IN DEPTH TOPIC: THE DETERMINANTS AND CONSEQUENCES OF OVERWEIGHT AND OBESITY

Introduction

According to the World Health Organization, childhood obesity is one of the most serious public health challenges of the 21st century [1]. Over the past thirty to forty years there has been a marked increase in the prevalence of childhood obesity in almost all countries for which data is available [2].

New Zealand is part of this global trend. The New Zealand Health Survey 2011/12 found that, among children aged 2–14 years, 20.7% were overweight and 10.2% were obese [3]. In boys, but not in girls, there was a statistically significant increase in the obesity rate in 2–14 year olds from the 2006/07 survey (8%) to the 2011/12 survey (10%). For 5–14 year old children, although the rate of obesity did not change from 2002 (9%) to 2006/07 (8%), from 2006/07 to 2011/12 there was a statistically significant increase (to 11%).

Childhood obesity carries significant physical and mental health risks both in the short term and the long term [4]. In the short term, childhood obesity may be associated with asthma [5], sleep apnoea [6,7,8],slipped femoral capital epiphysis (slipped upper femoral epiphysis) [9,10], being bullied, teased and socially marginalised [11,12,13,14,15], and having low self-esteem [16,17].Childhood obesity has been associated with emotional and behavioural problems even in pre-school children [18]. Many studies in children have documented the association between childhood obesity and most of the major risk factors for later cardiovascular disease: high blood pressure, dyslipidaemia, hyperinsulinaemia and/or insulin resistance, abnormalities in left ventricular mass and/or function, and abnormalities in endothelial function [4].

A number of large, long-running cohort studies have found that childhood overweight and obesity is associated with premature mortality in adulthood and with an increased risk of type 2 diabetes, stroke, coronary heart disease, and hypertension later in life [19]. It has been argued that this is largely because childhood overweight and obesity is a strong predictor of adult obesity [20,21]. Obese children have a moderately high probability of becoming obese adults (in the range 40–70%) [4] and adult obesity is associated with substantially increased risks of hypertension, dyslipidaemia, type 2 diabetes, coronary heart disease and stroke [22].This is the major reason why childhood obesity is considered to be a public health crisis [23].

Adult obesity is also associated with an increased risk of gallbladder disease, osteoarthritis, sleep apnoea and respiratory problems and some types of cancer [22]. The most convincing evidence for body fatness as a cause as a cause of cancer is for cancer of the oesophagus, pancreas, colon and rectum, breast (in post-menopausal women), uterus (endometrium), and kidney [24].

There is a general consensus among obesity experts that the obesity problem is not simply a personal issue of eating too much and doing too little but a problem that has its roots in a mismatch between basic human biology, the product of thousands of years of evolution, and modern society. It therefore requires tackling complex social and economic issues and changing public policy in many areas including food production, manufacturing and retailing, trade, urban planning, transport, healthcare, education and culture [23].

This in-depth topic aims to examine the determinants and consequences of childhood obesity and also to provide background information on the trends in childhood obesity prevalence (both globally and in New Zealand), defining and measuring obesity, and the natural history of obesity over the lifespan.

The sections covering these topics are arranged as follows:

- Factors contributing to childhood obesity

- Defining and measuring obesity
- The prevalence of childhood obesity
- The natural history of obesity over the life course
- The consequences of childhood obesity

Factors contributing to childhood obesity

Humans, like all animals, have a powerful biological drive to consume the food necessary for survival and rarely choose not to eat when they are hungry. They often, however, eat when they are not hungry and continue eating when they have already eaten enough. At a fundamental level, obesity is the result of mismatch between appetite, the drive to eat, and satiety, the drive to stop eating when sufficient food has been consumed. Eating behaviour is influenced by complex interactions between genetic, physiological, psychological and environmental factors that are incompletely understood but have been, and continue to be, the subject of much research.

From very early in life there are both genetic and environmental factors operating to influence a child's chances of becoming overweight or obese [25,26,27].

Genetics and parental weight status

Parental obesity is a strong predictor of a child's weight status [28]. The Avon longitudinal study of parents and children, which included 8234 children born in the U.K. in 1991 and 1992, found that a child was ten times more likely to be obese at age seven if both parents were obese than if neither parent was obese (adjusted odds ratio 10.4, 95% CI 5.1 to 21.3)[29]. Since parents are largely responsible for their child's food environment in early life it is difficult to separate out the genetic and environmental components of the influence of parental obesity [30].

Maes et al. reviewed over thirty twin, adoption and family studies and concluded that genetic factors are significant and explain between 20% and 90% of the variation in body mass index (BMI, defined as weight/height^2 with weight measured in kg and height in metres) [31]. It seems that there are many interacting genes involved in the predisposition to obesity, each with a small effect [32]. Single gene mutations causing morbid obesity in humans have been identified [33] but they are exceedingly rare [34].

There are a number of very rare syndromes with a Mendelian (single gene) pattern of inheritance, such as Prader-Willi syndrome and Bardet-Biedl syndrome, which include obesity as a clinical feature, often in association with mental retardation, dysmorphic features and organ-specific developmental abnormalities [35].

The hormone leptin, discovered in 1994, is secreted by adipocytes (fat cells) and is essential for regulating body weight through its effects on food intake and energy expenditure. [36] A few people who were homozygous for mutations in the gene encoding leptin, and consequently had congenital leptin deficiency, were subsequently identified in two families, in Pakistan [37] and in Turkey [38]. The clinical phenotype associated with congenital leptin deficiency includes hyperphagia (overeating), severe obesity, hypogonadism, and impaired immunity. Treating leptin-deficient people with daily injections of recombinant human leptin produces dramatic reductions in food intake and obesity [36,39]. Subsequent research has indicated that there are many different genetic defects affecting the downstream pathways from leptin signalling in the brain. All of these are associated with hyperphagia. This research has made it clear that human appetite and food intake are in part biologically determined and there are hopes it may lead to therapeutic options for regulating food intake in obese people [36].

