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

18 – 20 June 2014 - Geneva, SWITZERLAND
WHO Library Cataloguing-in-Publication Data


© World Health Organization 2014

All rights reserved. Publications of the World Health Organization are available on the WHO website (www.who.int) or can be purchased from WHO Press, World Health Organization, 20 Avenue Appia, 1211 Geneva 27, Switzerland (tel.: +41 22 791 3264; fax: +41 22 791 4857; e-mail: bookorders@who.int).

Requests for permission to reproduce or translate WHO publications – whether for sale or for non-commercial distribution – should be addressed to WHO Press through the WHO website (www.who.int/about/licensing/copyright_form/en/index.html).

The designations employed and the presentation of the material in this publication do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. Dotted and dashed lines on maps represent approximate border lines for which there may not yet be full agreement.

The mention of specific companies or of certain manufacturers’ products does not imply that they are endorsed or recommended by the World Health Organization in preference to others of a similar nature that are not mentioned. Errors and omissions excepted, the names of proprietary products are distinguished by initial capital letters.

All reasonable precautions have been taken by the World Health Organization to verify the information contained in this publication. However, the published material is being distributed without warranty of any kind, either expressed or implied. The responsibility for the interpretation and use of the material lies with the reader. In no event shall the World Health Organization be liable for damages arising from its use.

This publication contains the report of the first meeting of the ad hoc working group on science and evidence for ending childhood obesity and does not necessarily represent the decisions or policies of the World Health Organization.
BACKGROUND

1. The prevalence of obesity in children is increasing around the world. The reduction in childhood obesity is a critical target as part of a strategy to promote healthy life expectancy, not only for the child, but also for the next generation. Life-course studies suggest that interventions in early life, when biology is most ‘plastic’ and amenable to change, are likely to have sustained effects on health, particularly because they can influence responses to later challenges such as living in an obesogenic environment. Interventions aimed at preventing childhood obesity would lead to a reduction in co-morbidities in children, and to a reduction of the long-term burden of non-communicable diseases. Early life is a window in the life-course where there is strong political consensus that action is desirable, including considerations of vulnerability, equity, the rights of the child and gender issues. The combination of short-term direct and indirect benefits and longer-term effects in the primary prevention of non-communicable diseases (NCDs) creates a powerful economic and social argument for action. A focus on childhood obesity complements and potentiates, but does not replace, the need for secondary and tertiary prevention and treatment of obesity and its sequelae. If childhood obesity could be successfully addressed, the benefits derived would include improvements in maternal and child health, greater human capital gain through academic achievements in young people today and in the next generation, improvements in productivity, longevity, and health and wealth capital in many countries. Addressing childhood obesity thus has a compelling logic. New scientific insights offering novel opportunities for interventions in the pre-conceptional period, infancy and childhood can complement existing maternal and child health interventions and potentially augment their effectiveness. There needs to be clear guidance on what combinations of interventions are likely to be most effective in different contexts across the globe, and the surveillance and accountability mechanisms required for stakeholders at multiple levels.

INTRODUCTION

The nature and scope of the problem

2. 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 a range of health problems, including asthma, high blood pressure, musculoskeletal disorders, fatty liver disease, insulin resistance and type 2 diabetes, as well as obstructive sleep apnoea. In later life, they are at greater risk of obesity, type 2 diabetes, cardiovascular disease, some cancers, obstructive respiratory disease, mental, emotional and social health problems and reproductive disorders. In addition to these health risks, the rapid rise in obesity negatively impacts on their opportunity to participate in educational and recreational activities, and imposes a range of economic burdens at familial and societal levels. The rapidly rising rates of childhood obesity and subsequent increasing burden of disease and disability have grave social and economic consequences, contributing to the rising cost of health services, limiting economic growth and exacerbating health inequalities and inequities.
3. At the societal level, increasing levels of urbanisation, even in currently semi-rural settings, are resulting in lower levels of physical activity in work, changing patterns of transport and leisure activities requiring less energy expenditure, an overall more sedentary lifestyle, and adverse changes in dietary intake. In the well-recognized “nutrition transition” and more recent “physical activity transition”, traditional diets and lifestyles are replaced by more Westernized ones. Major changes are the shift from diets based on unprocessed foods to those with a high in fat, sugar and salt, and an increase in meat consumption at the expense of vegetables and fruit, and a decrease in habitual physical activity and an increase in sedentary behaviours (especially screen time). In many developing countries undergoing such changes in diets and lifestyles, food insecurity and undernutrition are often present alongside overnutrition (that is, excess energy intake in relation to energy needs leading to energy storage as fat, and obesity). Thus, many developing countries are increasingly suffering a “double burden” of undernutrition and overnutrition, and often both types of malnutrition exist concurrently in individuals (e.g. obesity and micronutrient deficiencies), households, and communities. Interventions to prevent overweight and obesity are therefore complementary to interventions to address undernutrition and poor development in children. Early child development interventions currently in place in many countries would provide a valuable programmatic link and an opportunity to integrate interventions aimed at reducing childhood obesity and improving child health.

The Life-Course Model

4. 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’ the 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. 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.

5. 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 (Fig 1). This ‘pathway
dependency’ includes 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 (GDM) which produce biological cues which
modulate the epigenetic processes, along with broader behavioural influences of
parents.

Figure 1 Life-course model of obesity and other NCD risk

![Life-course model of obesity and other NCD risk](image)

(From WHO Meeting Report: Nurturing human capital along the life course: Investing in early child development. 2013)

6. 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.

7. The biological processes of epigenetics and developmental plasticity are related to behavioural and wider contextual drivers of childhood obesity, and 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:

8. Although the life-course of the child commences at conception, new evidence from animal studies, epidemiology and a limited number of randomised 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 raises the prospect that “obesity begets obesity” and similarly, “healthy living begets healthy living”.

Types of risk factors

9. 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, emphasising however that the distinction between them is not absolute and within each there are multiple factors.

10. Biological risk factors include maternal malnutrition (unbalanced nutrition, including both under- and over-nutrition), obesity, stress before and during pregnancy, and conditions such as maternal glycaemia. These prenatal influences are exacerbated by a range of postnatal behaviours, including inadequate periods of exclusive breast-feeding; 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.

11. Behavioural risk factors overlap with these biological factors and include movement behaviours, which incorporate physical activity, sedentary behaviour and sleep, which are established in early childhood. Reduced opportunities for sport, increasingly mechanised 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 of meal times, and consuming large portions. Concerns over safety and the over-crowding 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 etc.

12. 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 of and changing nature of marketing to children, as recognized in World Health Assembly resolution WHA63.14: Marketing of
food and non-alcoholic beverages to children, is also a contributor to the wider environment.

**Relation to current global policy initiatives**

13. The life-course approach is encapsulated in clause 26 of the United Nations (Sept 2011) Political Declaration of the High-level Meeting of the General Assembly on the Prevention and Control of Non-communicable Diseases and the WHO Action Plan on the Prevention and Control of NCDs, presented at the World Health Assembly 2013. These note that maternal and child health is inextricably linked with NCDs and their risk factors, specifically as prenatal malnutrition and low birth weight create a predisposition to obesity, high blood pressure, heart disease and diabetes later in life; and that pregnancy conditions, such as maternal obesity and gestational diabetes, are associated with similar risks in both the mother and her offspring. This approach emphasizes that maternal health, including pre-conceptive, antenatal and postnatal care, breastfeeding and health and development promotion for children and adolescents are key.

14. In view of the importance of the life-course approach to reducing the risk of childhood obesity, it will be important to ensure that this perspective is incorporated into ongoing discussions of the Sustainable Development Goals (SDGs); these will build upon the Millennium Development Goals and converge with the post 2015 development agenda.

**Meeting Objectives**

15. This first meeting of the *ad hoc* Working Group on Science and Evidence was to initiate the development of the work needed to provide advice to the Director-General on four core areas:

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

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

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

   D. Monitoring and surveillance: what are the feasibility and mechanisms required to monitor and evaluate recommended policy options?
16. The *ad hoc* Working Group on Science and Evidence consists 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 Annex 1.

17. The meeting aimed to develop a brief situational analysis, outlining current knowledge about the mechanisms underlying childhood obesity, potential evidence-based interventions and areas where further data are needed, and a draft set of recommendations to the Director General focussing on the core areas.

18. The *ad hoc* Working Group on Science and Evidence will also propose recommendations to the Director-General regarding monitoring and surveillance. A proposed *ad hoc* Working Group on Implementation, Monitoring and Accountability will comprise experts in monitoring and accountability, together with representatives from the different stakeholders that have an important role in ending childhood obesity, including governments, civil society, groups representing children, advocates for child health and nutrition, international organizations, and industry.

19. This report synthesises the issues raised during discussions on 18-20 June 2014, the consensus reached on the draft recommendations to the Director-General and the agreed action points and working method of the Working Group.

20. The increasing prevalence and incidence of obesity in children requires immediate attention. Children with obesity suffer severe health consequences in childhood and are at high risk of becoming obese adults, with resulting increased risk of non-communicable diseases. The dramatic increase in the obese population will have considerable implications for society in the future, both in terms of health needs and burden and increasing inequalities in health due to the trends in obesity associated with lower socioeconomic status.

