

# Advances in Biochemistry & Applications in Medicine

## Chapter 9

# Childhood Obesity, an Emerging Threat to Global Public Health: A Nutraceutical Approach to Contain

*Muni Swamy Ganjayi; Dasari Sreenivasulu; Balaji Meriga\**

*Department of Biochemistry, Sri Venkateswara University, Tirupati, 517502, A.P. India*

*\*Correspondence to: Balaji Meriga, Department of Biochemistry, Sri Venkateswara University, Tirupati, 517502, A.P. India*

*Mobile: +91-9849086856; Email: balaji.meriga@gmail.com*

## Abstract

Global health reports indicate alarming levels of childhood obesity (CHOB) in recent decades across the world. Childhood obesity (CHOB) has a significant impact on both physical and psychological health and is a known precursor to metabolic disorders in adulthood. Apart from genetic aspects, changed lifestyle preferences, environmental factors, food habits, cultural aspects and declining physical activity are the prime causes of rising prevalence of obesity world over. In most populous and developing countries like China and India increased purchasing capacity of the middle classes, increased publicity and mushrooming of fast food centres, supermarkets, attraction of the children towards fried-roasted foods, increased conveyance facility and information technology has direct impact on food habits and contributed to growing over weight-obesity. Both obese children and adults are at increased risk for several health complications including hypertension, dyslipidemia, type 2 diabetes, CVDs, arthritis and infertility. Additional health complications associated with overweight-children include sleep apnea, asthma and liver diseases. Since it is difficult for children to have bariatric surgery or to be on synthetic drugs for a long time, a natural product based nutraceutical approach may find fit to deal with childhood obesity. This review discusses and updates various causative aspects and consequences of childhood obesity and necessary interventional options with emphasis on phytochemicals to contain CHOB.

**Keywords:** Childhood obesity; Causative factors; Nutraceuticals

## 1. Introduction

### 1.1. Epidemiology of childhood obesity

In recent decades prevalence and severity of childhood obesity has reached epidemic proportions, [1]. In 2016, it was estimated that globally 200 million children under 5 years were overweight, with more than 75% of overweight or obese children living in low and middle income countries [2,3]. While the incidence of obesity is not new to the developed countries, the main drivers of the escalating trends of childhood obesity in the developing countries are cheap foods with high content of sugar and fat, sedentary lifestyles, rapid nutritional transitions, increasing affluence, socioeconomic transitions, urbanization, mechanization and rural-to-urban migration [4]. For instance, the socio economic transitions in most populous countries like China and India have profound impact on world statistics of obesity and overweight [5]. In developing countries, the requirement for physical labour has considerably reduced due to mechanization, availability of advanced technologies, various implements, instruments and sophistication of life styles. Growing and rampant rural to Urban migration and consequent changes in lifestyles and food habits is an important cause for high incidence of obesity and other metabolic disorders.

Since 1986, several studies in preschool children show increasing obesity in most countries in Latin America and the Caribbean, along with the Middle East and North Africa, which is comparable with prevalence rates of childhood obesity seen in the United States [6]. Similar trends have also been observed in India, Mexico, Nigeria, and Tunisia over the past two decades [7]. Increase in the prevalence of overweight among older children and adolescents has been seen as well; from 6.4 to 7.7% between 1991 and 1997 in China, and from 16 to 24% between 2002 to 2007 in New Delhi, India [8,9].

### 1.2. Obesity parameters: Body mass index (BMI) as a measure of overweight and obesity

Overweight is defined as a body mass index (BMI) in the 25 to 29 kg/m<sup>2</sup> range, where as obesity is a BMI in excess of 30 kg/m<sup>2</sup>. Overweight and obesity result from an energy surplus over time that is stored in the body as fat. The definition of childhood overweight and obesity based on body mass index (BMI) is complicated but it is made clear by recent studies [10]. Due to differences in maturation and growth, the measurement of overweight and obesity in children and adolescents is very difficult. There are two periods when adiposity increases they are about the age of 5 to 7 years and early puberty. So, BMI in childhood changes substantially with age. The international cut-offs defined the BMI values at the age of 18 years but BMI reference of the WHO is based on growth standard and growth reference. Using LMS (Skewness L, Median M, Variation S) curves coefficients are related to the international child BMI cut-offs of thinness, overweight and obesity and they make it simple to compare them with

other methods like BMI cut-offs [11].

## **2. Contributing factors**

There are regional differences in the prevalence of childhood obesity that have occurred overtime [12] and in many countries childhood obesity depends on lifestyle behaviours such as physical activity and dietary intake, but childhood obesity may not depend on same lifestyle behaviour across the world [13,14]. Complex interaction of multiple behavioural, biological and environmental factors which adversely impact long time energy balance and this energy balance leads to obesity in children. The major contributing factors are showed in **Figure 1**.

### **2.1. Genetic background**

Obesity is developed by complex interactions between environment, behaviour and genetic predisposition. Of late, dietary and lifestyle changes are said to be major contributing factors to develop obesity, but previous studies reported the genetic basis for development of obesity [15-18]. There is growing evidence that genetic factors are cornerstone in the development of obesity [19]. Specific gene expression pattern of obesity may help to understand the pathogenic mechanisms of obesity and associated metabolic diseases [19].

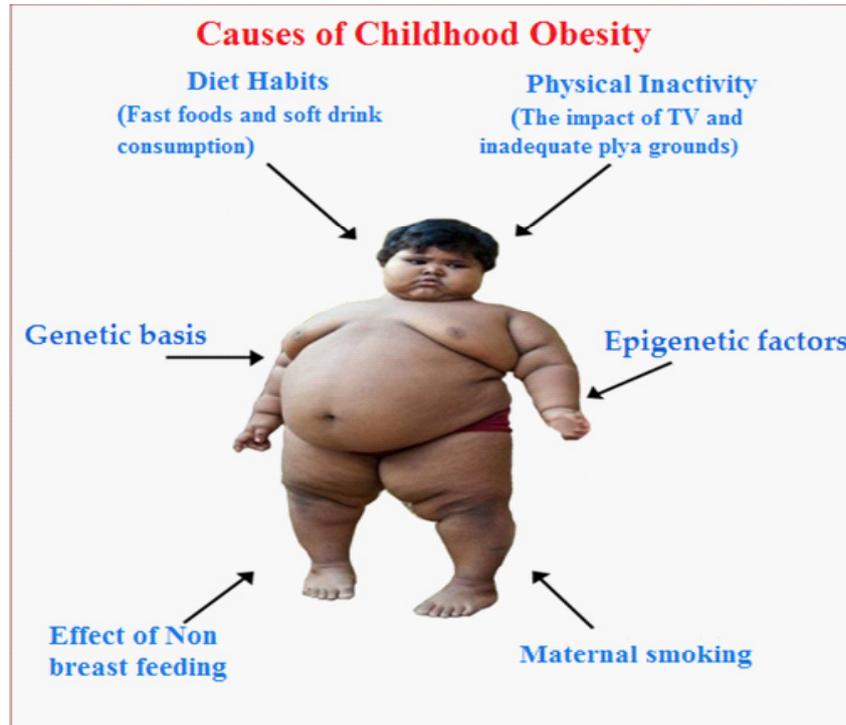
### **2.2. Epigenetic aspects: Environmental factors**

Influence of environment encompasses several aspects like social, cultural, economic and political factors [20]. The obesogenic environments are divided in to two stages- the micro and macro settings/sectors [21] and there are four types of environments like physical environment (what is available), economic environment (what are the costs), policy environment (what are the rules) and socio-cultural environment (what are the beliefs and attitudes) [20]. In recent decades, sea changes in environmental aspects have been greatly contributing to the tremendous raise of obese children rather than genetic aspects. Physical activity and television viewing may have independent effects on adiposity and cardiovascular risk factors [22]. Increased purchasing power, increased availability of readymade foods and decreased necessity for walking because of increased conveyance facilities are also their due role in this regard.

