

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

5,500

Open access books available

136,000

International authors and editors

170M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Obesity in School Children in India

Vangal Krishnaswamy Sashindran and Puja Dudeja

Abstract

The global prevalence of obesity has doubled from 1990 to 2015. Worryingly, the increase is more in children than in adults. In just three decades, the number of school-going children and adolescents with obesity has increased by 10-fold, and the International Association for the Study of Obesity (IASO) and International Obesity Task Force (IOTF) reckon that 200 million school children worldwide are either overweight or obese. The prevalence of obesity among 5- to 19-year-old Indian children, ranged between 3.6 and 11.7%. It is predicted that by 2025 there will be 17 million obese children in India. Urbanisation is the single most important factor linked to obesity in India. Epigenetic, dietary, familial, psychosocial, parental education and parental occupation are other important factors. About 50% of obese children will become obese adults. The prevalence of hypertension, type 2 diabetes dyslipidaemia and non-alcoholic fatty liver disease in children is also increasing parallelly. Prevention of childhood obesity is vital because it is near impossible to get children to lose weight and maintain it. A healthy diet and an active lifestyle should start from the pre-conception time itself and be continued through all stages of childhood.

Keywords: India, childhood, overweight, obesity, causes, consequences

1. Introduction

Non-communicable diseases are the biggest public health challenge of this century, and childhood obesity is an important part of this. Its rates have tripled in the US over the past 30 years. Today, 17% of US adolescents and 16% of children between 2 and 11 years are obese with prevalence being highest among Black and Hispanic children and adolescents [1]. Childhood obesity was so far thought to be a problem of the developed world, but it is increasingly being reported from middle- and low-income countries, especially from urban areas [2]. Obesity in children is not as easy to quantify as in adults. Body mass index is not an accurate measure of obesity in children. It is important to recognise childhood obesity and manage it, because if untreated, it can result in obesity in adulthood with all its attendant metabolic complications. Childhood obesity also has a deep psychosocial impact and is consistently associated with lower scholastic achievements. Weight once gained is difficult to lose, and hence prevention is important. This assumes greater significance with regards to children as compared to adults, because they are more susceptible to the constant bombardment by advertisements for energy-dense food [3]. Immuring them from an obesogenic environment is a priority and a challenge. A number of innovative programmes to tackle childhood obesity have

been tried in different countries, and each one of them has important lessons for public health specialists and health policy makers. The WHO's Commission for Ending Childhood Obesity opines that this problem requires, 'a whole-government approach in which policies across all sectors systematically take health into account, avoid harmful health impacts, and thus improve population health and health equity' [4]. The magnitude of the problem, causative factors, complications of childhood obesity and some solutions will be discussed.

2. Prevalence

2.1 Global prevalence

Obesity in school children is today an uncomfortable reality. The global prevalence of obesity has doubled between 1990 and 2015. Worryingly, the rate of increase in obesity in children is higher than that in adults in many countries [5]. Globally, about 10% of school children in the 5–17 age groups are obese or overweight [5]. The prevalence varies from 30% in America to less than 2% in sub-Saharan Africa. In just three decades, the number of school-going children and adolescents with obesity has increased by 10-fold, from 11 million to 124 million (2016 estimates). In addition to this, another 216 million children were estimated to be overweight though not obese in 2016 [6]. The International Association for the Study of Obesity (IASO) and International Obesity Task Force (IOTF) reckon that 200 million school children worldwide are either overweight or obese [7]. The problem probably starts early in childhood. A 2010 report estimated that 42 million children under the age of 5 were overweight and of these, 35 million lived in developing countries. While the prevalence of childhood obesity may be plateauing in some developed countries, it is showing a steep rise in developing countries of Asia and Africa. Within Asia, China has the highest number (15 million) followed by India (14 million) [4]. Even within nations, certain subgroups like children of migrants and indigenous populations are at greatest risk of obesity [8].

The NCDRisC study pooled data for 2416 population-based studies. This included data of 31.5 million children and adolescents aged 5–19 years. The mean BMI in 1975 was 17.2 and 16.8 kg/m² for girls and boys, respectively. It was lowest in South-East Asia and East Africa, and highest in Polynesia, Micronesia and English-speaking regions. The age-standardised mean BMI for children and adolescents increased all over the world from 1975 to 2016. The increase was 0.32 kg/m² per decade for girls and 0.40 kg/m² per decade for boys. The mean BMI for girls and boys in 2016 was 18.6 and 18.5 kg/m², respectively. The age-standardised mean BMI in 2016 was still lowest in South-East Asia and east Africa and highest in Polynesia and Micronesia. It was 16.9 and 17.9 kg/m² for girls and boys respectively South-East Asia and Africa and 23.1 and 22.4 kg/m² for girls and boys respectively in Polynesia and Micronesia. The regions with the largest absolute numbers of obese children and adolescents were East Asia, the Middle-east, North Africa, South Asia and English-speaking regions. It is predicted that if post-2000 trends continue, then by 2022, the number of obese children will outstrip those with moderate and severe underweight [3].

Peltzer reported that the prevalence of overweight/obesity in school children aged 13–15 years in seven ASEAN countries (excluding Brunei) was 9.9% [9]. The highest prevalence of overweight/obesity in all eight ASEAN countries was in Brunei Darussalam (36.1%), followed by Malaysia (23.7%). It was lowest in Myanmar (3.4%) and Cambodia (3.7%) [10]. Pengpid and Peltzer also studied the prevalence and factors affecting obesity in children in six Pacific island countries of

Oceania. Among the 10,424 children in the age group of 13–16 years, the prevalence of overweight and obesity was 24.3 and 6.1%, respectively. The researchers used a BMI of $>30 \text{ kg/m}^2$ to define obesity and defined overweight as a BMI between 25 and 29.9 kg/m^2 . Therefore, their estimate may not be quite accurate [11].

2.2 Indian prevalence

India is caught in a nutrition paradox where stunting and underweight coexist with overweight and obesity in children. National Family Health Survey-4 (2015–2016) reported the prevalence of stunting, wasting and underweight in children <5 years to be 38%, 21% and 36%, respectively. In this survey, overweight was defined as weight for height being more than 2 SD above the median of the reference population. By this definition, 2% of Indian children under the age of 5 were overweight. Unfortunately, data of older children are not forthcoming from this survey [12]. The prevalence of overweight/obesity among adolescent Indian children rose from 9.8% in 2006 to 11.7% in 2009 [13]. Lobstein and Jackson-Leach computed that there will be 17 million obese children in India by 2025 [14]. This trend is reported from all over India, both in urban and rural areas. Prevalence of overweight/obesity in children in Delhi increased from 16% in 2002 to 24% in 2006 [15]. A study done in the urban areas of Udupi in South India, found prevalence of overweight and obesity in school children to be 10.8 and 6.2%, respectively [16]. Another study from Central India found 3.1% (95% CI 2.5–3.8) of children between 10 and 17 years to be overweight and 1.2% (95% CI 0.8–1.8) to be obese and overall 4.3% were overweight/obese [17]. From Surat, in Western India, Gamit et al. reported the prevalence of overweight and obesity to be 10.2 and 6%, respectively [18]. In Kanpur, the prevalence of overweight and obesity were 4 and 2%, respectively. The authors attributed this low prevalence, when compared to other studies from North India, to local dietary habits [19]. A systematic review conducted by Gupta et al., reported that prevalence of overweight, among 5–19 years children, ranged between 6.1 and 25.2%, while that of obesity ranged between 3.6 and 11.7% [20]. Khadilkar et al., in 2010, estimated the combined prevalence of overweight and obesity to be 19.6% as per IOTF classification, while this was 27% according to WHO definitions [21]. Among adolescence, between 10 and 17 years, the percentage was 22.3 (as per IOTF cut off) and 29.8% (as per WHO cut off), and these age groups should be considered most vulnerable for adiposity [22]. The estimates will swing widely till all investigators adopt standard criteria for measurement of overweight and obesity.

3. Factors causing obesity in school children

Obesity is a result of imbalance between calorie intake and energy output. This may seem a facile explanation, but in reality it is due to a complex interplay of many factors. Biological factors can lead to childhood obesity through ‘mismatch’ and ‘developmental’ pathways [4].

3.1 Genetic factors

Obesity is probably polygenic in inheritance. BMI may be 25–40% inheritable. But, like for hypertension and diabetes, behavioural and environmental factors play a big role. Genetics may just account for less than 5% of all cases of childhood obesity [23]. Maternal undernutrition or malnutrition and placental insufficiency lead to epigenetic changes that put these children at greater risk for developing

overweight and obesity when exposed to energy-dense foods and sedentary lifestyles when compared to children born of mothers with adequate nutrition. Maternal obesity or hyperglycaemia can also cause epigenetic changes, which predispose the children to increased deposition of fat tissue. Recent studies reveal that paternal obesity can also have an adverse effect on the offspring through epigenetic effects [4].

3.2 Individual factors

3.2.1 Gender

The GDB Collaborators Group did not find any gender difference in prevalence of obesity in children <20 years. In countries with high-medium and high SDIs, the prevalence was higher in boys than in girls with the trend reversing in late adolescence [5]. The NCDRisC study also reported that gender disparity had narrowed considerably all over the world by 2016. They reported a flattening of the curve for both sexes in North-western Europe, high-income English-speaking countries and Asia-Pacific regions. The plateauing was also seen for boys in South-western Europe and girls in Central and Andean Latin America. In contrast, a rise in BMI was noted for both sexes in East and South Asia and for boys in South-east Asia. The prevalence of obesity was highest in Polynesia and Micronesia in both sexes being 25.4% in girls and 22.4% in boys. This was followed by the high-income English-speaking countries. Obesity prevalence was between 1 and 2% in Cambodia, Burkina Faso, Vietnam, Ethiopia, India, Madagascar, Republic of Congo, Japan, Nepal, Niger and Chad. The number of girls with obesity increased from 5 million in 1975 to 50 million in 2016. For boys, the numbers are 6 million and 74 million, respectively [3]. However, other studies do report differences. The prevalence of overweight/obesity in children and adolescents is more in boys than in girls in developed countries (23.8 and 22.6%, respectively). In developing countries, it is lesser in boys than in girls (12.9 and 13.4%, respectively) [24].

