Challenges for prevention of childhood obesity

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Global health scenario has seen a paradigm shift from communicable diseases to non-communicable diseases. Obesity is the entry point of non-communicable diseases. The etiology and management of these non-communicable diseases is entirely different from communicable diseases and the long term consequences are grave. The prevalence of childhood obesity has tripled since 1980, and data shows that almost one-third of children over 2 years of age are already overweight or obese. Obese children today are on track to have poor health throughout their adult lives. Obese children and adolescents are more likely to have risk factors associated with cardiovascular disease and diabetes, psychological problems, and orthopaedic problems than those who are not obese. Also overweight children and adolescents are more likely to become obese adults, with all the health problems that accompany obesity in adulthood. This accounts for a huge burden on health care costs. The fundamental reason for childhood obesity is an energy imbalance between the calories they consume and the calories they expend through activity. The important causes of this childhood obesity include an increased intake of energy dense foods that are high in fat and sugars alongside a trend toward decreased physical activity due to the sedentary nature of many forms of play, changing modes of transportation, and increasing urbanization. Other factors like environmental and social framework also play a role. Therefore a comprehensive approach is needed to tackle the problem and overcome the challenges for prevention of childhood obesity.

Epidemiology

Childhood obesity is a global epidemic and is one of the leading public health problems of this century.[1] The problem is steadily growing in urban settings in most of the low- and middle-income countries. The prevalence has increased at tremendously. Worldwide, it was estimated that in 2010, the population of overweight children under the age of five was over 42 million. Of these, around 35 million belonged to developing countries.[1] Global prevalence of childhood overweight and obesity increased from 4.2% (95% CI: 3.2%, 5.2%) in 1990 to 6.7% (95% CI: 5.6%, 7.7%) in 2010. By 2020, this trend is expected to reach 9.1% (95% CI: 7.3%, 10.9%), or approximately 60 million. Prevalence of childhood overweight and obesity in Africa is expected to reach 12.7% by 2020. The prevalence is lower in Asia, 4.9% in 2010, but the number of affected children is higher (18 million).[2] According to a meta-analysis, the prevalence of overweight was estimated to be 12.64% and that of obesity to be 3.39% in India.[3]

Overweight and obesity are classified using age and sex specific normograms for body mass index (BMI) for children and adolescents. According to CDC, children with BMI equal to or more than the age-gender-specific 95th percentile are classified as obese. Children with BMI more than or equal to the 85th but below 95th percentiles are classified as overweight and are at risk for obesity related complications.[4] In WHO growth reference charts for children and adolescents, overweight is classified as greater than one standard deviation body mass index for age and sex, and obesity as greater than two standard deviations body mass index for age and sex.[1]

Overweight and obese children are likely to continue to be obese in adulthood and more likely to develop noncommunicable diseases like diabetes and cardiovascular diseases at an earlier age. This will lead to a heavy financial burden. Prevention of childhood obesity is therefore a major public health concern. In the 66th World Health Assembly, the WHO member states have decided upon a voluntary global NCD target to stop the rise in diabetes and obesity.[1]

Consequences of childhood obesity

Medical science had proudly waged war against communicable diseases and conquered some of the infectious diseases by producing vaccines and antibiotics until it was faced with the epidemic of non-communicable diseases. Obesity is the starting point of non-communicable or lifestyle diseases and scientists are still struggling to determine the etiopathogenesis of these lifestyle diseases. The consequences of obesity are grave – hypertension, cardiovascular disease (CVD), diabetes and stroke which indicate that obesity is an

epidemic of serious public health importance. However, obesity leads to a wide variety of co-morbidities in adulthood. Childhood obesity can adversely affect nearly every organ system.