### **2.3. The impact of TV and advertising**

A special focus is given in the present paper on TV and advertising. Within the environmental context, the impact of TV viewing and advertising on children, their seating behaviour and health seems to have a potential association with their overweight or obesity problems. Television is suspected to be linked to a reduction in physical activity whilst advertising seems to promote an over consumption of sugar rich and high fat foods [23]. Most of the studies showed [24] a positive correlation between times spent watching television and nutritional status of the subjects involved.

Two main aspects have been considered by researchers' about the TV effects on children's obesity: (1) reduced energy expenditure linked to screen time [25] and (2) augmented energy intake driven by advertising and snacking in front of the TV (**Figure 2**). The first issue seems to be related to long hours of TV watching, influencing positive energy balance through displacement of physical activity. It has been suggested that youth may decrease their physical activity when sedentary behaviours are increased.



**Figure 1:** Causes of Childhood obesity



**Figure 2:** Children eating high calorie foods and drinks

## 2.4. Fast foods and soft drinks consumption

Fast food consumption is another leading suspect in the childhood obesity epidemic. Fast foods typically include all of the things that nutritionists warn against: ‘saturated and trans-fats’, high glycemic index, high energy density, and large portion sizes. This alarming trend should be of particular concern to health authorities and public communities. Usually,

food industries release attractive “messages” and “advertisements” to which children are easily attracted and thus increased consumption of high-fat, high-sugar foods (HFSS) can profoundly impact a child’s eating habits and weight status [14]. It is therefore undeniable fact that the food industry has successfully created a highly obesogenic environment. Consumption of meat foods like chicken and fish that are grown using growth boosters is also reported to have obesogenic effect. As children are an important asset of any nation, this disorder adversely affects the future generations as it results in metabolic syndrome and causes higher mortality rate in adulthood [26].

Further studies reveal that the soaring rates childhood obesity has been due to innate propensity of children to respond to external food advertising cues [27]. Some research supports the hypothesis, that there are no differences between obese and leaner individuals in their liking for sweetness in food products and subsequent food intake in relation to brand exposure [28]. It is equally important to note that the amount of television-viewing among children denotes the possibility of better fast food brand recognition which can be a threat to children’s health [9,29]. However, conflicting evidence revealed that obese children did not consume more than non-obese individuals when meals were branded with famous food logos [28]. Employing strategic branding on healthy foods may be a novel and effective way of helping children develop healthy eating habits and lifestyles from an early age [30].

## **2.5. Effect of smoking during pregnancy**

Many previous studies support the ‘fetal origins of adult diseases’ hypothesis for maternal smoking: these studies showed a positive association between maternal smoking during pregnancy and overweight in children aged between 5 and 16 years. One study found that maternal smoking during pregnancy was mainly related to childhood overweight in the upper percentiles [31]. The number of cigarettes smoked during pregnancy also appeared to be associated with childhood overweight. Syme et al. [32] used magnetic resonance imaging-based (MRI) studies to measure adiposity and reported that maternal smoking during pregnancy was not associated with fat distribution in early puberty but higher subcutaneous and intra-abdominal fat mass was noticed in late puberty [32,33]. Other studies on maternal smoking showed that over weight increased with age suggesting a lasting effect that may increase even further in puberty, adolescence and adulthood, with major health implications [34].

## **2.6. Effect of breast feeding**

Breast feeding has been associated with a decreased risk of obesity, along with other health benefits for the child and mother. According to the WHO recommendations, infants should be exclusively breast fed for the first six months, and breast feeding should be supplemented with additional foods for the first two years or beyond. Breast milk is considered the ideal food for infants, as it provides adequate energy and nutrients to meet the infants’ needs.

In addition, as breast milk is safe and contains antibodies, breast feeding could reduce the risk of neo natal infection, gastro-intestinal infection, and pneumonia during infancy [35,36].

In recent decades, there is a growing work culture among women of developing countries of Asia and African continents to work in industrial and service sectors. So, due to economic compulsions, work timings and other reasons the mothers are forced to skip breast feeding to their babies. Babies fed on tinned milk/formula milk are more prone to develop health complications including obesity ailments.

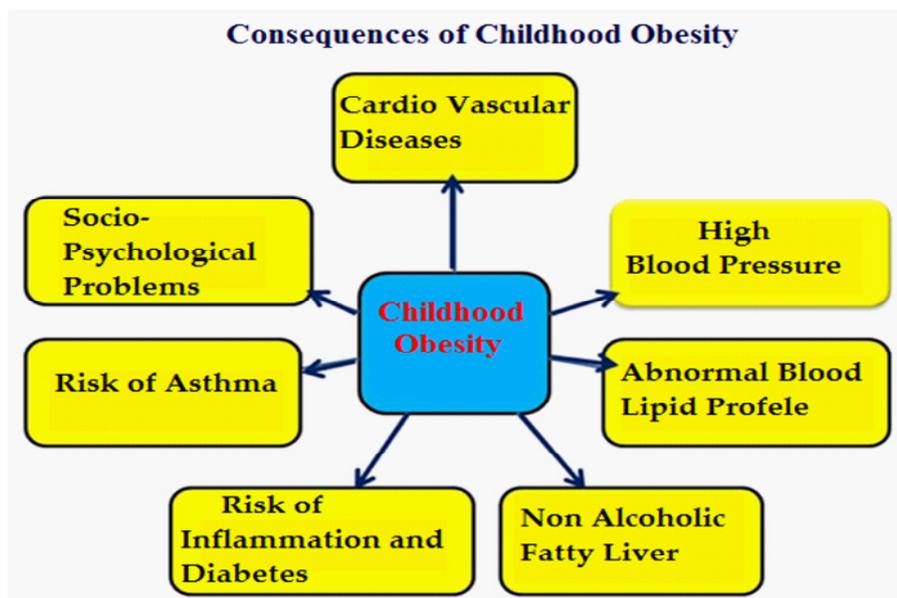
Prolonged breast feeding is directly related to a decreasing risk of obesity in children [37]. More particularly, children being breastfed for  $\geq 7$  years are significantly less likely to be obese in later childhood [38]. Moreover, breast feeding has long-term benefits through-out a child's life time. Usually, children who were breast fed have lower rates of overweight/obesity, type-2 diabetes, hypertension and are known score higher on intelligence tests than persons who were formula-fed [39]. Breast feeding has been identified as a protective factor for childhood obesity in many studies [40,41].

