Consideration of the evidence on childhood obesity for the Commission on Ending Childhood Obesity

Report of the Ad hoc Working Group on Science and Evidence for Ending Childhood Obesity

Geneva, SWITZERLAND

WHO Library Cataloguing-in-Publication Data

Consideration of the evidence on childhood obesity for the Commission on Ending Childhood Obesity: report of the ad hoc working group on science and evidence for ending childhood obesity, Geneva, Switzerland.

1.Obesity - prevention and control. 2.Child Welfare. 3.Exercise. 4.Food Habits. I.World Health Organization.

ISBN 978 92 4 156533 2 (NLM classification: WD 210)

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1. Introduction

The prevalence of childhood obesity is increasing in all countries, with the most rapid rise in low- and middle-income countries; the majority of overweight or obese children live in developing countries, where the rate of increase has been more than 30% higher than that of developed countries. Children who are overweight or obese are at greater risk of asthma and cognitive impairment in childhood, and of obesity, diabetes, heart disease, some cancers, respiratory disease, mental health, and reproductive disorders later in life. The consequences of the rapid rise in obesity include not only health consequences but also negative impacts on the opportunity to participate in educational and recreational activities and increased economic burden at familial and societal levels. The rapidly rising rates of childhood obesity and subsequent increasing burden of disease and disability has grave social and economic consequences, contributing to rising cost of health services and limiting economic growth.

Overweight and obesity are critical indicators of the environment in which children are conceived, born, and raised. Childhood obesity is driven by biological, behavioural, and contextual factors. Biological drivers include maternal malnutrition (including both under- and overnutrition) during pregnancy, and gestational diabetes. Inappropriate infant feeding behaviours include inadequate periods of exclusive breastfeeding and inappropriate complementary foods, as taste, appetite and food preferences are established in early life. Physical activity behaviours are also established in early childhood. Contextual and wider societal factors include socioeconomic considerations, nutritional literacy within families, availability and affordability of healthy foods, inappropriate marketing of foods and beverages to children and families, lack of education and reduced opportunity for physical activity through healthy play and recreation in an increasingly urbanized and digital world.

Childhood obesity is a critical target as part of a strategy to promote a healthy life expectancy. Life-course studies suggest that interventions in early life when biology is most “plastic” are likely to have sustained effects on health, particularly because it can influence responses to later lifestyle factors. Early life represents a phase in the life-course when most societies are able to intervene constructively. It is also an area in every society where there is strong political consensus that action is desirable, including considerations of equity. This combination of short-term direct and indirect benefits and longer-term effects on the primary prevention of noncommunicable disease creates a powerful economic and social argument for action.

Addressing childhood obesity has a compelling logic and the science offers many opportunities for intervention. However, at present there is no clear consensus on what interventions and which combinations are likely to be most effective in different contexts across the globe and no global accountability mechanism for stakeholders currently exists. If childhood obesity could be successfully addressed, the spill-over benefits could be enormous. In addressing childhood obesity as a specific target, it is likely that there would be improvements in both maternal and child health in

general, there would be benefits for cognitive development and a reduction in other comorbidities in children, the nutritional status in the whole family would improve and there would certainly be major effects on long-term burden of noncommunicable disease.

The Ad hoc Working Group on Science and Evidence

In order to develop a comprehensive strategy to address childhood obesity, in May 2014, the Director-General of the World Health Organization (WHO) established the Commission on Ending Childhood Obesity. This Commission comprises 15 eminent individuals from a variety of relevant backgrounds. To support the work of the Commission, two ad hoc working groups were also established – one on science and evidence and the other on implementation, monitoring and accountability for ending childhood obesity. The working groups reported to the Director-General who transmitted their findings to the Commission. In turn, the Commission requested further information from the working groups on specific topics through the Director-General.

The Ad hoc Working Group on Science and Evidence consisted of academics, researchers and experts in the diverse fields that relate to childhood obesity, including epidemiology, paediatrics, nutrition, developmental origins of health and disease, health literacy, marketing to children, health economics, physical activity, gestational diabetes, etc. The full list of working group members is available in the appendix of this report. The membership of the working group was posted online for public comment prior to being finalized and all members of the working group completed a declaration of interest.

Objectives of the Ad hoc Working Group on Science and Evidence for Ending Childhood Obesity

To provide advice to the WHO Director-General on three core areas:

1. Epidemiology and burden: what is the current and estimated prevalence of childhood obesity and what are the health and social consequences?

2. Economic impact: what are the economic implications of increasing childhood obesity, especially in low- and middle-income countries?

3. Interventions: what are the evidence-based, or currently applied, policy options for prevention of childhood obesity, and in what combination should interventions be applied in different country contexts and for different age groups?

The Ad hoc Working Group on Science and Evidence for Ending Childhood Obesity met on two occasions at WHO headquarters in Geneva, Switzerland: 18–20 June 2014 and 15–18 December 2014. The Commission had requested further information on nine topics of interest, and rapid review papers were prepared by the working group. For three of these review papers, additional expertise was solicited and contributions made by external authors.

This report is being published following the release of the Commission's final report in January 2016 and prior to its consideration at the World Health Assembly in May 2016. The objective is to make available, in one document, the written information that the Ad hoc Working Group on Science and Evidence prepared for the Commission. The bulk of the report consists of background papers prepared by teams of the Ad hoc Working Group on Science and Evidence members and other collaborators or authors. These papers are presented in the context of background information and conclusions developed by the overall working group.

2. CONCEPTUAL FRAMEWORKS

Two complementary conceptual frameworks were identified early in the working group discussions and were used extensively in gathering and interpreting evidence about causal pathways and potential opportunities for intervention on obesity in children and adolescents. One is the life-course model; the other is the total environmental assessment model for early childhood development. Key elements of each framework are shown graphically and summarized below.

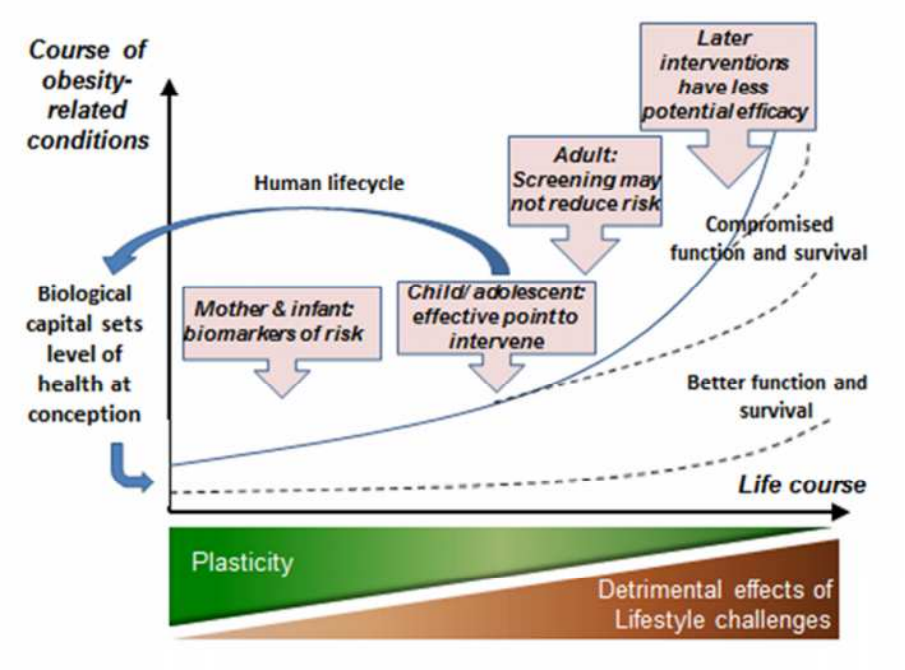
2.1 The life-course model

Science, based on animal and human studies, demonstrates how causal pathways for obesity originate in the earliest periods of life and persist throughout growth, development, and later years. New evidence from a range of fields shows that early life influences "prime" a child's responses to "obesogenic" environments. These alterations in susceptibility operate via integrated mechanisms, some of which are based on epigenetic processes. For example, they are involved in setting the body composition of the offspring in terms of numbers of fat cells. They also affect the physiological systems controlling appetite, food preference, metabolism, fat deposition, and insulin secretion and sensitivity. These epigenetic processes operate by modifying gene function without changing inherited genes themselves. This explains why, when considered in isolation, inherited fixed genetic patterns do not account for the major portion of attributable risk of obesity and its associated diseases in the population. These new mechanistic insights reinforce the idea that early life offers critical opportunities to intervene to reduce later risk of childhood obesity, providing a more optimistic view than the purely genetic deterministic approach.

These new scientific insights are captured by the life-course concept, a model which shows how risk at one time-point is influenced not only by the current challenge to health, for example the obesogenic environments now so prevalent, but also by the path which each individual took to reach their current position (Figure 1). This "pathway dependency" includes not only the cumulative exposure to risk factors, but also the degree to which the individual can respond to such challenges to maintain their health. Interventions to reduce risk and restore health in adults are likely to be less effective, and more costly, than earlier preventative interventions during developmental phases. The model also illustrates how risk can be passed from one generation to the next, for example through maternal diet or activity behaviours, body composition or conditions such as gestational diabetes

mellitus which produce biological cues which modulate the epigenetic processes in the offspring, along with broader behavioural influences of parents.

Figure 1. Life-course model of obesity and other noncommunicable disease risk



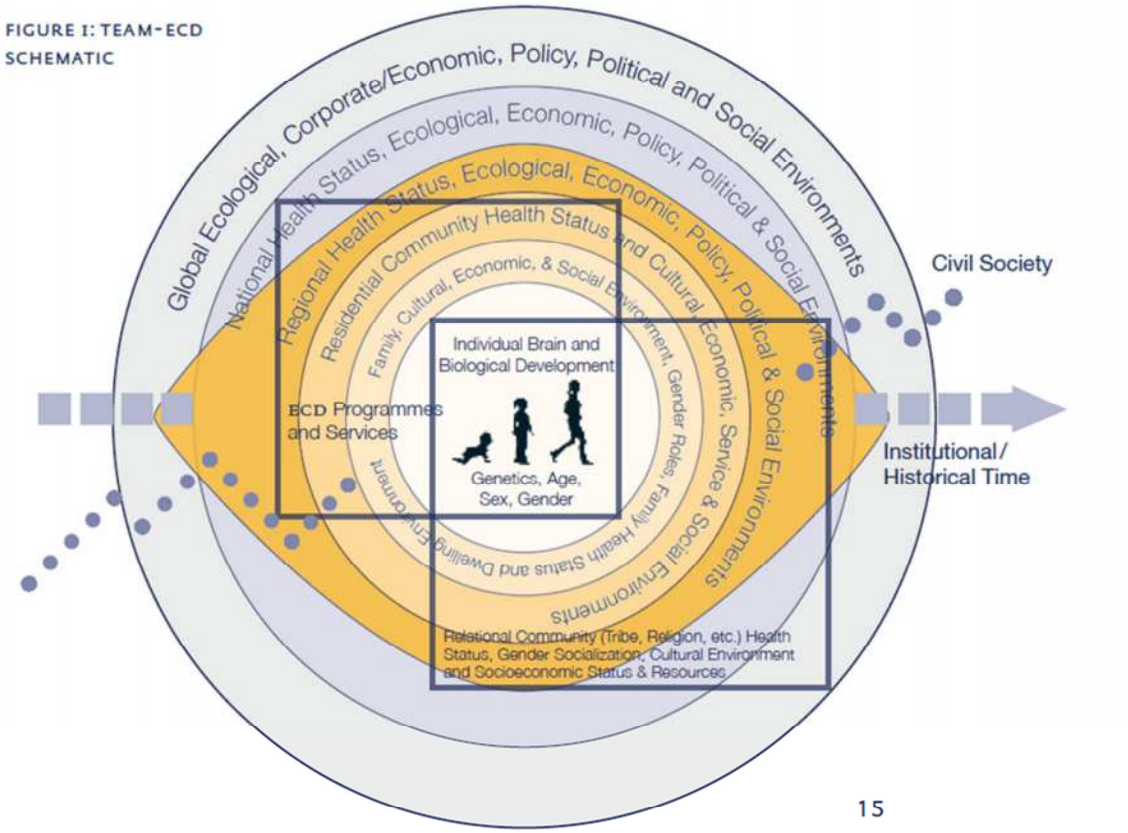
Source: WHO Meeting Report: Nurturing human capital along the life course: investing in early child development. 2013

Concepts related to this life-course approach include resilience, flexibility, and particularly, plasticity. The latter encompasses the idea that responding to an environmental challenge induces some change in the individual's phenotype, thus creating a step along the pathway of increasing risk. Plasticity is greatest during early development and declines with maturation, but the period over which decline occurs, and its rate of decline, varies between tissues, organs and systems, and between individuals. For some aspects of development, there are critical windows of plasticity, or sensitive periods, when phenotypic changes can occur. For example, animal experiments and human observations show that maternal nutritional balance, body composition and stress levels have effects on epigenetic processes in the developing fetus and newborn, altering the settings of the offspring's responses to subsequent challenges, such as an unbalanced diet. During these periods, future health can be adversely influenced by unhealthy environments and behaviours. However, these periods also offer valuable opportunities for interventions conferring long-term protection from risk.

2.2 The total environmental assessment model for early childhood development

The biological processes of epigenetics and developmental plasticity are related to behavioural and wider contextual drivers of childhood obesity. These are often not distinct, emphasizing that addressing the problem will require a systems-based and multisectoral and multijurisdictional approach, such as that shown in Figure 2. Such an approach has been utilized in relation to aspects of child development and should be applied to childhood obesity.

Figure 2. Early childhood development schematic



Source: Total environment assessment model for early child development. Evidence report. WHO 2007.

Although the life-course of the child commences at conception, new evidence from animal studies, epidemiology and a limited number of randomized controlled trials makes it clear that aspects of maternal and paternal nutrition, body composition, lifestyle behaviours and a wider range of environmental factors operating before conception influence the development and health of the fetus, infant and child. Accordingly, the future health of children is influenced not only by their future environment and behaviour, but also by historical risk factors which include, but are not limited to, inherited genetic predisposition. The trans-generational passage of risk suggests that "obesity begets obesity" and that "healthy living begets healthy living".

3. EPIDEMIOLOGY

3.1 Estimated prevalence and trends of childhood obesity worldwide

The increasing prevalence and incidence of obesity in children requires immediate attention. In 2014, globally, an estimated 41 million children under 5 years of age were affected by overweight or obesity: 48% living in Asia and 25% in Africa *(1)* (overweight being defined as the proportion of children with weight-for-height z-score values more than 2 standard deviations [SDs] and obesity more than 3 SDs, from the WHO growth standard median *(2)*). In Africa, the number of overweight or obese children has nearly doubled, from 5.4 million in 1990, to 10.3 million in 2014. The prevalence of infant, childhood and adolescent obesity may be plateauing in some settings, but in absolute numbers there are more overweight and obese children living in low- and middle-income countries than in high-income countries *(3)*.

Children with obesity suffer severe health consequences in childhood and are at high risk of becoming obese adults, with resulting increased risk of noncommunicable diseases. The dramatic increase in the obese population has considerable implications for society in the future, in terms of both health needs and burden, as well as increasing inequalities in health due to the trends in obesity associated with lower socioeconomic status.

3.2 Other epidemiological considerations

Epidemiological considerations for describing obesity in children and adolescents include tracking of patterns, as well as a range of obesity-associated behaviours related to dietary intake and movement behaviours (physical activity, sedentary behaviours and sleep) across and within countries and regions and over time. They also include characterization of key sources of variation in these patterns, such as age, gender, socioeconomic status, cultural, ethnic and urban–rural differences. Further focusing and refining epidemiological data collection can make a major contribution to meeting the target of preventing further increases in child obesity prevalence and to identifying optimal biological, behavioural, and environmental targets for, and the relative impact of, intervening at different times in the life-course.

There is a need to review carefully the strengths, limitations, and gaps in current data available to WHO noncommunicable disease and nutrition monitoring and surveillance frameworks, and make improvements to ensure accurate and timely identification of positive and negative changes in the dynamics of the obesity epidemic. Ideally, status and trend data will include: obesity prevalence (number and percentage of those currently obese); obesity incidence (number and rate of new cases of obesity that develop in a given time period); relevant behavioural data; data on obesity-relevant aspects of the environments to which children and adolescents are exposed during developmental periods; and the ability to associate these data with short- and longer-term health outcomes.

The issues, as set out below, were considered by the Ad hoc Working Group on Science and Evidence and are included here for future reference and consideration. Some are discussed in the rapid reviews prepared for the Commission, however, it was not possible, given the timeframe of the Commission's work, to cover all aspects:

- There is a need to evaluate the relevance and impact in low- and middle-income countries of childhood obesity risk factors and pathways that have been established in data from high-income countries.
- Given the marked inequities in obesity associated with both demographic characteristics, urbanization and socioeconomic status that have been documented within some countries, the extent to which these variations in obesity may be explicable, on the basis of currently recognized risk factors and broader determinants of health, deserves focused study. Knowledge of trends in obesity by socioeconomic status within countries (e.g. increasing prevalence in low socioeconomic groups in low- and middle-income countries) will be important for projecting future health-care burdens and needs.
- Key "windows of opportunity" – when interventions can prevent excess weight gain in a sustained manner – need to be identified. Quantification of the attributable risk of the various influences on obesity development is challenging but necessary, and needs to take into account that influences on obesity act as a complex system in which neither causes nor intervention effects operate via a single pathway.
- When considering obesity prevalence and trends for children in any given generation and, particularly in relation to the circumstances of socially disadvantaged populations within all countries, there is a need to have data sources and analyses that are sensitive to societal stages and trajectories related to urbanization, socioeconomic change, and associated nutrition, activity, epidemiologic, economic and technology transitions.
- It is important to move toward a focus on the full distribution of body weight in children rather than only focusing on the upper (i.e. obesity) and lower (i.e. undernourished) ends of the weight distribution, or on the crossing of boundaries between normal/overweight/obese. Changes in adiposity and body composition may be important considerations for health as part of the overweight continuum.
- A key aspect of this change in focus will be tracking trends of incidence, rather than only prevalence of obesity. Focusing on the entire distribution will enable better understanding of transitions from underweight to normal weight and from normal weight to overweight as potential precursors of eventual obesity. Moreover, it is important to examine shifts in the upper levels of the body mass index (BMI) distribution to gain insights into the severity of obesity (e.g. the increase in mean BMI of overweight and obese children). Measuring the reverse trends, although much less frequent, may provide insight for intervention strategies.
- Tracking new cases can provide important clues about aetiology by highlighting periods in the life-course when excessive weight gain is most likely to occur. Knowing the time of onset will also permit accounting for the duration of obesity in studies of trajectories and health impact.
- There is a sizable potential contribution of surveillance of obesity trends in children to identify the scale of problem, trends and risk factors. Even small improvements in surveillance systems can serve as an intervention, especially in situations where current surveillance is weak or absent.

- There is potential to identify new risk factors or patterns of vulnerability to risk factors in ways that can lead to new intervention strategies, for example subgroups of infants for whom lack of breastfeeding poses a greater-than-average risk of developing obesity (such as offspring of gestational diabetes mellitus pregnancies).
- There is potential to identify or clarify the roles of novel risk factors for obesity development. The relationship between sleep duration and quality and obesity development is one such pathway which appears to be critical, based on current evidence.
- Another area for increased emphasis in the epidemiology of obesity is the role of the digital revolution and social media, including its use as a marketing tool and in social networking, and the resulting increase in sedentary behaviours. However, such technology might be used to gather information to improve health behaviours in individuals.
- It is important to improve understanding of the features associated with the reverse transition from obesity to overweight or normal weight, including gender, age and enabling factors.
- Consistent with the life-course approach, epidemiological studies of the amplification of risk, or cumulative risk, due to carry-over effects from prior periods, are needed.
- The relevance of household composition and family structures to risks of childhood obesity needs exploration.

3.3 The health consequences of childhood obesity throughout the life-course[1]

Worldwide, the prevalence of combined overweight and obesity rose by 27.5% for adults and 47.1% for children between 1980 and 2013. Katzmarzyk et al. describe this as a challenging problem "unprecedented in evolutionary terms" and with the capacity to negate many of the health benefits that have contributed to the increased longevity observed in the developed world *(4)*.

According to Bhattacharjee et al. *(5)* the complexity of the obesity problem lies in the "multi-systemic nature" of the condition, with its increased risk for psychological disturbances, gastrointestinal complications, cardiovascular disease and diabetes, as well as the comorbidities of the latter two. Three of these comorbidities – high blood pressure, insulin resistance and dyslipidaemia – form the key elements of the metabolic syndrome and are increasingly being observed in children *(6)*. Adding to the complexity of the problem, and also to the need for primary prevention, are the long-term health consequences which extend from childhood into the later phases of the life-course. Obesity in childhood increases the likelihood of obesity in adulthood, and, although not true for all comorbidities, is strongly associated with contributing to cardiovascular disease and diabetes *(6, 7)*. These negative health consequences can present even if normal weight is attained after childhood, suggesting that early development leaves a permanent imprint. Longitudinal studies suggest that for some noncommunicable disease-related comorbidities, the increased risk for morbidity and mortality in adulthood are independent of adult BMI *(8)*.

[1] Authors: Mark Hanson; Anniza de Villiers; Rachel Rodin, Linda Adair, Fereidoun Azizi, Zulfi Bhutta.

The risk of childhood obesity morbidity and mortality, and the need for ending childhood obesity, should therefore be considered in terms of immediate, short-term and long-term consequences, first as a residual effect of being obese as a child, and as a state of obesity carried over from childhood into adulthood. The noncommunicable diseases and comorbidities to which childhood obesity predisposes, and the evidence available for the impact on health status through the life-course is highlighted in this report.

The estimated burden of childhood obesity-related diseases

When discussing the impact of childhood obesity on noncommunicable diseases throughout the life-course, it is important to consider the burden of childhood obesity-related diseases against the broader burden of disease across the life-course. A systematic analysis of the burden of disease in young people by Gore et al. *(9)* reports that those aged 10–24 years represent 27% of the world's population. According to these authors, the health of this group has been largely neglected in global public health because they are considered to be healthy. Unipolar depressive disease, lower-respiratory infections and road traffic accidents are the 10 most important causes for disability-adjusted life years (DALYs) in young people of both age groups 10–14 years and 15–19 years. Although the noncommunicable disease-related risk factors of high blood pressure, cholesterol and glucose, tobacco use, physical inactivity, and overweight and obesity do not contribute significantly to DALYs in these age ranges, they become major public health threats in later life *(9)*. Furthermore, as mentioned in the previous section, many of the obesity-related comorbidities are now presenting in childhood and adolescence, as the prevalence reported by various authors and presented in Table 1 clearly show *(10, 11)*.

The data presented in Table 1, and the known burden of noncommunicable diseases in later life, strongly suggest that strategies to combat the rising burden of noncommunicable diseases should start early in life and focus particularly on adolescence when risk factors and lifestyles are established. Although they might not affect health during this period per se, they may have a substantial effect later in life *(9, 13)*. In view of the increased prevalence of comorbidities during adolescence, prevention should probably start even earlier in life. The following sections look at the current evidence for obesity-related health consequences from early to later life.

A large body of literature documents the health consequences of childhood obesity and the associated conditions and comorbidities predisposing children to noncommunicable diseases. The following sections provide a brief overview of one or two of the most recent and relevant references, as well as some background to the mechanisms involved in health consequences of obesity. Table 2 provides evidence for the short-term health consequences of childhood obesity and Table 3 for the longer-term consequences.

Table 1. Estimated numbers of children in Europe, the USA and China with obesity-related disease indicators

Comorbidities	Lowest estimated prevalence among obese children (%)	NHANES (1999–2008): adolescents aged 12–19 years (%)	2009 China Health and Nutrition Survey: children aged 7–17 years
	Europe	**USA**	**China**
Raised triglicerides	21.5	-	42% had at least one of the following cardiometabolic risk factors: • pre-diabetes/diabetes (HbA1c, 5.7%), • hypertension, • high TC, • high LDL, • low HDL, • high TG, and • high CRP
Raised total cholesterol	22.1	-	
High LDL cholesterol	18.9	22	
Low HDL cholesterol	18.7	6	
Hypertension	21.8	14	
Impaired glucose tolerance	8.4	15	
Hyperinsulinaemia	33.9	-	
Type 2 diabetes	0.5	-	
Metabolic syndrome	23.9	-	
Hepatic steatosis	27.9	-	
Elevated aminotransferase	12.8	-	

CRP: C-reactive protein; HDL: high-density lipoproteins; LDL: low-density lipoproteins; TC: total cholesterol; TG: triglycerides;

Sources: Lobstein TJ *(10);* Flynn J *(11);* Jan S et al. *(12)*

Table 2. Evidence for short-term health consequences of childhood obesity

		Author/ publication year	Publication type	Description of evidence
	Immediate/short-term consequences			
	Birth to 2 years			
1	Delayed motor development	Slining et al., 2010 *(14)*	–	Motor delay was 1.80 times more likely in overweight infants compared with non-overweight infants (95% CI: 1.09–2.97) and 2.32 times as likely in infants with high subcutaneous fat compared with infants with lower subcutaneous fat (95% CI: 1.26–4.29). High subcutaneous fat was also associated with delay in subsequent motor development (OR: 2.27; 95% CI: 1.08–4.76).
	Childhood and adolescence			
1	Asthma and other breathing problems	Papoutsakis et al., 2013 *(15)*	–	Current evidence supports a weak yet significant association between high body weight and asthma. The link may be stronger in non-allergic asthma.
2	Obstructive sleep apnoea (OSAS)	Bhattacharjee et al., 2011 *(5)*	Review	The authors conclude that obesity and OSAS share many common pathways that lead to the induction of chronic inflammation. There seems to exist of a causative link between OSAS, obesity, and cardiovascular or metabolic disease, which begins as early as childhood, even in the presence of a priori a normal cardiovascular system or a normal metabolic reserve. The exact mechanisms are likely multifactorial and need to be studied further.
3	Dysglycemia, type 2 diabetes	Van Name et al., 2013 *(16)*	Review	The authors refer to several studies pointing to the role of ectopic fat accumulation and hepatic steatosis in youth and the development of type 2 diabetes. They conclude that since this is the first generation in which this phenomenon is so diffuse, longitudinal data showing the long-term natural history of the early onset of diabetes are not yet available.

4	Elevated blood pressure	Flynn, 2012 (*11*)	–	Cross-sectional studies clearly suggest a significant impact of obesity on the prevalence of childhood hypertension, as well as on absolute blood pressure levels. Various potential mechanisms are discussed by the author.
5	Dyslipidemia	Cook & Kavey, 2011 *(17)*	Review	Childhood obesity – especially abdominal obesity – is strongly associated with a high prevalence of the atherogenic combined dyslipidemia described by the authors. It is pointed out that although these cardiometabolic abnormalities have not resulted in measurable increases in total or LDL cholesterol, adult and paediatric data have revealed qualitative changes in LDL and HDL. cholesterol associated with elevated measures of atherosclerosis among adolescents and with clinical disease in adults.
6	Chronic inflammation	Stolzman & Bement, 2012 *(18)*	Review	With the increase in adiposity that occurs with weight gain, a persistent low-grade inflammatory state is created. The most commonly studied inflammatory markers associated with obesity are the cytokines, tumour necrosis factor α and interleukin-6, and the acute-phase reactant, C-reactive protein. This review concluded that further research is needed to better understand the complexity of the chronic inflammatory state associated with obesity.
7	Dental health: caries and periodontal disease	Katz et al., 2011 *(19)*	Review	Further studies are needed to support the hypothesis that obesity in children may be associated with increased rates of periodontal disease.
8	Musculoskeletal problems	1. Wearing et al., 2006 *(20)* 2. Paulis et al., 2014 *(21)*	Review/ Systematic review	1. The delineation of the effects of childhood obesity on musculoskeletal structure in terms of mass, adiposity, anthropometry, metabolic effects and physical inactivity, or their combination, has not been established. More specifically, there is a lack of research regarding the effect of childhood obesity on the properties of connective tissue structures, such as tendons and ligaments. 2. Overweight/obesity is associated with musculoskeletal pain, injuries and fractures in childhood. More high-quality prospective cohort studies are needed to study the nature of this relationship.
9	Accelerated	Fennoy, 2013	Review	Growth patterns of obesity during childhood are well described, documenting increased linear growth in early childhood associated with accelerated pubertal maturation resulting in normal

	maturation	*(22)*		adult height. Despite recent data suggesting that ghrelin and the growth hormone secretagogue receptor, as well as the insulin-like growth factors, their binding proteins and insulin have potential to be mediators of nutrient exposure and linear growth, it remains to be determined how these systems interrelate and determine growth.
10	Mental/psychological health	Pizzi, 2013 *(23)*	Review	The authors discuss salient multiple psychosocial sequelae associated with childhood obesity, including weight bias and victimization. According to the authors, an undeniable relationship exists between obesity and psychological difficulties and disorders; but, for example, in depression it is not known whether obesity is comorbid or contributory. What is known is that obesity for a significant number of children and adolescents have been shown to have short- and long-term psychological consequences and that it is often associated with negative self-image, low self-esteem, eating disorders, and low health-related quality of life.
11	Executive functioning	1. Miller et al., 2014 *(24)* 2. Reinert et al., 2014 *(25)*	–	There is some evidence for obesity-associated biomarkers that have associations with neurocognitive skills, specifically executive functioning skills. Reinert et al. (2014) suggest the need for further longitudinal studies.
11	Skin problems	Mahe et al., 2014 *(26)*	Review	Psoriasis is a chronic inflammatory skin disease that can affect nearly 1% of children, even during the first months of life. A link with obesity has been demonstrated by a few studies.
12	Non-alcoholic fatty liver disease (NAFLD)	Pacifico et al., 2011 *(27)*	–	Several studies in the paediatric population have reported independent associations between NAFLD and impaired flow-mediated vasodilatation and increased carotid artery intimal medial thickness – two reliable markers of subclinical atherosclerosis – after adjusting for cardiovascular risk factors and MetS. Therefore, the rising prevalence of obesity-related MetS and NAFLD in childhood may lead to a parallel increase in adverse cardiovascular outcomes.
13	Neurological	Yau et al., 2013 *(28)*	–	The authors, after studying 42 adolescents with, and 62 without, MetS, documented lower cognitive performance and reductions in brain structural integrity among adolescents with MetS. They argue that these findings indicate that it is plausible that obesity-associated metabolic disease, short of type 2 diabetes mellitus, may be mechanistically linked to lower the academic and professional potential of adolescents.

14	Renal abnormalities	Savino et al., 2010 *(29)*	Review	There is clear evidence that, in adults, excess body weight is significantly associated with an increased risk of kidney disease. A similar association has also been documented in obese children and adolescents, but there is a lack of large and long-term studies.
15	Gastroenterological problems	Luoto et al., 2013 *(30)*	Systematic review	The authors conclude that recent scientific advances point to an aberrant compositional development of the gut microbiota and low-grade inflammation as contributing factors, in conjunction with excessive energy intake. A high-fat/high-energy diet alters the gut microbiota composition, which reciprocally engenders excessive energy harvesting and storage. Further, microbial imbalance increases gut permeability, leading to metabolic endotoxemia, inflammation and insulin resistance. Local intestinal immunologic homeostasis is achieved by tolerogenic immune responses to microbial antigens. Causality in humans however remains to be proven.
16	Menstrual problems/reproductive function/polycystic ovary syndrome (PCOS)	Rosenfield, 2013 *(31)*	Review	Obesity may itself be a common unrecognized cause of adolescent ovulatory dysfunction. It seems to disrupt the ovulatory cycle by suppressing gonadotropins and by increasing insulin resistance. Obesity also can raise androgen levels, adipocyte type 5 17β-hydroxysteroid dehydrogenase, an enzyme that is up-regulated by insulin, forms T from circulating androstenedione; the expression of this enzyme in subcutaneous fat correlates with BMI and falls with weight loss in simple obesity. The authors conclude that obesity may cause a PCOS picture.

BMI: body mass index; CI: confidence interval; HDL: high-density lipoproteins; LDL: low-density lipoproteins; MetS: metabolic syndrome; T: testosterone; OR: odds ratio.

Table 3. Long-term health consequences of childhood obesity

		Authors	Source	Findings
	Long-term consequences			
1	Persistence of overweight/obesity	1. Lee, 2009 *(32)* 2. Singh et al., 2008 *(33)*	Review/ Systematic review	1. Nearly half of obese children proceeded to obese adults. 2. All included studies consistently report an increased risk of overweight and obese youth becoming overweight adults, suggesting that the likelihood of persistence of overweight into adulthood is moderate for overweight and obese youth. However, predictive values varied considerably.
2	Asthma and other breathing problems	Park et al., 2012 *(34)*	Systematic review	One study demonstrated indirectly positive association; the other indicated no association. Studies that use more robust designs and analytical techniques are needed to establish whether childhood obesity is an independent risk factor for adult disease.
3	Obstructive sleep apnoea (OSAS)	Inge et al., 2013 *(35)*	Retrospective study	Adolescent severe obesity had association with sleeping apnoea in adults (ARR: 1.2; CI: 1.08–1.34)
4	Dysglycaemia, type 2 diabetes	Park et al., 2012 *(34)*	Systematic review	Increased overweight in childhood and adolescence associated with increased risk in adulthood. This association, however, became not significant when adjusted for adult BMI.
5	Elevated blood pressure	Park et al., 2012 *(34)*	Systematic review	Two of the five studies reported findings adjusted for adult BMI. In one study, individuals who had been overweight at ages 8–15 years had five times the risk of hypertension in early adulthood compared with those who were normal weight (OR: 5.1, 95% CI: 1.4–18.1); after adjustment for BMI at ages 18–26, the effect size remained the same, but the CI was wider and included OR=1. In the other study, associations between BMI-SDS at ages 7, 11 and 16 and clinically assessed hypertension at age 45 were no longer observed after controlling for BMI at age 45 years.

6	Dyslipidaemia	Cook & Kavey, 2011 *(17)*	–	
7	Metabolic syndrome (MetS)	Lloyd et al., 2011 *(36)*	Systematic review	Of the four papers included in the systematic review that considered metabolic syndrome as an end point, none showed evidence of an independent association with childhood obesity.
8	Chronic inflammation	Codoner-Franch et al., 2011 *(37)*	Review	The authors identified a close link among obesity, a state of chronic low-level inflammation, and oxidative stress. In addition, the dysregulation of adipocytokines, which are secreted by adipose tissue and promoted by oxidative stress, act synergistically in obesity-related metabolic abnormalities. Adipocytokines link the local and systemic inflammation responses in the context of obesity.
9	Dental health: caries and periodontal disease	Chaffee et al., 2010 *(38)*	Systematic review	A consistent positive association was found coherent with a biologically plausible role for obesity in the development of periodontal disease. The authors however point out that with few quality longitudinal studies, there is an inability to distinguish the temporal ordering of events, thus limiting the evidence that obesity is a risk factor for periodontal disease.
10	Cardiovascular disease (CVD)	Park et al., 2012 *(34)*	Systematic review	Two of the studies reported, looked at the independent effect of childhood obesity. In one study (*n*=37 674), the HR for incident CHD at ages 25–45 for men with BMI in the top decile at age 17 (compared with the bottom decile) increased from 5.43 (95% CI: 2.77–10.62) to 6.85 (95% CI: 3.3–14.2) after adjustment for BMI in adulthood. Another study (*n*=181), which reported an association between overweight in adolescence and CHD mortality among men, found that this association was attenuated to null after adjustment for adult BMI.
		Lloyd et al., 2010 *(36)*	Systematic review	Several studies identified weak positive associations between childhood BMI and adult total cholesterol, low-density lipoprotein-cholesterol, triglyceride and insulin concentrations, these associations were ameliorated or inversed when adjusted for adult BMI or body fatness.

11	Dementia	Garcia-Ptacek, 2014 *(39)*	Review	The authors conclude that obesity and overweight as measured by BMI are important modifiable risk factors for dementia and AD. Evidence from observational studies seems to indicate that the relationship between BMI and mortality and BMI and cognition might be different depending on age group, sex, ethnicity and comorbidity. Most of the epidemiological studies confirm that obesity in midlife is a risk factor for the development of dementia, while high BMI in later stages of life is protective. The ongoing increase in obesity in midlife may contribute significantly to the future prevalence of dementia.
12	Non-acoholic fatty liver disease (NAFLD)	Girogio et al., 2013 *(40)*	Review	NAFDL is extremely prevalent (estimated to be between 3% and 10%) and predicted by obesity and male gender. NAFDL in children is strongly associated with several other features of the metabolic syndrome in children and increases the risk of developing CVD in adulthood.
13	Musculoskeletal problems	Wills et al., 2012 *(41)*	Cohort studies	Obesity from childhood throughout adulthood was associated with higher risk of knee osteoarthritis in adults.
		Macfarlane et al., 2011 (*42*)		
14	Cancers: kidney, breast, cervical and ovarian	Park et al., 2012 *(34)*	Systematic review	One study included showed higher BMI-SDS in childhood associated with increased odds of cancer (cases and mortality) while another reported higher BMI in adolescence associated with increased risk of cancer mortality in women only. One study showed no evidence of association.
	Kidney cancer	Bjorge et al., 2004 *(43)*	Cohort study	Adolescent overweight was associated with increased risk of renal cancer in boys. Confounded adult BMI was unknown.
	Cervical cancer	Bjorge et al., 2008 *(44)*	Cohort study	High BMI in adolescence was related to increased risk of cervical cancer in women without adjustment for adult size.
15	Polycystic ovary syndrome	Reilly et al., 2011 *(45)*	Systematic review	Child and adolescent overweight and obesity were associated with significantly increased risk of later polycystic ovary syndrome symptoms.

16	Infertility	Frisco & Weden, 2013 *(46)*	Review	Young women who were obese at baseline had higher odds of remaining childless and increased odds of underachieving fertility intentions than young women who were normal weight at baseline.
17	Reduced likelihood of breastfeeding	Lepe et al., 2011 *(47)*	Review	This review shows that in prospective studies, obese mothers are more likely to have delayed lactogenesis and reduced lactation. The delayed lactogenesis could be explained by the lower prolactin concentration showed in obese mothers at rest and after suckling.

AD: Alzheimer's disease; ARR: absolute risk reduction; BMI: body mass index; CHD: coronary heart disease; CI: confidence interval; HDL: high-density lipoproteins; HR: high rate; LDL: low-density lipoproteins; OR: odds ratio; SDS: standard deviation score.

Table 2 indicates that there are varying levels of evidence for the health consequences of obesity in early life. For most health consequences, authors recommend further studies to be conducted, however, delayed motor development during infancy, and dyslipidaemia in childhood, are two for which clear evidence exists. If, however, the estimated number of children with obesity-related disease indicators (as presented in Table 1) is considered together with the large number of noncommunicable disease-related conditions, for which there is some evidence that excess adiposity may be involved, the need for action is clear.

The evidence presented in Table 3 – showing that health consequences later in life are stronger than that for early life – is to be expected from the path dependency model of increasing disease risk across the life-course. However, there are indications that for some conditions there are no associations when data are adjusted for adult BMI or body fatness. Many of the studies included in the reviews presented in Table 2 have not made this adjustment. Nonetheless, a strong association exists between adult obesity and noncommunicable diseases such as cardiovascular diseases and diabetes, and studies consistently report an increased risk of overweight and obese youth becoming overweight adults.

Other issues to consider in noncommunicable disease risk and childhood obesity

In addition to the direct evidence for health consequences of childhood obesity, other factors relating to noncommunicable disease risk arising from obesity in early life need to be considered. These include:

Types of obesity and role of adipose tissue

Healthy ranges of measures of adiposity, such as BMI, vary, for example, across populations because body shape differs. Misra and Bhardwaj *(48)* point out that the phenotype of obesity and body fat distribution associated with insulin resistance and the metabolic syndrome are distinctive in people from south Asia. Cut-offs for measures of adiposity for this population had to be adjusted as they appear to be "metabolically obese" although they may have normal BMIs. According to these authors, this phenomenon could be partially explained by excess intra-abdominal and subcutaneous fat and ectopic fat deposition in various organs and body sites. This explanation is confirmed in a recent review by Bastien et al. *(49)* in which the authors suggest that the quality and function of adipose tissue is as important, or even more so, than its amount, in determining the overall health and cardiovascular risks of overweight/obesity. They describe adipose tissue as an endocrine organ "orchestrating crucial interactions" with vital organs and tissues such as the brain, the liver, skeletal muscle and the heart and blood vessels. Adipose tissue plays an important role in the fate of excess dietary lipids, which may determine whether or not body homeostasis will be maintained (metabolically healthy obesity [MHO]) or a state of inflammation/insulin resistance will be produced, with negative cardiovascular consequences. Visceral fat, especially, induces a variety of changes in cardiovascular structure and function. The effect of different patterns of fat deposition (e.g. central adiposity) is therefore important to consider in relation to noncommunicable disease risk. In another recent review, Roberson et al. *(50)* explore the metabolically healthy obese phenotype and suggest that more longitudinal cohort studies are needed to examine the transition between MHO and becoming metabolically unhealthy and obese.

In addition to different BMI ranges for different populations, Janssen et al. *(51)* point to the possibility that the obesity phenotype may change in a given population, reporting that body composition of present day Canadians has changed "more adversely" than body weight, with higher waist circumference and skinfold thicknesses for a given BMI now than observed in 1981. This is of concern as waist circumference is a stronger predictor of obesity-related morbidity and mortality risk than BMI *(51)*.

Pathways linking overweight/obesity to noncommunicable diseases

The response of an individual to, for example, an obesogenic environment, depends, in part, on their development. This is similar to the concept of path dependency in other fields, as described by Barnes et al. *(52)*. There is considerable ongoing research on how responses to noncommunicable disease risk track across the life-course, based on the concept that physiological responses are established during critical developmental windows. Whilst well known for processes such as visual function, the principle is now being applied to appetite, food preference, metabolic control and adipocyte number and type *(53)*. There may be common underlying mechanisms to such effects, for example the link with inflammatory processes described by various authors *(54, 55)*.

Wider social issues related to pathways involve ethnic differences *(56)* and amplification by migration in childhood *(57)*. It is not known how these factors influence the mechanisms underlying the pathway dependency.

The life-course approach for noncommunicable disease prevention

It is evident from the literature that a life-course approach is important when considering obesity treatment and prevention to reduce and prevent noncommunicable disease risk. While noncommunicable disease reduction in adults, through weight control, is proving difficult – as is shown by a recent review *(58)* – obesity reduction interventions in children aged 6–12 years had small if variable effects *(59)*. This contributes to the argument for earlier intervention and is also supported by evidence that obesity risk could be established in gestation or even preconception and possibly be influenced by parental diet, maternal obesity as well as gestational weight gain *(60)*.

Major challenges and gaps in knowledge

- Linkage between overweight/obesity and noncommunicable diseases operate across the normal range. It is not known whether the same pathways are involved across the range. More data are needed on the degree of attributable risk for various noncommunicable diseases resulting from childhood obesity.
- There appears to be no threshold effect, for example, for BMI and later risk.
- Further work is needed on the links between patterns of body fat deposition (in relation to gender, age, ethnicity, developmental influences) and risk.
- To what extent, and up to what age, are the effects reversible?
- What are best biomarkers of risk?

Summary

Although the level of attributable risk for many noncommunicable diseases arising from childhood obesity is not known, the concept of path dependency suggests that early interventions to improve responses to later noncommunicable disease risks, such as an obesogenic lifestyle, will have major long-term health benefits. There is evidence that interventions to reduce modifiable risk factors during both early development and childhood can reduce childhood obesity. These should be refined to be culturally-specific, linked temporally to optimize the path to health versus disease and taken to scale. There is a need to conduct more research in parallel to establish the underlying mechanisms and effective biomarkers of risk and intervention efficacy.

3.4 The psychosocial determinants and consequences of childhood obesity[1]

The psychosocial determinants of childhood obesity include caregiver and individual responses to innate factors and to a broad set of determinants in external contexts, as well as health knowledge and ability to act upon this knowledge in ways that mitigate obesity risks. Relevant environmental context determinants include environmental influences, socioeconomic factors, and cultural norms relating to eating/feeding and movement behaviour (i.e. physical activity, sedentary behaviour, and sleep), and body image. These determinants are of particular interest to the Commission's deliberations because of their potential relationship to differences in obesity prevalence and the nature of obesity interventions that are appropriate and potentially effective in low- and middle-income countries compared with high-income countries, and also in different sociodemographic and cultural groups within countries.

Obesity prevalence rates are higher in high-income countries compared with low- and middle-income countries. However, in absolute numbers there are more overweight and obese children living in low- and middle-income countries than in high-income countries *(3)*. There is no consistent pattern of change in rates of childhood overweight and obesity across low- and middle-income countries. There are better data on overall prevalence in particular countries and WHO regions. In Africa, the estimated prevalence rate of child overweight and obesity of 8.5% in 2010 (12 million children) is projected to increase to 12.7% by 2020. In Asia, the 2010 prevalence rate of 4.9% equates to approximately 18 million children *(61–63)*.

Two important factors distinguish emerging patterns of childhood obesity and overweight in low- and middle-income countries from the pattern seen in high-income countries. First, the nutritional profile of low- and middle-income countries is often characterized by a dual burden of both

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undernutrition and overnutrition. While rates of childhood overnutrition in these settings appear to be increasing, this does not necessarily mean that rates of undernutrition are decreasing. Rather, there is wide variation in the pattern and rate at which change in nutritional status occurs. Second, unlike the inverse relationship between socioeconomic status and childhood overweight and obesity that characterizes high-income countries, in low- and middle-income countries the highest rates of overweight and obesity are usually found among children of higher socioeconomic status.

Evidence from high-income countries[1]

Ethnicity and obesity prevalence

Variation in obesity prevalence among ethnic groups and socioeconomic strata provides clues to differences in psychosocial, cultural, and other environmental context influences on obesity. Ethnic differences in obesity prevalence and trends within high-income countries are exemplified by data from black, white, and Hispanic children in the USA, for example, the higher prevalence among Hispanic (22.4%) and non-Hispanic blacks (20.2%) versus whites (14.1%) *(64)*, with disparities evident as young as 2 years of age. Children in these ethnic minority populations also bear a disproportionate share of the burden of obesity-related comorbidities *(65)*. Obesity trends over time also differ. In the USA, for example, although prevalence among some subpopulations, such as whites and those of higher socioeconomic status, may have peaked, ethnic and socioeconomic inequalities appear to be widening *(66)*. A review of studies from the United Kingdom on this topic concluded that children of south-east Asian origin may have a higher prevalence of obesity compared with Caucasian children; and, less convincingly, black (including black African and black Caribbean) children may also have a higher prevalence *(67)*. While this review concluded that children of Chinese origin in the USA appeared to have a lower prevalence of obesity relative to Caucasian children, this conclusion seems premature given that only one study of Chinese-origin children was included, and also given the inconsistent findings across studies for children of other ethnic origins.

First-nation children (i.e. children in aboriginal or indigenous populations in high-income countries) appear to be at increased risk of overweight and obesity. Higher obesity prevalence in these children compared with the general population has been reported in New Zealand, Australia, the USA and Canada *(68)*. Research from Austria, Germany, the United Kingdom, the Netherlands, New Zealand and Australia consistently reports that migrant children or children speaking a non-local language at home had higher prevalence of overweight and/or obesity than non-migrant children. In a recent study involving seven European countries, higher BMI, waist circumference and overweight and obesity prevalence was observed in children aged 10–12 years of non-native origin (defined by language spoken at home and parental country of birth) in Belgium, Hungary, the Netherlands, Norway, Slovenia and Spain. The only country where this trend was not observed was Greece, where Greek-origin children of the 10–12 year age range, as well as those aged 1–5 years, had a higher prevalence of overweight and obesity than those of non-native origin *(69)*.

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Ethnic differences in risk factors for childhood obesity by developmental stage

Maternal obesity is one of the strongest and most reliable predictors of later obesity in children, and pre-pregnancy obesity is more common among non-Hispanic black and Hispanic women compared with non-Hispanic white women in the USA *(70)*; among non-European migrants, compared with Europeans living in the Netherlands *(71)*; and among black and south-Asian women (using an Asian-specific BMI criteria) compared with white women in the United Kingdom *(72)*. Thus, with the tendency being for obese children to become obese adults, ethnic minority children are born into a cycle of obesity more often than their white counterparts. Opportunities to interrupt this cycle and reduce ethnic inequalities in obesity exist at every stage of the life-course. For example, evidence suggests that reducing the prevalence of obesity risk factors before and during pregnancy, infancy, and early childhood could close the gap in obesity inequalities in mid-childhood *(73)*. Interventions in early life are therefore crucial in stemming the rise of obesity and related inequalities.

Previous studies have examined ethnic differences in pregnancy-related risk factors for childhood obesity. In a national study by Chu et al. *(74)*, black and Hispanic women were more likely than white women to begin their pregnancies already overweight or obese and gained less weight during pregnancy. The same pattern was observed in non-European migrants in the Netherlands *(71)*. One study has found that Hispanic women have a higher risk of gestational diabetes. Women of Asian origin have been found to be at increased risk of a range of pregnancy-related complications including gestational diabetes, at a similar or lower pre-pregnancy BMI than white women *(75)*.

A recent study by Taveras et al. has shown that ethnic differences in risk factors for obesity exist prenatally and in early childhood *(76)*. In this study, children from under-represented ethnic groups were found to have a higher risk of various obesity risk factors compared with their white counterparts. Specifically, black and Hispanic children had higher odds of their mothers experiencing antenatal depression, weight gain during infancy, introduction of solid foods before 4 months of age, maternal restrictive feeding, television in their bedrooms after age 2, intake of sugar-sweetened beverages and “fast foods”. Conversely, black and Hispanic children had lower odds of protective factors including exclusive breastfeeding and sleep for more than 12 hours per day during infancy. Findings across other high-income countries show similar early life risk for non-native groups. In Australia, Chinese-speaking women have lower intention and initiation rates for breastfeeding than English-speaking women, but better continuation rates, to 8 weeks *(77)*. In the United Kingdom, Vietnamese immigrants express lack of confidence to initiate breastfeeding *(78)*. Together, these findings may help to explain ethnic inequalities in early childhood obesity rates while justifying the need for early childhood interventions to reduce these inequalities in obesity prevalence.

Several studies of older children in the USA and the United Kingdom have found obesity-related risk factors to be more prevalent among ethnic minority groups. Previous studies have found higher levels of television viewing and more televisions in bedrooms, higher consumption of sugar-sweetened beverages, increased “fast food” consumption, and lower levels of physical activity among black, Hispanic and Asian children and youth compared with white children. Similar patterns

are reported in European studies of immigrant children. In Norway, dietary intake, dieting behaviour, physical activity and screen use differed by region of parental birth in immigrant children *(79)*. A German study reported television viewing, in combination with maternal education, explained almost all variance in obesity prevalence between immigrant and non-immigrant children *(80)*. A recent study involving seven European countries (Belgium, Greece, Hungary, the Netherlands, Norway, Slovenia and Spain), showed non-native children had higher intakes of sugar-sweetened beverages, more breakfast-skipping and screen time, and less time spent engaging in sports activities and sleep than their native counterparts. The only obesity-related risk factor showing an opposite association was walking to school, which was more prevalent in non-native children. While the magnitude of effects differed between the seven countries, trends were generally consistent *(69)*. In Australia, children of south-east Asian origin are at significantly higher risk of being inactive than native-born children *(81)*.

Socioeconomic status and position and obesity in high-income countries and emerging economies

Socioeconomic status is a multidimensional construct that is known to exert a profound influence on health. While there are some mixed findings from individual studies, generally there is evidence of inverse associations between socioeconomic position and overweight/obesity prevalence in children, whether socioeconomic position is assessed by neighbourhood or individual level indicators. This has been reported in 21 of 24 high-income countries included in a large data-pooling study spanning child and adolescent cohorts (of age 11, 13 and 15 years) *(82)* and from additional child studies in Denmark, France, Germany, the United Kingdom, Spain, Sweden, Australia and New Zealand. Inverse associations between BMI and socioeconomic position have also been reported in data from Spain, Belgium, and Greece. It is suggested that the relationship between socioeconomic position and overweight/obesity is approximately linear, rather than an effect observed only in the most deprived. Importantly, it appears that the inverse association between socioeconomic position and overweight/obesity has increased over recent decades, evidenced both by increasing consistency in findings from individual studies post 1990 and a secular trend of increasing strength of associations reported in consecutive data collections in the United Kingdom, Australia and France.

No obvious difference in overweight/obesity prevalence by socioeconomic position was evident in data from other studies in Greece or the Russian Federation. It is possible that the lack of association in these samples is due to the younger cohorts studied (1–5-year-olds from Greece and 6–7-year-olds from the Russian Federation), with socioeconomic gradients not developing until later in childhood. However, given the inverse associations observed in other samples of younger children, for example from Australia and France, this seems an unlikely explanation. A more plausible explanation is that there are cultural, economic transitions or other factors explaining the lack of associations observed in the Greek and Russian Federation samples. The same pattern was noted in migrant children living in Germany (the opposite pattern to that observed for German-born children).

While findings of individual studies are mixed, there is no strong evidence for sex differences in associations between socioeconomic position and obesity in children, despite sex being a moderating factor in this association for adults. Consistent with what is observed in adults, there

appears to be no association between socioeconomic position and obesity in non-Hispanic black children in the USA. The exact role of socioeconomic position in the relationship between ethnicity and obesity is unclear. Singh et al. *(83)* found that the magnitude of the effects of socioeconomic position on obesity among 10–17-year-olds varied by ethnicity. For example, the effects of socioeconomic position on the odds of obesity were larger for Hispanics than non-Hispanic whites and blacks. Additionally, after adjusting for maternal education, household income, and children's food security status, the increased odds of obesity risk among Hispanic children aged 3 years did not change substantially, suggesting that the increased prevalence of obesity in Hispanics, compared with blacks or whites, was not explained by ethnic differences in socioeconomic indicators. Taveras et al. *(76)* recently found that socioeconomic factors did not explain most ethnic differences in prenatal and early childhood risk factors for obesity. In this study, adjusting for socioeconomic factors markedly attenuated (> 30%) ethnic differences in smoking during pregnancy, maternal depression, breastfeeding initiation and duration, and having a television where the child sleeps. However, adjusting for socioeconomic position did not completely eliminate observed ethnic differences in obesity risk factors. Thus, existing evidence does not suggest that differences in socioeconomic status primarily explain ethnic inequalities in obesity.

Explanatory factors linking ethnicity and socioeconomic position to obesity

It has been suggested that differences in overweight and obesity prevalence by ethnicity and socioeconomic position are likely to be due, in large part, to differences in the proximal behaviours of diet, physical activity and sedentary behaviour observed by ethnicity or socioeconomic position. Further, intergenerational influences on childhood obesity prevalence (e.g. via intrauterine environment), and on family food and activity environments, are likely to play an important role. There is also some evidence to suggest ethnic and socioeconomic differences in overweight/obesity prevalence may also be explained by corresponding differences in body satisfaction, weight-related attitudes and beliefs, values, health knowledge, sociocultural norms, parental feeding practices and neighbourhood characteristics (e.g. less availability of healthy foods and sporting facilities in low socioeconomic position neighbourhoods). Differences in these postulated explanatory values begin early in life, with ethnic or socioeconomic discrepancies in some of these factors evident by the time children are in their preschool years (~3–5 years).

Contribution of culture and acculturation to ethnic differences in obesity risk factors in high-income countries

It is important to examine culture and acculturation as underlying factors that may contribute to ethnic inequalities in obesity. Cultural issues including differing perceptions of desirable body weight and body satisfaction have also been posited to explain ethnic differences in obesity prevalence. Studies in the United Kingdom indicate that black children have significantly higher body esteem than Asian children *(84, 85)*; this corresponds with strong adult evidence that black women are less concerned with their body weight and more accepting of being overweight than their white counterparts.

Accumulating evidence has shown a change in health status that correlates with more time spent in the USA. In particular, studies have shown changes in traditional diet components across generations. Across generations, the diets of Latin American migrants, in comparison with the diets of whites, have transitioned from being better to worse, with Latin American migrants consuming less fruits and vegetables and drinking more soda across generations; high fat and high sugar foods, not in the traditional Hispanic diet, were regularly available for children, suggesting that children may have experienced transitions in food preferences. Immigration and nativity status may also interact with other socioeconomic factors. For example, income was inversely associated with BMI among kindergarten children of USA-born Hispanic and white parents. On the other hand, income was positively associated with BMI among foreign-born Hispanic families *(86)*. Cultural differences among immigrants may explain the different associations and interactions.

Culture may also play a role in shaping parental perceptions of their children's health status. Mothers may have different preferences for what they consider a "healthy" child. In some cultures, mothers may view thinness as a reflection of poor health and malnutrition. For example, some evidence suggests that Hispanic mothers may perceive heavier children as healthier children. Thus, they may encourage their children to eat more. A qualitative study also showed that Hispanic mothers may perceive a child who is "not hungry" as worrisome. Such culturally-defined perceptions of body image may influence parenting strategies and decisions regarding eating habits, impacting the amount of food children eat and their risk of overweight and obesity. At the same time, some parents may not perceive their children as obese. In a study including overweight to very obese African–American children aged 5–10 years, although 90% of boys and 80% of girls were obese or very obese, only 30% of their parents classified their child as being very overweight *(87)*. However, lack of recognition by parents of a child's obesity status is also commonly reported in non-ethnic-specific populations and, without studies comparing such perceptions across cultural groups, this cannot be assumed to be a contributing factor.

A useful framework for conceptualizing the interplay between cultural factors and obesity-related risk factors in the association between ethnicity and obesity has been proposed by Kumanyika and colleagues *(68)*. Their Community Energy Balance Framework brings together individual cultural and environmental contextual influences to provide a model for considering ethnically-appropriate intervention strategies, likely to improve the effectiveness of strategies aiming to reduce the risk of obesity among at-risk ethnic groups.

Other psychosocial risk factors for obesity

Behavioural problems have been shown to be associated with increased obesity prevalence in children. Further, depression, chronic stress (abuse or neglect) and significant behavioural problems in childhood or adolescence have been shown to predict the development of obesity in adulthood. The evidence base in this area for pre-adolescent children remains small.

Hemmingsson recently proposed a causal model *(88)* whereby socioeconomic disadvantage causes psychological and emotional distress that leads to maladaptive coping strategies (including obesity-promoting behaviours and biological adaptations) and subsequently to overweight and obesity. Within his definition of psychological and emotional distress, he includes low self-esteem, low self-worth, depression, anxiety, and heightened sensitivity to stress. He suggests that children in socioeconomically disadvantaged families are particularly at risk of distress due to their exposure to heightened parental stress, discord, lack of parental support, negative belief systems and general insecurity, putting them at increased risk of overweight and obesity.

Summary

In high-income countries, children in ethnic minority populations, including migrant and first-nation children appear to be at increased risk of overweight and obesity. This may, in part, be due to different environments and lifestyles and, for migrants, different adaptations to a non-traditional environment and lifestyle. Evidence related to particular ethnic groups within populations varies.

Socioeconomic status appears to be inversely associated with overweight and obesity prevalence among children in high-income countries. This association appears to be approximately linear, rather than applying only to those children classified as most deprived. There are some exceptions of countries classified as high income but reporting no association, or an opposite gradient; however, these countries appear to be those with more recent economic transitions. While little research to date has considered psychosocial risk factors for obesity, there is evidence supporting the targeting of prevention efforts to children experiencing behavioural problems, depression and chronic stress in addition to the universal strategies directed to children in the population at large.

Evidence from low- and middle-income countries[1]

The production of childhood overweight and obesity at the individual, family and local community level involves relationships among the individual child, caregivers, family, and the interactions between these, set in the context of local social and cultural norms, framed within broader sociopolitical and environmental realities. Particularly important are locally- and nationally-framed eating behaviours, norms for physical activity, and body size, according to gender and at different stages of childhood. Physical and psychosocial development in childhood are closely intertwined in all societies; but the majority of research carried out on the psychology of learning behaviours related to food in early childhood has been carried out in high-income countries.

Determinants of childhood overweight and obesity in low- and middle-income countries can be considered at the levels of the individual, family and local community, and in environmental and wider sociopolitical contexts. It is useful to consider these within a life-course framework, given that overweight and obesity in early childhood, later childhood, and adolescence are likely to have different psychosocial and developmental determinants and will vary according to context across

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low- and middle-income countries. Additionally, earlier life experiences will influence the likelihood of becoming overweight or obese in later life. As shown in Poland (see Box A), longitudinal studies in low- and middle-income countries show that prevalence rates of childhood obesity and overweight follow a variety of patterns from early childhood through to adolescence *(62, 63)*.

Box A. Case study: gender differences in childhood obesity trajectories in Poland

It is important to separate different stages of childhood when examining rates of obesity and overweight, as well as changes in political–economic circumstances. The example of Poland is illustrative of this. While gender differences are small in early childhood, there are slightly higher rates of overweight and obesity among males than females in adolescence (89). A study of children in Cracow, comparing childhood overweight and obesity before and after the economic transition which began in 1990, shows rates of overweight and obesity to have increased more in females than males before 1990; but while rates continued to increase among males after 1990, they only continued to do so among pre-adolescent females, showing little change among adolescent females (90). The absence of continued increases in overweight and obesity rates among adolescent females may be due to sociocultural pressures associated with the transition to a free market economy in Poland. The post-communist economic transition took place very quickly in Poland. Among the many upheavals, adolescents have been exposed, by mass media, to Western standards of beauty which strongly emphasize thinness among females. Such exposure has created a general desire among girls to be thin and/or to lose weight, and among boys to increase muscle mass. The extent to which females attempt to achieve an idealized, thin body as portrayed by the media, is far greater in adolescence than in childhood, and gender differences in rates of obesity and overweight may represent societal differences in the level and type of agency that children have over their diet, activity patterns, and bodies.

There are few studies in low- and middle-income countries on the links between antenatal nutrition, infant feeding and childhood overweight or obesity. An antenatal diet, deficient in protein energy and micronutrients, is associated with offspring obesity in adulthood, and antenatal supplementation with zinc, folate and iron may decrease childhood fatness *(91)*. The protective effects of breastfeeding demonstrated in high-income countries may not be easily extrapolated to low- and middle-income country settings given that studies in high-income countries generally compare breastfeeding with formula feeding whereas in some settings the early introduction of other foods may be more relevant *(91)*. In the few studies in low- and middle-income countries that have examined this relationship, the association between breastfeeding and lower-body fatness across childhood holds; but these studies have been undertaken in societies where the cultural norm of breastfeeding is under challenge. There is also limited evidence from low- and middle-income countries of the effects of the timing of introduction of complementary foods on childhood overweight or obesity outcomes; a systematic review of studies (mostly from high-income countries) found some evidence for an association between complementary feeds before four months of age and childhood overweight *(92)*. Both formula feeding and early introduction of complementary

feeds are potentially linked to rapid weight gain in infancy, which may be associated with obesity in later life. In many societies, rapid infant weight gain is culturally desirable.

Cultural and behavioural shifts that could contribute to obesity and noncommunicable diseases include poor quality diets, reduced physical activity, increases in sedentary behaviours, and, possibly, insufficient sleep. In Latin America and the Caribbean, as in the USA, dietary consumption has shifted towards more highly-refined carbohydrates with higher glycaemic loads, as well as reduction in some healthful foods such as beans and whole grains. Substantial evidence also suggests that higher consumption of sugar-sweetened beverages can increase the risk of weight gain and cardiometabolic diseases. Sugar-sweetened beverage consumption and consumption of foods with added sugar have increased dramatically among populations in Mexico and the Caribbean. Most concerning is the early introduction of sugar-containing liquids and foods to infants and the potential for habituation to sweetened foods and drinks. Among very young children, modifiable risk factors for excess weight gain include: type and duration of infant feeding; timing of introduction and quality of complementary foods; parental responsiveness to infant hunger and satiety cues; sugar-sweetened beverage intake; and infant sleep duration.

