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Child Obesity

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Introduction

Childhood obesity, sometimes just referred to as overweight, is increasingly becoming a global public health concern (Kelishadi 2007; Kuczmarski et al. 2002; Reilly et al. 2003; World Health Organization 2010). Childhood obesity has debilitating consequences which may be short-term (for the obese child) or long-term (for the adult who was obese during childhood years). This review presents an overview of the definition and measurement of child obesity, development and factors associated with development of childhood obesity, magnitude of child obesity, consequences of child obesity and link to disability, and prevention and control of childhood obesity.

Definition and measurement of child obesity

Obesity reflects an imbalance between energy intake and expenditure, with the excess energy being stored as fat. Obesity in children is often measured using body mass index (BMI), defined as weight in kilograms divided by height in metre squared; as both weight and height, which are used in determining BMI are easy to measure. The International Obesity Task Force (IOTF) has recently come up with recommendations for determining obesity in children. This is based on data from six countries in both developed and developing countries: US, UK, the Netherlands, Hong Kong, Singapore and Brazil. The IOTF recommends absolute age and sex specific cut-offs for BMI in children defined to pass through a BMI of 25 and 30 kg/m2 at 18 years, for overweight and obesity respectively (Cole et al., 2000). Weight-for-height z-scores have also been used in defining child obesity. Other measurements may include waist circumference, skinfold thickness, and percentage of body fat using techniques such as multi-frequency bioelectrical impedance analysis (BIA) underwater weighing (densitometry), and magnetic resonance imaging (MRI).

Development of child obesity

The mechanism through which obesity develops is not fully understood and it is understood to be a condition with manifold causes (Davison and Birch 2001; Grundy 1998). It is generally believed that obesity results from caloric and fat intake increase, and decline in physical activity levels. Obesity arises when energy intake exceeds expenditure. Nutrition transition is believed to be the driving force behind the global obesity epidemic (Food and Agriculture Organisation (FAO) of the United Nations 2004, 2006; Popkin 2003; Popkin 2002). Nutrition transition refers to changes in diet

composition, commonly accompanied by changes in physical activity levels. Specifically, it refers to a shift from traditional diets mostly derived from plant-based food sources, to high energy dense foods and higher reliance on processed foods. The plant-based foods are low in fat and are high in fibre and their production is labour intensive. The modern high energy dense foods are high in fat and sugars, are often processed and prepared outside of the home, requiring less labour.

Developmental programming leading to the development of obesity and critical stages during which obesity develops, have been described. Genetic factors predispose individuals to the development of obesity. However, lifestyle preferences, environmental factors, and cultural environment are believed to play fundamental roles in the rising prevalence of obesity globally (Davison and Birch 2001; Grundy 1998). The system through which the different contextual factors interact to influence development of obesity in children has been described using Ecological Systems Theory (EST) (Davison and Birch 2001).

Developmental factors

Critical periods for the development of obesity and related complications include the early life period (prenatal and perinatal), the period of adiposity rebound during mid-childhood years; i.e. between the age of 5 and 7 years and adolescence period. (Dietz 1994)

Early life period

Prenatal and perinatal undernutrition or overnutrition influences adiposity later in life. Based on follow-up studies of infants (Braddon et al. 1986; Seidman et al. 1991), Dietz strongly hypothesised that low birth weight related to exposure to undernutrition during the first trimester confers a higher risk of later adiposity and hypertension or diabetes to the child. On the other hand, exposure of infants to maternal overnutrition may confer a higher risk of obesity but a reduced risk of subsequent morbidity to infants (Dietz 1994). In a later review, Martorell (2001) (Martorell et al. 2001) also concluded that overnutrition, represented by gestational diabetes or high birth weight is associated with subsequent fatness. On the other hand, association between poor nutrition during early life and later adiposity was inconclusive; with some studies indicating that children with a low birth weight were more likely to have higher adiposity than children with normal birth weight. Transition from nutritional scarcity to abundance or from rural to urban areas during early childhood and later life respectively, has been linked to the association between low birth weight and obesity (Martorell et al. 2001).