Studies from different parts of India also show that it is more common in boys than in girls. Ramachandran et al. reported the prevalence of overweight to be 17% in boys and 15.8% in girls in the age group 13–18 years from Chennai [25]. In the study from Udipi in South India, the prevalence of overweight and obese were 11.0 and 7.1%, respectively, in boys and 10.6 and 5.4%, respectively, in girls [16]. Researchers from Wardha, in Central India, reported the prevalence of overweight/obesity to be 4.4% in boys and 4.3% in girls [17]. In Pune, in Western India, the prevalence of overweight in children in the age group 9–16 years was 27.5% for boys and 20.9% for girls [26]. In Surat, the prevalence of overweight and obesity in boys was 12.4 and 8.2%, respectively, and in girls it was 7.2 and 2.7%, respectively [18]. Kapil et al. reported the prevalence of obesity in children aged 10–16 years in Delhi to be 8.0% for boys and 6.0% for girls [27]. Chhatwal et al. also found higher prevalence of obesity among boys than girls. They attributed it to the cultural advantages that boys enjoy in India. They get larger helpings of food at home, snack more as they have greater freedom to go out and also participate negligibly in doing household chores. The gender disparity was highest in the most affluent socio-economic groups. Among children going to the affluent school, the prevalence of overweight was 25% in boys and 16.6% in girls ($p = 0.001$). The prevalence of obesity was 19.9 and 13.3%, respectively ($p = 0.003$). The prevalence was similar among boys and girls in the lower-income schools. They also found that only at the age of 15 years was prevalence of obesity/overweight more in girls than boys. This is explained by the pubertal hormonal surge and growth spurt that occurs earlier in girls than in boys [28]. These findings are reiterated in the systematic review of 28 studies by

Ranjani et al. who found that overweight and obesity were slightly more common in adolescent boys than girls in India [2].

3.2.2 Age

There is a distinct age pattern in childhood obesity. In their longitudinal study on 8544 children, Whitaker et al. reported that the prevalence of obesity was 13% at 9 years and dropped to 9% at 14 years and then increased again [29]. The GDB Collaborators Group also reported that the prevalence of obesity decreased with age till 14 years and then increased [5]. In the American NHANES 1999–2000 survey, the prevalence of overweight/obesity did not vary much in the age groups of 6–11 and 12–19 years (15.3 and 15.5%, respectively) [30]. The findings by Chhatwal et al. in Ludhiana mirrored Whitaker's study. Prevalence of obesity declined from 18.5% at 9 years to 7.6% at 14 year-end then again spiked at 15 years to 12.1% [28]. Ranjani et al. found that prevalence of obesity in under-fives was less than 2% across India. In children >5 years, it varied from 2 to 8%. Overweight rates were about 2× higher and were higher in North and East India than in South India. Among adolescents, the overall prevalence of overweight and obesity ranged between 3 and 24.7% and 1.5–28% respectively [2].

3.2.3 Dietary factors

With globalisation, the dietary mores of Indian children has also started changing rapidly. Gulati et al. found that a majority of children surveyed in four urban centres preferred to eat out; they felt that home food was 'old-fashioned'. Almost half of them also had their evening meals while watching television [31, 32]. Adolescents associate 'junk food' with independence and convenience and consider health food options odd [33].

In the Udupi study, there was no statistical correlation between frequent consumption of carbonated drinks and being overweight/obese [16]. But in Oceania, Pengpid and Peltzer found that consumption of >1 carbonated drink per day increased OR for obesity by 1.32 (95% CI 1.09–1.61, $p < 0.01$) [11].

A drastic behavioural change is seen in Indian children in senior secondary schools. Those interested in taking competitive examinations stop physical activity totally and adopt a sedentary lifestyle. They enjoy eating packaged food items to relieve stress. These are usually energy-dense high fat, sugar and salt (HFSS) foods. Both nocturnal snacking and consumption of HFSS foods in the breaks between study sessions can lead to significant weight gain. Snacks rich in refined sugars and fats are habit forming and children get addicted to their flavours and tastes. This perspective is supported by a growing body of neuroscience researchers who have demonstrated that the chronic consumption of energy-dense foods brings about changes in the brain's reward pathways that are central to the development and maintenance of habits. While it is difficult to precisely define 'eating behaviour', such habits are associated with pleasure centre in the brain (neural reward circuitries)—characterised by symptoms such as loss of control while eating, over consumption and/or binge eating, continued consumption of high calorie foods despite the knowledge of its negative consequences and inability to cut down despite the desire to do so [34, 35]. The vicious cycle of obesity in school children is shown in **Figure 1**.

The reinforcing value of food is higher among obese children than among children with normal weight. In general, bland foods are not eaten in excess; whereas, highly palatable foods are often consumed even after an individual's energy requirements have been covered. Some children fall prey to vicious cycle of impulsive

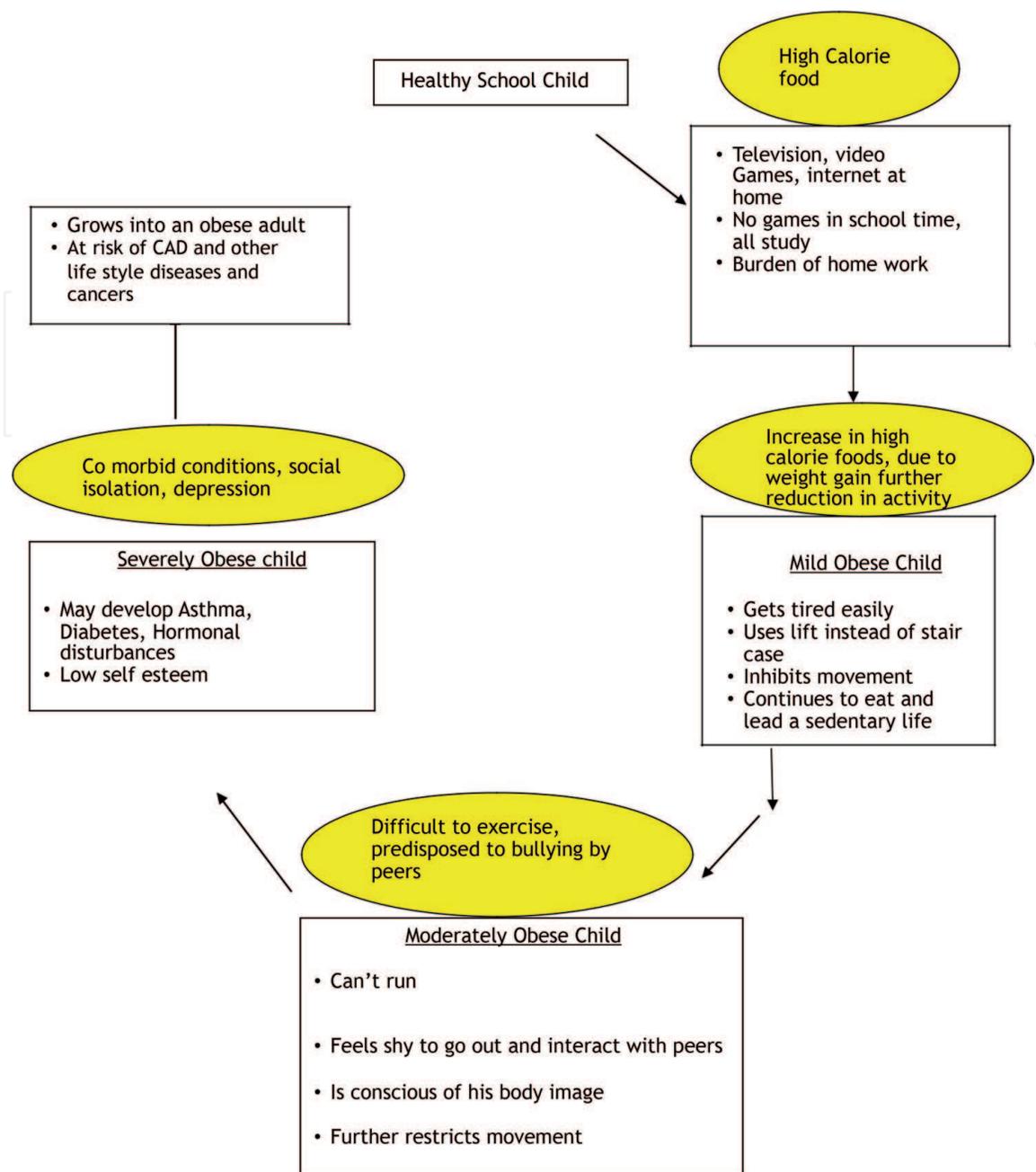


Figure 1.
Vicious cycle of obesity in school children.

eating. Highly impulsive children often do not think about the reactions or their consequences. Besides overeating, these children seem to be vulnerable to food triggers like the smell and taste of the food. It has been suggested that poor control of neural centres related to impulsivity and/or addiction could foster impaired control of food intake leading to overeating and subsequent obesity. Adaptive decision-making and the ability to delay gratification may positively influence eating behaviour, particularly in an energy-rich food environment, where conscious control of energy intake is essential for the maintenance of healthy body weight [36].

3.2.4 Physical activity

The Wardha study showed a good correlation between physical inactivity and childhood obesity. On univariate analysis, the odds ratio (OR) of obesity in school children was 2.064 if they played outdoors for less than 30 min a day. Step-down multiple logistic regression analysis showed OR for the same children

to be 2.133 (95% CI: 1.373–3.301) [17]. In the study from Oceania, sedentary behaviour defined as sitting for ≥ 3 h/d increased the odds of being overweight/obese OR1.17 (95% CI 1.03–1.34, $p < 0.05$) [11]. More frequent participations in sports correlated well with greater accretion of fat free mass. This finding was independent of ethnicity, individual, family, community and socio-economic factors. Walking or cycling to school also resulted in a lower fat mass index [37]. Kunwar et al. studied the prevalence of obesity in school children in a military station in North-Eastern India and concluded that the prevalence of obesity in garrison schools was lower due to the greater emphasis laid on games and physical activity [38].