21. 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, e.g. age, gender, socioeconomic status, cultural, ethnic and urban-rural differences. Further focusing and refining epidemiological data collection can make a major contribution to the ability to meet 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. Some key issues discussed were:

a. There is a need to review carefully the strengths, limitations, and gaps in current data available to the WHO NCD and nutrition monitoring and surveillance frameworks, and make improvements that will ensure accurate and timely identification of positive and negative changes in the dynamics of the obesity epidemic. Ideally, status and trend data will include: both obesity prevalence (number and percent of those currently obese) and 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.

b. 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.

c. Given the marked inequities in obesity associated with both demographic characteristics, urbanisation 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.

d. Key ‘windows of opportunity’ when interventions can prevent excess weight gain in a sustained manner need to be identified.

e. Quantification of the attributable risk of the various influences on obesity development is challenging but necessary, taking into account that influences on obesity act as a complex system in which neither causes nor intervention effects operate via a single pathway.

f. 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, socio-economic change, and associated nutrition, activity,
g. 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. Changes in adiposity and body composition may be important considerations for health as part of the overweight continuum.

h. 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 tails of the BMI distribution to gain insights into severity of obesity (e.g. mean BMI of overweight and obese children has increased). Measuring the reverse trends, although much less frequent, may provide insight for intervention strategies.

i. 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.

j. 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.

k. There is potential to identify new risk factors or patterns of vulnerability to risk factors in ways that can lead to new intervention strategies, e.g. subgroups of infants for whom lack of breastfeeding poses a greater than average risk of developing obesity (such as offspring of GDM pregnancies).

l. There is potential to identify or clarify the roles of novel risk factors for obesity development. The relationship between sleep duration and quality to obesity development is one such pathway which appears to be critical based on current evidence.

m. 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, social networking and information gathering to improve health behaviours, as well as its use by individuals and the resulting increase in sedentary behaviours.

n. It is important to improve our understanding of the features associated with reverse transition from obesity to overweight or normal weight, including gender, age and enabling factors.

o. Consistent with the life course approach, epidemiological studies of the
amplification of risk or cumulative risk due to carryover effects from prior periods are needed.

p. The relevance of household composition and family structures to risks of childhood obesity needs exploration.

22. The following research gaps were identified by the working group:

a. Data from existing country-level surveys should be used to conduct disaggregated analysis by specific childhood age groups and socioeconomic factors, and to determine the extent to which data need to be expanded in terms of geocoverage, frequency, data quality and fields to improve our understanding of childhood obesity.

b. The extent to which existing surveillance mechanisms should be expanded, possibly to monitor behaviours as well as disease burdens, needs to be determined.

c. The effect size and risk attributed to different risk factors should be better quantified, as this will be important in guiding interventions.

d. The attributable fraction of childhood obesity as a risk factor for adult obesity needs to be explored in different settings.

e. Monitoring the effectiveness of interventions, as well as the broader policy environment in countries will assist in understanding the dynamics of disease burden within and between countries, including key drivers and protective factors.

**Health and economic impact of childhood obesity**

23. Children depend on their families and society, and as such these have a responsibility to provide the optimal environment for healthy growth and development. This is recognized in international human rights instruments, such as the Convention on the Rights of the Child (Art 25), which obliges governments to fight disease and malnutrition by providing children with adequate nutritious food, clean drinking water and opportunities to play. These instruments recognize that childhood health lays the foundation for health in adulthood.

24. Some key issues discussed were:

a. There is an overwhelming benefit to intervening in early childhood because it has been demonstrated in developmental science that the costs of intervening later in life are greater and the intervention can be less effective. It is easier to modify risk
factors and optimise health early, due to factors such as plasticity, reversibility and conditioning.

b. There are already increasing economic costs (health care and associated costs) in dealing with adverse health consequences, including NCDs in childhood and these will increase over time with continuing obesity.

c. There are direct costs to the individual in terms of well-being and life expectancy.

d. There are indirect costs of childhood obesity which include lower educational attainment and lost productivity in adulthood.

e. Some of these costs are borne by individuals, some by society. These can be viewed as health capital for individuals and families, societies and countries. In a broader context, concepts such as healthy active living can also serve as a way to develop social capital through, for instance, community activities.

f. Personal lifestyle is influenced by the behaviour of others in the community. This suggests that the community can prove to be an effective agency for lifestyle improvement.

g. Policy-makers consider economic benefits in terms of impact on GDP, but this ignores significant components of the wealth of nations that are associated with health and associated social capital.

h. Investments to promote healthy behaviours, social norms and environment to prevent and or reduce obesity will have significant health, social and economic benefits.

i. There is reasonably good evidence on most of these factors from high income countries, but relatively little from low income countries.

j. The trans-generational effects of a poor developmental environment (including undernutrition) followed by an adequate/excessively rich environment (mismatch) leads to obesity and this has consequences for NCD risk. Obesity in one generation is linked to increased risk of obesity in the next. Thus there are trans-generational health capital benefits from preventing childhood obesity.

k. Failure to address childhood obesity will exacerbate inequalities in health, both within and between countries.

l. Clinical studies suggest a trend of earlier age of onset of adverse health consequences such as type 2 diabetes, cardiovascular disease, respiratory or joint problems.
25. A number of key research gaps were identified by the working group:

a. There are limited data, and only in high income countries, of the health care costs of obesity and related conditions in children and adolescents, but not on the indirect costs or on the costs in low and middle income countries. One of the limitations of the available studies on the economic impact of childhood obesity is that most of them consider brief time frames (1-2 years) and most do not cover adult life. It is, however, evident that children who are obese often remain obese in adulthood and thus, a life-long approach is required in measuring the economic impact.

b. Quantitative estimates of costs and benefits to the individual, the economy and the wealth of nations could be developed. Such estimates would be an invaluable tool for policy development.

c. Evidence on the economic impact (cost) of childhood obesity is useful to describe the scale of the problem and its impact on various economic actors. In order to inform policy-making on how to allocate scarce resources to prevent childhood obesity, more evidence is required on the efficiency (viz. cost-benefit analysis) of various interventions in different settings and particularly in low- and middle-income countries.

d. The estimates of the cost of childhood obesity require further study. A model is under development (see Appendix 1), which could be explored to further this work.

Mechanisms underlying childhood obesity

26. The working group first considered the evidence about causal pathways that potentially provide insights on where and when to intervene to prevent childhood obesity or stabilize childhood obesity levels. Given the focus on the life-course and the potential to identify novel intervention pathways or strengthen support for existing interventions, separate consideration was given to evidence on early life mechanisms underlying childhood obesity. The perspective was that, to optimize the reduction of risk factors for childhood obesity, there is a need to recognize that many factors may be already operating prior to pregnancy, and therefore that addressing the pre-conceptional period as a window of opportunity may be critically important.

27. This new perspective arises from a triangulation of evidence from animal experiments in a range of species with different developmental profiles; from randomized controlled trials involving pre-conceptional interventions, with outcomes related to pregnancy and the neonate but not yet childhood obesity; and from a range of observational studies in human populations.

28. It is important to note that ‘obesity begets obesity’ in more than one way. Prevention or control before reproductive age has beneficial effects for the next generation, an important consideration for long-term benefits to populations. Such a mechanism can also operate in a social and behavioural context, such that family members and close contacts might benefit from intervention in one person.
29. The biology of risk usually operates in a U shaped manner, being more detrimental at the lower and upper ends of exposure. This introduces a need to define optimal levels of intervention to avoid harm by doing too little or too much. However, some risk factors (tobacco and environmental toxins) are always harmful and need to be eliminated.

30. During discussions, the following issues were raised:

a. Many of the risk factors for obesity and therefore possible interventions are also linked to other adverse outcomes.

b. Susceptibility to the traditional risk factors was previously thought to be due to a combination of fixed genetic effects plus later lifestyle. Increasing evidence suggests that this genetic susceptibility is in part modifiable via epigenetic processes which are dynamic and affected by developmental environmental factors.

c. High risk groups of the population include those who are not being targeted in traditional health care delivery (for example indigenous populations), and whose exposure to detrimental social and environmental influences is greater (for example lower socio-economic groups, migrants and many adolescents); special efforts are required to identify and reach these groups with interventions.

d. Emphasis should be placed on the concept of balance for many markers of risk such as macro- and micronutrient compositions of the diet, body composition or weight gain, because risk is graded across the entire population, being greatest at the extremes of inadequacy and excess. Thus the association of many risk factors with obesity shows a U-shaped distribution. There is a need to define the optimal range of different factors, using available evidence or by additional research.

e. There is a need to distinguish between causal vs. associated risk factors, although there are limited data on many of the former. It will be important to identify risk factors that are likely to lie on the causal pathway, whilst recognizing that we cannot wait until the evidence has accumulated before intervening to reduce some factors which contribute substantially to risk even if they are currently believed to be associated rather than causal.

f. Food marketing is known to be a driver of childhood obesity. The problem arises because food processing increases the market value of foods (e.g. potato chips sell for much more than potatoes), but typically involves adding fat, salt and/or sugar which reduces nutritional value. Marketing budgets, therefore, greatly favour unhealthy options. This marketing affects children directly (pre-schoolers, for example, have been shown to have a strong sense of brand loyalty) and can also work indirectly through parents being encouraged to buy specific foods for their children – whether these be infant formula options, complementary foods or processed foods as a whole. In both audiences marketing can also create false norms and cause confusion about healthy dietary decision making.
g. Health literacy may be a key to being able to intervene in these different risk factors early, including pre-pregnancy. However, knowledge does not necessarily change behaviour. Strategies such as broader environmental change (numerous aspects, working at family, school, local community, broader food and movement behaviour environment etc.) social marketing and improvements in the health service are all important enablers and barriers to being able to put this knowledge into practice.

h. Ideally, interventions will be integrated and address multiple risk factors.

31. The working group identified that there is a need to review research on the effects of food marketing on pre-school children. This would cover the promotion in three sectors: infant formula, complementary foods and processed foods more generally. The first two would comprise research with caregivers, but the third would also take in studies with the children themselves.