O'Rahilly and Farooqi observed that all known monogenetic causes of obesity are associated with disruption of the hypothalamic pathways in the central nervous system [40] and have a profound effect on food intake and satiety [41]. They suggest that the evidence indicates that the major effect of genes on obesity is just as likely to be via an impact on hunger, satiety and food intake as via an impact on metabolic rate or fat deposition.

Prenatal influences

There is increasing evidence from both human and animal studies that prenatal and neonatal exposures can influence a child's risk of becoming an obese adult, developing metabolic syndrome (the combination of hypertension, insulin resistance, type 2 diabetes, dyslipidaemia and obesity) and developing cardiovascular disease [42,43]. The fetal origins hypothesis [44], also known as the developmental origins hypothesis, is that cardiovascular disease and type 2 diabetes may have their origins in under or over-nutrition in utero. The fetus makes adaptive responses to cues from the mother about her health or physical state. These responses may include changes in metabolism, hormone production and tissue sensitivity to hormones which may affect tissue and organ development and lead to persistent alterations in metabolic and physiologic set points [45]. Epigenetic changes in DNA (changes that do not involve changes in the DNA sequence but involve either methylation of DNA or modifications of chromatin) occurring in response to environmental stimuli alter gene expression and are believed to be a mechanism by which the fetus is "programmed" for later cardiac and metabolic disease, mostly on the basis of evidence from animal studies [46].

Whether being born to a mother with gestational diabetes increases the risk of childhood overweight and obesity may depend on whether or not the mother is also overweight or obese. A high pre-pregnancy BMI increases the risk of developing gestational diabetes. A 2009 systematic review and meta-analysis found that, compared to women with normal BMI, the unadjusted pooled odds ratios of developing gestational diabetes for overweight, moderately obese and morbidly obese women were 1.97 (95% CI 1.77 to 2.19), 3.01 (95% CI 2.34 to 3.87) and 5.55 (95% CI 4.27 to 7.21) respectively [47]. A 2011 systematic review considered 12 studies examining the association between gestational diabetes and childhood overweight and obesity [48]. The review authors found that most studies had methodological limitations. Only three studies adjusted for confounders. The two studies that adjusted for pre-pregnancy BMI found that, after this adjustment, there was no statistically significant association between gestational diabetes and offspring overweight and obesity.

Birth weight and early weight gain

Three recent systematic reviews have examined the relationship between birthweight and long term risk of overweight or obesity. Schellong et al. performed a meta-analysis of 66 studies (with various definitions of overweight and obesity, mostly related to BMI) and found a positive linear relationship between birthweight and later (in childhood, adolescence or adulthood) risk of overweight [49]. Low birthweight (<2500g) was associated with a decreased risk of overweight (Odds ratio 0.67; 95% CI 0.59–0.76) while high birthweight (>4000g) was associated with an increased risk of overweight (OR = 1.66; 95% CI 1.55–1.77). Zhao et al. performed a meta-analysis of fifteen studies reporting on the relationship between birthweight and overweight or obesity in adults [50]. They found that low birthweight (<2500g) was not associated with an increased risk of overweight/obesity (OR 1.17; 95% CI 0.94–1.46) but high birthweight (≥ 4000g) was associated with an increased risk of both overweight/obesity (OR = 1.46; 95% CI 1.27–1.68) and obesity (OR 1.43; 95% CI 1.25–1.64). Yu et al. included 33 studies in their review focusing on the association of birthweight with obesity in children and adolescents, most of which had been conducted in China [51]. They performed a meta-analysis of the results of 20 of them. They also concluded that high birthweight (>4000g) was associated with obesity (14 studies, OR = 2.07; 95% CI 1.91–2.24) and that low birthweight (<2500g) had a negative association with obesity (10 studies, OR = 0.61; 95% CI 0.46–0.80). The odds ratio for obesity increased gradually with increasing birthweight across all birthweight categories and 3500g was the threshold value above which the risk of obesity was increased.

The association between birthweight and the risk of type 2 diabetes in adulthood has been extensively studied. Whincup et al. carried out a quantitative systematic review of this issue using data from 30 reports (31 populations; 6090 diabetes cases; 152,084 individuals) [52]. After data from two Native American population with a high prevalence of maternal diabetes and one other population of young adults was excluded, the pooled

odds ratio for type 2 diabetes after adjustment for age and sex was 0.75 (95% CI, 0.70–0.81) per kilogram. The odds ratio was a little greater after adjustment for current BMI and adjustment for socio-economic status made almost no difference. Birthweight is therefore inversely related to type 2 diabetes risk across the normal range of birth weight.

Low birth weight may not be a risk factor for metabolic syndrome and subsequent type 2 diabetes if it is due to prematurity alone rather than failure to grow in utero. A recent systematic review by Parkinson et al. considered studies of adults (mean age 19.6 years, range 18–45 years) who were born pre-term babies (mean gestational age 32 weeks) that had measured markers of metabolic syndrome, including BMI, blood pressure, fasting glucose and lipid profiles [53]. There were no differences between preterm and term-born adults in most markers, including BMI, waist-to-hip ratio and per cent fat mass, but the adults born pre-term had higher blood pressure, more so in women. Young adults born pre-term had increased plasma low-density lipoprotein which could indicate an increased risk of later atherosclerosis and cardiovascular disease.

Low birthweight babies who have early rapid “catch-up” growth in their first two years are fatter than other children by age five [54]. Many large cohort studies have indicated that, among all children, after adjustment for age, sex and birthweight, more rapid weight gain in infancy is associated with an increased risk of subsequent obesity [55,56,57].

A possible explanation for the apparently paradoxical finding that the higher the birthweight the lower the risk of diabetes and cardiovascular disease but the higher the risk of adult overweight and obesity (which are risk factors for both diabetes and cardiovascular disease) is that a higher birthweight programmes greater lean mass in later life and that at given level of adult BMI, having had a low birthweight is associated with a higher percentage of body fat [58,59,60]. Further studies investigating body composition in infants may help to clarify this issue [61].

