Available online at http://www.journalijdr.com

IJDR

ISSN: 2230-9926

International Journal of Development Research
Vol. 09, Issue, 04, pp. 27297-27311, April, 2019

RESEARCH ARTICLE OPEN ACCESS

MANAGEMENT OF OBESITY IN CHILDREN

***Dr. Lamyaa Imran Ali, Dr. Abdulrahman Ismael Dakhel and Dr. Tareq Kannan Mohammed**

Central Teaching Paediatric Hospital, Baghdad, IRAQ

ARTICLE INFO

Article History:

Received 09th January, 2019
Received in revised form
21st February, 2019
Accepted 06th March, 2019
Published online 30th April, 2019

Key Words:

Obesity, Child, Treatment.

ABSTRACT

Obesity and overweight is the most prevalent nutritional disorder among children and adolescents in the United States and worldwide. Approximately 21-24% of American children and adolescents are overweight, and another 16-18% is obese; More than one billion people worldwide are affected by obesity. The study was a case-control study.

- Overweight/obesity were more common among males, no significant correlation was found with sex.
- There was highly significant protective effect of breastfeeding against development of obesity.
- Limitation of physical activity and watching TV for > 2 hours duration is a significant risk factor for overweight/obesity.
- There was no significant positive correlation between birth weight and/or history of prematurity and overweight/obesity.
- There was a significant correlation observed among paternal education, formula feeding, number of meals or snacks/day, and frequency and amount of consumption of saturated fat, dairy products, fruits/vegetables and grains with overweight/obesity.
- There was a significant correlation between high income and overweight/obesity.
- Although psychological effect was obvious on children in the study, this could not be assessed statistically.

Citation: Dr. Lamyaa Imran Ali, Dr. Abdulrahman Ismael Dakhel and Dr. Tareq Kannan Mohammed. 2019. "Management of obesity in children", *International Journal of Development Research*, 09, (04), 27297-27311.

INTRODUCTION

Obesity and overweight is the most prevalent nutritional disorder among children and adolescents in the United States and worldwide. Approximately 21-24% of American children and adolescents are overweight, and another 16-18% is obese; More than one billion people worldwide are affected by obesity (Kimm SY 2015). Obesity in children is a complex disorder. Its prevalence has increased so significantly in recent years that many consider it a major health concern of the world (Fiore, 2006). The National Health and Nutrition Examination Survey (NHANES) indicates that the prevalence of obesity is increasing in all pediatric age groups, in both sexes, and in various ethnic and racial groups. Many factors, including genetics, environment, Socioeconomic status, Family size, parental/sibling obesity, television viewing, computer games and other sedentary behavior, lifestyle, and eating habits, are believed to play a role in the development of obesity (Biddle, 2004).

****Corresponding author: Dr. Lamyaa Imran Ali,***
Central Teaching Paediatric Hospital. Baghdad, IRAQ

However, more than 90% of cases are idiopathic; less than 10% are associated with hormonal or genetic causes (Fiore, 2006).

Definitions 4 no single definition of obesity in childhood and adolescence has gained universal approval. For children and adolescents 2 to 20 years, the term "overweight" rather than "obesity" is currently used by the Centers for Disease Control and Prevention (CDC) and generally defined as a BMI at or above the 95th percentile of sex-specific BMI-for-age values from the 2000 CDC growth charts, In children and adolescents, the term "at risk of overweight" is defined as a BMI between the 85th and 95th percentiles.

Body mass index: A variety of methods are available to measure body fatness and body thinness (World Health Organization, 2009). Commonly used techniques for the accurate estimation of body fatness include underwater weighing, dual-energy X-ray absorptiometry (DXA), total body water, total-body electrical conductivity, total body potassium, and computed tomography, however, the use of most of these methods is limited to research settings because of their complexity and cost (Goran, 1998). The most frequently used tools in public health evaluations and clinical screening are anthropometric-based measurements such as skin fold-thickness or circumference measurements or various height and weight indexes such as ht/wt or BMI index (WHO, 2009). BMI is the one most commonly recommended for screening overweight and obesity in children and adolescents (Goran, 1998). BMI is calculated as weight in kilograms divided by height in meters squared or by dividing weight in pounds by height in inches squared and multiplying by a conversion factor of 703 (Keys, 1972). Normal values for BMI vary with age, sex, and pubertal status, and standard curves representing the 5th through the 95th percentiles for BMI in childhood and adolescence were generated using data from the 1988-1994NHANE, Consensus committees have recommended that children and adolescents be considered overweight or obese if the BMI exceeds the 85th or 95th percentiles, or exceeds 30 kg/m^2 at any age (Fiore, 2006).