Unlike the general trend seen in high-income countries, maternal education may increase the likelihood of childhood overweight and obesity in low- and middle-income countries, given that education is associated positively with higher socio-economic status. Infant and child feeding are informed by the health beliefs and behaviours of the parents, but also potentially of the grandparents, extended family and community. As is the case in China (see Box B), there may be gendered differences in child feeding and family food allocation, as well as in levels of physical activity and child perception of body image as shaped by sociocultural context *(93)*. In some low- and middle-income countries, girls may engage less than boys in outdoor activities or sport; they may be more involved with domestic chores, and may have less freedom than boys to participate in activities outside of the home *(94)*. Post-puberty, girls may be more likely to demonstrate concern about diet and body image, but this will vary across different low- and middle-income countries *(93)*. There are clear gaps in knowledge of gender differences in risk for overweight and obesity in low- and middle-income countries.

Box B. Case study: gendered effects on changes in obesity risk in China

Of the low- and middle-income countries, China has experienced the fastest changes in diet and physical activity over the past decade. These include increased consumption of animal products, oils, fried foods and snack foods, and a shift to increased screen time and decreased physical activity. Although this transition has unfolded differently across different regions, there is an overall convergence of urban and rural experiences (95). Of interest in the Chinese example are the potential effects of the government's low fertility policy on childhood overweight and obesity outcomes via child-caregiver dynamics (96). First, the positive effects of sibship on obesity risk seen in some other low- and middle-income countries does not appear to apply in China, where single children and children with siblings are treated similarly given that households usually have few children who have

similar access to resources. Second, the higher prevalence of overweight and obesity in boys than in girls may reflect sociocultural values in which obesity in boys is not viewed as detrimental or unbearable, while girls prefer a slender shape and are more likely to control their weight compared with their male counterparts. The Chinese 2005 NYRBS (National Youth Risk Behaviour Surveillance) reported that, nationally, 4.3% of boys and 2.7% of girls consumed "soft" drinks frequently; 23.6% of girls and 9.1% of boys tried to lose weight by restricting diet, and 29.1% of Chinese boys spent 2 hours per day playing computer games, which was 2.0 times higher than in girls (97). Third, parental expectations of academic excellence mean that Chinese children devote considerable hours to after-school study at the expense of sport and other extracurricular activities (98).

This example illustrates the importance of interpersonal factors in childhood overweight and obesity risk, and how these are context-specific and culturally informed.

Children in many low- and middle-income country settings live in increasingly obesogenic environments, as diets change and levels of physical activity decrease secondary to urbanization *(94)*. Comparisons between children living in urban and rural areas in low- and middle-income countries bear out the association between urban residence and increased rates of childhood overweight and obesity. Dietary change in urban settings in low- and middle-income countries is largely characterized by an increased availability of cheap, energy-dense foods, facilitated by trade liberalization and the expansion of "fast food" outlets *(99)*. There is likely to be wide variation in the consumption of "fast foods" and processed snack foods across low- and middle-income countries, but there are some commonalities. A significant proportion of children below the age of two years in low- and middle-income countries consume processed snack foods *(100)*. This is more common in Asia than in Africa, and with those living in urban areas and from wealthier families. Among school-going children, these foods may also be accessed at school vending machines, cafeterias, and in the areas surrounding schools at both formal and informal outlets. Children and adolescents in low- and middle-income countries are thought to find "Western" packaged commodities attractive and desirable, and those from affluent families may be more likely to access convenience foods given that they might be given daily allowances *(94)*. The example from Kuwait is illustrative of this (see Box C). There are few studies in low- and middle-income countries on schoolchildren's knowledge of the health-related effects of poor nutrition. Factors contributing to low levels of physical activity among children vary across low- and middle-income countries, but broadly include: high-density urban living with little recreational space; risk to safety which may restrict children's use of outdoor space; a lack of facilities in schools and neighbourhoods; increased availability of mechanized transport; increased levels of television viewing and internet use which displace other play activities; and increased emphasis on academic performance rather than participation in physical activities and sports. An association between short sleep duration and childhood obesity is demonstrated in high-income countries *(101)*, and this may well apply in low- and middle-income country settings, but has not been studied in these contexts. Similarly, there is no data on the impact of social media on childhood overweight and obesity in low- and middle-income countries.

Box C. Case study: the westernization of children's diets and obesity risk in Kuwait

Kuwait is a state where tradition and modernity sit alongside each other in the production of childhood overweight and obesity. Obesity is one outcome of the very rapid economic and social changes that have taken place since the discovery of oil in the 1930s and the high rates of economic growth since then. Rates of childhood overweight and obesity are among the highest in the world, and are primarily among the wealthy. The traditional role of food in social relations remains very strong, and is merged with plentiful availability of the highly energy-dense foods through which sociality is practiced. It is further facilitated by the convenience of regular eating in restaurants and the consuming of "fast foods". Maintaining adequate levels of physical activity is largely impossible in everyday life because, for much of the year, habitual activity is structured by the built environment which, because of the high temperatures, keeps people indoors and in motorcars (102). Unlike much of the Western world, in Kuwait, adolescent obesity is not associated with impaired health-related quality of life (103). This may reflect cultural differences in attitudes towards obesity in much of the Middle East, where large body size continues to be favoured as a measure of status (104).

In association with societal changes related to food systems, the relationship between socioeconomic status and obesity is changing in some countries, such as South Africa (see Box D).

Box D. Case study: the changing relationship of socioeconomic status and obesity in South Africa

The relationship between socioeconomic status and the prevalence of childhood overweight and obesity in South Africa is shifting to resemble more closely the inverse relationship seen in high-income countries. There is wide variation in the prevalence of childhood overweight and obesity across regions in South Africa, but an overall trend to an increase in rates of childhood overweight and obesity in the past two decades (105). This co-exists with persistently high rates of child stunting, and this dual burden is often found within the same low-income communities (106). This pattern may reflect the divergent effects of globalizing processes within food systems on the rich and the poor, as well as different cultural perceptions of optimal child growth and feeding. South Africa's inclusion into global markets following political change has facilitated increased foreign direct investment in the production and distribution of food products, the expansion of supermarket chains and "fast food" outlets, and the increased availability of packaged foods in the informal retail sector (107). While supermarkets provide staple and packaged foods at lower cost to the population, healthier foods are consistently more expensive than nutrient-poor alternatives (108). National survey data suggest that most South Africans consume a diet of low diversity and that price primarily determines food choices (105). As such, high-income groups benefit from access to a wide variety of healthy foods, while in low-income groups there is increased consumption of processed foods high in fat and salt as the most efficient cost-per-calorie options. Children access these in the home, at school, and from informal vendors in the neighbourhoods around school. Poor, energy-dense nutrition may be exacerbated by infant overfeeding by parents who value fatness as a sign of health (109).

Major challenges and gaps in knowledge

The mismatch hypothesis, which postulates that childhood obesity is an outcome of a pattern of intergenerational cycles of famine and subsequent energy abundance, is especially pertinent in low- and middle-income countries that have experienced rapid socioeconomic transitions in the recent past (*110*). This mismatch might be set up by conditions produced by war, where famine ends and food supplies resume at the end of conflict. The historical contexts that have created conditions for environmental mismatch are locally distinct, but common to most low- and middle-income countries is a recent absorption into an ever more globalized food system. Integration into the global economy is accompanied by increased foreign direct investment in production and distribution of processed foods, concentration of corporations that dominate food industries, and an intensification of food marketing practices in low- and middle-income countries *(99)*. The observed relationship between market-liberal policies and high obesity rates in high-income countries *(111)* appears to hold in low- and middle-income country settings. There is variation in low- and middle-income country policy response, but overall there are few low- and middle-income countries (Brazil, Chile, Colombia, Malaysia, Republic of Korea, South Africa and Thailand) with established policies on food labelling and the commercial promotion of food to children *(112)*. Feeding programmes in low- and middle-income countries may also inadvertently contribute to increased rates of childhood overweight and obesity if they have not been reformulated to account for the dual presence of undernutrition and overnutrition. Programmes need to provide supplementation in early infancy to promote catch-up growth and prevent stunting, while minimizing the inadvertent promotion of overnutrition in the general paediatric population *(110)*.

High quality evidence on the psychosocial and developmental aspects of overweight and obesity in low- and middle-income country settings is sparse, and it is likely that there is wide variation in the patterns of emergence of childhood overweight and obesity across these settings. Future research might address:

- the gendered differences in childhood obesity risk in different low- and middle-income country contexts;
- the health knowledge of both parents and children;
- the impact of peers, social networks and social media on diet and perceptions of body image;
- the formulation of appropriate nutrition programmes; and
- how and when the inversion between childhood overweight and obesity and socioeconomic position takes place and the temporal dynamics of this inversion relative to the nutrition and physical activity transitions occurring in low- and middle-income countries.

Summary

Differences in obesity prevalence and the nature of interventions that are appropriate and potentially effective in different settings, sociodemographic and cultural groups, need to be considered when developing obesity prevention and control strategies. In high-income countries, children in ethnic minority populations, including migrants and first-nation children, appear to be at increased risk of overweight and obesity. This may be, in part, due to the different environments and

lifestyles and, for migrants, different adaptations to a non-traditional environment and lifestyle. In high-income countries socioeconomic status is often, but not always, inversely associated with overweight and obesity prevalence among children, in an approximately linear manner, rather than applying only to children classified as deprived. The exceptions to this association are particularly in population subgroups with more recent economic transition. Patterns of obesity prevalence in low- and middle-income countries, where the largest numbers of children affected by obesity are found, tend to be opposite to those observed in low- and middle-income countries, i.e. highest rates are associated with higher socioeconomic status. Obesity also co-exists with a continuing prevalence of undernutrition in many low- and middle-income countries.

This section highlights some of the complex array of psychosocial, cultural and environmental context variables that influence the picture of obesity in various populations and subpopulations. Although the dynamic, macrosocietal processes leading to a global epidemic of childhood obesity are of the same general nature, various context specific factors influence the nature and intensity of their impact on obesity prevalence and trends within and across populations and over time. As outlined in other background papers, the critical set of components to be addressed can be identified along with some recommended approaches to interventions. However, the mix of relevant influences in any given situation argues against thinking in terms of “rule-of-thumb” or standard packages that can be applied even regionally or nationally without substantial tailoring and giving special attention to high-risk subgroups.

The unfolding of the epidemic of childhood obesity suggests that no society offers cultural protection at a level that can withstand the global drivers of the epidemic. However, tailoring of interventions should attempt to identify aspects of culture and lifestyles that can be leveraged as *assets* for obesity prevention as well as those that pose particular liabilities. Integration with other family- or child-relevant nutrition/health problems or interventions taking place or needed in these contexts will be important, particularly where there is a double burden of undernutrition and overnutrition and where the advice or traditions relating to interventions may be conflicting. Transitional situations in which sociocultural attitudes assign high social status and positive value to westernized eating patterns and sedentary lifestyles will pose particular challenges for the counteracting effects of obesogenic influences on parent/caregiver and child behaviours. These challenges will be compounded where larger body sizes are also socially acceptable, or even positively viewed (and where thin body size is shunned as a sign of poor health). The responses of boys and girls to the same environments may differ depending on the backdrop of gender differences in social roles, expectations, and privileges, as well as gendered aspects of parenting; these will also vary by the child’s developmental stage. Potential differences in responses may occur based on a family’s or community’s place on the socioeconomic spectrum (absolute or relative to the larger society) and related to the timing and pace of processes such as urbanization and economic development. As such, interventions need to be specifically tailored to address the specific issues in particular settings or groups.

4. ECONOMIC EVALUATION OF CHILDHOOD OBESITY[1]

4.1 A conceptual framework

Improvements in a person's health provide private and direct benefits, such as:

(1) contributing directly to well-being (a healthy person feels good);

(2) enabling greater productivity (a healthy person works better and for work longer hours than an unhealthy person);

(3) contributing to longevity (a healthy person can be expected to live longer than someone who is not healthy).

and indirect, more wide-reaching benefits, such as:

(4) Improvements in health benefitting others in the community (and more generally in the outside world), with people being able to engage more fully with someone who is healthy than with someone who is not.

Measuring the benefits of health

The benefits of (1) and (3) are direct contributions to an individual's well-being (feeling better; living longer), while (2) is an indirect contribution (larger income, greater social engagement) and (4) is a contribution to others' well-being. (1) and (3) and (4) are ends, while (2) is a means to those ends. International surveys, in which respondents are asked to assess their lives in terms of personal happiness and life satisfaction, have consistently revealed that among people everywhere, health appears as a major factor in personal well-being *(113–116)*.[2]

Over the years, economists have developed elaborate methods to estimate the benefits and costs of public investment in physical infrastructure, education, health, and the environment.[3] Some methods require asking respondents to report their willingness to pay for benefits (reported preference); other methods are designed to allow the investigator to infer the benefits by observing a person's behaviour, (revealed preference). The latter method involves a form of “reverse engineering”.

In the context of public investment on health, studies that rely on revealed preference, record expenditures on health to ascertain a person’s willingness to pay for them. Studies carried out calculate the benefits of (3) using figures to estimate the value of a statistical life (from a revealed preference for reducing the risk of death). Other studies estimate the benefits of (2) by calculating the value of output lost when workers are absent owing to illness *(117)*. The methods deployed for estimating benefits (2) and (3) are routinely used to measure (4) in the case of reducing

[1] Authors: Partha Dasgupta, Rachel Rodin and Frank Chaloupka.

[2] World Values Survey, European Values Survey, and Gallup World Poll are among the most prominent such undertakings. Scholars have studied them not only for what they tell us, but also for their consistency and reliability.

[3] “Costs” are to be thought of as negative benefits.

communicable diseases.[1]

Despite their use, however, these methods are inadequate, one reason being that people neither buy health nor vote on health. Goods and services that contribute to health are bought, and public programmes supplying those goods and services are voted on. Another reason is the lack of information on how the goods purchased and the activities pursued translate into aspects of life that are valued. Most people have inadequate knowledge of the goods, services and lifestyles that enable them to lead healthy lives. Studies have shown, for example, that in developing countries even middle-class households are unaware of the benefits of drinking filtered water *(118)*. However, any assessment of risks to health may be prone to bias. There is a tendency for people to imagine they are not part of a statistical distribution of people. That bias cuts across low-, middle- and high-income countries *(119, 120)*.

Obesity, in general, and childhood obesity, in particular, raise yet further issues. In countries with strong welfare provision, some of the costs of ill health are borne by the state. To rely on revealed preference in those countries would be to underestimate the desire for good health there. To imagine that medical services are like any other consumable good is to make a category error: the customer does *not* know best, nor do they necessarily act in their own interest, even if they know what is best for them. For these reasons, the methods developed by economists to ascertain the quantitative value of the earlier three types of benefits are usually supplemented by professional opinion of their value.

Although certain decisions, such as crying, smiling and crawling, are made by young children themselves, other decisions, such as those relating to play time, sleep time and food intake, are made on their behalf by adults. Greater autonomy is gained with age. Ending childhood obesity thus involves the participation both of the young and (perhaps especially) of adults.

Sustainable development and childhood obesity

Eradicating childhood obesity should form part of any national or international programme on sustainable development, with the WHO initiative being included in reports that supplement the United Nations Sustainable Development Goals.

“Sustainability analysis” involves evaluating the change in an economy across the passage of time. In contrast, "policy analysis" involves evaluating change in an economy owing to a change in policy (e.g. a change in tax policy or investment in a health programme).

[1] To date, there are only a few published studies on (1). An exception is Finklestein et al. (2013). The study made use of survey data on happiness.

The report of the Brundtland Commission *(121)* highlights that “sustainable development” is a pattern of development in which an economy's productive capacity (including its wealth *(122–124)*), relative to population size and structure, does not decline over time. “Wealth” is the social worth of a nation's stock of capital assets. The latter include manufactured capital (roads, buildings, machines and equipment), natural capital (sub-soil resources, ecosystems), and human capital (education, skills, and health). Thus, by sustainable development there is a requirement that this inclusive measure of wealth, relative to population, does not decline. In the same way that private firms prepare balance sheets, national governments should prepare wealth accounts. In the absence of wealth accounts, the idea of sustainable development will remain only an idea, lacking an empirical foundation.

Health as a capital asset

In order to compare the significance of an economy's various capital assets, the assets have to be expressed in a common currency, such as dollars – or any chosen commodity or consumable good. Health capital is health status expressed in common currency.

As set out earlier, "policy analysis" involves evaluating the change that occurs in an economy owing to a change in policy (e.g. investment in ending child obesity). To determine whether a policy, such as an investment project, is worth undertaking or should be rejected, it is necessary to estimate both its effect on wealth and its expected wealth increase. A change in wealth equals a project's present discounted value (PDV) of net social benefits (i.e. social profits) *(125)*.[1] Thus a unified method, that involves studying changes in wealth, can be used for both sustainability analysis and policy analysis *(126)*.

Quantitative estimates suggest that health – by a large margin – is the most significant component of a nation's wealth; health contributes substantially to the variations in wealth between nations. Global estimates of the value of a statistical life range from hundreds of thousands to several millions of US dollars. The value of a statistical life is an obvious first stage for valuing increases in life expectancy that accompany improvements in health. Even when adjusting the value of a statistical life for disabilities that come with age, the value of health remains two to three times greater than the accumulated value of all other forms of capital *(122)*.

A person's health status is an aspect of their human capital. As noted previously, human capital is an aggregate measure of health, education, and skills; the components of human capital, with the exception of output, cannot be traded.

[1] The PDV method is widely used for projects evaluated for their social profitability.

When economists estimate a person's wealth, the inclusion of human capital is restricted to education (e.g. years of schooling, converted into future earnings). This is a lacuna that should be corrected by macro-economists.

Social cost–benefit analysis of ending childhood obesity

An evaluation of the costs and benefits to society of a programme for ending childhood obesity, first requires an analysis of the incidence and consequences of childhood obesity, should such a programme be absent.

As with other programmes (or projects), a programme for ending childhood obesity involves a feasibility report containing: estimates of investment outlay; future costs distributed over several years (to cover for designing the programme, establishing clinics and publicity etc.); anticipated increases in programme expenditure and recurrent expenditure; and estimates of the flow of benefits.

Although forecasts of investment project costs may be prone to bias (due to costs frequently being greater than anticipated), the methods of estimating them are uncontroversial. However, the benefits of programmes, including those associated with ending childhood obesity, can pose novel problems.

An estimation of benefit and costs requires two classes of quantitative figures for: (i) the "factual" side of the programme's output, and (ii) the "value" of that output. Item (i) will consist of estimates of: (a) the proportion of children in each cohort that are found to be obese; (b) the proportion among those who will be targeted; and (c) the proportion among the targeted group who would not develop disease in their lifetime as a result of the programme. Item (ii) involves estimating the benefit accruing to those who avoid developing, for example, diabetes as a result of the programme.

Section 2 of this report discusses methods of estimating (ii); Annex 2 shows a model of how the figures pertaining to (i) can be estimated. Using, for example, a child aged "A", who is obese, epidemiological studies provide figures for the proportion of such children who will never succumb to diabetes nor other noncommunicable diseases linked to obesity. This in turn allows for an estimate of the proportion of obese children aged "A", who will be found to develop such disease at *some* future date. Although the age at which the latter group may develop disease is unknown, epidemiological studies contain information that enables an estimate of the proportion of obese children aged "A", who will develop disease at age "A+t". It is known that such individuals have a reduced life-expectancy. Epidemiological studies can be used to estimate the expected reduction in life expectancy of, for example, a diabetic, aged "A". Taken together with information on the annual social cost of treating a diabetic, (i) is thus complete.

The formal model, presented in Annex 2, uses the above information to estimate the PDV of social

costs saved and the expected life years gained under the programme for each cohort of children (A = 0,1,...). If the sum of PDVs for each cohort exceeds the investment costs, the programme should be accepted, but only after all potential programmatic improvements have been explored. If the PDV falls short of the investment costs, the programme should be rejected.

To calculate the PDV of a (public sector) programme, future costs and benefits – as expressed in the value of consumable goods – must equal current costs and benefits. The social value of a unit of future benefit or cost "t" years hence, relative to a unit of current benefit or cost, is known as the "social discount factor" for year "t". The percentage rate at which the social discount factor declines with "t" is known as the "social discount rate for year 't'". Most studies assume the social discount rate to be constant (3–5% per year), although recently some governments – for example the Treasury of the Government of the United Kingdom – have recommended a declining rate. Much has been written on the rates at which future benefits and costs should be discounted; but whatever rate (or rates) is used to discount a programme for ending childhood obesity, it should correspond to a government's discount rate (or rates) for all projects under its jurisdiction (aside from differences in risk categories).

4.2 A review of the literature on the economic implications of childhood obesity[1]

The literature on the economic consequences of childhood obesity is scarce compared with that on the economic burden of adult obesity *(127, 128)*. Four systematic reviews (*129–132*) cite fewer than 20 studies, mostly covering the impact on health-care expenditures. However, as the evidence on lifetime health costs of childhood obesity is developing – such as on the early onset of "adult" diseases, and tendency for childhood obesity to track into adulthood, as well as on the clustering of obesity within family and social networks – studies examining economic costs will follow.

Reviewed evidence suggests that early onset of obesity in childhood and adolescence has significant economic costs to individuals and society if a life-time perspective is chosen. Although short-term incremental economic costs of obesity in childhood may be relatively small, early onset of obesity and related chronic diseases impair individual life-time educational and labour market outcomes and place a significant long-term burden on health-care systems, employers, and society as a whole.

Studies on children and adults that break down costs by the level of BMI find a gradient, or higher costs for higher levels of BMI, for both health-care expenditures and for productivity losses *(133–136)*. This finding suggests benefits from delaying onset and reducing the magnitude of obesity, and conversely greater societal and individual costs for early onset of childhood obesity that progresses unchecked.

[1] Authors: Rodin Rachel Rodin with the WHO Collaborating Centre on Chronic Non Communicable Disease Policy (Olga Milliken, Vivian Ellis) on behalf of the Ad hoc Working Group on Science and Evidence for Ending Childhood Obesity.

Studies on the economic costs of childhood obesity are largely limited to the impact on health-care expenditures, mostly during childhood and mostly from the USA *(128, 130–132, 137)*. While offering a limited perspective on economic burden, these nonetheless identify significant costs that may likewise be considered a lower bound. However, given differences in children's BMI trajectories, as well as in social, cultural and institutional environments, significant caution is needed when extrapolating these findings to costs in developing countries. Trasande and Chatterjee (2009), for example, using panel data of 19 613 children aged 6–19 years in the USA found that children who were obese or overweight during two years of the study had elevated health-care expenditures *(138)*. The authors concluded that the overall impact if extrapolated to the entire US population would be $14.1 billion in additional expenditures annually. From the lifetime perspective, Finkelstein et al. *(129)* estimated an incremental lifetime medical cost of obese 10-year-olds in the USA of $14 billion. For Mexico, Anaya and Melendez (2009) estimated the incremental lifetime health-care costs incurred by obese and overweight children aged between 5 and 11 years, and concluded that an extra $41 billion will be spent on those who will become obese, and an extra $35 billion on those who will become overweight compared with those who will remain normal weight (*139*). It is difficult to compare estimates across studies. Differences in costs accounted for, measurement of obesity and overweight and length of observation period, as well as differences in health-care systems across countries can partially explain differences in findings.

As life-time studies on the subject are scarce, it is necessary to draw from the literature on economic consequences of adult obesity to provide a low bound for the future cost of childhood obesity today. Unobjectionably, the economic cost of adult obesity is substantial *(140–143)* and may be higher than the economic cost of heavy drinking (Sturm 2002, cited in *(144)*). Most studies on the economic consequences of obesity use the "cost-of-illness" approach (originated by Rice in 1967 *(145)* revised *(146, 147)*) and examine the impact on national health-care expenditures (direct costs) and on economic productivity (indirect costs or productivity losses due to morbidity and premature mortality). For example, production losses and extra health-care expenditures due to adult obesity are estimated to amount to a fraction of a percentage point of Gross Domestic Product in most countries, over 1% for the USA and over 4% in China *(140, 141)*. Recent systematic reviews have found that between 2–5% of total annual health-care expenditures are attributable to overweight and obesity in European countries *(128)*. Similarly, the report of the Organisation for Economic Co-operation and Development (OECD) *(141)* cites 1–3% in most OECD countries. Zhao et al. 2008 cite 3.7% for China in 2003 *(148)*. The exception is the USA, where estimates are higher, cited as ranging from 5–10% and even 20% of health-care expenditures *(128, 141)*. Indirect costs of adult obesity are measured through a range of methodologies which make comparisons difficult, even within a country. However, all show that the productivity costs of obesity are comparable or higher than related health-care expenditures (*127*).

Potentially significant components of economic costs of childhood obesity, such as the impact on unpaid caregivers (parents), future labour market outcomes, and the intangible value of health lost

to childhood obesity, have received only cursory attention[1] in the economic burden literature *(134, 149)*. Although some studies point towards the pathway from obesity to lower educational/skill attainment *(149–151)* and, in particular, that due to poorer mental health *(152)*, more research establishing causal relationships and associated costs are required in this area.

Some studies look at reductions in health-related quality of life as a measure of the full health cost of obesity to an individual. Most did not examine a value to society or an individual of the reduction in non-market social participation and enjoyment of life, which are often referred to as intangible costs *(149, 153–156).* Some studies used a range of values between $20 000 and $200 000 per unit of QALY to present a dollar value of life and quality of life lost due to obesity *(156–158)*.

Importantly, a life-time model which would build pathways from childhood obesity to adult health and associated economic costs is missing from the current literature. For example, from an individual and society perspective this model could establish causal links from obesity and associated physical and mental health to early childhood development and educational attainment with economic consequences for the individual employment and career choices. There are a few current studies that model a life-time (but, limited) impact of obesity using cross-sectional data *(129, 139)*. Longitudinal data are required to gain a better understanding of BMI trajectories, and health risks, as well as associated health-care and other costs over an individual lifetime.

5. RISK PATHWAYS AND POTENTIAL FOCAL AREAS FOR INTERVENTIONS

5.1 An overview of potentially modifiable risk factors

Childhood obesity is driven by interactions between biological, behavioural and contextual factors. For simplicity, and to draw upon the available evidence, the working group used this classification of determinants, emphasizing, however, that the distinction between them is not absolute and within each there are multiple factors.

Biological risk factors include maternal malnutrition (unbalanced nutrition, including both undernutrition and overnutrition, obesity, stress before and during pregnancy, and conditions such as maternal hyperglycaemia. These prenatal influences are exacerbated by a range of postnatal behaviours, including inadequate periods of exclusive breastfeeding and infant feeding behaviours, including inappropriate complementary foods and caregiver feeding style. As indicated above, these set the responses of the child, adolescent and adult to aspects of the obesogenic environment, including inappropriate amounts of dietary sugar, fat and salt, lack of physical activity and excessive sedentary behaviour.

[1] Two German studies (Kesztyus et al. 2014 and Breitfelder et al. 2011) examined the impact on caregivers (parents) in terms of work absences and potential productivity losses. Neither study found statistically significant differences across BMI groups.

Behavioural risk factors overlap with these biological factors and include movement behaviours, which incorporate physical activity, sedentary behaviour and sleep and which are established in early childhood. Reduced opportunities for sport, increasingly mechanized transport and increased screen-based entertainment (which typically promotes sitting and may disrupt healthy sleep habits) reduce physical activity in children. Behavioural risk factors related to diet include those that tend to contribute to calorie overconsumption, such as consumption of sugary beverages, snacking on highly processed, energy-dense foods outside meal times, and consuming large portions. Concerns over safety and overcrowding in some urban communities, increase stress in both parents and children. Related behaviours therefore include a range of psychosocial factors, such as stress, parenting behaviours, the influence of peers and siblings.

Contextual and wider societal factors include: socioeconomic considerations; changes in employment patterns; nutritional literacy within families; availability and affordability of healthy foods; wider availability of energy-dense foods; increased use of processed foods as part of lifestyle changes; eating patterns within families; reduced opportunity for physical activity through healthy play and recreation in an increasingly urbanized and digital world; automobile dependency; increased opportunities for sedentary behaviour; and possible interruption of sleep. The built and social environment in which children live is increasingly obesogenic. The influence and changing nature of marketing to children – as recognized in the World Health Assembly resolution on the marketing of food and non-alcoholic beverages to children (WHA63.14) – is also a contributor to the wider environment.

5.2 The development of taste and flavour preferences during early childhood[1]

Because the flavour senses are the major determinants of whether young children will accept a food (i.e. they eat what they like), they take on even greater significance in addressing this issue since many chronic illnesses that afflict modern society, including obesity, derive, in large part, from poor food choices, dictated by flavour preferences. Food habits begin to be shaped by experiences very early in life and are more difficult to change in later life.

Against recommendations of health authorities worldwide, people eat too much sugar and salt, and too few fruits and vegetables, even and especially among children. It is important to consider why modern patterns of food choice are antithetical to health, and why it is so difficult to develop good food habits and to change bad habits. This section summarizes evidence that helps answer these questions and explains why and how the basic biology of children can predispose them to consume excess calories when exposed to obesity-promoting environments. Inborn, evolutionarily-driven taste preferences make children vulnerable to the current food supply rich in added sugars and salt

[1] Author: Julie A. Mennella, PhD, Monell Chemical Senses Center, Philadelphia, PA, USA, on behalf of the Ad hoc Working Group on Science and Evidence for Ending Childhood Obesity.

and cause them initially to reject the healthful flavours of many vegetables, some of which taste bitter (e.g. broccoli). The section also summarizes evidence on how children learn to like "healthy" foods and the detrimental consequences of their not being exposed to the flavours of such foods as early as in utero and while being breastfed – and thus why policies that enable children and their families to be exposed to the foods and flavours of healthy diets are so critical.

The focus is on early life, in contrast with other reviews that focus on attempts to modify food habits of older children and adults, thus missing sensitive periods that may modulate long-term food habits, appetite, and growth. The information gleaned from these key findings is that any attempt to tackle childhood obesity must accommodate the unique vulnerability of children to the modern food system as a critical first step, and that policies that provide pregnant and lactating mothers the foundation for healthy food habits for themselves, for their children and for other members of their families will, therefore, have an equally critical role.

Evidence base

As noted earlier, two factors conspire to increase the likelihood that children will consume obesogenic diets: (a) inborn, evolutionarily-driven taste preferences that make them vulnerable to the unhealthy aspects of the current food supply; and (b) the detrimental consequences of not being exposed to flavours of healthful foods when young *(159)*. More specifically:

1. Evolution has shaped the taste of foods initially preferred or rejected by children – humans most likely evolved in an environment where the primary challenge was to obtain sufficient nutrients to survive and reproduce, while avoiding the abundant poisons found in many plants. In response to this challenge, sensory systems evolved to detect and prefer the once rare energy-rich (carbohydrate-rich) and sodium-rich foods that taste sweet or salty while rejecting the potentially toxic ones that taste bitter.
2. Children have a heightened preference for sweet and salty tastes during childhood and adolescence, compared with adults. This may have ensured the acceptance of nature's first food – mother's milk – and subsequently other energy-rich, sweet-tasting foods such as fruits, which are also abundant in vitamins, and ensured an adequate intake of sodium needed during periods of maximal growth.

Today many children live in environments abundant in highly processed and palatable foods, rich in added sugars, non-nutritive sweeteners, and salt. There is a mismatch between their biology and the environment in which they currently live; thus, children's natural taste predispositions draw them to processed foods that taste sweet and salty making them especially vulnerable to poor food choices.

The sense of taste is not the only component of flavour; the perceptions arising from taste and smell, in particular, are often confused and misappropriated *(160)*. Flavour is a product of several sensory systems and results from the perceptual integration of signals from taste (e.g. sweet, salt, bitter, sour, umami), olfaction (aromatic volatiles of, e.g. strawberries, garlic, and asparagus, perceived retronasally), and the trigeminal system (e.g. coolness of mint, burning of chilli peppers). These

anatomically independent senses are generally well developed at birth and continue to change throughout childhood and adolescence, controlling decisions that are among the most important they make: whether to eat or drink something or to reject it *(159)*.

The biology of taste

The ontogeny of taste has been the focus of scientific investigations for more than a century (see references *161* and *162* for review). This knowledge base, which addresses a range of behavioural outcomes, indicates that humans are born with well-developed taste and olfactory systems that, by the last trimester of gestation, can convey information to the central nervous system and organize a variety of behaviours. (Little work has focused on the trigeminal component of flavour.) Because these senses continue to mature throughout childhood and adolescence, children live in different sensory worlds than adults, with heightened preferences for sweet and salt, and increased sensitivity and rejection for bitter, which sweet and salt can partially mask or block (*163, 164*). The following focuses on the evidence base, specifically for sweet, salty and bitter tastes, in view of the preponderance of evidence, and because many of the processed foods that children are overeating are rich in added sugars, non-nutritive sweeteners, and salt. (For more detailed reviews, see references *161* and *162.*)

Sweet taste

Children do not have to learn to like sweet tastes, they are born attracted to the predominant taste quality of mother's milk. At birth, infants can differentiate varying degrees of sweetness and will consume a greater volume of a solution that tastes sweeter *(165)*. Their faces relax when tasting something sweet, and this relaxation is often accompanied by smiling *(166, 167)*, sucking *(168)* and hand-to-mouth movements *(169)*. Tasting something sweet leads to the activation of pleasure-generating brain circuitry *(170)* and can be an analgesic, blunting pain *(171)*; this circuitry is the same as, or overlaps with, that which mediates the addictive nature of such drugs as alcohol and opiates.

Recently, two Cochrane Database Systematic Reviews examined all the reputable studies that had been carried out in this international field of science, and concluded that sucrose was an effective analgesic for infants and can reduce pain from such procedures as circumcision and heel prick *(172, 173)*. This further highlights how sweet taste is not only preferred but blunts expressions of pain. Because the non-nutritive sweetener, aspartame, mimicked the calming effects of sucrose *(174, 175)*, it was concluded that afferent signals from the mouth, rather than metabolic consequences of ingesting sugars, are responsible for the analgesic properties of sweet tastes.

Evidence of the proclivity that infants have for sweet tastes also comes from (i) cross-cultural studies, and (ii) leading chronic early childhood diseases that result from overconsumption of sweet tasting liquids. In some cultures, there is (or was) a fear of feeding colostrum *(176)* resulting in other liquids or foods being fed to infants during the early postpartum period. There is a commonality in these pre-lacteal feeds in that many taste sweet (e.g. sweetened teas, sugar water, pre-masticated bananas) *(177)*. Feeding sugar water in hospital nurseries decades ago was perhaps a vestige of this

ancient practice: sugar water not only is readily accepted by babies, but it also results in a quiet nursery, due to its ability to inhibit crying and pain *(178)*. Of relevance, one of the most common diseases of childhood – baby bottle tooth decay *(179, 180)* – is due to chronic feeding of sugars, especially putting a child to bed with a bottle of sweetened juice or milk *(181–183)* or frequent use of sugar-sweetened medicines and beverages *(183, 184)*. To end such practices, it is necessary first to understand the caregivers' perceptions and beliefs regarding feeding sweetened drinks to babies, and how such practices reflect the norms of their culture. Caregivers feed their children what they themselves are eating and drinking and children learn what foods are appropriate not only through their own experiences, but also by watching others.

The heightened preference for sweet taste is universally evident among children (see *(178)* for review). Compared with average adult sweet preferences, children, on average, prefer a more intense sweet sensation, with the adult pattern emerging only during mid-adolescence and coinciding with the cessation of physical growth *(185, 186)*. In a study of 930 participants living in the USA, children selected as most preferred, a 0.54 molar sucrose concentration *(187)* – equivalent to ~11 teaspoonfuls of sugar in 230 ml of water (a typical cola has a 0.34 molar sugar concentration, the level typically preferred by adults). The intensity of children's most-preferred sweetness, as measured in the laboratory, has real-world significance because it relates to their preferred levels of sugar in beverages, and food such as breakfast cereals.

Experimental evidence suggests that some children may be more vulnerable to sweets than others, especially those with a family history of alcoholism *(188)* and those of certain ethnic or cultural backgrounds *(187)*. For example, black children and adolescents living in Philadelphia, USA, preferred a more concentrated sweet taste sensation than did white children, and this difference persisted even when statistical adjustments were made for gender and socioeconomic variables such as family income and parental education *(187).* Research suggests several explanations: first, some children may exhibit faster short-term taste adaptation after exposure to sweets (i.e. they "get used to" the taste), leading to preferences for more concentrated sweetness over time. Secondly, known ethnic variation in the allele frequency of polymorphisms in the sweet receptor gene may contribute to these differences *(189)*. Thirdly, the practice of feeding sugar water during infancy (prevalent, e.g. among urban black mothers in the USA) has been associated with heightened sweet preferences during later childhood *(190)*. Children learn from an early age what should (in the sense of expectation), and should not, taste sweet. Further, the higher consumption of sugar-sweetened beverages among African–Americans *(191)* may be due to a combination of biology, early exposure, and marketing practices that disproportionately target advertisements for sweetened beverages to black American audiences and communities, and especially to youth *(192)*.

Sweet tastes continue to blunt expressions of pain beyond infancy. Two research laboratories, one in Canada *(193)* and the other in the USA *(190, 194)*, determined how sugar affects children's tolerance to painful stimuli using the cold stimulus test – a classical experimental model for pain induction in both children and adults that measures time to reported pain onset in a hand submerged in cold water.

Tasting concentrated sugar water, but not water alone, delayed children's reporting of pain onset. The more children liked the particular intensity of sweetness, the better it worked for increasing pain tolerance. However, sucrose was not an effective analgesic for children who exhibited depressive symptoms or were overweight or obese *(188, 194)*, despite the finding that they preferred high levels of sucrose as well as sweet-tasting foods and candies *(194)*. Taken together, these data suggest that, due to their heightened positive hedonics of sweet taste, children, as a group, are more vulnerable to a food marketing environment that promotes foods rich in added sugars. Children of certain ethnic or sociocultural groups, or those with a family history of alcoholism, who are overweight or obese, or who are depressed, may be at particular risk of excess intake of sweets or candies. They may be eating more of these to feel the pleasure of sweet tastes.

While children are born liking sweet taste, experimental studies in children reveal that the sensation of sweetness is also context-dependent and acquires meaning through associative learning *(195, 196)*. Children whose mothers reported routinely adding sugar to their foods were significantly more likely to prefer apple juices with added sugar and cereals with higher sugar content than were similar-age children whose mothers reported never adding sugar to foods at home *(197)*. Through familiarization, children develop a sense of what should, or should not, taste sweet *(198)*. Our food supply now includes non-nutritive sweeteners that provide sweetness with fewer calories *(199)*. Despite their widespread use, little is known of the impact of non-nutritive sweeteners on children's acceptance, growth, and eating patterns *(200)* and obesity *(201)*. For example, as noted above, non-nutritive sweeteners have the same analgesic effect in children as do sugars *(174, 175)*, perhaps because they trigger the same reward centres of the brain *(202)*. An environment replete with sweetened beverages and foods, which is evident even in foods geared for infants and toddlers *(203)*, teaches multiple contexts in which sweet taste should be experienced. Thus, caregiver practices should be addressed by policies aiming to reduce early exposures to processed sweets. Policies and practices regarding sweetness in food should take into account that, although non-nutritive sweeteners reduce calories in sweetened foods, they nonetheless teach children that those particular foods should taste sweet.

Salty taste

As with sweet taste, age-related changes occur for salty taste *(159)*. While the ability to detect salt does not emerge until infancy *(204)*, children prefer more concentrated salt solutions and saltier foods with the adult pattern emerging during adolescence *(186, 205)*. Preferences for salt and sweet tastes are not only elevated during childhood but are also related to each other *(186)*, as well as to how much the child is growing: children who are growing, as evidenced by higher levels of a biomarker for bone growth, prefer sweeter and saltier solutions *(185, 186)*. There is evidence that the amount of salt an infant consumes, not only affects later salt taste preferences *(206, 207)*, but also blood pressure 15 years later *(208)*.

From a culinary perspective, salt has many desirable properties; it improves the sensory attributes of many foods that humans consume, and it is now inexpensive and abundant in many parts of the world *(209)*. Although no studies to date have been conducted in children, research in adults

suggests that decreasing, as well as increasing, sodium intake shifts preferences for salt accordingly. After consuming a diet with a 30–50% overall reduction in sodium content for 2 to 3 months, adult volunteers gradually developed a preference for foods with lower salt levels *(210)*. Not only do they acclimate to a lower salt diet, but they may also perceive foods containing the previous amount of salt as too salty *(211, 212)*. On the other hand, when adults were placed on a higher-salt diet, they shifted preference upward to like more salt in their foods *(213)*. Based on a recent review of this evidence base *(209)*, decreasing the desire for salt should be studied in children, for whom it might be particularly effective and have long-term benefits *(209)*.

Bitter taste

Of the five basic tastes (sweet, salt, bitter, sour, umami), bitter taste is the most diverse, both psychophysically (e.g. sensitivity to a particular trigger) and genetically (e.g. numbers of receptors, range of receptor sensitivity, and numbers of genetic variations) *(214)*. Whereas the family of sweet receptors is small *(215)*, with only three known genes, to date approximately 25 different bitter receptors have been identified *(216)*. Bitter taste is thought to have evolved as a deterrent to ingesting toxic substances, and it protected the plant producing the bitter toxins from being ingested *(217)*.

The ontogeny of bitter taste perception has been less studied than that of sweet perception, but it is accepted that infants will gape, wrinkle their noses, shake their heads, flail their arms, and frown when a bitter-tasting solution is placed in the oral cavity *(167, 218)*. Beginning at around two weeks of age, an infant will consume less and suck less while tasting a bitter-tasting (urea) solution *(219)*. There is some evidence that children may be more sensitive to some bitter tastes than are adults. Using identical psychophysical methodologies for adults, adolescents, and children, children with bitter-sensitive genotypes were found to be more sensitive to a bitter taste than were adults with the same genotype *(220)*, with the changeover occurring during adolescence *(221)*. Thus, for some children, childhood may be a time of heightened bitter sensitivity.

During childhood, the rejection of bitter is evident in children's rejection of medicines and bitter-tasting foods such as some green vegetables *(222)*. Liquid formulations aimed at children often include sweeteners and salts which can block or mask some bitter tastes for both children and adults *(163, 164)*. While children initially reject certain foods with bitter components (e.g. dark green vegetables) *(223)*, they can learn to like these foods (although it will often be easier to introduce fruits than green vegetables). As will be discussed, infants can learn to like vegetables, but encouraging older children to eat (or even try) these foods becomes more difficult the older they are when first introduced.

It is interesting to note, from a culinary perspective, that the two tastes that are most preferred (salt and sweet) can block or mask some bitter tastes for children *(163, 164)*. A little salt or sweet taste may go a long way in making healthy foods, such as vegetables, more palatable, thus decreasing a child's perception of the bitterness and increasing their liking of certain vegetables.

Early learning about food and flavours

Although humans generally have inborn, positive responses to sugar, and negative responses to bitter, "our biology is not our destiny", as is witnessed by the diverse world cuisines. Early experiences with nutritious foods, even those that taste bitter, and with flavour variety, can maximize the likelihood that, as children grow, they will prefer the flavours of a healthy diet because they have learned to like the flavours, textures, and variety of foods such a diet contains. Cross-cultural studies, for example, suggest that dietary history affects preferences for bitter-tasting foods without changing sensitivity to the taste *(224)*. Humans learn to like the foods with which they are familiar, and to which they have been exposed from an early age.

Early-life exposures – both biological and social – correlate with trajectories of health in adulthood decades later *(225)*. Many health initiatives address childhood obesity in part by encouraging good nutrition early in life. While much research has focused on the effects of the nutrient quality of the diet or on the long-term effects of early growth, relatively little attention has been paid to another important feature of nutrition: how humans learn to like the flavours of foods. (Note that the use of "flavour" here refers to food volatiles that are perceived retronasally – i.e. travel from the back of the mouth to the back of the nasal cavity, such as vanilla, garlic, and many others.)

First flavour experiences

Building on an evidence base in animals, research has systematically studied the transfer of volatiles from the maternal diet to amniotic fluid – the first flavours infants experience – and to mother's milk (see *(226)* for review). There can be confidence in these findings because of the rigour of the methods used and the consistency of findings among mammals. Research, over two decades, has shown that a wide variety of flavours from substances either ingested (e.g. fruits, vegetables, spices) or inhaled (e.g. tobacco or perfumes) by the mother are transmitted to her amniotic fluid and breast milk. Variations in flavour from mother to mother and from feeding to feeding suggest that breastfeeding, unlike formula feeding, provides the infant with a potentially rich source of sensory variety, if the mother eats a varied diet. The types and intensities of flavours experienced may be unique for each infant and characteristic of the culinary traditions of the family.

These fluids share flavour profiles with foods eaten by the mother, which suggests that breast milk forms a "bridge" between flavours experienced in utero and those experienced from solid foods and which helps infants to make the important transition from an all-milk diet to one containing solid foods (see *(227)* for review) and leading to greater acceptance of those foods *(228)*. This pattern makes evolutionary sense because the foods that a mother eats when she is pregnant and nursing are likely to be the foods to which the child will have the earliest exposure, and thus should prefer, since mothers typically feed children foods that are part of their own diet and culture. Thus, mothers eating diets rich in healthy foods can help their children develop healthy food preferences.

Infant formulas

Sensory experiences of food flavours in breast milk in children whose mothers eat a varied diet may explain why children who were breastfed tend to be less picky *(229)* and, as they learn the flavours of the foods they are offered, more willing to try new foods during childhood *(230–232)*. However, many infants worldwide receive infant formula, either exclusively or as a supplement to breast milk, and this percentage increases steadily as the infant ages.

From the perspective of flavour, breast and formula feeding are striking contrasts on many levels *(233)*. As with the infant's previous amniotic environment, human milk contains transmitted flavours from the diet of the mother. In contrast, formula-fed infants are usually exposed to a constant and monotonous flavour profile during formula feeding that does not contain salient sensory information about the maternal diet. Infants fed formula learn to prefer its unique flavour profile and initially may have more difficulty accepting the flavours of fruits and vegetables not experienced in formula. Moreover, during formula feeding there is a dissociation between what the mother is eating and the flavour of the formula milk she and other caregivers feed to her infant.

Although diet, in early infancy impacts more than flavour preferences throughout the first year of life, an increasing body of literature demonstrates that it affects later life health outcomes as well. Breast milk is by far the preferred source of nutrition during infancy, with infant formula considered the next most suitable feeding alternative. However, not all formulas are alike in flavour or dynamics of feeding (e.g. satiation or growth), and such differences may be related to compositional differences among the formulas. Numerous studies have shown that formula-fed infants (who mostly are fed cow's milk formula) tend to weigh more by the end of the first year of life and have a greater risk of obesity than do breastfed infants (see *(233)* for review). However, a recent study found that although infants, randomized to cow's milk formula during infancy, consumed more formula to satiation and, based on the WHO standards, their growth trajectories were accelerated, the weight gain for those randomized to extensively hydrolysed protein formula was normative *(234)*. Not only can formula-fed infants signal satiation, but they are sensitive to some components of the formula in determining how much they consume during a formula meal *(234, 235)* (see *(233, 236)* for review). Therefore, when evaluating the effect of early diet composition on growth and health outcomes, it may not be appropriate to group all formula-fed infants together with respect to certain health outcomes such as obesity *(236)*.

Complementary foods: flavour learning

How to introduce vegetables and fruits to the diets of infants is surrounded by much medical and cultural lore, but little research; this is summarized below. The research indicates that, regardless of whether breastfed, formula-fed, or both, infants can learn to accept healthy foods during complementary feeding. Infants learn through (a) repeated exposure (8–10 exposures) to a particular food and (b) exposure to foods that vary in both flavour and texture. This, in turn, promotes willingness to eat, not only introduced foods, but also other, novel foods *(236, 237)*. Exposing infants to multiple sensory contrasts (between- and within-meal flavour variety) also provides more opportunities to develop conditioned flavour preferences based on the post-ingestive

reinforcing effects of nutritious foods *(238, 239)*. Looking at the food is not sufficient – the child has to taste the food to learn to like its flavour *(240)*.

Although the facial reactivity of infants during feeding is related to intake, it is governed by different neural substrates; thus, actual intake can increase sooner than facial expressions change following repeated exposures to a new food *(237, 241)*. This means that infants may continue to display facial expressions of distaste (e.g. grimace) even though they are increasing their intake of a food with repeated exposure. Therefore, caregivers who focus on the infant's willingness to eat the food, not the facial expressions made during feeding, will be more successful in enabling their children to like a range of healthy foods *(237)*. As the child grows, providing repeated experience with small tastes of the target food (e.g. vegetables) either alone, or with a favoured accompaniment, can have a lasting impact on liking and intake, even in older-aged children *(242)*.

Because consumption of vegetables (and some fruits) is so low in many families, many children are deprived of the sensory experiences (and parental and peer modelling) needed to learn to like these foods. In animal models, early taste deprivation remodels the central nervous system *(243)*. Fruits are typically more accepted than vegetables by children, and while some progress has been made in recent years to increase total fruit intake, little progress has been made for vegetables *(244)*. While vegetables, in particular, are most cited by children as being a food they do not like *(223)* – and some children may be more sensitive to bitter tastes than others *(220, 245)* – children can learn to like these foods through repeated exposure and variety when they are young and then through parental and social modelling. This becomes a more difficult task during later childhood when children often refuse to eat (or even try) these foods to learn to like them.

Evidence from longitudinal studies suggests that food habits established during infancy track into later childhood and adolescence (see *159, 232, 246*). The strongest predictors of what foods young children eat are (a) whether they like the taste, (b) how long they were breastfed and whether their mothers ate these foods when pregnant or breastfeeding, and (c) whether they have been eating these foods from an early age *(230, 232, 247–249)*. For many children, by the time they reach 2 years of age, they have completed the transition to "table foods" and are consuming diets similar to those of other family members. As children make this transition, early experiences with nutritious foods and flavour variety may maximize the likelihood that they will choose a healthier diet because they like the tastes and variety of the foods it contains. These foods need to be part of the family's diet so that once the preference develops, with continued exposure, the preference for these foods can be maintained, and the child can thus learn to like more complicated flavours and textures. Ultimately, the goal is gradually to accustom children to a varied diet that meets nutritional needs for growth and development and provides them with opportunities to learn to like and prefer a variety of healthy foods.

Feeding behaviours and satiation

While this review focused on how early taste and flavour experiences during milk and complementary feeding can provide children with the experiences to learn to like and accept healthy foods, it is acknowledged that feeding occurs in an environmental context (*250, 251*) and that some aspects of feeding, including satiety and satiation, are heritable (*252*).

Much of the interest in early feeding behaviours has focused on the two predominant modes of feeding: breastfeeding and bottle feeding. There is evidence that, as a group, bottle-fed infants consume a greater volume during each feeding and over the course of a day compared with breast-fed infants *(253)*. In turn, bottle-fed infants are more likely than breastfed infants to exhibit rapid rates of growth during the first year postpartum *(254–256)*, a known risk factor for childhood and adult obesity and other comorbidities *(257–261)*. One hypothesized mechanism underlying associations between bottle-feeding, overfeeding, and excessive weight gain is that mothers (or fathers) exert more control (and thus infants less control) during bottle-feeding than breastfeeding. This may be attributable, in part, to the abilities of bottle-feeding parents to feed in response to environmental cues (e.g. the amount of milk or formula in the bottle) rather than in response to infant hunger and satiation cues *(262–264)*.

Despite this widely held hypothesis, research, albeit limited, suggests that bottle-feeding does not uniformly place infants at risk of overfeeding and excess weight gain. Bottle-fed infants can communicate hunger and satiation, but some do so more clearly than others *(265)*. Likewise, some parents are more responsive to their infants' hunger and satiation cues than others *(266)*. Furthermore, emerging evidence reveals that, particularly for cow's milk formula and extensively hydrolysed formulas, the different free amino acid content may affect how much formula an infant consumes during a meal (i.e. satiation) (see *234–236* for review), which may have effects on growth trajectories during the first year of life *(267)*. In other words, for formula-fed infants, what is in the bottle may be just as important as how they are fed and who it is fed by.

In large-scale observation studies, formula-fed infants are often categorized into one large group; thus research is needed to examine how infant formulas of different composition affect growth, or why growth for some formula-fed infants, but not all, differs substantially from the "gold standard" of breastfeeding. Such knowledge is significant because many mothers worldwide feed their infants formula milk while in hospital, either exclusively or as a supplement to breast milk, and this percentage increases steadily as the infant grows. Since early diet can programme risks for later obesity and other chronic diseases *(268)*, it is imperative to determine the mechanisms underlying growth differences from the perspective of both the infant and mother during milk feedings, complementary feedings and then when they eat foods at the table. The evidence suggests that mothers vary in their control over their infants' feeding, their ability to read their infants' cues and their feeding practice styles (e.g. restrictive), all of which can impact feeding *(269, 270)*. Likewise, children vary, from an early age, in their feeding and appetite traits (e.g. size, duration, rate of feeding), temperament and their signalling of satiation *(250, 265)*. The development and validation

of methods to assess the behaviours of both members of the dyad is needed to understand the modifiable factors associated with overfeeding and rapid rates of growth *(234, 235, 249, 265)*.

Optimum timing of interventions

The importance of influencing the eating patterns of young parents or parents-to-be cannot be underestimated, since parents often feed their children what they themselves are eating (see *271–273*). One strategy is to enable caregivers, and mothers in particular, to eat (and learn to enjoy) more fruits and vegetables and other healthy foods. Children whose mothers were instructed to eat more fruits and vegetables tried more vegetables themselves *(274)*, and the more fruits and vegetables eaten by the mother, the more likely their child was to follow their example *(273)*.

Although the specific foods women crave may be a function of their culture or geographic location, cravings for foods such as fruits and vegetables often increase during the last trimester of pregnancy and during lactation *(275–277)*. Pregnancy and lactation are often times for dietary change; women often seek health advice and change their diets and habits *(278)* or have access to food, nutrition and information resources that were not available to them prior to pregnancy *(279)*. This transition to motherhood is associated with positive changes in food-choice behaviours *(280)* and other behaviours (e.g. smoking cessation *(281)*), thus leading many to suggest that this is an optimal time to build on this potential motivator to effect dietary change. Women may be motivated to change their own unhealthy food habits during pregnancy and lactation; this may be an opportunistic time to influence the food habits of women during the transition to motherhood as well as the next generation. Feeding good tasting, healthy foods to the mother will maximize the likelihood that these foods form part of the family diet. As previously noted, early experiences with nutritious foods and flavour variety should increase the chance that, as infants grow, they will enjoy a more healthy diet because they learn to like the tastes. The best predictor of what foods children eat is whether they like the taste *(282)*.

This also suggests that, for optimum results, the type of healthy foods fed to infants should be similar to those eaten by the mother and other family members. Certainly, first foods are often specially prepared (pureed or mashed and then gradually increased in texture), but, as noted earlier, for many children in the world, by the time they reach 2 years of age, they are consuming diets similar to those of other family members. There may be a disconnection between mother and infant with respect to the development of flavour preferences when the young child consumes only specially-prepared foods that are not in the family diet. Once the child stops being fed these specially-prepared foods, mothers will likely feed them what they themselves are eating.

Summary

The evidence base reviewed herein focuses on five important and interrelated issues, all of which have implications for the work of the Commission: (i) understanding the unique vulnerability of children to the modern food system; (ii) understanding how food habits have their beginnings during early life; (iii) the importance of encouraging healthy food variety in families and limiting children's

exposure to unhealthy foods; (iv) understanding how breastfeeding and formula feeding differ in flavour learning, initial acceptance of foods in the maternal diet, satiation and growth, and (v) understanding how the differing composition of currently-marketed infant formulas have differential effects on infant flavour learning, food acceptance, satiation and growth, and implications for later health and discordance in infant dietary experiences with their mothers (Table 4). Children (and their families) need "an environment that encourages healthy preference learning in early life" *(283)*.

First, the evidence shows that children are particularly vulnerable to overconsuming processed foods and sugar-sweetened beverages – important and independent causal factors in the childhood obesity epidemic. In many parts of the world, a mismatch exists between children's physiology and their current food environment; many live in an environment with easily accessible food: it is inexpensive, good tasting, and served in large portions. Further, the increased levels of sugar, fat, and salt in processed foods cater to children's natural taste predispositions. Attempts to limit consumption of sweet-tasting foods and beverages may be more difficult for some children because of the individual differences in the inherent hedonic value of sweet taste and how sweet foods make them feel. This struggle parents have in modifying their children's diets to reduce added sugars and salt appears to be, in part, a function of children's biologically-based preference for these tastes when they are initially and repeatedly exposed to them. However, the frequency to which the child is exposed to such foods and their salience within a given cultural framework are also driven by multiple aspects of food marketing and their interplay with extant cultural beliefs, preferences and norms. This emphasizes the need for strategies to alter the current patterns of exposure to sweet and salty foods, particularly for children, all of whom are vulnerable – some more than others – and the need to target children's proclivity for sweets or candies to healthy, good tasting fruits.

Secondly, the evidence shows that food habits have their beginnings during early life. While it is not easy to change the basic ingrained biology of children for liking sweet taste (e.g. sweets or candies) and avoiding bitterness (e.g. broccoli), an individual's earliest flavour experience – in the womb, via breast milk, and during early feedings – has long-term effects on food choice. Children can learn to like healthy foods, and the earlier they are exposed to them, the more successful the outcomes. A little sugar or salt can reduce the bitter tastes of some foods, such as vegetables, and this can increase the likelihood of children at least trying those foods. Research is needed to determine effective strategies, in terms of timing, content, and outcomes, to increase genuine liking for the tastes of healthy foods.

Thirdly, the feeding of infants and children cannot be separated from the feeding of their mothers or caregivers. Feeding involves more than providing calories: family and culture are defined, since food habits, integral to all cultures, have their beginning during early life. A key policy response, therefore, is to provide the foundation for healthy food habits for entire families, not solely children. Because pregnancy and lactation are often times when women seek health advice and change their diets and habits, the transition to motherhood can be associated with positive changes in food choice and other behaviours (e.g. smoking cessation). Research is needed to determine whether encouraging

these positive changes in mothers will increase their own liking of these foods and, in turn, expose their developing infants to healthy flavours in utero and in the mother's milk.

Fourthly, from the perspective of flavour, formula feeding is in marked contrast to breastfeeding. The range and types of flavours that infants experience and learn to prefer in formula are quite different from the wide variety of flavours infants experience and learn to prefer in breast milk. The effects of early experiences with flavours are persistent and long-lived and are specific to the flavour profile experienced, and this may be discordant with the family diet. However, formula-fed infants can learn to like new flavours once solids are introduced to their diet via different types of learning, including repeated exposure, familiarity and experience with variety. In terms of the dynamics of feeding, not all formulas are alike in terms of satiation and growth. Because breastfeeding imparts more control of the feeding situation for the infant than bottle feeding, understanding how the composition of formula impacts the signalling of satiation and how mothers can interpret those signals and not overfeed is key for the development of evidence-based approaches to responsive bottle feeding. In addition, understanding which formulas place infants at greater risks for faster rates of growth – and why, is needed to provide sound guidance and policy. Likewise, understanding the mother–infant dynamics of responsive bottle feeding, and which mothers and infants are at highest risk, is essential for preventing accelerated weight gain during infancy – a risk factor for obesity. Researchers should be encouraged to specify formula types when studying the effects of bottle feeding versus breastfeeding.

Table 4. Summary of evidence base and possible recommendations to promote healthy eating habits during pregnancy, lactation and early life

Developmental stage	Knowledge gleaned from evidence base	Policy goal for mothers or caregivers
Prenatal	1. Flavours of the foods mothers eat are transmitted to amniotic fluid and influence later flavour preferences.	**1. Provide and encourage a wide array of healthy foods during pregnancy.** Every fetus has a unique flavour experience depending on the mother's diet. Mothers should be encouraged to eat healthy foods (and avoid unhealthy foods such as those rich in added sugars) since her infant is being introduced to new foods and flavours.
Infancy/breastfeeding	2. Sensory experiences with food flavours begin in utero and continue during breastfeeding. Flavours of foods mothers eat transmit to breast milk and influence later flavour preferences.	**2. Encourage mothers to each a wide array of healthy foods during lactation.** Every breastfed infant has a unique flavour experience depending on the mother's diet. There is a continuity of flavour experiences in amniotic

	Children who were breastfed by mothers who eat a varied diet tend to be less picky and more willing to try new foods during childhood.	fluid, breast milk and then when mothers feed their infants the healthy foods they ate during pregnancy and lactation (see Steps 4–8).
Formula feeding	3. Children who are formula fed have flavour experiences that are independent of the mother and unlike that experienced in utero. They have more difficulty initially accepting flavours of fruits and vegetables and some children may be more sensitive to bitter tastes than others. Formula-fed infants can signal hunger and satiation.	**3. Be aware that infant formula provides a monotone flavour experience for infants.** While infants learn to prefer the flavours of their formula, they may have a more difficult time initially accepting fruits, vegetables and other foods (e.g. meats) since they have not experienced the same varied flavours as breastfed infants. However, formula-fed infants can learn to accept initially rejected foods (see Steps 5–8). Mothers to learn to read their infant's signals so as not to overfeed.
Complementary feeding	4. Breastfed infants are more accepting of new foods such as cereals when flavoured with mother's milk.	**4. Encourage mothers to prepare the infant's cereal with mothers' milk** to provide familiar and preferred flavours.
	5. Infants are more accepting of the flavours of the foods eaten by their mother during pregnancy and lactation upon initial exposure. In general infants have a proclivity for sweet and salt tastes and a rejection of bitter taste which reflects their biology.	**5. Encourage mothers to offer the infant the same healthy foods that were eaten by them during pregnancy and lactation** to provide a continuity of flavour experiences. Avoid feeding sugar water and teas. Learn to read signals given by the infant.

	6. Regardless if infants were formula fed, breastfed, or both, they learn to like both fruits and vegetables (and other healthy foods) by repeated exposure to those foods, by experience of a variety of such foods and then parental and social modelling. The acceptance by infants of a novel food increases after 8–10 exposures to that food (meaning tasting, not just seeing the food). Some children are more sensitive to bitter tastes than others.	**6. Encourage the mother to offer new foods multiple times.** The baby's initial rejection of a new food (especially vegetables and some fruits) may not be an indication of dislike. Fruits will generally be liked more than vegetables (especially green vegetables) but children learn to like the foods they are familiar with, and have been exposed to, from an early age.
	7. The facial reactions of infants during feeding are governed by different neural substrates than intake; actual intake can increase sooner than facial expressions after repeated exposure or exposure to variety.	**7. Encourage mothers to focus on the infant's willingness to eat the food, not the facial expressions made during feeding**. Infants need to taste healthy food to learn to like the taste; it may take longer to observe changes in facial expressions than in intake. Learn to read signals given by the infant so they can be provided with tasting experiences to learn to like healthy foods.
	8. Infants who were fed a variety of fruits were more accepting of novel fruit; those fed a variety of vegetables were more accepting of a novel vegetable and novel meat.	**8. Introduce a wide variety of healthy foods.** Opportunities to taste a variety of foods promote the infants' willingness to try new foods.
	9. The ability to detect and prefer sweet foods is evident early in life and largely reflects biology. Tasting something sweet gives children pleasure, calms them, and blunts expressions of pain. In addition, the sensation of sweetness and saltiness is context-dependent and can acquire meaning though associative learning. Early exposure to sweetened foods teaches children multiple contexts in which they expect to experience sweet tastes	**9. Limit exposure to added sugars, non-nutritive sweeteners and salt.** Children have a proclivity for sweet and salt tastes and are at risk of over-consuming these foods and beverages, not just because they taste good, but because it gives them pleasure. Early experiences of foods with added sugars, non-nutritive sweeteners and salt can programme children to like these unhealthy foods and to not like the unsweetened (or unsalted) versions.