Modifiable risk factors for childhood obesity

32. The discussion of mechanisms leads directly to a discussion of which pathways, and where along those pathways, modifications are possible that would change the course of risk to follow. At the population level, the important developmental factors which influence risk of childhood obesity operate in all individuals but across a wide range. The risk is greatest at the extremes of exposure to such factors, leading to a U-shaped distribution when markers of exposure are plotted against outcome. This is seen for example in such plots for birthweight (as a measure prenatal nutrition), maternal body composition, gestational weight gain or folate status against a range of health outcomes in the child. This distribution leads to the concept that interventions should be aimed at optimising the health of the population as a whole by shifting the levels of factors which prime the development of the next generation towards the median. This concept, established for public health policy by Geoffrey Rose (Rose, 1985) has not been applied to the developmental origins of childhood obesity. Additional risk factors which are known to disrupt human development, such as parental smoking, toxic and endocrine disruptor chemical exposure or some infections, do not show such a U-shaped distribution and ideally these should be eliminated from the developmental environment. In addition, there is a close relationship between maternal glucose during pregnancy and excess adiposity at birth, across the entire range of maternal glucose levels, highlighting the impact of maternal hyperglycaemia on obesity risk in the offspring.

33. The following Tables 1-3 outline the biological, behavioural and contextual risk factors for each important life stage. These are not listed in order of importance and the list may be incomplete, requiring further consideration by the working group.
Table 1: Modifiable risk factors in the pre-conceptional period

<table>
<thead>
<tr>
<th>Biological - genotype to phenotype and causal or correlated</th>
<th>Behavioural (*may also be biological rather than solely learned)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal obesity</td>
<td>Parental behaviours, including smoking, alcohol use, low physical activity, sedentary behavior</td>
</tr>
<tr>
<td>Paternal obesity</td>
<td>Dietary patterns</td>
</tr>
<tr>
<td>Maternal nutritional status (amount and balance of macro-, micronutrients eg. balanced B12 and folate)</td>
<td>Overuse of nutritional supplements (eg folic acid)</td>
</tr>
<tr>
<td>Maternal body composition</td>
<td></td>
</tr>
<tr>
<td>Use of assisted reproductive technologies</td>
<td></td>
</tr>
<tr>
<td>Maternal glycaemia</td>
<td></td>
</tr>
<tr>
<td>Maternal lipid metabolism</td>
<td></td>
</tr>
<tr>
<td>Young age or physical immaturity (adolescent pregnancy)</td>
<td></td>
</tr>
<tr>
<td>Short maternal stature</td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Modifiable risk factors during gestation

<table>
<thead>
<tr>
<th>Biological - genotype to phenotype and causal or correlated</th>
<th>Behavioural (*may also be biological rather than solely learned)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal gestational weight gain</td>
<td>Maternal behaviours as above</td>
</tr>
<tr>
<td>Maternal diet (micro- and macronutrients)</td>
<td>Stress</td>
</tr>
<tr>
<td>Maternal body composition: fat and skeletal muscle mass</td>
<td>Depression</td>
</tr>
<tr>
<td>Maternal workload</td>
<td></td>
</tr>
<tr>
<td>Maternal stress</td>
<td></td>
</tr>
<tr>
<td>Maternal glycemia, and gestational diabetes</td>
<td></td>
</tr>
<tr>
<td>Pre-eclampsia</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
</tr>
<tr>
<td>Glucocorticoid or other drug treatment</td>
<td></td>
</tr>
<tr>
<td>Pre-existing maternal or paternal diabetes</td>
<td></td>
</tr>
<tr>
<td>Multiple conception</td>
<td></td>
</tr>
<tr>
<td>Parity</td>
<td></td>
</tr>
<tr>
<td>Infection and inflammation: STIs, malaria, HIV</td>
<td></td>
</tr>
</tbody>
</table>
Table 3: Modifiable risk factors in infancy

<table>
<thead>
<tr>
<th>Biological - genotype to phenotype and causal or correlated</th>
<th>Behavioural (*may also be biological rather than solely learned)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational age/degree of prematurity</td>
<td>Age-specific dietary patterns</td>
</tr>
<tr>
<td>Birthweight</td>
<td>Caregiver’s infant feeding behaviours and dietary quality</td>
</tr>
<tr>
<td>Anthropometry and body composition</td>
<td>(breast feeding, timing of introduction of complementary foods,</td>
</tr>
<tr>
<td>Genetic and epigenetic factors</td>
<td>caregiver responsive feeding)</td>
</tr>
<tr>
<td>Hormone levels and growth factors (IGF, C-Peptide)</td>
<td>Food preference</td>
</tr>
<tr>
<td>Linear growth rate</td>
<td>Nurturing behaviour</td>
</tr>
<tr>
<td>Breast/formula feeding</td>
<td>Stimulation</td>
</tr>
<tr>
<td>Taste development</td>
<td>Activity</td>
</tr>
<tr>
<td>Appetite control/satiety set point</td>
<td>Sleep patterns</td>
</tr>
<tr>
<td>Metabolic set point</td>
<td>Child care</td>
</tr>
<tr>
<td>Development of gut microbiome</td>
<td>Level of physical activity</td>
</tr>
<tr>
<td>Infection</td>
<td></td>
</tr>
</tbody>
</table>

Modifiable risk factors after infancy

34. Consideration of modifiable risk factors after infancy begins with priming by risk from earlier periods along with persistence and evolution of these carry over risk factors, and new risks that are specific to the subsequent developmental periods. It is therefore important to re-assess the nature, levels, and balance of these risks by developmental stage and the biological, behavioural, familial and environmental exposures that are characteristics of those stages. In general, pathways that lead to eating behaviours associated with excess intake or energy in relation to need or patterns of activity and sedentary behaviour that result in insufficient energy output are of interest. Some examples of potentially modifiable risk factors that have been established in certain population or that are potentially relevant on a theoretical basis are shown in Table 4 along with examples of familial, sociocultural and environmental influences that may be important targets or considerations for efforts to enable changes at the individual level. These contextual factors may influence exposure to risk factors, sensitivity to risk factors, or the response to interventions.
Table 4: Modifiable risk factors in childhood

<table>
<thead>
<tr>
<th>Behavioural</th>
<th>Familial and Social</th>
<th>Broader Environmental Context</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Frequent consumption of sugary beverages</td>
<td>• Parenting practices</td>
<td>• Upstream food supply factors (e.g. agriculture and trade)</td>
</tr>
<tr>
<td>• Low intake of fruits and vegetables</td>
<td>• Parental role models</td>
<td>• Food supply – food composition, availability, access, affordability</td>
</tr>
<tr>
<td>• Frequent consumption of calorically-dense snacks</td>
<td>• Gender roles</td>
<td>• Marketing of high calorie, low nutrition packaged foods to children and parents – intensity and forms</td>
</tr>
<tr>
<td>• Eating while watching TV</td>
<td>• Peer influences</td>
<td>• Built environments related to physical activity</td>
</tr>
<tr>
<td>• High frequency/long duration of TV watching (screen time)</td>
<td>• Social norms about eating and physical activity or inactivity</td>
<td>• Health systems (e.g. health professional training and service delivery in antenatal, postnatal, infant and child/adolescent health care)</td>
</tr>
<tr>
<td>• Eating in restaurants</td>
<td>• Food traditions and preparation practices</td>
<td>• “Street food” vending and its economic role</td>
</tr>
<tr>
<td>• High use of digital devices</td>
<td>• Food provision in schools and child care settings</td>
<td></td>
</tr>
<tr>
<td>• Eating before bed</td>
<td>• Limited income</td>
<td></td>
</tr>
<tr>
<td>• Insufficient outdoor play</td>
<td>• Physical activity provision in schools and child care settings</td>
<td></td>
</tr>
<tr>
<td>• Long periods of uninterrupted sitting</td>
<td>• Limited education</td>
<td></td>
</tr>
<tr>
<td>• Poor sleep quality and quantity</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Interventions to prevent or reduce childhood or adolescent obesity

35. The culminating question addressed by the Working Group was: what can be done to prevent childhood obesity or mitigate its persistence into later life? That is, what are the policy options for prevention of childhood obesity, what is the evidence of their effectiveness, and in what combination should those viewed as potentially usefully applied in different contexts? Childhood was taken to refer to birth to 18 years of age. Obesity prevention refers to ways to influence behaviours related to eating and movement, including physical activity, sedentary behaviour and sleep and the provision or facilitation, by caregivers as well as health professionals of these behaviours in children in a direction that is favourable to an appropriate pattern of weight gain, healthy development and growth and the prevention of adverse health consequences during and after childhood.

36. A particular emphasis should be placed on identifying early life interventions beginning in the pre-conceptional period. From a theoretical perspective and by reference to the benefits of early life interventions in other health spheres, interventions during this period will set the foundation for obesity prevention.
37. Given the plethora of ‘risk factors’ in the literature, there is a need to distinguish between ‘causal’ and ‘only associative’ factors, so that the chances of success of intervention are maximised. Considerations of effect size and attributable risk may allow prioritisation of interventions.

38. Individuals ultimately control what they eat and how active they are, but these behaviours are shaped by biological, physical, economic and social environments, including early life effects on appetite, metabolism and body composition. Moreover, many of these behaviours are established in early life, when individuals cannot make conscious choices or reflect a mixture of conscious and unconscious choices in subsequent years. Thus interventions should also support population-level approaches to promote healthier behaviours, including through engaging actors in sectors outside the health system which play key roles in shaping the physical, economic and social environments that drive individual behaviours related to later risks.

39. The group noted the following guiding principles for interventions:

- A life-course approach is essential, and was endorsed by the UN Political Declaration of the High-level Meeting of the General Assembly on the Prevention and Control of Non-communicable Diseases (document A/66/L.1).
- Early life interventions, beginning with the pre-conceptional period, are of particular importance since susceptibility to later environmental effects is in part established in utero, and plasticity is greater at younger ages.
- Since risks accumulate from the prenatal period throughout childhood and adolescence, the range of interventions must target all developmental stages. Given the futility of isolated interventions, continued and reinforcing interventions at each key developmental stage are essential.
- Obesity is a complex, multifactorial problem; thus interventions must be developed to reduce risk at multiple levels: individual/family, community, the broader food and physical activity environments.
- In developing and prioritizing interventions, we should build on what is known from other child health initiatives, such as for maternal nutrition. Research in maternal and child health and early childhood development has contributed knowledge on how to promote healthy growth and development: we need to build on this with the outcome of child obesity also in mind. It will be useful to identify well-established strategies for addressing child nutrition in general that also have implications for child obesity. These may include multiple micronutrient and macronutrient balance, breastfeeding promotion and improving the quality of weaning foods.
f. Research on prevention of child obesity in preschool- and school-age children needs to be considered with special attention to the contexts of low and middle income countries. Interventions should be in accord with fundamental rights instruments, not least the UN Convention on the Rights of the Child to promote safe and healthy facilities, standards of nutrition, mass media, health care, play and recreation. For all interventions, the best interest of the child shall be a primary consideration.¹

g. While interventions require a solid evidence base, we cannot wait to act: understanding from knowledge of basic biology and well-conducted observational studies and parallel studies of other health issues may be of particular importance in development of novel strategies.