Breastfeeding

It is difficult to assess the effect of breast feeding on obesity risk. It is unethical to conduct a randomised controlled trial (RCT) of breast feeding versus formula feeding and observational studies are subject to numerous confounders. There has been a large RCT (17,046 infants) of a breastfeeding promotion intervention in Belarus which found that, although the intervention led to substantial increases in the duration and exclusivity of breastfeeding, it did not reduce measures of adiposity at age 6.5 years in the intervention group, or increase stature, or reduce blood pressure [62].

There are plausible reasons to believe breastfeeding should lower obesity risk: breast milk contains hormones such as leptin, adiponectin and ghrelin which may affect long term appetite regulation; infants being breast fed are typically better than those being bottle fed at signalling to their mothers that they are full and so may develop better self-regulation of energy intake; breastfed infants gain weight more slowly than formula fed infants; infant formula contains more protein than breast milk and randomised controlled trials have shown that protein-enriched formulae produce more rapid weight gain, increased adiposity and some later adverse cardiometabolic consequences in childhood and adolescence; and many cohort studies have shown an association between having been breastfed or longer duration of breastfeeding and reductions in obesity risk [63].

Owen et al. reviewed a large number of observational studies and found that breastfeeding was associated with a slightly lower mean BMI in later life but adjustment for socioeconomic status, maternal smoking in pregnancy and maternal BMI in 11 studies abolished the effect [64]. They concluded that mean BMI is slightly lower in those who were breastfed but this result is likely to be strongly influenced by publication bias and confounding factors and that, while there are good reasons for mothers to breastfeed, doing so is not likely to reduce their children’s mean BMI in later life. Another 2004 systematic review by Arenz et al. included a meta-analysis of data from nine studies (over 69,000 children) that adjusted for at least three of the factors birthweight, parental overweight, parental smoking, dietary factors, physical activity and socioeconomic status [65]. The adjusted odds ratio calculated from the data of these studies was 0.78 (95% CI

0.71–0.85) and the study authors concluded that there was a small but consistent protective effect of breastfeeding against obesity risk in later childhood. Weng et al. performed a meta-analysis of ten studies published between 2003 and 2009, five of which had found a protective effect of breast feeding and five of which had not, to obtain a pooled odds ratio for the effect of having been ever breast fed on being overweight in childhood of 0.85 (95% CI 0.74–0.99) [66].

Sleep

There is evidence from many cross-sectional studies around the world of a link between sleep duration and obesity in all age groups [67]. A 2008 systematic review by Chen et al. reported on three cohort studies, 12 cross-sectional studies and two case-control studies in children and adolescents [68]. Six had been conducted in the U.S., five in Europe, four in Asia and one each in Australia and Canada. Numbers of participants varied from 150 to 8,941 but most studies had over 1,000. The review authors found that there was strong evidence of an association between short sleep duration and childhood obesity and that early life shortness of sleep seems to be associated with greater risk. The association between short sleep and obesity may be stronger in boys as this was found in large studies in Japan and Australia. In general, the studies in children aged < 10 years found an inverse association between sleep duration (according to parental report) and obesity but those in adolescents were inconsistent. Based on a meta-analysis of 11 studies, the pooled odds ratio of overweight/obesity for shorter vs. longer sleep duration (based on each individual study's criteria) was 1.58 (95% CI 1.26–1.98). Based on three studies, gender-specific odds ratios were 2.50 (95% CI 1.91–3.26) for boys and 1.24 (95% CI 1.07–1.45) for girls. Another systematic review by Patel and Hu, which included ten of the same studies and also three smaller studies, two of which did not adjust for any confounders, stated that the studies' results suggest that, in children, short sleep is strongly and consistently associated with current and future adiposity but that major limitations in study design precluded definitive conclusions [69].

An Otago study investigating the relationship between sleep duration and measures of adiposity in adolescents found an effect in boys, but not in girls. In boys each hour increase in average nightly sleep duration was associated with decreases of 1.2% for waist circumference, 0.9% for waist-to-hip ratio 4.5% for fat mass index and 1.4% for fat-free mass index in multivariate models. Results were similar for weekday and weekend night sleep duration [70]. The Prevention of Overweight in Infancy (POI.nz) study is currently in progress in Dunedin. This is a four-arm randomised controlled trial of food, activity and sleep interventions for preventing the development of overweight from infancy [71].

Environmental factors in childhood

Many of the changes in family lifestyles that have occurred over the last few decades are likely to be contributing to childhood overweight and obesity but it is difficult to assess the impact of any one factor in isolation. Factors that are probably significant contributors to what has been termed the "obesogenic environment" are: increased consumption of food away from home, processed food, fast food, sugar-sweetened beverages and fruit juices; fewer children walking or biking to school; increased television watching, computer use and video game playing ("screen time"); and decreased participation in organised sport [72]. Reduced physical activity and increased sedentary behaviour may be both causes and consequences of obesity [27].

It is unclear what relative contributions excess consumption and insufficient physical activity make to childhood obesity. A recent review identified 26 cross-sectional and longitudinal studies examining the relationship of factors related to energy intake and energy expenditure to childhood obesity [73]. The reviewers found that there was wide variation in data quality between studies and concluded that, on the basis of current evidence, there is no consensus on the main driver of the increase in childhood obesity prevalence over recent decades.

A recent Australian study used data from the Longitudinal study of Australian Children to examine whether patterns of behaviour, based on physical activity, diet, screen time and sleep time, were associated with obesity in 1833 children aged 6–7 years [74]. Using latent

class analysis the researchers identified three behavioural profiles: healthy (27.7%), sedentary (24.8%) and short sleepers/unhealthy eaters (47.5%).

The healthy profile was associated with the lowest rates of sleeping <10 hours per night, low levels of screen time and high levels of physical activity, and it also had the highest intake of fruit and vegetables and the lowest intake of high fat foods and high sugar drinks. The sedentary profile was characterised by long screen time (> 2 hours per day) and low rates of physical activity (< 1 hour per day). The short sleepers/unhealthy eaters profile was characterised by the highest rate of short sleep (27.2%) and greater consumption on high fat foods and sugary drinks. Compared to the healthy profile, at two year follow up both the sedentary profile (odds ratio = 1.59, 95% CI 1.06–2.38) and short sleepers/unhealthy eaters profile (odds ratio = 1.47; 95% CI 1.03–2.13) had elevated odds of obesity.