### **2.7. Changed school environments: without adequate playgrounds**

More particularly in developing countries like India, mushrooming of private schools and colleges with inadequate play grounds could be widely noticed. As a result, children are not adequately exposed to playing games and sports regularly and hence do not get required physical activity. In school going children, increased snacking frequency, consumption of junk foods, soft drinks, milk products and ice creams that contains excessive sugar, fat and preservatives, coupled with steady decline in physical activity have been major contributing factors for rising rates of obesity [42]. Surprising observation is that some school managements allot more time periods for teaching and learning and give less time for physical activity because they are more concerned about students scoring higher makes from their school. Among apparently healthy school going children the higher prevalence of obesity is unnoticed and in obese children the systolic and diastolic hypertension was higher [42].

### **3. Consequences of childhood obesity**

Many of the outcomes associated with obesity which were previously thought of as diseases of adults are now affecting children as well. Outcomes related to childhood obesity include hypertension, diabetes, dyslipidemia, CVDs, non-alcoholic steatohepatitis, obstructive sleep apnea, and orthopedic problems in addition to social and psychological problems (**Figure -3**). Hence, obesity is popularly described as “New World Syndrome”.



**Figure 3:** Consequences of childhood obesity

### 3.1. Cardiovascular diseases (CVDs)

The World Health Organization (WHO) reported in 2008 that 17.3 million deaths worldwide were due to CVDs [43]. A major contributor to CVDs is atherosclerosis which is a dynamic process that can begin in childhood and develop or regress, depending on the presence or absence of a range of risk factors including obesity, inflammation, hyperglycemia, hypertension and hyperlipidemia [44,45]. The International Obesity Task force estimates that approximately 40-50 million school aged children are obese [2].

Abdominal obesity in children is associated with low grade inflammation, a significant contributor to the development of atherosclerosis [44]. Both BMI and WC correlate with intra-abdominal fat in primary school aged children [47] and are used as clinical measures to identify CVD risk [48]. Obese children are also at increased risk of hypertension and dyslipidemia as they age [49,50]. Overweight boys with high dietary intakes of fat and carbohydrate in particular had significantly more CVD risk factors than girls.

### 3.2. Blood pressure alterations

The prevalence rates of hypertension and obesity are increasing worldwide in children [51]. Increased blood pressure leads to damage of capillaries and tissues in brain, heart, kidneys etc. Administration of antihypertensive drugs like diuretics, ACE inhibition, adrenergic receptor antagonist, renin inhibitors and vasodilators on children for longer period is not suggestive as they cause side effects. One report quoted that, the blood pressure lowering effect of docosa hexanoic acid (DHA), observed in adults, could be mediated by ATP release from the endothelium, which increases vasodilation by stimulating the release of nitric oxide, and by the decrease in nor-adrenaline levels [52].

A systematic review stated that breast feeding has a small protective effect against high

systolic blood pressure, although residual confounders had to be eliminated [53]. One of the plausible mechanisms that have been suggested to explain this protective effect is represented by the presence of long chain poly unsaturated fatty acids (LCPUFAs), including DHA, which are important structural components of the vascular endothelium [53].

### **3.3. Abnormal blood lipid profile**

The alterations of blood lipid profile associated with metabolic syndrome are usually characterized by increased triglycerides (TG), very-low-density lipoproteins (VLDLs), low density lipoproteins (LDL) and reduced High density lipoproteins (HDL) [54,55]. Visceral obesity and insulin resistance could be key factors in the promotion of atherogenic dyslipidemia by increasing the synthesis of TG-rich VLDLs in the liver [56].

Long term administration of statins or other anti hyperlipidemic drugs have side effects on children and adults. A study performed on 32 obese children showed that plasma phospholipids, DHA content was negatively associated with VLDL-triglyceride, a major factor involved in the development of metabolic syndrome [57].

### **3.4. Non-alcoholic fatty liver disease (NAFLD)**

In children of industrialized countries, Non-alcoholic fatty liver disease NAFLD is the most common chronic liver disease reaching prevalence up to 80% in obese or overweight children [58]. NAFLD includes different diseases ranging from “simple” liver steatosis, with pathological accumulation of fat, non-alcoholic steatohepatitis (NASH), with different degree of inflammation and fibrosis to end-stage liver disease with cirrhosis and hepato-cellular carcinoma [59].

Only one study showed that in obese children with single-nucleotide polymorphism (SNP), 276G>T at adiponectin gene, the increased liver echogenicity could be associated with higher levels of n-6 PUFA in plasma phospholipids (Presented at 44<sup>th</sup> ESPGHAN Annual Meeting, Sorrento) [60]. However, some trials evaluated the effect of extracts supplementation on paediatric NAFLD ([61,62]. A reduced liver hyper-echogenicity was observed in children with NAFLD after DHA supplementation for 12 and 24 months [61].

Breast feeding might be protective against NASH and liver fibrosis, suggesting a long-lasting effect of breast milk [63]). The authors speculated that DHA supplied by breast milk, could be protective, acting as a Peroxisome proliferator-activated receptor (PPAR)-agonist, a transcription factor involved in protection against fibrosis [60,64].

### **3.5. Risk of diabetes mellitus**

Childhood obesity is a condition where children below the age of 14 years have high

BMI. Many of them are prone to diabetes when they become adults. Day by day we witness that the effect of junk food is clutching the children's future into neuroleptic malignant syndrome (NMS). It seems that children of very low age are suffering from either type-1 or type-2 diabetes. Type-1 diabetes is generally found in children and was initially termed as juvenile diabetes. About 10% of children worldwide is suffering from type-1 diabetes only. The diabetic figures of Asia and African subcontinents are shocking. The increased longing for consumption of polished rice and wheat foods, dairy products, ice creams, stored-preserved products, fast foods and other junk foods by school children has been the major cause of raise in diabetes. Added to this, decreased preference for sports and games at schools and home resulted in decreased physical activity [65].

With changed typical physical work and dieting activity, the BMI of the persons is changing to considerable extent in every decade and it has been noticed in Americans mostly [66]. Eating disorders are the main reasons of obesity, as obese person when eat more become more fat and then the tendency to lose the fat will get decreased. Moreover, the fat person feels sleepy all the time and lazy too which does not allows him/her to work and they remain in the same lying/siting posture for long time. Not only children alone, but with the changing times eating habits are also changing due to which obesity has been considered as the centre of metabolic syndrome as it causes different chronic disorders which some-times may leads to death also [67].

### **3.6. Risk of newly diagnosed asthma**

An association between a greater BMI and increased risk of asthma in both children and adults has been repeatedly shown in prospective, population-based studies [68]. Previous studies demonstrated significant associations between overweight/obesity with asthma and eczema [69].

The age of the weight gain may play a role, as pronounced weight gain in early life was identified as a risk factor to develop asthma before 10 years of age [70]. Two types of asthma in obese subjects can be distinguished by the age of onset and clinical presentation. Early onset of asthma in obese cases before the age of 12 years might occur in boys and girls and is characterized by severely decreased pulmonary function, significant airway hyper-responsiveness, and poor asthma control. These patients are atopic; serum immunoglobulin E (IgE) is increased, airway inflammation is eosinophilic, and fraction of exhaled nitric oxide (FeNO) is high [71]. In contrast, obese late-onset asthmatics become symptomatic after the age of 12 and are predominantly females without atopic characteristics. Compared to early-onset asthmatics, they have little airway obstruction with less airway hyper-responsiveness and better asthma control.