	(often through added sugars, non-nutritive sweeteners).	Such excessive sugar and salt intake may have long-term effects on their health.
Toddlerhood/preschool years	10. Children are more likely to eat a new food if their parents are eating the same type of food rather than when parents are merely present, or are eating a different food.	**10. Be a good role model. Eat healthy foods.** The combination of repeated exposures to a variety of novel foods and social modelling by you and other family members eating of those foods can help promote children's acceptance and liking.
	11. Children as a group prefer sweeter and saltier tastes than do adults. Certain children may be more vulnerable than others to sugar-enriched foods: those of certain ethnicities or sociocultural backgrounds; those with a family history of alcoholism; those who are overweight or obese or who are depressed. Salts and sugars can sometimes block or mask bitter taste in children, who are more sensitive to some bitter tastes than adults.	**11. Limit intake of foods with added sugars and salts and encourage intake of healthy foods.** Be a good role model and limit your, and your children's, intake of foods high in added sugars, non-nutritive sweeteners and salt. As an alternative, offer your children good tasting fruits. Easy access to sugars, non-nutritive sweeteners, and salt in processed foods and beverages cater to their natural taste predispositions and makes them especially vulnerable to poor food choice and overeating. However, the addition of a little sugar or salt may help greatly in making healthy foods, such as vegetables, more palatable to your children.

Major challenges and gaps in the knowledge

A major challenge for health initiatives, aimed at decreasing obesity in both children and adults, is that neither adults nor children are achieving the recommendations; the unhealthy eating habits that afflict many adults are widespread among the youngest members of society. Because early experiences with food can set the stage for healthy eating habits, research on optimum feeding practices may contribute to improved health outcomes. The following lists gaps in knowledge that influence our ability to develop effective approaches for obesity prevention and improved, enjoyed diets, starting with infancy and early childhood:

1. Children need particular protection; their biology favours sugars and salts not just because of their pleasant tastes but because both sweet and salt tastes can block the aversive aspects of unpleasant (bitter) tastes and blunt expressions of pain. Achieving recommendations to reduce added sugars and salt in children's diets likely faces a biological challenge during periods of growth, emphasizing the need for research and evidenced-based strategies to improve the healthfulness of children's earliest food and food-related exposures and, ultimately, their diets.

2. While many cultures use sugar and salt in recipes to reduce the bitterness of healthy foods, such as vegetables, it is concerning that sugars or non-nutritive sweeteners are added to processed foods (with preferred flavours such as chocolate) at levels beyond those needed to encourage appropriate intakes. Manufacturers are tapping into the child's proclivity for sweet tasting foods and thus increasing their risks for overconsumption. By demonstrating the unique vulnerability of children to the modern food system, the development of evidence-based, corrective policies and strategies targeted to this developmental stage, is critical. It is also important to understand the biological substrate and source of individual or subgroup differences to reduce risks for addictions, obesity, and other intake-related morbidities.

3. Understanding the interplay between the biology of the child, the culturally-influenced beliefs and perceived norms of the caregiver, and the associated food marketing environment will provide a better understanding of ways to counteract the widespread use of sweets and candies.

4. Attempts to limit consumption of sweet foods and beverages may be more difficult for some children because of the individual differences in the inherent hedonic value of sweet taste and the accompanying feelings. Research on how to modify approaches to fit individual needs may improve efficacy.

5. Research suggests that there are optimum periods during early life when experience with flavours promotes greater liking and enjoyment. Further research establishing these optimum periods and interventions may inform strategies and recommendations to reduce added sugars and salt and increase healthy foods such as fruits, vegetables, meats, etc. in children's diets.

6. Taste and odours are not the only components of flavour. During childhood, children learn to appreciate the texture of the foods in the adult diet. Further research is needed to examine this neglected aspect of food preferences in children.

7. The foods of infants and children cannot be viewed separately from those of their caregivers. Mothers' preferences shape the early feeding environments, and infants and children learn what food is available, and what food the mother likes. Because many adults around the world have unhealthy eating habits, many children will be deprived of early experiences with healthy foods and the opportunity to learn to like them. Research is needed to better understand: (a) how to improve

dietary habits of women during pregnancy and postpartum; (b) how infants learn to like foods; (c) the processes of satiation and satiety during infancy and its impact/stability during development; and (d) how mothers recognize and interpret their infant's satiation and satiety cues. It is important that the diets of mothers and their children should not be dichotomized or defined as reflecting "bad" or "good" mothering but, rather, as a continuum of parenting.

8. Animal model studies suggest that epigenetically-programmed changes in brain reward systems, obesity, and unhealthy food habits, may occur in offspring because of the types of foods (e.g. high sugar, high fat "junk food" diets) that their mothers eat during pregnancy and lactation *(284)*. Research is needed to address these issues in humans.

9. Infancy may be an important time for parents to establish healthy beverage practices for their children. Research on feeding practices and beliefs can be used to inform strategies to reduce this source of added sugar (and salt) intake among children. It cannot be underestimated how sweets bring children pleasure and the reward that is felt by the adults in giving the child such joy. Therefore, a fuller understanding of why mothers and other family members feed sweetened foods and beverages and their interpretations of their infant's hedonic responses is key.

10. Due to increased usage of non-nutritive sweeteners, the food supply now provides sweet taste with fewer calories. Understanding how the growing use of low-calorie sweeteners in products for growing children affects their food preferences and behaviours and metabolism may indicate which interventions and practices promote healthier eating habits overall, rather than merely fewer calories per serving. Such understanding can also inform potential policies related to the use and labelling of non-nutritive sweeteners in food manufacturing.

11. The long-term consequences are not known of children learning to associate sweet taste with certain foods that typically are not sweet but have been processed to taste sweet. Although there are studies in adults, no research to date has examined whether preferences for sweet (or salt) tastes can be shifted downward in children. Consequently, there are no evidence-based strategies for "re-teaching" children to like a particular food in its natural, unsweetened or unsalted form. It is likely that during infancy and childhood, the salt and sweet environment – and any changes in it that result from lowering the overall levels in the food environment – will have profound effects.

12. Supporting breastfeeding initiation and duration are key. However, many infants are also fed infant formula, which places them at elevated risks for accelerated weight gain during infancy, a risk factor for obesity. However, formula-fed infants are not a homogeneous group in terms of flavour learning, growth or satiation. Thus, when evaluating the effect of early diet composition, it is not appropriate to group all formula-fed infants together in one group when comparing them with breastfed infants. Research is needed to determine the effect of infant diet composition among formulas and between formulas and breast milk on (a) energy intake; (b) energy expenditure; and (c) satiation, to better understand their interrelationships to early growth and obesity risk.

13. Since many infants are fed breast milk and formula, understanding the role of mixed feeding on flavour learning and growth is also an important area for research.

14. Understanding the maternal–infant dynamics of responsive feeding, and which mothers/parents/ caregivers and infants are at highest risk for behaviours that predispose to obesity, is essential for preventing accelerated weight gain during infancy, a risk factor for obesity. The focus of future research should be on both caregivers and children and their interaction to determine: (i) sources of individual differences in the impact of early feeding practices (breastfeeding, formula feeding, mixed feeding) and styles of the mothers (e.g. responsive feeding; reading infant signals); and (ii) individual differences in the infants' display of communicating satiation and satiety to the caregiver and whether that tracks throughout childhood.

5.3 The role of nutrition literacy in the prevention of childhood obesity[1]

Health literacy is defined as the ability of individuals to access and understand health information and services and to use this to inform health decisions; it has become a focus of attention because of its strong association with objectively measured health status *(285–287)*, including links to obesity *(288)*. Importantly, health literacy is considered a better predictor of health than age, income, employment, ethnicity, or education level *(289)*. As health literacy includes conventional literacy skills, and adult literacy skills are often too poor to understand health messages, there is concern that poor parental literacy skills may be an important contributor to child health disparities *(290)*, with implications for childhood obesity prevention strategies.

Surprisingly, despite the level of interest in the role of health literacy as a predictor of health, much of the scientific literature concerning it does not explicitly mention food and nutrition *(289)*. Nutrition information is complex and can require high levels of cognitive skills *(291)*. A definition of nutrition literacy that corresponds to Nutbeam's primary description of "functional health literacy" will be used therefore in analysing the effectiveness of intervention strategies *(286)*. Nutrition literacy is a relatively new construct that draws heavily on notions of health literacy. The definition of nutrition literacy is: "the degree to which individuals can obtain, process, and understand the basic nutrition information and services they need to make appropriate nutrition decisions" *(292)*.

Guttersrud et al. refer to the "functional" component of nutrition literacy as the proficiency in applying basic literacy skills, such as reading and understanding food labelling, and grasping the essence of nutrition information and guidance *(293)*. Interventions to improve nutrition literacy tend

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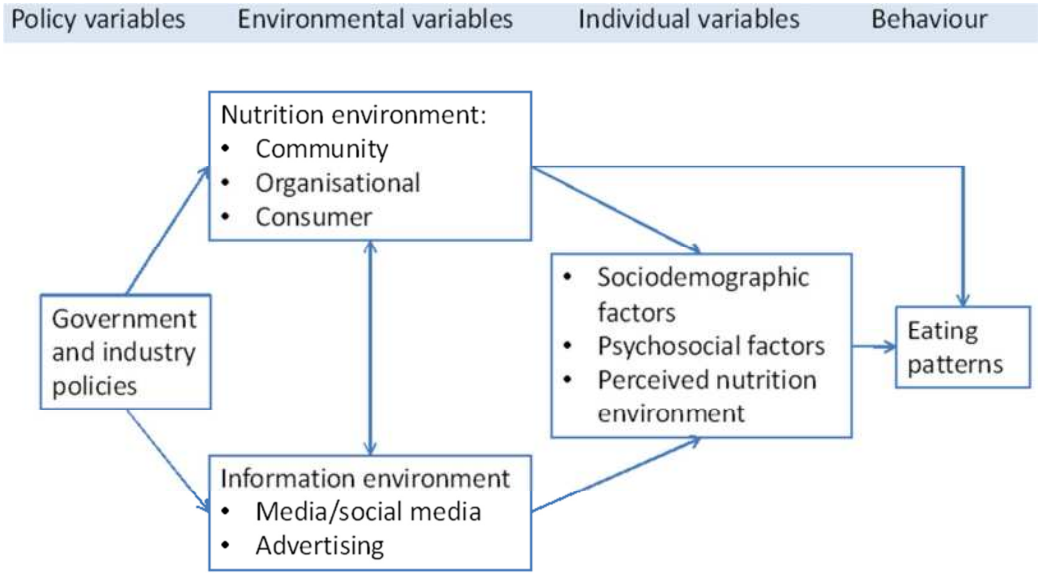
to focus on educating and informing people about their nutrition needs and how to make best use of nutrition information. Providing education and increasing people's understanding of nutrition is intended to increase their skills and capacities – their self-efficacy – and enable them to exert more control over their diets and eating behaviour. Self-efficacy itself has been shown to have an independent effect on health and on health behaviours *(294)*. In Glanz et al.'s conceptual model (modified in Figure 3) showing the way in which the nutrition environment influences individual eating patterns, nutrition literacy therefore acts as an individual level variable *(295)*. It is one of the psychosocial factors which mediate the relationship between environmental variables, such as the nutrition environment provided by schools and within the home, or the accessibility and types of food outlets, and individual eating patterns.

Figure 3. Conceptual model of how nutrition environment influences individual eating patterns



Source: adapted from Glanz et al. *(295)*

As nutrition literacy is a newly defined construct, there is little published evidence of its role in mediating healthy food choices or the extent to which interventions to change nutrition literacy are effective in improving dietary behaviour. However, a number of instruments have now been developed to measure nutrition literacy which will enable a better understanding of its role in the future *(291, 296)*. This short overview focuses on studies that assess nutrition literacy directly, but also considers more general reviews of health literacy in which diet and obesity-related behaviours are included as outcomes. There has been a reliance principally on systematic and other reviews to evaluate the role and importance of nutrition literacy together with nutrition knowledge, one of its key components, to consider opportunities for prevention of childhood obesity. Most of the evidence considered comes from studies carried out in high-income settings, which has implications for extending the findings to other cultures and contexts.

Nutrition literacy

The ability of parents to process and use nutrition information effectively is likely to be an important influence, both on their own, and their children's, dietary behaviour *(297, 298)*. Interventions to improve health literacy, including effects on nutrition and physical activity, have been shown to be effective in changing health behaviours *(299)*. In the first review of the role of health literacy in relation to nutrition behaviours and dietary outcomes, Carbone and Zoellner *(289)* considered 13 studies, six of which were experimental. Some benefits of higher literacy levels were observed, for example in two of the descriptive studies health literacy skills were associated with greater accuracy in estimation of portion sizes and trust in nutrition information sources. Furthermore, interventions to make nutrition education materials more readable, or use of strategies to enhance reading comprehension, resulted in improvements in nutrition knowledge. However the effects of these interventions on eating behaviour were mixed, and no studies reported significant changes in BMI. The authors comment that methodological and other differences across the literacy interventions included, make it difficult to draw conclusions regarding their effectiveness *(289)*.

More recently the role of "food literacy" has been considered in a systematic review of studies of adolescents *(300)* and in a review of adolescent food literacy programmes (*301*). Food literacy is variably defined in the literature *(302)*, although it has elements that are common to nutrition literacy, and is used synonymously for the purpose of this overview. In the systematic review, 13 studies were included, two of which were intervention studies. Overall, this review suggested that greater food literacy was linked to beneficial effects on dietary behaviours in adolescents, and could play a role in shaping youth eating behaviours. In eight studies there was a positive association between food literacy and dietary intake; for example, adolescents who had greater food knowledge and who were more frequently involved in food preparation had healthier dietary practices, and there was some evidence of sustained benefits in a longitudinal study that included five-year follow-up *(300)*. In comparison, the conclusions of the review of 23 adolescent food literacy programmes were less positive *(301)*. Although most studies reported some positive changes, these did not translate into significant improvements in diet quality or in cooking frequency at home. In common with Carbone and Zoellner's review *(289)*, methodological differences and issues of study quality were also highlighted as limitations, particularly the lack of randomized-controlled trials *(300)*. Perhaps the most significant issue, however, is that the studies reviewed evaluated aspects of food literacy (e.g. food knowledge) and none used a comprehensive tool to evaluate its effects. Vaitkeviciute and colleagues stress the need to develop validated tools to measure food literacy in order to strengthen future research in this area *(300)*. Consistent with this suggestion, studies that have assessed food literacy have shown links to healthier dietary behaviour – for example, using the Newest Vital Sign[1] tool to evaluate understanding of food labels in a low-literacy population, higher scores were shown to predict higher healthy eating index scores and lower consumption of sugar-sweetened beverages *(303)*.

In the context of prevention of childhood obesity, a range of interventions aimed at decreasing obesity in young children has been tested *(304)*. Some approaches have been shown to be effective

[1] The Newest Vital Sign is a screening tool available in English and Spanish that identifies patients at risk for low health literacy.

in improving dietary intake and parental knowledge about nutrition although, to date, none has examined effects of interventions that integrate a literacy-sensitive approach *(305)*.

Nutrition knowledge

Nutrition information is widespread, often provided within the school curriculum as well as by numerous health promotion and other agencies *(306)*. Nutrition knowledge is an integral component of nutrition literacy, and has been assessed in many studies. Knowledge is influenced by age, gender and level of education, with higher levels reported in middle-aged people, in women, and in those with higher education. However, the impact of nutrition knowledge on food consumption and nutrient intake is complex. Furthermore its specific contribution may be influenced by demographic and environmental factors *(307)* and differ across settings. A number of studies (e.g. those included in the reviews cited in the previous section *(300, 301)*) suggest that increasing nutrition knowledge alone may be ineffective in improving dietary behaviour. The most recent summary, of studies to evaluate the influence of nutrition knowledge on dietary intake, is a systematic review published by Spronk and colleagues in 2014 *(306)*. Evidence from 29 published studies was examined, mostly carried out in community settings; none had a randomized-controlled design. The majority of the studies included (n=19) found some positive associations between greater knowledge and markers of diet quality, most commonly judged in terms of fruit and vegetable consumption, although associations were modest ($r < 0.5$).

A comparable message of limited links between knowledge and dietary practice has also come from a recent review of population adherence to, and knowledge of, national nutrition guidance in the USA. Haack & Bycker carried out a systematic review of 31 studies evaluating the food guide pyramid and "MyPlate" over the period 1992–2013 *(308)*; 22 studies examined adherence to the national guidance, six examined knowledge, and three examined both adherence and knowledge. Although knowledge was variable across studies, most reported high rates of awareness of the national guidance, with additional evidence of participants having increased knowledge of the nutrition guides over time. However, overall rates of adherence to the guidance were low; there was no relationship between knowledge and adherence and, importantly, no improvement in adherence over time. The authors concluded that nutrition guides or campaigns are ineffective if individuals cannot apply the guidance to enable appropriate food choices, making incorporating behavioural strategies key to the utility of future guidance. Similar messages regarding the mismatch between nutrition knowledge and dietary practice are also described in other settings, including the United Kingdom *(309)*.

Food and menu labelling

Making healthy food choices may be more difficult in settings where energy-dense foods are readily available and marketed in ways that make it challenging to understand their nutritional content. In high-income settings, where there is increasing use of pre-prepared meals, nutritional labelling, to provide information on types and amounts of nutrients in the food, may be a way to enable consumers of these foods to make healthier choices. This approach to increasing nutrition literacy has international support *(310)*. In recent years more explicit nutritional information has been added

to food labels, including text or colour coding (e.g. “traffic lights”) to aid interpretation by putting nutrient content in the context of nutrient need or other guidance. This may make the information easier to understand, and should help consumers to make healthier food choices *(311)*.

A number of systematic reviews published over the past decade have shown that most consumers look at food labels, although difficulties in their interpretation are common, and a link between use of food labels and purchasing decisions is not well-established *(312, 313)*. However, nutrition labels are regarded as a highly credible source of information, and in a recent systematic review, Campos and colleagues *(314)* describe a consistent link between the use of food labels and healthier diets in both cross-sectional and longitudinal studies. There is also some experimental evidence of a positive effect of provision of nutritional information on food labels on dietary choices, although this is not a consistent finding in all studies *(314)*. Overall, the effectiveness of labels may be explained by their greater use among people who already have healthier preferences and greater intention to eat healthier foods *(283)*, with the risk that widespread dependence on labels could contribute to widening of current inequalities in health. The impact of nutritional labelling on health inequalities between high-income and low- and middle-income countries is therefore also a concern. To date, the effectiveness of food labelling to support and promote healthier dietary behaviours is not clearly established.

Menu labelling is a recent development, most commonly used to show the energy content of menu items in restaurants, canteens and other food outlets. There is some evidence that inclusion of the calorie count can affect food purchased *(315)*, but the impact of calorie labelling may depend on the characteristics of the consumer as well as the type of food outlet – for example negligible effects were found in a study of fast-food outlets, whereas benefits have been shown in coffee shops and work-based canteens *(283, 315)*. A systematic review and meta-analysis published in 2014 *(316)*, provides a current overview of the value of menu labelling as an influence on calories purchased or consumed. Seventeen studies were evaluated in this review; seven were quasi-experimental and ten were experimental. The review showed that menu labelling alone did not decrease calories selected or consumed. However, when additional information was added, such as contextual information to aid interpretation, labelling was effective in reducing calories consumed *(316)*. Sinclair and colleagues conclude that further studies are needed to determine the optimal approach for the provision of menu-based information, particularly for consumers who have limited food and health literacy skills.

Nutrition education and counselling

In their substantial review of interventions to increase health literacy, Taggart et al. identified 13 studies that used nutrition education to improve nutrition and nutrition behaviours *(299)*. Six of the seven studies that assessed nutrition knowledge showed increases attributable to the nutrition education programmes; and six of the total group of 13 nutrition education interventions they reviewed, seemed to produce significant and beneficial effects on diet. Of all the types of intervention they reviewed, Taggart et al. identified group education as the most likely to increase knowledge. Overall, however, the review suggests that whilst it may be possible to increase nutrition

knowledge with nutrition education programmes, there is no guarantee that this increase in knowledge will translate into changes in dietary behaviour *(299)*.

Some suggest that there is an argument for focusing efforts to increase aspects of health literacy in those who have low general literacy, because of its association with adverse health outcomes. In a systematic review of 20 interventions to improve health outcomes in those of low literacy, Pignone et al. identified four nutrition education interventions employing different educational strategies *(317)*. Only one of the four reported increases in nutrition knowledge and accompanying improvements in diet, referring to a reduced calorie intake from saturated fat. This intervention involved a series of six specially-designed nutrition classes. Another intervention, comprising eight nutrition education classes, failed to show any effect on self-reported dietary outcomes or on participants' ability to read food labels. By implication, improving the nutrition knowledge and diets of those with low literacy levels may require more support than simply educational classes.

The review by Taggart et al. *(299)* also examined the impact of individual counselling on health literacy and health behaviours, and found that of the three interventions they identified, only one appeared to result in increases in knowledge, and none produced improvements in nutrition or nutrition behaviours. This said, most of the individual counselling interventions were brief, consisting of only one counselling session. This was not true of other health behaviours, such as physical activity, which appeared to be more likely to increase following individual counselling.

Both the Pignone et al. *(317)* and Taggart et al. *(299)* reviews described attempts to address nutrition literacy in adults and are included here because of the assumption that the nutrition literacy of parents will affect children's likelihood of obesity. There is a large, additional literature on nutrition education in schools, which is described in other sections of this report. To summarize, a Cochrane review *(59)* and a review by the American Academy of Nutrition and Dietetics *(318)*, conclude that nutrition education can be effective in having small, but beneficial, effects on children's body mass, particularly if the curriculum on healthy eating is integrated into a wider school curriculum and if interventions combine nutrition education with increased opportunities for physical activity. The American Academy review also concludes, however, that, "There is insufficient evidence to draw conclusions about the effectiveness of school-based nutrition education interventions alone to address adiposity in children", while at the same time emphasizing that "children are inundated with messages promoting consumption of high-energy foods, so it is important to intensify and sustain the dose of nutrition education" *(318)*.

There is a slightly different message from a review of nutrition education in preschool children. Hesketh and Campbell identified 23 interventions studies, all of which involved education to improve diet or increases in physical activity *(319)*. Two thirds of the studies examined were successful in modifying some aspect of diet or activity behaviour, and the most successful were those that included both nutrition and physical activity as integral parts of the intervention. Again this would seem to suggest that targeting obesity prevention through nutrition education alone is not as effective as a combination approach.

Computer-mediated interventions to increase nutrition literacy

There is considerable interest in the use of interactive media to increase nutrition literacy. Methods include computer programmes for entertainment and on-line educational tools *(320–322)*, as well as mobile phone technology to support behaviour change *(323)*. Evaluations of their effectiveness have provided some promising results. In a systematic review of 15 studies of computer-mediated, obesity-related nutrition education interventions in adolescents, positive changes in diet, physical activity, knowledge and self-efficacy were shown *(320)*. For example, a computer tailored multiple behaviour intervention delivered to 1182 high school students was effective in increasing fruit and vegetable consumption, and the intervention group also reported higher physical activity levels *(324)*. However, the overall finding was that changes were small, and there was also little evidence that they were sustained *(320)*. Hou and colleagues *(321)* conducted a wider review of internet-based interventions to promote a range of health behaviours. Most included provision of web-based intervention, tailored feedback, regular emails, self-assessment and goal-setting. All studies that examined nutrition outcomes, with or without physical activity, reported positive improvements, and the authors concluded that the studies reviewed showed that internet-based interventions are effective in increasing targeted health or behavioural outcomes. However it may be difficult to isolate the role of computer-mediated delivery of nutrition information within studies that also included other components such as personalized feedback. New studies are essential to understanding the value of interactive media for the promotion of nutrition literacy and healthier behaviours.

Summary

An increasing understanding of the role of appropriate nutrition and its links to good health is likely to be central to the success of strategies to improve diet. The studies included in this overview point to effectiveness of interventions that aim to improve nutrition knowledge and understanding. However, the impact of these interventions on dietary behaviour is less clear – consistent with the widely recognized gap that exists between knowledge and practice. Nutrition literacy goes beyond the acquisition of knowledge to the ability to "process, and understand basic nutrition information and services to make appropriate nutrition decisions". The reviewed studies provide limited evidence that improvements in knowledge translate into better dietary decisions, limiting the potential of provision of information alone as a strategy to promote healthy diets in childhood and to prevent obesity. Nutrition literacy is one of many influences on food choices. Undoubtedly, good nutrition knowledge and understanding are essential to enabling appropriate food purchasing decisions and healthier dietary behaviours. And their roles are recognized in the NOURISHING food policy framework *(325)* for healthy diets and prevention of obesity *(326)*. However, improving nutrition literacy alone may not be effective in improving dietary behaviours. It is unlikely that literacy can be separated from the powerful role of context, which may undermine individuals' efforts to eat healthily *(326)*, as well as personal factors such as self-efficacy, that enable knowledge and understanding to be translated into healthier food choices. While nutrition literacy would be expected to play a role in effective interventions to promote healthier diets and prevent obesity, current evidence suggests that supportive food environments may be key to their success.

5.4 The obesogenic impact of global marketing and advertising aimed at children[1]

There are many reasons for the current epidemic of childhood obesity, but there is ample research to show that unhealthy food promotion is one important and independent causal factor. This section summarizes this evidence base and explains why it is sound and is likely to be a conservative estimate of the problem. It also discusses why children are particularly vulnerable to such promotion and why unhealthy foods get disproportionate promotional support. It concludes that any attempt to tackle childhood obesity must encompass measures to reduce radically children's exposure to unhealthy food promotion, and that such measures are intuitively sound and are associated with no adverse health-related consequences. It also argues that, in combination with these measures, social marketing interventions will also have an important role to play.

The evidence base

There is strong scientific evidence that food and beverage marketing is contributing to the childhood obesity epidemic. Over a decade ago two comprehensive reviews were conducted, one by the United Kingdom Food Standards Agency *(327)* and the other by the US Institute of Medicine *(328)*. These reviews systematically examined all the reputable studies that had been carried out in this field at that time and concluded that:

(i) There is a lot of food advertising to children. Studies examining the extent and nature of food promotion consistently conclude that food is promoted to children more than any other type of product.

(ii) Processed foods that are high in salt, sugar and fat dominate advertising. They comprise five principal categories: sugary breakfast cereals, "soft" drinks, confectionary, savoury snacks and products from fast-food outlets. By contrast there is very little promotion of unprocessed foods, such as fruit and vegetables, wholegrain products and milk. Thus the advertised diet is much less healthy than the recommended diet.

(iii) Research shows that children enjoy and engage with the advertising, which typically focuses on themes of fun, fantasy, novelty and flavour. Food marketers target children because they may have their own money to spend and also because they can influence parental purchasing decisions. In addition, there is evidence that children are used by marketers as a bridgehead into low- and middle-income countries where adults may be more resistant to westernised diets *(329)*. There is also evidence from these countries *(330)* that children now have influence on the overall spending decisions of the family for food and snacks, as well as other products, and that this makes them particularly interesting to marketers and vulnerable to marketing (see below).

(iv) To determine whether there is a direct causal link between such advertising and the food-related knowledge, attitudes and behaviour of children, requires complex studies using a range of methodologies. Many such studies have been carried out, and careful review of these shows

[1] Authors: Gerard Hastings, Amandine Garde, Frank Chaloupka and John Reilly.

that food advertising has the following effects: undermining food knowledge and confusing children as to what are healthy and unhealthy foods; stimulating a preference for unhealthy foods; encouraging the purchase of, and pestering for, unhealthy foods; and making children more likely to consume unhealthy foods. This unhealthy eating is directly linked to the recent increases in childhood obesity and weight gain, and related health-harms.

All these effects have been shown to be statistically independent of other factors, such as parenting behaviours, peer influence and television viewing. They are also present for both category and brand choices – thus advertising does not only persuade children to eat, for example, brand "A" burgers rather than brand "B" burgers, but to eat burgers per se *(331)*.

These findings have since been replicated and confirmed by many further studies and reviews. In 2012, for example, a review published in the journal *Appetite* concluded: *"*Food promotions have a direct effect on children's nutrition knowledge, preferences, purchase behaviour, consumption patterns and diet-related health. Current marketing practice predominantly promotes low-nutrition foods and beverages *(331)"*.

The link between advertising and young people's consumption has been empirically established in two other sectors: tobacco and alcohol. The harms to health of energy-dense foods, alcohol and tobacco have become known as "industrial epidemics" *(332)* because of the extent to which they are driven by commercial interests. As a recent paper in *The Lancet* explains: "through the sale and promotion of tobacco, alcohol, and ultra-processed food and drink (unhealthy commodities), transnational corporations are major drivers of global epidemics of noncommunicable diseases*" (333).* Childhood obesity is a precursor of these noncommunicable diseases.

Marketing strategies targeting children, especially for processed foods and snacks, have also been linked to household disposable income of the middle class in low- and middle-income countries. The growing consumption pattern of the middle class is not homogeneous and is also one of the driving forces for increasing demands for ready-to-eat food items. In several rapidly-transitioning societies, the family environment has changed due to mothers entering the workforce and consequent perception among them that these food items could be measures to relieve them of constraints on their time.

Exposure is the key concern

Attempts have been made to determine whether certain creative approaches used by advertisers (e.g. celebrity endorsement or movie tie-ins) have a particular power over children, and therefore warrant specific regulatory attention. However, establishing this link has proved difficult. Results can vary between campaigns – the celebrity or movie being used, for instance, can be more or less influential. Also, as Box E shows, digital marketing is rapidly blurring any distinction between medium and message. Most importantly, the evidence base shows that it is food promotion in general – not just a specific type of content – that is harming children.

The focus thus has to be on exposure. The best way to protect children from unhealthy food promotion is to reduce the amount they see, whatever the approach being used.

BOX E. Digital online marketing

While television often remains a frequently-used medium for marketing purposes, the Internet and other forms of digital marketing (not least smartphones) have increased rapidly during the last decade and are expected to increase their share of total advertising expenditure in the coming years. For example, estimates of advertising expenditure in Western Europe indicate that Internet spending is expected to rise from 20% of total advertising expenditure to 30% over the period 2010–2015, while Internet marketing expenditure in the United Kingdom already exceeds television advertising expenditure.[1]

Children access the Internet from an early age,[2] *and often without parental or any adult supervision. Furthermore, as is with television advertising, the types of foods marketed to children online or via other new media tends to be unhealthy. In addition, the Internet and other forms of digital marketing allow food companies to develop marketing strategies which are far more immersive, interactive and integrated than they are able to do on television or other more traditional media. To examine these aspects further:*

Immersive: the online environment often portrays marketing opportunities as entertainment, making it particularly difficult for children to distinguish marketing from content, to the point that marketing sometimes becomes content. For example, major food companies have internet sites offering a range of games promoting their goods, services and brands ("advergames"). These games, which are often intended for children without always referring explicitly to the goods, services and brands thus promoted, are highly immersive. They are designed to be entertaining and rely on children playing for long periods of time, sometimes with several repeat visits. They are a particular cause for concern as they tend to operate "under the cognitive radar", i.e. without children being aware that they are the targets of unhealthy food marketing. A systematic review of the impact of advergames on children was carried out in 2012, highlighting their influence on children's dietary choices. Moreover, it also emphasizes that "research that tests the effects of advergames on children, rather than their understanding of commercial intent, concurs that older and more experienced children are just as affected by advergames as younger children".[3] *Several studies have since confirmed the poor nutritional value of the foods marketed to children via advergames.*

Interactive: media such as the Internet and smartphones enable companies to gather information and adapt their marketing strategies to target each potential customer as individually and effectively as possible. In particular, the increasing use of "behavioural marketing" (coupled with geolocalization technology) personalizes the connection between a brand and its customers. This trend is compounded by the rapid development of social networking sites which allows food manufacturers to ensure that their brands are referred to, "liked" and promoted by children themselves without

[1] WHO Europe, *Marketing of foods high in fat, salt and sugar to children: Update 2012-2013*, Copenhagen, 2013, at p. 5.
[2] For example, in 2011, in the United Kingdom, 65% of children aged 5–7 years and 85% of children aged 8–11 years accessed the Internet through home computers. Several of them also own a smartphone: 1 in 50 children aged 5–7 years and 1 in 8 children aged 8–11 years: *Children and parents: media use and attitudes*. London, Ofcom, 2011.
[3] A. Nairn and H. Hang, *Advergames, it's not child play*, Family and Parenting Institute, London, 2012, pp. 14 and 15.

them always realizing that they have been recruited as "brand ambassadors". This form of "viral marketing" in turn enables the brands to create a mix of social impressions which incorporate both "paid" and "earned" media. However, social media marketing increases advertisement recall, awareness of the product or brand and intent to purchase.[1] Even though children may have to declare that they are over a certain age to register on networking sites and message services, evidence suggests that they often register on such sites under false ages and access them at a much younger age which, in turn, raises questions around the effectiveness of age-verification,[2] and gives rise to both public health and data protection concerns.

Integrated: the Internet has significantly increased the opportunities available to food operators to ensure that their strategies are always more integrated. For example, in the USA, a survey reviewed 130 food company websites: 48% had designated children's areas, featuring a variety of marketing techniques including advergaming, interactive programmes, branded spokes-characters and tie-ins to other products. Of the companies with child-oriented sites, 87% promoted unhealthy food.[3] Beyond their own websites, major food operators have created powerful alliances with companies such as Google in order to increase their presence on search engines and emails, as well as Facebook, Twitter and YouTube and other user-generated content sites.

Digital online marketing has become an integral component of the "marketing mix" of food brands: it has not replaced other forms of more traditional food marketing; rather, it has been added to them to increase brand presence across a larger, more diverse range of media. With digital opportunities, food marketing has become more ubiquitous and multifaceted than ever.

Methodological rigour

Unpacking the effects of a phenomenon as ubiquitous and multifaceted as food marketing is difficult, and researchers have used a variety of different methods to meet the challenge. These include interviews and surveys (with young people, adults and experts), controlled experiments (e.g. exposing pupils to specific advertisements), natural experiments (monitoring what happens when something in the food environment changes as a result, say, of government policy) and statistical analysis of official data (e.g. on advertisement spending and obesity trends). All of these methods have their drawbacks: interviewees can be unreliable; controlled experiments too artificial; natural ones too messy; and official statistics incomplete. On the other hand, they can each shed useful light on the problem.

In these circumstances, it is important not to rely on any one type of study, but to examine the totality of the evidence – and this emphasizes the need for rigorous sifting and review methods. To meet this challenge, procedures have been borrowed from medical science, where great care is needed to ensure that particular treatments are especially safe and effective, and to guarantee that every possible source of evidence is identified and properly evaluated. Electronic databases have made the search process extremely efficient, and objective inclusion and exclusion criteria can then

[1] Advertising effectiveness: understanding the value of a social media impression. New York, Nielsen, 2010.
[2] ASA Compliance Survey, *Children and advertising on social media websites*, London, July 2013.
[3] A. Henry and M. Story, "Food and beverage brands that market to children and adolescents on the internet: a content analysis of branded websites", *Journal of Nutrition Education and Behaviour* (2009) 23:150.

be applied to determine which studies are worthy of consideration. The precise methods of this search and evaluation process are laid down in detailed protocols, so that anyone can appraise the review and, if necessary, replicate them to verify conclusions. The best reviews, including the ones cited above, use these rigorous and transparent systematic review methods.

Understating the problem

Two further factors suggest that the evidence base likely *underestimates* the effect that food marketing has on children. First, the studies have looked at *direct* effects on individual children, and so do not address *indirect* and *social* influences. For example, promotion for "fast food" outlets may not only influence the child, but encourage parents to take them for meals and reinforce the broader idea that this is a normal and desirable behaviour. Research in China, for instance, showed that young people patronized the newly arrived Pizza Hut outlets because they were the fashionable place to be seen, despite not actually liking pizza *(334)*. Research in recent years has shown that these "social norming" effects are particularly powerful *(335)*. Secondly, the literature focuses principally on television advertising, which, while important, is only one of many channels companies use to promote food products and services. Television commercials are, for example, combined with techniques such as sports sponsorship, celebrity endorsement, billboards, public relations and, increasingly, digital promotions to construct the most persuasive overall communications effort.

This mix of messages is, in turn, combined with new product development, ubiquitous distribution and attractive pricing strategies to develop the most persuasive marketing effort. Box F, for example, illustrates how price can influence consumption.

All of these techniques feed into long-term marketing strategies that focus on building evocative brands, often over many decades. Against this strategic context, studies looking at the impact of current advertising can, inevitably, only tell part of the story.

BOX F. Food and beverage pricing

Pricing strategies are a key component of food and beverage marketing activities. Prices reflect a variety of factors, from the costs of production and distribution, to the degree of competition in marketplaces. Increasing globalization of the processed food industry has increased the market power of food and beverage companies, giving them greater ability to manipulate prices, generating greater revenues that can be used for increased advertising and other marketing efforts.

Extensive economic evidence demonstrates that the fundamental economic "law of demand" applies to food and beverage consumption, with increases in the price of a given product leading to reductions in the quantity consumed of that product, and vice versa. Economists use the "price elasticity of demand" to quantify this relationship, with price elasticity defined as the percentage change in consumption resulting from a 1% increase in price. How responsive demand is to price varies across products. A 2010 systematic review of 160 studies concluded that the price elasticities

for food and non-alcoholic beverages ranged from -0.27% to -0.81%, with the demands for food away from home, "soft" drinks, juices, and meats being relatively more responsive to price (with a 10% price increase reducing consumption by 7–8%), and the demands for eggs, sweets/sugars, cheese, and fats/oils being relatively less responsive to price (with a 10% price increase reducing consumption by 3–5%) (336). The basic finding that changes in food and beverage prices affect consumption is confirmed in two recent systematic reviews of more recent research (337, 338). One of these reviews, focused on English-language studies published from January 2007 to March 2012, concluded that the demand for more narrowly-defined categories of foods and beverages was more responsive to price (337). For example, this review concluded that the demand for sugar-sweetened beverages would fall by over 12% in response to a 10% increase in price, while demand for "soft" drinks overall would fall by 8.6% following a 10% price increase.

While the evidence is clear that a change in price for a given product leads to changes in consumption of that product, the impact on nutritional quality of the overall diet and on weight outcomes is less clear, given the potential for substitution among products in response to changes in relative prices. For example, an increase in the price of carbonated sodas, if prices for other beverages remain unchanged, will lead to a significant decline in soda consumption, but will likely result in increased consumption of other beverages, including sports, energy, and juice drinks. A growing evidence-base suggests that changes in the prices of, at least, some categories of foods and beverages do lead to changes in weight outcomes, with higher "fast food" and sugar-sweetened beverage prices and lower fruit and vegetable prices associated with improved weight outcomes among children and adults, particularly those in lower-income families (337).

The evidence on the impact of prices on food and beverage consumption, nutritional quality of the diet, and weight outcomes, coupled with the extensive evidence on the public health benefits of higher tobacco taxes (339) has led to growing interest in using fiscal policies to promote healthier eating and curb the obesity epidemic and its consequences. In 2011, for example, the United Nations recommended the use of fiscal measures to improve diets and health at its High-level Meeting on Non-communicable Diseases (340). More recently, the Pan American Health Organization's action plan for the prevention of obesity among children and adolescents called on Member States "to increase the price of sugar-sweetened beverages and energy-dense nutrient-poor products through taxation policies" (341).

Governments have begun to use fiscal policies, including taxes on less healthy products and subsidies for healthier products, in efforts to address unhealthy diets and obesity. A variety of programmes have been implemented to reduce the prices of healthier options through subsidies on fruits, vegetables, low-fat snacks, and other products, with evaluations of these programmes consistently showing that the subsidies lead to increased purchase and consumption of the subsidized products (342). Similarly, a number of governments have adopted taxes on selected products or nutrients. For example, in September 2011, Hungary adopted a tax on packaged products with excess sugar, salt or fat, while raising its previously implemented "soft" drink tax. In October 2011, Denmark introduced a "fat tax" on processed foods containing more than 2.3% saturated fat, as well as on butter, milk, cheese, oil, pizza and meat, but repealed the tax a year later. In 2012, France introduced a new tax on all beverages containing added sugars or artificial sweeteners, and extended the tax to include energy drinks in 2013. Effective from January 1, 2014, Mexico implemented a one peso per litre tax on sugar-sweetened beverages and an 8% tax on "junk foods" (non-essential foods high in sodium, added sugars, and/or solid fats), including salty snacks, candy, ice cream, and more. Early evidence

based on the experiences with food taxes in the European Union confirms that these taxes lead to reduced consumption of taxed products, as well as product reformulation that lowers the sugar, fat or salt content of various products, while at the same time leading to substitution to untaxed or lower taxed products and/or cheaper brands of taxed products (343).

One recent systematic review of the evidence on food/beverage taxes and subsidies concluded that these policies do lead to improvements in the consumption patterns that contribute to obesity and noncommunicable diseases (338). It noted that the evidence was strongest for sugary beverage taxes and for subsidies of healthier foods. Similarly, it concluded that taxes based on nutrient profiling (e.g. "junk foods") had a greater impact than targeted nutrient taxes (those based on fat, sugar or salt) and that the former were less likely to have unintended consequences from substitution to other nutrients and were less likely to apply to foods recommended in dietary guidelines. At the same time, it noted that there is some evidence that subsidies alone can lead to increases in overall calorie consumption.

Child vulnerability

Children need particular protection; society recognizes that they are more vulnerable to exploitation than adults. In the case of advertising, the extent of this vulnerability has often focused on a child's cognitive development: at what age are they able to fully understand the nature of advertising? This will obviously vary from child to child, but it is generally agreed that although the first recognition of advertising begins around the age of 4 or 5 years, it is probably not until early adolescence (about 12 years) that children fully understand the persuasive purpose and partiality of advertising, and acquire the skills to question and counter-argue. This suggests that advertising to children and young adolescents is inherently unfair.

More recent research on advertising adds an additional concern, demonstrating that as well as the cognitive influence of an explicit sales pitch (e.g. "buy our soda because it is refreshing"), advertising also uses implicit, emotional levers (e.g. "our soda is a synonym for happiness"). Such subtle methods are especially effective with small, everyday purchases – including many food items – which are often made without any great thought or purely on impulse. Because they are indirect, even adults find it difficult to counter these advertising effects; children, who are still developing emotionally, are likely to be especially susceptible.

This vulnerability is also likely to be even greater in low- and middle-income countries, where sophisticated media communications and mass marketing are relatively novel. The major food producers are multinational businesses and their promotional campaigns are reaching into the poorest countries with marketing which "mirrors the strategies, techniques and channels deployed in high-income countries" and promotes "foods new to the indigenous food culture, such as fast-food, dairy products in Asia, and carbonated soft drinks" *(331)*. Empirical data is now confirming this tendency *(344)*.

Why is the advertised diet unhealthy?

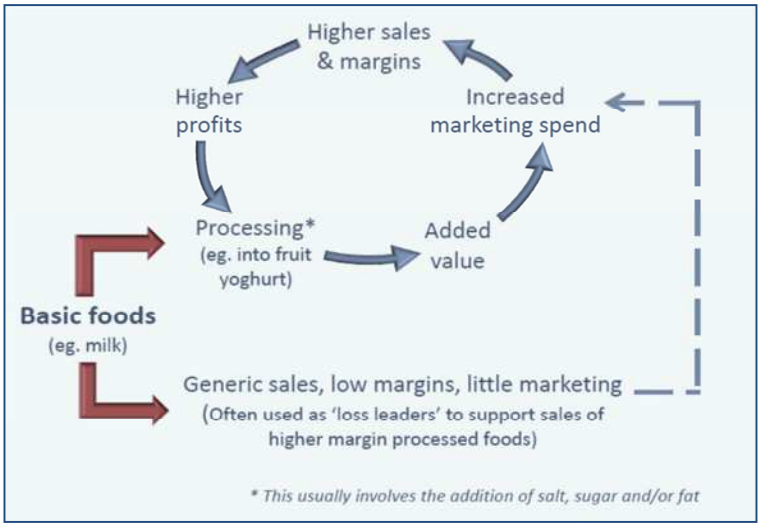
It is useful to consider why the advertised diet is so unhealthy. The explanation lies in food processing and the profits this delivers.

Marketers seek to "add value" to their products so that they can sell them at a higher price and thus maximize their returns. In the case of food products, "adding value", typically means "processing". A simple potato will sell for a few pence per pound, but when sliced, fried and salted into crisps or chips, the price multiplies. Similarly, milk is a supermarket loss-leader but becomes a premium product when converted into creamy fruit yoghurt. It is this difference in price between the basic and processed product that generates marketing budgets. If fresh milk sells for $1 per litre, but when converted into yoghurt sells for $3 per litre, much of the $2 difference can be spent on marketing. Furthermore, yoghurt has a longer "shelf life" than milk and is easier to transport and this further extends the potential investment in marketing. Thus crisps/chips and yoghurt are supported by extensive promotion and evocative branding, but potatoes and milk are not.

Unfortunately, although processing increases the monetary value of basic foodstuffs, in most instances the nutritional value is reduced. This is because, as in the case of potatoes and milk, processing involves adding some combination of salt, sugar and fat – each of which is a key marker of an unhealthy diet. Conversely, in the manufacture of white bread, processing involves the removal of good constituents, as with wholegrain. As a paper in *The Lancet* explains: *"*ultra-processed foods" such as *"*burgers, frozen pizza and pasta dishes, nuggets and sticks, crisps, biscuits, confectionery, cereal bars, carbonated and other sugared drinks, and various snack products … are typically energy dense; have a high glycaemic load; are low in dietary fibre, micronutrients, and phytochemicals; and are high in unhealthy types of dietary fat, free sugars, and sodium*" (333)*.

This powerful circular dynamic (Figure 4) of processing natural foods to add value, thereby generating promotional budgets to encourage consumption, means that, in the absence of regulation, food promotion will inevitably tend to favour unhealthy food choices. On the other hand, if rules were introduced requiring that only healthy foods (well-validated nutrient-profiling systems can identify these) get promotional support this could help rebalance the food market to combat obesity.

Figure 4. Why is marketing unhealthy?



Ways forward

An extensive body of peer-reviewed research shows that food promotion is one of the key drivers of the current epidemic in obesity. This is because a) advertising and all the related marketing communications are powerful influences, and b) for commercial reasons; promotional efforts tend to favour unhealthy foods and beverages. These effects are not the result of any particular type of promotional appeal (e.g. cartoon characters or celebrity endorsements), but the cumulative effect of all food promotion: in essence, as noted above, the advertised diet is much less healthy than the recommended diet.

A key policy response therefore is to reduce the exposure of children to unhealthy marketing. Experience suggests that any such controls need to be comprehensive and radical – partial restrictions on advertising in certain media, or at specific times, result only in greater exposure from other media and at other times. For example, an independent evaluation of the United Kingdom's restrictions on unhealthy food advertising around children's television programmes concluded that, "Despite good adherence to the restrictions, they did not change relative exposure of children to HFSS [high fat, salt and sugar] advertising and were associated with an increase in relative exposure of all viewers to HFSS advertising. Stronger restrictions targeting a wider range of advertisements are necessary to reduce exposure of children to marketing of less healthful foods" *(345)*. Experience from tobacco control shows that comprehensive controls on advertising have a significant protective effect on young people, discouraging both the onset and continuance of smoking *(346)*.

More positively, if, as noted above, nutrient profiling is used to identify healthy foods and promotion is limited by regulation to these foods, food promotion could move from being part of the problem to being part of the solution.

In addition, the discipline of "social marketing" offers hope. This takes ideas such as advertising, branding and customer orientation, which commercial marketers use to mould consumption behaviour, and uses them to influence social and health behaviours. There is good evidence *(347, 348)* to show that social marketing interventions can successfully encourage both healthy eating and greater physical activity – thus making a useful contribution to fighting obesity.

However, it is important to recognize that social marketing is not an alternative to the adequate regulation of commercial food promotion. The disproportionately high budgets of food producers and retailers mean that social marketing cannot compete with unsympathetic commercial marketing.

Fundamental rights as a lever for change

Several United Nations (UN) bodies have called for a rights-based approach towards marketing restrictions, on the grounds that fundamental rights should enhance, rather than undermine, the noncommunicable disease prevention and control agenda. As Olivier De Schutter, former UN Special Rapporteur on the Right to Food, argued:

> It is unacceptable that when lives are at stake, we go no further than soft, promotional measures that ultimately rely on consumer choice, without addressing the supply side of the food chain. Food advertising is proven to have a strong impact on children, and must be strictly regulated in order to avoid the development of bad eating habits early in life […] There is 'no reason why the promotion of foods that are known to have detrimental health impacts should be allowed to continue unimpeded' *(349)*.

Other UN bodies have also interpreted fundamental rights as a rationale for restricting the marketing of unhealthy foods to children. Thus, the UN Committee on the Rights of the Child called on UN Member States to address not only tobacco consumption, but also alcohol consumption and obesity. In particular, it noted that "children's exposure to 'fast foods' that are high in fat, sugar or salt, energy-dense and micronutrient-poor, and drinks containing high levels of caffeine or other potentially harmful substances should be limited. The marketing of these substances – especially when such marketing is focused on children – should be regulated and their availability in schools and other places controlled."[1] The UN High Commissioner for Human Rights similarly stressed that "obesity […] and substance use […]" were among "the areas requiring sustained and immediate attention" *(350)*. UN Member States should therefore "prioritize issues that have received little attention to date […] They should ensure adequate attention to the underlying determinants of child health, including, inter alia, access to minimum safe and nutritionally adequate food, basic shelter, housing, sanitation, safe and potable water and a healthy and safe environment." *(351)* More

[1] At para. 47 of the General Comment N° 15 (2013) on the right of the child to the enjoyment of the highest attainable standard of health. The Committee on the Rights of the Child has also expressed its concerns relating to growing childhood obesity in General Comment N° 17 (2013) interpreting Article 31 of the CRC on the right of the child to rest, leisure, play, recreational activities, cultural life and the arts: Growing dependence on screen-related activities is thought to be associated with reduced levels of physical activity among children, poor sleep patterns, growing levels of obesity and other related illnesses" (at para. 46).

recently, Anand Grover, the UN Special Rapporteur on the Right to Health, highlighted that the pervasive marketing of unhealthy foods has contributed to the increased consumption of such foods, before outlining a number of policies to increase the availability and accessibility of healthier food options, including through the regulation (as opposed to the self-regulation) of marketing and promotion of unhealthy foods:

> Owing to the inherent problems associated with self-regulation and public–private partnerships, there is a need for States to adopt laws that prevent companies from using insidious marketing strategies. The responsibility to protect the enjoyment of the right to health warrants State intervention in situations when third parties, such as food companies, use their position to influence dietary habits by directly or indirectly encouraging unhealthy diets, which negatively affect people's health. Therefore, States have a positive duty to regulate unhealthy food advertising and the promotion strategies of food companies. Under the right to health, States are especially required to protect vulnerable groups such as children from violations of their right to health *(352)*.

The principle that all actions by public authorities should be undertaken in the best interest of the child calls for the imposition of tougher restrictions on the marketing of unhealthy food to children. The right to health; the right to adequate food; the right to education; the right to play; the right of the child to be free from economic exploitation – all support the argument that States should restrict the exposure of children to unhealthy food marketing *(353)*. It is all the more important that these rights are systematically invoked if they are to be used as a "sword" – i.e. to actively promote the highest attainable standard of health – rather than merely as a "shield" – i.e. to oppose challenges of industry operators that food marketing restrictions would infringe their right to free commercial expression *(28)*.

Summary

The evidence base shows that unhealthy food marketing is an important and independent causal factor in the childhood obesity epidemic. Efforts to combat this problem have to include robust measures to limit children's exposure to such marketing; there are no adverse health-related consequences to such measures, and evidence from tobacco control shows that they have significant protective potential. In combination with these measures, social marketing also offers a promising way forward.

6. INTERVENTIONS TO ADDRESS CHILDHOOD OBESITY

6.1 Preconception and pregnancy: reducing obesity risk[1]

Primary prevention of child obesity requires a life-course perspective with attention to intergenerational risk factors and preconception health. A focus on young girls is crucial, because their health and nutritional status prior to, as well as during, pregnancy influence fetal growth and body composition, metabolic and hormonal pathways, and newborn weight and adiposity. Periconceptional diet and other lifestyle factors affect the embryonic development even before a couple know that they have conceived. The developmental trajectory of new human beings, and thus their biological capabilities for health or susceptibility to diseases, starts to be entrained within the first week after fertilization. The importance of the embryonic period is apparent from a range of animal studies *(354),* and in humans from the long-term health consequences of assisted reproductive technologies *(355)*. Awareness of pregnancy may lead to a woman's first contact with a health-care provider, making preventive measures outside the health-care sector important. In this respect, socioeconomic factors are important, and it is critical in many societies to engage the support of partners and other family members. In many societies, a substantial proportion of pregnancies are unplanned and many women do not change their diet or their lifestyle when they know that they are pregnant *(356)*. Prudence and parsimony in the approach to primordial prevention of obesity and comorbidities therefore demand that the maternal biological environment, within which conception occurs, is optimized long before the likelihood of conception *(356)*. Recent evidence suggests that paternal diet, body composition and behaviour can also affect offspring development and health; it is thus important to involve partners and other family members in primordial prevention initiatives.

Birth outcomes, including gestational age, birth weight and length, and body composition reflect a wide range of parental influences, and strongly predict future child adiposity. Risk of excess childhood adiposity is, in theory, modulated by developmental entrainment of appetite, arrangement of intermediary metabolism around fuel partitioning, and physical activity energy expenditure. Birth characteristics are sentinel indicators of developmental impact on underlying physiology and metabolism. Thus, child adiposity is related to birth characteristics, and to continuing effects of prenatal maternal and fetal factors interacting with postnatal exposures. It is important to understand better, and more fully, how preconception and pregnancy behaviour and environmental factors relate to infant birth outcomes.

Most attention in this context has been given to nutrition. Maternal nutrition, before and during very early pregnancy, influences the nature and strength of signals that mould developmental biology. This first crucial point of interaction between genetics, epigenetics, and the parental environment lays the groundwork for embryonic and fetal development, which is subsequently modulated by nutritional status throughout the rest of the pregnancy. These very early influences

[1] Authors: Linda Adair, Narendra Arora, Mark Hanson, Ronald Ma, Terrence Forrester, Chitteranjan Yajnik, and Zulfiqar Bhutta.

determine crucial aspects of fetal tissue and organ development (e.g. number of pancreatic beta cells or skeletal muscle mass) and alter gene expression and metabolic function in pathways related to regulation of growth and metabolism *(357–360).* Two pathways of risk for childhood obesity should be considered: one is dominated by maternal and fetal undernutrition, manifested as maternal short stature, low pre-pregnancy BMI, inadequate gestational weight gain, and/or micronutrient deficiencies and fetal growth restriction; the second involves maternal and fetal overnutrition associated with maternal overweight and obesity and unbalanced diet, but also with conditions such as gestational diabetes mellitus and excess fetal growth. Each of these developmental pathways is exacerbated by later exposure of the child to an obesogenic environment.

Undernutrition

A pattern of maternal undernutrition and high rates of infection is more prevalent in low- and middle-income countries, but also occurs in more affluent groups among women with the kinds of poor quality diets, prior to and during pregnancy, that are associated with fetal adaptive responses and greater adiposity in young children *(361, 362)*. Maternal short stature, often the result of childhood stunting, is consistently associated with lower infant birth weight *(361)*. Systematic reviews of high quality observational cohort studies from diverse regions estimated that pre-pregnancy underweight (BMI < 18.5 kg/m^2) increases the risk of low birth weight by 47%, of being born small-for-gestational-age by 81% *(363)*, and of preterm birth by 32% *(364)*. Inadequate gestational weight gain independent of, and interacting with, pre-pregnancy weight status is similarly related to lower offspring birth weight, and higher risk of small-for-gestational-age *(365, 366)*. Low gestational weight gain is a risk for these poor outcomes even among overweight or obese women *(367)*. Despite lower birth weight, it has been suggested that small-for-gestational-age infants have relative deficits in skeletal muscle mass, but not adiposity (the “thin-fat” baby), with important implications for later adiposity and diabetes risk *(368)*. Periconceptional maternal micronutrient status also influences the development of the placenta and fetus, and affects numerous regulatory processes with implications for postnatal energy metabolism and adiposity *(359, 360)*. Circulating micronutrients at conception and during pregnancy may influence expression of genes regulating growth *(357)*. In the context of adiposity, particular attention has been paid to micronutrients that influence DNA methylation of IGF and other growth-regulating genes *(359)*. Unbalanced maternal diet is associated with epigenetic effects on offspring, measurable at birth *(369)*, and substantial effects on levels of adiposity at 6–9 years of age. Deficiencies of iodine, iron, and zinc are significantly associated with increased risk of low-birth-weight, small-for-gestational-age and/or preterm birth *(360)*.

Maternal overweight and obesity

Maternal overweight in pregnancy is well known to be associated with detrimental effects on birth outcomes and later offspring health *(370)*. Compared with women of normal weight, obese women enter pregnancy more insulin resistant *(371)* and are more likely to develop gestational diabetes mellitus. Based on 70 studies of ~700 000 women, maternal overweight was found to double, and obesity to triple, the risk of gestational diabetes mellitus *(372)*. Maternal obesity and gestational diabetes mellitus create an intrauterine environment with high levels of glucose and insulin, resulting in faster fetal growth, altered fetal and newborn glucose regulation, insulin resistance, and

increased risk of being born large-for-gestational age or with macrosomia (birth weight > 4 kg) *(372–374)*. There are independent and synergistic effects of maternal obesity and glycaemia on birth outcomes, with influence of glucose across the full distribution of values as well as effects of maternal diet via glycaemic load and index *(375)* and effects of obesity independent of maternal glucose *(376)*. Untreated or inadequately treated gestational diabetes may double the risk of childhood obesity *(377)*. Pre-pregnancy overweight increases the risk of having a large-for-gestational-age infant by 53%, and obesity doubles the risk *(363)*. Pregnancy weight gain, in excess of recommendations, increases the risk of macrosomia and large-for-gestational-age *(378)*. Gestational weight gain and maternal over-nutrition during pregnancy prime the fetus for an increased lifetime risk of obesity *(379, 380)* because maternal excess weight may alter hypothalamic control mechanisms that regulate body weight, pancreatic endocrine functions and metabolic programming. Overeating during pregnancy may also be associated with the emotional reasons of "eating for the two", and consequently lead to maternal overweight and associated ill effects on the fetus. There is now also evidence for paternal adiposity influencing offspring adiposity *(381)*.

Preconception and pregnancy nutrition in relation to child obesity

An extensive literature relates size and maturity at birth to childhood BMI and adiposity. In general, larger size and higher adiposity at birth are associated with higher child adiposity and higher risk of child obesity. Preconception maternal obesity, and excess gestational weight gain are independently associated with adiposity in children and adolescents *(382, 383)*. Several innovative studies comparing siblings have shown that such effects are independent of shared genetic factors *(384)*, leading to the conclusion that prenatal exposure to an obesogenic environment has persistent effects, increasing risk of obesity in childhood. Other factors, such as parity, interact with maternal weight status influences. There is very little specific evidence of longer-term effects of maternal micronutrient status, prior to and during pregnancy, on obesity in the offspring. Studies in Pune, India found that in children aged 6 years, adiposity was directly related to maternal folate, while low maternal vitamin B_{12} status predicted higher insulin resistance *(368)*. Follow-up of offspring whose mothers participated in micronutrient supplement trials is discussed below.

While maternal nutritional status is one of the most important determinants on fetal development, other factors are influential; these include toxic exposures from maternal smoking *(385)*, environmental chemicals, *(386, 387)* severe stress *(388–390)*, young maternal age, parity, and birth spacing. A recent meta-analysis showed that maternal smoking during pregnancy was associated with higher childhood obesity (OR 1.6; 95% CI: 1.37–1.88) compared with paternal smoking (OR 1.23; 95% CI: 1.10–1.38) *(391)*. Higher effect estimates with maternal smoking during pregnancy may indicate intrauterine effects on the fetus. The interaction, especially in low- and middle-income countries, of endemic and episodic infection is a potent work modulator of fetal development, but the information in this area is particularly inadequate. Maternal infections such as malaria *(392)* or genitourinary infections *(393, 394)* may alter developmental trajectory in early pregnancy and birth outcomes. Infections act via inflammation and micronutrient disturbances, and measurable effects on the offspring include intrauterine growth restriction and prematurity, both of which have negative implications for the health of the offspring in the short and longer term. Fetal insults from pre-eclampsia predispose individuals to an increased risk of metabolic diseases including obesity in

adulthood *(395)*. High maternal load, with determinants of fetal harm from negative energy balance and stress also contributes to developmentally induced risk of ill health in offspring, in both the short and longer term. Evidence suggests that a short birth interval is causally associated with malnutrition in mothers, particularly among those living in low- and middle-income countries *(396)*. Interpregnancy intervals of less than 6 months are associated with a higher incidence of maternal anaemia and stillbirth, whereas intervals of greater than 60 months are associated with an increased risk of pre-eclampsia *(396)*. Furthermore, pregnancy in adolescence is another risk factor, since adolescent girls are not yet physically mature and may lack the knowledge, skills and support they need to support a healthy pregnancy *(397)*. Adolescent pregnancy is associated with increased risk of stillbirth, neonatal death, preterm birth, low-birth-weight and asphyxia *(398)*. There are also some reports that first-born children have, as adults, a greater fat mass in relation to maternal BMI than subsequent children, and that risk of metabolic disease is higher in preterm infants and in multiple pregnancies.

Implications for interventions

Given the importance of maternal preconceptional nutritional status at both ends of the distribution (inadequacies and excesses), in addition to nutrition during pregnancy, interventions need to be aimed at optimizing early linear growth and body composition and micronutrient status in young girls so that they enter the childbearing years in good health. All women of childbearing age (and their partners) need to have optimal weight and micronutrient status. Interventions should aim to eliminate micronutrient deficiencies, ensure adequate energy and protein intakes, promote weight loss for those who are overweight or obese, and optimize weight gain during pregnancy. As the vast majority of developmental modulation by undernutrition and infection occurs in low- and middle-income countries, there is also the need to focus on controlling infections in these settings, especially during childbearing years in women. Smoking, excessive alcohol consumption and lack of physical activity and sedentary lifestyle need to be discouraged. In all societies these issues relate to improving a sense of empowerment and self-efficacy, particularly in girls and young women. The key issues were raised in a WHO report in 2006 *(399)* and included: access to continued secondary education after marriage/childbirth; delaying first pregnancy until at least four years after menarche; diet before and during pregnancy and suckling; reduction of physical exercise levels in pregnancy; and smoking cessation. Recognizing that some of these measures are not delivered effectively through formal education alone, and that in some societies school attendance of young girls is limited after marriage, underscores the need for holistic interventions at a societal level. These also require environmental interventions which are possible through multisectoral collaboration only and involve a range of actors.

The evidence base

Interventions to prevent or correct undernutrition (prevent stunting, eliminate underweight and micronutrient deficiencies)

Numerous nutrition supplementation trials, primarily in low- and middle-income countries or at-risk populations in high-income countries, have aimed to improve micronutrient status as well as increase birth weight, lower rates of preterm birth, low-birth-weight and small-for-gestational-age births, and/or reduce neonatal mortality. As yet there are no published studies designed specifically

to assess whether nutritional supplementation of undernourished women will prevent later development of offspring obesity, but several trials with this aim have been initiated in India *(400)*. Thus, evidence from studies of birth outcomes is needed to allow inference of *potential benefits* for reducing later obesity. Interventions to improve maternal nutrition will prevent, or correct, the conditions that lead to fetal priming of childhood obesity risk, and to size and body composition at birth. These are related to later body composition, patterns of fat deposition, appetite and food preference, metabolic control, and risk of overweight and obesity.

Results of supplementation trials are often based on an estimation of main or group average effects. An important issue to consider is whether interventions have different effects depending on the nutritional status of the recipients. For example, benefits are expected to be greatest among those most deficient or at risk.