3.2.5 TV watching

Sedentary lifestyle is associated with higher adiposity. Every additional hour of TV-time per day increases the prevalence of obesity in children by 2% [23]. Govindan et al. found that watching TV for >2 h/d was significantly associated with obesity (boys OR 1.19, $p < 0.01$, girls OR1.19, $p < 0.01$) [39]. The National Institutes of Health, US also consider TV-time of >2 h as a definite risk for obesity [40]. TV watching (passive screen time) is unhealthier than active screen time (computer and video games) [41]. Hours of TV watching also directly correlate with increased intake of foods frequently advertised on TV like sweets, sweetened beverages, cookies, chocolates, sweetened cereals and salted snacks [42]. This habit of watching TV or playing games on mobile phones starts at an earlier age. During preschool period, most mothers have a tendency to feed children by distracting them. They let them watch cartoons on television or play games on mobile phones. This makes their job of feeding easier as the children do not resist feeding. In the process, they tend to overeat, being too distracted to signal the feeders that they are full. This tendency to overfeed themselves continues into adolescence also.

3.2.6 Psychosocial factors

Pengpid and Peltzer looked for impact of loneliness, lack of close friends, anxiety and worrying, suicidal ideation and bullying on occurrence of overweight/obesity in school-going children in Oceania. The OR for being overweight with lack of close friends was 0.72 (95% CI 0.56–0.92, $p < 0.01$) and with suicidal ideation OR was 1.42 (95% CI 1.21–1.66, $p < 0.001$). The association with other factors was not significant on multivariate regression analysis [11]. The lifetime risk for anxiety disorders is higher for obese adolescents in comparison to non-obese ones [43]. There is a bi-directional relationship between eating disorders and depression making both difficult to treat [44].

3.2.7 Familial factors

The family has a significant role to play in the development of childhood obesity. The influence starts even before the child is born. The first reported causal association of intra-uterine undernutrition resulting in obesity in the offspring later in life was from the observation of children born to women who were pregnant during the Dutch famine [45]. Intra-uterine malnutrition may lead to a higher susceptibility to excess weight gain due to increased fat stores, short stature and a preference for foods high in fats [46]. This is the 'mismatch' effect alluded to earlier. A phenomenon known as 'mirror imaging' between the mother and her baby is observed in India. The baby's birth size is predicted by the mother's size before pregnancy. Despite this, birth weight has been found to be a poor indicator of adiposity in the

foetus. In Caucasian populations, birth weight is related to lean mass rather than to fat mass; whereas, in India, babies have more fat mass as compared to Europoid babies at every ponderal index. Babies with both low- and high-birth weights have a high risk of obesity in later life in India [47].

Maternal and paternal heights are independent predictors of childhood obesity. The highest adiposity is seen in children who exceed mid-parental height. Parental height may serve as a composite indicator of genetic factors, nutrition and growth during childhood [47, 48]. Many studies in India have shown that the mother's BMI correlates with the child's weight. This may be due to genetic factors and environmental factors such as shared diets and eating habits [31, 49]. Raskind et al. found that every overweight child had an overweight mother. The combination of overweight mother and child formed 11% of their study population which was one of the highest in Asian countries [50]. Rebecca Kuriyan studied the role of familial and sibling factors on abdominal adiposity in children in an urban location in South India. Multiple regression models showed that overweight status of father or mother was associated with the younger sibling having abdominal obesity (OR = 1.45 and 1.81, respectively). If both parents were overweight, the OR for the younger sibling having abdominal obesity was 1.63 (95% CI: 1.33–2.99). If the older sibling had abdominal obesity, then the OR for the younger sibling to have the same was 3.22 (95% CI: 2.30–4.50). When siblings were of the same sex, the odds were higher (OR 3.55, 95% CI 2.24–5.65) than if the siblings were of different sexes (OR 2.73, 95% CI: 1.67–4.46). The association was stronger among male sibling pairs (OR 4.18, 95% CI: 2.21–7.93) than female sibling pairs (OR 2.85, 95% CI 1.42–5.72). All the above findings were statistically significant ($p < 0.01$). These effects are probably due to behaviour modelling among siblings and this is strongest in same sex siblings. The younger child is likely to follow the elder sibling in a number of behavioural traits like academic engagement, smoking, alcohol use, substance abuse and sexual behaviour [48]. Not only are dietary habits of children modelled on those of their parents and peers, but they also pick up food preferences, intake and willingness to try new foods from them. The key to good dietary preferences is the availability and repeated exposure to a variety of healthy foods. Authoritative feeding refers to parents who determine what foods are offered and then allowing children to choose. This leads to a positive attitude to healthy eating. However, authoritarian restriction of 'junk foods' works in the opposite way. It increases the children's desire for these foods and leads to weight gain. It is also known that families that eat together consume healthier foods. There is a greater tendency to consume fast foods in single parent families or where both parents work. Eating out regularly and 'TV dinners' are both associated with higher intake of dietary fats [51, 52].

Peer support at school, supervision of studies by parents, parental connect with children and bonding have all been studied. Pengpid and Peltzer founds that odds for overweight/obesity correlated with peer support at school (OR 1.28, 95%CI 1.08–1.53, $p < 0.01$), parental support (OR 1.33, 95%CI 1.13–1.58, $p < 0.001$) and parental bonding (OR 1.31, 95%CI 1.12–1.54, $p < 0.001$) [11].

3.2.8 Socio-economic factors

There are many commonalities in socio-economic factors associated with childhood obesity from studies across India. Urban background has the strongest association. Familial economic status, educational levels of parents and job profile of parents are other important factors. The type of school that children study in also is an important determinant. Strangely, data on religious denominations is conflicting.

Urbanisation is the strongest risk factor for obesity in India. Obesity is three times commoner in cities as compared to rural areas. Development of good roads and satellite television have blurred the divide between cities and villages. Youth migrating from villages to metros to study and work take back urban food habits and norms back to their villages. Thus, Indian villages are getting urbanised in their habits. This phenomenon is referred to as rurbanisation or urbanisation in situ [47]. Bharathi et al. reported that the OR for obesity in school children from urban areas as compared to rural areas was 3.046 (95% CI: 1.662–5.582) [17]. Rapid urbanisation has led to ‘McDonaldisation’ of society in terms of an increase in the culture of eating out and eating fast foods. An important contributory factor to this change has been increasing financial independence of women, who are now spending less time in their kitchens. Such households often pack energy-dense convenient food in school tuck boxes and also offer the same as snacks between meals. This results in excess consumption of calories by the children and leads to fat gain.

Higher socio-economic status is another risk factor. The GDB Collaborator Group found prevalence of obesity to be higher in countries with higher socio-demographic index (SDI). A relative increase of 20% was found in the prevalence of obesity in children of both sexes between 1980 and 2015 in low SDI countries. The increase was greatest in countries with middle SDI [5]. An American study compared school children from two neighbouring communities, the first with a median household income of US\$ 28610 and the second with US\$ 46299. The prevalence of obesity was 22.2 and 12.6%, respectively ($p = 0.01$) [53]. Chhatwal et al. found a direct correlation between socio-economic status and overweight/obesity in children from three different schools in Ludhiana, Punjab. Prevalence of obesity was 15.5% in children from socio-economic class I (highest income group), 2.7% in children from class IV and 0% in children from class V (lowest income group.) [28]. Eagle et al. observed that as the average American household income decreased, frequency of consumption of fried food and TV or video time/week increased. Consumption of vegetables and moderate/vigorous physical exercise also proportionately decreased [54].

Kaur et al. reported three times higher number of overweight children (15.3%) and obese children (6.8%) in high-income schools as compared to children in lower-income schools [55]. Prevalence of overweight/obesity in children aged 14–17 years from urban Delhi was 29% in private schools and 11.3% in government-funded schools [15]. In Udupi, the percentage of children with BMI in overweight/obese range were 6.9%, 10.9% and 31.2% for government, aided and unaided/private schools, respectively ($p < 0.001$) [16]. Ramachandran et al. reported an overweight/obesity prevalence of 4.5% in low-income schools and 22% in better-off schools in Chennai [25]. Another interesting factor related to schools is the provision of free lunches or mid-day meals (in India). Govindan et al. reported that regular consumption of school lunches was associated with obesity in both boys and girls (boys OR 1.29, 95% CI 1.01–1.64, $p = 0.04$, and girls OR 1.27, 95%CI 1.00–1.62, $p = 0.05$) [39].

The effect of religion on childhood obesity is varied. In Udupi, the odds of overweight/obesity in children from Muslim or Christian homes was significantly higher (for Muslims the AOR was 2.26, and for Christians it was 1.60) in comparison to children from Hindu homes [16]. The authors attributed this to a focus on vegetables in the Hindu diet and a lack of meat [16]. In Wardha, however, the OR for obesity was 1.730 ($p = 0.036$) for Hindu children when compared to children from other communities [17]. Here, it probably was influenced by greater affluence of Hindus in comparison to Muslims and Christians.

Myths related to obesity in school children	Reality
Fat children are healthy, as they are not undernourished	Fat children are not healthy inside. About 28% of fat children have syndrome X. They have a risk for chronic lifestyle diseases
With age, children will gain height and loose fat	Majority of obese children become obese adults
So what, if a child is obese! Obesity is a problem of adults and not of children	Fat children are at risk of developing early diabetes. Girls may develop polycystic ovarian syndrome
Heart disease starts at old age	Hardening and blockage of the arteries starts at 11 years in boys and 15 years in girls
Children do not develop high blood pressure or high cholesterol	Many children will have high blood pressure and low HDL-cholesterol
Children should eat, drink and be merry. Childhood will not return	Children should enjoy being active as such energy will not come again later
Children by nature are physically active	Time on TV, Internet and studies leaves little time for play. Many do not participate in sports activity in mandatory sports periods in schools.
Observing a child to be fat is considered inauspicious	Obesity in children should be viewed with concern

Table 1.