### 3.7. Risk of psychosocial problems

Overweight in children and adolescents may be associated with a range of psychological and social problems that have a considerable deleterious impact on the behavioural development and quality of life in them. By the ages of about 5 years, obese boys have more conduct problems, hyperactivity, inattention problems and peer relation problems than normal weight children. Interestingly, obese girls of these respective ages present only with more peer relation problems in comparison to normal weight girls, indicating obese boys to be at a greater risk for the development of emotional and behavioural problems already at an early age [72]. The school and social performance of obese children, their academic and extracurricular activities and their overall quality of life are often less favourable compared to their normal weight peers. This is a multi-faceted problem, related to greater school absenteeism, less nutritious diet and physical activity, more emotional and behavioural problems, less favourable neuropsychological functioning and overall psychosocial stress.

By contrast, negative body esteem is usually correlated with the severity of obesity. Normal weight children tend to show negative attitudes towards their overweight peers. Obese youth are usually considered less popular than normal-weight teens, and overweight during childhood and adolescence may be associated with low esteem, considerable societal victimization and peer teasing. Moreover, obese children themselves perceive negatively many of their own characteristics and attach them to their overweight [73].

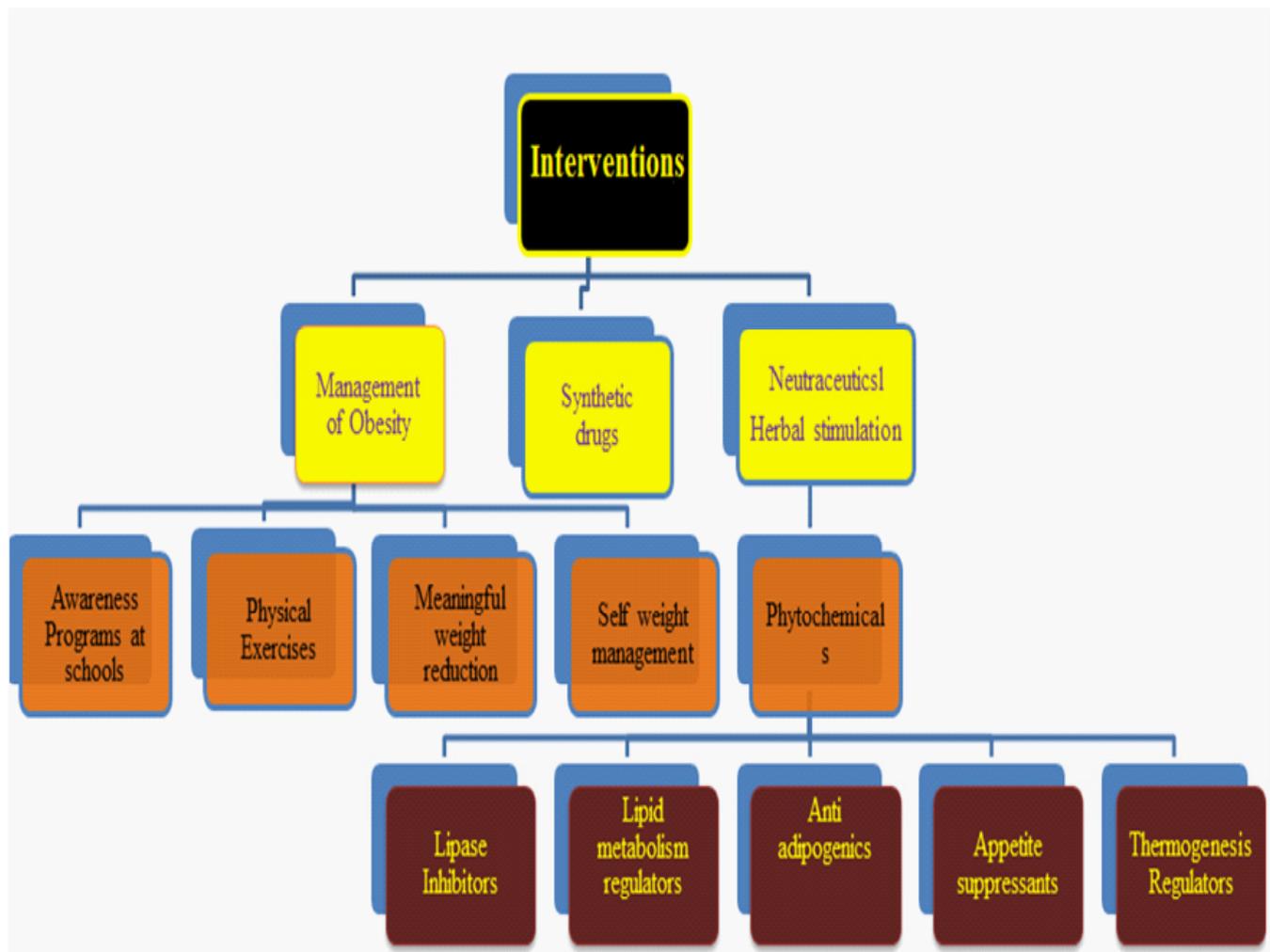
### 3.8. Risk of metabolic syndrome

The worldwide epidemic of childhood obesity is responsible for the occurrence in paediatric disorders once mainly found in adults, such as the Metabolic Syndrome (MS). The prevalence of childhood obesity has been increasing in the last four decades with an estimated sixty millions of children being overweight worldwide by 2020 [74]. MS in children is commonly defined as the co-occurrence of three or more of the following features: severe obesity (high BMI), dyslipidemia (increase of triglycerides and decrease of HDL), hypertension and alterations of glucose metabolism such as impaired glucose tolerance (IGT) and type 2 diabetes [75]. This causes a large accumulation of lipids into the liver, which results in hepatic steatosis and higher triglycerides production. From a molecular point of view the link between lipid accumulation and insulin resistance seems to be represented mostly by diacylglycerol (DAG) [76]. In children aging between 5-8, only 16% can be regarded as metabolically healthy, while about 36% fulfilled the criteria for metabolic syndrome [77]. The metabolically healthy children are characterized by lower waist circumference, less visceral fat content, increased peripheral insulin sensitivity, less pronounced inflammatory status, lower melondialdehyde concentration etc.

## 4. Interventional Strategies

### 4.1. Awareness campaigns

Both sedentary life style and high energy diet contribute to increased global obesity rates for the past three decades [78,79]. Along with obesity, the associated ailments like diabetes and heart disease [80] are cause of public health concern in both children and adults. Hence, education campaigns and health promotion programs are recommended by the World Health Organization as prevention strategies of obesity [81,3]. While implementation of WHO recommended prevention strategies of obesity in multicultural countries, variation in cultural, religious, demographic and ethnic backgrounds needs to be considered [82-86]. For instance, a study showed that, the assimilated Iranian migrants were more active to accept and endorse the Australian culture of physical activity. Otherwise, this contributes to the development of obesity in migrant populations [87]. At nursery and school level the teacher/trainers need to bring greater awareness about the junk foods and emphasize on physical activity. The non-governmental organisations (NGOs) and other institutes need to play active role in this regard as the epidemic of childhood obesity affects the future of their countries. Information about new lifestyle and environmental interventions is necessary for implementation.