The cost of healthy eating

It has been claimed that healthy food is more expensive than "junk food" and that cost is a barrier to families being able to provide nutritionally adequate meals for their children [75]. A 2011 study by Regional Public Health in Wellington, entitled *Food Costs for Families: Analysis of the proportion of the minimum wage and income support benefit entitlements that families need to purchase a healthy diet,* found that in order to purchase a "basic" healthy diet families needed to spend 43%–89% of their net income after deduction of rent costs [76]. Others have claimed, however, that it is possible to achieve substantial improvements in nutrition without spending a great deal more money [77].

Food insecurity has been defined as: "the inability to acquire or consume an adequate diet quality or sufficient quantity of food in socially acceptable ways or the uncertainty that one will be able to do so" [78].The 2008/09 New Zealand Adult Nutrition Survey classified 59.1% of New Zealanders as living in households that were fully/almost food secure, 33.7% as living in households that were moderately food secure and 7.3% as living in households that had low food security, based on survey participants' answers to questions on eight facets of food security related to food affordability. This survey did not report on households with children specifically [79].

The 2002 National Children's Nutrition Survey asked adult members of the children's households about their household's food security. Overall, 77.8% of children's households could "always" afford to "eat properly" and 20.1% could "sometimes" afford to do so. There were marked increases in the proportion of households who could only sometimes afford to eat properly with increasing number of children (from 15.1% in those with ≤ 2 children to 37.3% in those with ≥ 5 children) and with increasing deprivation (from 5.5% in NZDep quintile I to 37.5% in NZDep quintile 5). The proportion of Māori children's households who could only sometimes afford to eat properly was 33.6%. The proportion of Pacific children's households in this category was 47.9% while the proportion of European/Other children's households was 12.1% [80].

While it may seem counter-intuitive, a link between food insecurity and obesity has been found in a number of studies. Several recent reviews have looked at this issue in relation to children and adolescents [81,82,83,84]. There is a consensus that there is an association between food insecurity and obesity for adult women but for children the evidence is inconclusive.

There are particular challenges faced by Pacific people wishing to choose more healthy eating patterns. In Pacific societies food plays an important social and cultural role and the ability to provide plenty of food for the family and visitors is an integral part of Pacific identity [85]. Lanumata and others reported on a series of focus groups that they facilitated with Māori, Pacific and low-income New Zealanders to learn about their views on food security and physical activity [86]. They stated that there was "unanimous agreement amongst participants about the desire for better access to nutritious food in order to live healthier and longer lives". The Tongan participants reported that huge feasts were part of cultural functions such as birthdays and funerals. Along with cultural expectations regarding hospitality, cost was reported to be a major barrier to healthy eating for many

Pacific people. Participants also talked about the convenience of fast food and some people lacking the time or the knowledge and skills to prepare healthy food.

A recent report from the Office of the Auditor General comments on four focus groups which discussed the issue of child obesity and programmes to address it with Māori and Pasifika families of children aged five to fourteen in Auckland [87]. Māori parents reported poverty as a major obstacle to providing healthy food for their families. This was also a concern for Pasifika families but they saw the main issue as food and eating to excess being an integral part of their culture and a customary belief that being big was a sign of health, strength and being well cared for.

The prevalence of obesity in children and young people

A global perspective

Until relatively recently, most of humanity struggled against food shortage, disease and a hostile environment [88]. People lived in small groups and obtained most of their food from sources close to where they lived. Obesity was confined to a few high status individuals. In the poorest and least developed countries this is still the situation today but in developed and developing countries obesity is pervasive and is increasingly a greater problem for the lower socio-economic groups in society. This shift from the higher to the lower socio-economic groups occurred first in the U.S.A. and Europe but it is now occurring in developing countries also, most obviously in women [89,90]. In developing countries in the midst of the "nutritional transition" where diets are changing from locally produced fruits, vegetables and whole grains to highly processed, low cost, energy dense foods there is a "nutrition paradox" where over nutrition and under nutrition co-exist and overweight mothers may have stunted children [91].

From the 1970s to the end of the 1990s the prevalence of overweight or obesity in school age children doubled or trebled in many countries including Canada, the United States, Australia, Japan, the U.K., Finland, Germany, Greece, Spain, Brazil and Chile [2]. There is, however, some evidence that, over recent years, in a number of countries, obesity rates in children are no longer increasing and the prevalence of overweight and obesity may have plateaued [92,93,94].

New Zealand children: trends over time, ethnic and socio-economic disparities

Not only are there more obese children than there were but, on average, all children are heavier. Participants in the Family Lifestyle, Activity, Movement and Eating Study, born in Dunedin in 2001–2002 had a mean body mass index at age seven that was 0.84 kg/m^2 (95% CI 0.61 to 1.06) greater than that of the participants in the Dunedin Multidisciplinary Health and Development Study, born in 1972–1973 [95]. A comparison of body mass index in two groups of 12 year old Hawke's Bay schoolchildren who were part of an asthma prevalence study indicated that, over the 11 year period from 1989 to 2000, the geometric mean BMI increased from 18.1 kg/m^2 (95% CI 17.9 to 18.3) to 19.8 kg/m^2 (95% CI 19.6 to 20.0) in 2000, a relative increase of 9.2% (95% CI 7.6 to 10.9) [96].

As has been found in other developed countries [97,98,99], the New Zealand Health Survey 2011/12 found marked social and ethnic inequalities in childhood (2–14 years) obesity rates [3]. Pacific and Māori children (compared to European/other children) and children living in the most deprived areas (compared to those living in the least deprived areas) had significantly higher rates of both overweight and obesity. Similar disparities were found in the Youth '07 survey of 9,107 secondary school students [100]. More detail on the prevalence of overweight and obesity in New Zealand children can be found in the Overweight and Obesity chapter.