Preconception

Since maternal pre-pregnancy underweight, anaemia and other micronutrient deficiencies have been associated with increased risk of preterm birth, small-for-gestational-age and low birth weight *(364, 401–403)*, many interventions have targeted these nutritional problems. Interventions that optimize women's health before pregnancy, with the intent to improve maternal and newborn health outcomes, are collectively termed "preconception care". While preconception folic acid supplementation is effective in preventing recurrent neural tube defects, folic acid alone, or in combination with multiple micronutrient supplements, may not have any impact on preterm birth or low-birth-weight *(364)*. In contrast, a recent study in China found that multiple micronutrient supplementation from 3 months before pregnancy, through the first trimester, lowered rates of low-birth-weight *(404)*. Nutrition-sensitive interventions can be effective in preventing adolescent pregnancy and promoting birth spacing. Increasing correct and consistent use of effective contraception can prevent first pregnancy in adolescence by 15% and repeat adolescent pregnancy by 37% *(364, 405)*.

Overall the evidence suggests that women who receive preconception care and counselling are likely to develop positive health behaviours, such as daily pre-pregnancy multivitamin consumption, early entry into prenatal care, and cessation of alcohol use *(406)*. Community or health-care facility-based preconception care, including educating women about pregnancy and child care by building women's support groups, has shown improved behavioural outcomes, such as smoking cessation; increased use of folic acid; breastfeeding; greater odds of obtaining antenatal care; and lower rates of neonatal mortality *(364)*.

Periconception and pregnancy

Maternal nutrition supplementation trials have used high protein, balanced protein and energy, energy alone, single micronutrients, and multiple micronutrients. A recent collection of systematic reviews and meta-analyses evaluated the effects of a wide range of maternal periconception (up to 12 weeks' gestation) *(360),* and pregnancy interventions on birth outcomes *(407)*. In most cases, the evidence for effects was evaluated as weak and inconsistent.

Protein and energy: A review summarizing the effects of trials of dietary counselling to increase energy and protein intakes, balanced energy and protein supplementation (protein < 25% of kcal), high protein, and isocaloric protein supplements found no significant effects of dietary advice on birth weight, birth length or small-for-gestational-age; increased mean birth weight (41 g) and reduction of small-for-gestational-age (11%) with balanced protein-energy supplements; no effects of isocaloric supplementation on birth weight; and an increased risk of small-for-gestational-age with high protein *(408)*. In contrast, another review of 16 studies of balanced protein/energy reported a mean birth weight effect of 73 g, with greater benefits (100 g) in malnourished women; along with a 32% reduction in low-birth-weight, and 34% reduction in small-for-gestational-age *(409)*. The literature representing the impact of energy/protein supplementation more consistently shows small effects on birth weight within the range of maternal BMI, above the severely wasted levels associated with famine (BMI < 16 kg/m^2). This reflects the mechanisms at play to make available to the fetus, all nutrients held in store by an adult mother who is not herself growing. In cases of severe maternal undernutrition, protein energy supplements may have a greater impact *(408, 409)*.

Improving diet: A recent report of a large trial in India to improve women's diet quality during the pre-conception period and during pregnancy, found no main effect on infant birth weight, but suggested that early initiation of supplementation (more than 90 days before conception) had an effect in women who were not underweight *(400)*.

Micronutrients: Numerous studies report modest effects of single or multiple micronutrients on birth weight, risk of preterm birth, low-birth-weight and small-for-gestational-age. Systematic reviews of supplementation trials provide the following evidence. While highly effective for reducing maternal anaemia, iron supplements have inconsistent effects on risk of preterm birth and birth weight *(410, 411)*. Folic acid during pregnancy has no effect on preterm birth, but results in increased birth weight *(412)*. Zinc supplementation modestly reduces risk of preterm birth, but has no effect on birth weight *(413)*. Vitamin D reduces low-birth-weight, but not preterm birth *(414–416)*. Vitamin B6 supplementation modestly improves birth weight, but not risk of preterm birth or low-birth-weight, and supplemental vitamin C or vitamin E has no effect on birth outcomes *(417)*. Similarly, vitamin A does not influence birth weight or risk of preterm birth *(418)*. Compared with iron plus folic acid, multiple micronutrient supplementation reduces incidence of low-birth-weight by 14%, reduces small-for-gestational-age by 17%, and increases mean-birth-weight by about 53 g, but results in no difference in the overall risk of preterm birth *(419)*. Findings such as these have led to the recommendation to replace routine iron-folate supplementation with multiple micronutrients *(420)*. The use of fortified foods and beverages, especially those containing milk and essential fatty acids, in addition to multiple micronutrients has been estimated to improve birth weight by 60–73 g *(91, 421)*. There is weak and inconsistent evidence that supplementation with polyunsaturated fatty acids increases birth weight *(422)*.

Despite rich data on the theoretical importance of improving maternal nutrition to optimize birth outcomes, and the short-term success of nutrition interventions on birth outcomes, there has been very little follow-up of the longer term consequences of supplement trials for child obesity and related metabolic disease risks. Hawkesworth *(423)* reviewed the minimal evidence from offspring whose mothers participated in nutrition supplementation trials during pregnancy. Provision of protein-energy supplements to pregnant Guatemalan women and young children resulted in

increased height, weight, and fat-free mass among adolescent girls; however it was not possible to clearly distinguish the effects of prenatal versus postnatal supplementation. Protein-energy supplements provided to Gambian women increased birth weight by a mean of 136 g (and by 201 g for births during the "hungry" season), but despite these improvements in birth weight, there were no long-term effects on body fat, or fasting glucose, or insulin in the offspring. Follow-up of several micronutrient supplementation trials in Nepal found no effects of folic acid supplementation during pregnancy on haemoglobin A1c at age 6–8, while at age 2–3, children whose mothers received multiple micronutrients versus iron-folate had slightly higher triceps skinfold thicknesses. In a follow-up of > 3300 children aged 6–8 years whose mothers received folic acid, iron, and zinc supplements, the children were taller (0.64 cm; 95% CI: 0.04, 1.25), and had lower mean triceps skinfold thickness (-0.25 mm; 95% CI: -0.44, -0.06), subscapular skinfold thickness (-0.20 mm; 95% CI: -0.33, -0.06), and arm fat area (-0.18 cm(2); -0.34, -0.01). Offspring of supplemented mothers were not different in mean weight or BMI z-scores, waist circumference, or arm muscle area. Other types of micronutrient combinations, including a multiple micronutrient formulation, failed to show a growth benefit *(424)*. Another study in Peru found maternal zinc supplements during pregnancy were related to greater lean tissue in infants aged 4–12 months *(425)*.

Interventions to reduce maternal overweight and obesity, excess pregnancy weight gain and gestational diabetes mellitus

Owing to the substantial evidence linking maternal pre-pregnancy overweight and obesity, and excess gestational weight gain to obesity-related infant outcomes (higher birth weight, large size for gestational age, macrosomia, and increased risk of poor glucose control), numerous clinical trials have aimed to reduce pre-pregnancy weight or limit pregnancy weight gain as a means to improve pregnancy outcomes. Studies focus disproportionately on women in high-income countries. Types of interventions include: education/advice for lifestyle modification; dietary interventions (e.g. reduction of total energy intake, consumption of low glycaemic index diets); or physical activity/exercise interventions.

Clinical trials of interventions to reduce maternal weight gain have had limited impact on the incidence of gestational diabetes *(426)*. For example, in a cluster-randomized trial which randomized euglycaemic women with risk factors for gestational diabetes mellitus to intervention with intensive counselling on diet, physical activity and weight gain – versus a control group with no intervention – found no difference in incidence of gestational diabetes mellitus, although it was noted that the intervention group had lower birth weight and proportion of large-for-gestational-age, and the mothers had better diet and physical activity levels *(427)*. Nevertheless, meta-analyses have found that lifestyle intervention for overweight and obese pregnant women, including dietary counselling, physical activity or behavioural counselling, is associated with reduced pregnancy weight gain, although does not appear to have a significant effect on birth weight, macrosomia or large-for-gestational-age *(428)*. Likewise, a systematic review and meta-analysis of studies found that treatment of gestational diabetes mellitus results in less pre-eclampsia, shoulder dystocia and macrosomia, but has no effect on neonatal hypoglycaemia or future metabolic outcome *(429)*. However, most of the current published studies do not provide data on outcomes of the greatest clinical importance, such as future childhood obesity, and there is a paucity of studies addressing

childhood obesity specifically as the outcome of interest. It is also important to note that the impact of interventions during pregnancy on offspring adiposity may not be apparent until later in childhood. For example, it has been noted that macrosomic infants of diabetic mothers show "catch down" growth during infancy, only to show "catch up" at the time of adiposity rebound, and subsequently become obese *(430)*. In this context, although intensive treatment of gestational diabetes mellitus in the ACHOIS trial was associated with reduced macrosomia, no effect on offspring overweight and obesity was seen at 5 years of age *(431)*. In another large cohort, greater adherence to healthy eating guidelines was associated with reduced incidence of gestational diabetes mellitus *(432)*.

Overall, the studies demonstrate modest success, mostly in lowering pregnancy weight gain. The studies are focused primarily on pregnancy complications, birth outcomes and maternal health, and none were designed specifically for prevention of child obesity. In addition, there is a lack of studies in low- and middle-income countries where rates of maternal obesity and gestational diabetes mellitus are increasing rapidly. Nonetheless, the observational cohort studies of maternal obesity, excess gestational weight gain and poor glycaemic control during pregnancy, suggest that interventions to reduce maternal obesity and limit gestational weight gain will be effective in prevention of child obesity. Furthermore, many interventions were initiated during pregnancy, and measures such as reducing maternal obesity are likely to be more effective if initiated before pregnancy. Some have also suggested a need for greater awareness for clinicians to screen for gestational diabetes mellitus. Indeed, in low- and middle-income countries, where rates of gestational diabetes mellitus are quite high and increasing, there is often a lack of screening for gestational diabetes mellitus. However, a very recent review challenges the view that exposure to maternal hyperglycaemia increases risk of obesity and diabetes in the offspring, suggesting that the increased risk of obesity in the offspring of women with gestational diabetes mellitus or type 2 diabetes can be explained solely by their high BMI *(433)*.

Non-nutrition interventions

Other interventions have important implications for fetal health and subsequent obesity risk *(434)*. Maternal smoking during pregnancy is associated with lower birth weight and greater risk of preterm birth, but a 50% increase in risk of child obesity *(385, 391, 435)*. Preconception smoking cessation interventions may therefore be effective: Oken et al. *(385)* estimated that "in the US, where 11% of pregnant women currently smoke and child overweight is highly prevalent, about 715 000 US children may be overweight because their mothers smoked".

Toxic exposures during pregnancy can influence infant/child obesity risk. Particular concern in this area relates to toxic pollutants, exposure to smoke from indoor cooking, and endocrine disruptor chemicals. In some instances endocrine disruptor chemicals may act via effects on epigenetic control systems regulating endocrine function *(436)*, and this is an area of intense research and debate. Prenatal exposure to compounds such as bisphenol A can produce effects on offspring adiposity and cardiometabolic control in both animals and humans *(437, 438)*. As the effects of an endocrine disruptor chemical in isolation can be subtle, and since even low environmental levels of several endocrine disruptor chemicals can act together to affect development, more research on their long-term health effects is needed. A recent report from the Royal College of Obstetricians and

Gynaecologists on possible effects during pregnancy *(439)* seems to demonstrate the importance of the precautionary principle in this regard, but emphasizes the need for more evidence.

Delaying first birth and lengthening pregnancy intervals may also improve nutritional status at conception and reduce risk of intrauterine growth restriction *(440, 441)*. Recent initiatives, including the Bill and Melinda Gates Foundation's "Start with a Girl: A New Agenda for Global Health"*(442)*, and the Center for Global Development's "Girls Count: A Global Investment and Action Agenda" *(443)*, have focused on young girls as an important target for improving long-term offspring health. This emphasis is also important in light of the sharp increases in obesity in post-menarcheal girls in low- and middle-income countries *(444)*, putting young women at risk for pregnancies complicated by obesity as well as micronutrient deficiencies. Although the data on maternal stress is not consistent, it seems probable that reducing lifestyle stressors would be of benefit to an intervention package contributing to childhood obesity factors such as low support for breastfeeding and disrupted infant sleep patterns.

Major challenges and gaps in knowledge

- While there is substantial observational evidence from cohort studies showing associations of preconception and pregnancy nutritional status with birth outcomes, the evidence from well-designed randomized-controlled trials is weaker. In the absence of randomized-controlled trials, innovative observational designs may be alternative ways to generate more robust evidence.
- Preconception nutrition and health status greatly affects the pregnancy outcome. It is also necessary to identify the window before conception, when interventions are likely to be most effective.
- The relative impacts and efficiency of pre-, peri- and post-conception interventions (nutrition, micronutrients) need to be evaluated.
- Many studies focus on main effects, and in so doing, may underestimate benefits of interventions for the most at-risk women. Differential effects of interventions on the most nutritionally at-risk, or otherwise vulnerable, women need to be better evaluated.
- There is a serious lack of any quantification of effects of interventions on child obesity. Not enough is known about the relative importance of prenatal versus postnatal effects. Studies with follow-up into childhood, face the challenge of accounting for differential effects of postnatal environments, and of attributing outcomes to genetics, epigenetics, and environmental exposures.
- Awareness of the importance of preconception health is poor in both women and health-care providers *(445)*. This is especially true for at-risk groups such as teenage pregnancies and low socioeconomic status sections of the population, migrants etc.
- The recent neonatal series in *The Lancet* indicates that up to 50% of all newborn infants in south Asia might be small for date (i.e. not achieving their full intrauterine growth potential); this has serious implications and needs further elucidation on the definitions of small-for-gestational-age and related issues of prevention and interventions for mitigating the ill

effects including potential for childhood obesity. The issue is particularly important for low- and middle-income countries.

- Of high relevance for low-income countries are interventions to mitigate the influence of poor environmental hygiene and sanitation on fetal health, priming and outcomes.

Implications for future work

There is sufficient evidence to argue for a new initiative to promote health in girls and women before conception and throughout pregnancy. A holistic approach is needed. The major issues reviewed are summarized in Table 6.

The major elements should be:

1. Prevent malnutrition and promote awareness of healthy balanced nutrition across the spectrum from under- to overnutrition, to include macro- as well as micronutrients. This should be done before conception, through pregnancy and between pregnancies and should extend to partners.
2. Optimize body weight and composition and promote awareness of balance across the spectrum from under- to overweight. This should make allowance for ethnic differences in healthy BMI and pattern of fat deposition. Again this should be done before conception, through pregnancy and between pregnancies and should extend to partners.
3. Discourage smoking and reduce exposure to pollutants, environmental chemicals and toxins.
4. Integrate the initiative with others aimed at empowering young women and adolescents, promotion of education and health literacy through schools and wider society and, where appropriate, to those aimed at sexual health and communicable disease prevention.
5. Infection control, especially in low-income settings, can make an important contribution.
6. Intervention studies are needed during the preconception period and pregnancy to prepare and empower prospective mothers for breastfeeding and feeding for optimal growth and nutrition of the infant, particularly to reduce low-birth-weight and to be pro-active on feeding with human milk, assessing long-term impact from the childhood obesity and chronic diseases perspective.
7. The challenge of improving preconception health is related to that of promoting adolescent health and health literacy. Investments in adolescent health have been less than that for younger members of the population in recent decades.[1] This issue is also related to life-course approach social inequalities in health (e.g. the review of social determinants and the health divide in the WHO European Region[2]).
8. Addressing these issues is central to initiatives such as WHO's Global Action Plan 2013–2020[3], of which one of the over-arching principles and approaches advocates a life-course approach starting at preconception.

[1] *The Lancet*. Editorial, Nov. 9 2013 Adolescence: a second chance to tackle inequities. Also *Lancet* series.
[2] http://www.instituteofhealthequity.org/projects/who-european-review.
[3] http://www.who.int/nmh/events/ncd_action_plan/en/.

Table 6. Summary of issues reviewed

PERICONCEPTION				
Insult	**Why important/ mechanisms**	**Consequence**	**Implications for intervention**	**Potential evidence-based actions**
Maternal short stature	Marker of maternal nutritional history and intergenerational stressors	IUGR, LBW in offspring	Promote optimal linear growth in infancy through adolescence	Optimal breastfeeding, high quality weaning foods, MN supplements, WASH to decrease morbidity
Maternal MN deficiencies	Epigenetic regulation of metabolic pathways (e.g. methyl donors) Antioxidants: prevent oxidative stress Placental function Components of regulatory proteins	Altered glucose metabolism; insulin resistance; altered body composition	Optimize preconception folate, B-vitamin and antioxidant intakes. Focus on adolescent nutrition	Multiple micronutrient fortification or supplementation
Maternal underweight	Signalling: Setting of growth trajectories Establishment of placental structure and function Limited maternal stores to support fetal growth	IUGR, LBW in offspring; long-term disruption of metabolism; favouring fat storage	Increase maternal BMI **prior** to pregnancy	Food-based: Promotion of high quality, nutritionally-adequate diets Supplemental foods
Maternal obesity	Epigenetic changes in embryo Increased risk of GDM, hypertensive disorders of pregnancy Maternal IR and elevated TG change macronutrients delivered to fetus	Fetal macrosomia; Increased risk of offspring obesity; T2D and CVD later in life	Reduce maternal BMI **prior** to pregnancy	Lifestyle interventions for young women: diet and physical activity

Maternal smoking	Hypoxia and toxic exposures	LBW, IUGR	Smoking cessation **prior** to pregnancy	Behavioural modification or medication-assisted smoking cessation
Toxic exposures	Endocrine disruptors, growth inhibitors, etc.	Depends on toxin: obesity, reproductive development	Environmental controls: clear air, clean water, safe foods	
Stress	Corticosteroid secretion may affect fetal growth	IUGR	Need for social support	Counselling, peer-support programmes
Infections	Elevated maternal inflammatory levels	Preterm birth	Screening and treatment	

PREGNANCY				
Insult	**Why important/ mechanisms**	**Consequence**	**Implications for intervention**	**Potential evidence-based actions**
Inadequate pregnancy weight gain	Signalling of maternal status to early embryo, inadequate delivery of nutrients to fetus	IUGR, LBW Impaired organ growth and development Impaired brain development	Antenatal care, with monitoring of PGW and its causes	Maternal protein/energy diet supplements during pregnancy
Excess pregnancy weight gain	Altered hormonal milieu Hyperinsulinemia/ hyperglycemia IGF pathways	Fetal macrosomia Increased risk of obesity, offspring DM	Antenatal care, with monitoring of PGW and its causes, glucose monitoring	Diet-based strategies can reduce PWG, chances of gains > IOM recommenations, infant BW and likelihood of LGA
Inadequate maternal MN intake	Impaired organ growth and development Similar to preconception Establishment of placental morphology and function, epigenetic regulation, antioxidants, etc.	Deficits in brain development Organ-specific deficits Epigenetic changes in IGF axis, leading to IR, dyslgycemia	In resource-poor settings, diet alone may not meet needs	Maternal multiple micronutrient supplementation
Inadequate macronutrient intake	inadequate supply of nutrients to fetus, specific regulatory roles of protein, EFAs etc.	IUGR Skeletal muscle deficits) organ-specific deficits	Address food insecurity and seasonal food deficits	Food supplements

CVD: cardiovascular disease; DM: diabetes mellitus; EFAs: essential fatty acids; GDM: gestational diabetes mellitus; IOM: Institute of Medicine; IUGR: intrauterine growth restriction; IR: insulin resistant: IGF: insulin-like growth factor; LBW: low birth weight; LGA: large for gestational age; MN: micronutrient; PGW: pregnancy weight gain; TG: triglycerides; T2D: type 2 diabetes.

6.2 Obesity prevention in infants, preschool children, school-age children and adolescents[1]

The goals of obesity prevention and treatment in children and adolescents are highlighted in Table 7 *(446)*. Interventions are necessary at all developmental stages. However, the approaches to achieving these goals, and the settings in which they can be implemented to have high reach and impact in populations, vary by developmental stage (e.g. whether the child, the parent, or both, are primary focal points) and according to circumstances (e.g. which settings and influences are targeted depends on patterns of child care, parental lifestyles, and community characteristics and broader environmental contexts).

Table 7. Goals of obesity prevention and treatment in children and adolescents

Individual children and adolescents	Population of children and adolescents
• A healthy weight trajectory • A healthy diet (quality and quantity) • Appropriate amounts and types of physical activity • Achievement of physical, psychosocial and cognitive growth and developmental goals • A healthy body image and the absence of potentially-adverse weight concern or restrictive eating behaviours • For those affected by obesity, a reduction in level of overweight, improvement in obesity-associated comorbidities, and improvement in risk factors for excess weight gain	• Reduction in the incidence of childhood and adolescent obesity • Reduction in the prevalence of childhood and adolescent obesity • Reduction of mean population BMI levels • Improvement in the proportion of children and adolescents meeting dietary guidelines • Improvement in the proportion of children and adolescents meeting physical activity recommendations • Reduction in health-care costs associated with obesity in children and adolescents • Achievement of physical, psychological and cognitive growth and developmental goals

Source: adapted from Institute of Medicine, USA, 2012 *(446)*

A broad range of interventions is required

Obesity prevention, and, more broadly, prevention of noncommunicable diseases, require actions to create (or maintain, if still present) environments and policies across the multiple levels and sectors that

[1] Authors: Shiriki Kumanyika, Louise Baur, Mark Tremblay, John Reilly, Anniza de Villiers.

influence feeding, eating, physical activity and other movement behaviours *(447–450)*. These interventions enable and work together with individually-focused interventions that shape child, peer group, parent, and other caregiver behaviours in ways that protect against excess weight gain or, where appropriate, promote and support weight loss.

This section provides an overview of current evidence for interventions to prevent and treat obesity in individuals or communities from infancy and early childhood to adolescence. More detailed consideration is given to early life interventions because this is where new guidance and approaches are needed, and also because interventions implemented from the earliest ages will have the greatest potential for cumulative benefits at later stages. The report focuses on interventions working close to the child or family – generally acting within the health sector, or in family, education or early childhood settings.

The evidence base

The evidence assessment relates potentially to the following questions that can be asked for each developmental stage, and separately for prevention and treatment:

1. What are the characteristics of interventions that have been evaluated?
2. What is the evidence that they can be effective under ideal conditions or under the circumstances in which they have been implemented?

Annex 1 provides a summary of issues that arise when considering the types of evidence used.

Interventions for the prevention of obesity

The most recently updated Cochrane review of childhood obesity prevention studies was based on a March 2010 search of published literature, published in 2011 and reflecting articles published in English or another language through 2009 *(59)*. Both randomized and non-randomized studies were included if a control or comparison condition was included, and if they related to any type of intervention, policy or programme of $\geq$ 12 weeks' duration. Included interventions may have used educational, health promotion, or behavioural counselling approaches that focused on changing energy balance behaviours (physical activity or dietary intake) to prevent *excess* weight gain or obesity. Cluster-randomized studies by units (e.g. if schools were the unit of randomization instead of children within schools) that had fewer than six units were excluded.

The evidence base identified, using these criteria, included 55 studies – still a relatively small database, although a stated increase by 36 studies from the prior, 2005 update *(451)*, and a more definitive review with respect to conclusions about effectiveness. Studies were primarily from high-income countries, but included studies from Brazil, Chile, Mexico and Thailand. Other studies and reviews consulted to

complement the evidence from the Cochrane review were also primarily from the USA, Europe or Australia and New Zealand.

The authors of the 2011 Cochrane review concluded that there was "strong evidence to support beneficial effects of child obesity prevention programmes on BMI, particularly for the 6–12-year age range for which there was the most evidence". To provide further evidence relevant to children younger than age 6, the following summary of age-specific interventions draws on several reviews and studies published since the end date of the Waters et al. Cochrane review. The age groups are those used by Waters et al., however the period from birth to age 2 years has been treated separately.

Infancy and very early childhood: ~Birth–2 years

Waters et al. *(59)* did not identify any childhood obesity prevention studies beginning at birth. The earliest age of intervention in the studies of the 0–5 year age range cited by these authors was a home visiting programme delivered to Native American parents by an indigenous peer educator *(452)*. The study, conducted in the northern USA and Quebec and Ontario, Canada, included children aged 9 months to 3 years who were walking. Further recent studies providing additional information on the effectiveness of childhood obesity prevention interventions during infancy are highlighted below:

- The Early Prevention of Obesity in Children (EPOCH) Collaboration *(453)* combines evidence from four separate trials in Australia and New Zealand that together comprise more than 2000 women and infant dyads beginning between birth and 6 months of age *(454–457)*. Based on a preliminary report of the pooled results (Askie et al. abstract *(453)*), the mean BMI z-score, at 18–24, was significantly lower in the intervention versus the control group: respectively, 0.67 (95% CI: 0.60–0.74) and 0.80 (95% CI: 0.73–0.87); estimated difference was -0.13 (95% CI: -0.23 to -0.03; *P*=0.012) as observed and -0.10 (95% CI: -0.20–0.00; *P*=0.04) using intention-to-treat (multiple imputation) analysis. For secondary outcomes, significant, favourable changes were observed for breastfeeding duration and childrens' television viewing, but not for overweight/obesity prevalence, sleeping patterns or physical activity.

- The Healthy Beginnings Study, conducted in Sydney, Australia, illustrates the approach taken in one of the EPOCH interventions that yielded statistically significant results at 2 years of age and which conducted further child follow up through to age 5 in the absence of any further intervention (see Box G). The loss of any detectable advantage of the early life intervention is noteworthy and could indicate that environmental context factors affecting children in the study communities at ages 2–5 may overwhelm any benefits for parent or child behaviour that might have carried over from the intervention period.

Box G. The Healthy Beginnings Trial: an Australian early childhood obesity prevention programme, with longer-term follow-up.

The Healthy Beginnings Trial was conducted in a socially disadvantaged region of south-western Sydney, Australia, with 667 first-time mothers (intervention: 337, control: 330). The aim was to investigate the effectiveness of a home-based early intervention primarily on children's BMI at age 2 and, secondarily, on dietary behaviours, television time and physical activity of both children and their mothers. The intervention comprised eight home visits from early childhood, nurses delivering a staged home-based intervention, one in the antenatal period, and seven at 1, 3, 5, 9, 12, 18 and 24 months after birth. The intervention was developmentally staged and focused on supporting breastfeeding and healthy infant feeding, promotion of a healthy family lifestyle, active play and a healthy dietary intake.

At age 2, the intervention led to a reduction in child BMI (0.29 kg/m^2 difference), increased vegetable consumption, and decreased television viewing time (458).

The intervention and control participants were subsequently followed up for three more years <u>without further intervention</u> (459). The differences in children's BMI and BMI z-score between intervention and control groups at 2 years disappeared over time, so that by age 5, no significant differences were detected. Similarly, no significant effects of the early intervention on television viewing, physical activity and diets were detected over time. Such data suggest that the effect of this early life home visiting intervention on child BMI may not be sustained without further intervention, and that a range of interventions acting at different developmental stages may be required.

- A community-partnered, cluster-randomized trial of a community intervention to prevent overweight in American-Indian toddlers (aged 0–24 months) is underway in the USA: The Prevention of Toddler Obesity and Teeth Health Study (PTOTS) *(460)*. This study will test a multicomponent intervention "to promote breastfeeding, reduce sugar-sweetened beverage consumption, appropriately time the introduction of healthy solid foods, and counsel parents to reduce sedentary lifestyles in their children". Results of a small feasibility and acceptability study compared community-plus family interventions with community-wide interventions alone and found suggestive evidence that adding the family component was associated with greater improvements in breastfeeding, reduction in sugar-beverage consumption and BMI z-scores at age 18–24 months *(461)*.

- The effect of breastfeeding as a childhood obesity prevention strategy has been of interest for decades, and answers have been sought through the Promotion of Breastfeeding Intervention Trial (PROBIT) *(462)*. This cluster-randomized experiment assigned a set of 15 maternity hospitals and associated clinics in Belarus to continue usual breastfeeding practices and policies and 16 to an experimental condition based on the WHO/UNICEF Baby Friendly Hospital guidelines in 1996–97. In

the most recent report from PROBIT, nearly 14 000 of the originally-enrolled 17 000+ mother/daughter pairs provided follow-up data when the children were 11.5 years old. Although the experimental intervention had clear documented effects of increasing breastfeeding duration, exclusive breastfeeding, and overall prevalence during the first year of life, a significant effect on obesity prevention was not observed at this or the prior (age 6.5 years) time point *(463)*, indicating that this intervention alone would not likely prevent childhood obesity.

- Analyses of the long-term health effects of the Carolina Abecedarian Project (ABC Project) in the USA suggest that obesity-preventive interventions in the first year of life may be favourable for health during the early adult years (mid-30s) *(464)*. This randomized social experiment was designed to prevent mental retardation in children from socially disadvantaged communities by intervening in a cohort of children living near Chapel Hill, North Carolina. Most of the observed health benefits were associated with the first stage of the intervention at ages 0–5 and involved a nutrition and health-care component. Males in the treated group were less likely than those in the control group to be overweight during their preschool year and the associated health benefits appeared to persist in later life. No such effects were observed for the females.

Early childhood: ~2–5 years

Seven (of eight identified) studies in the age range 0–5 years were included in the meta-analysis in the Waters et al. 2011 review. Most were conducted with children in the preschool through kindergarten age range, often with mean ages $\geq$ 4 years, and used behavioural strategies to address eating and/or movement behaviours in child-care or preschool settings. Parental or family involvement varied. Some included low-income or ethnic minority populations, or a mix of low- and high-socioeconomic status locations including Thailand *(465)*, the USA *(466–468)*, and Scotland *(469)*. Exceptions were the above-cited home visiting in American-Indian children in the USA and Canada *(452)* and a study in a primary health-care setting in Germany *(470)*. Parental or family involvement varied. Meta-analysis results suggest a positive trend of improvement in BMI/BMI z-scores (-0.26 BMI units (95% CI: -0.053–0.00) compared with controls; *P*=.05). The effect was larger when the home-based and health-care based studies were removed to decrease heterogeneity (-1.08, *P*=0.0001). The authors estimate that the -0.26 reduction would amount to an average 1.6% reduction in average BMI which, if sustained, would be important at a population level.

Waters et al. further examined these studies, comparing those conducted in, or outside of, educational settings, with inclusion of the eighth study that was not in the meta-analysis *(471)*. They observed that effects were greater outside of educational settings (e.g. in home or health-care settings), perhaps due to greater parental involvement in these settings and that effects were observed more consistently for children from less advantaged backgrounds. Of possible relevance is a pilot and feasibility effectiveness trial of a school-based intervention for Latino children aged 2–5 years in Chicago which reported low attendance at parent sessions and a lack of clear benefit with respect to BMI at 1-year follow-up *(472)*.

Pre-adolescent children: ~6–12 years

The 2011 Cochrane review by Waters et al. *(59)* identified 39 intervention studies targeting the 6–9 year age range – many more than for those identified for younger (*n*=8) or older children (*n*=8) – and found the evidence to support interventions for this age range to be the strongest. Twenty-seven of the studies gave BMI or BMI z-score data that permitted inclusion in the meta-analysis, yielding an effect size of -0.15 (95% CI: -0.23 to -0.08, equivalent to an estimated reduction in average BMI in a population of this age by 0.8%). This effect size was not increased significantly by any exclusions used to reduce apparent heterogeneity. The majority of studies were conducted in educational settings (see Box H for the approaches used). The interventions used various approaches and no conclusions about the best approach could be drawn. However, in addition, the authors pointed out that these types of interventions – i.e. targeting only physical activity and dietary behaviours – address only some of the relevant influences and mention the potential need to address advertising, environmental factors and government or school policies.

Adolescents: ~13–18 years

Waters et al. *(59)* identified 8 studies for this age group and included 6 in the meta-analysis. The effect size was -0.09 BMI units (95% CI: -0.20–0.03), equivalent to an estimated 0.4% reduction in average BMI for a population in this age range. The non-significant effect size, together with the heterogeneity in the studies, precluded any firm conclusions about effectiveness of interventions in these populations, although there was a promising trend of a smaller increase in adiposity over time in the intervention group children.

Box H. Planet Health: a USA school-based intervention to prevent obesity and reduce obesity-conducive behaviours

Planet Health is a school-based intervention in the USA which is designed to reduce obesity in middle-school youth (grades 6–8). It was designed to reduce obesity by increasing energy expenditure while promoting key dietary behaviours consistent with dietary guidelines. The intervention focuses on four behavioural changes: reducing television viewing to less than 2 hours per day; increasing moderate and vigorous physical activity; decreasing consumption of high-fat foods; and increasing consumption of fruits and vegetables to 5 a day or more.

A distinctive aspect of Planet Health is the interdisciplinary curriculum approach, with intervention material infused into major subject areas and physical education, using grade- and subject-appropriate skills and competencies. This approach is designed to enhance efficiency by using classroom teachers, with minimal health education training, to implement the materials, and to enhance effectiveness by involving multiple classes, which often use different approaches to learning.

The initial Planet Health trial commenced in 1995 in Massachusetts and was conducted in 10 schools (5 intervention schools) over a 2-year period. In intervention schools there was a reduction in prevalence of obesity among girls compared with controls controlling for baseline obesity (odds ratio, 0.47; 95% CI: 0.24–0.93; P=0.03), with no differences found among boys. There was greater remission of obesity among intervention girls versus control girls (odds ratio, 2.16; 95% CI: 1.07–4.35; P=0.04). The intervention reduced television hours among both girls and boys, and increased fruit and vegetable consumption, and resulted in a smaller increment in total energy intake among girls. Reductions in television viewing predicted obesity change and mediated the intervention effect (473).

The Planet Health programme was shown to be cost-effective and cost-saving as implemented (474). At an intervention cost of US$ 14 per student, per year, it was estimated that the programme would prevent ~1.9% of the female students from becoming overweight adults. As a result, an estimated 4.1 quality-adjusted life years (QALYs) would be saved by the programme, and society could expect to save an estimated US$ 15 887 in medical care costs and US$ 25 104 in loss of productivity costs. These findings translated to a cost of US$ 4305 per QALY saved and a net saving of US$ 7313 to society. Results remained cost-effective under all scenarios considered, and remained cost-saving under most scenarios.

The Planet Health programme has also been shown to improve self-reported disordered weight-control behaviours in girls in both the short term (475) and at 3-year follow-up when implemented at scale (476).

Studies in community settings

Many of the studies in the 2011 Cochrane update were conducted in school settings. Bleich et al. reviewed studies conducted in community settings using methods recommended by the US Agency for Healthcare Research and Quality *(477)*. The review covered English-language published studies through to 11 August, 2012 and was limited to randomized-controlled trials, quasi-experimental, or natural experiments conducted in high-income countries which were designed to prevent obesity or excessive weight gain in 2–18-year-old children with follow-up of at least 1 year. Nine eligible studies were identified, of which 6 were conducted in the USA *(478–483)* and 1 each in Australia *(484)*, the Netherlands *(485)*, and Switzerland *(486)*. Key features of the approaches used in these studies are shown in the excerpted table below.

As shown, only one of the interventions was at the community level only. All of the others involved at least one other setting and two involved four different settings. A desirable intervention effect was reported for the studies by Chomitz, de Sliva-Sanigorski, Economos, and Sallis *(479, 480, 483, 484)*, all of which focused on a combination of diet and activity changes. The authors concluded that there is moderately strong evidence that inclusion of a school component in a community-based intervention is effective for prevention of child overweight and obesity, in that 2 of the 3 studies that involved schools

found a statistically significant benefit. Evidence was insufficient to draw conclusions about the other approaches.

TABLE 1 Characteristics of the Included Studies Testing the Effect of Community-Based Childhood Obesity Prevention Programs (n = 9)

First Author, Year, Country	Setting(s)	RCT	Sample Size	Sample Age (Range or Mean y)	Girls (%)	Intervention Time: Elements Included in the Intervention	Follow-up Period[a] (mo)
Chang, 2010, United States[30]	Community, school, primary care, and child-care components	No	4595	2–17	NR	24 mo: social marketing, strategic partnerships, knowledge mobilization, strategies in multiple sectors	NR
Chomitz, 2010, United States[24]	Community, school	No	1858	8	48	36 mo: city policies, community awareness campaigns, physical education enhancements, food service reforms, farm-to-school-to-home programs, family outreach, BMI and fitness reports	36
de Silva-Sanigorski, 2010, Australia[31]	Community, home, primary care, child care	No	43 811	2–3.5	49	48 mo: community capacity building and environmental changes to increase healthy eating and active play	48
Economos, 2007, United States[29]	Community, school, home	No	1178	7	NR	10 mo: physical activity options and availability of healthy foods before, during, and after school; social marketing; family outreach and engagement	36
Eiholzer, 2010, Switzerland[23]	Community only	Yes	46	13	0	4 mo: resistance exercise program	12
Klesges, 2010[28]	Community, home	Yes	303	9	100	20 mo: group behavioral counseling (obesity prevention program) or self-esteem and social efficacy (alternative intervention)	24
Robinson, 2010, United States[27]	Community, home	Yes	261	9	100	24 mo: dance classes and reduced screen use; health education	24
Sallis, 2003, United States[25]	Community, school	Yes	24 schools (mean enroll-ment: 1109)	NR	49	24 mo: physical education, physical promotion throughout school day, changes to school food service, social marketing	24
Singh, 2009, Netherlands[26]	Community, school	Yes	1108	13	53	8 mo: education in biology and physical activity, environmental change options for schools (physical education classes, changes to school cafeteria)	20

The sample size of each study represents the number of children included in the analysis with both baseline and follow-up data available. NR, not reported.
[a] From start of intervention

Source: Bleich et al. *(477)*

An example of community-based interventions being implemented in several countries in shown in Box I below.

Box I. EPODE (Ensemble Prévenons l'Obesité des Enfants – Together Let's Prevent Childhood Obesity)

EPODE is the world's largest obesity prevention network with 25 programmes in 15 countries reaching 150 million people. The mission of the EPODE International Network (http://www.epode-international-network.com/) is to reduce childhood obesity prevalence through sustainable strategies based on community-based programmes. EPODE employs a coordinated, capacity-building approach for communities to reduce the prevalence of childhood obesity by delivering programmes on a local level that create everyday norms and settings for children to enjoy healthy eating, active play and recreation. The EPODE philosophy includes no stigmatization of people, behaviour or food; a positive step-by-step approach focused on pleasure of eating and exercise; consideration of modern eating and physical activity patterns; and strengthening the community spirit of sharing, bonding and social cohesion. It focuses on changing social norms through integrated efforts from the four pillars of the EPODE methodology: 1) a multistakeholder approach 2) social marketing 3) scientific evaluation and a scientific evaluation board and 4) public–private partnerships (487) .

Evidence of the effectiveness of the EPODE approach originated from a repeated, cross-sectional, school-based survey conducted on schoolchildren aged 5–12 years from two intervention towns (Fleurbaix and Laventie [FL]) and two comparison towns (Bois-Grenier and Violaines) in northern France (488). After an

initial increase, trends in mean BMI and prevalence of overweight started to reverse. Compared with 2002, the age-adjusted odds ratio for overweight in FL was significantly lower in 2003 and 2004 (but for girls only). In the 2004 school year, the overweight prevalence was significantly lower in FL (8.8%) than in the comparison towns (17.8%, P< 0.0001). The authors concluded that over a long period of time, interventions targeting a variety of population groups can have synergistic effects on overweight prevalence. Since the original study, the EPODE approach has been implemented in many other countries with emerging evidence of success (487, 489). The EPODE movement provides hope that, with political commitment, a strategy of whole-of-community engagement, coordinated efforts and careful evaluation, it is possible to reverse trends towards increasing overweight by actions at the community level.

Summary of prevention studies

Here, the most detailed consideration has been given to evidence relating to interventions in the birth to 2-year age range. This age group has not been covered by existing systematic reviews because most of the evidence was published after the last Cochrane review update. Of necessity, impressions to date are, therefore, based on a limited search for, and review of, relevant studies, of which some are still in progress. With this caveat, the conclusions are that interventions that begin at or within a few months of birth and that include but are not limited to breastfeeding promotion can benefit obesity prevention in the first 1 or 2 years of life. Some of these interventions have included families of children in socially disadvantaged communities. The elements included in the Healthy Beginnings study (Case example 1) illustrate how these interventions may be approached. The Healthy Beginnings follow-up data suggest that any subsequent benefits, e.g. after age 2, may depend on whether age-appropriate interventions are provided during the ensuing years. As long as the environmental contexts for appropriate energy-balance behaviours continue to be heavily weighted toward excess energy consumption and inadequate or inappropriate movement behaviours (see subsection below on movement behaviours), children may need to be exposed to a coherent sequence of age-appropriate interventions in order to achieve and maintain a healthy weight.

Conclusions about children older than 2 years are taken from the 2011 Cochrane update. Evidence relevant to children aged 2–5 years suggests that interventions in child-care or school settings can be effective, but there is a need for greater clarity on the most effective approaches in specific settings and contexts, including socioeconomic contexts, and more studies overall on which to base conclusions. The evidence supporting interventions in children aged 6–12 years is strong and the trends observed in the younger and older children are promising. Most of the relevant interventions have been conducted in school settings although for children aged 2–5 years, home and health-care settings might allow for more parental involvement and larger effect sizes.

Guidance for preventive interventions

The authors of the 2011 Cochrane review on obesity prevention noted that there was some evidence for positive outcomes in disadvantaged populations, no evidence of widened health inequalities, and no evidence that these interventions were associated with adverse effects. They concluded that no further studies are needed of short-term interventions for children aged 6–12 years, but suggested that future research should focus on longer-term sustainability of interventions. Recommendations on which specific interventions to implement more widely, based on current evidence, were based on components that appear to be associated with beneficial impacts, although the authors could not distinguish among them, as follows (verbatim from p. 35 of Waters et al. *(59)*):

- Curriculum on healthy eating, physical activity and body image integrated into regular curriculum;
- More sessions for physical activity and the development of fundamental movement skills throughout the school week;
- Improved nutritional quality of foods made available to students;
- Creating an environment and culture that support children eating nutritious foods and being active throughout each day;
- Providing support for teachers and other staff to implement health promotion strategies and activities (e.g. professional development, capacity building activities);
- Engaging with parents to support activities in the home setting to encourage children to be more active, eat more nutritious foods and spend less time in screen-based activities

Annex 2 discusses two of the frameworks that can be used when considering approaches to interventions.

Interventions influencing movement behaviours

Generally, comprehensive intervention studies that target both healthy eating and physical activity are effective for both the prevention *(477, 490)* and treatment *(491)* of childhood obesity *(492)*. There is also evidence that exercise alone can be an effective treatment *(493)*. Intuitive, cross-cultural *(494)* and secular trend *(495)* evidence provides compelling support for the need to preserve and promote habitual healthy movement behaviours as part of a comprehensive obesity prevention strategy and for health promotion more generally.

Historically, the benefits of physical activity (e.g. moderate-to-vigorous physical activity) to overall health have dominated discussions. However, emerging evidence suggests that a more integrated conceptualization of all movement-related behaviours (e.g. moderate-to-vigorous physical activity, light/incidental physical activity, sedentary behaviours, sleep) is needed to achieve population energy

balance and recalibrate the global movement behaviour frame of reference *(496, 497)*. Published guidelines for children and youth around the world focus on moderate-to-vigorous physical activity despite an accumulating body of evidence showing that light-intensity physical activity *(498)*, sedentary behaviour *(499–502)* and adequate sleep *(503)* are important independent predictors of health indicators (including obesity) and that these behaviours moderate the health impact of each other. Ignoring the totality of the movement continuum while focusing efforts exclusively on moderate-to-vigorous physical activity (accounting for < 5% of a 24-hour period), limits the potential to optimize the health benefits of movement behaviours and disregards fundamental cultural differences in countries at different stages of the physical activity transition *(494, 504)*. A global matrix of movement behaviours comparing 15 countries from 5 continents demonstrated the importance of unstructured, lifestyle-embedded activity (e.g. active play, active transportation, chores) in achieving recommended levels of physical activity and sedentary behaviours *(494)*. Population-based approaches targeting all movement behaviours, especially unstructured movement, are infrastructure-light and scalable and may have greater resonance among low- and middle-income countries as obesity prevention strategies.

Related to this, time spent outdoors is associated with increased physical activity *(505)*, and daylight savings/summer-time is a practical, population-based approach to increasing rates of physical activity *(506)*. Ironically, there is emerging evidence that active video games (AVGs) may serve as an effective intervention or adjunct for the promotion of healthier body weights among adolescents with obesity *(507)* but may not be effective in the prevention of obesity or in the promotion of physical activity among the general population *(508)*.

A comprehensive childhood obesity prevention or treatment intervention should promote and facilitate moderate-to-vigorous physical activity, light/incidental physical activity, outdoor time, and good sleep hygiene, while discouraging extended sedentary behaviours. For children and adolescents affected by obesity, AVGs may be a stepping stone to greater engagement in the activities listed above.

Boxes J and K highlight two interventions in preschool settings in Europe which particularly target movement behaviours.

Box J. The ToyBox Study: a pan-European childhood obesity prevention programme.

The ToyBox Study developed an evidence and theory-based intervention which targeted four behaviours key to obesity prevention in children aged 4–6 years: (i) reduction in screen-time (television viewing, gaming); (ii) promotion of increased physical activity; (iii) reduction in sugar-sweetened drinks (by promotion of water consumption); and (iv) reduction of sweet/candy consumption (see: http://www.toybox-study.eu). ToyBox was based on changes to the preschool environment but also involved parents; it is a potentially generalizable/scalable and low-cost (€5–29 per child per year) intervention. ToyBox was evaluated in a cluster-randomized controlled trial in 6 European countries, Belgium, Bulgaria, Germany, Greece, Poland and Spain, in more than 300 preschools and more than 7000 children). The ToyBox Study ended in June 2014 and many of the outcome data are not yet available. However, the intervention was successful in reducing child screen-time and has been shown in an economic analysis (based on the impact screen-time changes have on reduced later risk of obesity and diabetes), to be cost-effective (costs reasonable for health gain) and dominant (health gains significantly greater than costs).

Box K. Participatory physical activity and sedentary behaviour intervention in German preschools

Participatory research – with greater involvement of stakeholders in interventions aimed at obesity prevention than has been traditional – is widely recommended. However, relatively few examples exist of participatory interventions in early childhood movement behaviours. One notable exception is from a German cluster-randomized controlled trial in 39 preschools (509), in which the intervention group parents and preschools developed and implemented their own project ideas. This intervention was successful in reducing preschool sedentary time and increasing physical activity (both measured objectively).

6.3 Interventions to treat those affected by obesity

There is a need for treatment of children and adolescents who are already affected by obesity. In this section, the evidence for treatment effectiveness is summarized, suggested recommendations for health sector response to the need for treatment services are made, and a case example highlighted.

Evidence for treatment effectiveness

Evidence reviews of childhood obesity show that behavioural lifestyle interventions can lead to positive outcomes in weight, BMI and other measures of body fatness *(491, 510–512)*. This is the case for both the adolescent (~12–18 years) and pre-adolescent (~5–12 years) age groups. Such an approach is the foundation for all treatment interventions.

Very few of the studies included in evidence reviews have been undertaken in low- or middle-income countries. For example, of the 54 family-based lifestyle interventions included in the 2009 Cochrane review on the treatment of childhood obesity, 51 were from high-income countries (30 in the USA; 12 in Europe; 4 each in Australia and Israel; and 1 in Japan), with only 2 from China and 1 from Brazil *(510)*.

Early childhood: ~2–5 years

There have been very few obesity treatment trials in the first few years of life, no doubt because obesity is more prevalent with increasing age. In a systematic review of weight management schemes for children under 5 years of age, incorporating papers published up to February 2009, no studies aimed at the treatment of overweight or obesity in this age group were identified *(513)*. A subsequent review of treatment studies of obese children aged 2–7 years that had been published up to August 2011 identified 9 randomized-controlled trials *(512)*. Most of these were in primary school-aged children, rather than in preschool-aged children, and almost all reported positive weight outcomes. The majority of interventions (*n*=7) targeted parents as the "exclusive agents of change" and all had at least a moderate intensity (i.e. weekly, or more frequent) of face-to-face sessions for part of their programme.

Pre-adolescent children: ~5–12 years

The evidence base is strongest in this age group, with more studies having been undertaken than in adolescents, and certainly than in younger children. In pre-adolescent children, meta-analyses show that family-targeted behavioural lifestyle interventions can lead to a mean BMI reduction of approximately 1.0 kg/m^2 at 6 months or more when compared with no treatment/wait list control or usual care *(514).* Parental involvement when managing obese pre-adolescent children is vital *(510, 512, 515–517)*. Most clinical practice guidelines or consensus statements on the management of paediatric obesity, highlight the importance of parental and family involvement, although few provide age-specific recommendations *(518)*.

Adolescents: ~12–18 years

In adolescents, family-based lifestyle interventions can lead to a mean reduction in BMI at 6 months, from baseline or more, of approximately 1.4 kg/m^2, as well as improvements in a range of cardiometabolic risk markers (e.g. blood pressure, blood lipids, fasting insulin) *(514)*. There is also modest effectiveness of a limited armamentarium of drug therapy (e.g. orlistat, metformin) as an adjunct to lifestyle intervention in adolescents with moderately severe obesity *(510)*. Bariatric surgery may also be appropriate in those with more severe obesity *(516, 517)*.

Key elements of effective obesity treatment interventions

While there is no evidence to support one specific treatment programme over another, the key elements of obesity treatment in children are well recognized: management of obesity-associated

comorbidities; parental involvement; whole-of-family lifestyle change; long-term behavioural change; change to a healthier dietary intake; increased physical activity; decreased sedentary behaviours; and long-term healthy weight maintenance strategies *(510, 515–517)*. For adolescents, the role of non-conservative therapies, such as drug therapy or bariatric surgery in those with severe obesity, should be considered in the context of a family-based lifestyle change programme *(516, 517)*.

Recommendations for health sector response

One of the difficulties in adapting the existing evidence on obesity treatment to "real-life" service provision is that, firstly, treatment services may not be available, and secondly, where they do exist, such services are often less well-resourced, and staff less experienced, than those in funded clinical trials. In addition, patients seen in usual health-care settings may be different from those who participate in clinical trials. For example, they may be more socially disadvantaged, or they, or their parents, may have a broader range of psychological or medical comorbidities, thus making engagement in treatment plans more difficult.

The health sector in each country varies considerably and will have different challenges in responding to the need for provision of treatment services for those affected by obesity. There is little written on models of health service delivery for provision of obesity treatment in children and adolescents. The 2013 United Kingdom's National Institute for Health and Care Excellence (NICE) guidelines on lifestyle weight management services for children and young people *(516)* make a number of recommendations in this regard. While they are United Kingdom-based, many of the key recommendations would apply to other countries. These recommendations include:

- Ensuring family-based, multicomponent lifestyle weight management services for children and young people are available as part of a community-wide, multi-agency approach to promoting a healthy weight and preventing and managing obesity. They should be provided as part of a locally agreed obesity care or weight management pathway.
- Dedicating long-term resources to support the development, implementation, delivery, promotion, monitoring and evaluation of these services.
- Raising awareness of local lifestyle weight management programmes.
- Ensuring lifestyle weight management health professional staff are trained and have the necessary knowledge and skills.

Annex 3 discusses two potential models of a staged, coordinated approach to provision of treatment services for those affected by obesity, both of which may be more relevant to high-income countries.

Box L, below, gives an example of a community-accessible treatment programme for children aged 7–13 years with overweight or obesity, and their parents, that has been delivered at scale.

Box L. The MEND Programme: an English treatment programme delivered at scale

The MEND (Mind, Exercise, Nutrition. Do it!) 7–13 programme was established in the United Kingdom as a family-based weight management intervention for families of children aged 7–13 years affected by overweight or obesity. It is a multicomponent programme that addresses diet and physical activity through education, skills training and motivational enhancement. The intervention requires a parent or carer, and the child, to attend all 20 sessions over 10 weeks.

The MEND 7–13 intervention was developed to be delivered in community settings, such as schools or leisure centres, by a wide range of health, physical activity and social care professionals. MEND 7–13 was previously demonstrated in a randomized-controlled trial to be effective in reducing the BMI of obese children at 6 months from baseline (519).

Between 2007 and 2010, the MEND 7–13 intervention was implemented on a large scale, with MEND programmes rolled out across all regions of England. The intervention was delivered by local community-based "delivery partner" organizations, and was evaluated using prospective service-level data (520).

More than 21 000 families with children who were overweight were referred or self-referred to the MEND programme, of whom almost 14 000 attended. Over the 10-week programme there were improvements in BMI (mean reduction of 0.76 kg/m^2), self-esteem and psychological distress. The reduction in BMI under service conditions was slightly, but not statistically significantly, less than that observed in the randomized trial of the same intervention.

Generally, BMI fell more in children with higher baseline BMI, or who were younger, male, white, from families with an employed primary earner or who lived in less deprived areas. BMI also fell more if the child attended more programme sessions and if the programme group was relatively small, suggesting a "dose" effect.

These results suggest that the intervention, although benefiting all groups to some extent, may also have the potential to widen existing ethnic and socioeconomic inequalities in childhood overweight and psychosocial outcomes. Such findings highlight the potential need to adapt programmes (for example, by modifying content, training and implementation) to make them more successful for groups who currently respond less well to the intervention.

Several evidence gaps in the treatment of those children already affected by obesity have also been highlighted *(510)*; these include:

- What interventions are most effective at different levels of severity, and different ages and developmental levels?
- What interventions are most effective for specific culturally- and linguistically-diverse groups?
- What are the most cost-effective and resource-effective methods of treating those with obesity in different health-care settings? What models of health-service delivery are most useful, and what works best for different health systems?
- What is the health-service usage and costs of children and adolescents affected by obesity in high-, middle- and low-income countries?
- What are the training needs of health professionals who manage children affected by obesity in different settings, countries and regions?

Major challenges and knowledge gaps

There are future challenges in developing the evidence base for both obesity prevention and treatment, and in implementing solutions:

- Interventions need to be developed that can be embedded into ongoing practice and existing systems, rather than implementing interventions that are resource-intensive and cannot be maintained in the long-term.
- Interventions will need to be suitable for low- and middle-income countries, and for the more poorly resourced sections of high-income countries. This has major implications for capacity-building.
- Reinforcement and resourcing of relevant existing policies and programmes will need to occur, such as those relating to breastfeeding and appropriate infant feeding, child-care environments and school environments.

Summary

The promotion of healthy active living behaviours should be considered a priority to prevent obesity development, to manage and treat existing obesity and to improve health and wellness among children of all body weights. Preserving and improving healthy active living behaviours, including healthy eating, healthy moving and healthy sleeping, should be the priority of any childhood obesity prevention strategy. Support at the individual, parental, community and policy levels is required to achieve a recalibration of socially normative behaviours associated with healthy active living.

6.4 Policies that address childhood obesity in diverse sectors of society[1]

The ongoing global obesity epidemic does not spare children. Globally, in 2010, an estimated 43 million preschool children (under age 5) were overweight or obese – a 60% increase since 1990 *(521)*. This public health challenge affects countries across all income groups *(522)*. The nutrition transition that is taking hold in low-and middle-income countries has contributed to rapid increases in childhood obesity rates, even in countries that are still combating undernutrition. The coexistence of undernutrition and overweight and obesity within the same country, community, and sometimes within the same household, presents a challenging new paradox for addressing poverty and promoting human development in low- and middle-income countries *(523)*. To date, the policy response has not kept pace with the evolution of this public health crisis. Nations are at different stages of implementing childhood obesity initiatives, but all have now recognized that childhood obesity is a "wicked" issue that can only be tackled through a "whole-of-government" and "whole-of-society" approach *(522)*.

The Political Declaration adopted at the United Nations High-level Meeting on the Prevention and Control of Non-communicable Diseases in 2011 raised the level of importance of multisectoral action as a cornerstone strategy for reducing the global burden of noncommunicable diseases. Additional guidelines for successful multisectoral action have since been developed, building on the literature and lessons learned from "Health in All" policy approaches and experiences over the past few decades *(524, 525)*. A large number of sectors (e.g. transport, agriculture, trade, health) have a contribution to make to address the determinants and risk factors of obesity in children and adults. Given that obesity is rising across all age groups, and that enabling family and community environments are critical to child health, preventive policies in the non-health sector may have their greatest preventive impact when they are not restricted to children alone. Government, civil society and the private sector all have important roles to play in advancing solutions that give primacy to public health.[2]

The global evidence base around non-health sector policies is growing, but major gaps remain, including in synthesizing the available evidence. In many regions of the world there is also a dearth of local evidence and technical resources available to guide and support policy-makers. Regional action plans can help fill some of these gaps. Of the six WHO regions, four, the European Region, the Region of the Americas, the South-East Asia Region and the Western Pacific Region, have a regional noncommunicable disease action plan that addresses, at least in part, the issue of childhood obesity. There are two examples of regional action plans focusing specifically on childhood obesity, one for Europe (the

[1] Authors: Shiriki Kumanyika, Rachel Rodin with the WHO Collaborating Centre on Chronic Non-Communicable Disease Policy (Catherine Dickson, Vivian Ellis, Robert Geneau, Ahalya Mahendra), on behalf of the Ad hoc Working Group on Science and Evidence for Ending Childhood Obesity.

[2] It is beyond the scope of this report to review the evidence on the governance and coordination mechanisms that enable effective multisectoral action.

European Union Action plan on childhood obesity 2014–2020[1]) and the other for the Americas (Plan of action for the prevention of obesity in children and adolescents 2014–2019[2]). These regional action plans are intended to guide Member States by proposing a range of evidence-informed measures that are voluntary and that should be taken forward by each country according to their own circumstances. Many of the recommendations are similar across regions, with a strong focus on creating and promoting environments that are conducive to healthy eating and active living (Annex 4, Table A4.1).

The complete range of available measures is quite large and includes interventions at the population, community and individual levels. There have been recent efforts to map out the roles of various sectors and policy actors in the development, adoption and implementation of obesity prevention initiatives *(524)*.

The uptake of preventive measures at the country level has increased in recent years. However, it is currently not possible to paint a precise picture of the level of uptake and degree of implementation country by country. The 2013 noncommunicable diseases capacity survey *(526)* provides information by region for a limited number of interventions only: restrictions on the marketing of foods and non-alcoholic beverages to children; legislation or self-regulating policies to limit saturated fatty acids and trans-fats from the food supply; and encouraging breastfeeding and the International Code on the Marketing of Breast-milk Substitutes. For all three measures, a clear gradient, by country income status, is typically observed, with high-income countries more likely than low- and middle-income countries to have adopted and implemented childhood obesity prevention initiatives. For example, 57% of countries in the European Region report having policies in place for reducing the impact on children of the marketing of foods and non-alcoholic beverages high in saturated fats, trans fats, free sugars or salt. The proportion drops to 3%, however, for the African Region, and 0% for the South-East Asia Region (Annex 4, Figure A4.1).

Overview of evidence available and key policy recommendations for each sector

This section summarizes the *evidence on the effectiveness of interventions* involving non-health sectors, and provide a sense of the *range of policy interventions* which have been recommended or implemented. In some cases, the policy recommendations are based on early or mixed evidence, reflecting the urgency to take action to stem the epidemic rather than wait for "perfect proof of what works, especially in the many areas where interventions are low risk" *(449)*. Continued emphasis on policy research and programme evaluation is needed.

Although similar questions are addressed for each sector, the summaries differ, as appropriate to the literature in each area. Attempted answers are given to such questions as: what makes it important to

[1] http://ec.europa.eu/health/nutrition_physical_activity/docs/childhoodobesity_actionplan_2014_2020_en.pdf
[2] http://www.paho.org/hq/index.php?option=com_docman&task=doc_view&Itemid=270&gid=28890&lang=en

intervene in this sector? (i.e. what determinants or underlying causes of childhood obesity could be addressed?); what is the range of policy interventions proposed and what is known about their effectiveness? Sectors referenced refer to government ministries and their associated civil society partners (nongovernmental organizations private sector, academia, community groups, etc.).

Built environment sectors: environment, transportation, infrastructure, development, urban planning, housing

Influence of the built environment on obesity

The built environment is recognized as a determinant of obesity due to its influence on food intake and physical activity *(527, 528)*. It is comprised of homes, buildings, streets, open spaces and infrastructure, including transportation systems, as well as their design, configuration and connectedness *(527)*. There is growing evidence that specific built environment characteristics have an impact on physical activity and the food environment, which are known to influence body weight *(529)*. The range of policy options from the literature is summarized in Panels 1 and 2. A more complete overview of the evidence is presented in Annex 4, Tables A4.2 and A4.3.

Panel 1 – Built environment and physical activity

Policy recommendations

- Locating schools within easy walking distance of residential areas (CDC).
- Create safe routes to schools by improving the built environment (e.g. implement Safe Routes interventions).
- Ensure open spaces and public paths can be reached on foot, by bicycle and using other modes of transport involving physical activity. They should also be accessible by public transport.
- Improve access to outdoor recreational facilities such as parks and green spaces.
- Ensure public open spaces and public paths are maintained to a high standard. They should be safe, attractive and welcoming to everyone.
- Enhance personal and traffic safety in areas where people are, or could be, physically active.
- Creation of, or enhanced access to, places for physical activity (e.g. trails or facilities) by reducing barriers to access, combined with informational outreach activities: strongly recommended.
- Communities, transportation officials, community planners, health professionals, and governments should make promotion of physical activity a priority, by substantially increasing access to places and opportunities for such activity.
- Improve access to public transportation.
- Point-of-decision prompts are recommended, for health benefits or weight loss.

Influence of the built environment on physical activity

The elements of the built environment most often associated with increased physical activity are density and design (including connectivity, proximity, land-use mix), and transportation systems *(527, 530)*. For example, people living in compact neighbourhoods are more likely to engage in physical activity, since there are multiple convenient opportunities nearby to do so as part of their daily activities. Design essentially refers to the functionality of the neighbourhood, and includes the presence of good street connectivity, sidewalks and bicycle paths which enable access to neighbourhood amenities (stores, workplaces, schools, public spaces). The presence of many different types of amenities (land-use mix) and the presence of public transportation stops (transportation) are also important. Research has demonstrated that these elements of neighbourhood design, and the way land is developed and used, may affect transport choice (automobiles, public transport, walking or bicycling) *(319, 531)*. This type of physical activity for utilitarian purposes – active transport – is

an important contributor to meeting the daily physical activity requirements that many public health agencies have established for healthy living. The literature on built environment and physical activity emerges primarily from developed countries.

Influence of the built environment on food environments

The "food environment" refers to the type of food available, how much it costs, and how it is marketed in specific settings. Geographic access to healthy, affordable and nutritious food is a key domain of the community nutrition environment linked to the built environment. Measures have been developed to quantify the density of food outlets ("fast food" outlets, restaurants, grocery stores, convenience stores), the different types of food outlets that are available (variety) as well as how close these outlets are to neighbourhoods (proximity), since these three components can influence the food consumed by residents. Research in the area of food environment is relatively new, and as with research on built environment and physical activity, primary studies are predominantly observational in nature. A large amount of heterogeneity in the terminology and methodology is used in the literature. However, this line of inquiry is sufficiently promising to merit consideration and investment from a public policy perspective.

Panel 2 – Built environment and food

Policy recommendations

- Increase community geographic access to healthy foods retailers, through a variety of policy levers, such as tax incentives, land use and zoning regulations.
- Improve transportation routes to healthy food retailers.
- Restrict access to unhealthy foods around schools through zoning regulations, and make potable water available in school environments.

Education

This section addresses broad-based programmes and policies to prevent childhood obesity in preschool and school settings. The main focus was on reviewing studies where the target group was the general student population (which inherently contains a mixture of weight profiles). The range of policy options from the literature is summarized in Panels 3, 4 and 5. A more complete overview of the evidence is presented in Annex 4, Table A4.4.

Panel 3 – Education: overarching recommendations

Policy recommendations

- Adopt a coordinated "whole-school approach" including assessing the entire school environment and implementing policies and practices to support healthy weights, diet, and the promotion of physical activity.
- Integrate teaching on physical activity and healthy eating with changes to the physical and food environments.
- Use community and parental engagement to amplify the impact of child-care and school-based interventions.
- Address the whole school, regardless of weight status.
- Include pre- and post-care, and extracurricular activities.
- Support and train teachers and other staff to implement health promotion strategies and activities.