Some common myths in India society related to obesity in school children.

4. Community/society level factors

Structural elements like road, transportation, structure of buildings, playgrounds, parks and public spaces influence obesity in children. Research conducted over the past decades provides increasing evidence that there is a direct correlation between easy access to supermarkets laden with cheap and readily available HFSS food and sweetened carbonated drinks and obesity in children. Increased concretisation and loss of public spaces and parks in cities have led to decrease opportunities for sports and a more sedentary lifestyle. Many poorer neighbourhoods are considered unsafe for children because of drug pedlars and predatory adults. Parents prefer to keep their children indoors. Even when they have to go out or attend school, the parents drop them by car. Walking or cycling to school often considered a healthy activity is thus lost [23].

Children are bombarded by commercials of confectionary, chocolates, sweetened cereals and fizzy drinks on TV, billboards and magazines. Celebrity endorsements are a big influence on children. Nothing can be more telling than endorsement of a fizzy drink by a cricketer or football legend. Most cultures also use sweets and food as inducements for good behaviour or rewards. This further reinforces the habit of HFSS food intake.

Some common food myths related to children are shown in **Table 1**.

5. Consequences of obesity in school children

At least 30% of obesity begins in childhood and 50–80% obese children become obese adults [17]. Obesity is linked to greater and earlier mortality. The GDB collaborator Group found that the lowest overall risk of death was in the BMI range of 20–25 kg/m² [5]. As in adult metabolic syndrome, hyperinsulinemia is a key to most of the complication of childhood obesity also. Some of the consequences of childhood obesity are enumerated in **Table 2**.

Physical	Psychological	Social
Respiratory and sleep problems	Low self-esteem, increased depressive symptoms and unhealthy dietary practices	Low participation in social activities
Insulin resistance		Lack of social support due to less interaction with peers
High blood pressure		
Dyslipidemia		
Musculoskeletal problems		
Gall stones		
Fatty liver		

Table 2.
Hazards of obesity in school children.

5.1 Hyperinsulinemia

There is an early leaning towards adult South-Asian phenotype in children, probably starting in neonatal period. Hyperinsulinemia and its attendant metabolic perturbations are more common in Asian neonates than in Caucasian ones [56]. Kuriyan and colleagues studied the waist circumference of Indian urban middle-class children between the ages of 6–16 years and found that their waist circumference was more than that of age- and sex-matched British children [57]. About one-third of the urban-Asian children have insulin resistance. The odds for hyperinsulinemia in one study were OR 4.7 (95% CI 2.4–9.4) in overweight children, OR 6.4 (95% CI 3.2–12.9) with high percentage body fat, OR 3.7 (95% CI 1.9–7.3) with high waist circumference, OR 6.8 (95% CI 3.3–13.9) with high waist hip ratio and OR 4.5 (95% CI 1.8–11.3) for sum of four skin-fold thicknesses (Sigma 4SF). Multiple logistic regression analysis showed that percentage body fat and Sigma 4SF were independent predictors of hyperinsulinemia with ORs being 3.2 and 4.5, respectively [58]. This being so, the risk of chubby children becoming obese adults is real.

5.2 Paediatric metabolic syndrome

In parallel with increasing prevalence of childhood obesity, the prevalence of paediatric metabolic syndrome, hypertension and type 2 diabetes mellitus are also increasing. Insulin resistance has been clearly been implicated in pathogenesis of hypertension, coronary artery disease and polycystic ovarian syndrome [59]. A study from Shimla in Himachal Pradesh showed prevalence of metabolic syndrome in school children to be 3.3% with odds of having metabolic syndrome being significantly linked to male sex, higher family monthly income, sedentary lifestyle and snacking in the evening [60]. The prevalence of metabolic syndrome in a study by Singh et al. in Chandigarh was 4.2%, and they did not find any difference between the sexes [61].

5.3 Childhood diabetes

The rise in prevalence of childhood obesity has mirrored the rise in prevalence of type 2 diabetes among children. For a long time, only type I diabetes was associated with childhood. The myth was shattered in 1979, when type 2 diabetes was described in children of Pima Indians. Type 2 diabetes accounts for 80% of childhood diabetes in Japan. The prevalence of type 2 diabetes in Japanese children rose from 0.2 to 7.3 per 100,000 children between 1976 and 1995 [62]. Ehtisham et al. observed that prevalence of type 2 diabetes in white UK children was significantly

less than that in South-Asian children (0.10/100,000 vs. 1.42/100,000, $p < 0.001$). According to them, the relative risk for type 2 diabetes in an Asian child compared to a white child in UK was 13.7-fold. Family history of diabetes, prevalence of obesity in the family, female preponderance and pubertal onset were some of the other significant associations that they noted apart from the ethnic clustering [63]. Ripamonti et al. found the prevalence of impaired glucose tolerance (IGT) to be 11% among the 398 obese Italian children studied [64]. Wabitsch et al. studied 520 obese children in Germany, and noted the prevalence of IGT and type 2 DM to be 2.1 and 1.5%, respectively [65]. Vijayalakshmi Bhatia observed that type 2 DM accounted for 10% of diabetes detected in children between the ages of 10–18 [66]. A higher BMI, and increased truncal and abdominal fat were important determinants of hyperglycaemia and insulin resistance [67, 68]. The GDB Collaborators Group found diabetes to be the second commonest cause of BMI-related deaths and DALY. It accounted for 9.5% of all deaths at a BMI of $\geq 30 \text{ kg/m}^2$ and 4.5% occurred at a BMI of $< 30 \text{ kg/m}^2$ [5].

5.4 Dyslipidaemia

Nearly 17% of obese adolescent American youth have abnormal non-HDL-cholesterol. Class III obesity is associated with high total cholesterol (19%), low HDL-cholesterol ($\leq 19\%$) and high triglycerides (29%) [69]. The commonly observed pattern of dyslipidaemia in obesity is one with increased triglycerides (TG), decreased HDL-cholesterol (HDL-C) and high normal or mildly raised LDL-cholesterol (LDL-C). The NHANES data indicate that in the US 42.9% of children with BMI > 95 th percentile have this pattern of dyslipidaemia [70]. Obesity is consistently associated with low HDL-cholesterol values [71]. In obese children and adolescents, there is a positive correlation between BMI and levels of VLDL and LDL-cholesterols [72]. The Princeton follow-up study conducted family lipid surveys between 1973 and 1978. Of all the children surveyed, 808 agreed to be a part of a CVD survey when contacted in 1998 and 19 live subjects reported CVD events. These events occurred at a mean age of 37.1 ± 4.9 years. BMI and TG levels in childhood correlated significantly with occurrence of CVD events ($p = 0.012$ and 0.0001 , respectively) [73].

5.5 Hypertension

Childhood obesity is associated with future development of hypertension. In fact, elevated BMI even in infancy is associated with high BP later [74, 75]. Risk of hypertension is two-fold with obesity and four-fold with severe obesity [76]. The prevalence of hypertension in children in the US varies from 3 to 5%, but it rises to about 25% in children with obesity [77]. Yadav et al. found the prevalence of pre-hypertension and hypertension to be 1.14 and 2.57%, respectively, among school children in the age group of 10–16 years in Kanpur, North India. Among overweight children 6.25% were pre-hypertensive and 12.5% were hypertensive and among obese children, the figures were 14.28% and 42.5%, respectively. The association in both cases was statistically significant [19]. Vedavathy and Sangamesh studied prevalence of hypertension in school children between the ages of 11–19 in Bangalore. Pre-hypertension and stage I hypertension were found in 3.6% of the students. The prevalence was significantly higher in children with BMI $> 23 \text{ kg/m}^2$. Family history of hypertension and obesity were significantly associated with pre-hypertension and stage I hypertension in the children ($p < 0.001$) [78]. An epidemiological survey of school children in Delhi found the prevalence of hypertension (systolic, diastolic or both) to be 11.9% in boys and 11.4% in girls.

There was a positive correlation between hypertension and BMI [79]. In another study from North India, Gupta et al. surveyed 3851 children between the ages of 5 and 15 and found that 292 were obese. The prevalence of hypertension was 0.34% among the obese children and 0.16% among the non-obese children [80]. In another study from Ludhiana, where 5000 children in the age group of 5–17 years were studied, the prevalence of hypertension in obese children was 3.5% and among normal ones 0.23% [81]. Hypertension in obese children results from an interplay of numerous factors which include, increased intravascular volume, increased cardiac output, increased sympathetic tone, increased steroid production, increased sodium intake and increased sodium retention and hyperinsulinemia. Among adolescents, hormonal changes and biological maturation are additional contributory factors [82, 83]. By the time hypertension is diagnosed in childhood, 20–40% have left ventricular hypertrophy. This correlates with high carotid intimal-media thickness (cIMT), which is a known predictor of heart-failure in childhood [77].

5.6 Cardiovascular disease

In relation to high BMI, cardiovascular disease (CVD) was the leading cause for death and disability-adjusted life-years (DALY). It accounted for 2.7 million deaths and 66.3 million DALY. Among the obese, 41% of deaths and 34% of DALY were due to CVD [5]. Current adolescent overweight prevalence is estimated to increase future adult obesity by 5–15% by 2035, and this will result in an additional 100,000 CVD cases [84]. Cardiovascular disease associated with obesity is usually secondary to hypertension (already discussed above) and atherosclerosis. Atherogenesis is mainly due to sub-intimal deposition of LDL-cholesterol particles. The combined atherogenicity of childhood obesity is an ideal scenario for this to occur. High circulating levels of small LDL-cholesterol particles and decreased clearance of the same by LDL receptors increases risk of their entrapment in the sub endothelial matrix. Further, low levels of HDL-cholesterol limit reversal of cholesterol transport. An objective measure of the damaging potential of atherogenicity of combined dyslipidaemia in childhood is the carotid intimal-media thickness (cIMT). The Young Finns study followed up children from childhood for 21 years. Children with combined dyslipidaemia had significant cIMT compared to normo-lipidaemic controls even after adjusting for other factors [85]. The Childhood Cardiovascular Cohort (i3C) was a 23.4 years follow-up of 2893 children aged 12–18 years across three continents. i3C data showed a strong correlation between childhood obesity, hypertension and dyslipidaemia with high cIMT in adulthood. Obesity increased the risk for high cIMT 3.7 (2.0–7.0)-fold and hypertension by 1.9 (1.3–2.9)-fold [86]. Another study showed that 90% of children with high cIMT also had left ventricular hypertrophy [87]. Obesity has been associated with a 50% increase in heart-failure incidence among young adults (18–34 years) from 1987 to 2006 in the US [88]. The frequency of stroke and renal failure are also higher in young adults with history of childhood obesity [89, 90]. In a recent study published in JAMA, positive associations were observed between consumption of artificially sweetened soft drinks and death due to circulatory diseases (>2 drinks/day vs. <1 glass per month; HR 1.52; 95%CI 1.30–1.78, $p = 0.001$) [91].