40. Relevant highlights of the Working Group deliberations in relation to each of these areas follow.

Macroeconomic and contextual factors that promote obesity:

41. The childhood obesity epidemic is still relatively new, and the key drivers have been in the macro-environment which has become obesogenic. Specifically energy dense food has become ubiquitous and sedentary living much more prevalent driven in part by rapid urbanisation and globalisation. What has not changed is the aspiration of parents, educators, health providers and policy makers to enable children to flourish. Interventions should concentrate on a) making the environment healthier and b) supporting these key actors – especially parents – to achieve their aspirations. Moreover the focus should always be on prioritising the needs, empowerment and potential of children themselves.

42. There have been many efforts to identify effective and cost-effective environmental interventions to promote healthier eating, increase activity and reduce childhood obesity. Table 5 below presents the most promising approaches. They cut across the life course and enable the key actors – especially parents and children - to make healthful choices.

¹ See UN Convention on the Rights of the Child http://www.ohchr.org/EN/ProfessionalInterest/Pages/CRC.aspx Article 3-1 Best interests of the child; Article 3-3. Facilities that are safe, healthy etc.; Article 17. Mass media; Article 24, d to f. Standard of health; Article 27-3. Nutrition; Article 31-1; Play, recreation;
Table 5: Potential policies approaches and interventions to promote healthy eating

<table>
<thead>
<tr>
<th>Reduce unhelpful influences</th>
<th>Increase helpful influences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proposed Framework Convention on Nutrition</td>
<td>Marketing healthy options</td>
</tr>
<tr>
<td>Look back provisions (eg set targets for reductions in childhood obesity and fine industry if they don’t make it)</td>
<td>Institutional meals standards</td>
</tr>
<tr>
<td>Mandatory Standards:</td>
<td>Social enterprises to support healthy eating</td>
</tr>
<tr>
<td>• Product formulation (eg reduced sugar, salt, fat)</td>
<td>Health and media literacy interventions</td>
</tr>
<tr>
<td>• Pricing (eg price promotions on healthy options)</td>
<td>Social marketing on healthy eating and activity</td>
</tr>
<tr>
<td>• Advertising and promotions</td>
<td>Training of healthcare and childcare professionals</td>
</tr>
<tr>
<td>• Distributions (eg not in schools)</td>
<td></td>
</tr>
</tbody>
</table>

43. Some key areas for further understanding and development include:
   a. More information around the impact of taxes and pricing policies such as subsidies.
   b. Learning from natural experiments in the areas of policy interventions, for example Mexico’s taxes on unhealthy foods.
   c. Potential interventions around the increased availability of processed foods.
   d. The use of information and education campaigns (known to increase knowledge and consumption of healthy food) in order to support positive parenting, as well as the training of health professionals.
   e. The role fundamental rights can play in helping prevent child obesity.

**Early Life Nutrition and Health Interventions:**

44. There is emerging evidence that the pre-conceptional health status and wellbeing of the woman and her partner have important and long-lasting influences on the predisposition of her future child to obesity. Key issues for early life interventions relate to how to take fuller advantage of new scientific advances in the understanding of the aetiology and vulnerability to obesity to develop new and better apply available interventions. Points made in the discussion were as follows.
a. Interventions aimed at optimizing pre-conceptional health can impact childhood obesity, for example prevention of under- as well as over- nutrition and encouragement of healthy movement behaviours. Some interventions can be effective just before conception (e.g. glycaemic control) while others will require intervention earlier in the woman’s life cycle (e.g. reduction of stunting).

b. In view of the U-shaped distribution of risk, interventions should be targeted at optimising factors such as maternal BMI, micro- and macro-nutrient status. Interventions should also aim to eliminate other factors for which there is no optimal range, e.g. infection, toxic exposure, smoking and second hand smoke exposure (especially in relation to women in some developing countries).

c. Interventions and policy instruments, including the International Code on Marketing of breastmilk supplements and guidance for the composition of complementary foods are among the strongest potential influences to optimize nutrition in early childhood and are relevant to both undernutrition and obesity.

d. Policies to decrease cigarette use are also relevant to early life interventions given the association of maternal smoking with poorer infant health.

e. Interventions could be achieved through education, improving health literacy, and other types of health promotion among adolescent girls and boys, in addition to promoting access to health care services.

f. Women and girls in more disadvantaged social groups are at particular risk and may be less likely to access health care and be exposed to interventions. Particular attention needs to be paid to such groups.

Interventions later in childhood to reduce childhood obesity:

45. Interventions have been developed based on settings for access, e.g. homes, schools and child care settings, community settings other than schools, and whole communities. Many have been shown to be effective in modifying eating or physical activity or sedentary behaviours, although showing effects on BMI is more difficult. Whereas the key issues for interventions for children under age two years relate to how to take fuller advantage of new scientific advances in the understanding of the aetiology and vulnerability to obesity, issues for children aged two years and over relate more to how to further refine design and implementation of the types of interventions that have already been identified. Issues that arose in the discussion were as follows:

a. There is a strong evidence base for integrated interventions within schools (curriculum, school foods, physical activity opportunities, family and community engagement, teacher training) influencing food and movement behaviours (Population-based approaches to childhood obesity prevention, WHO 2012).

b. There is also growing evidence that community based interventions to
prevent obesity are able to engage communities, modify some aspects of the food and physical activity environment and so improve some food and movement behaviours.

c. However, to date this evidence is largely from high income countries and further data are needed from low and middle income settings. Whether these interventions modify the critical behaviours in contexts that are different from those where the intervention was generated is often unknown. Very few studies have been done in low and middle income countries.

d. Identifying characteristics of successful interventions and ways to scale and transfer them should be a high priority.

e. The impact of any given intervention will be affected by carry-over of risk or protection from earlier in life; how this affects the design and delivery of interventions is unclear; the issue may be particularly relevant to achieving health equity related to healthy weight.

f. Some interventions will work better on overweight than obesity or in children of certain ages. It will be important to target interventions accordingly for greatest effectiveness.

g. The failure of many apparently well-designed and well-documented interventions is informative in itself in that it provides an opportunity to identify key influences on the ability to intervene effectively. Potential reasons for failure suggested by case studies include insufficient adoption of an intervention; failure to disseminate an intervention; a ‘dose’ of intervention that has transitory rather than durable effects, resulting in attenuation of effects over time in the context of countervening forces; or an intervention that was incomplete in the sense of addressing the minimum set of relevant drivers.

h. Accepting that no single intervention is likely to be effective at reducing childhood obesity, any menu of potential interventions should identify those most likely to be complementary and mutually reinforcing. Evaluation of the effects of interventions should follow the same principle.

i. Regardless of other considerations, having a multisectoral national or regional level plan with clear commitments and lines of accountability is critical to addressing issues across the life course; annual reporting of results of surveillance in the form of report cards was discussed as an incentive to action.

j. Related to the above, the likelihood of action at either government or individual levels in the absence of adequate resources was a consideration. Ways to generate funding for new initiatives or to link incentives to mandates
should be identified.

k. Identifying key developmental transition points or opportunities for universal access might increase the efficiency of interventions.

l. In addition, the following issues to consider in the treatment of child and adolescent obesity were highlighted
   i. The increased prevalence of child and adolescent obesity highlights the need for effective treatment of affected people, as well as primary prevention.
   ii. Systematic reviews of treatment of child and adolescent obesity show that behavioural lifestyle interventions can lead to positive outcomes in weight, BMI, other measures of body fatness and a range of cardio-metabolic risk markers. Severe obesity, especially in adolescence, may require additional forms of therapy.
   iii. In high income countries, overweight and obese children and adolescents present more frequently to primary, secondary and tertiary health care services than might be expected from the background prevalence of this condition, although they rarely present primarily for the problem of obesity. Unfortunately, they are unlikely to have this problem specifically addressed by the clinician. There are no data on health service presentations by obese children in LMICs.
   iv. Given the high prevalence of paediatric obesity in most westernized and rapidly westernizing countries, and its chronicity, there is a need for coordinated models of care for health service delivery. No country has yet established a cost-effective comprehensive model of care for obesity management. Different models of service delivery may be required for different countries and may require training and upskilling of a range of health professionals.

**RECOMMENDATIONS**

46. While there are several knowledge gaps in terms of the epidemiology, mechanisms and effectiveness of interventions to reduce childhood obesity, there are potential interventions which can be integrated into maternal, reproductive and child health programmes. These can further inform and complement efforts to address the research gaps identified.

47. **Epidemiology:**
   a. Further focusing and refining of epidemiological data collected could identify the optimal biological, behavioural and environmental conditions for the development of obesity in children. The tracking of patterns of obesity and the
associated behaviours across and within countries and regions, noting in particular the age, gender, socioeconomic, cultural, ethnic and urban vs. rural demographics would inform the development of potential interventions.

b. The relevant risk factors and pathways for obesity in children, hitherto primarily developed for high income countries, need to be investigated for low and middle income countries.

c. The effect sizes and attributable risk associated with these factors need to be established.

d. Data on the incidence and age at onset of obesity, plus factors associated with reverse transition from obesity to overweight, or overweight to normal weight, can further inform our understanding of childhood obesity.