Growth and development

Complications of childhood obesity include early onset of thelarche and menarche in girls, pubertal advancement in boys and adverse effects on maturation and alignment of growing bones in both.[5,6]

Metabolic

Deranged lipid profile occurs among obese children and adolescents. The pattern observed includes increased serum low-density lipoprotein (LDL)–cholesterol and triglycerides and decreased high-density lipoprotein–cholesterol levels.[7] Thee is evidence that soon obesity related type 2 diabetes will become the commonest form of newly diagnosed diabetes in adolescents in the next decade.[8] Atherosclerosis occurs in the obese child and almost half of children with BMI ≥ 97th percentile have one or more of the conditions which comprise the metabolic syndrome.[9]

Nutritional

Increased BMI has been associated with decreased vitamin D levels in children.[10] Overweight and obese children are also more likely to develop iron deficiency.[11]

Systemic

Increased BMI in childhood and adolescence is associated with a high risk of cardiovascular morbidity in adulthood.[7] Hypertension and left ventricular hypertrophy occur. Pulmonary disorders, including obstructive sleep apnoea and reactive airway disease are more common.[12] Orthopaedic problems, including fractures, musculoskeletal discomfort, impaired mobility, and lower limb malalignment may occur more frequently.[13] Serious orthopaedic complications of childhood obesity may include tibia vara (Blount's disease or adolescent bowing of the legs) and slipped capital femoral epiphyses. In females, polycystic ovarian disease(PCOD) has also been obsereved in the obese children. Gallstones, non-alcoholic steatohepatitis(NASH), hepatic fibrosis and cirrhosis are also common in adulthood in obese children. Pseudotumor cerebri has been obsereved among neurological complications.[14]

Psychosocial

Obesity in adolescence largely gives rise to psychosocial problems like obsessive concern about body image, rejection, withdrawl, low self esteem and also depression in adulthood.[14]

Etiopathogenesis

Obesity is largely preventable, but lack of knowledge regarding etiopathogenesis of obesity has resulted in preventive measures leading to variable results in different populations.

Genetic

a. Leptin signaling pathway genes- Inactivating mutations that affect these genes give rise to 3 or 4% of early-onset obesity. Adipose tissues produce leptin and it binds to leptin receptors in the arcuate nucleus in the brain. Increasing fat mass causes a rise in leptin concentrations rise.[15] Fasting lowers leptin, and a decrease in leptin is a signal that stimulates food seeking behaviors and also reduced energy utilization. Restoration of normal leptin concentration leads to decrease in food intake and regulation of appetite. Inactivating mutations that affect both alleles of leptin gene result in increased intake of food and severe, early-onset obesity by causing very low (<5 ng/mL) serum leptin concentrations.[15]

b. Pro-opiomelanocortin (POMC)—In some leptin-responsive hypothalamic neurons, leptin stimulates the production of POMC, which is the precursor for ACTH, alpha, beta, and gamma melanocyte-stimulating hormone (MSH), beta-lipoprotein, and beta-endorphin. Alpha-MSH binds to the melanocortin receptors MC3R and MC4R in the arcuate nucleus and regulates appetite and energy balance. Researchers have identified inactivating mutations of POMC that prevent its cleavage into alpha-MSH or ACTH. These patients have hyperphagia (secondary to absent signaling at MC3R and MC4R) and obesity.[16]

c. FTO—Recent Genome-Wide Association Studies have reported that SNPs in FTO (fat mass and obesity associated) gene are associated with increased body mass index children and adults.[17]

d. PPAR-γ—Peroxisome proliferator-activated receptors (PPAR) regulate metabolism and storage of fat and are involved in differentiation of adipocytes from precursors. A rare gene mutation is associated with morbid obesity.[18]

e. Lipoprotein lipase (LPL) gene - In Asian Indians, the T93G single nucleotide polymorphism(SNP) of the Lipoprotein lipase (LPL) gene is associated with obesity.[19] Indians have increased body fat known as 'Asian Indian phenotype' and central body obesity inspite of low rates of obesity as defined by body mass index (BMI).[20]

f. MC4R gene - Researchers have found associations of SNP rs12970134 near *MC4R* gene with waist circumference and also with insulin resistance. Homozygotes for the risk allele of rs12970134 have apporxiamtely 2 cm increased waist circumference. Common genetic variation near *MC4R* is associated with risk of obesity.[21]

Syndromic

Multiple genetic syndromes are associated with obesity, although patients with these syndromes do not present with obesity related complaints.Syndromes for hyperphagia are the Prader Willi, Bardet Biedl, and Alstrom syndromes.[22]