Sub-clinical vascular inflammation may also be a contributory factor to development of CVD. In Indian adolescents, raised C-reactive protein (CRP) levels are seen in 13% of all subjects, 22% of overweight and 25% of obese ones. CRP levels have a strong association with percentage body fat, WHR, waist circumference and triceps skin-fold thickness [92].

5.7 Non-alcoholic fatty liver disease (NAFLD)

Non-alcoholic disease is a component of metabolic syndrome. It is being increasingly recognised since abdominal ultrasonography has become a common diagnostic tool. The overall prevalence of NAFLD in children is about 10%, which includes a prevalence of 17% in teenagers and 40–70% in obese children. Steatosis *per se* is benign and self-limiting but it can progress to non-alcoholic steatohepatitis (NASH) in 3–5% of patients [93]. In the US, about 12–24% of the children are obese and 10–25% of these have elevated serum transaminases. That means that 1–4% of children, in the US, are obese and have deranged transaminases and are at risk for NAFLD [94]. A more accurate estimation comes from an autopsy study done in San Diego. Steatosis was found in 9.6% of children between 2 and 19 years of age and in 38% of obese children autopsied between 1993 and 2003 [95]. Prevalence of NAFLD in obese children in China is estimated to be between 20 and 77% [96]. The latest theory on pathogenesis of NAFLD is a ‘multi-hit’ one where there is an interplay of numerous factors like hepatic fat accumulation, insulin resistance, oxidative stress due to genetic and epigenetic factors, unfavourable lifestyles, gut microbiota, gut-liver axis dysfunction, and trace element deficiency and fluctuations [97, 98]. The best predictors for NASH in obese individuals with no evidence of other liver disease are elevations of AST or ALT to >200 IU/L, or any elevation of AST (>46 IU/L) and ALT (>35 IU/L) for greater than 6 months. These criteria identified 100% cases of NASH in a small study [99]. If detected early, hepatic steatosis is reversible. Weight loss is the most effective treatment. A 10% reduction in weight can normalise AST, ALT values and decrease ultrasound evidence of fatty liver on 30 months follow-up [100]. Compliance to weight loss and lifestyle changes is poor, especially in children. A number of medical and surgical therapies are emerging. Medical approaches include micronutrient supplementation (Vit E, Vit D, PUFAs, choline), probiotics, anti-obesity medication, metformin, cysteamine, antioxidants, obeticholic acid, growth hormone, lipid-lowering agents and hormones like adiponectin, resistin and TNF alpha. Surgical approach is mainly bariatric surgery [93]. These therapeutic measures could serve as boosters for the weight loss and lifestyle programmes which are difficult to sustain on their own.

5.8 Other disorders

5.8.1 Endocrine

Adipocytes secrete hormones which have paracrine and exocrine actions. The common abnormalities noted include increased production of steroid hormones, decreased progesterone secretion in females and decreased testosterone secretion in males. There is also a strong association between obesity and occurrence of polycystic ovarian syndrome. A study from Bombay looked at the prevalence of PCOS among 600 girls in the age group of 15–19 years from under privileged backgrounds. The prevalence of PCOS by Rotterdam criteria was 22.5%. PCOS was more common in obese than non-obese girls ($p = 0.002$) [101]. In comparison to Caucasians, Asian girls develop PCOS at an earlier age, are more symptomatic and have more fasting hyperinsulinemia and lower insulin sensitivity [102].

5.8.2 Orthopaedic

The most serious orthopaedic disability associated with childhood obesity is slipped capital epiphysis of the femur. Blount disease is another disorder that has

a strong association. About 50–80% of children with Blount disease are obese. Minor problems like flat feet, knock knees and frequent ankle sprains are also more common [83].

5.8.3 Psychosocial

Obesity is described as ‘one of the most stigmatising and least socially acceptable conditions of childhood’. [103]. Not surprisingly, low self-esteem and poor body image adversely impacting scholastic performance are common in obese children. They are bullied and excluded from group activities, especially sports and games, because they are slower and less agile. They get marginalised and withdraw from society. This makes them more sedentary and also predisposes them to eating disorders which further worsen their obesity [104].

6. Interventions to tackle obesity in school children

The plateauing of BMI of children and adolescents in high-income countries may be due specific initiatives by governments, community groups, schools and individuals and increased public awareness about the hazards of childhood obesity. Findings from the i3C study identified obesity, hypertension and dyslipidaemia as the three key modifiable factors in childhood that result in high cIMT in adulthood. All can be addressed effectively by lifestyle modification and medication. Obesity is easily apparent but difficult to treat on a sustained basis due to a variety of reasons. Hypertension and dyslipidaemia are more difficult to identify due to confusing cut-offs and multiple guidelines. Lifestyle modification is easy to suggest but difficult to implement. Care-givers and parents lack sufficient knowledge about dietary modifications and exercise programmes to advise children. Depression, low self-esteem, lack of peer and parental support and rebellious nature of adolescents make implementation of lifestyle changes difficult. Thus, a multi-modal approach to childhood obesity is required. Even modest loss of weight can lead to significant reduction in TG levels and increase in HDL-C levels. Even without weight loss, regular exercise training can have the same effect on serum lipids [105].

The WHO has included tackling childhood obesity as one of its priority areas. It has suggested a three-pronged strategy: reducing the risk of obesity by addressing critical elements in the life course, tackling obesogenic environment and norms and lastly treating obese children to improve their current and future health.

There is enough evidence to support the fact that undernutrition during pregnancy, excessive weight gain during pregnancy, hyperglycaemia during pregnancy and smoking and exposure to other toxins increase the risk of obesity in the child during infancy and childhood. Even obesity in fathers can increase risk of obesity in the child through epigenetic factors. Ensuring good health during pregnancy and a safe delivery are both important for the child’s health. The WHO guidelines include dietary advice to prospective parents before conception and during pregnancy, avoidance of smoking, alcohol and other toxins. Early detection of gestational diabetes and hypertension, monitoring and managing gestational weight gain are other important measures suggested. Ensuring that the baby is exclusively breast-fed during the first 6 months of life and that breast feeding is continued even after complementary feeding is started are important to prevent malnutrition and deposition of excess fat in the baby. The magnitude of the gap between policy and implementation can be gauged from the Indian NFHS 2015–2016 data which show that despite recommendations for exclusive breast feeding up to an age of 6 months, only 55% of Indian infants were actually

exclusively breast-fed. Many children in this age group were given other foods like plain water (18%), other milk (11%) and complementary foods (10%). Breast feeding can be encouraged by educational programmes for pregnant women, legislation and regulations to provide feeding rooms and time for breast feeding in work places, and by giving new mothers maternity leave. Nations should implement the International Code of Marketing of Breast-milk Substitutes and other World Health Assembly resolutions to promote breast feeding. During infancy and childhood, parents and care-givers should be given guidance and support to encourage consumption of a variety of healthy foods, avoidance of sugar-sweetened milks and juices and energy-dense nutrient-poor foods. Enforcing regulations on marketing of complementary foods and beverages and subsidising healthy foods are other measures [5, 12].

Further, there should be government regulations on the types of foods and beverages that can be served in school meals. Provision of safe potable drinking water in schools is another basic requirement. Teaching children and parents about healthy eating and having an active lifestyle should be an integral part of school curriculum at all stages. Cookery class in schools is another way of teaching both children and parents about healthy food options. Making time in school schedules for play-time, encouraging sports by providing space and facilities both at schools and in communities will enable children to be physically active. A mandatory physical education programme is another good option. The government's role also includes enforcing ban of sale of HFSS foods and sweetened drinks in schools and around school premises.

Regular health check-ups and growth monitoring should also be an integral part of school life. Children who are overweight or obese should have easy access to treatment including psychotherapy, medications for hypertension, diabetes and dyslipidaemia and even bariatric surgery in extreme cases. Treatment of children should include parents or care-givers. This will provide the much needed emotional and psychological support these children will require to lose weight and change their lifestyles. Multi-specialty teams are required for a successful child-health programme. Successful programmes to tackle childhood obesity usually involve parents, teachers, community members, non-governmental organisations and private players. Some of the more successful international programmes are listed below:

1. Focus on school health education and changing lifestyle:

- a. Project Healthy Schools: This is a project set up by University of Michigan in collaboration with middle schools, community organisations and donors. It aims to educate and encourage children to eat healthy and lead healthy lives. The programme comprises of 10 health education activity modules that focus on eating more fruits and vegetables, consuming less sweetened drinks and sugary foods, eating slowly and eating less fatty food, being more active every day and spending less time in front of the screen [106].

