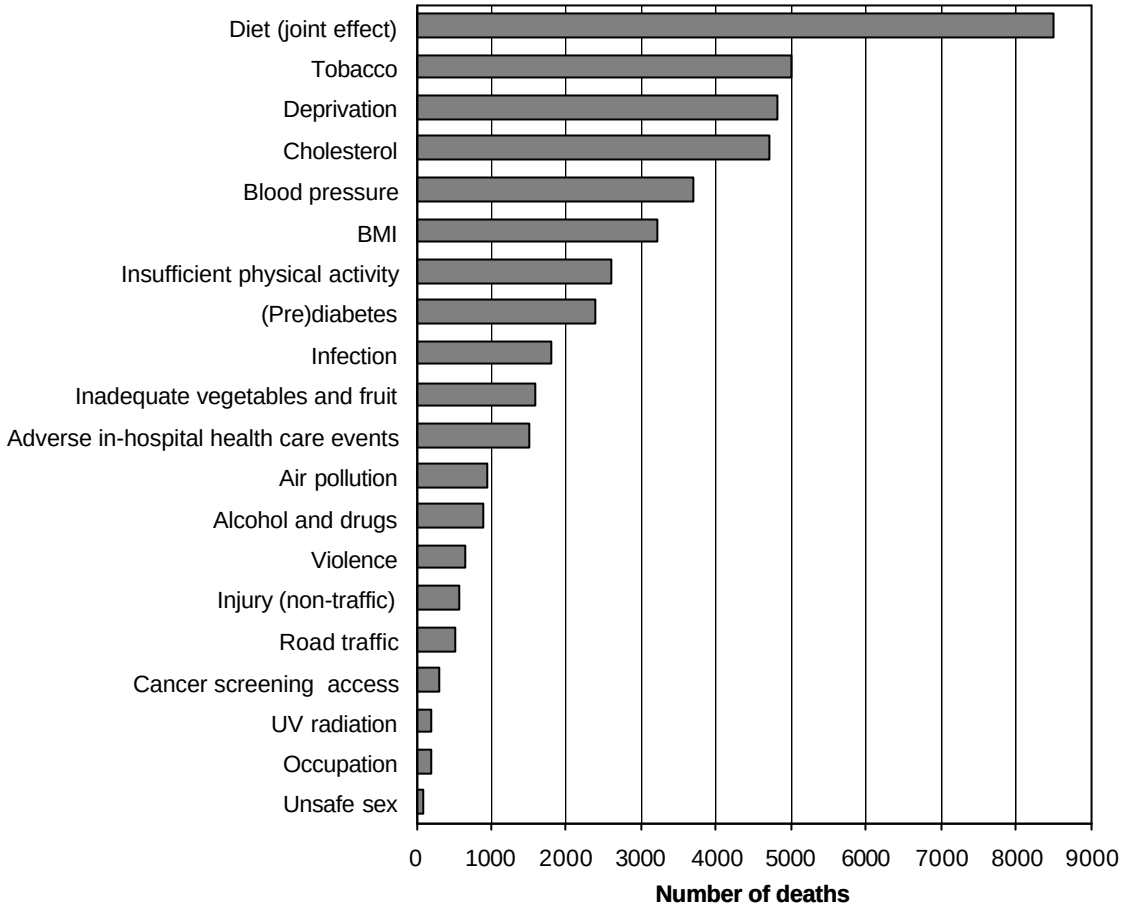
Unhealthy diets and physical inactivity are the leading cause of cardiovascular disease and type 2 diabetes. Diet and physical activity influence health both together and separately. Unhealthy diets and physical inactivity lead to disease through multiple mechanisms besides those resulting from overweight and obesity. While the effects of diet and physical activity on health often interact, particularly in relation to obesity, there are additional health benefits from physical activity that are independent of nutrition and diet. There are also significant nutritional risks that are unrelated to obesity. Physical activity is a fundamental means of improving the physical and mental health of individuals.[7]

Healthy habits are established early in life. Healthy children are more likely to grow into healthy adults. Lifestyle choices for children are made, and habits formed, within the context of their family or whänau, and also within the broader context of the structural features of society, economy and environment. Choices for many are limited by socioeconomic constraints. In New Zealand it is estimated that at least one third of all children lack access to sufficient economic and social resources that would allow a minimum adequate standard of living and participation in society.

Although there is evidence of tracking of obesity and other cardiovascular risk factors from childhood to adulthood, the fact that this tracking is not complete is evidence that these factors are reversible, and gives hope that effective interventions are possible. Also, the fact that tracking generally increases in strength with increasing age suggests preventative strategies should begin in early life. The health sector has an important role to play in reversing these trends, but implementation of sustainable changes to improve health outcomes and reduce disparities between population groups requires collaboration between sectors, local government, and industry to create new social norms and environments that are supportive of healthy lifestyles. Monitoring and evaluation of interventions are key to increasing our understanding and ensuring desired outcomes are achieved.

Appendix

Figure 4: Top 20 causes of death, by risk factor, New Zealand, 1997



Notes: Components of diet included in the analysis were energy intake, saturated fat intake, sodium intake, and vegetable and fruit intake. BMI = body mass index.

Source: Tobias M. 2004. Looking Upstream: Causes of death cross-classified by risk and condition New Zealand 1997. *Public Health Intelligence Occasional Bulletin Number 20*. Wellington: Ministry of Health.

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