Developmental programming during the prenatal and perinatal period that may have implications for the development of obesity and obesity-related health risks later in life, is well illlustrated (Cripps et al. 2005; Gardner and Rhodes 2009; Gillman et al. 2008; Oken et al. 2008). Early life insults, for example exposure to prenatal maternal smoking, gestational undernutrition or overnutrition, inadequate breastfeeding duration and inadequate infant sleep duration may result in a child's programming that may lead to energy imbalance. A famous hypothesis with regards to developmental programming is the "thrifty phenotype hypothesis" also known as the "Barker hypothesis", proposed by

Hales and Barker in 1992 (Hales and Barker 1992). It states that adverse influences during early life, especially during the intrauterine period, can lead to permanent changes in metabolism and physiology which may further result in heightened risk of disease later in the life-course. In response to poor prenatal nutrition, the compromised foetus adopts survival strategies to maximize survival during the postnatal period. Metabolic programming occurs in a way that enhances survival in conditions of poor postnatal nutrition. If the foetus is born to conditions of poor nutrition, it adapts well to the environment. On the other hand, if the foetus is born to conditions of nutritional abundance, this conflicts with earlier programming leading to adverse consequences including obesity, Type 2 diabetes and other metabolic syndrome features. (Hales and Barker 1992)

Various mechanisms through which programming occurs have been suggested. These include disruptions in organ function which may result in alterations in the secretion and sensitivity of insulin; disruptions in appetite regulation due to dysfunction of the central nervous system and increase in the size and/or number of fat cells or changes in adipose tissue function (Gardner and Rhodes 2009; Muhlhausler et al. 2008; Whitaker and Dietz 1998). In response to its environment, a foetus may make physiological adaptations in preparation for postnatal life. For example, early insults may programme appetite regulatory mechanisms and may result in long-term consequences with regards to intake of food and food preference in later life. Developmental programming also has implications for energy sensing and the regulation of energy expenditure. Therefore, in individuals who are developmentally programmed, excess energy is progressively stored rather than sensed and regulated, increasing the risk of obesity and obesity-related morbidity in the life course (Gardner and Rhodes 2009).

Period of adiposity rebound

The time of adiposity rebound, between the age of 5 and 7 years represents another critical period for subsequent development of adiposity (Dietz 1994; Rolland-Cachera et al. 1987; Siervogel et al. 1991). BMI increases in the first year of life but reduces in subsequent years. However, from about five years of age, BMI begins to increase again. This period, where BMI begins to increase again is referred to as the period of adiposity rebound. The time when this adiposity rebound occurs may have significant implications on obesity later in the life course. The risk of increased BMI and adiposity in adolescence and adulthood is higher if adiposity rebound begins at an early age (before 5.5 years) than if it is average (6-6.5 years) or late (after 7 years) (Rolland-Cachera et al. 1987).

Adolescence

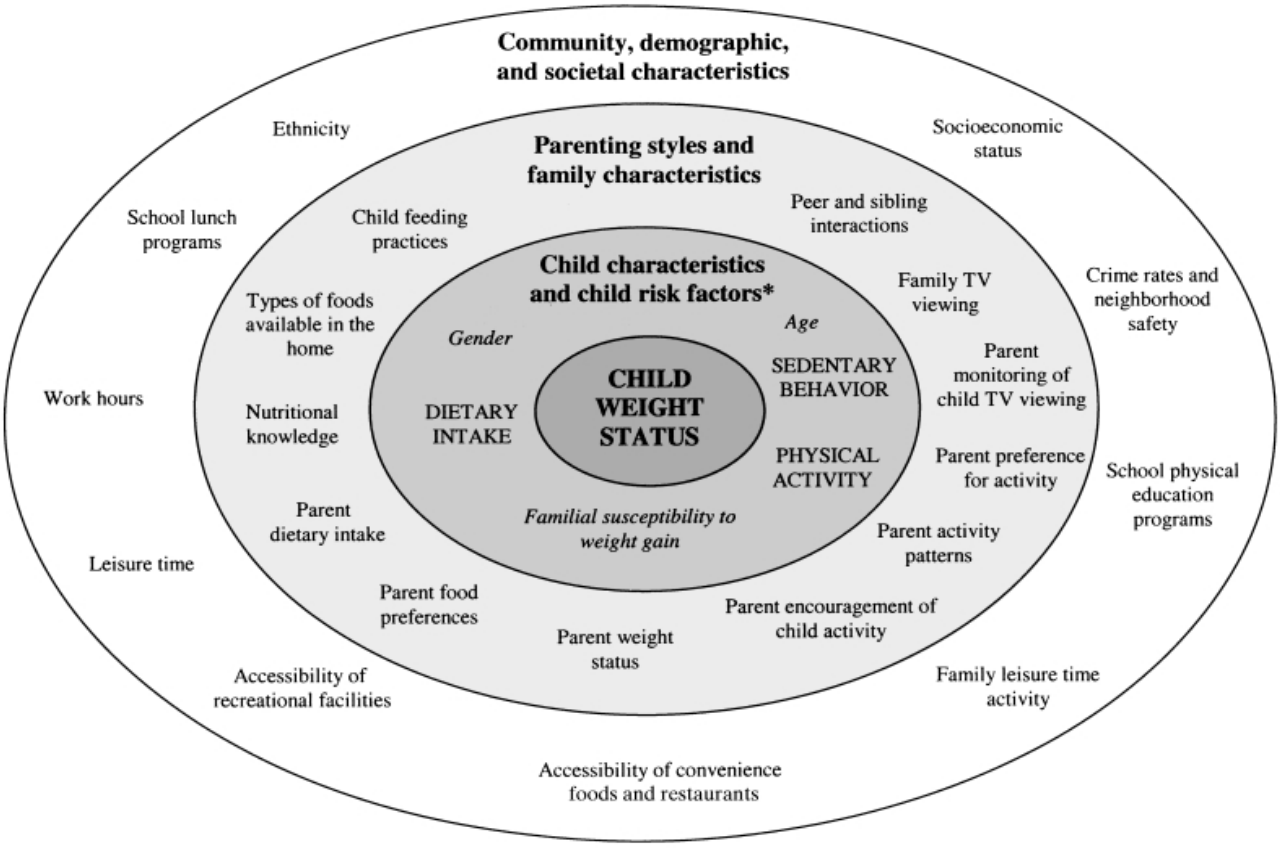
The final proposed critical stage for the development of obesity is during adolescence (Braddon et al. 1986; Dietz 1994; Mossberg 1989; Must et al. 1992). Obesity that has its onset during adolescence will persist in approximately half of the adolescents into adulthood (Dietz 1998a). The risk of obesity development is seemingly higher and persists longer in females than in males (Braddon et al. 1986). Adolescence also appears to be a critical period for the onset of obesity-related morbidity (Dietz 1998a; Mossberg 1989; Must et al. 1992).