Although study results have been mixed, intervening in preschool and child-care settings (for 0–5 years of age) to improve diet, increase physical activity and/or reduce sedentary behaviour can impact these behaviours, and adiposity, successfully and significantly *(319)*. Early trials often employed more than one strategy (for example, increasing structured physical activity time and training teachers), however, these tended to lack a home-based/parental component or modifications to the food or built environment *(319)*. The success of more recent programmes has been attributed to the application of a greater range of intervention components across more settings, and to the incorporation of environmental modifications *(318)*. For example, recent systematic reviews by Hoelscher et al. *(318)* and Laws et al. *(532)* report on studies showing: (i) lower prevalence of overweight and obesity among 2 year olds and 3.5 year olds after a comprehensive, multisetting, nutrition and physical activity programme was put in place in Australia (Romp & Chomp); (ii) lower obesity prevalence following a 3-year programme to reduce energy content of nursery-school food by 10% in Chile; (iii) reductions in BMI z-score among low-income populations in France after two combined parent/preschool interventions; (iv) a significant increase in physical activity and reduction in body fat following a multicomponent intervention that included educational classes, physical activity sessions, changes to the preschool environment and information sessions for parents in Switzerland; and (v) improvements in physical activity, but not BMI, in the multicomponent "Healthy and Ready to Learn" programme *(318)*.

Panel 4 – Education: school environment

Policy recommendations

- Establish school environments that support healthy eating choices and physical activity throughout the school day.
- Establish nutrition standards for schools that include a healthy diet with limited fat intake.
- Provide high quality school meals and healthy, appealing food /beverage choices outside school programmes.
- Create a pleasant, sociable environment for mealtimes with staff supervision.
- Ban sugar-sweetened beverages (including flavoured/sweetened milk) and limit the portion size of 100% juice.
- Make drinking water freely available to students in dining areas and throughout the day.
- Create and support school gardens.
- Plan building layout, recreational spaces.

Panel 5 – Education: programmatic

Policy recommendations

- "Hands-on" activities that encourage touching and tasting foods.
- Gardening programmes are promising for increasing children's fruit and vegetable consumption.
- Schools should offer opportunities to be physically active throughout the day (e.g. during play times, lunch breaks, pre- and post-school activities) and there should be at least one hour daily of school-based physical activity.
- Schools should try to increase the amount of time students spend doing moderate to vigorous physical activity, either by increasing the duration, or intensity, of activity in physical education classes.
- Teacher training in physical education modules is recommended from K-12.
- Children should be encouraged to develop movement skills (regardless of ability or disability) by having the appropriate opportunities, equipment and classes.

School-based programmes (for ages 6–18 years) can also improve diet, reduce sedentary behaviour, and increase physical activity; overall, study design, participants, intervention types and outcome measures have been heterogeneous *(533–536)*. In addition, school-based approaches are effective for reducing BMI in children, even though effects are mild. A recent meta-analysis of studies found an overall standard mean difference of -0.076 (95% CI: -0.123–0.028; $P < 0.01$) in BMI between intervention and control groups *(490)*. BMI appears to be more difficult to influence in teenage years, with reviews citing intervention-related differences that are not statistically significant *(490)*. Considering that maintenance of a healthy weight in general paediatric populations may be sufficient to stem the rising tide of obesity, the impact of programmes with small population-level effects (including BMI stabilization) warrants further exploration *(537, 538)*.

An overarching conclusion from the literature is that the most successful trials involved parental support, lasted at least 1 year, and were comprehensive (e.g. defined as targeting all of physical activity, sedentary behaviour, healthy eating and unhealthy eating or incorporating a range of educational/environmental techniques) *(490, 534)*. Further evidence on effective intervention components is provided in Annex 4, Table A4.4.

Although the evidence reviewed provides important guidance and rationale for policy-makers, many interesting and innovative community approaches may be underway which are not captured in the formal literature; the successes and failures of these programmes may hold the key to adapting known approaches to specific local and cultural contexts. Further, although BMI is of great interest, as the most frequently-used proxy measure for adiposity, changes in behaviours achieved through broad-based programmes may have more important effects than childhood BMI for later life stages. The appropriate balance of interventions to prevent BMI from increasing, and to instil life-long, individual, family and community-based patterns of healthy living remains in question. The long-term impacts of societal efforts require consideration and evaluation as part of ongoing research and programme evaluation.

Agriculture and trade

Food systems have undergone a profound transformation in recent decades, resulting in altered dietary patterns globally. The epidemic rise in obesity, which started in the late 1980s and early 1990s in the USA and other leading high-income countries, progressed to middle- and then, more recently, to low-income countries in parallel with strong changes in local food systems. Recent trends in food production, processing, trade, marketing, and retailing have contributed to the rising occurrence of diet-related noncommunicable diseases around the world *(539, 540)*. The consumption of ultra-processed food and drink products has steadily risen in high-income countries over the past few decades and the same trends are now also evident in lower-income countries *(541, 542).* The increase in the consumption of these products are accompanied by declines in the dietary share of fresh or minimally-processed foods *(543, 544)*. Agricultural and trade policies are some of the powerful forces behind this transition through their respective, and also synergistic, effects on the supply and demand factors affecting food affordability, availability and quality. The range of policy options from the literature is summarized in Panels 6, 7 and 8. A more complete overview of the evidence is presented in Annex 4.

Agriculture

There are a number of agricultural policies and interventions that have been recommended to promote healthy diets globally (see full table and evidence in Annex 4, Table A4.5) .

Panel 6 – Agriculture

Policy recommendations

- Strengthen and improve local and regional food systems, in part through agricultural policies and incentives that promote local food production and processing. Incentives can include forming grower cooperatives, instituting revolving loan funds, and building markets for local farm products through economic development. Additional incentives include, but are not limited to, farmland preservation, marketing of local crops, zoning variances, subsidies, streamlined license and permit processes, and the provision of technical assistance.
- Use of incentives to promote the production of fruits and vegetables for local markets.
- Adoption of programmes to protect fruit and vegetable farmers from natural disasters in a manner comparable to programmes that are available for farmers producing major commodity crops.
- In the USA, a recommendation has been made for the President to appoint a Task Force on Agriculture Policy and Obesity Prevention to evaluate the evidence on the relationship between agriculture policies and the American diet.
- Review national policies and investments and integrate nutrition objectives into food and agriculture policy, programme design and implementation, to enhance nutrition sensitive agriculture, ensure food security and enable healthy diets.
- Support urban agriculture in order to improve access to fresh and healthy foods, in a way that minimizes risks to the environment.
- Improve food supply chains (traditional and modern) to increase the local availability, affordability, diversity and nutritional quality of foods.
- Develop local solutions to reduce post-harvest food losses.
- Preserve and promote agricultural biodiversity/promote the diversification of crops, including underutilized traditional crops.

Over the past few decades, market factors and agricultural policies have increased the demand for exportable cash crops, sometimes with the unintended consequence of reducing the diversity of fresh produce available at the local level. Agricultural policies, often through both production and input policies, have promoted farmers' capacity to increase production, generally of the kind of commodities – corn, wheat, cotton, rice, milk, and later soybeans – that lend themselves to large-scale production, easy storage, and long-distance shipping *(545)*.

The extent to which agricultural subsidies (as a form of production policy) are partly to blame for the current obesity epidemic is still a matter of debate. One side of the argument links the presence of subsidies to the predominance of “cheap foods and calories” through the overproduction of commodities that are the basic ingredients of processed, energy-dense foods and “soft” drinks *(546, 547)*. Other studies suggest that the effect of these agricultural policies is negligible owing to the weak relationship between farm-gate prices and retail prices *(548, 549)*. A recent report in the USA concludes that subsidy removal as a means to combat the overconsumption of unhealthy foods and beverages is an ineffective obesity prevention strategy, not being sufficient, as a stand-alone measure, to affect the price or production of these products, and could harm small and midsized family farmers in the process *(550)*.

Only one systematic review has been identified that focuses on the empirical evidence linking agriculture-based food price policies with nutrition outcomes. The authors report that there is currently no direct evidence that agricultural policies that directly influence the price of food affect rates of undernutrition. However, three studies that evaluated the effect of these policies on overnutrition suggested that they had a small effect on adult weight and risks of nutrition-related chronic disease *(551)*.

There is more of a consensus and supporting evidence for (i) the use of incentives to promote the production of fruits and vegetables for local markets *(548, 552)* and (ii) value chain-based interventions enhancing the production of wholesome foods in general and reducing post-harvest losses (still as much as 30–40% in some low- and middle-income countries), so that the products become more available and affordable to the poor *(553)*. More research investments are needed to better understand the range of agricultural policy levers that can be used to facilitate the “farm to fork” process in different settings.

Trade

International trade agreements are contributing to the development of an increasingly global food system through four main pathways: (i) the opening of domestic markets towards international food trade and Foreign Direct Investments; (ii) the subsequent entry of transnational food corporations and their global market expansion; (iii) global food advertising; and (iv) restrictions to the policy space – for example, trade agreements can restrict governments from introducing policies and laws to address noncommunicable diseases (restrictions on food labelling, etc.) *(554–556)*.

These changes have accelerated in recent years and research efforts are now underway to understand the impact of trade agreements on various dimensions of local food environments (food availability, nutritional quality, price and promotion of foods) *(556, 557).* It is a complex research agenda, given that the effects of trade agreements on food environments, both negative and positive, can be context-specific. However, the evidence base about the impact of trade agreements on public health is slowly growing, including in mapping the risks and benefits of future large trade agreements like the Trans-Pacific Partnership Agreement *(557)*. This evidence is important since decision-makers of the World Trade Organization (WTO) have traditionally interpreted only a narrow range of public health concerns as legitimate reasons for trade restrictions *(557)*.

Panel 7 – Trade (relevant for all countries)

Policy recommendations

- Promote "policy coherence" between health policies and trade agreements and strengthen capacity to undertake rigorous health impact assessments in relation to free trade agreements.
- Adopt safeguards on the level of foreign ownership of agricultural land and of local food production and for trade agreements to protect national and local food sovereignty in more general terms.
- There are trade agreements where tobacco and alcohol were excluded from tariff reduction following submissions on the health and financial implications of their inclusion. Explore a similar scenario in relation to food and obesity prevention.
- Conduct research to identify policy instruments and measures that can be used to support local food systems and obesity prevention strategies triggering trade disputes.
- People and their livelihoods need to be placed at the centre of all international policy and development efforts. The right to adequate food, including nutritional considerations, should be part of the overall goal and policy of all institutions regulating or deregulating international trade, investments, development loans and external debt relief.
- Provisions could be made to exclude health and social security from trade and investment agreements, as a form of safeguard to protect the right of democratically elected governments to maintain policy space for regulation, licensing, cost-containment and limiting or reversing commercialization, where this is in the public interest.
- The Codex Alimentarius can be an appropriate mechanism to promote healthier diets worldwide and fair practices in food trade through its role in setting standards on labelling, and, to a lesser extent, food composition.
- Research could inform the development of future Codex standards. For example, on food labelling, the INFORMAS study proposes a new taxonomy that goes beyond Codex Alimentarius definitions.

The strength of the analyses so far has been to look at trends of supply and demand over long periods, although it remains difficult to link trade, diets and noncommunicable disease risk factors with country-level data *(558)*. Studies have linked trade liberalization to the nutrition transition in the Pacific Islands (PICs) *(559)*. Fat consumption has dramatically increased over the past 50 years in PICs, a period during which the total fat supply increased by as much as 80% *(560)*, particularly through increased imports of vegetable oils, margarine, butter, meat, chickens and canned meat *(561)*. The North American Free Trade Agreement (NAFTA) has also been identified as a contributing factor to the obesity epidemic in

Mexico. Directly and indirectly, the USA has exported increasing amounts of corn, soybeans, sugar, snack foods, and meat products into Mexico over the last two decades. Facilitated by NAFTA, these exports are one important way in which agriculture and trade policy in the USA influences Mexico's food system and ultimately health outcomes such as childhood obesity *(562)*. The availability of unhealthy products in Mexico, even in remote locations, has risen significantly over the past few decades. Studies have showed that caloric intake from sugar-sweetened beverages doubled in Mexico between 1999 and 2006 across all age groups *(563, 564)*. In Brazil, the entry of multinational retailers into Brazilian markets has increased competition and reduced prices, particularly for processed foods, for which large retailers have an advantage due to economies of scale. In turn, rising consumption of processed foods has been observed during the 1990s in conjunction with rising consumption of fat and sugar, and declining consumption of traditional cereals and beans in Brazil *(554)*.

There is less literature about the solutions that could be effective to mitigate the negative effects of trade agreements on public health. Various reports note the need for "policy coherence" between health policies and trade agreements in order to ensure that they are formulated in ways which enable both trade objectives and public health objectives to be met at the same time *(555)*. There is an explicit acknowledgment that there is currently a lack of capacity to undertake rigorous health impact assessments in relation to free trade agreements. There are also increasing calls for having safeguards on the level of investment of foreign ownership of local food production and for trade agreements to protect national and local food sovereignty[1] in more general terms *(556)*. It has also been recommended to broaden the scope of the Aid for Trade programme in order to tackle the issue of low fruits and vegetables supply and intake in low- and middle-income countries. The Aid for Trade model is based on increasing productivity by improving markets rather than subsidies.

Food/nutrition

The widespread availability of energy-dense, nutrient-poor foods has been identified as a key driver of the global obesity epidemic *(565)*. In response, international multilaterals such as the United Nations Food and Agriculture Organization (FAO) and WHO, governments, and civil society have recommended and undertaken a wide range of policy interventions to modify the food environment, and in turn, promote healthier eating. The food environment has been defined broadly as "the collective physical, economic, policy and sociocultural surroundings, opportunities and conditions that influence people's food and beverage choices and nutritional status" *(565)* (see Annex 4, Figure A4.2). Influencing the food environment therefore demands a collaborative multisectoral and multistakeholder approach engaging actors across the food sector *(566)*. This perspective is echoed by internationally recognized bodies of experts within the fields of nutrition, food policy and public health *(446, 449, 549, 566–569)*. Adopting a "multicomponent" approach is considered promising for having a greater reach and impact. The range

[1] Food sovereignty is the right of peoples to healthy and culturally-appropriate food, produced through ecologically sound and sustainable methods, and their right to define their own food and agriculture systems. It puts those who produce, distribute and consume food at the heart of food systems and policies rather than the demands of markets and corporations.

of policy options from the literature is summarized in Panels 9–12. A more complete overview of the evidence is presented in Annex 4.

Based on a comprehensive review of the evidence linking the food environment and patterns of diet, physical activity and body composition *(570)*, the World Cancer Research Fund (WCRF) has recommended a set of food policies to promote healthy diets within a framework called NOURISHING *(569)*. NOURISHING is an acronym for the food policy areas that make up a comprehensive approach for the food sector to address obesity and diet-related noncommunicable diseases. This includes: food labelling; changing food quality and availability; economic measures; restrictions on marketing; modifying the built environment; multisectoral and food system initiatives (e.g. agriculture/trade); and behaviour change communications through education, public awareness campaigns and clinical approaches (see Annex 4, Table A4.9) *(569)*.

Panel 8 – Trade: specific to developing countries

Policy recommendations

- The WTO Agreement on Agriculture (AoA) and the Doha Development Agenda (DDA) give special rights to developing countries. Not all countries are willing, or able, to take advantage of the available policy instruments under the AoA and DDA.
- Broaden the scope of the Aid for Trade programme in order to tackle the issue of low fruits and vegetables supply and intake in low- and middle-income countries. The Aid for Trade model is based on increasing productivity by improving markets rather than subsidies.

Similar authoritative groups or reports recommending changes to the food environment as part of a multipronged approach to obesity include: EATWELL[1] *(567)*; WHO[2] *(566)*; FAO[3] *(549)*; the Institute of Medicine (IOM)[4] *(446)*; the McKinsey Global Institute[5] *(449)*; and the American Heart Association (AHA)[6] *(568).* Further research and evaluation of existing interventions will be necessary to obtain a clearer understanding of the effects and impact of nutrition related interventions.

This section focuses on key recommendations on food labelling and quality, as well as public information.

Nutritional labelling is described as "the provision of nutrition information, in a standardized format on foods sold" *(566)*, for example, requiring producers to indicate energy, total fat, saturated fat, protein, carbohydrate, sugar and salt content for processed foods *(566)*. Nutritional labelling has also been applied to restaurant menus to promote healthy choices on meals consumed outside of the home *(567)*. The use and visibility of food labels is increased by placing information on the front of packages *(314)*, and the understanding of labels

Panel 9 – Labelling

Policy recommendations

- Front-of-package labelling.
- Simple information (e.g. traffic light scheme) with consistent format and placement.
- Mandated nutrition fact panels or front-of-pack labels/icons.
- The most critical nutrition components for front of-package labelling are: calories, saturated and trans fats, sodium, and added sugars.
- Clear information on portion size with nutritional information per portion.
- Endorsement schemes (such as a healthy choice symbols) identifying products on front of package that meet specific standards.
- Point of purchase schemes identifying and promoting healthy food options.
- Regulate nutrient and health claims that can be made on packages or to promote food items (e.g. permissible fat content to market food as "low in fat").
- Quick service restaurant menu labelling.
- Aggregate nutritional content information on food purchased, and traffic light labels provided at checkout.
- Use menu labelling alongside other interventions to influence healthier menu choices.
- Monitor real-life menu labelling experiments to expand the evidence base.

[1] EATWELL is a project funded by the European Commission to improve nutrition policy based on evidence around interventions to promote healthy eating. EATWELL's recommendations promote policies to enhance the availability of healthier choices and restrict foods with less nutritional value.

[2] The WHO Population-Based Approaches to Childhood Obesity Prevention tool was developed for WHO Member States following the 2009 Population-based Prevention Strategies for Childhood Obesity Forum and Technical Meeting. It identifies priority areas countries can engage in, examples of population-based approaches to preventing childhood obesity, and an overview of comprehensive prevention interventions likely to be most effective at national and regional levels.

[3] The background paper developed by Corinna Hawkes for the Second International Conference on Nutrition (INC2) captures an overview of the nutrition interventions which influence consumer behaviour and the evidence of the effects of these actions since the last ICN in 1992.

[4] The Institute of Medicine's report Accelerating Progress in Obesity Prevention: Solving the Weight of the Nation, funded by the Robert Wood Johnson Foundation, assesses the issue of obesity in the United States and provides a suite of recommendations that include the food sector among others.

[5] The McKinsey Global Institute's report, Overcoming Obesity: An Initial Economic Analysis, identifies 74 interventions to address obesity across a range of sectors including food. Many of these interventions were modelling to assess their impact if rolled out at the national level in the United Kingdom with plans to similarly test these interventions through models of rolling them out in emerging markets.

[6] The American Heart Association's scientific statement, "Population approaches to improve diet, physical activity, and smoking", was developed through a systematic review and grading of current scientific evidence of population approaches to improve dietary habits, increase physical activity, and reduce tobacco use.

can be improved by using simple graphics such as a traffic light system *(314)*.

Improved quality of the food supply includes improving access to healthy foods, including fruits and vegetables, and the mandatory, or voluntary, restriction of specific nutrients such as fat, sugar, or sodium (sodium, although not directly implicated in obesity, has been implicated in other diet-related noncommunicable diseases), the reformulation of foods to reduce their content of sugars, fats and/or sodium, and portion size reduction *(571)*. The WCRF suggests that food reformulation is an equitable approach, having the greatest impact on those who eat the largest quantities of unhealthy foods *(571)*. However, in some environments, where processed foods are rarely eaten, reformulated food products may promote the consumption of more processed foods *(572).* Thus, reformulation may be a better strategy in countries where the consumption of processed foods is already high. Policies to reduce salt and trans-fatty acid contents have shown reduced dietary intake *(567)*. To date, little information is available regarding the impact of policies to reduce saturated fatty acid and sugar content in foods *(567)*. Portion size reduction has been linked with reduced energy intake *(570)*.

Informed choice and public information campaigns to improve access to nutritional information, as well as having the tools, such as education, to interpret food-related information, are a key factor in influencing healthy food choices *(569)*. However, it must be noted that informed choices are not necessarily always healthy choices – many interlinked factors, aside from nutrition and health, influence what people consume; thus information alone is not sufficient to address child obesity. Social marketing and public information campaigns are supported and delivered through various forms of media, and across several channels, to increase knowledge and change attitudes towards diet and physical activity, and often run parallel with community-based activities *(566)*. Public information campaigns have been shown more likely to be effective if they are longer running, use multiple channels and targeted specific foods *(568)*.

Dietary guidelines advise the general population on food consumption for an adequate and healthy diet and may also address more broadly meals and eating patterns (i.e. when, where, how, with whom) for better health and well-being. An example of guidelines taking this broad approach are Brazil's new dietary guidelines, released in 2014 *(573)*. Guidelines may be food-based – translating the recommendations on nutrient intakes and population dietary targets into a recommended mix of foods that make up a healthy diet (e.g. a food pyramid or plate demonstrating portion size) *(574)*. Public education guidelines may also target specific foods or dietary requirements (e.g. China's "Guidelines on Snacks for Chinese Children and Adolescents" or Mexico's "Beverage Guidelines for Healthy Hydration") *(574)*. Dietary guidelines can be used as an educational tool to inform individuals on healthy foods to eat more frequently, and less healthy foods to avoid or to eat less frequently and in smaller amounts. Guidelines can also be used to inform and guide other policies (e.g. guidelines can provide a classification system to identify foods to be labelled as healthy or unhealthy choices; they can set standards for the food industry to use for product formulation, and can be used by institutions, such as

schools, to plan balanced menus for their students) *(566)*. Evidence suggests that public awareness of guidelines varies by population and setting, and although awareness appears to be increasing, being aware of guidelines does not necessarily mean that users understand them *(575)*. There is limited evidence demonstrating that dietary guidelines are linked to improved dietary behaviour *(575)*.

Panel 10 – Quality of food supply

Policy recommendations

- Improve access to healthy foods, including fruits and vegetables (see Agriculture and Trade sections)
- Introduce voluntary, or regulated, reformulation of food products to reduce specific nutrients (e.g. reduce salt, fat, trans fats, energy density, sugar content).
- Reduce portion size of processed meals, dishes, snacks, foods, and drinks.
- Reduce number of calories served to children in quick service restaurants.

Panel 11 – Informed choice and public information campaigns

Policy recommendations

- Longer-term campaigns.
- Simple targeted messages.
- Frequent exposure to messaging.
- Comprehensive public-health campaigns promoting healthy eating habits across various media, such as television, radio and social media.
- Implement public information campaigns in conjunction with other measures within a comprehensive obesity prevention strategy, including increasing availability, labelling or reformulation.

Panel 12 – Dietary guidelines

Policy recommendations

- Evidence-based national guidelines on healthy eating that are updated regularly, adapted for specific populations, and effectively communicated to the population.
- Develop and implement national food-based dietary guidelines.
- Educate the public on food-based guidelines.
- Efforts should be made to keep recommendations evidence-based and to grade evidence in a way that is easy for guideline users to understand.

Fiscal measures to curb obesity and promote healthy weights

The adoption of fiscal measures for obesity prevention is of high media interest *(449)* and has been recommended to countries for consideration by the WHO *(310, 566)*. The range of policy options from the literature is summarized in Panel 13. A more complete overview of the evidence is presented in Annex 4, Table A4.8.

So far, synthesis reviews consistently report that little evidence about effectiveness of the use of food taxes as a public health strategy to reduce obesity is available. There is somewhat better evidence for subsidies to promote consumption of healthy foods such as fruits and vegetables *(576)*. Evidence of the effectiveness of tax and other financial incentives to promote physical activity is mixed, with stronger evidence that financial incentives can shift behaviour at the individual level *(572)*. Recommendations endorsing fiscal measures, in most cases, are transparent about the limited evidence base, making the case for the recommendation on logic *(449)*, the need to research and evaluate *(572)*, or a "leap of faith" *(576)*.

Panel 13 – Fiscal measures

Policy recommendations

- The introduction of fiscal measures to shift consumption away from unhealthy foods and beverages should be considered. The most important criteria for considering a policy is the potential for harm such a policy might cause, rather than the extent of its impact on obesity.
- The introduction of a tax on sugar-sweetened beverages and unhealthy food. Conduct context-specific studies (e.g. price and cross-price elasticity of demand) to establish feasibility, risks and potential impact.
- Subsidies on healthy foods, especially fruit and vegetable subsidies, primarily targeting children and low-income households.
- Flexible financing or tax credits to support health promoting food and beverage retailing and distribution policies.
- Financial incentives to promote physical activity.
- Systemic and individual financial incentives and disincentives to promote physical activity.
- Combined or multipronged fiscal measures.
- Combined or multipronged approach, especially for children and adolescents, of changing relative prices by both taxing less healthy, energy-dense foods and subsidizing healthier, less energy-dense foods.
- The acceptable level of tax/subsidy and the most effective fiscal policy, or combination of policies, is likely to be context-dependent.
- Health impact assessments recommended to determine the unintended effects of new and/or existing fiscal measures designed to target other sectors. Removal of subsidies to unhealthy foods.

In a 2013 OECD review, Sassi et al. *(577)* summarize the economic basis and general guidance for the use of fiscal measures as public health interventions. "From a public health standpoint, taxes on health-related commodities may be beneficial when one or more of the following conditions are met: (a) they contribute to correcting important externalities; (b) they have the potential to shift consumption towards healthier patterns; (c) they can justify the use of incremental resources in the fight against the diseases linked with the consumption of the commodities in question; (d) they contribute to reducing health disparities; and, (e) the benefits associated with any, or all, of the above are not offset by the potentially negative impacts." Sassi et al. outline several negative impacts to be avoided, mitigated or balanced, including administration costs, regressive financial impacts on consumers, impacts on the

broader economy, tax avoidance and illicit trade. The risk that consumption may shift towards unhealthy substitute products is also noted.

In contrast to the, currently, relatively weak evidence supporting food and beverage taxes, taxation-related price increases are established to be effective in changing behaviour and health outcomes as part of comprehensive tobacco control. The success of tobacco control taxation policies, despite the imperfect information on their effectiveness available at the time of their introduction, has encouraged expert consensus panels to make recommendations for similar specific beverage and food taxes (e.g. Faulkner et al. (2011)) *(576)*. While there remain significant evidence gaps in linking price increases for unhealthy foods and beverages to decreases in weight, Andreyeva et al. (2010) found there is a large literature, particularly for the USA, showing the linkages between prices and food and beverage consumption that provide potential for public health benefits. They caution, however, that there are "observed gaps in research related to substitutions between healthy and unhealthy foods" *(336).* Moodie et al. (2013) explain the "uncertainty and gaps in the evidence" by noting that the "fiscal policies such as taxes and subsidies have been driven largely by imperatives to raise revenue or increase supply, rather than to change population behaviours. Its use specifically for the purposes of improving diets is still relatively untested". Moodie et al. conclude that, "Whilst more evidence is needed, the introduction of fiscal policies as obesity prevention measures should be considered." *(572)*. Faulkner et al. (2011) reach consensus that, "It is likely that policies need to be implemented in the face of an incomplete evidence base."*(576)*. McKinsey (2014) likewise argues for all obesity interventions that "society should also be prepared to experiment with possible interventions. In many intervention areas, impact data from high-quality randomized-control trials are not possible to gather. So, rather than waiting for such data, the relevant sectors of society should be pragmatic, with a bias toward action, especially where the risks of intervening are low." *(449)*.

Research and evaluation of the "natural experiments" in the relationship between price and consumption, and in use of fiscal measures, is broadly recommended, especially for emerging economies and developing countries and for vulnerable populations. Escobar et al. (2013) conclude that "Upcoming research should estimate price elasticities in low- and middle-income countries and identify potential health gains from taxes combined with subsidies of healthy food." *(578)*. Moodie et al. (2013) note that "Equity considerations highlight the paucity of evidence around the differential impact of taxes and subsidies on different sociodemographic or ethnic groups."*(572)*.

While several reviews note that high-income countries have increasingly initiated these natural experiments, the 2013 WHO Noncommunicable Diseases Country Capacity Survey shows that these innovations are more broadly distributed than previously reported (Table 8). For example, Mexico and Fiji have both implemented a comprehensive policy response to obesity that includes fiscal measures. These natural experiments, across diverse country contexts, may provide the opportunity to address some of the identified gaps in evidence of effectiveness and the optimal policy mix.

Table 8. Summary of reported obesity-related fiscal measures in the 2013 Noncommunicable Diseases Country Capacity Survey (*n*=178)

WHO region/ country income	[A] Taxation incentives to promote physical activity	[B] A subsidy on healthy foods	[C] Taxation of high fat foods	[D] Taxation of high sugar foods/non-alcoholic beverages	A and C or D	B and C or D	A and B	All four fiscal measures
AFR	3	2	2	5	0	1	1	0
AMR	4	6	0		0	0	3	0
EMR	1	0	1	2	0	0	0	0
EUR	4	2	2	8	1	0	1	0
SEAR	1	0	0	1	0	0	0	0
WPR	2	3	0	3	1	1	2	0
Low income	0	0	1	4	0	0	0	0
Lower-middle income	3	4	2	3	0	1	1	0
Upper-middle income	3	4	1	6	1	1	3	0
High income	9	5	1	6	1	0	3	0
All	**15**	**13**	**5**	**19**	**2**	**2**	**7**	**0**

AFR: African Region; AMR: Region of the Americas; EMR; Eastern Mediterranean Region; EUR: European Region; SEAR: South-East Asia Region; WPR: Western Pacific Region.

7. SUMMARY OF EVIDENCE REVIEWS

7.1 Rationale

A large body of literature documents both the short-term (during childhood) and long-term (in adulthood) health consequences of childhood obesity on noncommunicable diseases. In terms of the more immediate consequences of obesity, there is clear evidence of delayed motor development during infancy, a range of cardiometabolic risk factors in childhood (e.g. dyslipidaemia, high blood pressure, insulin resistance, glucose intolerance), both minor and more major orthopaedic complications, non-alcoholic fatty liver disease, obstructive sleep apnoea, a range of psychosocial complications and, less commonly, type 2 diabetes. The interrelationship between obesity and immune function can have effects on multiple organ systems. Studies consistently report an increased risk of children affected by overweight or obesity becoming adults with overweight or obesity. There is, in turn, a strong association between adult obesity and noncommunicable diseases, such as cardiovascular diseases, type 2 diabetes and some cancers. Childhood obesity, in itself, can also be a risk factor for later cardiovascular disease, diabetes and cancers. Although the level of attributable risk for many noncommunicable diseases arising from childhood obesity is not known, the concept of path dependency suggests that early interventions to improve responses to later noncommunicable disease risks, such as an obesogenic lifestyle, will have major long-term health benefits.

The literature on the economic consequences of childhood obesity is scarce compared with that on the economic burden of adult obesity. However, the evidence on the lifetime health costs of childhood obesity is developing, such as the early onset of adult diseases and a tendency for childhood obesity to track into adulthood. The evidence reviewed, suggests that early onset of obesity in childhood and adolescence has significant economic costs to individuals and society if a life-time perspective is taken. Early onset of obesity and related chronic diseases impair individual life-time educational and labour market outcomes and place a significant long-term burden on health-care systems, employers and society as a whole. A life-course model that could build pathways from childhood obesity to adult health and well-being, and associated economic costs, is missing from the current literature, and is being developed by the Ad hoc Working Group on Science and Evidence.

The psychosocial determinants of childhood obesity include caregiver and individual responses to innate factors and a broad set of determinants in external contexts (e.g. socioeconomic factors, cultural norms relating to eating/feeding, movement, sleep behaviours and body image) as well as health knowledge and ability to act upon this knowledge in ways that mitigate obesity risk.

Differences in obesity prevalence and the nature of interventions that are appropriate and potentially effective in different settings, sociodemographic and cultural groups, need to be considered. In high-income countries, children in ethnic minority populations, including migrants and first-nation children

appear to be at increased risk of overweight and obesity. This may be, in part, due to the different environments and lifestyles, as migrants make different adaptations to a non-traditional environment and lifestyle. Socioeconomic status appears to be inversely associated with overweight and obesity prevalence among children in high-income countries, in an approximately linear manner, rather than applying only to children classified as deprived. There are some exceptions to this association, particularly in countries with more recent economic transition.

In low- and middle-income countries, the highest rates of childhood obesity are found among children of higher socioeconomic status. In these settings the mismatch hypothesis – which postulates that childhood obesity is an outcome of a pattern of intergenerational cycles of undernutrition and subsequent energy abundance – is especially pertinent. Integration of low- and middle-income countries into the global economy is accompanied by increased foreign direct investment in production and distribution of processed foods, and intensification of food marketing practices. Increasing urbanization is associated with decreased physical activity and an increasingly sedentary lifestyle. High quality evidence on psychosocial and developmental aspects of overweight and obesity in low- and middle-income country settings is sparse and it is likely that there is wide variation in the patterns of emergence of childhood obesity.

The evidence base is clear that the marketing of unhealthy, ultra-processed food is causally related to childhood obesity. Any attempt to tackle childhood obesity has, therefore, to include a reduction in the exposure of children and adolescents to such foods and their marketing. To achieve this, it may be necessary to: a) move beyond voluntary measures and employ statutory instruments; b) distinguish healthy from unhealthy foods using independent nutrient profiling (e.g. the PAHO system); and c) regulate all elements of the marketing mix (marketing communications; price promotions; distribution and point-of-sale display; and product design) so that all of these tools are used to support healthy foods only.

7.2 Potential interventions

The Ad hoc Working Group on Science and Evidence discussed the availability of relevant expert consensus recommendations that could be potential reference points for providing updates or aid in the identification of potential ways to reinforce, strengthen or add value to promising approaches. Updating guidance is needed as new evidence emerges. The obesity epidemic is relatively recent but has required timely action. Policy and practice proceed ahead of the science on the basis of the best available evidence at any given point in time. In addition, the epidemic is “a moving target”; population-level forces that cause, or perpetuate, obesity continue to evolve with consequent implications for the relevance of existing evidence and new types of evidence needed. The changing media landscape, with respect to where and how foods and beverages are advertised, is a case in point. Strengthening, or

adding value to, existing guidance is critical to filling gaps and increasing the comprehensiveness and impact of ongoing efforts. The following are examples from a selection of recent documents noted at the Second Meeting of the Ad hoc Working Group on Science and Evidence.

The United States Institute of Medicine Committee on Accelerating Progress in Obesity Prevention was charged with "providing direction on what recommendations, strategies, and actions should be implemented in the short term to accelerate progress in obesity prevention over the next 10 years". The process of developing recommendations included review of nearly 800 previously published recommendations or strategies, assessment of the potential contribution of each to obesity prevention, and selection of a set of strategies considered to have the broadest potential reach and impact.

Guiding principles for selecting strategies to recommend, included the potential for the actions to: be widespread and sustained; drive cultural and societal changes to improve environmental influences on physical activity and food intake; come from multiple sources; and be taken at multiple levels with the involvement of multiple sectors. Each recommendation chosen included an assessment of the potential for high impact; timeliness of effects and ability to promote equity; and feasibility of identifying measurable outcomes. Recommendations (for the USA) were organized according to a conceptual framework, with five domains or settings, as shown below:

Recommendation 1: Physical activity – communities, transportation officials, community planners, health professionals, and governments should make promotion of physical activity a priority by substantially increasing access to places and opportunities for such activity.

Recommendation 2: Food environment – governments and decision-makers in the business community/private sector should make a concerted effort to reduce unhealthy food and beverage options and substantially increase healthier food and beverage options at affordable, competitive prices.

Recommendation 3: Message environment – industry, educators, and governments should act quickly, aggressively, and in a sustained manner on many levels to transform the environment that surrounds the American people with messages about physical activity, food, and nutrition.

Recommendation 4: Health-care and work environments – health-care and health service providers, employers, and insurers should increase the support structure for achieving better population health and obesity prevention.

Recommendation 5: Schools – federal, state, and local government and education authorities, with support from parents, teachers, and the business community and the private sector, should make schools a focal point for obesity prevention.

Each recommendation was accompanied by 3–5 key strategies that were, in turn, illustrated with relevant potential actions. The overall set of recommendations, strategies, and actions was felt to have potential for accelerating progress within each domain and, synergistically among domains. The report is

USA-focused, but some elements can be relevant to other high-income countries. A strong focus on children is evident in these recommendations, although the committee also recognized the importance of addressing obesity-promoting factors affecting the general population.

The World Cancer Research Fund report on Policy and Action for Cancer Prevention developed recommendations for improving diet, nutrition, and physical activity worldwide, including the identification of evidence specifically linking most of these actions to "body fatness, physical activity, and foods and drinks that promote weight gain". The report has a global focus on obesity prevention and includes some recommendations that are relevant to childhood. Recommendations were directed to 11 "actor groups": multinational bodies; civil society organizations; governments; built environment and food industries; media; schools; workplaces and institutions; health and other professionals; and society in general. The report emphasized the importance of synergy among actions of these different groups.

The 2014 Pan American Health Organization's *Plan of action for the prevention of obesity in children and adolescents (341)* was also reviewed and found highly relevant for the work of the Ad hoc Working Group on Science and Evidence's. This is one of two WHO regional action plans with a specific focus on childhood obesity (the second is from the WHO European Region). The five-year plan sets out four main lines of action to help countries reduce rates of child obesity:

1. Breastfeeding promotion: The plan urges countries to promote breastfeeding in their primary health-care services, through the certification of "Baby-Friendly Hospitals," and through stronger enforcement of the International Code of Marketing of Breast-milk Substitutes. Research suggests that longer breastfeeding can reduce rates of obesity and overweight by approximately 10%. Breastfeeding also helps mothers lose weight after pregnancy.

2. Better food and more physical activity in schools: Schools should prevent access to high-calorie, low-nutrient processed food products and sugar-sweetened beverages, and provide students with alternatives of healthier foods and water. Schools should also set aside at least 30 minutes for physical activity during each school day.

3. "Junk food" taxes and restrictions on marketing: Increased taxes can help reduce consumption of sugar-sweetened beverages and unhealthy food products by raising their prices. The plan also urges restrictions on the advertising of unhealthy foods to children and regulations that mandate easy-to-read, front-of-package nutrition labels.

4. Increased access to recreational spaces and nutritious foods: The plan calls for initiatives such as "Sunday bikeways" ("Ciclovías recreativas" in Spanish), which open up city streets on weekends for bicycling and recreation, and urges programmes to support small- and medium-sized farms to help increase the availability of fresh foods.

For practical reasons, the Ad hoc Working Group on Science and Evidence subgroups agreed to a "review of reviews" approach for evidence relevant to the Commission's request for further information about potentially effective interventions to end childhood obesity. This approach was considered expedient given that time did not allow for comprehensive reviews of primary evidence. It was also conservative in that existing good quality published reviews have usually benefitted from extensive evaluation by teams of experts over many months, rated study quality, and synthesized findings, and have already undergone peer-review. However, as noted below, the limitations of reviews based on systematic reviews of narrowly selected randomized trials when applied to public health evidence were also considered.

It is clear from the background and current guidance summarized above that obesity prevention, and prevention of noncommunicable diseases more broadly, require actions to create (or maintain, if still present) environments and policies across the multiple levels and sectors that influence feeding, eating, physical activity and other movement behaviours. These interventions enable and work together with individually-focused interventions that shape child, peer group, parent, and other caregiver behaviours in ways that protect against excess weight gain or, where appropriate, promote and support weight loss. The need for multilevel, multisectoral and synergistic approaches has important implications for the nature of evidence that will be relevant and how the currently available and emerging evidence should be viewed.

Specifically, when considering the types of evidence used to assess approaches to obesity prevention, two traditions have tended to dominate, each with its own limitations. The first, the causal model for studying obesity, targets the factors causing disturbances of energy balance. Using such an approach, much of the resultant evidence base has tended to focus on individual level outcomes and has taken a reductionist view, neglecting broader community contexts and potential explanatory influences. The second tradition emphasizes the use of randomized-controlled trials for determining the effectiveness of obesity interventions. This approach, which works well in more contained and homogeneous clinical settings, has challenges when confronted by the more heterogeneously distributed background and cultural variables that typically influence responses to a health intervention's effect at a population level.

Because of these limitations, public health researchers have argued that different criteria must be applied to build the evidence base for obesity prevention. Evidence evaluation must take into account the need: a) to implement at relatively large scale and across communities and regions; b) to evaluate interventions that comprise multiple components operating synergistically; and c) for generalizability, transferability and sustainability. There is a need for a systems-oriented framework to examine obesity issues that can address such issues as articulated in the Institute of Medicine's L.E.A.D. framework: a) Locate evidence – identify and gather the types of evidence that are potentially relevant to the questions; b) Evaluate evidence – apply standards of quality as relevant to different types of evidence; c)

Assemble evidence – select and summarize the relevant evidence according to considerations for use; and d) inform Decisions – use evidence in the decision-making process.

The assessment of interventions in the earliest periods of life (preconception, early gestation and pregnancy) faced the challenge of combining evidence from studies related to reducing risks of maternal undernutrition, micronutrient deficiency, and overnutrition and obesity, as they may confer risks to the fetus or predispose to risk in infancy. Additional evidence derives from studies of pre-existing maternal (and to a lesser extent paternal) disease such as type 2 diabetes, and from conditions in pregnancy such as gestational diabetes. Such studies have not routinely used childhood obesity as an outcome. Conventional randomized-controlled trials are used in studies of these issues but are only possible where experimentation is appropriate, e.g. nutritional supplementation or efforts to foster appropriate patterns of pregnancy weight gain. Some of the evidence that is potentially the most relevant, scientifically, is from animal models and, while there is no reason to believe that the fundamental biological processes (e.g. epigenetic mechanisms) revealed by these do not operate in humans, nonetheless they cannot be directly used to support population-based health strategies. Longitudinal studies are important potential resources, and several are ongoing, but must be interpreted with sensitivity to potential confounding variables.

The assessment of interventions in children was the most straightforward, although still complex. The review was guided by the following two questions: what are the characteristics of interventions that have been evaluated at each developmental stage within this period? and what is the evidence that they can be effective under ideal conditions or under the circumstances in which they have been implemented? Evidence to answer these questions for children from birth to about age 2 was limited by the availability of relevant evidence reviews of studies with long-term follow up, resulting in the need to supplement with primary review of emerging evidence. Pertinent to assessing the effectiveness of early childhood interventions to prevent obesity in countries where stunting is prevalent, as stunting and obesity co-exist and likely have some overlapping causal factors, this topic was not addressed in the reviews consulted. Several relevant systematic reviews relevant to interventions in school-age children and adolescents were identified as well as one review of community-based interventions. While these were informative, particularly as to potential effectiveness of comprehensive school-based interventions, the evidence identified was generally not informative from a systems perspective and likely underestimates the overall potential for coordinated interventions undertaken in whole communities.

The need for an expanded perspective on identifying relevant evidence is especially relevant to the task of identifying evidence on the effectiveness of interventions involving non-health sectors and gaining a sense of the range of policy interventions which have been recommended or implemented. Sectors considered in the Ad hoc Working Group on Science and Evidence subgroup's review refer to government ministries and their associated civil society partners (nongovernmental organizations, private sector, academia, community groups, etc.). In some cases, the policy recommendations are

based on early or mixed evidence, reflecting the urgency to take action to stem the epidemic rather than wait for "perfect proof of what works, especially in the many areas where interventions are low risk." Questions considered relevant for this aspect of the review included: what makes it important to intervene in this sector? (i.e. what determinants or underlying causes of childhood obesity could be addressed?); what is the range of policy interventions proposed and what is known about their effectiveness? It was emphasized that continued emphasis on policy research and programme evaluation is needed.

The Ad hoc Working Group on Science and Evidence compiled evidence from systematic reviews, original papers and reports, both terms of causality and implementation. Every attempt was made to make these reviews definitive, although given time constraints this cannot be guaranteed. In addition, evidence was assembled on the basis of expert opinion, of particular importance where the links between causes and childhood obesity (as opposed to other strongly related risk factors) or between likely beneficial intervention and childhood obesity as an outcome have not been proven through rigorous methodology, but were nonetheless considered to be very probable. In places, illustrative examples and case studies are included. This should not be taken to imply that the Ad hoc Working Group on Science and Evidence recommends these approaches or sponsors per se.

Primary prevention of child obesity requires particular attention to preconception health and nutrition, as well as maternal health and nutrition, during pregnancy. Strong evidence links preconception maternal weight and macro- and micronutrient status, metabolic health, fitness, and behavioural risk factors, such as tobacco use at conception, to fetal growth and body composition, metabolic and hormonal pathways, and newborn weight and adiposity. These fetal and infant characteristics, in turn, strongly influence later obesity risk. Interventions therefore need to be aimed at ensuring that women and their partners enter pregnancy with a healthy body weight, a healthy, balanced diet with absence of micronutrient deficiencies, good metabolic health, a healthy lifestyle and absence of infections and toxic exposures. Since maternal malnutrition affects fetal development in ways that increase susceptibility to the obesogenic environment, the large suite of interventions shown to be effective for improving maternal health and reducing low birth weight, preterm births, and neonatal mortality can be called upon to also decrease obesity risk in the longer term. These would include strategies such as multiple micronutrient or balanced protein and energy supplementation for at-risk women prior to and during pregnancy.

At the other end of the nutritional spectrum, strong evidence links maternal pre-pregnancy overweight and obesity, excess gestational weight gain and gestational diabetes to higher birth weight, large size for gestational age, macrosomia, and increased risk of poor glucose control in the offspring. A large number of clinical trials using education/advice for lifestyle modification, dietary interventions (reduction of total energy intake, consumption of low glycaemic index diets) or physical activity/exercise interventions have shown modest effects on lowering pregnancy weight gain and reducing pregnancy complications.

There is very limited evidence from low- and middle-income countries, where rates of maternal obesity and gestational diabetes mellitus are increasing rapidly. Non-nutritional interventions, including elimination of maternal smoking toxic exposures and promotion of healthy physical activity, also have great potential for the reduction of child obesity risk.

Individual and population level goals of obesity prevention from birth through to adolescence are multifaceted and include not only a healthy weight trajectory, but also healthy physical, psychosocial, and cognitive growth and development, avoidance of obesity-related comorbidities, health protective eating habits and movement patterns, and a healthy body image. With respect to physical activity, emerging evidence suggests that a more integrated conceptualization of all movement-related behaviours (e.g. not only moderate to vigorous physical activity, but also light/incidental physical activity, sedentary behaviours, sleep) is needed to achieve population-wide energy balance and recalibrate the overall movement behaviour frame of reference.

Interventions to prevent obesity in the earliest years of life (birth to ~age 2 years) focus on: parent/caregiver behaviours related to appropriate child feeding (breastfeeding, timing of complementary foods, composition of complementary foods and adherence to recommendations regarding sugary beverages and snack foods high in fat, sugar, and salt); provision for adequate physical activity and sleep and avoidance of an excess of sedentary behaviours. Particularly detailed consideration was given to evidence on interventions in this age range because of the new science indicating that this is a particularly critical period and because the some of the behavioural targets or developmental issues differ from those relevant once children gain more independence and autonomy. However, this age group has not been covered well by existing systematic reviews. Impressions to date are, therefore, based on a limited search for and primary review of relevant studies, of which some are still in progress and all are from high-income countries. With this caveat, the conclusions are that interventions that begin at, or within, a few months of birth, and that include, but are not limited to, breastfeeding promotion, can benefit obesity prevention in the first 1 or 2 years of life. Of note, some of these interventions have included families of children in socially disadvantaged communities and suggest favourable effects on equity. Benefits after the age of 2 years, may depend on whether age-appropriate interventions are provided during the ensuing years. Interventions in home and family settings may be the most relevant in many contexts. However, interventions delivered in child-care centres or community-based informal care systems are also relevant to children aged 0–2 years in some settings.

Evidence relevant to children aged 2–5 years suggests that interventions in child-care or school settings can be effective, but there is a need for greater clarity about the most effective approaches in specific settings and contexts – including socioeconomic contexts – and more studies overall on which to base conclusions. The evidence supporting interventions in children aged 6–12 years is strongest, and the trends observed in the younger and older children are promising. Most of the relevant interventions

have been conducted in school settings, although for children aged 2–5 years, home and health-care settings might allow for more parental involvement and larger effect sizes. Community-based interventions are also of interest for reaching children and adolescence. There is moderately strong evidence that inclusion of a school component in a community-based intervention is effective for prevention of child overweight and obesity, in that 2 of the 3 studies that involved schools found a statistically significant benefit. Evidence was insufficient to draw conclusions about the other approaches.

Goals of obesity treatment in children and adolescents are the same as for prevention. In addition, when children are already overweight or obese, goals include reduction in the level of overweight, improvement in obesity-related comorbidities and improvement in risk factors for excess weight gain. Evidence reviews of childhood obesity show that family-focused behavioural lifestyle interventions can lead to positive outcomes in weight, BMI and other measures of body fatness. This is the case for both the adolescent (~12–18 years) and pre-adolescent (~5–12 years) age groups. Such an approach is the foundation for all treatment interventions. However, very few of the studies included in evidence reviews have been undertaken in low- and middle-income countries.

The health sector in each country varies considerably and will have different challenges in responding to the need for provision of treatment services for those affected by obesity. There is little written on models of health service delivery for provision of obesity treatment in children and adolescents. The 2013 United Kingdom National Institute for Health and Care Excellence guidelines on lifestyle weight management services for children and young people make a number of recommendations in this regard. While they are United Kingdom-based, many of the key recommendations would apply to other countries. These recommendations include:

a) Ensure family-based, multicomponent lifestyle weight management services for children and young people are available as part of a community-wide, multi-agency approach to promoting a healthy weight and preventing and managing obesity. They should be provided as part of a locally-agreed obesity care or weight management pathway.

b) Dedicate long-term resources to support the development, implementation, delivery, promotion, monitoring and evaluation of these services.

c) Raise awareness of local lifestyle weight management programmes

d) Ensure lifestyle weight management health professional staff are trained and have the necessary knowledge and skills.

The global evidence base around the role and effectiveness of non-health sector policies in obesity prevention is growing, but major gaps remain. In many regions of the world there is also a dearth of local evidence and technical resources available to guide and support policy-makers. The built environment is recognized as a determinant of obesity due to its influence on food intake and physical

activity. There is growing evidence that specific built environment characteristics have an impact on physical activity, such as density (compact neighbourhoods favour physical activity and active transport), design (e.g. the functionality of a neighbourhood in terms of amenities) and transportation (including the provision of safe walking and cycling routes). The food environment refers to the food availability, affordability and marketing in specific settings. Research in this area is relatively new and predominantly of observation in nature. However, it is sufficiently promising to merit consideration and investment from a public policy perspective.

Results of interventions undertaken in the education sector have been mixed – intervening in preschool/child-care settings with young children (aged 0–5 years) and school settings with older children to improve diet and movement behaviours can be successful in improving these behaviours and adiposity. The success of recent programmes has been attributed to the application of a greater range of intervention components, including home-based and parental support components, which were comprehensive and lasted at least one year.

Recent trends in food production, processing, trade, marketing and retailing have contributed to the rise in diet-related noncommunicable diseases. Trade and agriculture policies are powerful forces behind the transition from fresh, minimally processed to ultra-processed food and drink products. Although there is still debate on the effects of agricultural subsidies on overnutrition, there is more consensus and supporting evidence for the use of incentives to promote the production of fruit and vegetables for local market. Nutrition-labelling on pre-packaged foods are a cost-effective population-level intervention. However, governments need to explore new formats for nutrition-labelling and consider an easily-understood labelling system that can be used globally. There is also some evidence that reformulation of foods to reduce trans fats and salt content can reduce intake, which may be of particular relevance in areas where there is already frequent consumption of ultra-processed foods.

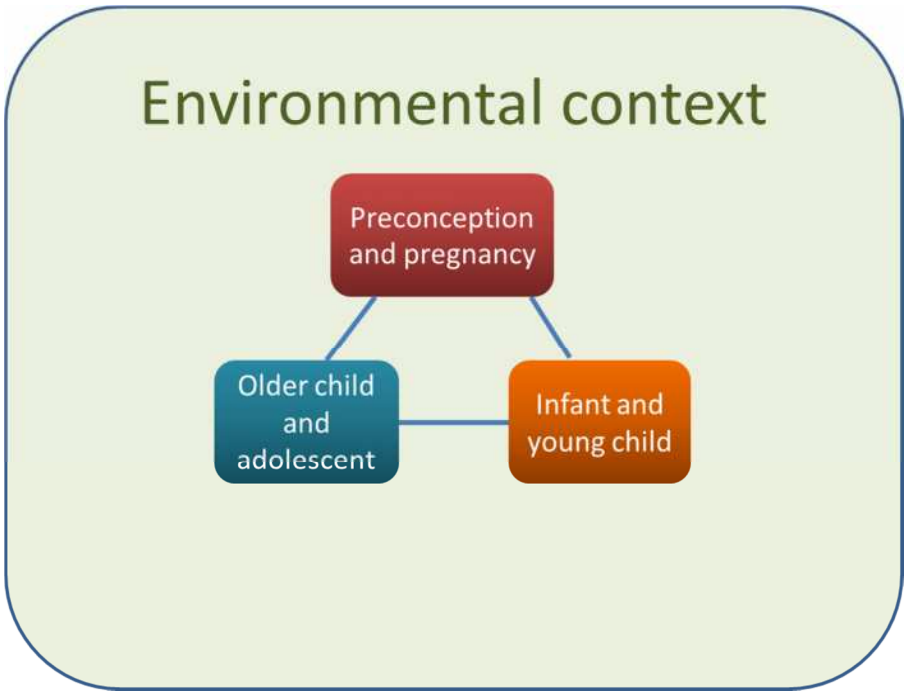
Financial incentives can change behaviour at the individual level. A growing evidence base suggests that changes in the prices of at least some categories of foods and beverages, do lead to changes in weight outcomes, with higher “fast food” prices and sugar-sweetened beverage prices and lower fruit and vegetable prices associated with improved weight outcomes among children and adults, particularly those in lower-income families. Evidence on the effectiveness of tax and other financial incentives to promote physical activity is mixed, with greater evidence at the individual level. Combined or multipronged approaches to taxation on unhealthy and subsidies on healthy food may increase effectiveness and mitigate any negative impact.

8. CONCLUSIONS

Childhood and adolescent obesity necessitates a life-course approach to a chronic risk situation that requires a set of concurrent interventions tailored to the developmental context as well as the physical, social, and economic and wider environment.

The conceptual framework for intervention perspectives that have emanated from the Ad hoc Working Group on Science and Evidence deliberations to date, is shown in Figure 5. As shown, three sensitive periods of the life-course are identified as new or enhanced points of intervention that can influence obesity risk in the context of the encompassing obesogenic environment. Path dependency for obesity risk which develops across the life-course, from conception through infancy and childhood, adolescence and into the pre-conception and pregnancy periods for the next generation, necessitates coordinated and connected interventions at each point, the specific components depending on population characteristics. The life-stage groups require specific intervention packages that should be viewed as sequential and cumulative, as are the underlying risks within each period. Interventions in preconception and pregnancy include those addressed to potential parents of any age and gender. Environmental context refers to interdependent, multilevel and multisectoral modifiable influences on eating and movement across life-stages. Selected influences in this realm are key targets for change in any efforts to end childhood obesity. All the interventions must be placed in the context of interrelated environmental influences. Simultaneously addressing modifiable aspects of these environmental influences is an important part of the strategy.

Figure 5. Simplified schematic overview of the Ad hoc Working Group on Science and Evidence perspectives on interventions



Note: Environmental context includes: global, national and regional; public and private sector; economic and social policies; political and social environments related to food systems and movement behaviours; residential, community, family and cultural environments related to health; health and social services; education; employment; gender roles; resources; and living conditions.

Proposed package of intervention

The Ad hoc Working Group on Science and Evidence stresses that the suggested package of interventions is not intended to be encyclopaedic. Rather, it focuses on existing guidelines where there is a need for more effective implementation and monitoring. It also provides new avenues for intervention, pulling together some key features of WHO guidance in relevant areas, and indicating how these can be leveraged and enhanced from the perspective of the Commission on Ending Childhood Obesity.

The Ad hoc Working Group on Science and Evidence's review of evidence identified a preliminary package of interventions to address the forces and influences that predispose children to gain or retain excess weight, consistent with the overall perspective depicted schematically in Figure 5. These interventions (policies or programmes or both) are seen to be interdependent and mutually reinforcing and will be most effective if applied simultaneously. The evidence indicates that previous intervention strategies, which have not been implemented in conjunction with other reinforcing interventions at different life stages, may have produced limited results because childhood obesity risk is known to be influenced by previous exposure and intergenerational effects.

Recommendations based on these proposed interventions would need to be supported by international agencies, national governments and various subnational jurisdictions, through citizen and community actions, private industry, and nongovernmental organizations, and by sharing responsibility and resources among and across different societal sectors.

The Ad hoc Working Group on Science and Evidence proposes that initiatives resulting from the recommendations of the Commission should be identified as contributing substantially to the United Nations Secretary-General's proposals for Sustainable Development Goals (especially 2, 3, 4 and 5 in the current draft)[1].

The Ad hoc Working Group on Science and Evidence has identified some new pathways for intervention while others emphasize the integration or more effective implementation of current initiatives from a childhood obesity-oriented perspective. All of these recommendations are supported by a combination of logic models and the best available evidence, as highlighted in the annexed papers. For each intervention, a brief rationale is provided indicating how these interventions can advance or add value to current approaches.

Goals of interventions to end childhood obesity

The goals of the interventions are to: a) prevent children and adolescents from developing obesity; and b) treat pre-existing obesity in children and adolescents, in order to reduce the risk of morbidity and mortality due to noncommunicable diseases, the psychosocial effects of obesity both in childhood and adulthood and the transgenerational risk of obesity**.**

Proposed strategic objectives

1) To shift environmental exposures and community capacity to support healthier eating and movement and sleep-related behaviours from preconception through adolescence.
2) To target healthy fetal development and maternal health status with respect to noncommunicable disease prevention as part of overall strategies for optimizing fetal development and birth outcomes.
3) To target healthy child development with respect to obesity and noncommunicable disease prevention as part of overall strategies for optimizing child development in the first five years of life.
4) To take full advantage of educational settings and related community contexts to foster healthy eating and movement environments and behaviours for children and

[1] https://sustainabledevelopment.un.org/post2015/transformingourworld

adolescents while embedding school-oriented efforts into broader whole-community approaches.

A suite of interventions acting across the life-course is needed in order to prevent obesity. Breastfeeding needs to be promoted and supported, and strategies implemented that either preserve or normalize healthy eating, activity and sleep behaviours in infants, children and adolescents. These interventions need to occur in the home, early childhood care, health and education settings, and schools. Whole-of-community interventions also offer great promise for obesity prevention. Preconception and antenatal care to a) diagnose and manage health issues that may impact on fetal health and pregnancy outcomes, and b) encourage healthy eating and movement behaviours, can contribute to reducing the risk of childhood obesity and should be strengthened.

Interventions for promoting a healthier environment

a. **Eliminate exposure of children and adolescents to the marketing of unhealthy foods and beverages through a variety of strategies**.

Rationale: Unhealthy food marketing is causally related to childhood obesity. Triangulation of several different types of quantitative evidence leads to a clear rationale for taking action in this domain as it relates to children, and extant voluntary efforts suggest that this evidence of a need for change is widely agreed. Any attempt to tackle childhood obesity should, therefore include a reduction in exposure of children and adolescents to this marketing (including all 4 "P"s of marketing: products, place, promotions, and prices). This strategy is critical for changing the mix of foods available for purchase, their availability and ease of acquisition, the mix of food products promoted, and food pricing at the point of purchase. The available evidence also supports the case that regulatory and statutory approaches are needed to ensure that changes reach the desirable level and apply to forms of marketing that are not currently covered under voluntary codes. Whether this evidence can be extended to cover exposure of adolescents or of adults (e.g. parents and caregivers) who influence what children eat is doubtful. Assessment of voluntary efforts to change food marketing exposures during the recent decade indicate the need to identify unhealthy foods using independent nutrient profiling (e.g. the PAHO system or overall WHO system if developed).

b. **Run large-scale, sustained social marketing programmes, including, but not limited to, mass media campaigns, with the aim of replacing the current unhealthy commercial marketing environment with a health-promoting one.**

Rationale: Reducing the exposure of children to marketing of unhealthy foods should be accompanied by marketing of healthy foods in order to achieve the goals of healthier eating norms and behaviours. Similar considerations need to be applied to redress the excessive use of screen-based and other sedentary games. The rationale for adding major social marketing campaigns to complement actions is theoretical and based on the Ad hoc

Working Group on Science and Evidence members' knowledge regarding precedents for mounting effective social marketing campaigns. A formal review of potential interventions in this domain could be a next step.

c. Implement fiscal policies, such as taxes on sugary beverages and high-fat, high-sugar manufactured snack foods and subsidies for healthy foods. These strategies could be directly linked to other childhood obesity prevention strategies.

Rationale: Overall, the rationale for and effectiveness of taxation strategies are well-supported by the available evidence in other areas of health promotion. The available evidence makes a solid case for applying this approach to products such as sugary beverages and snack foods. Debate continues about the priority that should be given to taxation strategies in relation to obesity prevention, although the debate appears to centre around feasibility and acceptability of taxation and whether the societally positive effects are sufficient to offset these potential regressive effects on low-income consumers.

Effectiveness of subsidies for healthy food products is also well supported. No reviews of interventions that require tax revenues to be earmarked for health promotion were identified although such provisions are built into some policies, e.g. in Mexico; French Polynesia. The Ad hoc Working Group on Science and Evidence view that it might be possible to also use this strategy for increasing physical activity or reducing sedentary behaviour is theoretical at this point.

d. Create safe, physical-activity friendly communities, which enable the use of active transport and improved access to natural spaces and places/facilities for active living.

Rationale: Recent evidence shows that physical activity declines from the age of school entry, and less than 20% of the global population is sufficiently active by the age of 13–15 years. Low physical activity is now the social norm in most countries, is an important driver of the obesity epidemic, and has a wide range of other adverse consequences (e.g. for risk of diabetes, cardiovascular disease, obstetric complications, mental health and well-being). Recent evidence suggests that obesity, in turn, reduces physical activity, creating a vicious cycle of increasing body fatness and declining physical activity. The rationale for, and effects of, various strategies are supported by numerous association studies (cross-sectional); a more limited number of evaluation studies (e.g. using controlled, before and after designs); and also by logic that may foster consensus even in the absence of overwhelming evidence of causality. The evidence in this domain does not always include assessment of child-specific outcomes but is often interpreted as including positive effects on children.

e. Promote "policy coherence" between health and trade/investment policies and trade/agriculture agreements in order to ensure that international trade/investment law promotes – rather than hinders – public health objectives, and the prevention of childhood obesity more specifically.

Rationale: There is ample evidence documenting the importance of this domain in shaping the context for dietary change and the need to take corrective action. Studies have linked trade liberalization to nutrition transition in, for example, Brazil, Mexico and Pacific Islands, and, where trade agreements and the entry of multinational retailers has increased the availability and reduced prices of ultra-processed food and drink products, increased their consumption and rates of obesity. Guidance on the best set of approaches to intervene in this complex arena cannot be identified based on the information reviewed and may not yet exist. This is not a child specific strategy but can be considered of critical contextual relevance as a part of the mix for addressing childhood obesity.

f. Health-care systems should provide family-based, multicomponent lifestyle weight management services for children and young people affected by obesity as part of a community-wide, multi-agency approach to promoting a healthy lifestyle and preventing and managing obesity. These services should be provided as part of a contextually relevant/locally agreed obesity care or weight management pathway. This also requires measures to ensure that health professional staff are trained and have the necessary knowledge and skills to provide these services.