Defining and measuring obesity

In order to be able to make meaningful comparisons between obesity rates in different population groups and to monitor trends in obesity over time it is essential that a consistent definition of obesity is used. The World Health Organization defines overweight and obesity as "abnormal or excessive fat accumulation that may impair health" [101]. It is not possible to measure total body fat precisely in a living person so various measures are

used as proxies. The techniques that have been developed to measure body fat are complex and have mostly been used only in research settings. They include hydrodensitometry (under water weighing), air displacement plethysmography, CT and MRI scanning, dual-energy X-ray absorptiometry (DEXA/DXA), and bioelectrical impedance methods [102].

For clinical use and for population surveys and screening, the measures used to assess body fatness include skinfold thickness, waist circumference, waist-to-hip ratio and body mass index [102]. Body mass index (BMI), defined as weight/height^2 (with weight is measured in kg and height in metres) is the most widely used tool for monitoring obesity. Definitions of overweight and obesity based on BMI values depend on specified cut-off points above which BMI values indicate overweight or obesity. For adults, the World Health Organization has defined obesity as a BMI ≥ 30 kg/m^2 and overweight as a BMI ≥ 25 kg/m^2.

A child's BMI is typically plotted on a BMI-for-age reference chart which has percentile lines marked on it to indicate the percentage of children in a reference standard (non-obese) population whose BMI is at or below a given level at each age. Whether or not a child is labelled as being overweight or obese depends somewhat on the reference standard used and the percentiles chosen as cut-offs [103]. Children who have a BMI greater than or equal to the 95th percentile for their age are commonly classified as being obese, and those whose BMI is at or above the 85th percentile but less than the 95th percentile as being overweight [104] but there is no universal agreement on BMI cut-off points for defining obesity in children [105,106].

The World Health Organization's Child Growth standards are widely used for monitoring obesity in children aged 0–5 years [103,107]. The growth charts that are used by Well Child/Tamariki Ora providers (and are in the Well Child/Tamariki Ora Healthbook) are based on the 2006 World Health Organization's standards and were originally developed for use in the U.K. [108,109,110,111].

For older children widely used reference standards (especially for research purposes) are the BMI-for-age percentile charts of the World Health Organization [112,113] and the BMI-for-age obesity and overweight cut-off charts and tables of the International Obesity Taskforce [114]. Some countries, including the U.S.[115] and the U.K. [116] use their own reference standards.

Research studies often report results as BMI z-scores, also known as BMI SDS (standard deviation scores), which indicate how may units of the standard deviation a child's BMI is above or below the average BMI for their age group and sex [117]. For example, a child with a z-score of 2.0 has a BMI that is 2.0 standard deviations above the average. Charts, tables or software can be used to convert children's BMI values into BMI percentiles or z-scores [118]. Although z-scores and percentiles can be converted to each other the commonly used cut-off points are not precisely comparable: a z-score of +2 corresponds to the 97.7th percentile and the 85th percentile corresponds to a z-score of 1.04 [119].The advantage of using z-scores is that they are useful for monitoring changes in weight in patients with BMI values above the 99th percentile, and in a research setting they enable the calculation of a meaningful average result for a group that includes children of different ages.

Should the same BMI values be used to define obesity in different ethnic groups?

There had been considerable debate about whether the same BMI cut-off points should be used to define obesity in different ethnic groups, both internationally [120] and in New Zealand [121,122]. Tyrrell et al. measured obesity rates in 2273 Auckland according to both BMI and bioelectrical impedance measures in the late 1990s. These children attended primary schools with a high proportion of Māori and Pacific Island pupils. There were clear differences in obesity rates between different ethnic groups: Pacific Island 24.1%, Māori 15.8%, and European 8.6%, but no clinically relevant ethnic differences in the relationship between BMI and body composition in children who had BMIs in the normal range (<30). The small, but statistically significant effect of ethnicity on this

relationship was attributable to the number of Pacific Island children with BMI>30. Tyrrell et al. stated that this skewing did not justify the use of different BMI percentiles for the different ethnic groups [122].

Using a subsample of participants in the 2002 New Zealand Child Nutrition Survey (643 children aged 5–14 years), Rush et al. investigated the relationships between BMI, body fatness, ethnicity, age and blood lipids. They found that Māori and Pacific Island girls had a lower per cent body fat (as determined by bioimpedance analysis) for the same BMI compared to European girls, but for boys there were no ethnic differences [123]. A later Auckland study of 1,247 primary school children, which included 147 East Asian and 117 South Asian children, found that, although the level of body fat at a given BMI appeared similar among Māori, European and East Asian boys and girls, South Asian children averaged 3.0–5.1% (boys) and 4.2–6.1% (girls) more body fat than the remaining four ethnic groups. When adjustment was made for this difference in body fat percentage, the prevalence of obesity in South Asian children rose from 5.1% to 21.4% [124]. Using a larger sample of 1676 girls with a wider age range (5–16 years), the same authors found that, compared to European girls, for a fixed BMI and age, South and East Asians averaged 4.2% and 1.3% more body fat, while Pacific Islanders averaged 1.8% less [125]. Using stepwise multiple regression they developed a series of ethnic-specific BMI cut-off points which they considered would provide a more accurate indication of overweight or obesity in New Zealand girls [126].

The limitations of BMI in clinical practice

While BMI is very useful for making comparisons between different population groups or for monitoring a population over time, it has some limitations as an indicator of body fatness at the individual level [106]. It does not take account of where on the body fat is deposited. It is high visceral fat that is associated with insulin resistance and risk of diabetes and cardiovascular disease [127]. A high BMI may be due to high muscle mass in an athletic child or adolescent who does not have excess body fat [104]. Body mass index in older children is affected by degree of sexual maturity. Early maturing girls tend to have a higher BMI than less mature girls of the same age while the opposite is the case for boys [128].

In the clinical setting, for identifying children who may be candidates for weight management intervention, BMI is a complement to, and not a substitute for, clinical examination. Visual inspection alone may be unreliable for identifying obese, and particularly overweight, children because, as being overweight is increasingly the norm, children with BMIs above the 85th or 95th percentile may not appear very different from other children [129]. Plotting BMI on a BMI-for-age chart is useful for tracking a child's weight status over time since it can take account of the fact that a child is growing in height but improvements in health status following adopting a healthier diet and increasing physical activity may not lead to improvements in BMI if fat is replaced with muscle.