Ecological factors

In a review of literature, Davison (2001) has described an ecological model of predictors of paediatric overweight/obesity (Davison and Birch 2001), (Figure 6), using the Ecological Systems Theory (EST) (Bronfenbrenner and Morris 1988). Under EST, human development is conceptualised to occur when interaction occurs within and among contexts. It emphasises the importance of consideration of the context(s) in which a person lives, in understanding the emergence of a characteristic. With regards to childhood development, its context(s) include the family/home and the school, which are found in larger social contexts that include the community and wider social and political environments. In addition to these contexts, child characteristics including gender and age, interact with familial and societal characteristics to influence child development (Bronfenbrenner and Morris 1988; Davison and Birch 2001).

As shown in the model (Figure 1), behavioral patterns of a child include dietary intake, sedentary behaviours and physical activity, referred to as child risk factors in the model, may predict child's weight status. Child characteristics including gender, age and susceptibility to gaining weight moderate the impact of child risk factors on the development of overweight/obesity. Additionally, parenting styles including restrictions and encouragement and family characteristics, including the dietary patterns of the parents, child feeding practices and sibling and peer interactions, shape the development of child risk factors. Further, school environment characteristics, for example structured activity periods and school-feeding programmes, and community and the wider societal and environmental factors including availability of recreational space and facilities, influence the weight of the child. They influence a child's weight status through influencing the child risk factors and family factors.

Figure 1: Ecological model of predictors of childhood overweight



Source: Davison 2001(Davison and Birch 2001)

Dietary patterns

Excessive energy intake without reciprocal energy expenditure may lead to high fat storage in the body - hence the risk of overweight/obesity. Energy intake has been associated with increased BMI in children in various studies (Berkey et al. 2000).

The association between dietary patterns and child weight status may depend on child characteristics such as age, gender, rate of growth and familial susceptibility to weight gain. The energy needs of children differ for boys and girls as well as in relation to rate of growth. Furthermore, the timing of growth spurts differs by sex, especially during adolescence. Overweight/obesity increases with age, particularly during adolescence (Cameron and Getz 1997; Kruger et al. 2006), and may reflect the effect of sexual maturation or other factors such as increased sedentary behaviour with age (Hardy et al. 2007). Many studies document the higher prevalence of overweight/obesity among girls rather than boys, particularly during adolescence (Cameron and Getz 1997; Jinabhai et al. 2007; Kelishadi 2007). Studies have further documented a positive association between a

child's overweight/obesity and parental BMI or fatness (Gibson et al. 2007; Mihas et al. 2009), which may reflect genetic susceptibility.