48. Economic impact:

a. Early intervention, when there is greater opportunity to modify both biology and behaviour due to plasticity, reversibility and conditioning, has the potential to have greater economic impact than interventions later in the life course. The efficiency of interventions in both high and low and middle income countries requires further study.

b. The economic costs of NCDs later in life are significant and are borne by individuals, families and society. There are the direct costs to the health care system, indirect costs in terms of reduced productivity and educational attainment, and losses to individuals and families in well-being and life expectancy. The economic impact of childhood obesity requires further investigation, particularly for low-and-middle-income countries.

c. Investments to promote healthy behaviours can have significant health, social and economic benefits, which should be estimated to support policy development.

d. The contribution of health issues such as childhood obesity to the wealth of nations has been insufficiently emphasised. A more inclusive approach would capture the value of health and of preventing childhood obesity in a broader societal way compared to what has been recorded by cost-of-illness studies so far.

e. Obesity begets obesity and there are trans-generational effects of poor development; as such, childhood obesity cannot be ignored.
f. Obesity can exacerbate inequalities in health, both within and between countries.

49. Interventions:
   a. Widespread obesity in populations is preventable. Whilst recognising that obesity has heritable components, obesity development encompasses a combination of genetic, epigenetic, behavioural and environmental risk factors and responds to changes in these factors. The appreciation of the potential for behavioural and environmental factors to influence obesity levels is demonstrated by the recent marked increases in obesity around the world. These trends are reversible.
   
   b. Childhood obesity is a complex, multifactorial condition and interventions are required at multiple levels: these include individual, family, community and the broader food and movement behaviour environments.
   
   c. Interventions should adopt a life-course approach. Whilst risks accumulate over time, the responses to them are influenced by early developmental processes.
   
   d. Early life interventions, commencing before conception, take greatest advantage of the potential to use developmental plasticity to prevent or reverse inadequate responses to later risks such as the obesogenic environment.
   
   e. Interventions should target all key developmental stages and be continuous and reinforcing, rather than isolated.
   
   f. There is currently a paucity of data from low and middle income countries to guide the development of interventions to tackle childhood obesity.
   
   g. In working to prevent childhood obesity, data are also required on the health service usage by obese children and adolescents in high, middle and low income countries and the models of health service delivery and training needs of health professionals in different settings, countries and regions.

50. Monitoring and surveillance:
   a. The working group was not able to consider fully monitoring and surveillance during this first meeting and would request that monitoring and surveillance be considered at a subsequent meeting. In addition, the working group recommends the establishment of a working group to consider implementation, monitoring, and accountability, to support the work of the Commission.
CONCLUSIONS, ACTION POINTS AND NEXT STEPS

The *ad hoc* Working Group on Science and Evidence has identified a number of research gaps and areas requiring further study before being able to submit a full report to the Commission on Ending Childhood Obesity. A model is under development to consider the health and economic impact of childhood obesity (see Appendix 1).

The Working Group will seek to address these knowledge gaps and may request further support from the Secretariat, the Director-General and the Commission to permit this within the time frame of the Commission.
## ANNEX 1: Members of the ad hoc Working Group on Science and Evidence

### List of Members

<table>
<thead>
<tr>
<th>Name</th>
<th>Position/Institution</th>
<th>Contact Information</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Professor Linda S. Adair</strong></td>
<td>Professor, Department of Nutrition, University of North Carolina</td>
<td>Tel: +1 919 962-6154, Fax: +1 919 966 9159, Email: <a href="mailto:Linda_adair@unc.edu">Linda_adair@unc.edu</a></td>
</tr>
<tr>
<td><strong>Dr Narendra Kumar Arora</strong></td>
<td>Executive Director, The INCLEN Trust International &amp; CHNRI</td>
<td>Tel: +91 11 47730000, Fax: +91 11 47730001, Email: <a href="mailto:nkarora@inclentrust.org">nkarora@inclentrust.org</a>, <a href="mailto:nkmanan@yahoo.com">nkmanan@yahoo.com</a></td>
</tr>
<tr>
<td><strong>Dr Fereidoun Azizi</strong></td>
<td>Director, Endocrine Research Center, Shahid Beheshti University of Medical Sciences (SBUMS), Professor of Internal Medicine, Endocrinology and Metabolism, Taleghani Medical Center, Shaheed Beheshti University of Medical Sciences, Tehran, Iran</td>
<td>Tel: +98(21) 22409309, Fax: +98(21) 22402463, Email: <a href="mailto:Azizi@endocrine.ac.ir">Azizi@endocrine.ac.ir</a></td>
</tr>
<tr>
<td><strong>Professor Louise Baur</strong></td>
<td>Discipline of Paediatrics &amp; Child Health, Co-Director, Physical Activity, Nutrition &amp; Obesity Research Group, Sydney School of Public Health, Paediatrics &amp; Child Health, C29 - Children's Hospital Westmead, The University of Sydney, NSW 2006, Australia</td>
<td>Tel: +61 2 9845 3382, Fax: +61 2 9845 3389, Email: <a href="mailto:louise.baur@health.nsw.gov.au">louise.baur@health.nsw.gov.au</a></td>
</tr>
</tbody>
</table>
Professor Zulfiqar A. Bhutta *
Department of Paediatrics and Child Health
Aga Khan University Hospital, Karachi
SDtyadium Road, P.O. Box 3500
Karachi 74800
Pakistan

Tel: +1 461-813-7654 ext. 328532
Fax: +1 416-813-8763 ext. 208763
Email: zulfiqar.bhutta@sickkids.ca

Dr Frank J. Chaloupka
Professor of Economics
Institute for Health Research and Policy
University of Illinois at Chicago (MC 275)
444 Westside Research Office Bldg.
1747 West Roosevelt Road
Chicago, IL 60608
USA

Tel: +1 312 413 2367
Fax: +1 312 996 2703
Email: fjc@uic.edu

Professor Partha Dasgupta
Emeritus Professor, Frank Ramsey Professor
Emeritus of Economics
Faculty of Economics, University of Cambridge
United Kingdom

Tel: +1 647 346-9158
Fax: +1 416.813.8763
Email: Partha.Dasgupta@econ.cam.ac.uk

Dr Anniza de Villiers
South African Medical Research Council
Francie van Zijl Drive
Parowvallei, Cape
PO Box 19070, 7505 Tygerberg
South African

Tel: +27 21 938 0242
Fax: +27 21 933 5519
Email: anniza.de.villiers@mrc.ac.za

Professor Terrence Forrester
Chief Scientist
UWI Solutions for Developing Countries
The University of the West Indies
25 West Road
Mona Campus, Kingston 7
Jamaica

Tel: 876 977-6803
Fax: 876 970 2682
Email: Terrence.Forrester@uwimona.edu.jm
Professor Amandine Garde *
The Liverpool Law School
Law Building
Chatham Street
Liverpool, L69 7WW
United Kingdom

Professor Mark Hanson
British Heart Foundation Professor
Director, Institute of Developmental Sciences
Academic Unit of Human Development and Health
University of Southampton
Medicine, Southampton General Hospital
Mailpoint 887, South Academic Block
Tremona Road, Southampton, SO16 6YD
United Kingdom

Professor Gerard Hastings
Director
Institute for Social Marketing and Centre for Tobacco Control Research
University of Stirling and the Open University
Stirling FK9 4LA
United Kingdom

Professor Shiriki Kumanyika
Co-chair, International Obesity Task Force of the International Association for the Study of Obesity
Professor of Epidemiology
Department of Biostatistics and Epidemiology
Perelman School of Medicine
University of Pennsylvania
8th Floor Blockley Hall
Philadelphia PA 19104-6021
USA

Professor Ronald Ching-Wan Ma
Department of Medicine and Therapeutics
The Chinese University of Hong Kong
Prince of Wales Hospital, Shatin
Hong Kong SAR
China
Professor Carlos A. Monteiro *
Department of Nutrition
School of Public Health
University of Sao Paulo
Av. Dr. Arnaldo 715
Sao Paulo 01246-907
Brazil
Tel: +55-11-3061-7854/01/05
Fax:
Email: carlosam@usp.br

Professor John Reilly
University of Strathclyde
Physical Activity for Health
Course Support Team
Psychological Sciences and Health
Room 670 Graham Hills Building
40 George Street, Glasgow, G1 1QE
United Kingdom
Tel: +44 141 548 4235
Fax:
Email: john.j.reilly@strath.ac.uk

Dr Rachel Rodin
Scientific Director, WHO Collaborating Centre on Noncommunicable Disease Policy
Senior Medical Advisor, Centre for Chronic Disease Prevention
Public Health Agency of Canada
130 Colonnade Road, A.L. 6501H
Ottawa, Ontario K1A 0K9
Canada
Tel: +1 613-954-8651
Fax: +1 613-941-9502
Email: Rachel.Rodin@phac-aspc.gc.ca

Dr Mark Tremblay
Director, Healthy Active Living and Obesity Research (HALO)
Scientist and Professor, Department of Pediatrics
University of Ottawa
CHEO Research Institute
401 Smyth Road
Ottawa, ON
K1H 8L1
Canada
Tel: + 1 613 737 7600 x 4114
Fax: +1 613 738 4800
Email: mtremblay@cheo.on.ca

Report of the first meeting of the Ad hoc Working Group on Science and Evidence
Professor Wenjuan Wang
Director
Unit of Obesity and Metabolic Diseases Prevention and Control
National Center of Noncommunicable Diseases Prevention and Control (NCNCD)
Chinese Center for Disease Control and Prevention (China CDC)
Nanwei road 27, Dongcheng District
Beijing, 100050
China

Tel: +8610-63026630
Fax: +8610-63042350
Email: wwj63131779@126.com

Professor Chittaranjan S Yajnik
King Edward Memorial Hospital Diabetes Unit
Sardar Moodliar Road
Pune 411011
India

*Unable to attend

Tel:
Fax:
Email: diabetes@vsnl.com
csyajnik@hotmail.com
Day 1:  
18th June 2014

Opening Session

Welcoming remarks by Dr Timothy Armstrong, on behalf of the ADG of the Non-communicable Diseases and Mental Health cluster and Family and Women’s Health cluster, who were unfortunately unable to attend.