Prenatal

Appetite and satiety function and regulation begin in utero or in early neonatal periods. Appetite/satiety mechanisms are programmed in response to an changes in pregnancy/newborn environment which may affect infant, childhood and adult appetite "set points". Dysfunctional appetite is a consequence of maternal environmental influences during critical periods of development. Children born with low birth weight paradoxically have an increased risk for adult obesity and diabetes, and this is exaggerated by increased weight gain during infancy. Low birth weight infants also develop hoarding behavior, reduced energy use (couch potato), and a tendency towards adipogenesis.[23] Gestational diabetes and maternal smoking also lead to obesity in adulthood.

Feeding practices

Breastfeeding results in 10–20% reduction in prevalence of obesity in childhood or adulthood, also manifesting a dose-response relationship.[24] Studies have reported that introduction of solids at less than 4 months causes increased infant weight gain.[25]

Sleep

Short sleep duration in children causes obesity. A meta-analysis reported an odds ratio of 1.89 of being obese if a child sleeps less.[26] Another study showed that sleep duration of less than 12 hours during 6–24 months was associated with overweight and obesity in preschool children.[27]

Parenting factors

Parents and family members influence the development of children's, eating, activity and sedentary behavior. These behaviors develops at an early age and show can be tracked into adulthood. Behaviors are learnt early and persist into later life.[23]

Child factors

Studies have revealed that that television viewing is associated with overweight and obesity. Number of hours a child spends watching TV is associated with increased risk of obesity in preschool children.[28] While watching TV, children see advertisements for food products which affects their food preferences and leads to geater energy intake.[28]

Nutrition factors

Studies have shown that consumption of sugar-sweetened beverages has increased rapidly among children. From 1977 to 2001, intake of sugar-sweetened beverages rose from 3.0 to 6.9% among children aged 2–18 years in the US.[29] Sugar-sweetened beverage consumption starts at a young age and increases to adulthood and has been associated with greater caloric intake and increased BMI.

Over the last 30 years, consumption of fast food has paralleled the increase in prevalence of childhood obesity. From 1970s to 1990s, consumption of fast food by children increased 5-fold. Greater consumption of fast food has been found to be associated with poorer diet quality among children and is associated high energy density and obesity.[30]

Physical activity

Regular physical activity has multiple health benefits for children. There is substantial evidence that children who are active early in life have a reduced risk for obesity in schooling years and adolescence.[31]

Prevention

Prevention of childhood obesity is definitely the best and most cost effective approach to curb the global epidemic of obesity. Prevention trials have been criticized for frequently not being based on sound theory of behaviour change and for having inadequate feasibility and pilot work.[32]

Butte and Ellis calculated that an energy deficit of >250 kcal/day is required to prevent further weight gain in 90% of overweight children – equivalent to a child walking an additional 1–2 hours/day at 1.9 km/hour – or consuming roughly one-fifth fewer calories than usual per day.[33]

The approach to prevention needs to be multi-pronged and challenges at every level- individual, household, institutional, community, and health care level, need to be addressed in order to achieve the goal of reduction in childhood obesity[Fig. 1].