Family characteristics including parenting styles may also influence a child's dietary patterns. Studies have found similar dietary patterns between children and their parents (Davison and Birch 2001; Vauthier et al. 1996). Despite these similarities in familial dietary patterns, a genetic explanation is not supported by research; individuals living in the same household tend to exhibit similar dietary patterns irrespective of genetic relationship (Vauthier et al. 1996). The similarities in child-parent dietary patterns may therefore be reflective of environmental factors. The pathways through which parents may shape their children's dietary practices may include maternal nutritional knowledge, types of food availed by the parent to the child, parental child feeding practices and parental modelling of eating behaviour (Davison and Birch 2001; Gibson et al. 1998). Maternal nutritional knowledge and general health awareness affects diet composition availed to the child as well as portion sizes. Both are positively associated with the intake of fruits and vegetables by the child, and negatively associated with total energy and fat intake by the child (Gibson et al. 1998). Parents may act as role models for their children; children may prefer to eat the foods taken by their parents (Fisher et al. 2001). Again, parental feeding control may also affect a child's feeding patterns (Fisher and Birch 1999). Parental overweight/obesity status is also associated with dietary patterns of the child. Studies have found that mothers who are overweight/obese are more likely to feed their children on snacks and high energy dense foods. A child's feeding patterns may also be shaped by peers and siblings (Davison and Birch 2001). Community, demographic, as well as wider societal and environmental factors including ethnicity, socioeconomic status, work status, and availability of convenience foods influence child-parent feeding patterns. The participation of women in the workforce has reduced the time available for meal preparation, resulting in more consumption of convenience foods. Foods available in the community/environment including supermarkets is reflected in the types of foods provided to children by their parents (Davison and Birch 2001).

Physical activity patterns

Many studies have associated lower levels of physical activity with increased overweight/obesity in children (Davison and Birch 2001; Hernandez et al. 1999). However, a few other studies have found no association (Davison and Birch 2001; Treuth et al. 1998). Physical activity patterns in children are influenced by a combination of child characteristics and family, peer and sibling physical activity patterns, which are in turn influenced by community and wider societal and environmental factors including school physical education programmes and availability of recreational facilities.

Child characteristics including age and gender influence a child's likelihood of participating in physical activities and sporting activities. Research indicates that boys are generally more physically active, participate more in sports and are more physically fit compared to girls (Lindquist et al. 1999; Sallis et al. 2000; Vilhjalmsson and Kristjansdottir 2003). There is also a documented decline in physical activity with age, from childhood to adolescence, in physical activity including participation in sports, particularly in girls (Goran et al. 1998). This decline in physical activity with age may be

explained by pubertal onset and the accompanying physical, social and emotional changes (Lindquist et al. 1999). The gender difference in the decline may be due to the belief among girls that sports and physical activity are not feminine, which strengthens as they approach puberty (Goran et al. 1998).

Family characteristics also shape children's physical activity patterns. Evidence indicates that the participation of parents in physical activity is positively associated with children's and adolescents' physical activity (Vilhjalmsson and Thorlindsson 1998). Physical activity patterns among siblings and peers may also influence a child's activity patterns especially during adolescence (Vilhjalmsson and Thorlindsson 1998). Family influence on child's physical activity is in turn influenced by community, wider societal and environmental factors including ethnicity, socio-economic factors, work status, accessibility to recreational facilities and security. School characteristics including physical activity programmes and recreational facilities in school influence a child's physical activity patterns. Ethnic differences may be explained by differential socio-economic status. Higher physical activity levels have been documented in higher socio-economic groups as compared to lower socio-economic groups (Davison and Birch 2001; Vilhjalmsson and Thorlindsson 1998).

Sedentary behaviour

Sedentary behaviours such as TV viewing, watching videos and playing computer games may put children at risk of overweight/obesity as evidenced by several studies (Hernandez et al. 1999; Lajous et al. 2009). On the other hand, there is also some evidence suggesting that sedentary behaviour is not related to child's obesity (Biddle et al. 2004; Fogelholm et al. 1999). Sedentary behaviour may be associated with overweight/obesity due to its association with reduced physical activity (Hardy et al. 2009; Sallis et al. 2000). However, several studies have found that the association between sedentary behaviour and child weight status is independent of socio-economic and physical activity (Davison and Birch 2001). This then indicates that the influence of sedentary behaviours on child's weight status is not merely the result of physical activity displacement. The association of TV viewing to child's weight status independent of physical activity status may be associated with other factors including for example food advertisement and the consequent request by children for the purchase of advertised foods (Morgan et al. 2009). Length of TV viewing by children has been associated with higher consumption of fast foods, and other high energy dense foods and lowered intake of fruits and vegetables (Taveras et al. 2006).