Meeting participants introduced themselves and summarised their Declaration of Interests. The meeting Chairs, Prof. Mark Hanson and Prof. Shiriki Kumanyika were elected and the proposed programme of work accepted.

In addition, the attendance of observers, in particular Prof. Sir Peter Gluckman, co-Chair of the Commission was approved.

Dr Timothy Armstrong then gave a brief update on the establishment of the WHO Commission on Ending Childhood Obesity (ECHO) by the Director-General. This high level commission on Childhood Obesity has been convened, not only in recognition of the huge problem that exists and the economic impact of later consequences of noncommunicable diseases, but also the social, educational and developmental impact of obesity during childhood. In addition, this Commission will work across the organisation and engage with non-state actors, including industry and civil society.

The Commission will be supported by two ad hoc Working Group, this group on Science and Evidence and, if suggested by this group and accepted by the Commission, an ad hoc Working Group on Implementation, Monitoring and Accountability, which might meet in September.
A brief outline was given of the following areas, based on the background paper, and this was followed by discussion in plenary.

1. Epidemiology of infant and young child obesity

During discussions the issue of using the term obesity, which can be a loaded term, but may capture the imagination of some, but be seen as irrelevant by other member states, was raised. The WHO mandated targets for the reduction of obesity and it was felt this would be an appropriate terminology to continue using.

The importance of recognising the dual burden of under- and over-nutrition experienced by some countries was highlighted.

The issues with using BMI as a measure of obesity in children and adolescents were raised. This is a conservative measure, adjusted for age and gender in children and complicated by the fast growth velocity in children and adolescents. As such it was suggested that in the report some examples be given of actual weights of children falling into various categories at different ages. It was felt that more data are needed on the body composition of children in relation to BMI, especially as over time greater adiposity is observed for any given BMI in the general population.

2. Health and economic impact of childhood obesity

During discussions the direct and indirect cost of childhood obesity were discussed. There are more data available from high income countries on the direct health care costs of childhood obesity in both childhood and when continued obesity in into adulthood results in NCD. This data is lacking for low and middle income countries and would should to be sought.

The issue of who bears the cost of obesity, whether families, employers or the state was raised. The involvement of the private sector in these calculations was suggested. The lack of free health care for children over 5 in many countries highlights this as a particularly vulnerable population group.

The trans-generational costs of obesity should be explored.

Following this, two groups were formed to draft recommendations and research gaps.
Drafting groups feedback

a. Epidemiology of infant and young child obesity

The group rapporteur, Prof. Shiriki Kumanyika, presented the bullet points developed during the drafting group work, which form the basis of the discussions and will be further expanded to provide recommendations for consideration by the Director-General. In addition, the group highlighted knowledge gaps which they suggest should be addressed, in order to further inform the report to the Commission.

b. Health and economic impact of childhood obesity

The group rapporteur, Dr Rachel Rodin, outlined the discussion points of the drafting group and the areas requiring further deliberation and potential research considerations.

Day 2:
19th June 2014

Brief outline and discussion

A brief outline was given of the following area, based on the background paper, and this was followed by discussion in plenary.

3. Mechanisms of childhood obesity

Issues discussed included the need for interventions to target particular points of modifiable risk, the influence of maternal nutrition on body size, composition and adiposity and the potential importance of measuring other markers, not just birth weight, as there may be differences in endocrine priming. The association between GDM and low vitamin B12 status was mentioned, including the exaggerated risk with high folate. The U shaped curve of risk and the importance of finding the optimal balance. In addition, the maternal programming of a girl’s ova in utero means that early interventions have the potential to influence 3 generations.

The developmental influences on appetite, including birthweight, indicate the potential for modification through breastfeeding and complementary feeding practices. Physical activity appears to also be influenced by birthweight.

The issues relating to the microbiome and the influence of this on digestion and metabolism.
Drafting groups  
c. Modifiable risk factors for childhood obesity: pre-conception, *in utero* and early life  
d. Modifiable risk factors for childhood obesity: age 2+ (childhood and adolescence)  

The drafting group rapporteurs presented back the deliberations of the drafting groups.

The cut-off age of 2 years was discussed. This is in line with standard WHO cut-off ages relating to breastfeeding and complementary feeding practices, and as such would be a suitable cut-off age for consideration. However, it was recognised that many of the marketing challenges faced by children are now encountered by very young children and as such there may be overlap in terms of interventions in these age groups.

Brief outline and discussion  
A brief outline was given on the potential interventions, based on the background paper, and this was followed by discussion in plenary.

4. Evidence-based interventions to prevent childhood obesity  
The following issues were raised during discussions included the importance of treating children already affected by obesity or overweight and secondary and tertiary interventions including health systems strengthening.

Drafting groups  
e. Interventions for childhood obesity: pre-conception, *in utero* and early life  
f. Interventions for childhood obesity: age 2+ (childhood and adolescence)  

The drafting group rapporteurs presented the deliberations of the drafting groups.

Day 3:  
20th June 2014  
The Zero Draft of the meeting report was presented and discussed. Drafting groups were assigned to provide additional text for each section and potential figures were discussed.

It was agreed that the first draft of the meeting report would be circulated by the middle of next week, allowing meeting participants and those members of the working group unable to attend the first meeting to comment, prior to submission to the WHO Director-General.

Closing remarks  
Dr Timothy Armstrong thanked the working group for their contributions to the process.
APPENDIX 1: Health economics model

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

Appendix

Dynamics of Personal Health Status: A Formalisation

Revised: 25 June 2014

[Note: The formal model presented here captures the dynamics of an individual's health status. Such a model is useful not only because it represents the ideas expressed by our Group in a quantitative manner, but also because it is an essential tool for estimating the relative importance of the factors contributing to a person's health status.]

1 Introduction

It is a truism that a person's health status is multi-dimensional. That means it is a vector of her attributes. The person's health status is in turn one component, albeit a vitally important component, of the human capital that is embodied in her. The remaining components are her disposition (more generally her psychological make-up), her cognitive abilities, her level of education, and so on. The totality of components jointly contributes to the person's ability to be and do. That ability is circumscribed by prices and incomes, social obligations and norms of behaviour, and all the other constraints that limit her ability to be the kind of person she would like to be and to do the various things she would like to do.

The individual is embedded in a socio-ecological environment. There are other people, in addition to her, who influence that environment. Although in this Appendix we focus on the dynamics of an individual's health status, we should remember that she is one among many. There is a corresponding health dynamics associated with each of the other individuals who together shape the environment. We do not produce the integrated model here. To do so would introduce additional notation, but it would be otherwise un-illuminating. We provide hints on how the integrated model would be constructed (Note-5). And we draw attention to the effects of others on the dynamics of the health of the person we study here (Note-6 on epidemics).²

² Because a single individual is under scrutiny here, we do not have to identify her with a label.
Although the individual's health status is a *vector* of her attributes, even epidemiologists are obliged to simplify if they are to make progress. Therefore, with all the caveats that are routinely made by experts, we assume that that those multiple attributes can be aggregated into a *scalar* (real number), which we label as $H$. We could, if we chose, interpret $H$ as BMI.

Health is a *capital asset*; and a person's health status $H$ is a component of her human capital. In order to compare the significance of an economy's various capital assets with one another, they have to be expressed in a common currency. That common currency is typically monetary, say, dollars. But the currency could have been any chosen commodity, or a basket of commodities, for example, a basket of consumption goods. *Health capital is health status expressed in that common currency.*

When economists estimate someone's wealth, they include her human capital, but unfortunately restrict the idea of human capital to education (e.g. years of schooling, expressed in dollars). This is a lacuna that should be corrected by macro-economists. Below we expand on the idea of health capital as a component of an economy's wealth and identify the sense in which sustainable development should mean an expansion of an economy's wealth per capita. But it is as well to note here that moves to end childhood obesity should be seen as an utterly necessary part of the search for "sustainable economic development". It would be more than just an irony if the Sustainable Development Goals currently being prepared at the United Nations were not to include health goals.

2 The Life Course Model: The deterministic case

Time is discrete and denoted by $t$. We want to forecast the person's *life course*. But to do that one must start somewhere. The starting date is to a large extent a matter of choice; it depends on the purpose behind the exercise. Here we interpret $t = 0$ to be the person's date of birth. Her health status at birth is therefore her inheritance. That inheritance will have been shaped by her experiences in the womb, by her mother's experiences even before the person was conceived, and so on - up the generations. (Those influences are in all probability more attenuated the farther back we trace the person's history.) We write the person's health status at $t \geq 0$ as $H(t)$. So $H(0)$ is her inheritance. Our Report elaborates on the significance for $H(0)$ of her life in the womb.³

The person's life course unfolds recursively, period by period. Starting at any arbitrary

³ An alternative move would be to interpret $t = 0$ as the moment of conception. But then we would lose the ability to interpret $H$ as BMI. That is why we are not making that move here.
date \( t \) in the life of the person, at which point her health status is \( H(t) \), we want to forecast \( H(t+1) \). We should then interpret the difference between \( H(t+1) \) and \( H(t) \) as the "net improvement" in the person's health status over the period \([t, t+1]\). That net improvement would be a "net worsening" if \( H(t+1) < H(t) \). Equivalently, we would say the person's health status will have improved if \( H(t+1) > H(t) \). A population's health, when aggregated into a scalar index and expressed in terms of a common currency with all other capital assets, is a component of the economy's overall wealth (the other components being the value of the stock of manufactured capital, education, natural capital, and so on). We should therefore conclude that childhood obesity contributes negatively to the wealth of nations. And because sustainable economic development requires that wealth per capita does not decline over time, childhood obesity reduces an economy's ability to pursue a sustainable development path.\(^4\)