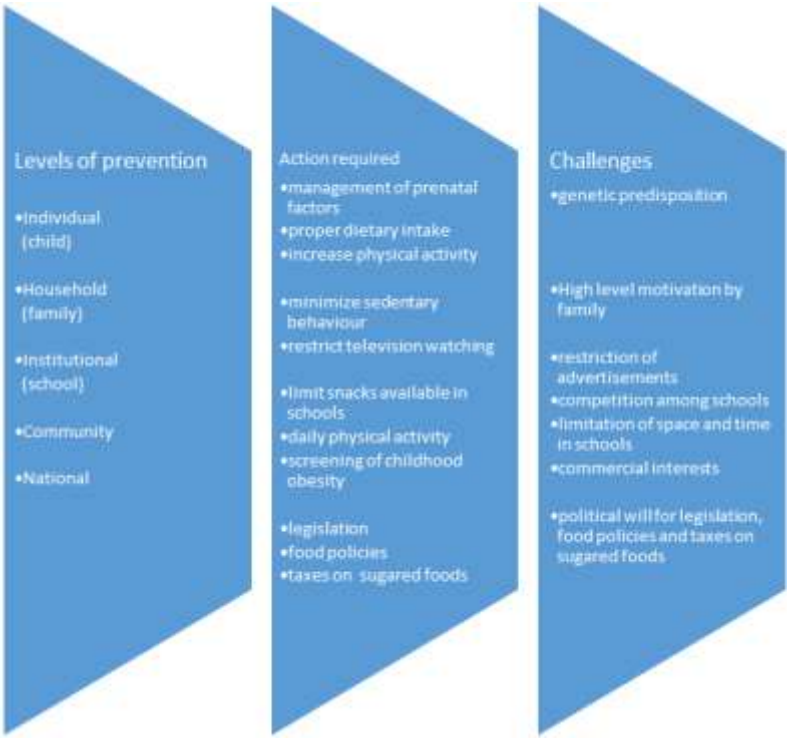

Fig.1: Showing the action required at different levels of prevention and the challenges for prevention of childhood obesity

Individual level prevention

At young age, mothers need to be targeted for prevention of foetal over-nutrition as a consequence of gestational diabetes or maternal obesity. It has also been suggested that breast-feeding might prevent childhood obesity. In older children, proper diet and increased physical activity should be emphasized to prevent childhood obesity. However, genetic predisposition plays n important role in the proportion of weight gain that occurs following a given calorie intake.

Household (family) level prevention

Childhood obesity is not a single problem that occurs in isolation. It exists in the family and prevention means a high level of motivation by the entire family. Children adopt behaviours that are followed by their parents. Unlike communicable diseases where antibiotics are to be popped and the disease is cured, prevention of obesity entails controlling the appetite and modifying sedentary behaviours and that too on a long term basis. Behaviour change is considered the 'gold standard' for preventing and managing childhood obesity.[36] Including families in treatment involves parents as role models to influence their child's weight-related behaviors.[37] Clinicians thereby bring about a change in child nutrition and activity indirectly through the parents. Inclusion of the family, namely parents, into obesity intervention is commonly called 'family-based' treatment. Both children and parents must be targeted for behavior change and weight loss.[38] Clinicians counsel for limiting television viewing or decreasing intake of sugar-sweetened beverages, but families fail to adopt these practices. Obesity treatment faces the challenge of high rates of attrition, which can be as high as 91%.[3] High level motivation is needed to resist the temptation of sugar-sweetened food stuffs and to follow a active lifestyle in these modern times.

Institutional (school) level prevention

Prevention programs include altering the caloric content of school meals, restricting the availability of energy dense snack foods and increasing physical activity. Given the rising competition among schools, modern amenities like availability of snacks in the school premises has become a pre-requisite in high profile schools. Secondly, advertisements and television promotions have made the children demand these foods. Limitation of time and space is a challenge towards incorporating a daily schedule of physical activity for all the students at school.

Community-level prevention

Mass media campaigns are needed for preventing childhood obesity at the community level.[4] Mentioning caloric values on restaurant menu cards, especially at fast food restaurants is being considered as a strategy for preventing childhood obesity. Centers for Disease Control developed a marketing campaign in the US using advertisements to promote physical activity in children aged 9–13 years, wherein children's physical activity (assessed by self-report) increased, but effects on BMI could not be assessed.[40]

National level prevention

Food policies need to be improved to include principles of balanced diet and reduction of BMI. Another strategy is government involvement in several countries for levying increased taxes on sugared beverages and fast foods.[41] Public health surveillance and screening for childhood obesity needs to be done. Legislation for compulsory BMI assessment of children in public schools with annual reporting to parents is being experimented in the US and UK and can be tried in developing countries as well.[42]

In conclusion, only a comprehensive approach can be helpful in the prevention of childhood obesity. Measures taken by the government and motivation by the families and the community are required to combat obesity in children. Given the grave consequences of childhood obesity, it is the need of the hour that all join hands to prevent and control this modern epidemic.

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