**Figure 4:** Interventional strategies

## 4.2. Management of childhood obesity

Management of weight in the child includes strategies to both reduce overweight/obesity and promote sustained change. Most weight loss programs are based on promoting behavioral change in the child and parent. As in the adult, caloric intake must be less than energy expended for weight loss to occur. Therefore, the key to weight loss in the children is making changes in both diet and exercise. Behavioural interventions included meeting with individual case managers, group and individual counselling sessions, self-management training, individualized adherence strategies, and clinical support help to manage obesity. Although many family physicians are pessimistic about their ability to influence patients to make necessary lifestyle changes in order to achieve weight loss, research suggests that patients are more likely to attempt weight loss when their primary care physicians recommend it [88]. In another study, patients who lost weight credited their physicians with having helped them by explaining the health risks of obesity, making physical activity recommendations, and providing referrals to weight-loss groups or programs.

It is also essential to keep in mind that while pharmaceutical agents can help patients achieve clinically meaningful weight loss, the medications must generally be continued to maintain the reduction [89]. Multivitamins contain fat-soluble vitamins to offset potential losses from fecal fat excretion [90]. Physical exercise and activity are particularly important for maintaining weight loss over the long term (and for preserving lean body mass during dieting) [89].

## 4.3. Synthetic drugs

Although some drugs/formulations are available in the market, only a few drugs are approved by FDA and European commission, and some of them are withdrawn due to their side effects. So it not advisable for children to take such drugs for a long time. In view of the difficulties in implementing dietary restriction and physical exercise regularly and in place of pharmaceutical approach, nutraceutical approach gained moment to treat childhood obesity.

## 4.4. Nutraceuticals

Nutraceuticals are products derived from food sources with extra health benefits in addition to the basic nutritional value found in foods. In this context, an effective nutraceutical is that which can increase energy expenditure and/or decrease caloric intake is desirable for body weight reduction. Several phytochemicals have been reported for their anti lipidemic and anti obesity activities (**Figure 4**) [45]. Herbal stimulants, such as caffeine, ephedrine, chitosan, ma huang-guarana, and green tea are effective in facilitating body weight loss [91]. However, their use is controversial due to their ability to cause side-effects. Green tea extract and 5-hydroxytryptophan may promote weight loss, while the former increases the energy expenditure,

the latter decreases appetite. [91,92].

Active ingredients of fruits, vegetables, and other edible plants, comprising of flavonoids, saponins, tannins, glucosinolates, phenols, phytates, phyto-estrogens are capable of efficiently combating metabolic syndrome. Anti-inflammatory properties contribute to counteract the obesogenic state [93]. Dietary phytochemicals (**Figure 4**) act on various targets associated with obesity ailments [94]. Some probable mechanisms of action of these plant derived products are reduction in adipose tissue mass by inhibition of precursor cell proliferation, enhancing apoptosis of fat cells and hindering the absorption of triglyceride by reducing formation of pancreatic lipase and amylase. Some work as appetite suppressants, interfere in lipid metabolism, decrease intake of energy and enhance energy expenditure [45,95,96].

For example asparagus is also effective in weight control. It works by flushing out toxins and other wastes from the body [97]. Tomatoes, the low calorie vegetables are consumed raw as well as cooked. Lycopene, the active constituent in tomato is famous for its anti-oxidant and anti-carcinogenic properties. Tomatoes are good for health as they also help to lose weight [98].

Oats, a popular breakfast item, is rich in antioxidants and other minerals. Fibre in oats brings down the cholesterol level [99]. Blueberries, rich in anthocyanins are responsible for breaking down fats and sugars and thus curtail extra fat from our body [100]. The most popular beverage in the world next to water is green tea, obtained from the leaves of *Camellia sinensis*, helps in reducing weight. Other fruits and vegetable like papaya, kera, grapes, lemen, curry leaves, carrot spices like piper, cumin seeds, cinnamon etc., possesses anti-obesity effects [101,45].

## 5. Conclusion

Recent medical and health reports have shown childhood obesity as an emerging threat to the global health in 21st century and termed it “New World Syndrome”.

The present study focused on various causative factors for raising prevalence of obesity world over. One of the major contributing factors for alarming world statistics on childhood obesity is increased purchasing power of millions of middle class people in most populous countries like China and India which has direct bearing on changed food habits and life styles. Other reasons include decreased preference for physical activity in schools and avoiding of breast feeding by mothers for various reasons. Since children are vulnerable and cannot be put on pharmacological intervention for a long time, nutraceutical approach has been highlighted in this study. Several fruits, vegetables and spices like cucumber, tomato, curry leaves, bitter guard, moringa, piper, cumins seeds, cinnamom etc., have significant beneficial effects against obesity ailments. Finally the parents at home and teachers at school need to educate children

and emphasize to avoid junk foods, encourage physical activity and include anti-obesity nutraceuticals in regular diet because today's healthy children are tomorrow's wealthy citizens.

## 6. References

1. Ellulul Mohammed, Yehia Abed, Asmah Rahmat, Yazan Ranneh and Faisal Ali. Epidemiology of obesity in developing countries: challenges and prevention. *Glo Epi Obes* 2014; 2: 2052-5966.
2. International Obesity Task Force. for Saudi, Canada, South Africa, Australia and NZ estimates. 2013.
3. World Health Organization. Childhood overweight and obesity. 2014
4. Aziz S, Noorulain W, Zaidi UE, Hossain K, Siddiqui IA. Prevalence of overweight and obesity among children and adolescents of affluent schools in Karachi. *J Pak Med Assoc.* 2009; 59: 35-38.
5. Hasani-Ranjbar, S., Z. Jouyandeh and Abdollahi, M. A systematic review of anti-obesity medicinal plants - an update. *J Diab Metab Disord.* 2013; 12, 28-38.
6. 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.
7. Wang Y, Monteiro C and Popkin BM. 2002. Trends of obesity and underweight in older children and adolescents in the United States, Brazil, China, and Russia. *Am J Clin Nutr.* 2002; 75: 971-977.
8. Bhardwaj S, Misra A, Khurana L, Gulati S, Shah P and Vikram NK. Childhood obesity in Asian Indians: a burgeoning cause of insulin resistance, diabetes and sub-clinical inflammation. *Asia Pac J Clin Nutr.* 2008; 17: 172-175.
9. Ranjani H, Pradeepa R, Mehreen TS, Anjana RM, Anand K, Garg R, Mohan V. Determinants, consequences and prevention of childhood overweight and obesity: An Indian context. *Ind J Endo and Met.* 2014 ; 18: S17.
10. Rolland-Cachera MF. . Childhood obesity: current definitions and recommendations for their use. *Int J Pediatr Obes.* 2011; 6: 325-331.
11. T. J. Cole & T. Lobstein. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. *Pediatr Obes.* 2012; 7: 284-294
12. Wang Y, Lim H. The global childhood obesity epidemic and the association between socio-economic status and childhood obesity. *Int Rev Psychiatry.* 2012; 24; 3: 176-188.
13. Haug E, Rasmussen M, Samdal O, Iannotti R, Kelly C, Borraccino A, Vereecken C, Melkevik O, Lazzeri G, Giacchi M, et al. Overweight in school-aged children and its relationship with demographic and lifestyle factors: results from the WHO-collaborative health behavior in school-aged children (HBSC) study. *Int J Public Health.* 2009; 54:167-179.
14. Williams AM, Suchdev PS. Assessing and Improving Childhood Nutrition and Growth Globally. *Pediatr Clin.* 2017; 4: 755-768.
15. Blakemore, A. I. F., & Froguel, P. Is Obesity Our Genetic Legacy? *J Clin Endo Metab.* 2008; 93: S51-56.
16. Farooqi, I. S. Genetic and hereditary aspects of childhood obesity. *Best Pract. Res. Clin. Endo Metab.* 2005; 3: 359-374.
17. O'Rahilly, S., & Farooqi, I. S. 2008. Human obesity as a heritable disorder of the central control of energy balance. *Int J Obes (Lond).* 2008; 32: 55-61.
18. Llewellyn, C. H., van Jaarsveld, C. H. M., Boniface, D., Carnell, S., & Wardle, J. Eating rate is a heritable phenotype related to weight in children. *Am J Clin Nutr.*, 2008; 88: 1560-1566.