2. Promoting better dietary choices:

- a. Pick a tick initiative: This was started in 1989 by the Australian National Heart Foundation. Healthy food packets could be distinguished from others by presence of a symbol along with the nutrition panel. Apart from helping people select healthy foods, the programme also championed vigorously against excessive salt intake [107].

b. Ensemble-prévenons-l'obésité-des-enfants (EPODE): This literally translates as: 'Together Let's Prevent Childhood Obesity'. This was a community initiative in which intervention were done in 10 French towns in France for children aged 5–12 who were overweight or at risk of weight gain. The approach was 'positive, concrete and stepwise' learning process with no stigmatisation of any culture, food habits, overweight and obesity. It targeted sales of healthy foods in schools, advertisements with respect to food and drinks on TV, internet and schools, mandatory nutritional information on nutrition label, subsidy on healthy foods through agricultural reforms and training for health professionals so that they were able to recognise and diagnose obesity risks in infancy, childhood and adolescence [108].

c. Healthy weight, Healthy lives: An initiative in England to tackle childhood obesity and hence later reduce obesity in adults. It was launched by the government in 2008. The various initiatives in this programme were promoting healthy growth and development of children, promoting healthier food choices, encouraging a more active lifestyle, incentivising good health and offering personalised help and support [109].

3. Promoting a more active lifestyle:

a. US White House Task Force on childhood obesity: This task force gave support to parents, adherence to limits on screen time and quality child care settings with nutritious food and ample opportunity for young children to be physically active. It also emphasised labels on food so that parents can make healthy food choices, improved school environment and lowered price of healthy foods. It improved access to safe parks, playgrounds and indoor and outdoor recreational facilities for children.

b. Tri-Policy initiative: This was done in Canada by creating a supportive environment to increase physical activity in children, early action to detect risk of overweight and obesity and promote availability and accessibility of nutritious foods and decrease the marketing of foods and beverages high in fat, sugar and/or sodium to children.

4. Government policy initiatives

a. New South Wales initiative in Australia: It focused on restoring the energy balance for the population, with a specific focus on children, young people, and their families by reducing the factors that give rise to an 'obesogenic' environment. It emphasised coordination between governments initiatives to tackle obesity along with industry and non-profit organisations.

b. Regulations regarding food advertisements: Brazil prohibits advertisements which are intended to influence children or adolescents to consume HFSS foods. Ireland has strictly banned using celebrities, icons and personalities to promote food products which target children. Norway prevents food advertisements on channels for children under 18 and South TV permits advertising for specific food categories only before, during and after programmes shown between 5 and 7 pm.

6.1 Actions taken in India to reduce childhood obesity in school children

Food Safety and Standards Authority of India (FSSAI) proposed a ban on sale of HFSS foods in school canteens in 2016. It also suggested categorisation of food items as 'green' or healthy foods constituting 80% of the food items, 'red' or common HFSS foods that should not be made available in schools; and 'yellow' category foods that should be eaten sparingly and could be made available in small portions and less frequently. In response to this, Maharashtra state government issued a notification instructing schools to stop serving HFSS food in their canteens. However, the same has not been implemented in other states. The Central Board of Secondary Education (CBSE) has also issued an advisory to all its affiliated schools to ensure that no HFSS food items are available in school canteens and within 20 m of their premises.

Ministry of Health and Family Welfare launched the School Health Programme in 2008 under the National Rural Health Mission (NRHM). The programme also aims to address physical and mental health needs of the children through nutritional interventions, yoga and counselling.

Food Safety and Standards Act, 2006 has provisions to prohibit advertisements that are misleading. These are monitored by the Advertisement Standards Council of India (ASCI). However, the advisories on misleading advertisements are not being strictly implemented. Kaushal et al. reported that the prevalence of misleading advertisements is about 60% [110]. A majority (90%) of these were for HFSS foods. The common methods of non-compliance included promotion of a food item with free gifts (57%), using celebrity endorsement on the food packaging (19%), making false claims (14%) and appealing with cartoons (10%). Apart from restrictions on food advertisements, we also need to strengthen nutrition labelling laws, and educate both parents and children about interpreting nutrient labelling. In India, food packages already have a labelling for vegetarian and non-vegetarian products. HFSS could also be labelled using colour codes as suggested above.

Family and parental guidance play an important role in preventing children from becoming obese. Family involvement can be pivotal in increasing physical activity, healthy eating patterns and decreasing sedentary time. Educating families and stakeholders about healthy eating pattern will help in cutting down on junk food in home and school, and increase healthy eating, thereby decreasing the risk of developing obesity.

Community level programs like CHETNA (Childrens' Health Education Through Nutrition and Health Awareness) and MARG (Medical education for children/adolescents for realistic prevention of obesity and diabetes and for healthy living) are a beginning. These programmes focus on building nutritional awareness and promoting increased physical activity, through pamphlets, lectures skits and group activities targeting both parents and children. They now cover 500,000 children in 15 towns/cities in North India [15]. Since children spend a significant amount of their day in schools, activities involving their peers in schools are smart ways of getting them interested. The government has recently introduced fitness test for all school children and compulsory physical activity during school hours. A 'Fit India' campaign has been announced by the prime-minister and a number of sports and film celebrities are part of the campaign. But much more needs to be done.

7. Conclusion

Obesity in Indian school children is a cause of concern. To tackle this menace, a sustained multi-pronged approach is required, where all stake holders join hands.

The strategy starts at the pre-conception time, continues during pregnancy, infancy and childhood. Apart from promoting healthy eating and an active lifestyle, it also includes active case finding among overweight and obese children and aggressive management of diabetes, hypertension and dyslipidaemia apart from weight loss. Legislations targeting infant feeds, HFSS, artificially sweetened soft drinks, nutrient labelling and food advertisements are important. Government have to be serious about implementing these legislations and they should also formulate imaginative programmes to target childhood nutrition and lifestyle.

IntechOpen

IntechOpen

Author details

Vangal Krishnaswamy Sashindran^{1*} and Puja Dudeja²

1 Principal Medical Officer, HQ Central Air Command, Prayagraj, India

2 Colonel (Pensions), Office of Director General Armed Forces, New Delhi, India

*Address all correspondence to: vksashindran@gmail.com

IntechOpen

© 2020 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. 

References

- [1] Ogden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index in US children and adolescents, 2007-2008. *JAMA*. 2010;**303**:242-249
- [2] Ranjani H, Mehreen TS, Pradeepa R, Anjana RM, Garg R, Anand K, et al. Epidemiology of childhood overweight & obesity in India: A systematic review. *The Indian Journal of Medical Research*. 2016;**143**(2):160
- [3] NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: A pooled analysis of 2416 population-based measurements studies in 128.9 million children, adolescents, and adults. *The Lancet*. 2017;**390**:2627-2642
- [4] Report of the Commission on ending childhood obesity. World Health Organization, Geneva. 2016
- [5] The GBD. Obesity collaborators. Health effects of overweight and obesity in 195 countries over 25 years. *The New England Journal of Medicine*. 2015, 2017;**377**:13-27
- [6] NCD Risk Factor Collaboration (NCD-Ris C). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: A pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. *Lancet*. 2017;**390**:2627-2642
- [7] World Health Organisation, World Obesity Federation (2018). Taking Action on Childhood Obesity WHO
- [8] Taveras EM, Gillman MW, Kleinman K, Rich-Edwards JW, Rifas-Shiman SL. Racial/ethnic differences in early-life risk factors for childhood obesity. *Pediatrics*. 2010;**125**:686-695
- [9] Pengpid S, Peltzer K. Overweight, obesity and associated factors among 13-15 years old students in the Association of South-East Nations member countries, 2007-2014. *The Southeast Asian Journal of Tropical Medicine and Public Health*. 2016;**47**(2):250-262
- [10] Peltzer K, Pengpid S. Leisure time physical inactivity and sedentary behaviour and lifestyle correlates among students aged 13-15 in the association of Southeast Asian nations (ASEAN) member states, 2007-2013. *International Journal of Environmental Research and Public Health*. 2016;**13**(2):217
- [11] Pengpid S, Peltzer K. Overweight and obesity and associated factors among school-aged adolescents in six Pacific island countries in Oceania. *International Journal of Environmental Research and Public Health*. 2015;**12**:14505-14518. DOI: 10.3390/ijerph12114505
- [12] National Family Health Survey-4(2015-16). International Institute of Population Sciences. Mumbai
- [13] Gupta DK, Shah P, Misra A, Bharadwaj S, Gulati S, Gupta N, et al. Secular trends in prevalence of overweight and obesity from 2006 to 2009 in urban asian Indian adolescents aged 14-17 years. *PLoS One*. 2011;**6**(2):e17221
- [14] Lobstein T, Jackson-Leach R. Planning for the worst: Estimates of obesity and comorbidities in school-age children in 2025. *Pediatric Obesity*. 2016;**11**(5):321-325
- [15] Bharadwaj S, Misra A, Khurana L, Gulati S, Shah P, Vikram NK. Childhood

obesity in Asian Indians: A burgeoning cause of insulin resistance, diabetes and sub-clinical inflammation. *Asia Pacific Journal of Clinical Nutrition*. 2008;**17**(S1):172-175

[16] Gautam S, Jeong H. Childhood obesity and its associated factors among school children in Udipi, Karnataka, India. *Journal of Lifestyle Medicine*. 2019;**9**(1):27-35

[17] Bharati DR, Deshmukh PR, Garg BS. Correlates of overweight and obesity among school going children of Wardha city, Central India. *The Indian Journal of Medical Research*. 2008;**127**:539-543

[18] Gamit SS, Moitra M, Verma MR. Prevalence of obesity and overweight in school going adolescents of Surat city, Gujarat, India. *International Journal of Medical Science and Public Health*. 2015;**4**(1):42-47

[19] Yadav KS, Yadav MB, Yadav C. Prevalence of overweight, obesity and hypertension among school going children in district Kanpur, Uttar Pradesh, India: A longitudinal study. *International Journal of Contemporary Pediatrics*. 2019;**6**(1):159-162

[20] Gupta N, Goel K, Shah P, Misra A. Childhood obesity in developing countries: Epidemiology, determinants, and prevention. *Endocrine Reviews*. 2012;**33**(1):48-70

[21] Khadilkar VV, Khadilkar AV. Revised Indian academy of pediatrics 2015 growth charts for height, weight and body mass index for 5-18-year-old Indian children. *Indian Journal of Endocrinology and Metabolism*. 2015;**19**(4):470