The natural history of obesity over the life course

Cross-sectional population surveys, including the most recent New Zealand Health Survey [130], indicate that the proportion of people who are overweight or obese increases with age up until old age, where it levels off or declines [131,132].

Longitudinal cohort studies provide the best methods for studying the tracking of children's weight status over time and investigating the childhood predictors of adult overweight and obesity and adult cardiovascular disease and diabetes [30,133]. In the epidemiological literature, "tracking" is the name often given to the concept of persistence or relative stability of a risk factor over time [134,135,136].

A 2008 systematic review by Singh et al. reported on longitudinal studies investigating the persistence of childhood and adolescence overweight [133]. The review included 25 longitudinal studies of retrospective or prospective design, 13 of which were considered to be of high quality. Most of the high quality studies were published after 2001.

All of the studies included in the review reported that overweight or obese children and adolescents had an increased risk of becoming overweight or obese adults. Four studies

included analyses stratified for different levels of body composition (BMI) and these studies showed that persistence of overweight increased with increasing degree of overweight. Most of the studies that included several measurements during childhood and adolescence showed that persistence of weight status into adulthood increased with age. Some studies reported that greater persistence of weight status in girls than in boys but others reported contrasting findings. Overall, the high quality studies indicated that the risk of overweight children becoming overweight or obese adults is at least twice that of children of normal weight with the highest odds ratio reported by a high quality study being 10. For obese children, the relative risks or odds ratios were generally greater. For overweight adolescents, one high quality study reported a relative risk for adult overweight of 4.3 and one high quality study reported odds ratios for adult obesity of 15 for boys and 12 for girls. Three high quality studies reported percentages of overweight adolescents who became overweight adults which ranged from 22% to 58%. Overall the percentage of obese adolescents who became overweight or obese adults was higher than the percentage of obese children. In three high quality studies it ranged from 24% to 90%.

The review authors concluded that there was strong evidence for “moderate” persistence of childhood and adolescent overweight into adulthood but there was considerable variation in predictive values. They noted that the study subjects had grown up in a less obesogenic environment than the current one which may mean that the persistence of overweight will be different for today’s children. They also noted that the prevalence of overweight in adults is much higher than it is in children indicating that most overweight adults were not overweight as children.

An earlier 1993 review by Serdula et al. also concluded that obese children are at increased risk of becoming obese adults [137]. Differences between studies in study design, definitions of obesity and analytic methods made it difficult to quantify the correlation between childhood and adult obesity.

The consequences of childhood obesity

There are both short term and long term consequences of childhood obesity. The short term consequences that may affect a child’s quality of life are largely limited to severely obese children. They include sleep problems, asthma, type 2 diabetes, polycystic ovary syndrome, orthopaedic disorders (including slipped upper femoral epiphysis) and psychological and social distress [138]. In addition, a number of markers for subclinical coronary artery disease and atherosclerosis are measureable in obese children including elevated blood pressure and serum concentrations of total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol [139]. The long term consequences of childhood obesity are probably mostly related to the fact that obese children have a high probability of becoming obese adults with increased risks of metabolic syndrome, type 2 diabetes and cardiovascular disease, since adults who have normal BMI but were obese as children have similar cardiovascular risk profiles to those who have always had normal BMI [140].

New Zealand data on children’s and young people’s hospital admissions for slipped upper femoral epiphysis, type 2 diabetes and bariatric surgery is presented in the consequences of obesity chapter beginning on **page** Error! Bookmark not defined..

Sleep problems

Obstructive sleep apnoea is one of the most serious problems that can affect obese children and it is more common among those who are severely obese. Parents may notice loud snoring with pauses in breathing, restless sleep and daytime sleepiness. Disturbed sleep can result in poor attention, poor school performance and bed-wetting. Obstructive sleep apnoea can lead to right ventricular hypertrophy and pulmonary hypertension. Diagnosis is made via polysomnography. Treatment options include removal of tonsils and adenoids if these are enlarged and continuous positive airway pressure (CPAP) therapy during sleep [141].

Obesity hypoventilation syndrome occurs in severely obese patients when the weight of fat on the chest and abdomen impairs breathing. The symptoms are similar to those of obstructive sleep apnoea [141].

Asthma

According to a 2013 review by Papoutsakis et al., the recent evidence from the epidemiological literature indicates that there is a weak but significant association between high body weight and asthma in children [5]. The association appears to be more pronounced where there is central obesity and where the asthma is not related to allergies. Some prospective studies have found that high body weight precedes asthma symptoms. A 2012 Cochrane review identified no published RCTs on the effect of either weight loss or weight gain on asthma symptoms in children but stated that a few studies were in progress [142].

Type 2 Diabetes

Type 2 diabetes is one of the most serious complications of childhood obesity [141]. Most overweight children have significant metabolic abnormalities due to insulin resistance even if they have no evidence of type 2 diabetes [143]. It has been reported that the incidence of type 2 diabetes in children < 15 years in Auckland increased five-fold from 1995 to 2007, from 0.5 per 100,000 to 2.5 per 100,000 (representing about 10% of all new cases of diabetes among children and adolescents in the Auckland region) [144]. The average annual incidence over the period was 1.3 per 100,000 overall and there were significant ethnic disparities with an average annual incidence (per 100,000) of 0.1 in Europeans and 3.4 in both Māori and Pacific children. Unlike type 1 diabetes, type 2 diabetes is often asymptomatic and therefore diagnosis requires laboratory testing. Obese or overweight children with a family history of diabetes, especially Māori and Pacific children, are at particular risk of developing type 2 diabetes.

Polycystic Ovary syndrome

Obese adolescent women may develop Polycystic Ovary syndrome (POCS) which is the combination of polycystic ovaries (visible on ultrasound) with hyperandrogenism (which can result in severe acne), anovulatory menstrual cycles or oligomenorrhea, and hirsutism (excess facial and body hair). Not only does POCS lead to fertility and cosmetic problems, it is also a risk factor for future metabolic syndrome, type 2 diabetes and cardiovascular disease [143].