19. Puiu Maria, Chirita Emandi Adela and Arghirescu Smaranda. Genetic Disorders. *Gent and Obes.* 2013; 10: 5772-52403.
20. Delavari, M.; Sonderlund, A.L.; Mellor, D.; Mohebbs, M.; Swinburn, B. Exploring obesogenic environments: Design and development of migrant obesogenic perception of the environment questionnaire (MOPE-Q). *BMC Pub Health.* 2014; 14: 567–578.
21. McLennan, W.; Podger, A.S. National Nutrition Survey Users' Guide, 1995; Australian Bureau of Statistics and Commonwealth Department of Health and Family Services: Canberra, Australia, 2008.
22. Ekelund U, Brage S, Froberg K, Harro M, Anderssen SA, Sardinha LB, Riddoch C, Andersen LB. TV viewing and physical activity are independently associated with metabolic risk in children: the European youth heart study. *PLoS Medicine.* 2006; 12: e488.
23. Carter OB, Patterson L, Donovan RJ, Ewing MT, Roberts CM. Children's understanding of the selling versus persuasive intent of junk food advertising: implications for regulation. *Soc Sci Med* 2011; 72: 962-968.
24. Adams J, Tyrrell R, Adamson A, White M. Socio-economic differences in exposure to television food advertisements in the UK: a cross-sectional study of advertisements broadcast in one television region. *Public Health Nutr.* 2012; 3: 487-494.
25. Scaglioni S, Arrizza C, Vecchi F, Tedeschi S. Determinants of children's eating behavior. *Am J Clin Nutr.* 2011; 6: 2006S-2011S.
26. World Health Organization 2011.
27. Bruce, A. S., Lepping, R. J., Bruce, J. M., Cherry, B. C., Martin, L. E., Davis, A. M., Brooks, W. M., & Savage, C. R.. Brain responses to food logos in obese and healthy weight children. *The J. Pediatr.* 2012; 1-6.
28. Keller, K. L., Kuilema, L. G., Lee, N., Yoon, J., Mascaro, B., Combes, A., Deutsch, B., Sorte, K., & Halford, J. C. G. The impact of food branding on children's eating behavior and obesity. *Phy. & Beha.* 2012; 106: 379-386.
29. Gunnarsdottir, I., & Thorsdottir, I. Should we use popular brands to promote healthy eating among children? *Pub Health Nutr.* 2010; 1: 1-4.
30. Lai Siew Tim, Zuhrah Beevi and Reiko Yeap. Effects of Fast-Food Branding on Children's Taste Preferences. *South. Asia Psy J.* 2014; 2: 39-56.
31. Beyerlein A, Toschke AM, von Kries R. Risk factors for childhood overweight: shift of the mean body mass index and shift of the upper percentiles: results from a cross sectional study. *Int J Obes.* 2010; 34: 642–648.
32. Koshy G, Delpisheh A, Brabin BJ. Dose response association of pregnancy cigarette smoke exposure, childhood stature, overweight and obesity. *Eur J Public Health.* 2011; 21: 286–291.
33. Syme C, Abrahamowicz M, Mahboubi A, et al. Prenatal exposure to maternal cigarette smoking and accumulation of intra-abdominal fat during adolescence. *Obesity.* 2010; 82: 1021–1025.
34. Timmermans. SH, Mommers M, Gubbels JS, Kremers SPJ, Stafleu A, Stehouwer CDA. Prins MH, Penders J, and Thijs C, Maternal smoking during pregnancy and childhood overweight and fat distribution: the KOALA Birth Cohort Study. *Pediatr Obesity.* 2013; 9: 14-25
35. Stolzer JM: Breast feeding and obesity: a meta-analysis. *Open J Prev Med.* 2011; 1: 88–93.
36. Labayen I, Ortega FB, Ruiz JR, Loit HM, Harro J, Villa I, Veidebaum T, Sjostrom M: Association of exclusive breast-feeding duration and fibrinogen levels in childhood and adolescence. *Arch Pediatr Adolesc Med.* 2012, 166: 56–61.
37. Kar SS, Kar SS. Prevention of childhood obesity in India: Way forward. *J Nat Sci, Biol and Med.* 2015; 6(1): 12.