Rationale: Many children and young people are already affected by obesity, even in low- and middle-income countries. Thus, health services also need to respond to the need for effective treatment of children and young people already affected by obesity. The inadequacy of obesity treatment in health systems can be readily documented. There is currently no coordinated approach to clinical service delivery. Expert guidelines suitable for adaptation to different contexts are available, at least for children aged 5 years and over. This will include the provision of family-based lifestyle intervention programmes in primary, secondary and tertiary clinical care settings.

g. Improve access to healthy foods through strengthening of local and regional food systems, by, for example, encouraging support for local farmers/agriculture, production, processing and retailers.**

Rationale: Influencing the food environment requires a collaborative approach to food production, processing and accessibility, availability and affordability. Where access to healthy foods is limited, ultra-processed foods are often the only alternative available and affordable. There is ample documentation of aspects of food systems that can be potential targets for change, i.e. why action is needed and appropriate. The Ad hoc Working Group on Science and Evidence did not identify estimates of the potential specific impact of these changes at the consumer or child level.

h. Build legal capacity within all sectors of government to ensure childhood obesity prevention policies are compliant with national and international laws including treaties and agreements.

Rationale: The law provides significant opportunities to improve the habitable environment and thus promote healthier lifestyles. Nevertheless, these opportunities can only be maximized if the constraints that the law imposes on policy-makers are understood and adequately taken into account at all stages of the policy process. In the expert opinion of the Ad hoc Working Group on Science and Evidence, policies intended to end childhood obesity (as any other policies) must comply with a range of higher norms derived from constitutional law, international trade and investment law, and fundamental rights law. If they do not, they may be annulled in a judicial review action. Consequently, all sectors of government responsible for the adoption of relevant policies must have sufficient legal capacity to ensure that these policies can withstand legal challenges.

i. Ensure that all policies comply with the UN Convention on the Rights of the Child and take the best interest of the child as a primary consideration.

Rationale: The United Nations has adopted several legally binding international human rights treaties and agreements that are used as a framework for applying human rights. Through these instruments, the principles and rights they outline become legal obligations on those UN States choosing to be bound by them. In particular, the United Nations Convention on the Rights of the Child has recognized that children are a vulnerable group of society requiring special protection. Thus, Article 3(1) provides that "in all actions concerning children, whether undertaken by public or private social welfare institutions, courts of law, administrative authorities or legislative bodies, the best interests of the child shall be a primary consideration". Several articles of the Convention and other fundamental rights instruments could be invoked to end childhood obesity, including the right to the enjoyment of the highest attainable standard of health, the right of the child to engage in play and recreational activities appropriate to the age of the child, the right of the child to be protected from economic exploitation, the right to adequate food. In recent years, the Committee on the Rights of the Child and other bodies entrusted with the interpretation of fundamental rights have explicitly called on UN States to address obesity, and childhood obesity more specifically, as part of their commitments to apply fundamental rights.

Interventions for preconception and pregnancy (fetal outcomes)

j. Integrate current guidance for preconception and antenatal care with guidance for noncommunicable disease prevention applicable to this life stage.

Rationale: Current guidance for preconception and antenatal care incorporates measures to prevent other forms of fetal malnutrition. Evidence shows that maternal undernutrition, maternal overweight or obesity, excess pregnancy weight gain, maternal hyperglycaemia

(including gestational diabetes), smoking or exposure to toxins are modifiable pre-conceptional or gestational influences that increase the likelihood of obesity during infancy and childhood. Guidelines that address both undernutrition and obesity risk are clearly needed, and there is, as yet, no evidence on the content or effectiveness of interventions that do this in settings where undernutrition is prevalent. Some evidence is available to support the development of interventions to address subsequent childhood obesity risk commencing during preconception and pregnancy in high-income countries; also, these interventions prevent other adverse pregnancy outcomes. Likewise, there is some evidence for preconception interventions reducing the risk of developing gestational diabetes. There is evidence for the beneficial effects of exercise programmes in pregnancy on maternal BMI, gestational weight gain and birth outcomes linked to risk of childhood obesity, although the effect size varies. The recommendation to target this phase of the life-course is also based on substantial basic science evidence and the Ad hoc Working Group on Science and Evidence's expert opinion that it will contribute to improving health and nutritional literacy and preparation for pregnancy and parenthood.

Interventions that integrate guidance related to all forms of malnutrition should address undernutrition, overnutrition and specific nutritional deficiencies, relevant to the double burden associated with transitions to urban and westernised lifestyles in low- and middle-income countries. Implications include the need to screen for and appropriate management of pre-existing diabetes mellitus and hypertension in pregnant women, and early diagnosis and effective management of gestational diabetes and pregnancy-induced hypertension as well as depression and mental health problems; gestational weight gain pattern, dietary quality and movement behaviours; and avoiding elective caesarean section deliveries for non-medical reasons wherever possible. This period is also a good opportunity for promoting awareness of the importance of exclusive breastfeeding and healthy complementary infant feeding and other aspects of parenting which affect the development of infant appetite, food preference, cardiometabolic control, growth and neurocognitive development.

Interventions for infants and young children

k. Integrate targets related to healthful eating/feeding and movement behaviours with current guidance on best practices for parenting and child care during the first 5 years of life. This could involve strengthening existing Infant and Young Child Feeding guidelines (ICYF) to include a focus on specific categories of foods (e.g. sugar-sweetened beverages or poor quality, high energy-dense foods) for prevention of excess weight gain.

Rationale: Established guidance for early child feeding primarily targets undernutrition. Although guidelines that address both undernutrition and obesity risk are clearly needed in

some countries, evidence related to intervention content and effectiveness for addressing both types of malnutrition may not exist. The evidence to support early interventions to prevent obesity in high-income countries is still emerging but looks very promising. Evidence supports interventions in preschool and child-care settings for children aged 2–5 years for early child feeding, activity patterns, media exposures, and sleep – all of which help to promote healthy behaviours and weight trajectories in this period of life. These recommendations are compatible with recommendations for the prevention of undernutrition while adding additional dimensions. Breastfeeding and appropriate complementary feeding (timing of introduction and nature of the foods used) are favourable for preventing all forms of malnutrition in infancy and early childhood. Several strategies in this age group have also supported parents and caregivers to ensure minimal television/screen viewing, encourage active play, establish healthy eating behaviours and diets, promote healthy sleep routines and role model healthy parental and family lifestyles. Each element of this suite of interventions appears important, and all support healthy child growth and development.

l. States should respect, protect, and promote women's rights to breastfeed their children, according to guidelines, using regulatory measures as needed.

Rationale: Breastfeeding is core to preventing undernutrition, and evidence supports its potential value as part of a comprehensive strategy for childhood obesity prevention. It may also be beneficial for postnatal weight management in women. Given changes in women's lifestyles and roles, the ability to breastfeed outside of the home and sustain breastfeeding when a mother returns to work will be critical to enable achievement of recommendations. Policies that establish rights of women and responsibilities of employers are needed. Some are in place but they should be universal in contexts where needed.

m. Use whole-of-community approaches to improve/strengthen processes and environments for early child care and education and parenting support in:

- **communities at large;**
- **the curricula and eating/physical activity environments in formal child-care settings or institutions;**
- **health-care delivery (e.g. primary or paediatric care); and**
- **through social institutions and community leaders (e.g. religious leaders).**

These strategies should explicitly seek to empower parents, other caregivers and community leaders to foster healthful eating and movement behaviours in environmental contexts that currently support and promote unhealthy behaviours. Approaches that integrate advice on healthy eating and movement behaviours for children with services that address other social and health needs of families and children may provide economies and foster sustainability.

Rationale: The evidence shows that interventions to improve child nutrition and movement behaviours are most effective if these are comprehensive and involve parents and the

community at large. Societal changes and transitions require a more deliberate and concerted approach to interventions in this domain, including support for parents and other caregivers to enable them to contribute to the recommended behaviour changes.

Interventions for older children and adolescents

n. **Strengthen the infrastructure for implementing whole-of-school programmes that promote healthy eating, physical activity and reduce sedentary behaviours, including environmental modification (e.g. addressing available food choices and recreational spaces) and engage parents and the community, including health-care providers and community health workers. Programmes should use evidence-based approaches, appropriately adapted to context. Given that schools are under the jurisdictions of educational authorities and the importance of sustainability, this strategy should include partnerships and resource-sharing between education and health ministries. The programmes require provision of appropriate training and resources for schools and school-based community activities, and statutory changes to curricula. They should ensure clarity of message and economy of scale by building on existing platforms, for example for promotion of reproductive health and communicable disease prevention. The programmes should ensure the engagement of older children and adolescents as actors.**

Rationale: There is a relatively large and convincing evidence base to support interventions in school settings and the wider community for pre-adolescent and adolescent children as an obesity prevention strategy. Qualitative assessments suggest that their effectiveness on obesity prevention behaviours and outcomes is related to a) quality of implementation and b) positioning school-based efforts within the context of broader community efforts. The most frequently mentioned challenge to implementation is competition with the schools' primary mission.

o. **Improve local access to healthy food retailers, restrict unhealthy food access around schools (e.g. through zoning) and increase access to potable water in schools.**

Rationale: Energy-dense foods and sugar-sweetened beverages are important drivers of the obesity epidemic in school-age children and adolescents globally, acting to both cause and maintain overweight and obesity. Increasing access to, and promotion of, lower energy-dense foods, and to water as an alternative to sugar-sweetened drinks, are actions necessary to make the environment less obesogenic, and to establish healthier behavioural norms.

References

1. UNICEF, WHO, World Bank. Levels and trends in child malnutrition: UNICEF-WHO-World Bank joint child malnutrition estimates. UNICEF, New York; WHO, Geneva; World Bank, Washington DC: 2015.
2. WHO Multicentre Growth Reference Study Group. WHO child growth standards based on length/height, weight and age. Acta Paediatr. 2006;Suppl 450:76–85.
3. Ng M, Fleming T, Robinson M, Thomson B, Graetz N, Margono C, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the global burden of disease study 2013. Lancet. 2014;384:766–81.
4. Katzmarzyk PT, Barlow S, Bouchard C, Catalano PM, Hsia DS, Inge TH, et al. An evolving scientific basis for the prevention and treatment of pediatric obesity. Int J Obes (Lond). 2014;38:887–905.
5. Bhattacharjee R, Kim J, Kheirandish-Gozal L, Gozal D. Obesity and obstructive sleep apnea syndrome in children: a tale of inflammatory cascades. Pediatr Pulmonol. 2011;46:313–23.
6. Litwin SE. Childhood obesity and adulthood cardiovascular disease: quantifying the lifetime cumulative burden of cardiovascular risk factors. J Am Coll Cardiol. 2014;64:1588–90.
7. Nader PR, O'Brien M, Houts R, Bradley R, Belsky J, Crosnoe R, et al. Identifying risk for obesity in early childhood. Pediatrics. 2006;118:e594–601.
8. Kelsey MM, Zaepfel A, Bjornstad P, Nadeau KJ. Age-related consequences of childhood obesity. Gerontology. 2014;60:222–8.
9. Gore FM, Bloem PJN, Patton GC, Ferguson J, Joseph V, Coffey C, et al. Global burden of disease in young people aged 10–24 years: a systematic analysis. Lancet. 2011;377:2093–102.
10. Lobstein T, Jackson-Leach R. Estimated burden of paediatric obesity and co-morbidities in Europe. Part 2. Numbers of children with indicators of obesity-related disease. Int J Pediatr Obes. 2006;1:33-41.
11. Flynn J. The changing face of pediatric hypertension in the era of the childhood obesity epidemic. Pediatr Nephrol. 2013;28:1059–66.
12. Yan S, Li J, Li S, Zhang B, Du S, Gordon-Larsen P, et al. The expanding burden of cardiometabolic risk in China: the China Health and Nutrition Survey. Obes Rev. 2012;13:810–21.
13. Craigie AM, Lake AA, Kelly SA, Adamson AJ, Mathers JC. Tracking of obesity-related behaviours from childhood to adulthood: a systematic review. Maturitas. 2011;70:266–84.
14. Slining M, Adair LS, Goldman BD, Borja JB, Bentley M. Infant overweight is associated with delayed motor development. J Pediatr. 2010;157:20–5.e1.
15. Papoutsakis C, Priftis KN, Drakouli M, Prifti S, Konstantaki E, Chondronikola M, et al. Childhood overweight/obesity and asthma: is there a link? A systematic review of recent epidemiologic evidence. J Acad Nutr Diet. 2013;113:77–105.
16. Van Name M, Santoro N. Type 2 diabetes mellitus in pediatrics: a new challenge. World J Pediatr. 2013;9:293–9.
17. Cook S, Kavey RE. Dyslipidemia and pediatric obesity. Pediatr Clin North Am. 2011;58:1363–73, ix.
18. Stolzman S, Bement MH. Inflammatory markers in pediatric obesity: health and physical activity implications. Infant Child Adolesc Nutr. 2012;4:297–302.
19. Katz J, Bimstein E. Pediatric obesity and periodontal disease: a systematic review of the literature. Quintessence Int. 2011;42:595–9.
20. Wearing SC, Hennig EM, Byrne NM, Steele JR, Hills AP. The impact of childhood obesity on musculoskeletal form. Obes Rev. 2006;7:209–18.

21. Paulis WD, Silva S, Koes BW, Middelkoop M. Overweight and obesity are associated with musculoskeletal complaints as early as childhood: a systematic review. Obes Rev. 2014;15:52–67.
22. Fennoy I. Effect of obesity on linear growth. Curr Opin Endocrinol Diabetes Obes. 2013;20:44–9.
23. Pizzi MA, Vroman K. Childhood obesity: effects on children's participation, mental health, and psychosocial development. Occup Ther Health Care. 2013;27:99–112.
24. Miller AL, Lee HJ, Lumeng JC. Obesity-associated biomarkers and executive function in children. Pediatr Res. 2014;77:143–7
25. Reinert KR, Po'e EK, Barkin SL. The relationship between executive function and obesity in children and adolescents: a systematic literature review. J Obes. 2013;2013:820956.
26. Mahe E, Gnossike P, Sigal ML. [childhood psoriasis]. Arch Pediatr. 2014;21:778–86.
27. Pacifico L, Nobili V, Anania C, Verdecchia P, Chiesa C. Pediatric nonalcoholic fatty liver disease, metabolic syndrome and cardiovascular risk. World J Gastroenterol. 2011;17:3082–91.
28. Yau PL, Castro MG, Tagani A, Tsui WH, Convit A. Obesity and metabolic syndrome and functional and structural brain impairments in adolescence. Pediatrics. 2012;130:e856–e64.
29. Savino A, Pelliccia P, Chiarelli F, Mohn A. Obesity-related renal injury in childhood. Horm Res Paediatr. 2010;73:303–11.
30. Luoto R, Collado MC, Salminen S, Isolauri E. Reshaping the gut microbiota at an early age: functional impact on obesity risk? Ann Nutr Metab. 2013;63 Suppl 2:17–26.
31. Rosenfield RL. Clinical review: Adolescent anovulation: maturational mechanisms and implications. J Clin Endocrinol Metab. 2013;98:3572–83.
32. Lee YS. Consequences of childhood obesity. Ann Acad Med Singapore. 2009;38:75–7.
33. Singh AS, Mulder C, Twisk JW, van Mechelen W, Chinapaw MJ. Tracking of childhood overweight into adulthood: a systematic review of the literature. Obes Rev. 2008;9:474–88.
34. Park MH, Falconer C, Viner RM, Kinra S. The impact of childhood obesity on morbidity and mortality in adulthood: a systematic review. Obes Rev. 2012;13:985–1000.
35. Inge T, King WC, Jenkins TM, Courcoulas AP, Mitsnefes M, Flum DR, et al. The effect of obesity in adolescence on adult health status. Pediatrics. 2013;132:1098–104.
36. Lloyd LJ, Langley-Evans SC, McMullen S. Childhood obesity and risk of the adult metabolic syndrome: a systematic review. Int J Obes (Lond). 2012;36:1–11.
37. Codoner-Franch P, Valls-Belles V, Arilla-Codoner A, Alonso-Iglesias E. Oxidant mechanisms in childhood obesity: the link between inflammation and oxidative stress. Transl Res. 2011;158:369–84.
38. Chaffee BW, Weston SJ. Association between chronic periodontal disease and obesity: a systematic review and meta-analysis. J Periodontol. 2010;81:1708–24.
39. Garcia-Ptacek S, Faxen-Irving G, Cermakova P, Eriksdotter M, Religa D. Body mass index in dementia. Eur J Clin Nutr. 2014;68:1204–09.
40. Giorgio V, Prono F, Graziano F, Nobili V. Pediatric non alcoholic fatty liver disease: old and new concepts on development, progression, metabolic insight and potential treatment targets. BMC Pediatr. 2013;13:40.
41. Wills AK, Black S, Cooper R, Coppack RJ, Hardy R, Martin KR, et al. Life course body mass index and risk of knee osteoarthritis at the age of 53 years: evidence from the 1946 British birth cohort study. Ann Rheum Dis. 2012;71:655–60.
42. Macfarlane GJ, de Silva V, Jones GT. The relationship between body mass index across the life course and knee pain in adulthood: results from the 1958 birth cohort study. Rheumatology (Oxford, England). 2011;50:2251–6.
43. Bjørge T, Tretli S, Engeland A. Relation of height and body mass index to renal cell carcinoma in two million Norwegian men and women. Am J Epidemiol. 2004;160:1168–76.
44. Bjørge T, Engeland A, Tverdal A, Smith GD. Body mass index in adolescence in relation to cause-specific mortality: a follow-up of 230,000 Norwegian adolescents. Am J Epidemiol. 2008;168:30–7.

45. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. Int J Obes (Lond). 2011;35:891–8.
46. Frisco ML, Weden M. Early adult obesity and U.S. Women's lifetime childbearing experiences. J Marriage Fam. 2013;75:920–32.
47. Lepe M, Bacardi Gascon M, Castaneda-Gonzalez LM, Perez Morales ME, Jimenez Cruz A. Effect of maternal obesity on lactation: systematic review. Nutr Hosp. 2011;26:1266–9.
48. Misra A, Bhardwaj S. Obesity and the metabolic syndrome in developing countries: focus on south Asians. Nestlé Nutr Inst Workshop Ser. 2014;78:133–40.
49. Bastien M, Poirier P, Lemieux I, Despres JP. Overview of epidemiology and contribution of obesity to cardiovascular disease. Prog Cardiovasc Dis. 2014;56:369–81.
50. Roberson LL, Aneni EC, Maziak W, Agatston A, Feldman T, Rouseff M, et al. Beyond BMI: the "metabolically healthy obese" phenotype & its association with clinical/subclinical cardiovascular disease and all-cause mortality – a systematic review. BMC Public Health. 2014;14:14.
51. Janssen I, Shields M, Craig CL, Tremblay MS. Changes in the obesity phenotype within Canadian children and adults, 1981 to 2007–2009. Obesity. 2012;20:916–9.
52. Barnes W, Gartland M, Stack M. Old habits die hard: path dependency and behavioral lock-in. Journal of Economic Issues. 2004;38:371.
53. Hanson MA, Gluckman PD. Early developmental conditioning of later health and disease: physiology or pathophysiology? Physiol Rev. 2014;94:1027–76.
54. Skinner AC, Steiner MJ, Henderson FW, Perrin EM. Multiple markers of inflammation and weight status: cross-sectional analyses throughout childhood. Pediatrics. 2010;125:801–9.
55. Singer K, Eng DS, Lumeng CN, Gebremariam A, Lee JM. The relationship between body fat mass percentiles and inflammation in children. Obesity. 2014;22:1332–6.
56. Taveras EM, Gillman MW, Kleinman KP, Rich-Edwards JW, Rifas-Shiman SL. Reducing racial/ethnic disparities in childhood obesity: the role of early life risk factors. JAMA Pediatr. 2013;167:731–8.
57. Schooling M, Leung GM, Janus ED, Ho SY, Hedley AJ, Lam TH. Childhood migration and cardiovascular risk. Int J Epidemiol. 2004;33:1219–26.
58. Booth HP, Prevost TA, Wright AJ, Gulliford MC. Effectiveness of behavioural weight loss interventions delivered in a primary care setting: a systematic review and meta-analysis. Fam Pract. 2014;31:643–53.
59. Waters E, de Silva-Sanigorski A, Hall BJ, Brown T, Campbell KJ, Gao Y, et al. Interventions for preventing obesity in children. Cochrane Database Syst Rev. 2011:CD001871.
60. Nader PR, Huang TT, Gahagan S, Kumanyika S, Hammond RA, Christoffel KK. Next steps in obesity prevention: altering early life systems to support healthy parents, infants, and toddlers. Child Obes. 2012;8:195–204.
61. Black RE, Victora CG, Walker SP, Bhutta ZA, Christian P, de Onis M, et al. Maternal and child undernutrition and overweight in low-income and middle-income countries. Lancet. 2013;382:427–51.
62. de Onis M, Blossner M. Prevalence and trends of overweight among preschool children in developing countries. Am J Clin Nutr. 2000;72:1032–9.
63. de Onis M, Blossner M, Borghi E. Global prevalence and trends of overweight and obesity among preschool children. Am J Clin Nutr. 2010;92:1257–64.
64. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. JAMA. 2014;311:806–14.
65. Wang Y, Baker JL, Hill JO, Dietz WH. Controversies regarding reported trends: has the obesity epidemic leveled off in the United States? Adv Nutr. 2012;3:751–2.
66. Olds T, Maher C, Zumin S, Peneau S, Lioret S, Castetbon K, et al. Evidence that the prevalence of childhood overweight is plateauing: data from nine countries. Int J Pediatr Obes. 2011;6:342–60.

67. El-Sayed AM, Scarborough P, Galea S. Ethnic inequalities in obesity among children and adults in the UK: a systematic review of the literature. Obes Rev. 2011;12:e516–34.
68. Kumanyika S, Taylor WC, Grier SA, Lassiter V, Lancaster KJ, Morssink CB, et al. Community energy balance: a framework for contextualizing cultural influences on high risk of obesity in ethnic minority populations. Prev Med. 2012;55:371–81.
69. Brug J, van Stralen MM, Chinapaw MJ, De Bourdeaudhuij I, Lien N, Bere E, et al. Differences in weight status and energy-balance related behaviours according to ethnic background among adolescents in seven countries in Europe: the ENERGY-project. Pediatr Obes. 2012;7:399–411.
70. Weden MM, Brownell P, Rendall MS. Prenatal, perinatal, early life, and sociodemographic factors underlying racial differences in the likelihood of high body mass index in early childhood. Am J Public Health. 2012;102:2057–67.
71. Gaillard R, Durmus B, Hofman A, Mackenbach JP, Steegers EA, Jaddoe VW. Risk factors and outcomes of maternal obesity and excessive weight gain during pregnancy. Obesity (Silver Spring). 2013;21:1046–55.
72. Heslehurst N, Sattar N, Rajasingam D, Wilkinson J, Summerbell CD, Rankin J. Existing maternal obesity guidelines may increase inequalities between ethnic groups: a national epidemiological study of 502,474 births in England. BMC Pregnancy Childbirth. 2012;12:156.
73. Taveras EM, Gillman MW, Kleinman KP, Rich-Edwards JW, Rifas-Shiman SL. Reducing racial/ethnic disparities in childhood obesity: the role of early life risk factors. JAMA Pediatr. 2013;167:731–8.
74. Chu SY, Callaghan WM, Bish CL, D'Angelo D. Gestational weight gain by body mass index among US women delivering live births, 2004–2005: fueling future obesity. Am J Obstet Gynecol. 2009;200:271 e1–7.
75. Retnakaran R, Hanley AJ, Connelly PW, Sermer M, Zinman B. Ethnicity modifies the effect of obesity on insulin resistance in pregnancy: a comparison of Asian, south Asian, and Caucasian women. J Clin Endocrinol Metab. 2006;91:93–7.
76. Taveras EM, Gillman MW, Kleinman K, Rich-Edwards JW, Rifas-Shiman SL. Racial/ethnic differences in early-life risk factors for childhood obesity. Pediatrics. 2010;125:686–95.
77. Homer CS, Sheehan A, Cooke M. Initial infant feeding decisions and duration of breastfeeding in women from English, Arabic and Chinese-speaking backgrounds in Australia. Breastfeed Rev. 2002;10:27–32.
78. Sharma A, Lynch MA, Irvine ML. The availability of advice regarding infant feeding to immigrants of Vietnamese origin: a survey of families and health visitors. Child Care Health Dev. 1994;20:349–54.
79. Kumar BN, Holmboe-Ottesen G, Lien N, Wandel M. Ethnic differences in body mass index and associated factors of adolescents from minorities in Oslo, Norway: a cross-sectional study. Public Health Nutr. 2004;7:999–1008.
80. Kuepper-Nybelen J, Lamerz A, Bruning N, Hebebrand J, Herpertz-Dahlmann B, Brenner H. Major differences in prevalence of overweight according to nationality in preschool children living in Germany: determinants and public health implications. Arch Dis Child. 2005;90:359–63.
81. Dassanayake J, Dharmage SC, Gurrin L, Sundararajan V, Payne WR. Are Australian immigrants at a risk of being physically inactive? Int J Behav Nutr Phys Act. 2011;8:53
82. Due P, Damsgaard MT, Rasmussen M, Holstein BE, Wardle J, Merlo J, et al. Socioeconomic position, macroeconomic environment and overweight among adolescents in 35 countries. Int J Obes (Lond). 2009;33:1084–93.
83. Singh GK, Kogan MD, Van Dyck PC, Siahpush M. Racial/ethnic, socioeconomic, and behavioral determinants of childhood and adolescent obesity in the United States: analyzing independent and joint associations. Ann Epidemiol. 2008;18:682–95.

84. Duncan MJ, Al-Nakeeb Y, Nevill AM. Body esteem and body fat in British school children from different ethnic groups. Body Image. 2004;1:311–5.
85. Duncan MJ, Al-Nakeeb Y, Nevill AM, Jones MV. Body dissatisfaction, body fat and physical activity in british children. Int J Pediatr Obes. 2006;1:89–95.
86. Balistreri KS, Van Hook J. Socioeconomic status and body mass index among hispanic children of immigrants and children of natives. Am J Public Health. 2009;99:2238–46.
87. Young-Hyman D, Schlundt DG, Herman-Wenderoth L, Bozylinski K. Obesity, appearance, and psychosocial adaptation in young African American children. J Pediatr Psychol. 2003;28:463–72.
88. Hemmingsson E. A new model of the role of psychological and emotional distress in promoting obesity: conceptual review with implications for treatment and prevention. Obes Rev. 2014;15:769–79.
89. Jodkowska M, Oblacinska A, Tabak I. Overweight and obesity among adolescents in Poland: gender and regional differences. Public Health Nutr. 2010;13:1688–92.
90. Chranowska M, Koziel S, Ulijaszek S. Changes in BMI and the prevalence of overweight and obesity in children and adolescents in Cracow, Poland, 1971–2000. Econ Hum Biol. 2007;5:370–8.
91. Yang Z, Huffman SL. Nutrition in pregnancy and early childhood and associations with obesity in developing countries. Matern Child Nutr. 2013;9 Suppl 1:105–19.
92. Pearce J, Taylor MA, Langley-Evans SC. Timing of the introduction of complementary feeding and risk of childhood obesity: a systematic review. Int J Obes (Lond). 2013;37:1295–306.
93. Sweeting HN. Gendered dimensions of obesity in childhood and adolescence. Nutr J. 2008;7:1.
94. Gupta N, Goel K, Shah P, Misra A. Childhood obesity in developing countries: epidemiology, determinants, and prevention. Endocr Rev. 2012;33:48–70.
95. Gordon-Larsen P, Wang H, Popkin BM. Overweight dynamics in Chinese children and adults. Obes Rev. 2014;15 Suppl 1:37–48.
96. Yang J. China's one-child policy and overweight children in the 1990s. Soc Sci Med. 2007;64:2043–57.
97. Song Y, Wang HJ, Ma J, Wang Z. Secular trends of obesity prevalence in urban Chinese children from 1985 to 2010: gender disparity. PLoS One. 2013;8:e53069.
98. Shan XY, Xi B, Cheng H, Hou DQ, Wang Y, Mi J. Prevalence and behavioral risk factors of overweight and obesity among children aged 2–18 in Beijing, China. Int J Pediatr Obes. 2010;5:383–9.
99. Hawkes C. Uneven dietary development: linking the policies and processes of globalization with the nutrition transition, obesity and diet-related chronic diseases. Global Health. 2006;2:4.
100. Huffman SL, Piwoz EG, Vosti SA, Dewey KG. Babies, soft drinks and snacks: a concern in low- and middle-income countries? Matern Child Nutr. 2014;10:562–74.
101. Chen X, Beydoun MA, Wang Y. Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. Obesity (Silver Spring). 2008;16:265–74.
102. Al-Kandari YY. Prevalence of obesity in Kuwait and its relation to sociocultural variables. Obes Rev. 2006;7:147–54.
103. Boodai SA, Reilly JJ. Health related quality of life of obese adolescents in Kuwait. BMC Pediatr. 2013;13:105.
104. Moussa MA, Shaltout AA, Nkansa-Dwamena D, Mourad M, Alsheikh N, Agha N, et al. Factors associated with obesity in Kuwaiti children. Eur J Epidemiol. 1999;15:41–9.
105. Shisana O, Labadarios D, Rehle T, Simbayi L, Zuma K, Dhansay A. South African National Health and Nutrition Examination Survey (SANHANES-1). Cape Town: HSRC Press, 2013.
106. Steyn NP, Labadarios D, Maunder E, Nel J, Lombard C, Directors of the National Food Consumption S. Secondary anthropometric data analysis of the national food consumption survey in South Africa: The double burden. Nutrition. 2005;21:4–13.

107. Igumbor EU, Sanders D, Puoane TR, Tsolekile L, Schwarz C, Purdy C, et al. "Big food," the consumer food environment, health, and the policy response in South Africa. PLoS Med. 2012;9:e1001253.
108. Temple NJ, Steyn NP. The cost of a healthy diet: a South African perspective. Nutrition. 2011;27:505–8.
109. Rossouw HA, Grant CC, Viljoen M. Overweight and obesity in children and adolescents: the South African problem. South African Journal of Science. 2012;108:31–7.
110. Popkin BM, Adair LS, Ng SW. Global nutrition transition and the pandemic of obesity in developing countries. Nutr Rev. 2012;70:3–21.
111. Offer A, Pechey R, Ulijaszek S. Obesity under affluence varies by welfare regimes: the effect of fast food, insecurity, and inequality. Econ Hum Biol. 2010;8:297–308.
112. Hawkes C, Lobstein T. Regulating the commercial promotion of food to children: a survey of actions worldwide. Int J Pediatr Obes. 2011;6:83–94.
113. Helliwell JF. How's life? Combining individual and national variables to explain subjective well-being. Economic Modelling. 2003;20:331–60.
114. Diener E, Helliwell JF, Kahneman D. International differences in well-being Oxford: Oxford University Press; 2010.
115. Helliwell JF, Wang S. The state of world happiness. New York: Earth Institute, Columbia University, 2013.
116. Kahneman D, Riis J. Living, and thinking about it: two perspectives on life. In: Huppert FA, Baylis N, Keverne B, editors. The science of well-being. Oxford: Oxford University Press; 2005.
117. Finklestein A, Luttmer EFP, Notowidigdo MJ. What good is wealth without health? The effect of health on the marginal utility of consumption. Journal of the European Economic Association. 2013;11:221–58.
118. Jalan J, Somanathan E. The importance of being informed: Experimental evidence on demand for environmental quality. Journal of Development Economics. 2008;87:14–28.
119. Kahneman D, Slovic P, Tversky A. Judgment under uncertainty: heuristics and biases. Cambridge: Cambridge University Press; 1982.
120. Kahneman D. Thinking, fast and slow. Princeton: Princeton University Press; 2011.
121. World Commission on Environment and Development. Our common future. New York: Oxford University Press; 1987.
122. Arrow KJ, et al. Sustainability and the measurement of wealth. Environment and Development Economics. 2012;17:317–55.
123. Arrow KJ, et al. Sustainability and the measurement of wealth: further reflections. Environment and Development Economics. 2013;18:504–16.
124. Dasgupta P, Mäler K-G. Net national product, wealth, and social well-being. Environment and Development Economics. 2000;5:69–93.
125. Little IMD, Mirrlees JA. Project appraisal and planning for developing countries. London: Heinemann; 1974.
126. Dasgupta P. Human well-being and the natural environment. Oxford: Oxford University Press; 2004.
127. Dee A, Kearns K, O'Neill C, Sharp L, Staines A, O'Dwyer V, et al. The direct and indirect costs of both overweight and obesity: a systematic review. BMC Res Notes. 2014;7:242.
128. Lehnert T, Sonntag D, Konnopka A, Riedel-Heller S, Konig HH. Economic costs of overweight and obesity. Best Pract Res Clin Endocrinol Metab. 2013;27:105–15.
129. Finkelstein EA, Graham WC, Malhotra R. Lifetime direct medical costs of childhood obesity. Pediatrics. 2014;133:854–62.

130. John J, Wenig CM, Wolfenstetter SB. Recent economic findings on childhood obesity: Cost-of-illness and cost-effectiveness of interventions. Curr Opin Clin Nutr Metab Care. 2010;13:305–13.
131. John J, Wolfenstetter SB, Wenig CM. An economic perspective on childhood obesity: recent findings on cost of illness and cost effectiveness of interventions. Nutrition. 2012;28:829–39.
132. Pelone F, Specchia ML, Veneziano MA, Capizzi S, Bucci S, Mancuso A, et al. Economic impact of childhood obesity on health systems: a systematic review. Obes Rev. 2012;13:431–40.
133. Batscheider A, Rzehak P, Teuner CM, Wolfenstetter SB, Leidl R, von Berg A, et al. Development of bmi values of German children and their healthcare costs. Econ Hum Biol. 2014;12:56–66.
134. Breitfelder A, Wenig CM, Wolfenstetter SB, Rzehak P, Menn P, John J, et al. Relative weight-related costs of healthcare use by children-results from the two German birth cohorts, GINI-plus and LISA-plus. Econ Hum Biol. 2011;9:302–15.
135. Finkelstein EA, DiBonaventura MD, Burgess SM, Hale BC. The costs of obesity in the workplace. J Occup Environ Med. 2010;52:971–6.
136. Kuhle S, Kirk S, Ohinmaa A, Yasui Y, Allen AC, Veugelers PJ. Use and cost of health services among overweight and obese Canadian children. Int J Pediatr Obes. 2011;6:142–8.
137. Trasande L, Elbel B. The economic burden placed on healthcare systems by childhood obesity. Expert Rev Pharmacoecon Outcomes Res. 2012;12:39–45.
138. Trasande L, Chatterjee S. The impact of obesity on health service utilization and costs in childhood. Obesity (Silver Spring). 2009;17:1749–54.
139. Anaya P, Melendez G. Estimate costs of comorbidites in overweight and obese Mexican children aged between five and eleven years until death. Value Health. 2009;12:A135–A.
140. Muller-Riemenschneider F, Reinhold T, Berghofer A, Willich SN. Health-economic burden of obesity in Europe. Eur J Epidemiol. 2008;23:499–509.
141. Sassi F. Obesity and the economics of prevention. Fit not fat. Organisation for Economic Co-operation and Development publishing, Paris: 2010.
142. Schmier JK, Jones ML, Halpern MT. Cost of obesity in the workplace. Scand J Work Environ Health. 2006;32:5–11.
143. Thompson D, Wolf AM. The medical-care cost burden of obesity. Obes Rev. 2001;2:189–97.
144. Knai C, Suhrcke M, Lobstein T. Obesity in eastern Europe: an overview of its health and economic implications. Econ Hum Biol. 2007;5:392–408.
145. Rice BP. Estimating the cost of illness. Am J Public Health. 1967;57.
146. Cooper BS, Rice DP. Economic cost of illness revisited. Soc Secur Bull. 1976;39:21–36.
147. Rice DP. Cost-of-illness studies: fact or fiction? Lancet. 1994;344:1519–20.
148. Zhao W, Zhai Y, Hu J, Wang J, Yang Z, Kong L, et al. Economic burden of obesity-related chronic diseases in mainland China. Obes Rev. 2008;9 Suppl 1:62–7.
149. Kesztyus D, et al. Illness and determinants of health-related quality of life in a cross-sectional sample of schoolchildren in different weight categories. Ger Med Sci. 2014;12:1–9.
150. Cawley J, Spiess CK. Obesity and skill attainment in early childhood. NBER Working Paper, No 13997, National Bureau of Economic Research, 2008.
151. Datar A, Sturm R. Childhood overweight and elementary school outcomes. Int J Obes. 2006;30:1449–60.
152. Caird J, et al. Childhood obesity and educational attainment: a systematic review. London: EPPI-Centre, Social Science Research Unit, Institute of Education, University of London, 2011.
153. Halfon N, Larson K, Slusser W. Associations between obesity and comorbid mental health, developmental, and physical health conditions in a nationally representative sample of US children aged 10 to 17. Acad Pediatr. 2013;13:6–13.
154. Konnopka A, Bodemann M, Konig HH. Health burden and costs of obesity and overweight in Germany. Eur J Health Econ. 2011;12:345–52.

155. Tsiros MD, Olds T, Buckley JD, Grimshaw P, Brennan L, Walkley J, et al. Health-related quality of life in obese children and adolescents. Int J Obes (Lond). 2009;33:387–400.
156. Wang LY, Denniston M, Lee S, Galuska D, Lowry R. Long-term health and economic impact of preventing and reducing overweight and obesity in adolescence. J Adolesc Health. 2010;46:467–73.
157. Dor A, Ferguson C, Langwith C, Tan E. A heathy burden: the individual costs of being overweight and obese in the United States. Washington, DC: Department of Health Policy, School of Public Health and Health Services, the George Washington University.
158. Trasande L. How much should we invest in preventing childhood obesity? Health Aff (Millwood). 2010;29:372–8.
159. Mennella JA. Ontogeny of taste preferences: Basic biology and implications for health. Am J Clin Nutr. 2014;99:704S–11S.
160. Rozin P. "Taste-smell confusions" and the duality of the olfactory sense. Percept Psychophys. 1982;31:397–401.
161. Forestell CA, Mennella JA. The ontogeny of taste perception and preference throughout childhood. In: Doty RL, editor. Handbook of olfaction and gustation. 2nd ed. New York: Marcel Dekker, Inc.; 2003. p.823–946.
162. Ganchrow JR, Mennella JA. The ontogeny of human flavor perception. In: Doty RL, ed. Handbook of olfaction and gustation. 2nd ed. New York: Marcel Dekker, Inc.; 2003. p.823–946.
163. Mennella JA, Reed DR, Mathew PS, Roberts KM, Mansfield CJ. "A spoonful of sugar helps the medicine go down": bitter masking by sucrose among children and adults. Chem Senses. 2015;40:17–25.
164. Mennella JA, Reed DR, Roberts KM, Mathew PS, Mansfield CJ. Age-related differences in bitter taste and efficacy of bitter blockers. PLoS One. 2014;9:e103107.
165. Desor JA, Maller O, Turner RE. Taste in acceptance of sugars by human infants. J Comp Physiol Psychol. 1973;84:496–501.
166. Steiner JE. Facial expressions of the neonate infant indicating the hedonics of food-related chemical stimulii. Taste and development: the genesis of sweet preference. Washington DC: US Government Printing Office, 1977.
167. Rosenstein D, Oster H. Differential facial responses to four basic tastes in newborns. Child Dev. 1988;59:1555–68.
168. Maone TR, Mattes RD, Bernbaum JC, Beauchamp GK. A new method for delivering a taste without fluids to preterm and term infants. Dev Psychobiol. 1990;23:179–91.
169. Barr RG, Quek VS, Cousineau D, Oberlander TF, Brian JA, Young SN. Effects of intra-oral sucrose on crying, mouthing and hand-mouth contact in newborn and six-week-old infants. Dev Med Child Neurol. 1994;36:608–18.
170. Berridge KC, Kringelbach ML. Affective neuroscience of pleasure: reward in humans and animals. Psychopharmacology (Berl). 2008;199:457–80.
171. Blass EM, Shah A. Pain-reducing properties of sucrose in human newborns. Chem Senses. 1995;20:29–35.
172. Stevens B, Yamada J, Ohlsson A. Sucrose for analgesia in newborn infants undergoing painful procedures. Cochrane Database Syst Rev. 2010:CD001069.
173. Stevens B, Yamada J, Ohlsson A. Sucrose for analgesia in newborn infants undergoing painful procedures. Cochrane Database Syst Rev. 2013:CD001069.
174. Barr RG, Pantel MS, Young SN, Wright JH, Hendricks LA, Gravel R. The response of crying newborns to sucrose: is it a "sweetness" effect? Physiol Behav. 1999;66:409–17.
175. Bucher HU, Baumgartner R, Bucher N, Seiler M, Fauchere JC. Artificial sweetener reduces nociceptive reaction in term newborn infants. Early Hum Dev. 2000;59:51–60.
176. Riordan J, Wamback K. Breastfeeding and human lactation. Sudbury, MA: Jones and Barlett Learning; 2010.

177. Morse JM, Jehle C, Gamble D. Initiating breastfeeding: a world survey of the timing of postpartum breastfeeding. Int J Nurs Stud. 1990;27:303–13.
178. Mennella JA. The sweet taste of childhood. In: Firestein S, Beauchamp GK, editors. The senses: a comprehensive reference. Vol 4: olfaction and taste. San Diego, CA: Elsevier; 2008. p.183–8.
179. Roberts IF, Roberts GJ. Relation between medicines sweetened with sucrose and dental disease. Br Med J. 1979;2:14–6.
180. Pawar S, Kumar A. Issues in the formulation of drugs for oral use in children: role of excipients. Paediatr Drugs. 2002;4:371–9.
181. Feldens CA, Giugliani ER, Duncan BB, Drachler Mde L, Vitolo MR. Effect of a nutritional intervention on childhood caries: reply to Seow. J Evid Based Dent Pract. 2011;11:164-5.
182. Prakasha Shrutha S, Vinit GBG, Giri KY, Alam S. Feeding practices and early childhood caries: a cross-sectional study of preschool children in kanpur district, India. ISRN dentistry. 2013;2013:275193.
183. Feldens CA, Giugliani ER, Vigo A, Vitolo MR. Early feeding practices and severe early childhood caries in four-year-old children from southern Brazil: a birth cohort study. Caries Res. 2010;44:445–52.
184. Warren JJ, Weber-Gasparoni K, Marshall TA, Drake DR, Dehkordi-Vakil F, Dawson DV, et al. A longitudinal study of dental caries risk among very young low SES children. Community Dent Oral Epidemiol. 2009;37:116–22.
185. Coldwell SE, Oswald TK, Reed DR. A marker of growth differs between adolescents with high vs. low sugar preference. Physiol Behav. 2009;96:574–80.
186. Mennella JA, Finkbeiner S, Lipchock SV, Hwang LD, Reed DR. Preferences for salty and sweet tastes are elevated and related to each other during childhood. PLoS One. 2014;9:e92201.
187. Mennella JA, Lukasewycz LD, Griffith JW, Beauchamp GK. Evaluation of the Monell forced-choice, paired-comparison tracking procedure for determining sweet taste preferences across the lifespan. Chem Senses. 2011;36:345–55.
188. Mennella JA, Pepino MY, Lehmann-Castor SM, Yourshaw LM. Sweet preferences and analgesia during childhood: effects of family history of alcoholism and depression. Addiction. 2010;105:666–75.
189. Fushan AA, Simons CT, Slack JP, Manichaikul A, Drayna D. Allelic polymorphism within the TAS1R3 promoter is associated with human taste sensitivity to sucrose. Curr Biol. 2009;19:1288–93.
190. Pepino MY, Mennella JA. Factors contributing to individual differences in sucrose preference. Chem Senses. 2005;30 Suppl 1:i319–20.
191. Bleich SN, Barry CL, Gary-Webb TL, Herring BJ. Reducing sugar-sweetened beverage consumption by providing caloric information: how black adolescents alter their purchases and whether the effects persist. Am J Public Health. 2014;104:2417–24.
192. Powell LM, Wada R, Kumanyika SK. Racial/ethnic and income disparities in child and adolescent exposure to food and beverage television ads across the U.S. Media markets. Health Place. 2014;29:124–31.
193. Miller A, Barr RG, Young SN. The cold pressor test in children: Methodological aspects and the analgesic effect of intraoral sucrose. Pain. 1994;56:175–83.
194. Pepino MY, Mennella JA. Sucrose-induced analgesia is related to sweet preferences in children but not adults. Pain. 2005;119:210–8.
195. Beauchamp GK, Moran M. Acceptance of sweet and salty tastes in 2-year-old children. Appetite. 1984;5:291–305.
196. Birch LL, McPhee L, Steinberg L, Sullivan S. Conditioned flavor preferences in young children. Physiol Behav. 1990;47:501–5.
197. Liem DG, Mennella JA. Sweet and sour preferences during childhood: role of early experiences. Dev Psychobiol. 2002;41:388–95.
198. Beauchamp GK, Cowart BJ. Congenital and experiential factors in the development of human flavor preferences. Appetite. 1985;6:357–72.

199. Mattes RD, Popkin BM. Nonnutritive sweetener consumption in humans: effects on appetite and food intake and their putative mechanisms. Am J Clin Nutr. 2009;89:1–14.
200. Drewnowski A, Mennella JA, Johnson SL, Bellisle F. Sweetness and food preference. J Nutr. 2012;142:1142S–8S.
201. Swithers SE. Artificial sweeteners are not the answer to childhood obesity. Appetite. 2015;93:85–90.
202. Green E, Murphy C. Altered processing of sweet taste in the brain of diet soda drinkers. Physiol Behav. 2012;107:560–7.
203. Cogswell ME, Gunn JP, Yuan K, Park S, Merritt R. Sodium and sugar in complementary infant and toddler foods sold in the United States. Pediatrics. 2015;135:416–23.
204. Beauchamp GK, Cowart BJ, Moran M. Developmental changes in salt acceptability in human infants. Dev Psychobiol. 1986;19:17–25.
205. Beauchamp GK, Cowart BJ. Preference for high salt concentrations among children. Dev Psychol. 1990;26:539–45.
206. Harris G. Development of taste and food preferences in children. Curr Opin Clin Nutr Metab Care. 2008;11:315–9.
207. Stein LJ, Cowart BJ, Beauchamp GK. The development of salty taste acceptance is related to dietary experience in human infants: a prospective study. Am J Clin Nutr. 2012;95:123–9.
208. Geleijnse JM, Hofman A, Witteman JC, Hazebroek AA, Valkenburg HA, Grobbee DE. Long-term effects of neonatal sodium restriction on blood pressure. Hypertension. 1997;29:913–7.
209. Henney JE, Taylor CE, Boon CS. Strategies to reduce sodium intake in the United States. Washington DC: Institue of Medicine, 2010.
210. Bertino M, Beauchamp GK, Engelman K. Long-term reduction in dietary sodium alters the taste of salt. Am J Clin Nutr. 1982;36:1134–44.
211. Beauchamp GK, Bertino M, Engelman K. Modification of salt taste. Ann Intern Med. 1983;98:763–9.
212. Mattes RD. The taste for salt in humans. Am J Clin Nutr. 1997;65:692S–7S.
213. Bertino M, Beauchamp GK, Engelman K. Increasing dietary salt alters salt taste preference. Physiol Behav. 1986;38:203–13.
214. Meyerhof W, Batram C, Kuhn C, Brockhoff A, Chudoba E, Bufe B, et al. The molecular receptive ranges of human TAS2R bitter taste receptors. Chem Senses. 2010;35:157–70.
215. Nelson G, Hoon MA, Chandrashekar J, Zhang Y, Ryba NJ, Zuker CS. Mammalian sweet taste receptors. Cell. 2001;106:381–90.
216. Adler E, Hoon MA, Mueller KL, Chandrashekar J, Ryba NJ, Zuker CS. A novel family of mammalian taste receptors. Cell. 2000;100:693–702.
217. Glendinning JI. Is the bitter rejection response always adaptive? Physiol Behav. 1994;56:1217–27.
218. Steiner JE, Glaser D, Hawilo ME, Berridge KC. Comparative expression of hedonic impact: affective reactions to taste by human infants and other primates. Neurosci Biobehav Rev. 2001;25:53–74.
219. Kajiura H, Cowart BJ, Beauchamp GK. Early developmental change in bitter taste responses in human infants. Dev Psychobiol. 1992;25:375–86.
220. Mennella JA, Pepino MY, Reed DR. Genetic and environmental determinants of bitter perception and sweet preferences. Pediatrics. 2005;115:e216–22.
221. Mennella JA, Pepino MY, Duke FF, Reed DR. Age modifies the genotype-phenotype relationship for the bitter receptor TAS2R38. BMC Genet. 2010;11:60.
222. Mennella JA, Spector AC, Reed DR, Coldwell SE. The bad taste of medicines: overview of basic research on bitter taste. Clin Ther. 2013;35:1225–46.

223. Zeinstra GG, Koelen MA, Kok FJ, de Graaf C. Cognitive development and children's perceptions of fruit and vegetables; a qualitative study. Int J Behav Nutr Phys Act. 2007;4:30.
224. Moskowitz HW, Kumaraiah V, Sharma KN, Jacobs HL, Sharma SD. Cross-cultural differences in simple taste preferences. Science. 1975;190:1217–8.
225. Glass TA, McAtee MJ. Behavioral science at the crossroads in public health: extending horizons, envisioning the future. Soc Sci Med. 2006;62:1650–71.
226. Mennella JA. The chemical senses and the development of flavor preferences in humans. In: Hale TW, Hartman RE, editors. Textbook on human lactation. Amarillo, Texas: Hale Publishing; 2007. p.403–14.
227. Mennella JA, Trabulsi JC. Complementary foods and flavor experiences: setting the foundation. Ann Nutr Metab. 2012;60 Suppl 2:40–50.
228. Mennella JA, Jagnow CP, Beauchamp GK. Prenatal and postnatal flavor learning by human infants. Pediatrics. 2001;107:E88.
229. Galloway AT, Lee Y, Birch LL. Predictors and consequences of food neophobia and pickiness in young girls. J Am Diet Assoc. 2003;103:692–8.
230. Cooke LJ, Wardle J, Gibson EL, Sapochnik M, Sheiham A, Lawson M. Demographic, familial and trait predictors of fruit and vegetable consumption by pre-school children. Public Health Nutr. 2004;7:295–302.
231. Nicklaus S, Boggio V, Issanchou S. Food choices at lunch during the third year of life: high selection of animal and starchy foods but avoidance of vegetables. Acta Paediatr. 2005;94:943–51.
232. Skinner JD, Carruth BR, Wendy B, Ziegler PJ. Children's food preferences: a longitudinal analysis. J Am Diet Assoc. 2002;102:1638–47.
233. Trabulsi JC, Mennella JA. Diet, sensitive periods in flavour learning, and growth. Int Rev Psychiatry. 2012;24:219–30.
234. Mennella JA, Ventura AK, Beauchamp GK. Differential growth patterns among healthy infants fed protein hydrolysate or cow-milk formulas. Pediatrics. 2011;127:110–8.
235. Ventura AK, Beauchamp GK, Mennella JA. Infant regulation of intake: the effect of free glutamate content in infant formulas. Am J Clin Nutr. 2012;95:875–81.
236. Mennella JA, Trabulsi JC, Inamdar LB. The sensory world of formula-fed infants: differences among artificial milk feedings in flavor learning and satiation. In: Preedy VR, Watson RR, Zibadi S, editors. Handbook of dietary and nutritional aspects of bottle feeding. Wageningen: Wageningen Academic Publishers; 2014.
237. Forestell CA, Mennella JA. Early determinants of fruit and vegetable acceptance. Pediatrics. 2007;120:1247–54.
238. Mennella JA, Nicklaus S, Jagolino AL, Yourshaw LM. Variety is the spice of life: strategies for promoting fruit and vegetable acceptance during infancy. Physiol Behav. 2008;94:29–38.
239. Coulthard H, Harris G, Fogel A. Exposure to vegetable variety in infants weaned at different ages. Appetite. 2014;78:89–94.
240. Birch LL, McPhee L, Shoba BC, Pirok E, Steinberg L. What kind of exposure reduces children's food neophobia? Looking vs. tasting. Appetite. 1987;9:171–8.
241. Sullivan SA, Birch LL. Infant dietary experience and acceptance of solid foods. Pediatrics. 1994;93:271–7.
242. Anzman-Frasca S, Savage JS, Marini ME, Fisher JO, Birch LL. Repeated exposure and associative conditioning promote preschool children's liking of vegetables. Appetite. 2012;58:543–53.
243. Mangold JE, Hill DL. Extensive reorganization of primary afferent projections into the gustatory brainstem induced by feeding a sodium-restricted diet during development: less is more. J Neurosci. 2007;27:4650–62.

244. Kim SA, Moore LV, Galuska D, Wright AP, Harris D, Grummer-Strawn LM, et al. Vital signs: fruit and vegetable intake among children – United States, 2003–2010. Morb Mortal Wkly Rep. 2014;63:671–6.
245. Bell KI, Tepper BJ. Short-term vegetable intake by young children classified by 6-n-propylthoiuracil bitter-taste phenotype. Am J Clin Nutr. 2006;84:245–51.
246. Nicklaus S, Boggio V, Chabanet C, Issanchou S. A prospective study of food preferences in childhood. Food Quality and Preference. 2004;15:805–18.
247. Nicklaus S, Boggio V, Chabanet C, Issanchou S. A prospective study of food variety seeking in childhood, adolescence and early adult life. Appetite. 2005;44:289–97.
248. Resnicow K, Smith M, Baranowski T, Baranowski J, Vaughan R, Davis M. 2-year tracking of children's fruit and vegetable intake. J Am Diet Assoc. 1998;98:785–9.
249. Skinner JD, Carruth BR, Bounds W, Ziegler P, Reidy K. Do food-related experiences in the first 2 years of life predict dietary variety in school-aged children? J Nutr Educ Behav. 2002;34:310–5.
250. Fildes A, van Jaarsveld CH, Llewellyn C, Wardle J, Fisher A. Parental control over feeding in infancy. Influence of infant weight, appetite and feeding method. Appetite. 2015;91:101–6.
251. Larsen JK, Hermans RC, Sleddens EF, Engels RC, Fisher JO, Kremers SP. How parental dietary behavior and food parenting practices affect children's dietary behavior. Interacting sources of influence? Appetite. 2015;89:246–57.
252. Llewellyn CH, Trzaskowski M, van Jaarsveld CH, Plomin R, Wardle J. Satiety mechanisms in genetic risk of obesity. JAMA Pediatr. 2014;168:338–44.
253. Heinig MJ, Nommsen LA, Peerson JM, Lonnerdal B, Dewey KG. Energy and protein intakes of breast-fed and formula-fed infants during the first year of life and their association with growth velocity: the Darling study. Am J Clin Nutr. 1993;58:152–61.
254. Li R, Fein SB, Grummer-Strawn LM. Association of breastfeeding intensity and bottle-emptying behaviors at early infancy with infants' risk for excess weight at late infancy. Pediatrics. 2008;122 Suppl 2:S77–84.
255. Li R, Magadia J, Fein SB, Grummer-Strawn LM. Risk of bottle-feeding for rapid weight gain during the first year of life. Arch Pediatr Adolesc Med. 2012;166:431–6.
256. Mihrshahi S, Battistutta D, Magarey A, Daniels LA. Determinants of rapid weight gain during infancy: baseline results from the nourish randomised controlled trial. BMC Pediatr. 2011;11:99.
257. Baird J, Fisher D, Lucas P, Kleijnen J, Roberts H, Law C. Being big or growing fast: systematic review of size and growth in infancy and later obesity. BMJ. 2005;331:929.
258. Evelein AM, Visseren FL, van der Ent CK, Grobbee DE, Uiterwaal CS. Excess early postnatal weight gain leads to thicker and stiffer arteries in young children. J Clin Endocrinol Metab. 2013;98:794–801.
259. Ong KK, Emmett P, Northstone K, Golding J, Rogers I, Ness AR, et al. Infancy weight gain predicts childhood body fat and age at menarche in girls. J Clin Endocrinol Metab. 2009;94:1527–32.
260. Skilton MR, Marks GB, Ayer JG, Garden FL, Garnett SP, Harmer JA, et al. Weight gain in infancy and vascular risk factors in later childhood. Pediatrics. 2013;131:e1821–8.
261. Stettler N, Iotova V. Early growth patterns and long-term obesity risk. Curr Opin Clin Nutr Metab Care. 2010;13:294–9.
262. Crow RA, Fawcett JN, Wright P. Maternal behavior during breast- and bottle-feeding. J Behav Med. 1980;3:259–77.
263. Ventura AK, Pollack Golen R. A pilot study comparing opaque, weighted bottles with conventional, clear bottles for infant feeding. Appetite. 2015;85:178–84.
264. Dunn J. Feeding and sleeping. In: Rutter M, editor. The scientific foundations of developmental psychiatry. London: Heinemann Medical Books; 1980.

265. Ventura AK, Inamdar LB, Mennella JA. Consistency in infants' behavioural signalling of satiation during bottle-feeding. Pediatr Obes. 2015;10:180–7.
266. Adair LS. The infant's ability to self-regulate caloric intake: a case study. J Am Diet Assoc. 1984;84:543–6.
267. Hauser B, Keymolen K, Blecker U, Suys B, Bougatef A, Loeb H, et al. A comparative evaluation of whey hydrolysate and whey-predominant formulas. How well do infants accept and tolerate them? Clin Pediatr (Phila). 1993;32:433–7.
268. Gluckman PD, Hanson MA. Developmental and epigenetic pathways to obesity: an evolutionary-developmental perspective. Int J Obes (Lond). 2008;32 Suppl 7:S62–71.
269. Lumeng JC, Ozbeki TN, Appugliese DP, Kaciroti N, Corwyn RF, Bradley RH. Observed assertive and intrusive maternal feeding behaviors increase child adiposity. Am J Clin Nutr. 2012;95:640–7.
270. Rollins BY, Loken E, Savage JS, Birch LL. Effects of restriction on children's intake differ by child temperament, food reinforcement, and parent's chronic use of restriction. Appetite. 2014;73:31–9.
271. Birch LL, Anzman-Frasca S, Paul IM. Starting early: Obesity prevention during infancy. Nestle Nutr Inst Workshop Ser. 2012;73:81–94.
272. Savage JS, Fisher JO, Birch LL. Parental influence on eating behavior: conception to adolescence. J Law Med Ethics. 2007;35:22–34.
273. Worobey H, Ostapkovich K, Yudin K, Worobey J. Trying versus liking fruits and vegetables: correspondence between mothers and preschoolers. Ecol Food Nutr. 2010;49:87–97.
274. Fisher JO, Mitchell DC, Smiciklas-Wright H, Birch LL. Parental influences on young girls' fruit and vegetable, micronutrient, and fat intakes. J Am Diet Assoc. 2002;102:58–64.
275. Duffy VB, Bartoshuk LM, Striegel-Moore R, Rodin J. Taste changes across pregnancy. Ann N Y Acad Sci. 1998;855:805–9.
276. Pope JF, Skinner JD, Carruth BR. Cravings and aversions of pregnant adolescents. J Am Diet Assoc. 1992;92:1479–82.
277. Rayburn WF, Phelan ST. Promoting healthy habits in pregnancy. Obstet Gynecol Clin North Am. 2008;35:385–400, viii.
278. Anderson AS. Symposium on 'nutritional adaptation to pregnancy and lactation'. Pregnancy as a time for dietary change? Proc Nutr Soc. 2001;60:497–504.
279. Olson CM. Tracking of food choices across the transition to motherhood. J Nutr Educ Behav. 2005;37:129–36.
280. Olson CM, Bove CF, Miller EO. Growing up poor: Long-term implications for eating patterns and body weight. Appetite. 2007;49:198–207.
281. Lumley J, Oliver SS, Chamberlain C, Oakley L. Interventions for promoting smoking cessation during pregnancy. Cochrane Database Syst Rev. 2004:CD001055.
282. Resnicow K, Davis-Hearn M, Smith M, Baranowski T, Lin LS, Baranowski J, et al. Social-cognitive predictors of fruit and vegetable intake in children. Health Psychol. 1997;16:272–6.
283. Hawkes C, Smith TG, Jewell J, Wardle J, Hammond RA, Friel S, et al. Smart food policies for obesity prevention. Lancet. 2015;385:2410–21.
284. Grissom N, Bowman N, Reyes TM. Epigenetic programming of reward function in offspring: a role for maternal diet. Mamm Genome. 2014;25:41–8.
285. Kickbusch I, Pelikan JM, Apfel F, Tsouros AD. Health literacy: the solid facts. Geneva: World Health Organization, 2013.
286. Nutbeam D. Health literacy as a public health goal: a challenge for contemporary health education and communication strategies into the 21st century. Health Promotion International. 2000;15:259–67.
287. Nutbeam D. The evolving concept of health literacy. Soc Sci Med. 2008;67:2072-8.

288. James DCS, Harville C, Efunbumi O, Martin MY. Health literacy issues surrounding weight management among African American women: a mixed methods study. Journal of Human Nutrition and Dietetics. 2015;28:41–9.
289. Carbone ET, Zoellner JM. Nutrition and health literacy: a systematic review to inform nutrition research and practice. J Acad Nutr Diet. 2012;112:254–65.
290. Sanders LM, Shaw JS, Guez G, Baur C, Rudd R. Health literacy and child health promotion: implications for research, clinical care, and public policy. Pediatrics. 2009;124 Suppl 3:S306–14.
291. Gibbs H, Chapman-Novakofski K. Establishing content validity for the nutrition literacy assessment instrument. Prev Chronic Dis. 2013;10:E109.
292. Carbone ET. Measuring nutrition literacy: problems and potential solutions. J Nutr Disorders Ther. 2012;03.
293. Guttersrud O, Dalane JO, Pettersen S. Improving measurement in nutrition literacy research using rasch modelling: examining construct validity of stage-specific 'critical nutrition literacy' scales. Public Health Nutr. 2014;17:877–83.
294. Ross CE, Wu CL. The links between education and health. Am Sociol Rev. 1995;60:719–45.
295. Glanz K, Sallis JF, Saelens BE, Frank LD. Healthy nutrition environments: concepts and measures. Am J Health Promot. 2005;19:330–3, ii.
296. Diamond JJ. Development of a reliable and construct valid measure of nutritional literacy in adults. Nutr J. 2007;6:5.
297. Spence AC, Campbell KJ, Crawford DA, McNaughton SA, Hesketh KD. Mediators of improved child diet quality following a health promotion intervention: the Melbourne inFANT Program. Int J Behav Nutr Phys Act. 2014;11:137.
298. Yin HS, Sanders LM, Rothman RL, Shustak R, Eden SK, Shintani A, et al. Parent health literacy and "obesogenic" feeding and physical activity-related infant care behaviors. J Pediatr. 2014;164:577–83 e1.
299. Taggart J, Williams A, Dennis S, Newall A, Shortus T, Zwar N, et al. A systematic review of interventions in primary care to improve health literacy for chronic disease behavioral risk factors. BMC Fam Pract. 2012;13:49.
300. Vaitkeviciute R, Ball LE, Harris N. The relationship between food literacy and dietary intake in adolescents: a systematic review. Public Health Nutr. 2015;18:649–58.
301. Brooks N, Begley A. Adolescent food literacy programmes: a review of the literature. Nutrition & Dietetics. 2014;71:158–71.
302. Vidgen HA, Gallegos D. Defining food literacy and its components. Appetite. 2014;76:50–9.
303. Zoellner J, You W, Connell C, Smith-Ray RL, Allen K, Tucker KL, et al. Health literacy is associated with healthy eating index scores and sugar-sweetened beverage intake: findings from the rural lower mississippi delta. J Am Diet Assoc. 2011;111:1012–20.
304. Ciampa PJ, Kumar D, Barkin SL, Sanders LM, Yin HS, Perrin EM, et al. Interventions aimed at decreasing obesity in children younger than 2 years a systematic review. Arch Pediatr Adolesc Med. 2010;164:1098–104.
305. Sanders LM, Perrin EM, Yin HS, Bronaugh A, Rothman RL, Team GS. "Greenlight study": a controlled trial of low-literacy, early childhood obesity prevention. Pediatrics. 2014;133:E1724–E37.
306. Spronk I, Kullen C, Burdon C, O'Connor H. Relationship between nutrition knowledge and dietary intake. Br J Nutr. 2014;111:1713–26.
307. Wardle J, Parmenter K, Waller J. Nutrition knowledge and food intake. Appetite. 2000;34:269–75.
308. Haack SA, Byker CJ. Recent population adherence to and knowledge of United States federal nutrition guides, 1992–2013: a systematic review. Nutr Rev. 2014;72:613–26.
309. Barker M, Lawrence W, Robinson S, Baird J. Food labelling and dietary behaviour: bridging the gap. Public Health Nutr. 2012;15:758–9.