Child characteristics such as age and gender may influence the relationship between sedentary behaviour and risk of overweight/obesity. There is limited evidence on the differential effect of sedentary behaviour on the risk of overweight/obesity by age, albeit some evidence of decreasing risk by age during adolescence (Vicente-Rodriguez et al. 2008). Some studies indicate that girls exhibit higher rates of sedentary behaviour than boys, particularly in the case of TV viewing (Davison and Birch 2001).

Parenting styles and family characteristics may influence sedentary behaviour in children. Parents shape sedentary behaviour of their children by their own sedentary behaviour and

by controlling their children's sedentary behaviour such as TV and video viewing (Davison and Birch 2001). Community and wider societal level factors such as ethnicity, socio-economic status and availability of recreation facilities may also influence sedentary behaviour in children, particularly through their influence on parenting styles and family characteristics. Research has found lower parental monitoring and control of child's TV viewing among lower socio-economic groups (Davison and Birch 2001).

Magnitude of child obesity

Child overweight/obesity is increasingly becoming a global public health concern. The rate of increase in child overweight/obesity per year has been estimated at 0.5-07% per year (Popkin 2008). Obesity has traditionally been associated with developed countries, particularly the United States, but it is increasingly affecting many low- and middle-income countries (LMICs), least prepared in coping with the consequences. It is estimated that the number of overweight/obese children aged less than five years of age globally is 42 million in 2010, while close to 35 million (over 80%) of these live in the developing countries (WHO 2010).

Among preschoolers, de Onis et al. in 2000 (de Onis and Blossner 2000) reviewed 160 nationally representative surveys from 94 countries in developing countries collected between 1970 and 2000. They indicated that the prevalence of overweight (defined as weight-for-height > +2 SDs from the NCHS/WHO reference median) was 3.3% among those developing countries included. Latin America and the Caribbean had the highest prevalence (4.4%). With respect to UN sub-regions, North Africa had the highest prevalence (8.1%). Southern Africa ranked second (6.5%), driven by South Africa (6.7%). In some countries including Uzbekistan, Egypt, Peru, Argentina, Malawi, Nigeria, Qatar and Jamaica, the prevalence of overweight exceeded that of the United States (de Onis and Blossner 2000).

The prevalence of overweight/obesity is reported as higher in older children and adolescents as compared to preschoolers in the developing countries (Kelishadi 2007). As with younger children, in developing countries, there is variation across regions and across countries. Overweight and obesity in older children and adolescents is reportedly highest in children and adolescents in the Middle East and in Central and Eastern Europe (Kelishadi 2007). In Africa, a high prevalence of overweight and obesity has been reported in North Africa in countries such as Egypt (Jackson et al. 2003; Salazar-Martinez et al. 2006) and in the Southern African region particularly in South Africa (Jackson et al. 2003; Reddy et al. 2008).

Consequences of child obesity and link to disability

Child obesity has debilitating physical and psychological consequences which may be short-term (for the obese child) and long-term (for the adult who was obese during childhood years). Common short-term consequences of overweight/obesity during childhood and adolescence include heightened risk of psychosocial morbidity; ashma; orthopaedic difficulties; cardiovascular complications; type 2 diabetes; and to some extent type 1 diabetes (Reilly et al. 2003; Zametkin et al. 2004).

Obesity signifies the most important risk factor for the development of insulin resistance in children and adolescents (Caprio 2002). Childhood obesity is therefore the driving force behind paediatric metabolic syndrome risk that is becoming highly prevalent globally and within the LMICs (Kelishadi 2007). Studies have indicated that obesity in children is positively and significantly related with cardiovascular disease (CVD) risks (Berenson et al. 1998; Freedman et al. 1999; Reilly et al. 2003). Results of the Bogalusa Heart Study indicate that close to 60% of overweight children had at least one CVD risk factor while using overweight as a screening tool; while it identified 50% of children with two or more risk factors (Freedman et al. 1999). Significant clustering of cardiovascular risk factors in paediatric obesity has also been observed in many studies (Berenson et al. 1998; Reilly et al. 2003). Expert opinions have consistently reached similar conclusions with regards to effects of childhood obesity on the cardiovascular system, pointing out similarities between children and adults in lifestyle factors and biological mechanisms through which obesity leads to CVD risk (Dietz 1998b; Williams et al. 2002).