38. Jing Yan, Lin Liu, Yun Zhu, Guowei Huang and Peizhong Peter Wang. The association between breast feeding and childhood obesity: a meta-analysis. *BMC Public Health*. 2014; 14: 1-11.
39. Jimenez-Cruz A, Bacardi-Gascon M, Pichardo-Osuna A, Mandujano-Trujillo Z, Castillo-Ruiz O: Infant and toddlers' feeding practices and obesity amongst low-income families in Mexico. *Asia Pac J Clin Nutr*. 2010; 19: 316–323.
40. Chivers P, Hands B, Parker H, Bulsara M, Beilin LJ, Kendall GE, Oddy WH: Body mass index, adiposity rebound and early feeding in a longitudinal cohort (Raine study). *Int J Obes (Lond)*. 2010; 34: 1169–1176.
41. Labayen I, Ruiz JR, Ortega FB, Loit HM, Harro J, Villa I, Veidebaum T, Sjostrom M: Exclusive breastfeeding duration and cardiorespiratory fitness in children and adolescents. *Am J Clin Nutr*. 2012; 95: 498–505.
42. Anjankumar V S, Bhagyalakshmi V S, Rajesh T and Arumugam A. 2015. Prevalence of Hypertension Among Obese Children and Effect of Environmental Factors on Hypertension and Childhood Obesity: A School Based Study. *Int J Intg. MedSci*, 2015; 2: 99-103.
43. Global Atlas on Cardiovascular Disease Prevention and Control.
44. Francis, A.A.; Pierce, G.N. An integrated approach for the mechanisms responsible for atherosclerotic plaque regression. *Exp. Clin. Cardiol.* 2011; 16: 77–86.
45. Balaji M, Ganjayi MS, Kumar GE, Parim BN, Mopuri R, Dasari S. A review on possible therapeutic targets to contain obesity: the role of phytochemicals. *Obes research & Clin pract*. 2016; 10: 363-380.
46. Galcheva, S.V.; Iotova, V.M.; Yotov, Y.T.; Bernasconi, S.; Street, M.E. Circulating proinflammatory peptides related to abdominal adiposity and cardio metabolic risk factors in healthy prepubertal children. *Eur. J. Endo*. 2011; 164: 553–558.
47. Von Schnurbein, J.; Klenk, J.; Galm, C.; Berg, S.; Gottmann, P.; Steinacker, J.M.; Kratzer, W.; Brandstetter, S.; Wartha, O.; Peter, R.; et al. Reference values and early determinants of intra-abdominal fat mass in primary school children. *Horm Res Paediatr*. 2011; 75: 412–422.
48. Reinehr, T.; Wunsch, R. Relationships between cardiovascular risk profile, ultrasonographic measurement of intra-abdominal adipose tissue, and waist circumference in obese children *Clin Nutr*. 2010; 29, 24–30.
49. Juonala, M.; Magnussen, C.G.; Berenson, G.S.; Venn, A.; Burns, T.L.; Sabin, M.A.; Srinivasan, S.R.; Daniels, S.R.; Davis, P.H.; Chen, W.; et al. Childhood adiposity, adult adiposity, and cardiovascular risk factors. *N. Engl. J. Med*. 2011; 365: 1876–1885.
50. Tracy L. Schumacher, Tracy L. Burrows, Dylan P. Cliff, Rachel A. Jones, Anthony D. Okely, Louise A. Baur, Philip J. Morgan, Robin Callister, May M. Boggess and Clare E. Collins. Dietary Intake Is Related to Multifactor Cardiovascular Risk Score in Obese Boys. *Healthcare*. 2014; 2: 282-298
51. Ahern, D.; Dixon, E. Pediatric hypertension: A growing problem. *Prim. Care*. 2015; 42: 143–150.
52. Cottin, S.C.; Sanders, T.A.; Hall, W.L. The differential effects of EPA and DHA on cardiovascular risk factors. *Proc. Nutr. Soc*. 2011; 70: 215–231.
53. Horta, B.L.; Victora, C.G. Long-term effects of breastfeeding: A systematic review.
54. D'Adamo, E.; Guardamagna, O.; Chiarelli, F.; Bartuli, A.; Liccardo, D.; Ferrari, F.; Nobili, V. Atherogenic dyslipidemia and cardiovascular risk factors in obese children. *Int J Endo*. 2015; 912047: 1–912047: 9.
55. Cook, S.; Kavey, R.E. Dyslipidemia and pediatric obesity. *Pediatr Clin N Am*. 2011; 58: 1363–1373.
56. Pacifico, L.; Giansanti, S.; Gallozzi, A.; Chiesa, C. Long chain  $\omega$ -3 polyunsaturated fatty acids in pediatric metabolic syndrome. *Mini Rev. Med Chem*. 2014; 14: 791–804.

57. Saito, E.; Okada, T.; Abe, Y.; Kuromori, Y.; Miyashita, M.; Iwata, F.; Hara, M.; Ayusawa, M.; Mugishima, H.; Kitamura, Y. Docosahexaenoic acid content in plasma phospholipids and desaturase indices in obese children. *J. Athero Thromb.* 2011; 18: 345–350.
58. Nobili, V.; Alkhoury, N.; Alisi, A.; Della Corte, C.; Fitzpatrick, E.; Raponi, M.; Dhawan, A. Nonalcoholic fatty liver disease: A challenge for pediatricians. *JAMA Pediatr.* 2015; 169: 170–176.
59. Zhang, H.; Yang, H.; Lai, C.; Xu, X.; Huang, K.; Fu, J. Quantitative relationship between liver fat content and metabolic syndrome in obese children and adolescents. *Clin Endo.* 2015.
60. Verduci, E.; Radaelli, G.; Scaglioni, S.; Toni, N.; Banderali, G.; Riva, E. Liver echogenicity and polyunsaturated fatty acids (Pufas) in plasma phospholipids of obese children with SNP 276G>T at adiponectin gene. In Proceedings of the ESPGHAN—44<sup>th</sup> Annual Meeting, Sorrento, Italy, 25–28 May 2011.
61. Nobili, V.; Alisi, A.; Della Corte, C.; Rise, P.; Galli, C.; Agostoni, C.; Bedogni, G. Docosa hexaenoic acid for the treatment of fatty liver: Randomised controlled trial in children. *Nutr Metab Cardiovasc Dis.* 2013; 23: 1066–1070.
62. Nobili, V.; Carpino, G.; Alisi, A.; de Vito, R.; Franchitto, A.; Alpini, G.; Onori, P.; Gaudio, E. Role of docosahexaenoic acid treatment in improving liver histology in pediatric non alcoholic fatty liver disease. *PLoS ONE* 2014; 9, e88005:1–e88005: 9.
63. Nobili, V.; Bedogni, G.; Alisi, A.; Pietrobattista, A.; Alterio, A.; Tiribelli, C.; Agostoni, C. A protective effect of breastfeeding on the progression of non-alcoholic fatty liver disease. *Arch Dis Child.* 2009; 94, 801–805.
64. Verduci, E.; Lassandro, C.; Radaelli, G.; Soldati, L. Docosahexaenoic acid and non-alcoholic fatty liver disease in obese children: A novel approach? 2015.
65. Stapleton P. Beliefs about Causes of Obesity: A Comparison of Australian Doctors, Psychologists and Community Members. *J Obes Weight Loss Ther* 2015; 5: 246.
66. Faghri P, Stratton K, Momeni K. Sedentary Lifestyle, Obesity, and Aging: Implication for Prevention. *J Nutr Disorders Ther.* 2015; 5: 119.
67. Priya Kumar and Vilaas Raaj. Childhood Obesity: World's Messy Issue. *J Diab Metab.* 2015; 6: 4: 1-2.
68. Brumpton B, Langhammer A, Romundstad P, Chen Y, Mai X-M. General and abdominal obesity and incident asthma in adults: the HUNT study. *Euro Resp J* 2013; 41, 2: 323–329.
69. Mitchell E, Beasley R, Bjorksten B, Crane J, Garcia-Marcos L, Keil U. The association between BMI, vigorous physical activity and television viewing and the risk of symptoms of asthma, rhino conjunctivitis and eczema in children and adolescents: ISAAC Phase Three. *Clin & Exp Allergy.* 2013; 43; 1: 73–84.
70. Brüske I, Flexeder C, Heinrich J. Body mass index and the incidence of asthma in children. *Curr Opin Allergy Clin Immunol,* 2014; 14: 155–160.
71. Holguin F, Bleeker ER, Busse WW, Calhoun WJ, Castro M, Erzurum SC, Fitzpatrick AM, Gaston B, Israel E, Jarjour NN: Obesity and asthma: an association modified by age of asthma onset. *J Allergy Clin Immunol.* 2011; 127: : 1486–1493.
72. Griffiths C, Gately P, Marchant PR, Cooke CB: Cross-sectional comparisons of BMI and waist circumference in British children: mixed public health messages. *Obesity (Silver Spring)* 2012; 6: 1258–1260.
73. Yael Latzer and Daniel Stein. A review of the psychological and familial perspectives of childhood obesity. *J Eat Disor* 2013; 1: 7.
74. De Onis M, Blössner M, Borghi E. Global prevalence and trends of overweight and obesity among preschool children. *Am J Clin Nutr.* 2010; 92: 1257-1264.