310. World Health Organization. Global strategy on diet, physical activity and health. Geneva: World Health Organization, 2004.
311. Thorndike AN, Riis J, Sonnenberg LM, Levy DE. Traffic-light labels and choice architecture: promoting healthy food choices. Am J Prev Med. 2014;46:143–9.
312. Cowburn G, Stockley L. Consumer understanding and use of nutrition labelling: a systematic review. Public Health Nutr. 2005;8:21–8.
313. Kleef EV, Dagevos H. The growing role of front-of-pack nutrition profile labeling: a consumer perspective on key issues and controversies. Crit Rev Food Sci Nutr. 2015;55:291–303.
314. Campos S, Doxey J, Hammond D. Nutrition labels on pre-packaged foods: a systematic review. Public Health Nutr. 2011;14:1496–506.
315. Sonnenberg L, Gelsomin E, Levy DE, Riis J, Barraclough S, Thorndike AN. A traffic light food labeling intervention increases consumer awareness of health and healthy choices at the point-of-purchase. Prev Med. 2013;57:253–7.
316. Sinclair SE, Cooper M, Mansfield ED. The influence of menu labeling on calories selected or consumed: a systematic review and meta-analysis. Journal of the Academy of Nutrition and Dietetics. 2014;114:1375–88.e15.
317. Pignone M, DeWalt DA, Sheridan S, Berkman N, Lohr KN. Interventions to improve health outcomes for patients with low literacy. A systematic review. J Gen Intern Med. 2005;20:185–92.
318. Hoelscher DM, Kirk S, Ritchie L, Cunningham-Sabo L, Comm AP. Position of the academy of nutrition and dietetics: interventions for the prevention and treatment of pediatric overweight and obesity. J Acad Nutr Diet. 2013;113:1375–94.
319. Hesketh KD, Campbell KJ. Interventions to prevent obesity in 0–5 year olds: an updated systematic review of the literature. Obesity (Silver Spring). 2010;18 Suppl 1:S27–35.
320. Ajie WN, Chapman-Novakofski KM. Impact of computer-mediated, obesity-related nutrition education interventions for adolescents: a systematic review. J Adolesc Health. 2014;54:631–45.
321. Hou SI, Charlery SA, Roberson K. Systematic literature review of internet interventions across health behaviors. Health Psychol Behav Med. 2014;2:455–81.
322. Silk KJ, Sherry J, Winn B, Keesecker N, Horodynski MA, Sayir A. Increasing nutrition literacy: testing the effectiveness of print, web site, and game modalities. J Nutr Educ Behav. 2008;40:3–10.
323. Delisle C, Sandin S, Forsum E, Henriksson H, Trolle-Lagerros Y, Larsson C, et al. A web- and mobile phone-based intervention to prevent obesity in 4-year-olds (MINISTOP): a population-based randomized controlled trial. BMC Public Health. 2015;15:95.
324. Mauriello LM, Ciavatta MM, Paiva AL, Sherman KJ, Castle PH, Johnson JL, et al. Results of a multi-media multiple behavior obesity prevention program for adolescents. Prev Med. 2010;51:451–6.
325. World Cancer Research Fund. NOURISHING framework. Available at: http://www.wcrf.org/int/policy/nourishing-framework accessed 5 May 2016.
326. Roberto CA, Swinburn B, Hawkes C, Huang TTK, Costa SA, Ashe M, et al. Patchy progress on obesity prevention: emerging examples, entrenched barriers, and new thinking. Lancet. 2015;385:2400–9.
327. Hastings G, Stead M, McDermott L, Forsyth A, MacKintosh AM, Rayner M, et al. Review of research on the effects of food promotion to children – final report. Report to the Food Standards Agency. Glasgow: University of Strathclyde, Centre for Social Marketing, 2003.
328. McGinnis JM, Gootman JA, Kraak VI. Food marketing to children and youth. Threat or opportunity? Washington, DC: Institute of Medicine, National Academies Press; 2006.
329. Watson JL. Food as a lens: the past, present, and futures of family life in China. In: Jing J, editor. Feeding China's little emperors: food, children, and social change. Stanford: Stanford University Press; 2000. p.199–239.

330. Opree SJ, Buijzen M, van Reijmersdal EA, Valkenburg PM. Children's advertising exposure, advertised product desire, and materialism: a longitudinal study. Communication Research. 2014;41:717–35.
331. Cairns G, Angus K, Hastings G, Caraher M. Systematic reviews of the evidence on the nature, extent and effects of food marketing to children. A retrospective summary. Appetite. 2013;62:209–15.
332. Jahiel RI, Babor TF. Industrial epidemics, public health advocacy and the alcohol industry: lessons from other fields. Addiction. 2007;102:1335–9.
333. Moodie R, Stuckler D, Monteiro C, Sheron N, Neal B, Thamarangsi T, et al. Profits and pandemics: prevention of harmful effects of tobacco, alcohol, and ultra-processed food and drink industries. Lancet. 2013;381:670–9.
334. Anderson PM, He X. Culture and the fast-food marketing mix in the people's republic of China and the USA: implications for research and marketing. Journal of International Consumer Marketing. 1999;11:77–95.
335. Kenny P, Hastings G. Understanding social norms: upstream and downstream applications for social marketers. In: Hastings G, Angus K, Bryant C, editors. The sage handbook of social marketing. London: SAGE Publications Ltd; 2011.
336. Andreyeva T, Long MW, Brownell KD. The impact of food prices on consumption: a systematic review of research on the price elasticity of demand for food. Am J Public Health. 2010;100:216–22.
337. Powell LM, Chriqui JF, Khan T, Wada R, Chaloupka FJ. Assessing the potential effectiveness of food and beverage taxes and subsidies for improving public health: a systematic review of prices, demand and body weight outcomes. Obes Rev. 2013;14:110–28.
338. Thow AM, Downs S, Jan S. A systematic review of the effectiveness of food taxes and subsidies to improve diets: understanding the recent evidence. Nutr Rev. 2014;72:551–65.
339. International Agency for Research on Cancer. Effectiveness of tax and price policies for tobacco control. Lyon: International Agency for Research on Cancer, 2011.
340. Resolution adopted by the General Assembly: 66/2 – Political Declaration of the High-level Meeting of the General Assembly on the Prevention and Control of Non-communicable Diseases (2012).
341. Plan of action for the prevention of obesity in children and adolescents. Washington DC: Pan American Health Organization, World Health Organization Regional Office for the Americas, 2014.
342. Ruopeng A. Effectiveness of subsidies in promoting healthy food purchases and consumption: a review of field experiments. Public Health Nutr. 2013;16:1215–28:
343. European Competitiveness and Sustainable Industrial Policy Consortium. Food taxes and their impact on competitiveness in the agri-food sector. Rotterdam: ECSIP Consortium, 2014.
344. Lear SA, Teo K, Gasevic D, Zhang XH, Poirier PP, Rangarajan S, et al. The association between ownership of common household devices and obesity and diabetes in high, middle and low income countries. Can Med Assoc J. 2014;186:258–66.
345. Adams J, Tyrrell R, Adamson AJ, White M. Effect of restrictions on television food advertising to children on exposure to advertisements for 'less healthy' foods: repeat cross-sectional study. PLoS One. 2012;7.
346. Lovato C, Watts A, Stead LF. Impact of tobacco advertising and promotion on increasing adolescent smoking behaviours. Cochrane Database Syst Rev. 2011.
347. Hastings G. Marketing diet and exercise: lessons from mammon. Social Marketing Quarterly. 2003;8:32–9.
348. Stead M, Gordon R, Angus K, McDermott L. A systematic review of social marketing effectiveness. Health Educ. 2007;107:126–91.
349. The right to an adequate diet: the agriculture-food-health nexus, report presented at the 19th session of the United Nations Human Rights Council, 26 December 2011, A/HRC/1/9/59.

350. Annual report of the United Nations High Commissioner for Human Rights, 29 April 2013, A/HRC/23/59, at para 6.
351. United Nations Human Rights Office of the High Commission. The right of the child to the enjoyment of the highest standard of health, March 2013, at para 99.
352. Unhealthy foods, non-communicable diseases and the right to health, report presented at the 26th session of the United Nations Human Rights Council, 11 June 2014, A/HRC/26/31, at para 25.
353. Garde A, Friant-Perrot M. The regulation of marketing practices for tobacco, alcoholic beverages and foods high in fat, sugar and salt – a highly fragmented landscape In: Alemanno A, Garde A, editors. Regulating lifestyle risks: The EU, alcohol, tobacco and unhealthy diets: Cambridge University Press; 2014.
354. Watkins AJ, Lucas ES, Wilkins A, Cagampang FR, Fleming TP. Maternal periconceptional and gestational low protein diet affects mouse offspring growth, cardiovascular and adipose phenotype at 1 year of age. PLoS One. 2011;6:e28745.
355. Hart R, Norman RJ. The longer-term health outcomes for children born as a result of IFV treatment: Part I – general health outcomes. Hum Reprod Update. 2013;19:232–43.
356. Crozier SR, Robinson SM, Godfrey KM, Cooper C, Inskip HM. Women's dietary patterns change little from before to during pregnancy. J Nutr. 2009;139:1956–63.
357. Hochberg Z, Feil R, Constancia M, Fraga M, Junien C, Carel JC, et al. Child health, developmental plasticity, and epigenetic programming. Endocr Rev. 2010;32:159–224.
358. Heijmans BT, Tobi EW, Stein AD, Putter H, Blauw GJ, Susser ES, et al. Persistent epigenetic differences associated with prenatal exposure to famine in humans. Proc Natl Acad Sci USA. 2008;105:17046–9.
359. Wu G, Imhoff-Kunsch B, Girard AW. Biological mechanisms for nutritional regulation of maternal health and fetal development. Paediatr Perinat Epidemiol. 2012;26 Suppl 1:4–26.
360. Ramakrishnan U, Grant F, Goldenberg T, Zongrone A, Martorell R. Effect of women's nutrition before and during early pregnancy on maternal and infant outcomes: a systematic review. Paediatr Perinat Epidemiol. 2012;26 Suppl 1:285–301.
361. Godfrey KM, Haugen G, Kiserud T, Inskip HM, Cooper C, Harvey NC, et al. Fetal liver blood flow distribution: role in human developmental strategy to prioritize fat deposition versus brain development. PLoS One. 2012;7:e41759.
362. Hanson M, Fall C, Robinson S, Baird J. Early life nutrition and lifelong health: British Medical Association Board of Science; 2009.
363. Yu Z, Han S, Zhu J, Sun X, Ji C, Guo X. Pre-pregnancy body mass index in relation to infant birth weight and offspring overweight/obesity: a systematic review and meta-analysis. PLoS One. 2013;8:e61627.
364. Dean SV, Lassi ZS, Imam AM, Bhutta ZA. Preconception care: nutritional risks and interventions. Reproductive Health. 2104;11:s3–s15.
365. Institute of Medicine. Weight gain during pregnancy: re-examining the guidelines. Washington DC: National Academies Press; 2009.
366. Roland MC, Friis CM, Voldner N, Godang K, Bollerslev J, Haugen G, et al. Fetal growth versus birthweight: the role of placenta versus other determinants. PLoS One. 2012;7:e39324.
367. Catalano PM, Mele L, Landon MB, Ramin SM, Reddy UM, Casey B, et al. Inadequate weight gain in overweight and obese pregnant women: what is the effect on fetal growth? Am J Obstet Gynecol. 2014;211:137 e1–7.
368. Yajnik CS, Deshmukh US. Maternal nutrition, intrauterine programming and consequential risks in the offspring. Rev Endocr Metab Disord. 2008;9:203–11.

369. Drake AJ, McPherson RC, Godfrey KM, Cooper C, Lillycrop KA, Hanson MA, et al. An unbalanced maternal diet in pregnancy associates with offspring epigenetic changes in genes controlling glucocorticoid action and foetal growth. Clin Endocrinol (Oxf). 2012;77:808–15.
370. Eriksson JG, Sandboge S, Salonen MK, Kajantie E, Osmond C. Long-term consequences of maternal overweight in pregnancy on offspring later health: findings from the Helsinki birth cohort study. Ann Med. 2014;46:434–8.
371. Nohr EA, Vaeth M, Baker JL, Sorensen T, Olsen J, Rasmussen KM. Combined associations of prepregnancy body mass index and gestational weight gain with the outcome of pregnancy. Am J Clin Nutr. 2008;87:1750–9.
372. Torloni MR, Betran AP, Horta BL, Nakamura MU, Atallah AN, Moron AF, et al. Prepregnancy bmi and the risk of gestational diabetes: a systematic review of the literature with meta-analysis. Obes Rev. 2009;10:194–203.
373. Fall C. Maternal nutrition: effects on health in the next generation. Indian J Med Res. 2009;130:593–9.
374. Catalano PM, Presley L, Minium J, Hauguel-de Mouzon S. Fetuses of obese mothers develop insulin resistance in utero. Diabetes Care. 2009;32:1076–80.
375. Okubo H, Crozier SR, Harvey NC, Godfrey KM, Inskip HM, Cooper C, et al. Maternal dietary glycemic index and glycemic load in early pregnancy are associated with offspring adiposity in childhood: The Southampton Women's Survey. Am J Clin Nutr. 2014;100:676–83.
376. Catalano PM, McIntyre HD, Cruickshank JK, McCance DR, Dyer AR, Metzger BE, et al. The hyperglycemia and adverse pregnancy outcome study: associations of GDM and obesity with pregnancy outcomes. Diabetes Care. 2012;35:780–6.
377. Kim SY, Sharma AJ, Callaghan WM. Gestational diabetes and childhood obesity: what is the link? Curr Opin Obstet Gynecol. 2012;24:376–81.
378. Viswanathan M, Siega-Riz AM, Moos MK, Deierlein A, Mumford S, Knaack J, et al. Outcomes of maternal weight gain. Evid Rep Technol Assess (Full Rep). 2008:1–223.
379. Ludwig DS, Rouse HL, Currie J. Pregnancy weight gain and childhood body weight: a within-family comparison. PLoS Med. 2013;10:e1001521.
380. Wojcicki JM, Heyman MB. Let's move – childhood obesity prevention from pregnancy and infancy onward. N Engl J Med. 2010;362:1457–9.
381. Veena SR, Krishnaveni GV, Karat SC, Osmond C, Fall CH. Testing the fetal overnutrition hypothesis; the relationship of maternal and paternal adiposity to adiposity, insulin resistance and cardiovascular risk factors in Indian children. Public Health Nutr. 2013;16:1656-66.
382. Poston L. Maternal obesity, gestational weight gain and diet as determinants of offspring long term health. Best Pract Res Clin Endocrinol Metab. 2012;26:627–39.
383. Poston L. Gestational weight gain: influences on the long-term health of the child. Curr Opin Clin Nutr Metab Care. 2012;15:252–7.
384. Lawlor DA, Relton C, Sattar N, Nelson SM. Maternal adiposity – a determinant of perinatal and offspring outcomes? Nat Rev Endocrinol. 2012;8:679–88.
385. Oken E, Levitan EB, Gillman MW. Maternal smoking during pregnancy and child overweight: systematic review and meta-analysis. Int J Obes (Lond). 2008;32:201–10.
386. Janesick A, Blumberg B. Endocrine disrupting chemicals and the developmental programming of adipogenesis and obesity. Birth Defects Res C Embryo Today. 2011;93:34–50.
387. La Merrill M, Birnbaum LS. Childhood obesity and environmental chemicals. Mt Sinai J Med. 2011;78:22–48.
388. Hohwu L, Li J, Olsen J, Sorensen TI, Obel C. Severe maternal stress exposure due to bereavement before, during and after pregnancy and risk of overweight and obesity in young adult men: a Danish National Cohort Study. PLoS One. 2014;9:e97490.

389. Entringer S, Buss C, Swanson JM, Cooper DM, Wing DA, Waffarn F, et al. Fetal programming of body composition, obesity, and metabolic function: the role of intrauterine stress and stress biology. J Nutr Metab. 2012;2012:632548.
390. Ingstrup KG, Schou Andersen C, Ajslev TA, Pedersen P, Sorensen TI, Nohr EA. Maternal distress during pregnancy and offspring childhood overweight. J Obes. 2012;2012:462845.
391. Behl M, Rao D, Aagaard K, Davidson TL, Levin ED, Slotkin TA, et al. Evaluation of the association between maternal smoking, childhood obesity, and metabolic disorders: a national toxicology program workshop review. Environ Health Perspect. 2013;121:170–80.
392. Eisele TP, Larsen DA, Anglewicz PA, Keating J, Yukich J, Bennett A, et al. Malaria prevention in pregnancy, birthweight, and neonatal mortality: a meta-analysis of 32 national cross-sectional datasets in Africa. Lancet Infect Dis. 2012;12:942–9.
393. Schneeberger C, Geerlings SE, Middleton P, Crowther CA. Interventions for preventing recurrent urinary tract infection during pregnancy. Cochrane Database Syst Rev. 2012;11:CD009279.
394. Mardh PA. Influence of infection with chlamydia trachomatis on pregnancy outcome, infant health and life-long sequelae in infected offspring. Best Pract Res Clin Obstet Gynaecol. 2002;16:847–64.
395. Backes CH, Markham K, Moorehead P, Cordero L, Nankervis CA, Giannone PJ. Maternal preeclampsia and neonatal outcomes. J Pregnancy. 2011;2011:214365.
396. Conde-Agudelo A, Rosas-Bermudez A, Castano F, Norton MH. Effects of birth spacing on maternal, perinatal, infant, and child health: a systematic review of causal mechanisms. Stud Fam Plann. 2012;43:93–114.
397. Malabarey OT, Balayla J, Klam SL, Shrim A, Abenhaim HA. Pregnancies in young adolescent mothers: a population-based study on 37 million births. J Pediatr Adolesc Gynecol. 2012;25:98–102.
398. Bhutta ZA, Das JK, Bahl R, Lawn JE, Salam RA, Paul VK, et al. Can available interventions end preventable deaths in mothers, newborn babies, and stillbirths, and at what cost? Lancet. 2014;384:347–70.
399. World Health Organization. Promoting optimal fetal development. Report of a technical consultation. Geneva: World Health Organization; 2006.
400. Potdar RD, Sahariah SA, Gandhi M, Kehoe SH, Brown N, Sane H, et al. Improving women's diet quality preconceptionally and during gestation: effects on birth weight and prevalence of low birth weight-a randomized controlled efficacy trial in India (Mumbai Maternal Nutrition Project). Am J Clin Nutr. 2014;100:1257–68.
401. Han Z, Mulla S, Beyene J, Liao G, McDonald SD. Maternal underweight and the risk of preterm birth and low birth weight: a systematic review and meta-analyses. Int J Epidemiol. 2011;40:65–101.
402. Christian P, Lee SE, Donahue Angel M, Adair LS, Arifeen SE, Ashorn P, et al. Risk of childhood undernutrition related to small-for-gestational age and preterm birth in low- and middle-income countries. Int J Epidemiol. 2013;42:1340–55.
403. Salihu HM, Lynch O, Alio AP, Mbah AK, Kornosky JL, Marty PJ. Extreme maternal underweight and feto-infant morbidity outcomes: a population-based study. J Matern Fetal Neonatal Med. 2009;22:428–34.
404. Wang YF, Pei LJ, Song XM, Chen G, Zheng XY. Impact of periconceptional multi-micronutrient supplementation on gestation: a population-based study. Biomed Environ Sci. 2013;26:23–31.
405. Conde-Agudelo A, Rosas-Bermudez A, Kafury-Goeta AC. Effects of birth spacing on maternal health: a systematic review. Am J Obstet Gynecol. 2007;196:297–308.
406. Williams L, Zapata LB, D'Angelo DV, Harrison L, Morrow B. Associations between preconception counseling and maternal behaviors before and during pregnancy. Matern Child Health J. 2012;16:1854–61.
407. Special issue: Improving maternal, newborn, and child health outcomes through better designed policies and programs that enhance the nutrition of women. Paediatr Perinat Epidemiol. 2012;26:1–325.

408. Ota E, Tobe-Gai R, Mori R, Farrar D. Antenatal dietary advice and supplementation to increase energy and protein intake. Cochrane Database Syst Rev. 2012;9:CD000032.
409. Imdad A, Bhutta ZA. Maternal nutrition and birth outcomes: effect of balanced protein-energy supplementation. Paediatr Perinat Epidemiol. 2012;26 Suppl 1:178–90.
410. Pena-Rosas JP, De-Regil LM, Dowswell T, Viteri FE. Intermittent oral iron supplementation during pregnancy. Cochrane Database Syst Rev. 2012;7:CD009997.
411. Haider BA, Olofin I, Wang M, Spiegelman D, Ezzati M, Fawzi WW. Anaemia, prenatal iron use, and risk of adverse pregnancy outcomes: systematic review and meta-analysis. BMJ. 2013;346:f3443.
412. Lassi ZS, Salam RA, Haider BA, Bhutta ZA. Folic acid supplementation during pregnancy for maternal health and pregnancy outcomes. Cochrane Database Syst Rev. 2013;3:CD006896.
413. Chaffee BW, King JC. Effect of zinc supplementation on pregnancy and infant outcomes: a systematic review. Paediatr Perinat Epidemiol. 2012;26 Suppl 1:118–37.
414. Thorne-Lyman A, Fawzi WW. Vitamin d during pregnancy and maternal, neonatal and infant health outcomes: a systematic review and meta-analysis. Paediatr Perinat Epidemiol. 2012;26 Suppl 1:75–90.
415. Brannon PM. Vitamin D and adverse pregnancy outcomes: beyond bone health and growth. Proc Nutr Soc. 2012;71:205–12.
416. De-Regil LM, Palacios C, Ansary A, Kulier R, Pena-Rosas JP. Vitamin D supplementation for women during pregnancy. Cochrane Database Syst Rev. 2012;2:CD008873.
417. Dror DK, Allen LH. Interventions with vitamins B6, B12 and C in pregnancy. Paediatr Perinat Epidemiol. 2012;26 Suppl 1:55–74.
418. Thorne-Lyman AL, Fawzi WW. Vitamin a and carotenoids during pregnancy and maternal, neonatal and infant health outcomes: a systematic review and meta-analysis. Paediatr Perinat Epidemiol. 2012;26 Suppl 1:36–54.
419. Ramakrishnan U, Grant FK, Goldenberg T, Bui V, Imdad A, Bhutta ZA. Effect of multiple micronutrient supplementation on pregnancy and infant outcomes: a systematic review. Paediatr Perinat Epidemiol. 2012;26 Suppl 1:153–67.
420. Bhutta ZA, Imdad A, Ramakrishnan U, Martorell R. Is it time to replace iron folate supplements in pregnancy with multiple micronutrients? Paediatr Perinat Epidemiol. 2012;26 Suppl 1:27–35.
421. Yang Z, Huffman SL. Review of fortified food and beverage products for pregnant and lactating women and their impact on nutritional status. Matern Child Nutr. 2011;7 Suppl 3:19–43.
422. Martinez-Victoria E, Yago MD. Omega 3 polyunsaturated fatty acids and body weight. Br J Nutr. 2012;107 Suppl 2:S107–16.
423. Hawkesworth S. Conference on "multidisciplinary approaches to nutritional problems". Postgraduate symposium. Exploiting dietary supplementation trials to assess the impact of the prenatal environment on CVD risk. Proc Nutr Soc. 2009;68:78–88.
424. Stewart CP, Christian P, LeClerq SC, West KP, Jr., Khatry SK. Antenatal supplementation with folic acid + iron + zinc improves linear growth and reduces peripheral adiposity in school-age children in rural Nepal. Am J Clin Nutr. 2009;90:132–40.
425. Iannotti LL, Zavaleta N, Leon Z, Shankar AH, Caulfield LE. Maternal zinc supplementation and growth in peruvian infants. Am J Clin Nutr. 2008;88:154-60.
426. Ruifrok AE, van Poppel MN, van Wely M, Rogozinska E, Khan KS, de Groot CJ, et al. Association between weight gain during pregnancy and pregnancy outcomes after dietary and lifestyle interventions: A meta-analysis. Am J Perinatol. 2014;31:353–64.
427. Luoto R, Kinnunen TI, Aittasalo M, Kolu P, Raitanen J, Ojala K, et al. Primary prevention of gestational diabetes mellitus and large-for-gestational-age newborns by lifestyle counseling: a cluster-randomized controlled trial. PLoS Med. 2011;8:e1001036.

428. Oteng-Ntim E, Varma R, Croker H, Poston L, Doyle P. Lifestyle interventions for overweight and obese pregnant women to improve pregnancy outcome: systematic review and meta-analysis. BMC Med. 2012;10:47.
429. Hartling L, Dryden DM, Guthrie A, Muise M, Vandermeer B, Donovan L. Benefits and harms of treating gestational diabetes mellitus: a systematic review and meta-analysis for the US Preventive services task force and the national institutes of health office of medical applications of research. Ann Intern Med. 2013;159:123–9.
430. Yajnik CS. Fetal programming of diabetes: still so much to learn! Diabetes Care. 2010;33:1146–8.
431. Gillman MW, Ludwig DS. How early should obesity prevention start? N Engl J Med. 2013;369:2173–5.
432. Tobias DK, Zhang C, Chavarro J, Bowers K, Rich-Edwards J, Rosner B, et al. Prepregnancy adherence to dietary patterns and lower risk of gestational diabetes mellitus. Am J Clin Nutr. 2012;96:289–95.
433. Donovan LE, Cundy T. Does exposure to hyperglycaemia in utero increase the risk of obesity and diabetes in the offspring? A critical reappraisal. Diabet Med. 2014.
434. Lassi ZS, Mansoor T, Salam RA, Das JK, Bhutta ZA. Essential pre-pregnancy and pregnancy interventions for improved maternal, newborn and child health. Reprod Health. 2014;11 Suppl 1:S2.
435. Flower A, Shawe J, Stephenson J, Doyle P. Pregnancy planning, smoking behaviour during pregnancy, and neonatal outcome: UK Millennium Cohort Study. BMC Pregnancy Childbirth. 2013;13:238.
436. Skinner MK, Manikkam M, Guerrero-Bosagna C. Epigenetic transgenerational actions of endocrine disruptors. Reprod Toxicol. 2011;31:337–43.
437. Vom Saal FS, Nagel SC, Coe BL, Angle BM, Taylor JA. The estrogenic endocrine disrupting chemical bisphenol a (bpa) and obesity. Mol Cell Endocrinol. 2012;354:74–84.
438. Cagampang FR, Torrens C, Anthony FW, Hanson M. Developmental exposure to bisphenol a leads to cardiometabolic dysfunction in adult mouse offspring. J Dev Orig Health Dis. 2012.
439. Royal College of Obstetricians and Gynaecologists (RCOG). Chemical exposures during pregnancy: dealing with potential, but unproven, risks to child health. Scientific Impact Paper no 37. RCOG, 2013.
440. Gibbs CM, Wendt A, Peters S, Hogue CJ. The impact of early age at first childbirth on maternal and infant health. Paediatr Perinat Epidemiol. 2012;26 Suppl 1:259–84.
441. Wendt A, Gibbs CM, Peters S, Hogue CJ. Impact of increasing inter-pregnancy interval on maternal and infant health. Paediatr Perinat Epidemiol. 2012;26 Suppl 1:239–58.
442. Levine R, Temin M. Start with a girl: a new agenda for global health. Washington, DC: Center for Global Development; 2009.
443. Levine R, Lloyd CB, Greene M, Grown C. Girls count: a global investment & action agenda. Washington DC: Center for Global Development; 2009.
444. Kimani-Murage EW, Kahn K, Pettifor JM, Tollman SM, Klipstein-Grobusch K, Norris SA. Predictors of adolescent weight status and central obesity in rural South Africa. Public Health Nutr. 2011;14:1114–22.
445. Stephenson J, Patel D, Barrett G, Howden B, Copas A, Ojukwu O, et al. How do women prepare for pregnancy? Preconception experiences of women attending antenatal services and views of health professionals. PLoS One. 2014;9:e103085.
446. Institute of Medicine. Committee on Accelerating Progress in Obesity Prevention Food and Nutrition Board. Accelerating progress on obesity prevention. Solving the weight of the nation. Washington DC: Institute of Medicine, 2012.
447. Butland B, Jebb SA, Kopelman P, McPherson K, Thomas S, Mardell J, et al. Foresight. Tackling obesities: future choices – project report. London: Government Office for Science, 2007.

448. Chatterji M, Green LW, Kumanyika S. L.E.A.D.: A framework for evidence gathering and use for the prevention of obesity and other complex public health problems. Health Educ Behav. 2014;41:85–99.
449. McKinsey Global Institute. Overcoming obesity: an initial economic analysis. McKinsey & Company, 2014.
450. World Health Organization. Global action plan for the prevention and control of noncommunicable diseases 2013–2020. Geneva: World Health Organization, 2013.
451. Summerbell CD, Waters E, Edmunds LD, Kelly S, Brown T, Campbell KJ. Interventions for preventing obesity in children. Cochrane Database Syst Rev. 2005:Cd001871.
452. Harvey-Berino J, Rourke J. Obesity prevention in preschool Native-American children: a pilot study using home visiting. Obes Res. 2003;11:606–11.
453. Askie LM, Baur LA, Campbell K, Daniels LA, Hesketh K, Magarey A, et al. The Early Prevention of Obesity in CHildren (EPOCH) collaboration – an individual patient data prospective meta-analysis. BMC Public Health. 2010;10:728.
454. Wen LM, Baur LA, Rissel C, Wardle K, Alperstein G, Simpson JM. Early intervention of multiple home visits to prevent childhood obesity in a disadvantaged population: a home-based randomised controlled trial (healthy beginnings trial). BMC Public Health. 2007;7:76.
455. Taylor BJ, Heath AL, Galland BC, Gray AR, Lawrence JA, Sayers RM, et al. Prevention of overweight in infancy (poi.Nz) study: a randomised controlled trial of sleep, food and activity interventions for preventing overweight from birth. BMC Public Health. 2011;11:942.
456. Hesketh KD, Campbell K, Salmon J, McNaughton SA, McCallum Z, Cameron A, et al. The Melbourne infant feeding, activity and nutrition trial (infant) program follow-up. Contemp Clin Trials. 2013;34:145–51.
457. Daniels LA, Mallan KM, Battistutta D, Nicholson JM, Perry R, Magarey A. Evaluation of an intervention to promote protective infant feeding practices to prevent childhood obesity: outcomes of the NOURISH RCT at 14 months of age and 6 months post the first of two intervention modules. Int J Obes (Lond). 2012;36:1292–8.
458. Wen LM, Baur LA, Simpson JM, Rissel C, Wardle K, Flood VM. Effectiveness of home based early intervention on children's BMI at age 2: randomised controlled trial. BMJ. 2012;344:e3732.
459. Wen LM, Baur L, Rissel C, Simpson J, editors. Are the effects of an early childhood obesity prevention trial sustained over time after the intervention? 3-year follow-up of the healthy beginnings trial. Proceedings of The Obesity Society Annual Scientific Meeting; 2014: Boston.
460. Karanja N, Aickin M, Lutz T, Mist S, Jobe JB, Maupome G, et al. A community-based intervention to prevent obesity beginning at birth among American Indian children: study design and rationale for the PTOTS study. J Prim Prev. 2012;33:161–74.
461. Karanja N, Lutz T, Ritenbaugh C, Maupome G, Jones J, Becker T, et al. The tots community intervention to prevent overweight in American Indian toddlers beginning at birth: a feasibility and efficacy study. J Community Health. 2010;35:667–75.
462. Martin RM, Patel R, Kramer MS, Guthrie L, Vilchuck K, Bogdanovich N, et al. Effects of promoting longer-term and exclusive breastfeeding on adiposity and insulin-like growth factor-i at age 11.5 years: a randomized trial. JAMA. 2013;309:1005–13.
463. Kramer MS, Matush L, Vanilovich I, Platt RW, Bogdanovich N, Sevkovskaya Z, et al. Effects of prolonged and exclusive breastfeeding on child height, weight, adiposity, and blood pressure at age 6.5 y: evidence from a large randomized trial. Am J Clin Nutr. 2007;86:1717–21.
464. Campbell F, Conti G, Heckman JJ, Moon SH, Pinto R, Pungello E, et al. Early childhood investments substantially boost adult health. Science. 2014;343
465. Mo-suwan L, Pongprapai S, Junjana C, Puetpaiboon A. Effects of a controlled trial of a school-based exercise program on the obesity indexes of preschool children. Am J Clin Nutr. 1998;68:1006–11.

466. Dennison BA, Russo TJ, Burdick PA, Jenkins PL. An intervention to reduce television viewing by preschool children. Arch Pediatr Adolesc Med. 2004;158:170–6.
467. Fitzgibbon ML, Stolley MR, Schiffer L, Van Horn L, KauferChristoffel K, Dyer A. Two-year follow-up results for hip-hop to health jr.: a randomized controlled trial for overweight prevention in preschool minority children. J Pediatr. 2005;146:618–25.
468. Fitzgibbon ML, Stolley MR, Schiffer L, Van Horn L, KauferChristoffel K, Dyer A. Hip-hop to health jr. For Latino preschool children. Obesity (Silver Spring). 2006;14:1616–25.
469. Reilly JJ, Kelly L, Montgomery C, Williamson A, Fisher A, McColl JH, et al. Physical activity to prevent obesity in young children: cluster randomised controlled trial. BMJ. 2006;333:1041.
470. Keller A, Klossek A, Gausche R, Hoepffner W, Kiess W, Keller E. [selective primary obesity prevention in children]. Dtsch Med Wochenschr. 2009;134:13–8.
471. Jouret B, Ahluwalia N, Dupuy M, Cristini C, Negre-Pages L, Grandjean H, et al. Prevention of overweight in preschool children: results of kindergarten-based interventions. Int J Obes (Lond). 2009;33:1075–83.
472. Fitzgibbon ML, Stolley MR, Schiffer L, Kong A, Braunschweig CL, Gomez-Perez SL, et al. Family-based hip-hop to health: outcome results. Obesity (Silver Spring). 2013;21:274–83.
473. Gortmaker SL, Peterson K, Wiecha J, Sobol AM, Dixit S, Fox MK, et al. Reducing obesity via a school-based interdisciplinary intervention among youth: Planet Health. Arch Pediatr Adolesc Med. 1999;153:409–18.
474. Wang LY, Yang Q, Lowry R, Wechsler H. Economic analysis of a school-based obesity prevention program. Obes Res. 2003;11:1313–24.
475. Austin SB, Kim J, Wiecha J, Troped PJ, Feldman HA, Peterson KE. School-based overweight preventive intervention lowers incidence of disordered weight-control behaviors in early adolescent girls. Arch Pediatr Adolesc Med. 2007;161:865–9.
476. Austin SB, Spadano-Gasbarro JL, Greaney ML, Blood EA, Hunt AT, Richmond TK, et al. Effect of the planet health intervention on eating disorder symptoms in Massachusetts middle schools, 2005–2008. Prev Chronic Dis. 2012;9.
477. Bleich SN, Segal J, Wu Y, Wilson R, Wang Y. Systematic review of community-based childhood obesity prevention studies. Pediatrics. 2013;132:e201–10.
478. Chang DI, Gertel-Rosenberg A, Drayton VL, Schmidt S, Angalet GB. A statewide strategy to battle child obesity in delaware. Health Aff (Millwood). 2010;29:481–90.
479. Chomitz VR, McGowan RJ, Wendel JM, Williams SA, Cabral HJ, King SE, et al. Healthy living cambridge kids: a community-based participatory effort to promote healthy weight and fitness. Obesity (Silver Spring). 2010;18 Suppl 1:S45–53.
480. Economos CD, Hyatt RR, Goldberg JP, Must A, Naumova EN, Collins JJ, et al. A community intervention reduces BMI z-score in children: shape up somerville first year results. Obesity (Silver Spring). 2007;15:1325–36.
481. Klesges RC, Obarzanek E, Kumanyika S, Murray DM, Klesges LM, Relyea GE, et al. The Memphis Girls' health Enrichment Multi-site Studies (gems): an evaluation of the efficacy of a 2-year obesity prevention program in African American girls. Arch Pediatr Adolesc Med. 2010;164:1007–14.
482. Robinson TN, Matheson DM, Kraemer HC, Wilson DM, Obarzanek E, Thompson NS, et al. A randomized controlled trial of culturally tailored dance and reducing screen time to prevent weight gain in low-income African American girls: Stanford gems. Arch Pediatr Adolesc Med. 2010;164:995–1004.
483. Sallis JF, McKenzie TL, Conway TL, Elder JP, Prochaska JJ, Brown M, et al. Environmental interventions for eating and physical activity: a randomized controlled trial in middle schools. Am J Prev Med. 2003;24:209–17.

484. de Silva-Sanigorski AM, Bell AC, Kremer P, Nichols M, Crellin M, Smith M, et al. Reducing obesity in early childhood: results from Romp & Chomp, an Australian community-wide intervention program. Am J Clin Nutr. 2010;91:831–40.
485. Singh AS, Chin APMJ, Brug J, van Mechelen W. Dutch obesity intervention in teenagers: effectiveness of a school-based program on body composition and behavior. Arch Pediatr Adolesc Med. 2009;163:309–17.
486. Eiholzer U, Meinhardt U, Petro R, Witassek F, Gutzwiller F, Gasser T. High-intensity training increases spontaneous physical activity in children: a randomized controlled study. J Pediatr. 2010;156:242–6.
487. Borys JM, Le Bodo Y, Jebb SA, Seidell JC, Summerbell C, Richard D, et al. Epode approach for childhood obesity prevention: Methods, progress and international development. Obes Rev. 2012;13:299–315.
488. Romon M, Lommez A, Tafflet M, Basdevant A, Oppert JM, Bresson JL, et al. Downward trends in the prevalence of childhood overweight in the setting of 12-year school- and community-based programmes. Public Health Nutr. 2009;12:1735–42.
489. Borys JM, al. e. Epode-a model for reducing the incidence of obesity and weight-related comorbidities. US Endocrinol. 2013;9:32–6.
490. Sobol-Goldberg S, Rabinowitz J, Gross R. School-based obesity prevention programs: a meta-analysis of randomized controlled trials. Obesity (Silver Spring). 2013;21:2422–8.
491. De Miguel-Etayo P, Bueno G, Garagorri JM, Moreno LA. Interventions for treating obesity in children. World Rev Nutr Diet. 2013;108:98–106.
492. Williams AJ, Henley WE, Williams CA, Hurst AJ, Logan S, Wyatt KM. Systematic review and meta-analysis of the association between childhood overweight and obesity and primary school diet and physical activity policies. Int J Behav Nutr Phys Act. 2013;10:101.
493. Kelley GA, Kelley KS. Effects of exercise in the treatment of overweight and obese children and adolescents: A systematic review of meta-analyses. J Obes. 2013;2013:783103.
494. Tremblay MS, Gray CE, Akinroye K, Harrington DM, Katzmarzyk PT, Lambert EV, et al. Physical activity of children: a global matrix of grades comparing 15 countries. J Phys Act Health. 2014;11:S113–25.
495. Dollman J, Norton K, Norton L. Evidence for secular trends in children's physical activity behaviour. Br J Sports Med. 2005;39:892–7.
496. Chaput JP, Carson V, Gray CE, Tremblay MS. Importance of all movement behaviors in a 24 hour period for overall health. Int J Environ Res Public Health. 2014;11:12575–81.
497. Prochaska JO. Multiple health behavior research represents the future of preventive medicine. Prev Med. 2008;46:281–5.
498. Carson V, Ridgers ND, Howard BJ, Winkler EA, Healy GN, Owen N, et al. Light-intensity physical activity and cardiometabolic biomarkers in us adolescents. PLoS One. 2013;8:e71417.
499. Liao Y, Liao J, Durand CP, Dunton GF. Which type of sedentary behaviour intervention is more effective at reducing body mass index in children? A meta-analytic review. Obes Rev. 2014;15:159–68.
500. Salmon J, Tremblay MS, Marshall SJ, Hume C. Health risks, correlates, and interventions to reduce sedentary behavior in young people. Am J Prev Med. 2011;41:197–206.
501. Tremblay MS, Colley RC, Saunders TJ, Healy GN, Owen N. Physiological and health implications of a sedentary lifestyle. Appl Physiol Nutr Metab. 2010;35:725–40.
502. Tremblay MS, LeBlanc AG, Kho ME, Saunders TJ, Larouche R, Colley RC, et al. Systematic review of sedentary behaviour and health indicators in school-aged children and youth. Int J Behav Nutr Phys Act. 2011;8:98.
503. Owens J, Adolescent Sleep Working Group, Committee on Adolescence. Insufficient sleep in adolescents and young adults: an update on causes and consequences. Pediatrics. 2014;134:e921–32.

504. Katzmarzyk PT, Mason C. The physical activity transition. J Phys Act Health. 2009;6:269–80.
505. Active Healthy Kids Canada. Is active play extinct? The 2012 Active Healthy Kids Canada report card on physical activity for children and youth. Toronto: Active Healthy Kids Canada, 2012.
506. Goodman A, Page AS, Cooper AR, International Children's Accelerometry Database C. Daylight saving time as a potential public health intervention: An observational study of evening daylight and objectively-measured physical activity among 23,000 children from 9 countries. Int J Behav Nutr Phys Act. 2014;11:84.
507. Lamboglia CM, da Silva VT, de Vasconcelos Filho JE, Pinheiro MH, Munguba MC, Silva Junior FV, et al. Exergaming as a strategic tool in the fight against childhood obesity: a systematic review. J Obes. 2013;2013:438364.
508. LeBlanc AG, Chaput JP, McFarlane A, Colley RC, Thivel D, Biddle SJ, et al. Active video games and health indicators in children and youth: a systematic review. PLoS One. 2013;8:e65351.
509. De Bock F, Genser B, Raat H, Fischer JE, Renz-Polster H. A participatory physical activity intervention in preschools: a cluster randomized controlled trial. Am J Prev Med. 2013;45:64–74.
510. Oude Luttikhuis H, Baur L, Jansen H, Shrewsbury VA, O'Malley C, Stolk RP, et al. Interventions for treating obesity in children. Cochrane Database Syst Rev. 2009:CD001872.
511. Ho M, Garnett SP, Baur LA, Burrows T, Stewart L, Neve M, et al. Impact of dietary and exercise interventions on weight change and metabolic outcomes in obese children and adolescents: a systematic review and meta-analysis of randomized trials. Jama Pediatr. 2013;167:759–68.
512. Knowlden AP, Sharma M. Systematic review of family and home-based interventions targeting paediatric overweight and obesity. Obes Rev. 2012;13:499–508.
513. Bond M, Wyatt K, Lloyd J, Taylor R. Systematic review of the effectiveness of weight management schemes for the under fives. Obes Rev. 2011;12:242–53.
514. Ho M, Garnett SP, Baur L, Burrows T, Stewart L, Neve M, et al. Effectiveness of lifestyle interventions in child obesity: systematic review with meta-analysis. Pediatrics. 2012;130:e1647–71.
515. Barlow SE. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. Pediatrics. 2007;120:S164–S92.
516. National Institute for Health and Care Excellence. Weight management: lifestyle services for overweight and obese children and young people. NICE public health guidance 47, 2013. Available at: https://www.nice.org.uk/guidance/ph47 accessed 5 May 2016.
517. Scottish Intercollegiate Guidelines Network. Management of obesity: a national clinical guideline. Edinburgh: Scottish Intercollegiate Guidelines Network, 2010.
518. Shrewsbury VA, Steinbeck KS, Torvaldsen S, Baur LA. The role of parents in pre-adolescent and adolescent overweight and obesity treatment: a systematic review of clinical recommendations. Obes Rev. 2011;12:759–69.
519. Sacher PM, Kolotourou M, Chadwick PM, Cole TJ, Lawson MS, Lucas A, et al. Randomized controlled trial of the mend program: a family-based community intervention for childhood obesity. Obesity. 2010;18:S62–S8.
520. Fagg J, Chadwick P, Cole TJ, Cummins S, Goldstein H, Lewis H, et al. From trial to population: a study of a family-based community intervention for childhood overweight implemented at scale. Int J Obes (Lond). 2014;38:1343–9.
521. UNICEF, WHO, World Bank. UNICEF-WHO-World Bank joint child malnutrition estimates. UNICEF, New York; WHO, Geneva; World Bank, Washington DC: 2012.
522. Popkin BM. Nutrition, agriculture and the global food system in low and middle income countries. Food Policy. 2014;47:91–6.
523. Kimani-Murage EW. Exploring the paradox: double burden of malnutrition in rural South Africa: Glob Health Action 2013;6:19249.

524. Meiro-Lorenzo M, Villafana T, Harrit M. Effective repsonses to non-communicable diseases: embracing action beyond the health sector. Washington: The World Bank, 2011.
525. World Health Organization. Effective approaches for strengthening multisectoral action for noncommunicable diseases: discussion paper 1. Geneva: World Health Organization, 2012.
526. World Health Organization. Noncommunicable diseases progress monitor 2015. Geneva: World Health Organization, 2015.
527. Lake A, Townshend T. Obesogenic environments: exploring the built and food environments. J R Soc Promot Health. 2006;126:262–7.
528. Sallis JF, Glanz K. Physical activity and food environments: solutions to the obesity epidemic. The Milbank quarterly. 2009;87:123–54.
529. Frank LD, Schmid TL, Sallis JF, Chapman J, Saelens BE. Linking objectively measured physical activity with objectively measured urban form – findings from smartraq. Am J Prev Med. 2005;28:117–25.
530. Thornton LE, Pearce JR, Kavanagh AM. Using geographic information systems (gis) to assess the role of the built environment in influencing obesity: a glossary. Int J Behav Nutr Phys Act. 2011;8:71.
531. Saelens BE, Sallis JF, Frank LD. Environmental correlates of walking and cycling: Findings from the transportation, urban design, and planning literatures. Annals of behavioral medicine: a publication of the Society of Behavioral Medicine. 2003;25:80–91.
532. Laws R, Campbell KJ, van der Pligt P, Russell G, Ball K, Lynch J, et al. The impact of interventions to prevent obesity or improve obesity related behaviours in children (0–5 years) from socioeconomically disadvantaged and/or indigenous families: a systematic review. BMC Public Health. 2014;14:779.
533. Kahn EB, Ramsey LT, Brownson RC, Heath GW, Howze EH, Powell KE, et al. The effectiveness of interventions to increase physical activity. A systematic review. Am J Prev Med. 2002;22:73–107.
534. Khambalia AZ, Dickinson S, Hardy LL, Gill T, Baur LA. A synthesis of existing systematic reviews and meta-analyses of school-based behavioural interventions for controlling and preventing obesity. Obes Rev. 2012;13:214–33.
535. Kropski JA, Keckley PH, Jensen GL. School-based obesity prevention programs: an evidence-based review. Obesity. 2008;16:1009–18.
536. Van Cauwenberghe E, Maes L, Spittaels H, van Lenthe FJ, Brug J, Oppert JM, et al. Effectiveness of school-based interventions in europe to promote healthy nutrition in children and adolescents: systematic review of published and 'grey' literature. Br J Nutr. 2010;103:781–97.
537. Rose G. Sick individuals and sick populations. Int J Epidemiol. 1985;14:32–8.
538. Rose G. The strategy of preventive medicine. Oxford: Oxford University Press; 1992.
539. Nugent R. Chronic diseases in developing countries: health and economic burdens. Ann N Y Acad Sci. 2008;1136:70–9.
540. Nugent R. Bringing agriculture to the table: how agriculture and food can play a role in preventing chronic diseases. Chicago: The Chicago Council on Global Affairs, 2011.
541. Ma RC, Chan JC, Tam WH, Hanson MA, Gluckman PD. Gestational diabetes, maternal obesity, and the NCD burden. Clin Obstet Gynecol. 2013;56:633–41.
542. Monteiro CA, Moubarac JC, Cannon G, Ng SW, Popkin B. Ultra-processed products are becoming dominant in the global food system. Obes Rev. 2013;14 Suppl 2:21–8.
543. Martins AP, Levy RB, Claro RM, Moubarac JC, Monteiro CA. Increased contribution of ultra-processed food products in the Brazilian diet (1987–2009). Rev Saude Publica. 2013;47:656–65.
544. Moubarac JC, Batal M, Martins AP, Claro R, Levy RB, Cannon G, et al. Processed and ultra-processed food products: consumption trends in Canada from 1938 to 2011. Can J Diet Pract Res. 2014;75:15–21.
545. Wallinga D. Agricultural policy and childhood obesity: a food systems and public health commentary. Health Aff (Millwood). 2010;29:405–10.

546. Alston JM, Sumner DA, Vosti SA. Farm subsidies and obesity in the United States: national evidence and international comparisons. Food Policy. 2008;33:470–9.
547. Franck C, Grandi SM, Eisenberg MJ. Agricultural subsidies and the American obesity epidemic. Am J Prev Med. 2013;45:327–33.
548. Hawkes C, Friel S, Lobstein T, Lang T. Linking agricultural policies with obesity and noncommunicable diseases: a new perspective for a globalising world. Food Policy. 2012;37:343–53.
549. Hawkes C. Promoting healthy diets through nutrition education and changes in the food environment: an international review of actions and their effectiveness. Background paper for the International Conference on Nutrition (icn2). Rome: Food and Agriculture Organization, 2012.
550. Public Health Institute. Do farm subsidies cause obesity? Dispelling common myths about public health and the farm bill. Public Health Institute, 2011.
551. Dangour AD, Hawkesworth S, Shankar B, Watson L, Srinivasan CS, Morgan EH, et al. Can nutrition be promoted through agriculture-led food price policies? A systematic review. BMJ Open. 2013;3.
552. de Sa J, Lock K. Will European agricultural policy for school fruit and vegetables improve public health a review of school fruit and vegetable programmes. Eur J Public Health. 2008;18:558–68.
553. Henson S, Humphrey J. The influence of agro-food policies and programmes on the availability, affordability, safety and acceptability of food. Rome: Food and Agriculture Organization, 2013.
554. Thow AM. Trade liberalisation and the nutrition transition: mapping the pathways for public health nutritionists. Public Health Nutr. 2009;12:2150–8.
555. World Health Organization. Trade, trade agreements and non-communicable diseases in the Pacific Islands: intersection, lessons learned, challenges and way forward. Geneva: World Health Organization, 2011.
556. Friel S, Hattersley L, Snowdon W, Thow AM, Lobstein T, Sanders D, et al. Monitoring the impacts of trade agreements on food environments. Obes Rev. 2013;14:120–34.
557. Thow AM, Snowdon W, Labonté R, Gleeson D, Stuckler D, Hattersley L, et al. Will the next generation of preferential trade and investment agreements undermine prevention of noncommunicable diseases? A prospective policy analysis of the trans pacific partnership agreement. Health Policy. 2015;119(1):88–96.
558. Michelle Sahal Estimé BL. Trade as a structural driver of dietary risk factors for noncommunicable diseases in the pacific: an analysis of household income and expenditure survey data. Global Health. 2014;10:48.
559. Snowdon W, Thow AM. Trade policy and obesity prevention: challenges and innovation in the pacific islands. Obes Rev. 2013;14:150–8.
560. Hughes RG, Lawrence MA. Globalisation, food and health in Pacific Island countries. Asia Pac J Clin Nutr. 2005;14:298–306.
561. Friel S, Gleeson D, Thow AM, Labonte R, Stuckler D, Kay A, et al. A new generation of trade policy: potential risks to diet-related health from the trans-pacific partnership agreement. Global Health. 2013;9.
562. Clark SE, Hawke C, Murphy SME, Hansen-Kuhn KA, Wallinga D. Exporting obesity: US farm and trade policy and the transformation of the Mexican consumer food environment. Int J Occup Environ Health. 2012;18:53–65.
563. Barquera S, Campirano F, Bonvecchio A, Hernandez-Barrera L, Rivera JA, Popkin BM. Caloric beverage consumption patterns in Mexican children. Nutr J. 2010;9:47.
564. Barquera S, Hernandez-Barrera L, Tolentino ML, Espinosa J, Ng SW, Rivera JA, et al. Energy intake from beverages is increasing among Mexican adolescents and adults. J Nutr. 2008;138:2454–61.

565. Swinburn B, Sacks G, Vandevijvere S, Kumanyika S, Lobstein T, Neal B, et al. INFORMAS (international network for food and obesity/non-communicable diseases research, monitoring and action support): overview and key principles. Obes Rev. 2013;14:1–12.
566. World Health Organization. Population-based approaches to childhood obesity prevention. Geneva: World Health Organization, 2012.
567. Traill WB, Mazzocchi M, Niedźwiedzka B, Wills J, Shankar B. The EATWELL project: recommendations for healthy eating policy interventions across Europe. Nutrition Bulletin. 2013;38:352–57.
568. Mozaffarian D, Afshin A, Benowitz NL, Bittner V, Daniels SR, Franch HA, et al. Population approaches to improve diet, physical activity, and smoking habits: a scientific statement from the American Heart Association. Circulation. 2012;126:1514–63.
569. Hawkes C, Jewell J, Allen K. A food policy package for healthy diets and the prevention of obesity and diet-related non-communicable diseases: the NOURISHING framework. Obes Rev. 2013;14 Suppl 2:159–68.
570. World Cancer Research Fund/American Institute for Cancer Research. Policy and action for cancer prevention. Food, nutrition, and physical activity: a global perspective. Washington DC: American Institute for Cancer Research, 2009.
571. World Cancer Research Fund. NOURISHING framework. Improve food supply: Improve nutritional quality of the whole food supply. Available from: http://www.wcrf.org/int/policy/nourishing-framework/improve-food-supply , accessed 3 May 2014.
572. Moodie M, Sheppard L, Sacks G, Keating C, Flego A. Cost-effectiveness of fiscal policies to prevent obesity. Current obesity reports. 2013;2:211–24.
573. Saude Md. Dietary guidelines for the Brazilian population. Brasilia: Ministry of Health of Brazil, 2014.
574. World Cancer Research Fund. NOURISHING framework. Inform people: inform people about food & nutrition through public awareness. Available from: http://www.wcrf.org/int/policy/nourishing-framework/inform-people , accessed 4 May 2014.
575. Brown KA, Timotijevic L, Barnett J, Shepherd R, Lahteenmaki L, Raats MM. A review of consumer awareness, understanding and use of food-based dietary guidelines. Br J Nutr. 2011;106:15–26.
576. Faulkner GE, Grootendorst P, Nguyen VH, Andreyeva T, Arbour-Nicitopoulos K, Auld MC, et al. Economic instruments for obesity prevention: results of a scoping review and modified delphi survey. Int J Behav Nutr Phys Act. 2011;8:109.
577. Sassi F, Belloni A, Capobianco C. The role of fiscal policies in health promotion. Paris: OECD, 2013.
578. Escobar MAC, Veerman JL, Tollman SM, Bertram MY, Hofman KJ. Evidence that a tax on sugar sweetened beverages reduces the obesity rate: a meta-analysis. BMC Public Health. 2013;13.
579. Flynn MA, McNeil DA, Maloff B, Mutasingwa D, Wu M, Ford C, et al. Reducing obesity and related chronic disease risk in children and youth: a synthesis of evidence with 'best practice' recommendations. Obes Rev. 2006;7 Suppl 1:7–66.
580. Rattray T, Brunner W, Freestone J. The new spectrum of prevention: a model for public health practice. Martinez CA: Contra Costa Health Services. Public Health Division, 2002.
581. Kumanyika SK, Obarzanek E, Stettler N, Bell R, Field AE, Fortmann SP, et al. Population-based prevention of obesity: the need for comprehensive promotion of healthful eating, physical activity, and energy balance: a scientific statement from American Heart Association Council on Epidemiology and Prevention, Interdisciplinary Committee for Prevention (formerly the expert panel on population and prevention science). Circulation. 2008;118:428–64.
582. Department of Health. Supporting people with long term health conditions. UK: Department of Health Publications Policy and Guidance, 2007.

583. Feachem RG, Sekhri NK, White KL. Getting more for their dollar: a comparison of the NHS with California's Kaiser Permanente. BMJ. 2002;324:135–41.
584. Bricker SK, et al. School transportation modes. MMWR. 2002;51:704–5.
585. Timperio A, Ball K, Salmon J, Roberts R, Giles-Corti B, Simmons D, et al. Personal, family, social, and environmental correlates of active commuting to school. Am J Prev Med. 2006;30:45–51.
586. The Community Guide. Physical activity, environmental: community-scale urban design land use policies.
587. NICE. Physical activity and the environment. Guidance and guidelines.
588. Fenton M. Community design and policies for free-range children: creating environments that support routine physical activity. Childhood Obesity. 2012;8:44–51.
589. Frank LD, Saelens BE, Powell KE, Chapman JE. Stepping towards causation: do built environments or neighborhood and travel preferences explain physical activity, driving, and obesity? Soc Sci Med. 2007;65:1898–914.
590. Boehmer TK, Hoehner CM, Deshpande AD, Brennan Ramirez LK, Brownson RC. Perceived and observed neighborhood indicators of obesity among urban adults. Int J Obes (Lond). 2007;31:968–77.
591. McGinn AP, Evenson KR, Herring AH, Huston SL, Rodriguez DA. Exploring associations between physical activity and perceived and objective measures of the built environment. J Urban Health. 2007;84:162–84.
592. Black JL, Macinko J. Neighborhoods and obesity. Nutr Rev. 2008;66:2–20.
593. Ewing R, Brownson RC, Berrigan D. Relationship between urban sprawl and weight of United States youth. Am J Prev Med. 2006;31:464-74.
594. Rundle A, Diez Roux AV, Free LM, Miller D, Neckerman KM, Weiss CC. The urban built environment and obesity in New York city: a multilevel analysis. Am J Health Promot. 2007;21:326–34.
595. Tilt JH, Unfried TM, Roca B. Using objective and subjective measures of neighborhood greenness and accessible destinations for understanding walking trips and BMI in Seattle, Washington. Am J Health Promot. 2007;21:371-9.
596. Maas J, Verheij RA, Spreeuwenberg P, Groenewegen PP. Physical activity as a possible mechanism behind the relationship between green space and health: a multilevel analysis. BMC Public Health. 2008;8:206.
597. Task Force on Community Preventive S. Recommendations to increase physical activity in communities. Am J Prev Med. 2002;22:67–72.
598. Soler RE, Leeks KD, Buchanan LR, Brownson RC, Heath GW, Hopkins DH, et al. Point-of-decision prompts to increase stair use. A systematic review update. Am J Prev Med. 2010;38:S292–300.
599. Kruger DJ, Greenberg E, Murphy JB, DiFazio LA, Youra KR. Local concentration of fast-food outlets is associated with poor nutrition and obesity. Am J Health Promot. 2014;28:340–3.
600. Fraser LK, Edwards KL, Cade J, Clarke GP. The geography of fast food outlets: a review. Int J Environ Res Public Health. 2010;7:2290–308.
601. Mehta NK, Chang VW. Weight status and restaurant availability a multilevel analysis. Am J Prev Med. 2008;34:127–33.
602. Maddock J. The relationship between obesity and the prevalence of fast food restaurants: state-level analysis. Am J Health Promot. 2004;19:137–43.
603. Grafova IB. Overweight children: assessing the contribution of the built environment. Prev Med. 2008;47:304–8.
604. Timperio A, Salmon J, Ball K, Baur LA, Telford A, Jackson M, et al. Family physical activity and sedentary environments and weight change in children. Int J Pediatr Obes. 2008;3:160–7.
605. Morland KB, Evenson KR. Obesity prevalence and the local food environment. Health Place. 2009;15:491–5.

606. Khan LK, Sobush K, Keener D, Goodman K, Lowry A, Kakietek J, et al. Recommended community strategies and measurements to prevent obesity in the United States. MMWR Recomm Rep. 2009;58:1–26.
607. Institute of Medicine. Local government actions to prevent childhood obesity. Washington DC: National Academies Press, 2009.
608. Institute of Medicine. Early childhood obesity prevention policies. Washington DC: National Academies Press, 2011.
609. White House Task Force on Childhood Obesity. Solving the problem of childhood obesity within a generation: report to the President. Washington DC: 2010.
610. Cook-Cottone C, Casey CM, Feeley TH, Baran J. A meta-analytic review of obesity prevention in the schools: 1997–2008. Psychol Sch. 2009;46:695–719.
611. Sharma M. International school-based interventions for preventing obesity in children. Obes Rev. 2007;8:155–67.
612. Faith MS, Fontaine KR, Baskin ML, Allison DB. Toward the reduction of population obesity: macrolevel environmental approaches to the problems of food, eating, and obesity. Psychol Bull. 2007;133:205–26.
613. Jago R, Baranowski T, Baranowski JC. Fruit and vegetable availability: a micro environmental mediating variable? Public Health Nutr. 2007;10:681–9.
614. Swinburn BA, Caterson I, Seidell JC, James WPT. Diet, nutrition and the prevention of excess weight gain and obesity. Public Health Nutr. 2004;7:123–46.
615. Chriqui JF, Pickel M, Story M. Influence of school competitive food and beverage policies on obesity, consumption, and availability a systematic review. JAMA Pediatr. 2014;168:279–86.
616. Giles CM, Kenney EL, Gortmaker SL, Lee RM, Thayer JC, Mont-Ferguson H, et al. Increasing water availability during afterschool snack evidence, strategies, and partnerships from a group randomized trial. Am J Prev Med. 2012;43:S136–S42.
617. Ozer EJ. The effects of school gardens on students and schools: conceptualization and considerations for maximizing healthy development. Health Educ Behav. 2007;34:846–63.
618. Ganann R, Fitzpatrick-Lewis D, Ciliska D, Peirson LJ, Warren RL, Fieldhouse P, et al. Enhancing nutritional environments through access to fruit and vegetables in schools and homes among children and youth: a systematic review. BMC Res Notes. 2014;7:422.
619. Centers for Disease Control and Prevention (CDC). School health guidelines to promote healthy eating and physical activity. MMWR Recomm Rep. 2011;16:1–76.
620. Pekruhn C, Arlington VA. Preventing childhood obesity: a school health policy guide. National Association of State Boards of Education. 2009.
621. National Institute for Health and Care Excellence. Obesity: guidance on the prevention of overweight and obesity in adults and children. London: NICE, 2006.
622. Department of Health and Ageing Governement of Australia. Health and active school communities: a resource kit for schools. Canberra: Department of Health and Ageing, Australia, 2014.
623. European Union. EU action plan on childhood obesity 2014–2020. Brussels: European Union, 2014.
624. World Health Organization. Prevention and control of noncommunicable diseases: Implementation of the global strategy. Geneva: World Health Organization, 2008.
625. World Health Organization. School policy framework. Implementation of the WHO global strategy on diet, physcial activity and health. Geneva: World Health Organization, 2008.
626. EU working group sports and health. EU physiocal activity guidelines: recommended policy actions in support of health-enhancing physical activity. Brussels: EU Commission, 2008.