Orthopaedic disorders, including slipped upper femoral epiphysis

Children's cartilaginous bones and unfused growth plates predispose them to obesity-related orthopaedic problems [138]. Dislocation of the femoral growth plate, known as slipped upper femoral epiphysis (SUFE), can lead to permanent damage to the femoral head. At least 50% of patients with SUFE are obese and SUFEs occur at significantly younger ages among obese children than non-obese children [138]. Blount's disease involves bowing of the legs in response to unequal or early excess weight bearing. Treatment can require multiple surgical osteotomies. Severe infantile (under three years) Blount's disease seems to be associated with obesity with one study reporting that 12 out of 18 patients with Blount's disease at the Boston Children's Hospital Medical Center had weights above 120% of ideal body weight for their age and sex and all of the "obese" patients had infantile Blount's disease [145].

Psychological distress, stigmatisation and bullying

Parents of obese children often worry most about the emotional consequences of childhood obesity [87,146] and research indicates that there are good reasons for this. Obesity has been said to be one of the "most stigmatising and least socially acceptable conditions in childhood" [147]. One cross-sectional study of 106 obese children and adolescents (mean BMI 34.7 kg/m^2) referred to an academic children's hospital found that obese children and adolescents were more likely to have impaired health-related quality of life (QOL) than healthy children (odds ratio 5.5; 95% CI 3.4–8.7) and were similar to children and adolescents diagnosed with cancer (OR, 1.3; 95% CI, 0.8–2.3). Compared to healthy children, the obese children had impaired quality of life in all five QOL domains both by self-report and parent proxy report, as shown by unadjusted odds ratios which

ranged from 4.0 (95% CI 2.4–6.5) for self-reported school functioning to 13.6 (95% CI 8.2–22.5) for parent-reported overall psychosocial health (comprising emotional, social and school functioning) [147].

From a review of 17 studies, Tang-Peronard and Heitman concluded that overweight girls seemed to experience a greater degree of stigmatisation than overweight boys and were more often teased about their weight and bullied verbally, relationally and physically and also more socially marginalised with respect to friendships and romantic relationships [148]. A British prospective cohort study also found gender differences with respect to bullying [14]. At the age of 8.5 years, after adjustment for social class, obese boys were 1.66 (95% CI 1.04– 2.66) times more likely to be overt bullies and 1.54 (95% CI 1.12 to 2.13) times more likely to be overt victims. Compared to average weight girls, obese girls were 1.53 (95% CI 1.09–2.15) times more likely to be overt victims. The authors of this study suggested that the physical dominance of the obese boys in their peer group allowed some of them to become the perpetrators rather than the victims of bullying.

Childhood obesity may be associated with emotional and behavioural problems from a very young age. The British Millennium Cohort Study (11,202 children) used the Strengths and Difficulties Questionnaire to assess the relationship between obesity and behavioural problems in three and five year old children [18]. Adjusted linear and multinomial regression analyses indicated that at age three, compared to normal weight children, obese boys had more conduct problems, and obese girls had more prosocial behaviours. At age five, obese boys had more conduct problems, hyperactivity and inattention problems, peer relationship problems and total difficulties. Obese girls only had more peer relationship problems.

Later life consequences of childhood obesity

Most of the concern about rising rates of childhood obesity is based on the belief that society is facing a future tidal wave of obesity-related health problems that will overwhelm the health system [149,150]. As has been discussed earlier, obese children are at higher risk of becoming obese adults than normal weight children and this risk is greater for obese older children and adolescents than obese young children and for the most severely obese children [151]. Most overweight or obese adults, however, were of normal weight in childhood but gradually gained weight through adulthood to become overweight or obese in later life [152,153], so clearly childhood obesity is not the only predictor of adult obesity.

Reilly and Kelly conducted a systematic review of the more recent evidence (published from January 2002–June 2010) on the long term impact of child and adolescent obesity on premature mortality and physical morbidity in adulthood [19]. They identified five studies on the association between child or adolescent overweight or obesity and premature mortality, four of which found a significantly increased risk. All of the eleven studies on cardiometabolic morbidity reported that overweight and obesity were associated with increased risk of adult cardiometabolic disease (diabetes, heart disease, hypertension and stroke) with the hazard ratios ranging from 1.1 to 5.1. Nine studies examined other adult morbidity. Associations between child or adolescent overweight and cancer were inconsistent, but there were associations with increased adult risk of receiving a disability pension, asthma and symptoms of polycystic ovary syndrome. Reilly and Kelly stated that many of the studies in their review attempted to consider the issue of the relative contributions of child and adolescent obesity per se and the tendency of childhood obesity to persist into adult life by adjustment for current (adult) weight status. Some studies found that the associations between child and adolescent obesity and adult outcomes were attenuated after adjustment for adult weight status but others found such adjustment had a negligible effect.

Does childhood obesity have adverse consequences if adult weight is normal?

Although many studies have demonstrated positive associations between childhood obesity and adult cardiovascular risk Lloyd et al. have argued that it is uncertain whether there is an effect of child adiposity on adult cardiovascular risk that is independent of the degree of adult adiposity or whether the observed associations merely reflect the tracking

of childhood obesity into adulthood [20]. These authors undertook a systematic review of longitudinal studies (published prior to July 2008) investigating the association between childhood BMI and adult cardiovascular disease (CVD) with two objectives: firstly to report on the strength of the association between childhood obesity and adult CVD risk, and, secondly, to investigate whether the effects of childhood obesity are independent of adult BMI status. They identified 16 studies meeting their inclusion criteria, all but one published in the ten years prior to 2008. Most studies took account of age and gender but not socio-economic status and most treated childhood BMI as a continuous variable. There was considerable variation in age of childhood BMI measurements (ranging from two to 18 years) and adult outcome measurements (18 to 71 years).