75. Zimmet P, Alberti G, Kaufman F, Tajima N, Silink M, Arslanian S, et al. The metabolic syndrome in children and adolescents. *Lancet*. 2007; 369: 2059-2061.
76. Galbo T, Shulman GI. Lipid-induced hepatic insulin resistance. *Aging (Albany NY)*. 2013; 5: 582-583.
77. D. Weghuber, S. Zelzer, I. Stelzer, K. Paulmichl, D. Kammerhofer, W. Schnedl, D. Molnar, H. Mangge, High risk vs. “metabolically healthy” phenotype in juvenile obesity - neck subcutaneous adipose tissue and serum uric acid are clinically relevant. *Experimental and clinical endocrinology & diabetes: official journal, German Society of Endocrinology [and] German Diabetes Association*. 2013; 121: 384-390.
78. Finucane, M.; Stevens, G.; Cowan, M. National, regional, and global trends in body-mass index since 1980: Systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. *Lancet* 2011; 377: 557–567.
79. Sallis, J.F.; Floyd, M.F.; Rodríguez, D.A.; Saelens, B.E. Role of built environments in physical activity, obesity, and cardiovascular disease. *Circulation* 2012; 125: 729–737.
80. Fogelholm, M. Physical activity, fitness and fatness: Relations to mortality, morbidity and disease risk factors. A systematic review. *Obes Rev*. 2010; 11: 202–221.
81. Cecchini, M.; Sassi, F.; Lauer, J.A.; Lee, Y.Y.; Veronica, G.-B.; Chisholm, D. Tackling of unhealthy diets, physical inactivity, and obesity: Health effects and cost-effectiveness. *Lancet* 2010; 376: 1775–1784.
82. Rasmussen F, Hancox RJ (2014) Mechanisms of obesity in asthma. *Curr Opin Allergy Clin Immunol*. 2014; 14: 35–43.
83. Delavari, M.; Sønderlund, A.L.; Swinburn, B.; Mellor, D.; Renzaho, A. Acculturation and obesity among migrant populations in high income countries—A systematic review. *BMC Pub Health*. 2013; 13: 1471-2458.
84. Gortmaker, S.; Swinburn, B.; Levy, D.; Carter, R.; Mabry, P.L.; Finegood, D.T.; Huang, T.; Marsh, T.; Moodie, M.L. Changing the future of obesity: Science, policy, and action. *Lancet*. 2011; 378: 838–847.
85. Stevens, G.; Singh, G.; Lu, Y.; Danaei, G.; Lin, J.; Finucane, M.; Bahalim, A.; McIntire, R.; Gutierrez, H.; Cowan, M.; et al. National, regional, and global trends in adult overweight and obesity prevalences. *Popul. Health Metr*. 2012; 10: 1478-7954.
86. Summerbell, C.D.; Moore, H.J.; Vogeles, C.; Kreichauf, S.; Wildgruber, A.; Manios, Y.; Douthwaite, W.; Nixon, C.A.; Gibson, E.L. Evidence-based recommendations for the development of obesity prevention programs targeted at preschool children. *Obes Rev*. 2012; 13: 129–132.
87. Delavari Maryam, Anders Larrabee Sonderlund, David Mellor, Mohammadreza Mohebbi and Boyd Swinburn. Migration, Acculturation and Environment: Determinants of Obesity among Iranian Migrants in Australia. *Int. J. Environ. Res. Public Health*. 2015; 12: 1083-1098.
88. Kolasa KM, Collier DN, Cable K. Weight loss strategies that really work. *J Fam Pract*. 2010; 59: 378-385.
89. Casazza K, Fonatine KR, Astrup A, et al. Myths, presumptions, and facts about obesity. *N Engl J Med*. 2013; 368: 446-454.
90. Orlistat (Xenical) prescribing information. San Francisco, Calif.: Genentech, Inc., 2012.
91. Clegg ME, Golsorkhi M, Henry CJ. Combined medium-chain triglyceride and chilli feeding increases diet-induced thermogenesis in normal weight humans. *Eur J Nutr*. 2013; 52(6): 1579-85.
92. Whittle AJ, Lopez M, and Vidal-Puig A. Using brown adipose tissue to treat obesity-the central issue. *Trends Mol Med*. 2011; 17(8): 405-11.
93. Williams, D.J., D. Edwards, I. Hamernig, L. Jian, A.P. James, S. K. Johnson and Tapselle, L.C. Vegetables contain-

- ing phytochemicals with potential anti-obesity properties: A review. *Food Res Int.* 2013; 52: 323-333.
94. Holubkova, A., A. Penesovab, E. turdika, S. Mo ovskaa and Mikuovaa, L. Phytochemicals with potential effects in metabolic syndrome prevention and therapy. *Acta Chimica Slovaca.* 2012; 5: 186-199.
95. Chandrasekaran CV, M.A.Vijayalakshmi, K. Prakash, V.S. Bansal, Meenakshi J and Amit, A. Herbal Approach for Obesity Management. *Am J Plant Sci.* 2012; 3: 1003-1014.
96. Ong, S.L., S. Paneerchelvan, H.Y. Lai and Rao, N.K. In vitro lipase inhibitory effect of thirty two selected plants in Malaysia. *Asian J Pharm Clin Res.* 2014; 7: 19-24.
97. Dhara, R., P. Dhar and Ghosh, M. Dietary effects of diacylglycerol rich mustard oil on lipid profile of normocholesterolemic and hypercholesterolemic rats. *J Food Sci Technol.* 2013; 50: 678-686.
98. Maruyama, C., N. Kikuchi, Y. Masuya, S. Hirota, R. Araki and Maruyama, T. Effects of green-leafy vegetable intake on postprandial glycemic and lipidemic responses and  $\alpha$ -tocopherol concentration in normal weight and obese men. *J. Nutr Sci Vitaminol (Tokyo).* 2013; 59: 264-271.
99. Chang, H.C., C.N. Huang, D.M. Yeh, S.J. Wang, C.H. Peng and Wang, C.J. Oat prevents obesity and abdominal fat distribution, and improves liver function in humans. *Plant Foods Hum Nutr.* 2013; 68: 18-23.
100. Wu, T., Q. Tang, Z. Gao, Z. Yu, H. Song, X. Zheng and Chen, W. Blueberry and mulberry juice prevent obesity development in C57BL/6 mice. *PLoS One.* 2013; 8: e77585.
101. Andersen, C., S. Rayalam, M.A. Della-Fera and Baile, C.A. Phytochemicals and adipogenesis. *Biofactors.* 2010; 36: 415-422.