The effects of child obesity on psychosocial morbidity are particularly important in adolescents particularly girls. Obese children undergo teasing and stigmatization leading to higher rates of anxiety disorders, low self-esteem, depression, and other psychopathology (Zametkin et al. 2004). These may lead to other complications including compromised academic performance and other longer term effects later in the life course including lower productivity.

Child obesity has been tracked into adulthood (Bibbins-Domingo et al. 2007; Dietz 1998a; Reilly et al. 2003). For example, in the Bogalusa Heart Study, 77% of overweight children ((BMI ≥95th percentile), remained obese as adults (BMI ≥30 kg/m2) (Freedman et al. 2001). Additionally, other risk factors for CVD associated with child obesity have been tracked to adulthood (Eisenmann et al. 2004; Reilly et al. 2003). Common longer term consequences, experienced during adulthood include heightened morbidity such as from cardiovascular diseases and type 2 diabetes, heightened risk of premature death, and impaired social, educational and economic productivity particularly for women (Dietz 1998a; Reilly 2006; Reilly et al. 2003).

Prevention and control of child obesity

Prevention and control of child obesity are critical given the debilitating effects of child obesity. Given the confirmed enormous influence of child obesity on adult obesity (Bibbins-Domingo et al. 2007; Dietz 1998a; Freedman et al. 2001), it is important that interventions be initiated as early as possible to cub the global problem of obesity and its consequences. Children are the priority group for interventions due to various reasons including that weight loss in adulthood is difficult and there are more potential interventions for weight management for children than for adults. Intervention programs should focus on both energy consumption and energy expenditure. Research has indicated that focusing on encouraging free play and reducing sedentary behaviour is more effective than reducing food intake or focusing forced exercise in controlling weight gain among obese children (Caterson and Gill 2002).

Addressing the problem of child obesity successfully requires a multilevel and multi-sectoral approach directed to the multiplicity of the factors associated. With respect to the ecological model of predictors of childhood obesity, interventions should take into consideration the contexts in which a child lives including the familial context, the school context, the community and the larger societal environment (Davison and Birch 2001). Studies have shown that programmes that encourage weight loss among children and their parents have bigger long-term achievement compared to those that focus solely on child weight reduction (Brownell et al. 1983; Epstein et al. 1990). Familial factors such as parents' dietary practices, TV viewing practices, physical activity, nutritional knowledge, and child feeding practices; parental influence on child's physical activity; and parental monitoring of child's TV viewing need to be put in consideration. Community and the wider societal factors which influence development of child obesity such as availability of fast food outlets, regulation of food advertisement targeted at young children, availability of recreation facilities, security in the neighbourhoods and labour policies that influence time available for preparation of meals at home also need to be considered (Davison and Birch 2001).

Children generally spend most of their days in school, usually five days per week. Therefore schools, which act as central institutions for children socialization, are ideal locales for instituting obesity-prevention programs. Schools may play an important role in influencing children's food and physical environment. Various studies have found school-based interventions on physical activity knowledge and behaviour to be effective (Stone et al. 1998). Increase in sports participation and time for physical education need policy-based changes both at school level and at education sector level. Likewise, increase in active modes of transport for children to and from school e.g. walking and cycling require policy changes at school and government levels, as well as parental and community support (Swinburn and Egger 2002).

Conclusion

This review has described child obesity, its causes, its consequences and its prevention and control. Childhood obesity, is progressively becoming a global public health concern, experienced both in the developed and developing world. Child obesity is associated with incapacitating consequences, both physical and psychosocial, experienced during childhood and carried over to adulthood. Causes of obesity are not fully understood, but is believed to arise from a multiplicity of causes (Davison and Birch 2001; Grundy 1998). Obesity occurs when energy intake exceeds expenditure. Though genetic factors may predispose children to the development of obesity, contextual factors including lifestyle factors, environmental factors, and cultural environment, linked to nutrition transition occurring globally, are believed to play more important roles (Davison and Birch 2001; Grundy 1998). Development of obesity in children is influenced by several contextual factors including child characteristics and child risk factors, parenting styles and family characteristics, and community demographic and social characteristics. Given the contextual nature of factors associated with the development of childhood obesity, prevention and control must be multifaceted, taking into account the different contexts in which the child lives including the familial context, the school context, the community and the larger societal environment (Davison and Birch 2001). School-based intervention

programmes may particularly be effective given that children spend most of their days in school.

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