Eight studies considered the relationship between childhood BMI and adult blood pressure, six the relationship with carotid intima-media thickness (CIMT, a marker of atherosclerosis and increased risk of coronary heart disease and stroke), and three the impact on incidence of coronary or ischaemic heart disease and stroke. Lloyd et al. stated that only two studies reported evidence of an independent positive relationship between childhood BMI and adult blood pressure and neither of them included the relevant adjusted data (i.e. confidence intervals or correlation statistics). One of them was the study with the fewest participants and it measured adult blood pressure at a young age (18 to 26 years). For these reasons, Lloyd et al. considered that the evidence for an independent association between childhood BMI and adult blood pressure was weak. In contrast, three studies which adjusted for adult BMI provided evidence of a negative relationship between childhood BMI and adult blood pressure, and two of them measured adult blood pressure at older ages (45 and 50 years) and could therefore be considered to provide a better indication of lifetime risk of hypertension. Lloyd et al. stated that the findings of these studies suggest that it is those who had low BMI in childhood but became overweight as adults who have the greatest risk of high blood pressure. Two studies, both using data from the Bogalusa Heart Cohort, reported a positive independent association between childhood BMI and adult CIMT but Lloyd et al. considered that these studies had weaknesses in their design and interpretation. The remaining studies showed no association between childhood BMI and adult CIMT after adjustment for adult BMI although all except one measured CIMT in young adulthood. Lloyd et al. therefore concluded that there was little evidence for an independent relationship between childhood BMI and adult CIMT and the limited existing evidence should be considered weak.

Two studies reported a positive association between childhood BMI and mortality from coronary or ischaemic heart disease but neither adjusted for adult BMI and so did not provide evidence for an independent effect of childhood BMI. Another study showed that men who died from coronary heart disease had a higher childhood BMI than those who did not, but these men had a mean BMI on the sixtieth centile for the population as a whole and therefore were well within the normal weight range.

In summary, Lloyd et al. found that there was little evidence that childhood obesity is an independent risk factor for increased blood pressure, CIMT or cardiovascular disease morbidity or mortality and that what evidence existed was weak. They stated that the approach and targeting of obesity interventions requires careful consideration and that while it is important for interventions to target the stages of life which offer the greatest long-term benefits, it is important to avoid the potential for negative consequences of obesity prevention or treatment programmes if they coincide with critical stages of neurological, behavioural and physical development.

In another systematic review, Lloyd et al. looked at studies investigating the associations between childhood BMI and markers of adult metabolic syndrome and whether or not any associations were independent of adult BMI [21]. They identified eleven studies published up until July 2010 which fulfilled their inclusion and exclusion criteria and most of these (seven studies) did not adjust for adult BMI. While several studies found positive associations between childhood BMI and adult total cholesterol, low density lipoprotein cholesterol, triglyceride and insulin concentrations, these associations were attenuated or reversed after adjustment for adult BMI or body fatness. None of the four studies that

considered adult metabolic syndrome as an endpoint found evidence of an independent association with childhood obesity. Lloyd et al. concluded that there was little evidence that childhood obesity is an independent risk factor for adult metabolic syndrome or its markers. They noted that data from studies which adjusted for adult BMI showed a weak negative association between childhood BMI and adult metabolic variables with those who were at the lower end of the BMI in childhood, but obese in adulthood, at particular risk.

Juonala et al. analysed data from four prospective cohort studies (6328 subjects) that followed participants from childhood into adulthood (mean length of follow up 23 years): the Bogalusa Heart Study (conducted in the U. S.), the Muscatine Study (U.S.), the Childhood Determinants of Adult Health Study (Australia), and the Cardiovascular Risk in Young Finns Study (Finland) to determine whether childhood obesity increases cardiovascular risk independent of adult BMI [140]. They found that, compared to those who had normal BMI as children and became non-obese adults, subjects who had a consistently high BMI from childhood through adulthood had an increased risk of type 2 diabetes (relative risk, 5.4; 95% confidence interval 3.4 to 8.5), hypertension (RR 2.7; 95% CI 2.2 to 3.3), elevated low-density lipoprotein cholesterol levels (RR 1.8; 95% CI 1.4 to 2.3), reduced high-density lipoprotein cholesterol levels (RR 2.1; 95% CI 1.8 to 2.5), elevated triglyceride levels (RR 3.0; 95% CI 2.4 to 3.8), and carotid artery atherosclerosis (increased intima-media thickness of the carotid artery) (RR 1.7; 95% CI 1.4 to 2.2). In contrast, subjects who were overweight or obese during childhood but were non-obese as adults had risks of the outcomes that were similar to those of subjects who had a consistently normal BMI from childhood to adulthood ($p > 0.20$ for all comparisons).

Conclusions

The prevalence of childhood obesity has increased significantly over the last thirty years, in New Zealand and in other developed countries. This is seen by many as a public health crisis, heralding a future epidemic of adult obesity and a tidal wave of obesity-related health problems, including diabetes (and consequent renal failure), hypertension, cardiovascular disease and cerebrovascular disease, which could overwhelm the health system. This is because childhood obesity, particularly severe childhood obesity, is a strong predictor of adult obesity, although it must be remembered that most currently overweight and obese adults were not obese as children. While there are obesity-related physical health problems for children, these are mostly confined to the relatively few severely obese children. It is the social and mental health consequences of childhood obesity which are of most concern to parents. Obesity is a stigmatised condition and obese children may face social exclusion, teasing and bullying.

There are both genetic and environmental factors which contribute to a child's chances of being obese. The many different contributing factors interact to form a complex web of potential determinants of childhood obesity [27]. These factors include maternal under or over nutrition, maternal smoking, in-utero growth restriction, maternal diabetes, high birth weight, low birth weight followed by rapid catch-up growth, lack of breast feeding, short sleep duration, parental overweight and obesity, low parental education and/or socio-economic status, low physical activity, high screen time, unhealthy food and beverages, food marketing practices, food prices, food insecurity and a built environment that discourages outdoor play and walking and cycling.

Many children start on the pathway to obesity very early in life. Obesity is not evenly distributed among the child population. Children of obese parents are at high risk of obesity for both genetic and environmental reasons, since parents are largely responsible for the food available to young children. Obesity also disproportionately affects poor and Māori and Pacific children. The cost of healthy food is a barrier to healthy eating for many families.

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