Epidemiology: The rapid increase in the prevalence of childhood obesity has alarmed public health agencies, health care clinicians, health care researchers and general public.

United States statistics: (Ogden, 2006) On the basis of measured heights and weights from nationally representative samples of US children assessed approximately every 5 years, obesity prevalence has increased from 5% in 1963 to 1970 to 17% in 2003 to 2004.An estimated17%of children and adolescents 2 to19 years of age are overweight according to the 2003–2004 NHANES. Extreme obesity is a frequent finding in children and adolescent. The numbers show a shift towards extreme obesity.

International statistics (Eneli, 2008): International data reporting regarding childhood obesity varies, and accuracy may be less than optimal; however, 77% of the countries analyzed, the prevalence rate for children who were overweight was at least 10%.

Racial, sexual, and age differences in incidence (Baskin, 2005): Race and ethnicity are associated with increased rates of obesity in children and adolescents. Puerto Rican, Cuban American, and Native American preschoolers have an increased incidence of obesity; black, Native American, Puerto Rican, Mexican, and native Hawaiian school-aged children have the highest rates of obesity. Ethnic disparities in overweight prevalence are also observed in male or female children and adolescents, as in adults. Adolescent obesity is predictive of adult obesity, with 80% of teenagers who are obese continuing on to be obese as adults. Obesity is more likely to occur during specific periods of life, such as when children are aged 5-7 years and during adolescence (Ogden, 2006).

Etiology and Pathophysiology

Physiology: Obesity results from an imbalance between energy intake and energy expenditure. Excesses in adipose tissue mass also can be viewed as a pathological derangement in the feedback between energy intake and expenditure (Eckel, 2003). The development of adipose tissue in the fetus begins in the mid- to late third trimester of pregnancy. Early in life, the ability of adipose tissue stromal cells to differentiate into triglyceride-filled adipocytes is facilitated. Critical periods of adipocyte differentiation may include infant feeding, puberty, the administration of steroids, and by peroxisome proliferator-activated receptor-mediated adipocyte differentiation in adulthood (Ivkovic-Lazar, 2003). However, after suction lipectomy, the failure of adipocyte volume to change suggests that a new program of adipocyte differentiation becomes operational to return fat mass to baseline (Yost, 1993). Understanding the regulation of energy intake requires differentiating the short-term signals that control hunger, food intake, and satiety, as well as the long-term signals that relate to the defense of energy stores, lean tissue, or both. In short-term regulation, gastrointestinal signals provide important input to the brain. For the most part, hormones released from the stomach and intestine that affect food ingestion are inhibitory (Woods, 2004). One example is the recently identified polypeptide YY3-36, which is produced by the L cells of the small intestine.When YY3-36 is infused into lean or obese subjects, reductions in food intake by _30% are seen (Batterham, 2003). Ghrelin is an exception. This peptide is produced by the stomach and proximal small intestine, and its release stimulates food intake.