[22] Pillai RN. Modelling Studies for a 'Whole of Society (wos)' Framework to Monitor Cardio-Metabolic Risk Among Children (6 to 18 years). Ph.D. Dissertation. Università Degli Studi Di Verona. 2018. Available from:

http://www.inclentrust.org/inclen/wp-content/uploads/6_D2_Rakesh-Pillai_Thesis_03_Mar_18.pdf [Accessed: 10 June 2019]

[23] Anderson PM, Butcher KE. Childhood obesity: Trends and potential causes. *The Future of Children*. 2006;**16**:19-45

[24] Ng M, Fleming T, Robinson M, Thomson B, Graetz N, Margono C, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980-2013: A systematic analysis for the global burden of disease study 2013. *Lancet*. 2014;**384**:766-781

[25] Ramachandran A, Snehalatha C, Vinitha R, Thayyil M, Kumar CK, Sheeba L, et al. Prevalence of overweight in urban Indian adolescent school children. *Diabetes Research and Clinical Practice*. 2002;**57**(3):185-190

[26] Rao S, Kanade A, Kelkar R. Blood pressure among overweight adolescents from urban school children in Pune, India. *European Journal of Clinical Nutrition*. 2007;**61**:633-641

[27] Kapil U, Pathak P, Singh P, Dwivedi SN, Bhasin S. Prevalence of obesity among affluent adolescent school children in Delhi. *Indian Pediatrics*. 2002;**39**(5):449-452

[28] Chhatwal J, Vema M, Riar SK. Obesity among pre-adolescent and adolescents of a developing country (India). *Asia Pacific Journal of Clinical Nutrition*. 2004;**13**(3):231-235

[29] Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *The New England Journal of Medicine*. 1997;**337**(13):869-873

[30] Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends

in overweight among US children and adolescents 1999-2000. *JAMA*. 2002;**288**:1728-1732

[31] Gulati S, Misra A, Colles SL, Kondal D, Gupta N, Goel K, et al. Dietary intakes and familial correlates of overweight/obesity: A four-cities study in India. *Annals of Nutrition and Metabolism*. 2013;**62**(4):279-290. [PubMed: 23689065]

[32] Center for Disease Control and Prevention. Contributing Factors. 2010. Available from: http://www.cdc.gov//obesity/childhood/contributing_factors.html. [Accessed: 01 July 2014]

[33] Chapman G, Maclean H. "Junk food" and "healthy food": Meanings of food in adolescent women's culture. *Journal of Nutrition Education and Behavior*. 1993;**25**:108-113

[34] Vucetic Z, Reyes TM. Central dopaminergic circuitry controlling food intake and reward: Implications for the regulation of obesity. *Wiley Interdisciplinary Reviews: Systems Biology and Medicine*. 2010;**2**:577-594

[35] Stice E, Yoket S, Bohon C, Martin N, Stolen A. Reward circuitry responsively to food predicts future increases in body mass: Moderating effects of DRD2 and DRD4. *NeuroImage*. 2010;**50**(4):1618-1625

[36] Stice E, Spoor S, Ng J, Zald DH. Relation of obesity to consummatory and anticipatory food reward. *Physiology & Behaviour*. 2009;**97**(5):551-560

[37] Bosch LSMM, Wells JCK, Lum S, Reid AM. Associations of extracurricular physical activity patterns and body composition components in multi-ethnic population of UK children (the size and lung function in children study): A multi-level modelling analysis. *BMC Public Health*. 2019;**19**:573

[38] Kunwar R, Minhas S, Mangla V. Is obesity a problem among school children? *Indian Journal of Public Health*. 2018;**62**:153-155

[39] Govindan M, Gurm R, Mohan S, et al. Gender differences in physiologic markers and health behaviors associated with childhood obesity. *Pediatrics*. 2013;**132**:468-474

[40] National Institutes of Health; US National Library of Medicine. Screen Time and Children. Available at: <https://medlineplus.gov/ency/patientinstructions/000355.htm> [Accessed: 22 June 2016]

[41] Vuong B, Rogers R, Corriveau N, et al. Passive screen time associated with unhealthy dietary consumption and physiological characteristics: A closer look at childhood behaviors. *Journal of the American College of Cardiology*. 2014;**63**:A1292

[42] Story M, Neumark-stainzer D, French S. Individual and environmental influences on adolescent eating behaviours. *Journal of the American Dietetic Association*. 2002;**102**:S40-S51. [PubMed: 11902388]

[43] Britz B, Siegfried W, Ziegler A, Lamertz C, Herpertz-Dahlmann BM, Remschmidt H, et al. Rates of psychiatric disorders in a clinical study group of adolescents with extreme obesity and in obese adolescents ascertained via a population based study. *International Journal of Obesity and Related Metabolic Disorders*. 2000;**24**:1707-1714. [PubMed: 11126229]

[44] Rawana JS, Morgan AS, Nguyen H, Craig SG. The relation between eating- and weight-related disturbances and depression in adolescence: A review. *Clinical Child and Family Psychology Review*. 2010;**13**:213-230. [PubMed: 20632207]

- [45] Ravelli GP, Stein ZA, Susser MW. Obesity in young men after famine exposure in utero and early infancy. *The New England Journal of Medicine*. 1976;**295**:349-353
- [46] Bateson P, Gluckman P, Hanson M. The biology of developmental plasticity and the predictive adaptive response hypothesis. *Journal of Physiology*. 2014;**592**(11):2357-2368. [PubMed: 24882817]
- [47] Yajnik CS. Obesity epidemic in India: Intrauterine origins? *Proceedings of the Nutrition Society*. 2004;**63**:387-396
- [48] Kuriyan R, Rodgers N, Thomas T, Aravind J, Subramanian SV, Kurpad AV. The role of familial and sibling factors on abdominal adiposity: A study of south Indian urban children. *Asia Pacific Journal of Clinical Nutrition*. 2018;**27**(4):869-874
- [49] Gupta S, Kapoor S. Gender differences in familial aggregation of adiposity traits in Aggarwal Baniya families. *Eurasian Journal of Anthropology*. 2011;**2**(2):85-95
- [50] Raskind IG, Patil's SS, Haardorfer R, Cunningham SA. Unhealthy weight in Indian families: The role of the family environment in the context of the nutrition transition. *Population Research and Policy Review*. 2018;**37**(2):157-180
- [51] Sahoo K, Sahoo B, Choudhury AK, Sofi NY, Kumar R, Bhadoria AS. Childhood obesity: Causes and consequences. *Journal of Family Medicine and Primary Care*. 2015;**4**(2):187-192
- [52] Moens E, Braet C, Bosmans G, Rosseel Y. Unfavourable family characteristics and their associations with childhood obesity: A cross-sectional study. *European Eating Disorders Review*. 2009;**17**:315-323. [PubMed: 19452495]
- [53] Jackson EA, Eagle T, Leidal A, et al. Childhood obesity: A comparison of health habits of middle-school students from two communities. *Clinical Epidemiology*. 2009;**1**:133-139
- [54] Eagle TF, Sheetz A, Gurm R, Woodward AC, Kline-Rogers E, Leibowitz R, et al. Understanding childhood obesity in America: Linkages between household income, community resources, and children's behaviors. *American Heart Journal*. 2012;**163**:836-843
- [55] Kaur S, Sachdev HP, Dwivedi SN, Lakshmy R, Kapil U. Prevalence of overweight and obesity amongst school children in Delhi, India. *Asia Pacific Journal of Clinical Nutrition*. 2008;**17**(4):592-596
- [56] Yajnik CS, Lubree HG, Rege SS, Naik SS, Deshpande JA, Deshpande SS, et al. Adiposity and hyperinsulinemia in Indians are present at birth. *The Journal of Clinical Endocrinology and Metabolism*. 2002;**87**:5575-5580
- [57] Kuriyan R, Thomas T, Lokesh DP, Sheth NR, Mahendra A, Joy R, et al. Waist circumference and waist for height percentiles in urban south Indian children aged 3-16 years. *Indian Pediatrics*. 2011;**48**:765-771
- [58] Misra A, Vikram NK, Arya S, Pandey RM, Dhingra V, Chatterjee A, et al. High prevalence of insulin resistance in post-pubertal Asian Indian children is associated with adverse truncal body fat patterning, abdominal adiposity and excess body fat. *International Journal of Obesity and Related Metabolic Disorders*. 2004;**28**:1217-1226
- [59] World Health Organization, Department of Non-Communicable Disease Surveillance. Definition,

Diagnosis and Classification of Diabetes Mellitus and its Complications. Report of a WHO Consultation. Part 1: Diagnosis and Classification of Diabetes Mellitus. Geneva: World Health Organization. 1999

[60] Gupta A, Sachdeva A, Mahajan N, Gupta A, Sareen N, Pandey RM, et al. Prevalence of paediatric metabolic syndrome and associated risk factors among school-age children of 10-16 years living in district Shimla, Himachal Pradesh, India. *Indian Journal of Endocrinology and Metabolism*. 2018;**22**(3):373-378

[61] Singh R, Bhansali A, Sialy R, Aggarwal A. Prevalence of metabolic syndrome in adolescents from a north Indian population. *Diabetic Medicine*. 2007;**24**(2):195-199

[62] Kitagawa T, Owada M, Urakami T, et al. Increased incidence of non-insulin dependent diabetes mellitus among Japanese schoolchildren correlates with an increased intake of animal protein and fat. *Clinical Pediatrics (Phila)*. 1998;**37**:111-115

[63] Ehtisham S, Hattersley AT, Dunger DB, Barrett TG. First UK survey of paediatric type 2 diabetes and MODY. *Archives of Disease in Childhood*. 2004;**89**:526-529

[64] Ripamonti G, De Medici C, Guzzaloni G, Morn G, Ardizzi A, Morabito F. Impaired glucose tolerance in obesity in children and adolescents. *Journal of Physiology and Biochemistry*. 2003;**59**:217-223