627. Heath GW, Brownson RC, Kruger J, Miles R, Powell KE, Ramsey LT, et al. The effectiveness of urban design and land use and transport policies and practices to increase physical activity: a systematic review. Act Health Suppl 1. 2006:S55–76.
628. Siegel KR, Ali MK, Srinivasiah A, Nugent RA, Narayan KM. Do we produce enough fruits and vegetables to meet global health need? PLoS One. 2014;9:e104059.
629. Food and Agriculture Organization. The state of food and agriculture 2013: food systems for better nutrition. Rome: Food and Agriculture Organization, 2013.
630. Institute of Medicine. Sustainable diets: Food for healthy people and a healthy planet: workshop summary. Washington, DC: National Academies Press; 2014.
631. Food and Agriculture Organization. Second international conference on nutrition: framework for action. Rome: Food and Agriculture Organization, 2014.
632. Hood C, Martinez-Donate A, Meinen A. Promoting healthy food consumption: a review of state-level policies to improve access to fruits and vegetables. WMJ. 2012;111:283–8.
633. Thow AM, Priyadarshi S. Aid for trade: an opportunity to increase fruit and vegetable supply. Bull World Health Organ. 2013;91:57-63.
634. Rickard BJ, Okrent AM, Alston JM. How have agricultural policies influenced caloric consumption in the United States? Health Econ. 2013;22:316–39.
635. Hawkes C, Ruel M. The links between agriculture and health: an intersectoral opportunity to improve the health and livelihoods of the poor. Bull World Health Organ. 2006;84:98–-90.
636. Turner R, Hawkes C, Jeff W, Ferguson E, Haseen F, Homans H, et al. Agriculture for improved nutrition: the current research landscape. Food Nutr Bull. 2013;34:369–77.
637. Korth MJ, et al. What are the impacts of urban agriculture programs on food security in low and middle-income countries: a systematic review. Environmental Evidence. 2014;3:21.
638. Zezza A, Tasciotti L. Urban agriculture, poverty, and food security: empirical evidence from a sample of developing countries. Food Policy. 2010;35:265–73.
639. Food and Agriculture Organization. Growing greener cities. Rome: Food and Agriculture Organization, 2010.
640. Parfitt J, Barthel M, Macnaughton S. Food waste within food supply chains: quantification and potential for change to 2050. Philosophical Transactions of the Royal Society B-Biological Sciences. 2010;365:3065–81.
641. Frison EA, Smith IF, Johns T, Cherfas J, Eyzaguirre PB. Agricultural biodiversity, nutrition, and health: making a difference to hunger and nutrition in the developing world. Food and Nutrition Bulletin. 2006;27:167–79.
642. Lee K, Ingram A, Lock K, McInnes C. Bridging health and foreign policy: the role of health impact assessments. Bull World Health Organ. 2007;85:207–11.
643. Food and Agriculture Organization. The double burden of malnutrition. Case studies from six developing countries. Food and Nutrition Paper. 2006;84:1–334.
644. Food and Agriculture Organization. Impacts of foreign agricultural investment on developing countries: Evidence from case studies. Rome: Food and Agriculture Organization, 2014.
645. MacRae R. Do trade agreements substantially limit development of local/sustainable food systems in Canada? Canadian Food Studies Review. 2014;1:103–25.
646. von Tigerstrom B. How do international trade obligations affect policy options for obesity prevention? Lessons from recent developments in trade and tobacco control. Can J Diabetes. 2013;37:182–8.
647. Atkins VJ. Agricultural trade policy instruments to promote healthy diets in developing countries: an assessment of the opportunities within the framework of the WTO agreement on agriculture and the Doha development agenda. In: Trade, food, diet and health. Oxford: Wiley-Blackwell. 2010.

648. Yeshanew SA. International dimensions of the right to adequate food. Rome: Food and Agriculture Organization. 2014.
649. The proposed EU-Canada trade agreement raises health concerns in both Canada and European Union: Canadian Centre for Policy Alternatives. Available at: https://www.policyalternatives.ca/publications/reports/proposed-eu-canada-trade-agreement-raises-health-concerns-both-canada-and accessed 30 April 2016).
650. L'Abbe MR, Lewis J, Zehaluk C. The potential of the Codex Alimentarius to promote healthy diets worldwide – the Canadian experience of implementation. In: Trade, food, diet and health. Oxford: Wiley-Blackwell.
651. Kumanyika S. INFORMAS (International Network for Food and Obesity/non-communicable diseases Research, Monitoring and Action Support): Summary and future directions. Obes Rev. 2013;14 Suppl 1:157–64.
652. Institute of Medicine. Front-of-package nutrition rating systems and symbols. Washington, DC: The National Academies Press, 2010.
653. World Cancer Research Fund, American Institute for Cancer Research. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Washington, DC: American Institute of Cancer Research, 2007.
654. Lachat C, Nago E, Verstraeten R, Roberfroid D, Van Camp J, Kolsteren P. Eating out of home and its association with dietary intake: A systematic review of the evidence. Obes Rev. 2012;13:329-46.
655. Swartz JJ, Braxton D, Viera AJ. Calorie menu labeling on quick-service restaurant menus: An updated systematic review of the literature. Int J Behav Nutr Phys Act. 2011;8:135.
656. Traill WB, Bech-Larsen T, Gennaro L, Koziol-Kozakowska A, Khun S, Wills J, et al. Reformulation for healthier food: a qualitative assessment of alternative approaches. 2012.
657. Smit LA, Mozaffarian D, Willett W. Review of fat and fatty acid requirements and criteria for developing dietary guidelines. Ann Nutr Metab. 2009;55:44–55.
658. Harika RK, Cosgrove MC, Osendarp SJ, Verhoef P, Zock PL. Fatty acid intakes of children and adolescents are not in line with the dietary intake recommendations for future cardiovascular health: a systematic review of dietary intake data from thirty countries. Br J Nutr. 2011;106:307–16.
659. Aranceta J, Perez-Rodrigo C. Recommended dietary reference intakes, nutritional goals and dietary guidelines for fat and fatty acids: a systematic review. Br J Nutr. 2012;107 Suppl 2:S8–22.
660. WHO Regional Office for the Easter Mediterranean, FAO Regional Office for the Near East. FAO/WHO technical consultation on national food-based dietary guidelines. Cairo: World Health Organization/Food and Agricuture Organization, 2006.
661. Powell LM, Chaloupka FJ. Food prices and obesity: evidence and policy implications for taxes and subsidies. Milbank Q. 2009;87:22957.
662. Sturm R, An R. Obesity and economic environments. CA Cancer J Clin. 2014;64:337–50.
663. Jeffery RW. Financial incentives and weight control. Prev Med. 2012;55 Suppl:S61–7.
664. Thow AM, Jan S, Leeder S, Swinburn B. The effect of fiscal policy on diet, obesity and chronic disease: a systematic review. Bull World Health Organ. 2010;88:609–14.
665. Lobstein T, Brinsden H. Symposium report: the prevention of obesity and NCDs: challenges and opportunities for governments. Obes Rev. 2014;15:630–9.

ANNEX 1: Consideration of the types of evidence used to assess interventions

In an article which considered the types of evidence used to assess approaches to obesity prevention, Chatterjee et al. *(448)* suggested that two traditions have tended to dominate, each with its own limitations. The first, the causal model for studying obesity, targets the factors causing disturbances of energy balance. Using such an approach, much of the resultant evidence base has tended to focus on individual level outcomes and has taken a reductionist view, neglecting broader community contexts and potential explanatory influences. The second tradition emphasises the use of randomized controlled trials for determining the effectiveness of obesity interventions. This approach, which works well in more contained and homogeneous clinical settings, has challenges when confronted by the more heterogeneously distributed background and cultural variables that typically influence responses to a health intervention's effect at a population level.

Because of these limitations, public health researchers have argued that different criteria must be applied to build the evidence base for obesity prevention *(448, 579)*. Evidence evaluation must take into account the need: a) to implement at relatively large scale and across communities and regions; b) to evaluate interventions that comprise multiple components operating synergistically; and c) for generalizability, transferability and sustainability. Chatterjee et al. *(448)* put forward the need for a systems-oriented framework to examine obesity issues that can address such issues, championing the L.E.A.D. framework: a) **L**ocate evidence – identify and gather the types of evidence that are potentially relevant to the questions; b) **E**valuate evidence – apply standards of quality as relevant to different types of evidence; c) **A**ssemble evidence – select and summarize the relevant evidence according to considerations for use; and d) Inform **D**ecisions – use evidence in the decision-making process.

ANNEX 2: Conceptual frameworks for interventions to address obesity

Given the complexity of factors influencing obesity, several frameworks for considering approaches to interventions have been devised. Table A2.1 shows the "spectrum of prevention" *(580)* which characterizes and differentiates interventions at all of the levels that may be needed to address obesity at the population level. There are seven bands or levels, including environmental and policy strategies, community mobilization and individual education. Note that the more upstream bands (e.g. policy and legislation, or changing organizational practices) are important for enabling the effectiveness of interventions acting further downstream (e.g. those targeting families and individuals). It is also important to note that, while the upstream factors are important, most of the evidence is on downstream factors and interventions acting more proximal to the child and family.

Table A2.1. Components of a comprehensive "spectrum of prevention" as applied to obesity prevention

Prevention strategy	**Rationale**	**Potential examples related to childhood obesity**
Influencing policy and legislation	Both formal and informal policies have the ability to affect large numbers of people by improving environments in which the live, work and go to school, encouraging people to lead health lifestyles and providing for consumer protection	Regulation of food marketing of unhealthy foods and beverages directed towards children International Code of Marketing of Breast-milk Substitutes
Mobilizing neighbourhoods and communities	Particularly in low-income communities confronting more urgent concerns (violence, unemployment, drug use, the struggle to keep families together), engaging community members in developing agendas and priorities is essential	Whole-of-community obesity prevention sites, such as the EPODE (Ensemble Prevenons l'Obesité Des Enfants; Together Let's Prevent Obesity) network
Changing organizational practices	Modifying the internal policies and practices of agencies and institutions can result in improved health and safety, better services for clients and a healthier community environment; advocacy for such changes can result in a broad impact on community health	Protocols for health professional assessment of children Protocols for healthy eating and activity in child-care settings Improve park safety Healthy school canteens Baby Friendly Hospital Initiative.

Fostering coalitions and networks	Coalitions and networks, composed of community organizations, policy-makers, businesses, health/education/early childhood/ child-care professionals, and community residents working together, can be powerful advocates for legislation and organizational change and provide an opportunity for joint planning, system-wide problem solving and collaborative policy development.	Local project coalitions and advisory committees.
Educating professionals in the various service delivery sectors	Professionals within the health/education/early childhood/child-care sectors, and beyond, can encourage adoption of health behaviours, screen for health risks (health sector), contribute to community education and advocate for policies and legislation.	Training of health-care professionals in the assessment/prevention/ management of childhood obesity Training of education/early childhood/child-care sector staff University technical college curricula
Promoting community education	Community education can reach the greatest number of individuals possible with health education messages and also build a critical mass of people who will become involved in improving community health. This includes media advocacy – the use of mass media to shape the public's understanding of health issues.	Media campaigns School fitness events School and community gardens
Strengthening individual knowledge and skills	This involves working directly with individuals and families in the home, school, child-care or health settings providing information to promote well-being among children and family members. It also includes working with youth and adults to build their capacity in areas such as media advocacy, community mobilization and working with policy-makers to make positive changes to their community.	Home visiting by early childhood nurses Integrated curricula at school/early childhood/child-care settings User-friendly health promotion materials oriented to families with low literacy

Source: adapted from Kumanyika et al. (2008) *(581)* in turn adapted from Rattray et al. (2002) *(580).*

Table A2.2 summarizes approaches to population-based childhood obesity prevention highlighted in the 2012 WHO report *(566)*. Three broad components were identified: (i) structures within government to support childhood obesity prevention policies and interventions; (ii) population-wide policies and initiatives; and (iii) community-based interventions. A comprehensive childhood obesity prevention strategy will include aspects of each of the components.

Table A2.2. Schematic model demonstrating the framework for a childhood obesity prevention strategy

Structures to support policies and interventions	**Population-wide policies and initiatives**	**Community-based interventions**
Leadership; "Health in all" policies; Dedicated funding for health promotion; Noncommunicable disease monitoring systems; Workforce capacity; Networks and partnerships; Standards and guidelines.	Regulation of marketing of unhealthy foods and beverages to children; Nutrition labelling; Food taxes and subsidies; Fruit and vegetable initiatives; Policies to promote physical activity; Social marketing campaigns.	Multicomponent community-based interventions; Early child-care settings; Primary and secondary schools; Other community settings.

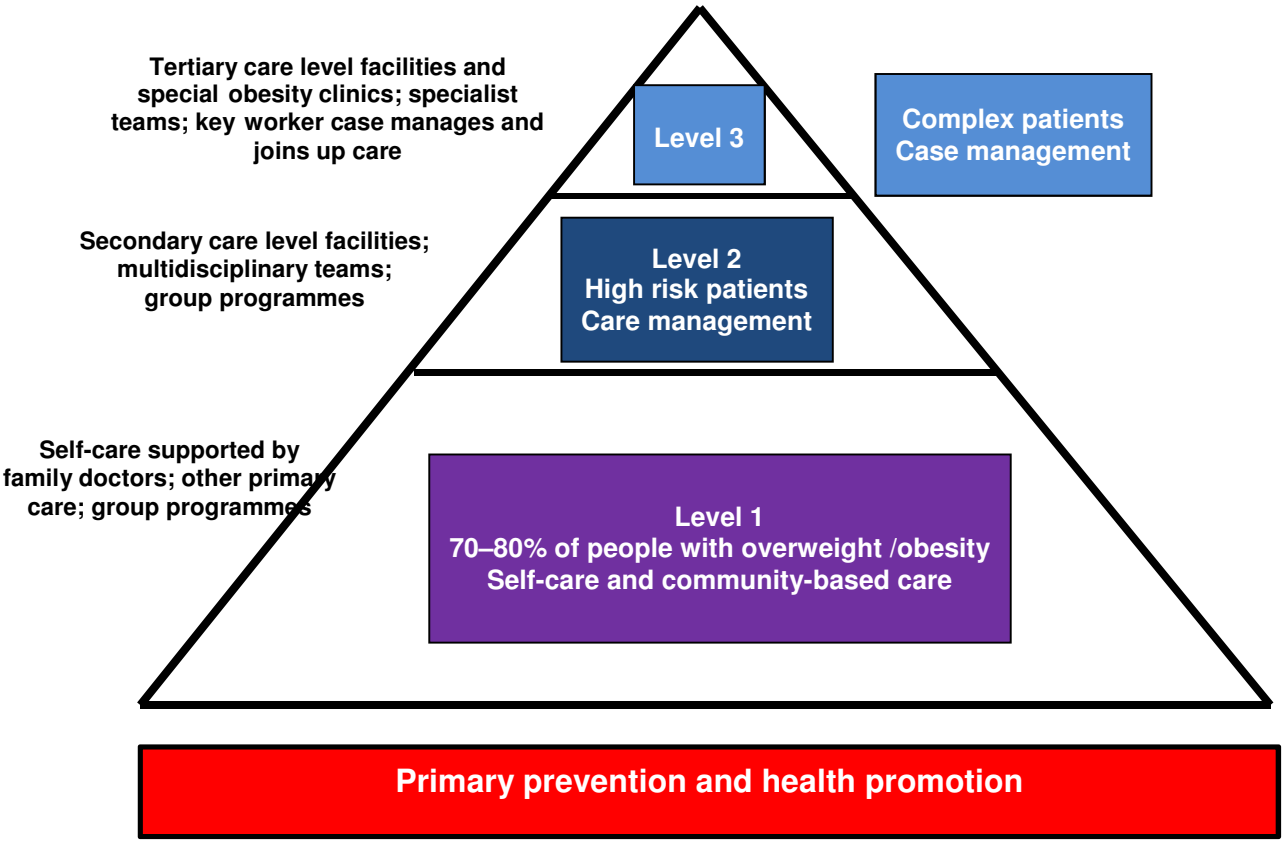
Source: adapted from WHO (2012) *(566)*.

ANNEX 3: Approaches to health service delivery for those affected by obesity

Figure A3.1 shows one potential approach to a coordinated model of health service delivery, which is adapted from the United Kingdom's National Health Service *(582)*, and Kaiser Permanente Chronic Disease Management Pyramid of Care *(583)*. This is based upon a tiered level of service delivery relating to severity of disease. Thus, while most people affected by the problem of obesity can be managed via self-care or family-based care, with support from primary care or community-based health service providers, there is a need for treatment by multidisciplinary care teams and possibly tertiary care clinics, for those who are more severely affected. Individual clinicians should be aware of the presence of other services within their geographic region, and the capacity of these to take referrals or to co-manage patients. These may include group programmes, individual consultations with allied health professionals or nurses, or specialised tertiary services.

Figure A3.1. Chronic disease management pyramid for paediatric overweight and obesity.

Adapted from the Kaiser-Permanente and United Kingdom National Health Service Chronic Disease Management Pyramid of Care



The 2007 US Expert Committee recommendations highlight a similar, staged approach to management of paediatric obesity *(515)*. The first stage, “Prevention Plus”, involves counselling for healthy lifestyle eating and activity habits for overweight and obese patients. The second stage, “Structured Weight Management”, includes more targeted recommendations for behavioural changes, supported by a structured meal plan, and involvement of a dietician. Stage 3, “Comprehensive Multidisciplinary Intervention”, increases the intensity of behaviour change and frequency of visits, and involves structured behaviour change supported by a multidisciplinary team of therapists, a service that is unlikely to be available in primary-care settings. The final stage, “Tertiary Care Intervention”, which is offered to severely obese young people, may include provision of pharmacotherapy or bariatric surgery. The patient’s age, severity of obesity and obesity-associated complications, and level of engagement with, and success of, previous interventions, should determine which stage of therapy is offered.

ANNEX 4: Policies that address childhood obesity in diverse sectors of society

Table A4.1. Regional action plans on childhood obesity

	Multisectoral action as an overarching principle	Strategic recommendations/lines of action

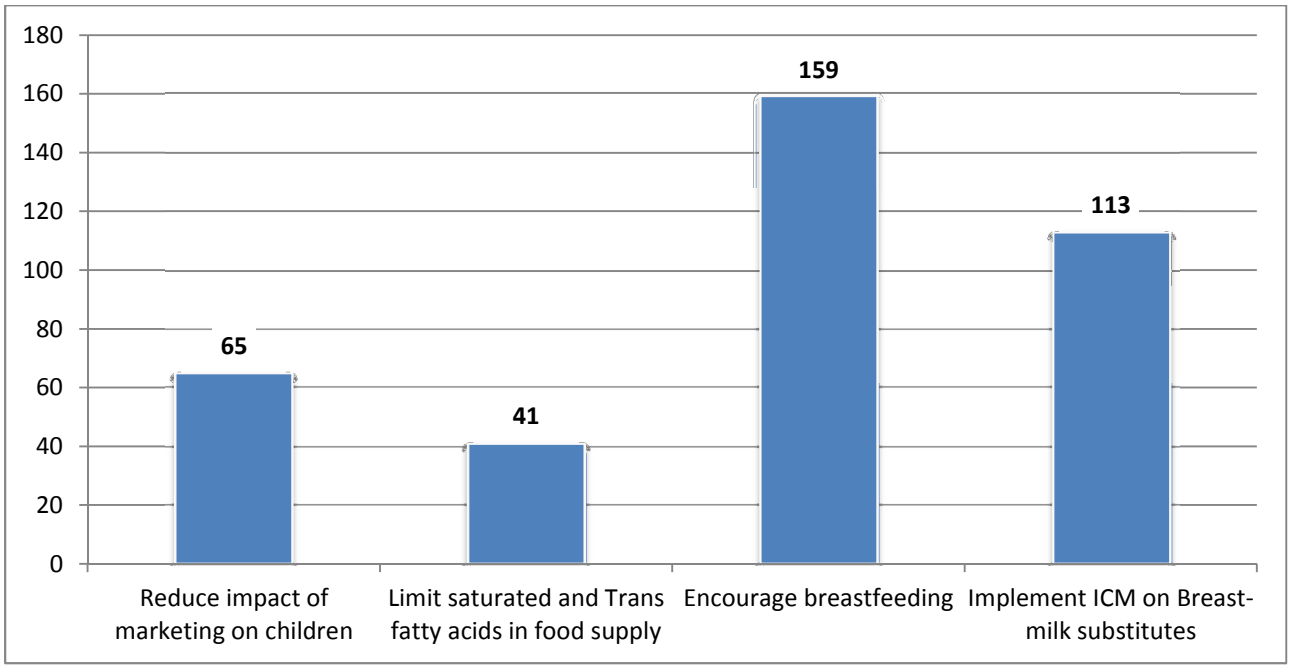
WHO Region of the Americas
 	
Plan of Action for the Prevention of Obesity in Children and Adolescents (2014–2019)[a]
	The overall goal is to halt the rise of the rapidly growing obesity epidemic in children and adolescents, so that there is no increase in current country prevalence rates. This goal requires a multisectoral life-course approach that is based on the social-ecological model and focuses on transforming the current obesogenic environment into opportunities for increased intake of nutritious foods and improved physical activity.	• Primary health care and promotion of breastfeeding and healthy eating.
		• Improvement of school nutrition and physical activity environments.
		• Fiscal policies and regulation of food marketing and labelling.
		• Other multisectoral actions:
		  ○ creating public spaces that promote physical activity;
		  ○ interventions to improve production, storage; and distribution systems of natural and whole foods.

EU Action Plan on Childhood Obesity (2014–2020)[b]	The overarching goal is to contribute to halting the rise in overweight and obesity in children and young people (0–18 years) by 2020. To achieve this goal, the active participation of a wide range of stakeholders is necessary. In addition to Member States and international organizations the Action Plan identifies civil society as a key stakeholder: 1) Nongovernmental organizations for health, education, family, consumer, and sport 2) The industry, including the retail, catering and agricultural sectors. 3) University and research institutes.	• Support a healthy start in life; • Promote healthier environments, especially in schools and preschools; • Make the healthy option the easier option; • Restrict marketing and advertising to children; • Inform and empower families; • Encourage physical activity; • Monitor and evaluate; • Increase research.

[a] http://www.paho.org/hq/index.php?option=com_docman&task=doc_view&Itemid=270&gid=28890&lang=en

[b] http://ec.europa.eu/health/nutrition_physical_activity/docs/childhoodobesity_actionplan_2014_2020_en.pdf

Figure A4.1. Policy uptake (number of countries) for selected noncommunicable disease policies (of 178 countries)



Source: Data from the 2013 Noncommunicable Disease Global Survey (unpublished).

Table A4.2. Overview of evidence on built environment and physical activity

Built environment and physical activity		
Evidence	Consensus policy recommendation	Proposed by
• There was a statistically significant relationship between children's physical activity and proximity to school *(584)* or road safety measures *(585)*.	Locating schools within easy walking distance of residential areas.	CDC *(586)*
	Create safe routes to schools by improving the built environment (e.g. implement Safe Routes interventions).	NICE *(587)*; Fenton *(588)*
	Ensure open spaces and public paths can be reached on foot, by bicycle and using other modes of transport involving physical activity. They should also be accessible by public transport.	NICE *(587)*
• There were a statistically significant associations between adults' physical activity and area walkability *(589)*, land use and aesthetics respectively *(590)*, but limited evidence on effect of traffic on physical activity *(591)*.	Improve access to outdoor recreational facilities such as parks and green spaces.	CDC *(586)*
• Neighbourhood features that discourage physical activity are associated with increased BMI *(592)*. • Significant associations between obesity/overweight and	Ensure public open spaces and public paths are maintained to a high standard. They should be safe, attractive and welcoming to everyone.	NICE *(587)*

Built environment and physical activity		
Evidence	Consensus policy recommendation	Proposed by
lower residential density or mixed land uses *(529, 593, 594).* • Lower BMI in residents of areas with higher greenness (*595*), proximity to open recreational facilities and access to natural environments *(596)*.	Enhance personal and traffic safety in areas where people are or could be physically active.	CDC *(586)*
• Neighbourhood features that discourage physical activity are associated with increased BMI *(592)*.	Creation of or enhanced access to places for physical activity (e.g. trails or facilities) by reducing barriers to access, combined with informational outreach activities: strongly recommended.	AJPM 2002 *(59, 533)*
	Communities, transportation officials, community planners, health professionals, and governments should make promotion of physical activity a priority by substantially increasing access to places and opportunities for such activity.	IOM *(446)*
	Improve access to public transportation.	CDC *(597)*
• There is strong evidence that point-of-decision prompts are effective in increasing the use of stairs *(598).*	Point-of-decision prompts are recommended, for health benefits or weight loss.	CDC (*597, 598*)

AJPM: American Journal of Preventive Medicine; CDC: United States Centers for Disease Control and Prevention; IOM: United States Institute of Medicine; NICE: United Kingdom's National Institute for Health and Care Excellence.

Table A4.3. Overview of evidence on built environment and food environments

Built environment and food environments		
Evidence	Consensus policy recommendation	Proposed by
• There was a significant association between the local concentration of "fast-food" outlets ("fast-food" restaurants and convenience stores), and BMI, and fruit and vegetable consumption among the general population and children between 3–14 years of age *(599–602).* Several studies have demonstrated significant associations between access to full service restaurants and being overweight or obese or eating fewer fruits and vegetables *(601, 603, 604)*. • Access to supermarkets or grocery stores is linked to lower obesity and overweight prevalence, as well as to lower BMI in the general population *(604, 605)*.	Increase community geographic access to healthy foods retailers, through a variety of policy levers such as tax incentives, land use and zoning regulations.	CDC *(606)*
	Improve transportation routes to healthy food retailers.	CDC *(606)*; IOM *(607, 608)*; White House Taskforce on Childhood Obesity *(609)*
	Restrict access to unhealthy foods around schools through zoning regulations, and make potable water available in school environments.	IOM *(446)*

CDC: United States Centers for Disease Control and Prevention; IOM: United States Institute of Medicine.

Table A4.4. Overview of evidence on education

Education		
Evidence	Consensus policy recommendations	Proposed by
Preschool age (0–5 years) • The preschool period has been identified as a sensitive and critical stage for establishing healthy eating and physical activity habits: young children are adaptable and weight in this period can set a pattern for the future *(319, 610)*. • Although study results have been mixed, intervening in preschool/child-care settings to improve diet, increase physical activity and/or reduce sedentary behaviour can be successful in significantly impacting these behaviours, as well as adiposity *(318, 319).* • In a systematic review, Hesketh and Campbell *(319)* note that successful interventions have aimed to improve not only parent/caregiver knowledge, but also skills and competencies. • Successful obesity reducing interventions were typically multicomponent, involved parents and the community, and attempted to address the food and physical activity environment *(318, 319)*. **School age (6–18 years)** *Influencing BMI among children* • Systematic reviews demonstrate the capacity for	*Overarching* Adopt a coordinated "whole-school approach" that includes assessing the entire school environment and implementing policies and practices to support healthy weights, diet, and the promotion of physical activity. Integrate teaching on physical activity and healthy eating with changes to the physical and food environments. Use community and parental engagement to strengthen the impact of child-care and school-based interventions. Support and train teachers and other staff to implement health promotion strategies and activities.	Academy of Nutrition and Dietetics *(318)* CDC *(619)* National Association of State Boards of Education *(620)* NICE *(621)*
	School environment • Establish school environments that support healthy eating choices and physical activity throughout the school day; • Establish nutrition standards for schools that include a healthy diet with limited fat intake;	CDC *(619)* Australian Government – Healthy and Active

Education		
Evidence	Consensus policy recommendations	Proposed by
school-based programmes to improve diet, reduce sedentary behaviour, and increase physical activity; overall, study design, participants, intervention types and outcome measures have been heterogeneous *(534–536, 611).* • Positively impacting BMI, and other measures of adiposity, has proven more elusive. Early reviews on the impact of school-based obesity prevention programmes on BMI showed mixed results, in part related to the types of studies selected for analysis *(490, 534)*. • Two recent meta-analyses present "convincing evidence" that school-based approaches can be effective for reducing BMI in children, even though effects are mild (e.g. overall standard mean difference of -0.076 [95% CI: -0.123 to -0.028; $P < 0.01$] in BMI between intervention and control groups) *(490, 610)*. Avoiding weight gain may significantly affect prevalence of obesity at the population level. *Influencing BMI among adolescents* • BMI is more difficult to influence in the teenage years; though the trend is towards the negative, Sobol-Goldberg's meta-analysis shows no statistically significant reduction in BMI (-0.039;	• High quality school meals and healthy, appealing food/beverage choices outside school programmes; • Create a pleasant, sociable environment for mealtimes with staff supervision; • Ban sugar-sweetened beverages (including flavoured/sweetened milk) and limit the portion size of 100% fruit juice; • Make drinking water freely available to students in dining areas and throughout the day; • Create and support School Gardens; and • Plan building layout, recreational spaces.	School Communities, A Resource Kit for Schools *(622)* NICE *(621)* EU 2014, Childhood Obesity Action Plan *(623, 624)*; WHO 2008 *(625)*
	Combined physical activity/healthy eating School programmes should give children the knowledge, attitudes, skills, and experiences required for engagement in healthy eating and physical activity. Interventions should be: • Ongoing • Multicomponent • Address the whole school regardless of weight status	CDC *(619)* Australian Government – Healthy and Active School Communities, A Resource Kit for Schools *(622)* Academy of Nutrition and Dietetics *(318)*

Education		
Evidence	Consensus policy recommendations	Proposed by
95% CI: -0.096–0.017; *P*=0.17) *(490)*. • In a Cochrane review, Waters et al. found a similar trend when they examined preventive interventions aimed at adolescents (13–18 years) delivered in diverse settings (school/community/home/after-care). BMI declined but the effect was not statistically significant (-0.09 kg/m2 [95% CI: -0.20–0.03]) *(588)*. *Which interventions work?* • The most successful trials involved parental support, lasted at least 1 year, and were comprehensive (i.e. defined as targeting all of physical activity, sedentary behaviour, healthy eating and unhealthy eating or containing all of the following components: "providing information on nutrition and physical activity, attitudinal changes, monitoring eating and physical activity, and environmental modifications (e.g. adding water coolers)") *(490, 534)*. • In its recent review, the Academy of Nutrition and Dietetics describes fair evidence (grade II) supporting combined nutrition-education and physical activity interventions as effective in improving adiposity measures, and behaviours contributing to childhood overweight, although results are heavily influenced by study design *(318)*.	• Incorporated into pre- and post-care, and extracurricular activities • Involve parents with advice and activities to enable them to create a healthy lifestyle at home • Fun! ○ Hands-on activities that encourage touching and tasting increase children's acceptance and gardening programmes are promising for increasing children's fruit and vegetable consumption	NICE *(621)*
	Physical activity Policies and programmes to encourage physical activity should be comprehensive and integrated; the involvement of school boards/advisory councils is recommended in development and implementation. Schools should offer opportunities to be physically active throughout the day (e.g. during play times, lunch breaks, pre-/post-school activities) and there should be (at least) one hour each day of school based physical activity.	National Association of State Boards of Education *(620)* EU Physical Activity Guidelines 2008 *(626)* CDC *(619)* CDC *(59, 597, 627)*

Education		
Evidence	Consensus policy recommendations	Proposed by
• Cook-Cottone et al. *(610)* reported that important features of successful interventions have included (all statistically significant findings): ○ Universality ○ Elementary, versus other grades, school setting ○ A range of intensities and durations > 12 weeks ○ More successful with high parental involvement (but still significant without), including a behaviour change goal for parents ○ Encourage a healthy diet ○ Try to reduce sedentary behaviour and increase physical education and activity breaks ○ Delivered by a school teacher – expert collaboration, regular classroom teachers, or physical education teachers ○ Community-based component *(449)* • Systematic reviews/research on interventions to alter the school physical activity and eating	Schools should try to increase the amount of time students spend doing moderate to vigorous physical activity, either by increasing the duration, or intensity, of activity in physical education classes. Teacher training in health-enhancing physical education modules is recommended from K-12[1]. Children should be encouraged to develop movement skills (regardless of ability or disability levels) by having the appropriate opportunities, equipment and classes.	NICE *(621)*

[1] http://www.k12.wa.us/healthfitness/Standards.aspx

Education		
Evidence	Consensus policy recommendations	Proposed by
environment show: ○ An association between the availability of fruits and vegetables and increased consumption *(612, 613)* ○ Availability and consumption of sugar-sweetened beverages within the school environment is linked to obesity and changes to the school food environment can lead to changes in student consumption and health outcomes *(614, 615)* ○ Availability of free potable water increases water consumption *(616)* ○ School Gardens are a source of greater understanding of food production and a promising practice to improve nutritional knowledge and healthy food consumption *(528, 617, 618)* ○ There is strong evidence that school-based physical education can increase levels of physical activity and can improve the overall levels of physical fitness *(597)*		

BMI: body mass index; CDC: United States Centers for Disease Control and Prevention; EU: European Union; NICE: United Kingdom's National Institute for Health and Care Excellence; WHO: World Health Organization.

Table A4.5. Overview of evidence on agriculture policies

Agricultural policies		
Evidence	Consensus policy recommendations	Proposed by
• Over the past few decades, market factors and agricultural policies have increased the demand for exportable cash crops, sometimes with the unintended consequence of reducing the diversity of fresh produce available at the local level. Agricultural policies, often through both production and input policies, have promoted farmers' capacity to increase production, generally of the kinds of commodities – corn, wheat, cotton, rice, milk, and later soybeans – that lend themselves to large-scale production, easy storage, and long-distance shipping *(545).* • Many countries have an agriculture system that fails to produce the mix and quantity of foods necessary for their citizens to consume a high quality diet. There is evidence to support the use of incentives to encourage the local production of healthy foods, more specifically fruits and vegetables. There is a supply gap for fruits and vegetables in many countries, but this is especially true in low-income countries *(548, 551, 628, 629).*	Strengthen and improve local and regional food systems, in part through agricultural policies and incentives that promote local food production and processing. Incentives can include forming grower cooperatives, instituting revolving loan funds, and building markets for local farm products through economic development. Additional incentives include but are not limited to farmland preservation, marketing of local crops, zoning variances, subsidies, streamlined license and permit processes, and the provision of technical assistance.	IOM *(630)*; FAO *(631)*; individual experts *(606, 632)*
	Use of incentives to promote the production of fruits and vegetables for local markets.	The Chicago Council on Global Affairs *(540)*
	In low- and middle-income countries, Aid for Trade programmes could be used to support the local production and supply of fruits and vegetables.	Individual experts *(633)*
	Adoption of programmes to protect fruit and vegetable farmers from natural disasters in a manner comparable to programmes that are available for farmers producing major commodity crops such as corn, soybeans, and wheat and loan and conservation	The Chicago Council on Global Affairs *(540)*

Agricultural policies		
Evidence	Consensus policy recommendations	Proposed by
	programmes aimed at fruit and vegetable producers.	
• There is mixed evidence about the links between agricultural subsidies and obesity. One side of the argument links the presence of subsidies to the predominance of "cheap foods and calories" through the overproduction of commodities that are the basic ingredients of processed, energy-dense foods and "soft" drinks *(545, 562)*. Studies suggest that the effect of these agricultural policies is negligible owing to the weak relationship between farmgate prices and retail prices *(550, 554)*. A recent report in the USA concludes that subsidy removal as a means to combat the overconsumption of unhealthy foods and beverages is an ineffective obesity prevention strategy, as it would not be sufficient, as a stand-alone measure, to affect the price or production of these products, and could harm small and midsized family farmers in the process *(550)*. A recent study also examined the effects of USA agricultural policies on prices and quantities of 10 agricultural commodities and nine food categories in the USA over time (1992, 1997, and 2002). The results indicate that – holding all other policies constant – removing USA subsidies on grains and oilseeds in the three periods would have caused caloric consumption to decrease minimally whereas removal of all USA agricultural policies (including barriers against imports of sugar and dairy products) would have caused total caloric intake to increase. The authors conclude that the influence of agricultural policies on caloric intake has diminished over time *(634)*. Only one systematic review has	There is a need to examine and monitor the implications of agriculture policy for obesity. In the USA, a recommendation has been made for the President to appoint a Task Force on Agriculture Policy and Obesity Prevention to evaluate the evidence on the relationship between agriculture policies and the American diet, and to develop recommendations for policy options and future policy-related research, specifically on the impact of farm subsidies and the management of commodities on food prices, access, affordability, and consumption.	IOM *(446)*; individual experts *(545)*

Agricultural policies		
Evidence	Consensus policy recommendations	Proposed by
been identified that focuses on the empirical evidence linking agriculture-based food price policies with nutrition outcomes. The authors report that there is currently no direct evidence that agricultural policies that directly influence the price of food affect rates of undernutrition. However, three studies that evaluated the effect of these policies on overnutrition suggested that they had a small effect on adult weight and risks of nutrition-related chronic disease *(551).*		
• Experts and international organizations emphasize the need to better align health and agricultural policies *(631, 635, 636).*	Review national policies and investments and integrate nutrition objectives into food and agriculture policy, programme design and implementation, to enhance nutrition sensitive agriculture, ensure food security and enable healthy diets.	FAO *(631)*
• There is evidence that urban agriculture projects can be a gateway to healthy foods but some studies have also highlighted urban health risks (e.g. irrigation may increase the risks for water-borne diseases) and implications for the environment (e.g. use of fertilizers and pesticides) *(637, 638)*.	Support urban agriculture in order to improve access to fresh and healthy foods, in a way that minimizes risks to the environment.	FAO *(639)*; individual experts *(637, 638)*
• Production and distribution of certain foods, like fruits and vegetables, tend to be very fragmented. Consumers don't necessarily have easy access to locally grown foods *(629)*.	Improve food supply chains (traditional and modern) to increase the local availability, affordability, diversity and nutritional quality of foods.	FAO *(629)*; individual experts *(548)*

Agricultural policies		
Evidence	Consensus policy recommendations	Proposed by
• Post-harvest losses (still up to 30–40% in some low- and middle-income countries) is a significant problem with negative impacts on healthy food availability and affordability. There is evidence of local solutions to address this challenge, but not on a large scale *(540)*.	Develop local solutions to reduce post-harvest food losses.	Chicago Council on Global Affairs *(540)*; individual experts *(640)*
• Loss of agricultural biodiversity has a negative impact on food security, diets and nutrition. There is a need to build production systems that deliver intensification without simplification- there is evidence available about the use of varietal mixtures, species intercropping and broader diversification strategies *(631, 641)*.	Preserve and promote agricultural biodiversity/ promote the diversification of crops including underutilized traditional crops.	FAO *(631)*; individual experts *(641)*

AJPM: American Journal of Preventive Medicine; CDC: United States Centers for Disease Control and Prevention; IOM: United States Institute of Medicine; NICE: United Kingdom's National Institute for Health and Care Excellence.

Table A4.6. Overview of evidence on trade policies

Trade policies		
Evidence	Consensus policy recommendations	Proposed by
• Experts and international organizations emphasize the need for policy coherence between health and trade policies - there is evidence that health impact assessments can be an effective tool for enabling greater policy coherence *(555, 642).*	Promote "policy coherence" between health policies and trade agreements in order to ensure that they are formulated in ways which enable both trade objectives and public health objectives to be met at the same time. Governments are encouraged to examine food and agricultural policies for potential health effects on the food supply. Strengthen capacity to undertake rigorous health impact assessments in relation to free trade agreements. The diet-related dimensions of interest include: – Trade in goods – Trade in services – Regulation of intellectual property rights (TRIPS and TRIPS Plus provisions) – Investment protections such as expropriation and investor state dispute settlement procedures – Government procurement – non-tariff barriers to trade (e.g. quotas, import/export licenses, administrative barriers) – Sanitary and phytosanitary measures	WHO *(310, 555)*; individual experts *(642)*
• Studies have linked trade liberalization with the quickening pace of nutrition transitions in all parts of the world. There are several provisions of trade agreements that can be a threat to local food production, systems and	There are increasing calls for having safeguards on the level of foreign ownership of agricultural land and of local food production and for trade agreements to protect national and	Individual experts *(556)*; FAO *(644)*

Trade policies		
Evidence	Consensus policy recommendations	Proposed by
environments (e.g. in the context of significant agricultural subsidies in high-income countries, local production of – often healthier – foods cannot compete in low- and middle-income countries) *(555, 562, 642, 643)*. However, there is also evidence that trading relationships and bilateral agreements can have a positive impact on the national food supplies *(559)*.	local food sovereignty in more general terms.	
	There are examples of trade agreements where tobacco and alcohol were excluded from tariff reduction following submissions on the health and financial implications of their inclusion. There is potential to explore a similar scenario in relation to food and obesity prevention.	Individual experts *(559)*
• Exploratory analyses of select trade agreements have shown that WTO Member States probably enjoy a significant degree of latitude in developing food regulations as part of an obesity prevention strategy, so long as those do not disproportionately affect imported products and therefore raise questions of discrimination. Similarly, there are different instruments and measures that can be used to support local/sustainable food systems without triggering trade disputes (*645)*.	Conduct research and analyses of trade agreements to identify policy instruments and measures that can be used to support local food systems and obesity prevention strategies triggering trade disputes.	Individual experts *(645, 646)*
	The WTO Agreement on Agriculture (AoA) and the Doha Development Agenda (DDA) give special rights to developing countries. Developing countries have the necessary scope to devote resources to agriculture in a manner which would provide incentives for the production, processing, marketing and consumption of certain crops relative to others, e.g. for the promotion of healthy diets. Not all countries are willing or able to take advantage of the available policy instruments under the AoA and DDA. In an environment characterized by policy coherence, partnerships with relevant stakeholders, good governance, and supportive legislative and regulatory measures, agricultural policies could play a meaningful role for	Individual experts *(647)*

Trade policies		
Evidence	Consensus policy recommendations	Proposed by
	obesity prevention.	
• The expansion of trade in agricultural commodities has potential impacts on the environment and on human health and nutrition, impacts that usually receive little attention in international trade discussions, despite their close relationship to the right to adequate food *(648).*	People and their livelihoods need to be placed at the centre of all international policy and development efforts. The right to adequate food, including nutritional considerations, should be part of the overall goal and policy of all institutions regulating or deregulating international trade, investments, development loans and external debt relief.	Individual experts *(648)*
• There has been evidence of negative impacts of trade agreements on policy space and institutional capacity since the establishment of the WTO. For example, the Technical Barriers to Agreement Trade (Agreement TBT) has important implications for governments' ability to regulate food marketing and labelling of processed foods. Investment and investor rights concerns in trade agreements can place greater constraint on domestic policy options and hence opportunities for governments to pursue nutrition and health goals *(556)*.	Provisions could be made to exclude health and social security from trade and investment agreements, as a form of safeguard to protect the right of democratically elected governments to maintain policy space for regulation, licensing, cost-containment and limiting or reversing commercialization, where this is in the public interest.	Individual experts *(555, 649)*
• Trade-related programmes and agreements could be used to boost the production of fruits and vegetables in many low-and middle-income countries *(633)*.	It has been recommended to broaden the scope of the Aid for Trade programme in order to tackle the issue of low fruits and vegetables supply and intake in low-and middle-income countries. The Aid for Trade model is based on increasing	Individual experts (*633*)

Trade policies		
Evidence	Consensus policy recommendations	Proposed by
	productivity by improving markets rather than subsidies.	
• Experts have highlighted the potential contributions of the Codex Alimentarius for obesity prevention at the global level. The work of the Codex Commission goes beyond creating means of removing barriers to trade. It also includes encouraging food traders to adopt voluntarily ethical practices as an important way of protecting consumers' health and promoting fair practices in the food trade. To this end, the Commission has published the Code of Ethics for International Trade in Food. A principal objective of the Code of Ethics is to stop exporting countries and exporters from dumping poor-quality or unsafe food on to international markets. International trade in food should be conducted on the principle that all consumers are entitled to safe, sound and wholesome food and to protection from unfair trade practices.	The Codex Alimentarius can be an appropriate mechanism to promote healthier diets worldwide and fair practices in food trade through its role in setting standards on labelling, and, to a lesser extent, food composition.	Individual experts *(650)*
	Research could inform the development of future Codex standards. For example on food labelling, the INFORMAS study proposes a new taxonomy that goes beyond Codex Alimentarius definitions to differentiate the following labelling components: ingredient list, nutrient declarations, supplemental nutrition information, nutrition claims and health claims.	Individual experts *(651)*

FAO: Food and Agriculture Organization of the United Nations; WHO: World Health Organization; WTO: World Trade Organization.

Table A4.7. Overview of evidence on food sector interventions

Food/nutrition		
Evidence	Consensus policy recommendation	Proposed by
Food packaging labelling		
• A systematic review of the use and understanding of pre-packaged prepared food labels and of the effect of labels on behaviour found that reported food label use was generally high (from 47% in the European Union to 82% in New Zealand) *(314).* • Young and middle aged adults were more likely to use and understand food labels than children and adolescents for whom interventions have had mixed results in terms of their effect on increasing the use of food labelling *(314)*. • Label use was shown to vary between subgroups (lower income and ethnic minorities use them less) *(314)*. • The understanding of labels has been shown to be limited with confusion reported around daily recommended amounts, percent of daily values, serving sizes and comparing labels between products; understanding has been shown to be generally higher among younger adults and those with higher income and education levels *(314)*. • • The understanding of labels can be improved with simple and easy to understand graphics, such as a traffic light system that indicates if a food contains high, moderate or	Front of package labelling.	EATWELL *(567)*; WHO *(566)*; WCRF *(569)*; IOM *(652)*
	Simple information (such as traffic light scheme) with consistent format and placement.	EATWELL *(567)*; WHO *(566)*; WCRF *(653)*; IOM *(652)*; McKinsey *(449)*
	Mandated nutrition facts panels or front-of-pack labels/icons	AHA *(568)*; McKinsey *(449)*
	The most critical nutrition components for front of package symbol labelling are: calories, saturated and trans fats, sodium, and added sugars.	IOM *(652)*
	Clear information on the package on portion size with nutritional information per portion.	McKinsey *(449)*
	Endorsement schemes (such as a healthy choice symbols) identifying products on front of package that meet specific standards.	WHO *(566)*
	Supermarket schemes identifying and	NOURISHING *(569)*;

low quantities of contents such as saturated fats or sugars *(314)*. • Some studies have found that front of packaging labelling is more effective and more frequently viewed than labels placed elsewhere on the package (*314*) and may particularly benefit those with limited nutrition education or understanding of food labels *(314)*. • Observational studies have found the use of nutritional labels to be linked to healthier diets, including lower fat and salt consumption *(314)* three longitudinal studies assessing the impact of the US Nutrition Labeling and Education Act found that after the act came into effect, frequent label users had a greater likelihood to consume a low-fat diet compared with both non-label-users in 1995 and label users in 1989, BMIs of label users decreased following implementation of the Act, and that purchases of low fat and low sodium foods increased significantly following implementation of the act *(314)*. • Disclosure of nutritional information through labelling may encourage food product reformulation in order for the product to look better on the label (e.g.: in order to claim that a product is "low in fat") *(567).*	promoting healthy food options at point of purchase. Regulate nutrient and health claims that can be made on packages or promoting food items (e.g. permissible fat content for a food marketed as "low in fat").	McKinsey *(449)*; AHA *(568)* NOURISHING *(569)*
Menu labelling		
• When eating outside of the home (i.e. in restaurants), higher levels of energy and fat and a lower micronutrient value have been shown to be consumed compared with food prepared and eaten at home *(654)*.	Quick service restaurant menu labelling. Aggregate nutritional content information on food purchased and traffic-light labels provided	WHO *(566)*; NOURISHING *(569)*; McKinsey *(449)* McKinsey *(449)*

• Evidence on the impact of menu labelling is limited. One systematic review of the effect of menu calorie-labelling on calories purchased found mixed results: 3 of 7 studies showed some reduction, 3 studies showed no change and one showed a mild increase of calories purchased when calorie-labelling is present compared with no labelling; when a reduction in calories was found, it was very modest and unlikely to be clinically significant *(655)*.	by "fast food" restaurant at checkout	
	Use menu labelling alongside other interventions to influence healthier menu choices (e.g. reformulation, reduced portion sizes, and education on the labelling scheme).	EATWELL *(567)*
	Monitor real-life menu labelling experiments to expand the evidence base.	EATWELL *(567)*
Improve the quality of the food supply		
• A series of case studies examining the impact of existing reformulation policies in Europe found that existing policies to reduce salt content have modestly reduced salt content in reformulated foods and salt consumption, though salt consumption levels remain above targets *(656)*. • In both Denmark, where trans-fatty acid reduction was mandatory, and the United Kingdom, where trans-fatty acid reduction was voluntary, population trans-fatty acid levels are reported to be below maximum daily recommended levels *(656)*; however, some reports have indicated that mandatory reductions are more effective and that under voluntary regulations, certain foods, that may be preferentially eaten by certain subpopulations, could still be high in trans fat *(567)*. • To date, little information is available regarding the impact of policies to reduce saturated fatty acid and sugar content	Improve availability of healthy foods in the food system, including fruits and vegetables (see Agriculture and Trade sections)	
	Voluntary or regulated reformulation of food products to reduce specific nutrients. E.g.:	McKinsey *(449)*; EATWELL (*567*); AHA *(568)*
	• Reduce salt and fat contents	NOURISHING *(569)* EATWELL *(567)*
	• Regulate or eliminate trans-fatty acids	EATWELL *(567)*; WHO *(566)*; NOURISHING *(569)*
	• Reduce energy density	
	• Develop beverages with reduced sugar content	IOM *(446)*

in foods *(567)*. • Experts suggest that it is important to address the issue of replacement nutrient selection in food reformulation: studies showing health improvements by removing saturated fats from the diet generally replace them with polyunsaturated fatty acids, fruits and vegetables or whole grains. The use of sugars and easily digested carbohydrates in place of saturated fatty acids is unlikely to have an effect on obesity or its related noncommunicable diseases and may even increase a food's overall energy content *(653, 657)*. • Package and serving sizes of processed foods, "fast foods" and other "convenience foods" have increased since the 1970s with larger portions promoted as better value *(653)*; availability of larger portion sizes has been linked with greater energy intakes and the reduction of portion sizes has been linked with reduced energy intake *(653)*.	However, to avoid implicitly promoting heavily processed foods in developing countries where they are rarely consumed, policies for reformulation should focus on countries where markets are already saturated with heavily processed food.	Moodie et al. *(333)*
	Reduce portion size of processed meals, dishes, snacks, foods, and drinks. E.g.: Remove "supersize" items from menus.	WCRF *(653)*; NOURISHING *(569)*; IOM *(446)*; McKinsey *(449)*
	Reduce the number of calories served to children in quick service restaurants.	IOM *(446)*
Informed choice and public information campaigns		
• Evaluations of public awareness campaigns promoting fruit and vegetable consumption have shown both an increase in fruit and vegetable consumption and an increase in public awareness *(568)*. • Shorter term campaigns have had some effect on public awareness but appear to have less reach to lower	Longer-term campaigns.	EATWELL *(567)*; IOM *(446)*; AHA *(568)*; Academy of Nutrition and Dietetics *(318)*
	Simple targeted messages.	WHO *(566)*; IOM (446); AHA *(568)*

socioeconomic groups or other minority groups and may not have an effect on behaviour *(568)*. • Public awareness campaigns utilizing multiple channels for messaging were more likely to be effective and impact behaviour *(568)*. • A systematic review found that campaigns were more likely to be successful if they targeted a specific food and ran for longer periods of time *(568)*. • Multicomponent campaigns that include public awareness campaigns along with other population strategies such as regulations or economic interventions are recommended; however, the specific impact of a public awareness campaign within a larger intervention is difficult to assess *(568)*.	Frequent exposure to messaging.	WHO *(566)*; IOM *(446)*; AHA *(568)*; Academy of Nutrition and Dietetics *(318)*
	Comprehensive public-health campaign promoting healthy eating habits across various media such as television, radio and social media.	McKinsey *(449)*; AHA *(568)*; Academy of Nutrition and Dietetics *(318)*
	Implement public information campaigns in conjunction with other measures within a comprehensive obesity prevention strategy. including increasing availability, labelling or reformulation.	EATWELL *(575)*; WHO *(566)*; WCRF *(653)*; AHA *(568)*
Dietary guidelines		
• A systematic review of food-based dietary guideline awareness, comprehension and use showed that public awareness of guidelines and recommendations varied across studies and populations but appeared to increase over time *(575)*. • There is an inconsistent relationship between consumer awareness and understanding of guidelines; studies have identified confusion among guideline users regarding portion sizes and abstract terms such as “low fat” and “low sugar” *(575)*. • There is limited evidence regarding the effect of guideline use, although one study from China demonstrated that the percentage of school children who ate a healthy breakfast	Evidence-based national guidelines on healthy eating that are updated regularly, adapted for specific populations and effectively communicated to the population.	WHO *(566)*
	Develop and implement national food-based dietary guidelines.	FAO/WHO *(660)*
	Educate the public on food-based guidelines.	NOURISHING *(569)*
	Efforts should be made to keep	Aranceta & Perez-Rodrigo

doubled following the promotion of the 1997 Guidelines for Chinese residents and Food Guide Pagoda *(575)*. • A systematic review of children's dietary intake studies covering 30 countries showed that in most countries the majority of children were consuming too much saturated fat and insufficient polyunsaturated fat compared with the WHO 2003 general population nutrient intake goals *(658)*. • The underlying information providing the basis for guidelines is important to consider: a recent systematic review of dietary guidelines suggests that the evidence behind some recommendations may be limited *(659)*.	recommendations evidence-based and to grade evidence in a way that is easy for guideline users to understand.	*(659)*

AHA: American Heart Association; FAO: Food and Agriculture Organization of the United Nations; IOM: United States Institute of Medicine; WHO: World Health Organization; WCRF: World Cancer Research Fund.

Table A4.8. Overview of evidence on fiscal measures

Fiscal measures		
Evidence	Consensus policy recommendations	Proposed by
Taxation to shift consumption from unhealthy foods and beverages (in general)		
• Most reviewers find low/weak evidence of effectiveness of fiscal measures on unhealthy foods. Even in the USA, where assessment of evidence on food and beverage taxes has been a focus, "Additional research is needed to be able to draw strong policy conclusions regarding the effectiveness of fiscal-pricing interventions aimed at reducing obesity" (Powell and Chaluptka 2009) *(661)*. There is evidence of relationship between price and consumption, particularly in the USA, but gaps in knowledge about substitute products, or the price/consumption relationship in developing countries. The relationship with health outcomes is less robust. Moodie et al. (2013) found a large proportion of the studies were modelling studies or "grey" literature *(572)*. • The regressive nature of consumption taxes, which have a greater negative economic impact on low-income households, is given significant attention by all reviewers. Balancing this, most look to the USA evidence that low-income consumers are likely to be more responsive to price changes and would "reap greater benefit" or have greater "health gain" (e.g. Escobar 2013) *(578)*.	While more evidence is needed, the introduction of fiscal policies as obesity prevention measures should be considered.	Moodie et al. *(572)* McKinsey *(449)* WCRF NOURISHING Framework 2013 *(569)* World Health Assembly resolution 66.10 *(450)* WCRF *(570)* EATWELL *(567)*

Fiscal measures		
Evidence	Consensus policy recommendations	Proposed by
	Where the empirical evidence is still not sufficiently strong, the most important criteria for considering a policy is the potential for harm such a policy might cause, rather than the extent of its impact on obesity."	Faulkner et al. *(576)* McKinsey *(449)* Sassi *(577)*
Tax on sugar-sweetened beverages (SSBs)		
• Several examples, of natural experiments that have been evaluated, involve modest taxes that were sufficient to see statistically significant changes in consumer behaviour, but either small or not significant changes in BMI (e.g. Faulkner et al. 2011) *(576)*. • Escobar et al. (2013) found evidence to suggest that, in the USA and other countries, an increase in price could be used to shift consumption from SSBs to healthier beverages (whole milk and fruit juice) *(578).*	"Three-quarters of the panel recommended moving forward with a tax on caloric sweetened beverages … such a recommendation was described as a "leap of faith" given the incomplete evidence base."	Faulkner et al. *(576)*
	"Taxing SSBs may reduce obesity. Future research should estimate price elasticities in low- and middle income countries and identify potential health gains and the wider impact on jobs, monetary savings to the health sector, implementation costs and government revenue. Context-specific cost-effectiveness studies would allow policy-makers to weigh these factors."	Escobar et al. *(578)*
Tax on unhealthy foods		
• Faulkner et al. (2011) *(576)* consensus panel's review, among others, viewed the evidential base for policy in this area to be more compelling than the evidence to support a beverage tax. In particular, they were swayed by the 4 longitudinal studies that suggest that low fast-food prices	Recommendation against proposing such taxes at this time, citing a number of difficulties with the design and implementation of food taxes that require further research before specific recommendations can be made.	Faulkner et al. (*576)*

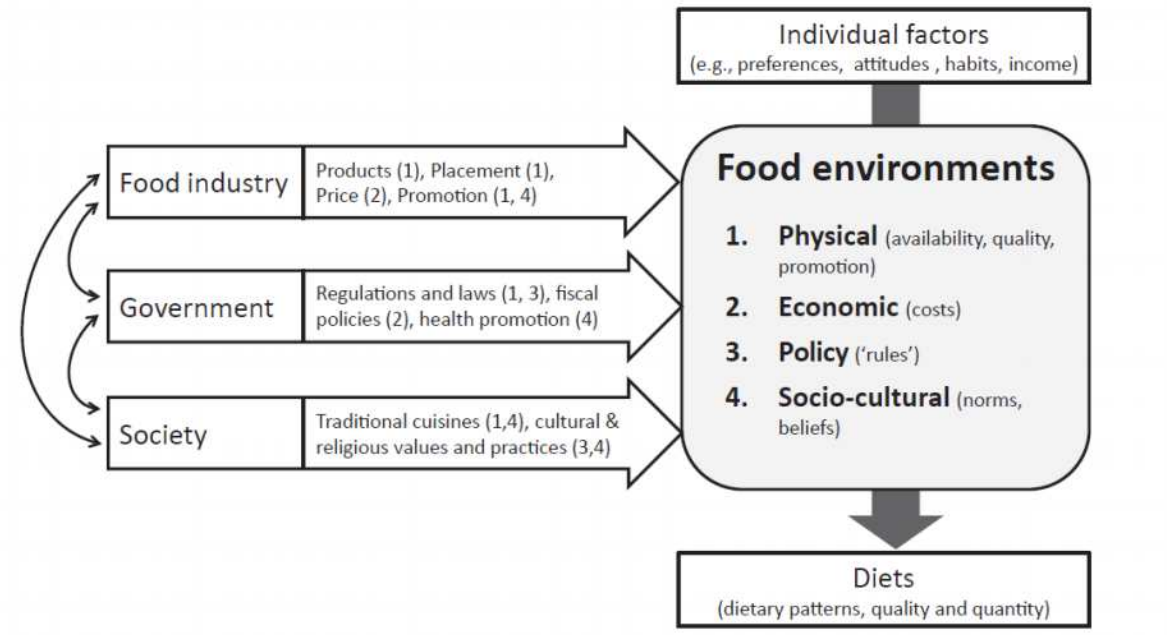
Fiscal measures		
Evidence	Consensus policy recommendations	Proposed by
increase weight outcomes and 6 studies employing cross-sectional data.		
Subsidies on healthy foods		
• Several groups/panels reviewed the same recent literature. The evidence clearly demonstrated a link between lower obesity risk and greater fruit and vegetable consumption although the mechanisms for this relationship are unclear. For example, Faulkner et al.'s (2013) consensus review could not "identify any study evaluating the impact of fruit and vegetable subsidies on overweight and obesity directly. However, there is evidence on the effect of prices of fruits and vegetables on weight outcomes, including 4 longitudinal studies and 3 cross-sectional studies *(576)*. Sturm and Datar found that in the USA changes in children's weight were positively related to the price of fruits and vegetables but not to changes in meat, dairy, or "fast-food" prices *(662)* Powell and Chaloupka (2009)'s USA subpopulation analysis suggest that children living in poverty and those at risk for overweight were more price sensitive compared with their non-poor and not-at-risk counterparts. For adolescents, there is weaker evidence that fruit and vegetable prices have an influence on body weight (*661*).	Healthy food, especially fruit and vegetable subsidies, primarily targeting children and low-income households.	Faulkner et al. *(576)* Powell and Chaloupka *(661)* WCRF International NOURISHING framework 2013 *(571)* EATWELL 2013
	Flexible financing or tax credits to support health promoting food and beverage retailing and distribution policies	IOM *(446)*
Financial incentives to promote physical activity		

Fiscal measures		
Evidence	Consensus policy recommendations	Proposed by
• Reviewers conclude that there is relative "paucity of data" on the effectiveness of fiscal measures to encourage physical activity. A review by Martin et al. argues that financial incentives offer a potentially large, but as yet unexplored, role in promoting walking and bicycling *(662)*. The interventions covered include both the use of positive financial incentives to promote active travel, as well as negative incentives to reduce vehicle transport. However, the authors highlighted the lack of empirical evidence in this area. • Another review by Jeffery (2012) focused on empirical studies between 1972 and 2010 which evaluated the use of financial incentives to promote weight control. The research supports the notion that financial rewards can be a motivating factor for people wishing to lose weight, particularly in the short term. However, the results across studies varied widely *(663).*	Systemic and individual financial incentives and disincentives to promote physical activity.	McKinsey (*449*) WCRF (*570*)
Combined or multipronged fiscal measures		
• On the basis of logic, and evidence of the effectiveness of healthy food subsidies targeted to low-income groups, Powell and Chaloupka, 2009 *(661)* and Faulkner et al. 2011 *(576)*, among others, also note that targeted subsidies of healthy foods could offset the regressivity of taxes to increase the prices of unhealthy foods while improving policy effectiveness. The optimal balance of taxes and	Combined/multipronged approach, especially for children and adolescents, of changing relative prices by both taxing less healthy, energy-dense foods and subsidizing healthier, less dense foods.	Powell and Chaloupka *(661)* Thow et al. *(338, 664)* Faulkner et al. *(576)*

Fiscal measures		
Evidence	Consensus policy recommendations	Proposed by
subsidies could be developed in consideration of country or sub-region specific estimates of the responsiveness of consumption behaviour to prices. • Some reviewers also note evidence that the effectiveness of fiscal measures can be reinforced by health promotion messaging. Thow 2010 notes that, "Being aware that a product has been taxed because it is unhealthy may discourage purchases".		WHO *(310)* McKinsey *(449)*
	The acceptable level of tax/subsidy and the most-effective fiscal policy or combination of policies is likely to be context-dependent.	Moodie et al. *(572)*
	Health impact assessments recommended to determine the unintended effects of new and/or existing fiscal measures designed to target other sectors. Removal of subsidies to unhealthy foods.	Lobstein et al. *(665)* WCRF *(570)*

IOM: United States Institute of Medicine; WHA: WHO World Health Organization; WCRF: World Cancer Research Fund

Figure A4.2. Food environments and their four main components; the major influences of the food industry, governments and society on food environments (and their interactions); and the interaction between individual factors and food environments to shape diets



Source: Swinburn et al. *(565)*.

Table A4.9. The NOURISHING framework

Domain		Policy Area	Policy Options/Actions
Food environment	N	Nutrition label standards and regulations on the use of claims and implied claims on foods	e.g. Nutrient lists on food packages; clearly visible "interpretive" and calorie labels; menu, shelf labels; rules on nutrient and health claims
	O	Offer healthy foods and set standards in public institutions and other specific settings	e.g. Fruit and vegetable programmes; standards in education, work, health facilities; award schemes; choice architecture
	U	Use economic tools to address food affordability and purchase incentives	e.g. Targeted subsidies; price promotions at point of sale; unit pricing; health-related food taxes
	R	Restrict food advertising and other forms of commercial promotion	e.g. Restrict advertising to children that promotes unhealthy diets in all forms of media; sales promotions; packaging; sponsorship
	I	Improve the quality of the food supply	e.g. Reformulation; elimination of trans fats; reduce energy density of processed foods; portion size limits
	S	Set incentives and rules to create a healthy retail environment	e.g. Incentives for shops to locate in underserved areas; planning restrictions on food outlets; in-store promotions
Food system	H	Harness supply chain and actions across sectors to ensure coherence with health	e.g. Supply-chain incentives for production; public procurement through "short" chains; health-in-all policies; governance structures for multi-sectoral engagement
Behaviour change communication	I	Inform people about food and nutrition through public awareness	e.g. Education about food-based dietary guidelines, mass media, social marketing; community and public information campaigns
	N	Nutrition advice and counselling in health care settings	e.g. Nutrition advice for at-risk individuals; telephone advice and support; clinical guidelines for health professionals on effective interventions for nutrition
	G	Give nutrition education and skills	e.g. Nutrition, cooking/food production skills on education curricula; workplace health schemes; health literacy programmes

Source: Hawkes et al. *(149)*.

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