How is \( H(t+1) \) related to \( H(t) \)? The discussion held at the first meeting of the Ad Hoc Working Group identified a series of items: (1) food items consumed by the person, (2) the activities she undertook, (3) the facilities to which she has access (e.g., medical care), and (4) the influence on (1)-(3) of the behaviour of others (peer group effects, norms of behaviour, customary obligations to oneself and others, and so forth). That means, for example, that if \( C \) is the vector of goods she consumes, the consumption patterns of others (which we denote by \( C^o \)) are factors influencing \( C \). But the choice of \( C \) is also influenced by her tastes, by prices and her wealth, the information she possesses of commodity characteristics (e.g. that processed food is commonly injurious to health), and so on. We may then denote the functional dependence of \( C \) on those factors by \( C(C^o, Q) \), where \( Q \) is the set of all other factors, including prices and incomes. Psychologists and sociologists have written extensively on the influences of others on one's own behaviour. That literature will prove to be of

---

importance in the design of public policy on childhood obesity.\(^5\)

Some commodities are desired by the person and are good (even essential) for her health if taken in moderation, but is at the margin harmful otherwise. In order to keep the notation tidy, we limit \(C\) to such goods. Consumption and lifestyle activities that are unambiguously bad for health we label by the vector \(A\). Similar activities by others are labelled as \(A^o\). If others influence the person we are studying here, the dependence is expressed as \(A(A^o)\). Government policies, including policies that are targeted at health, are denoted by the vector \(G\). And of course, each of those variables has a time index.

We may now represent the dynamics driving the person's health in the form of a life course (for \(t \geq 0\)) as driven by the recursive form

\[
H(t+1) = H(t) + \alpha(H(t),t)F(C(C^o(t)),G(t)) - \beta(H(t),t)K(A(A^o(t)),G(t)) - \delta(H(t),t).
\]

**Interpretation of equation (1):**

(a) \(F(.,.,.)\) is a function that translates \(C(t), C^o(t),\) and \(G(t)\) into a (scalar) flow. \(F\) contributes to the person's health status at date \(t+1\) and has non-negative values.

Example: In the simplest of nutrition models, \(F\) would be the energy laid down to contribute to next period's BMI.

**Note-1:** Habit formation, including "addiction", can be introduced by creating an index of past consumption, say \(Z(t)\), that influences the desire for current consumption. So we would have \(C(Z(t))\). If we were then to introduce the effect of one's peer group on her consumption and the joint effect on all of advertisement (soft drinks, snacks), thus extending the \(C\)-function to the form, \(C(Z(t),C^o(t))\) and which we comment on in Note-5 and Note-6, the model would allow the analyst to estimate the additional likelihood that an overweight child of age \(t\) will become obese at age \(t+1\).\(^6\)

(b) The contribution of \(F\) to the person's health status at \(t+1\) depends on her health status at \(t\). \(\alpha(H(t),t)\) is a positive (scalar) parameter displaying that dependence. The function is also age dependent (*note the relevance of that for our Group's discussion of plasticity*). It is

---


natural to suppose that $\partial \alpha/\partial H > 0$, which means that the person's ability to translate nutritional intakes into health improves with better health. Our Group discussed the functional form of $\alpha(H(t), t)$ at length. It was noted that there are values of $H$ where the slope of $\alpha$ with respect to $H$ (i.e. $\partial \alpha/\partial H$) changes sharply, implying that the person's health is at risk at those values of $H$ if diet and activities deteriorate even marginally.

(c) $K(\ldots)$ is a function that translates $A(t)$, $A^0(t)$, and $G(t)$ into a (scalar) flow that reduces the person's health status at date $t+1$. It assumes non-negative values. Smoking is a prime example.

Note-2: Habit formation can be introduced by creating an index of past activities that influences the desire for current activities, say $Y(t)$, that influences the desire for certain types of activities. We would then have $A(Y(t))$. If we were then to introduce the effect of one's peer group on her activities and the joint effect on all of advertisement (computer games), which we comment on in Note-5 and Note-6, we would be able to extend the $A$-function to the form, $A(Y(t), A^0(t))$. The extended model would allow the analyst to estimate the additional likelihood that an overweight child of age $t$ will become obese at age $t+1$.

(d) The contribution of $K$ to the person's health status at $t+1$ depends on her health status at $t$. $\beta(H(t), t)$ is a positive (scalar) parameter displaying that dependence. $\beta$ is also age dependent (note the relevance of that for our Group's discussion of plasticity). It is natural to suppose that $\partial \beta/\partial H < 0$, which means that the person's ability to withstand harmful activities improves with better health. Our Group discussed the functional form of $\beta(H(t), t)$ at length. It was noted that there are values of $H$ where the slope of $\beta$ (i.e. $\partial \beta/\partial H$) changes sharply, implying that the person's health is at risk at those values if the levels of those harmful activities increase even marginally.

(e) $\delta(H(t), t)$ is the rate at which the person's health would deteriorate if the factors in the $F$ and $K$ functions assumed those values at which $F = K = 0$. In simple nutrition models $\delta(H(t), t)$ would be the "resting metabolic rate" (dependent as it would be both on the person's BMI and her age).

Note-3: The dynamics embodied in equation (1) has a Markov structure, meaning that at any date $t$ the history of the person up to that date is summarized entirely in $H(t)$. One can question that. Our Group noted, for example, that "birth-weight" (the index the late David Barker used to summarize health status at birth) has shortcomings. But that is not an argument against a Markov structure; rather, it is an argument against using a scalar index to represent health status.
Note-4 In view of the remarks in (b), (d) and (e) concerning the shape of the functions \( \alpha(H(t),t) \) and \( \beta(H(t),t) \), systematic neglect of health in early years cannot easily be compensated for in future years. Thus, if health thresholds are crossed in early life, health status would perforce remain low in later years because of an inability to make up. Steep gradients in the functional forms of \( \alpha, \beta \) and \( \delta \) imply inter-temporal complementarities among the factors making for good health.\(^7\)

3 The Life History Model: The stochastic case

Thus far a deterministic model. But uncertainty is a necessary ingredient in models purporting to forecast a person's life course. Our Group repeatedly referred to "risk factors". We build stochasticity into the life course model by adding a stochastic term, \( \mu^* \), to the right hand side of equation (1). Thus, the equation is modified to read as

\[
H^*(t+1) = H(t) + \alpha(H(t),t)F(C(C^\circ(t)),G(t)) - \beta(H(t),t)K(A(A^\circ(t)),G(t)) - \delta(H(t),t) + \mu^*(H(t),t). \quad (2)
\]

We are to suppose that for every \( H(t) \) and \( t \), the range of values \( \mu^* \) can assume includes both positive and negative numbers. The former should be interpreted as shocks to the person's system that improve her health, while the latter should be interpreted as shocks that lead to a deterioration. The time interval over which equation (2) holds is \([t, t+1]\). In equation (2), \( H(t) \) is known and all the terms of the right hand side, barring \( \mu^* \), are known. Because \( \mu^* \) is not known at \( t \) with certainty, \( H(t+1) \) is not known with certainty. The actual realization of \( H^*(t+1) \) will depend on the realization of \( \mu^* \), which depends on the health status at \( t \) and the person's age (which is \( t \)). Our Group identified health thresholds at which the risk factor increases sharply. The way equation (2) captures that is by recognizing that, say, the mean of \( \mu^* \) declines sharply at certain values of \((H,t)\) pairs. (In addition, it could be that the variance of \( \mu^* \) increases at those pairs).

Note-5 The person we have been studying is one among many in the economy. We may label the representative person by the index \( i \) and number people as \( i = 1, 2, ..., N \), where \( N \) is the size of the population. Person \( i \)'s health status is denoted by \( H_i(t) \). And the economy's health status can then be represented by the \( N \)-tuple of figures, \( \{H_1(t), ..., H_i(t), ..., H_N(t)\} \). This \( N \)-tuple evolves over time as a consequence of the interplay of \( N \) recursive relationships, one

---

7 The most vivid (and extreme) example of "complemen tarities" is a pair of shoes: in the absence of a right (respectively, left) shoe, the value of the left (respectively, right) shoe is nil.

8 It is customary to represent stochastic terms by the "tilde" sign. My software doesn't allow me to transcribe the tilde sign in Word, so I am using the star sign "*" instead.
for each person (equation (2)).

**Note-6 (Epidemics)** Even though diabetes is a non-communicable disease, our Group frequently called sharp increases in the incidence of obesity in populations, "epidemics". This is an entirely justified usage of terms. *Epidemics are positively correlated individual outcomes.* In the case of obesity, the "contagion" in question is not a virus in the usual sense, but mutual influence in behaviour (i.e. the functional dependence of $C$ on $C^0$ and the functional dependence of $A$ on $A^0$ in equations (1) and (2)). Fads and fashions are examples, and they extend to food habits and leisure activities. But for fads and fashions to take off, there needs to be a trigger mechanism. Our Group identified advertisement of cheap and enticing food products as a trigger. It can be, of course, that people shift to a particular consumption item because its price has fallen, not because they are influenced by the consumption behaviour of their peers. Our propensity to be influenced by others' behaviour amplifies the shift.

**4 Ending Childhood Obesity: the normative analysis**

When the person we have been studying is young (before she reaches the "age of reason"), decisions affecting her well-being is made on her behalf. She may choose some of her activities (wailing, smiling, crawling), but others will be taken on her behalf (going out to play, when to go to sleep). And her food intake will be chosen on her behalf. As she matures into adulthood, she gains greater and greater autonomy (or so we assume here, for simplicity of exposition). So $C$ is, as a first approximation, her decision or the decision of her guardian; as is $A$. What about $G$? How is that chosen?