[65] Wabitsch M, Hauner H, Hertramf M, Muche R, Hay B, et al. Type II diabetes mellitus and impaired glucose regulation in Caucasian children and adolescents with obesity living in Germany. *International Journal of Obesity and Related Metabolic Disorders*. 2004;**28**:307-313

[66] Bhatia V, National Task Force IAP. Childhood prevention of adult diseases: Insulin resistance and type 2 diabetes mellitus in childhood. *Indian Pediatrics*. 2004;**41**:443-457

[67] Abate N. Insulin resistance and obesity. The role of fat distribution. *Diabetes Care*. 1996;**19**:292-294

[68] Lundgren H. Adiposity and adipose tissue distribution in relation to incidence of diabetes in women. Results from prospective population study in Gothenburg, Sweden. *International Journal of Obesity*. 1989;**13**:413-423

[69] Skinner AC, Perrin EM, Moss LA, Skelton JA. Cardiometabolic risks and severity of obesity in children and young adults. *The New England Journal of Medicine*. 2015;**373**:1307-1317

[70] Centers for Disease Control and Prevention. Prevalence of abnormal lipid levels among youths --- United States, 1999-2006. *MMWR. Morbidity and Mortality Weekly Report*. 2010;**59**:29-33

[71] Anderson KM, Wilson PW, Garrison RJ, Castelli WP. Longitudinal and secular trends in lipoprotein cholesterol measurements in general population sample. The Framingham offspring study. *Atherosclerosis*. 1987;**68**:59-66

[72] Guida F, Guzzaloni G, Moreni G, Morabito F. Lipid profile, adiposity in children and adolescents. *Minerva Pediatrica*. 1989;**41**:449-458

[73] Morrison JA, Glueck CJ, Woo JG, Wang P. Risk factors for cardiovascular disease and type 2 diabetes retained from childhood to adulthood predict adult outcomes: The Princeton LRC follow up study. *International Journal of Pediatric Endocrinology*. 2012;**2012**(1):6-14

- [74] Falkner B, Gidding SS, Ramirez-Garnica G, Wiltrout SA, West D, Rappaport EB. The relationship of body mass index and blood pressure in primary care pediatric patients. *The Journal of Pediatrics*. 2006;**148**(2):195-200 PMID:16492428
- [75] Perng W, Rifas-Shiman SL, Kramer MS, et al. Early weight gain, linear growth, and mid-childhood blood pressure: A prospective study in project viva. *Hypertension*. 2016;**67**(2):301-308 PMID:26644238
- [76] Parker ED, Sinaiko AR, Kharbanda EO, et al. Change in weight status and development of hypertension. *Pediatrics*. 2016;**137**(3):e20151662 PMID:26908707
- [77] Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR, et al. Clinical practice guideline for screening and management of high blood pressure in children and adolescents. *Pediatrics*. 2017;**140**:e20171904
- [78] Vedavathy S, Sangamesh. Prevalence of hypertension in urban school going adolescents of Bangalore, India. *International Journal of Contemporary Pediatrics*. 2016 May;**3**(2):416-423
- [79] Laroia D, Sharma M, Dwivedi V. Profile of blood pressure in normal school children. *Indian Pediatrics*. 1989;**26**:531-536
- [80] Gupta AK, Ahmed AJ. Childhood obesity and hypertension. *Indian Pediatrics*. 1991;**28**:810-816
- [81] Anand NK, Tandon L. Prevalence of hypertension in children. *Indian Pediatrics*. 1994;**49**:1065-1069
- [82] Feld LG, Springale JE, Was WR. Special topics in pediatric hypertension. *Seminars in Nephrology*. 1998;**18**:295-303
- [83] Kaur S, Kapil U, Singh P. Pattern of chronic diseases amongst adolescent obese children in developing countries. *Current Science*. 2005;**88**(7):1052-1056
- [84] Bibbins-Domingo K, Coxson P, Pletcher MJ, Lightwood J, Goldman L. Adolescent overweight and future adult coronary heart disease. *The New England Journal of Medicine*. 2007;**357**:2371-2379
- [85] Juonala M, Viikari JS, Ronnema T, Marniemi J, Jula A, Loo BM, et al. Associations of dyslipidemias from childhood to adulthood with carotid intima-media thickness, elasticity, and brachial flow-mediated dilatation in adulthood: The cardiovascular risk in young Finns study. *Arteriosclerosis, Thrombosis, and Vascular Biology*. 2008;**28**(5):1012-1017
- [86] Koskinen J, Juonala M, Dwyer T, Venn A, Thomson R, Bazzano L, et al. Impact of lipid measurements in youth in addition to conventional clinic-based risk factors on predicting preclinical atherosclerosis in adulthood: International childhood cardiovascular cohort consortium. *Circulation*. 2018;**137**:1246-1255. DOI: 10.1161/CIRCULATIONAHA.117.029726
- [87] Sorof JM, Alexandrov AV, Cardwell G, Portman RJ. Carotid artery intimal- medial thickness and left ventricular hypertrophy in children with elevated blood pressure. *Pediatrics*. 2003;**111**:61-66
- [88] Barasa A, Schaufelberger M, Lappas G, Swedberg K, Dellborg M, Rosengren A. Heart failure in young adults: 20-year trends in hospitalization, aetiology, and case fatality in Sweden. *European Heart Journal*. 2014;**35**:25-32
- [89] Sultan S, Elkind MS. The growing problem of stroke among young adults. *Current Cardiology Reports*. 2013;**15**:421

- [90] Vivante A, Golan E, Tzur D, Leiba A, Tirosh A, Skorecki K, et al. Body mass index in 1.2 million adolescents and risk for end-stage renal disease. *Archives of Internal Medicine*. 2012;**172**:1644-1650 [PMID: 24068459]. DOI: 10.1007/s00431-013-2157-6
- [91] Mullee A, Romaguera D, Pearson-Stuttard J, Viallon V, Stephen M, et al. Association between soft drink consumption and mortality in 10 European countries. *JAMA Internal Medicine*. 2019;**03**:E1-E12. DOI: 10.1001/jamainternmed.2019.2478
- [92] Arya S, Isharwal S, Misra A, Pandey RM, Rastogi K, Vikram NK, et al. C-reactive protein and dietary nutrients in urban Asian Indian adolescents and young adults. *Nutrition*. 2006;**22**:865-871
- [93] Clemente MG, Mandato C, Poeta M, Vajro P. Pediatric non-alcoholic fatty liver disease: Recent solutions, unresolved issues, and future research directions. *World Journal of Gastroenterology*. 2016;**22**(36):8078-8093
- [94] Mathur P, Das MK, Arora NK. Non-alcoholic fatty liver disease and childhood obesity. *Indian Journal of Pediatrics*. 2007;**74**(4):401-407
- [95] Schwimmer JB, Deutsch R, Kahen T, Lavine JE, Stanley C, Behling C. Prevalence of fatty liver in children and adolescents. *Pediatrics*. 2006;**118**:1388-1393
- [96] Chan DF, Li AM, Chu WC, Chan MH, Wong EM, Liu EK, et al. Hepatic steatosis in obese Chinese children. *International Journal of Obesity and Related Metabolic Disorders*. 2004;**28**:1257-1263
- [97] Berardis S, Sokal E. Pediatric non-alcoholic fatty liver disease: An increasing public health issue. *European Journal of Pediatrics*. 2014;**173**:131-139
- [98] Goyal NP, Schwimmer JB, et al. *Clinics in Liver Disease*. 2016;**20**:325-338 [PMID: 27063272]. DOI: 10.1016/j.cld.2015.10.003
- [99] Lerret SM, Garcia-Rodriguez L, Skelton J, Biank V, Kilway D, Telega G. Predictors of non-alcoholic steatohepatitis in obese children. *Gastroenterology Nursing*. 2011;**34**(6):434-437
- [100] Vajro P, Fontanella A, Perna C, Orso G, Tedescom VAD. Persistent hyperaminotransferasemia resolving after weight reduction in obese children. *The Journal of Pediatrics*. 1994;**125**:239-241
- [101] Joshi B, Mukherjee S, Patil A, Purandare A, Chauhan S, Vaidya R. A cross-sectional study of polycystic ovarian syndrome among young adolescent and young girls in Mumbai, India. *Indian Journal of Endocrinology and Metabolism*. 2014;**18**(3):317-324
- [102] Wijeyaratne CN, Balen AH, Barth JH, Belchetz PE. Clinical manifestations and insulin resistance (IR) in polycystic ovary syndrome (PCOS) among south Asians and Caucasians: Is there a difference? *Clinical Endocrinology*. 2002;**57**:343-350
- [103] Schwimmer JB, Burwinkle TM, Varni JW. Health-related quality of life of severely obese children and adolescents. *JAMA*. 2003;**289**:1813-1819
- [104] Budd GM, Hayman LL. Addressing the childhood obesity crisis. *The American Journal of Maternal Child Nursing*. 2008;**33**:113-117. [PubMed: 18327110]
- [105] Cook S, Kavey R-EW. Dyslipidemia and pediatric obesity. *Pediatric Clinics of North America*. 2011;**58**(6):1363-1373

[106] Rogers R, Krallman R, Jackson EA, DuRussel-Weston J, Palma-Davis LV, de Visser R, et al. Top ten lessons learned from project healthy schools. *The American Journal of Medicine*. 2017;**130**:990.e1-990.e7

[107] Dalton CB. The National Heart Foundation Pick the tick program. *The Medical Journal of Australia*. 1993;**158**(8):577

[108] Borys JM, Richard P, Ruault du Plessis H, Harper P, Levy E. Tackling health inequalities and reducing obesity prevalence: The EPODE community approach. *Annals of Nutrition & Metabolism*. 2016;**68**(Suppl 2):35-38. DOI: 10.1159/000446223 Epub 2016 Jun 16

[109] Healthy Weight, Healthy Lives: A Cross-government strategy for England. *PublicationsPolicyAndGuidance/DH_082378*

[110] Kaushal N, Dudeja P. Food advertisements boon or bane: A prevalence study of misleading food advertisements in India. *Journal of Childhood Obesity*. 2017;**2**(4):17-19