We interpret $G(t)$ to be a vector of government capital assets at $t$. $G(t)$ includes the stock of hospitals, research laboratories, doctors, nurses, and so on, at $t$. Health services at $t$ (which is a flow) should then be interpreted as the flow of services provided by the stock, $G(t)$. For simplicity of exposition we now concentrate on one component of that vector: government facilities for ending childhood obesity. We denote the stock of government assets pertaining to obesity by $G$, expressed in the common currency (e.g. dollars). It's a scalar aggregate.

Net increase in $G$ between $t$ and $t+1$ is $G(t+1)-G(t)$. The figure summarises the deployment of resources to ending childhood obesity. But there are competing uses for those resources. Economists have invented the tools of *social cost-benefit analysis* to help determine whether,

---

9 The private sector is important in health, but aside from differences in motivation that drives the decision maker, the analysis for the private sector is analogous to the one we study below.
all things considered, bringing about $G(t+1)-G(t)$ is desirable.

In some cases the decision to add to a particular type of government asset is reached by the prevailing political process (e.g. response to a regional demand for a new diabetes unit); in which case the analysis reduces to one of determining the most cost-effective way of doing so. A more elaborate method of analysis would seek to estimate the social worth of the project (e.g. whether, in view of competing claims on government resources, the new diabetes unit is justified). We sketch the more elaborate method of social cost-benefit analysis here. And for simplicity we focus on the deterministic case (equation (1)).

Imagine that the proposal is to expand the existing stock at $t$ by $\Delta G$. $\Delta G$ may be thought of as a new health unit targeting childhood obesity. So $\Delta G$ is the investment cost.

Note first that if the project is accepted, it would perturb the life course of the person we have been studying (whose age is $t$). Equation (1) would enable us to track the entire effect. Notice also that in determining how $\Delta G$ would perturb the person's life course, we would need to have an understanding of how the $C$'s and $A$'s in equation (1) get determined. The project evaluator therefore needs to have an understanding of the *behavioural* response to $\Delta G$ by people, over time. If that information were available, he could use equation (1) to determine $H(t+1)$ for the person in question. Analogously, he can determine the health status of all others in the economy (see Note-5). What remains is to value that change in terms of human well-being.

Good health brings three benefits to a person:

(i) It adds directly to the person's well-being (she feels good);

(ii) It enables the person to be productive (a healthy person works better and can work for longer hours than an unhealthy person);

(iii) It contributes to her longevity (a healthy person can be expected to live longer than an unhealthy person).

Items (i) and (iii) are direct benefits (they constitute aspects of a good life), while (ii) is an indirect benefit (a means to a better life). It is humanity's good fortune that good health offers the three benefits jointly (they are not in competition!). Economists have developed elaborate methods for estimating the value of each type of benefit, but in other contexts, some involve asking people to report their willingness to pay for the benefits ("reported preference"), while others estimate the value of the benefits to people by observing their behaviour ("revealed preference"). One way to estimate the combined benefit of improved health is by recording people's willingness to pay for better health (e.g., observing how much...
people spend on health). Some studies estimate the benefits enjoyed from (ii) by the output lost when workers are absent owing to illness. And there are studies that estimate the benefit enjoyed from (iii) by estimating the value of a statistical life.

Note-7. *All the above methods are inadequate.* The reason is that people don't buy health nor vote on health, they buy and vote on goods and services that contribute to health. And people typically have very incomplete information of the way the goods they purchase and the activities they pursue translate into the features of life they care about. Put another way, people can't be expected to possess adequate knowledge of the goods and services, nor the life style, that enable them to lead healthy lives (the $C$'s and $A$'s in equation (1)). To imagine that medical services are like any other consumption good is to make a category error: the customer does *not* know best. For these reasons, the methods developed by economists to ascertain the value of (i)-(iii) are usually supplemented by professional opinion of their value. It's no accident that the medical profession has a built-in authoritarian streak.

Consider person $i$. Let $P_i(t)$ be the value of $H_i(t)$, measured in the common currency (e.g. the market value of a representative basket of food items). $P_i(t)$ includes not only the value of health to person $i$, it also includes the value to others of $i$'s health (e.g. that the person isn't high maintenance for the government health sector). Call $P_i(t)H_i(t)$ person $i$'s *health capital*. Then $\sum P_i(t)H_i(t)$ is the aggregate health capital of the economy. The figure includes the health capital of people of all ages at date $t$. $\sum P_i(t)H_i(t)$ is a component of the economy's wealth at $t$.

In our example, the government is considering whether to inject $\Delta G$ into the diabetes unit. Using equation (1) sequentially on all the people in the economy, we may determine the change to the economy's health capital, $\Delta \sum P_i(t)H_i(t)$, occasioned by $\Delta G$. Call that change $\Delta B$ (that is, $\Delta B = \Delta[\sum P_i(t)H_i(t)]$). $\Delta B$ would represent the change in the economy's wealth, brought about by $\Delta G$. The net change in the economy's wealth would then be $\Delta B - \Delta G$. If it were estimated to be positive, the project would be recommended by the project evaluator; if it were negative, the project would be rejected.

That's the theory. In practice the way the government project evaluator calculates $\Delta B$ is by estimating the flow of benefits (items (i)-(iii)) to people over time that is occasioned by $\Delta G$. The flow of benefits is then discounted back to the present (date $t$). The present discounted value of the flow of benefits is what we have been calling $\Delta B$. The social cost-benefit rule is to accept the project if $\Delta B > \Delta G$ and to reject it if $\Delta B < \Delta G$.

Note-8: A great deal has been written on the rates at which future benefits and costs
ought to be discounted.\textsuperscript{10} Our example pertains to government expenditure, which means the project is in the public sector. The discount rate to be used in this case is known as the \textit{social} rate of discount. The government should use the \textit{same} rate to discount all the projects under its jurisdiction (aside from differences in risk categories).

\textbf{Note-9} If $\Delta B > \Delta G$ and the project is put into operation, the decision will have increased the economy's wealth. As was noted previously, health is a component of the wealth of nations. Improvements in health thereby are an essential feature of sustainable development.

\section*{5 Illustration of Social Cost Benefit Analysis of Project for Ending Childhood Obesity}

The illustration is stylised, but can be adapted easily to actual studies. The government’s proposed project is an initiative targeting childhood obesity. The investment outlay is $\Delta G$. For simplicity we focus on the \textit{over-weight newborn} (cohorts of ages 1-10 can be included in the same manner as we develop for the newborn). We thus consider the impact of the project on someone of age 0. We imagine that if the project is accepted, it will be implemented successfully.\textsuperscript{11}

Overweight children run the risk of becoming diabetic. The illness brings with it attendant costs, both to the diabetic and to others (including the government). To keep matters simple, we imagine that a person can be either a diabetic or not (there are no gradations) and that the per-period cost of being a diabetic is $x$. Diabetes shortens life. Imagine that someone born overweight is expected to live to age $T$; however, should a person fall prey to diabetes at age $t$, she would expect to die at age $T(t)$ years. Self-evidently, $T > T(t) > t$. Let $r (> 0)$ be the social rate of discount. (For simplicity we assume it is constant, although some governments now use a rate that declines over the project’s life.) In order to keep the notation tidy, we now assume that time is a continuous variable. Let the present discounted value of costs that are incurred when someone becomes diabetic at age $t$ be denoted by $D(t)$. It follows that

$$D(t) = x[1 - e^{-r(T(t) - t)}]/r.$$  

(3)

Notice that $D(t)$ is a function of $r$ (which is no surprise!), but it is also a function of the age ($t$) of the onset of diabetes (which is what we highlight here). That too should be no surprise: the older is the person, the fewer would be the required years of treatment.

However, whether an over-weight newborn will develop diabetes is uncertain.

\begin{flushleft}
\textsuperscript{10} The Secretariat at WHO have an extensive list of references to this literature.
\end{flushleft}

\begin{flushleft}
\textsuperscript{11} If there is doubt over the project's success, the project evaluator will need to articulate the probability of success and evaluate the project accordingly.
\end{flushleft}
Suppose epidemiological studies have estimated that the probability an over-weight newborn will become diabetic at some age is $\pi$, where $0 < \pi < 1$. (So $1-\pi$ is the probability that she will not become diabetic.) The problem is, the date of onset of the illness is unknown. Imagine now that epidemiological studies have estimated that the probability (rate) that the person will become diabetic at age $t$ is $\gamma e^{-\gamma t}$, where $\gamma > 0$. $\gamma$ is known as the hazard rate. (The hazard rate is the rate at which something happens to a person, conditional on that something not having happened to her until that date. $\gamma$ is the probability rate at date $t$ that the person will experience the onset of diabetes.)

But the proposed diabetes unit is being evaluated at $t = 0$. Moreover, it targets newborns. Using equation (3), it follows that the expected present discounted value of costs that are attributable to an over-weight newborn at $t = 0$ is $\int_0^T [D(t)\gamma e^{-\gamma t}] dt$. This is the expected loss in aggregate well-being at $t = 0$. The aggregate well-being we are alluding to here is the well-being of all who are alive and the potential well-being of future people. Let aggregate well-being at $t = 0$ be $V(0)$. We may then express the loss to aggregate well-being owing to the possible onset of diabetes to the cohort of newborns as $\Delta V(0)$. If the cohort of newborns at $t = 0$ is of size $M$, \[
\Delta V(0) = \int_0^T [MD(t)\gamma e^{-\gamma t}] dt. \tag{4}
\]

The public health project in question is designed to avert that loss. If it is found that $\Delta V(0) > \Delta G$, the project evaluator recommends the project. He recommends rejection if $\Delta V(0) < \Delta G$.

---

12 We are, for simplicity of notation assuming that the hazard rate is constant. In other words, we are assuming that the underlying stochastic process governing the onset of diabetes is a “Poisson process”. By our assumptions, $\int_0^T [\gamma e^{-\gamma t}] dt = \pi$. 