Ghrelin declines after a meal and rises before the next meal. Ghrelin is elevated in Prader-Willi syndrome (Cummings, 2002). Which is a genetic form of obesity accompanied by marked hyperphagia. Ghrelin is dramatically reduced after gastric bypass surgery (Cummings, 2002). Adipose tissue is critically involved in feedback regulation of energy balance by the production of a number of peptide hormones, and leptin and adiponectin are 2 of the most important. The absence of leptin produces massive obesity, and treatment of leptin-deficient individuals reduces food intake and body weight (Farooqi, 1999). Adiponectin is the most abundant hormone from fat cells, increases insulin sensitivity, and appears to be a cytokine that is anti-inflammatory (Ouchi, 2003). To a large extent, the signals directed by leptin and other adipose tissue–derived peptides are integrated in the hindbrain and mid-brain (dorsomedial hypothalamus) through various signals (monoamines, neuropeptide-Y, agouti-related peptide, melanocyte–stimulating hormone), which in turn send efferent signals for food seeking and modulation of function of various organs, including the pancreas and muscle (glycerol 3-phosphate dehydrogenase) (McMinn, 2000). Metabolism of the adrenal steroid in adipose tissue may provide a mechanism for the increase in visceral fat. When the enzyme 11-hydroxysteroid dehydrogenase type-1, which converts cortisol to the inactive cortisone in fat cells, is genetically disrupted, mice develop visceral obesity, This pathophysiology may also apply to humans (Masuzaki, 2001). Studying the relationship between the Circadian rhythm, sleep deprivation, obesity, and diabetis (the Circadian integration of metabolism and energetic) has been showen that eat at night might lead to increase body mass by shifting biological clock despite agood energy balance. Now it is possible to genetically interfere with these processes leading to transformation of biological timing the so called clock gene (Bass, 2010). New studies of the genetic factor association with obesity showed 18 new loci in addition to the previously confirmed 14 already known obesity susceptibility loci, these finding identify multiple loci that influence body fat distribution independent of overall adiposity and reveal strong gene-by-sex interaction and provide new insight into the rgeulation of human body weight (Speliotes, 2010).

Etiology: The development of childhood overweight is a multifactorial disorder involving genetic, biological, social, and environmental pathways (Salsberry, 2005). Prenatal characteristics and early-life feeding factors, including maternal weight before and during pregnancy, maternal smoking during pregnancy, infant feeding, and birth weight, have been associated with overweight during childhood (Donohoue, 2004).

Diet: Over the last decades, food has become more affordable to larger numbers of people as the price of food has decreased substantially relative to income and the concept of 'food' has changed from a mean of nourishment to a marker of lifestyle and a source of pleasure. It takes between 1–2 hours of extremely vigorous activity to counteract a single large-sized (i.e., ≥785 kcal) children's meal at a fast food restaurant. Frequent consumption of such a diet can hardly be counteracted by the average child or adult (Dehghan, 2005).

Calories intake (Dehghan, 2000) : Although overweight and obesity are mostly assumed to be the result of increase in caloric intake and there is no enough supporting evidence for such phenomenon; soft drinks intake has been associated with the epidemic of obesity and type 2 diabetes among children Food frequency methods measure usual diet, but estimate caloric intake poorly. Other methods such as 24-hour recall or food diaries evaluate caloric intakes more accurately, however, estimate short-term but not long-term intake.

Fat Intake: While for many years it has been claimed that the increase in pediatric obesity has happened because of an increase in high fat intake, contradictory results have been obtained by cross-sectional and longitudinal studies (Cole, 2000). Although fat eaten in excess leads to obesity, there is no strong enough evidence that fat intake is the chief reason for the ascending trend of childhood obesity (Troiano, 2000).

Other dietary factors (Carruth, 2001): There is a growing body of evidence suggesting that increasing dairy intake by about two servings per day could reduce the risk of overweight by up to 70%. In addition, calcium intake was associated with 21% reduced risk of development of insulin resistance among overweight young adults and may reduce diabetes risk. While it is possible that drinking soda instead of milk would result in higher intake of total energy, it cannot be concluded definitively that sugar containing soft drinks promote weight gain because they displace dairy products.

Physical Activity (Tremblay, 2003): It has been hypothesized that a steady decline in physical activity among all age groups has heavily contributed to rising rates of obesity all around the world. Physical activity has been found to strongly influence weight gain in a study of monozygotic twins. Numerous studies have shown that sedentary behaviors like watching television and playing computer games are associated with increased prevalence of obesity. Furthermore, parents report that they prefer having their children watch television at home rather than play outside unattended because parents are then able to complete their chores while keeping an eye on their children.

Maternal Smoking: Several investigators have reported an increased risk of obesity in childhood or adulthood after intrauterine exposure to maternal cigarette smoking (Montgomery, 2002) Disproportionate weight gain associated with prenatal tobacco exposure may be due to the infant's self-regulation of food intake after birth, showing that small-for-date babies take more bottle milk per kilogram bodyweight than large-for-date babies. Alternatively, endocrine imbalances could occur through disrupted programming of endocrine axis at critical developmental periods, as proposed by the fetal origins hypothesis (that gestation is a critical period in the development of obesity) (Power, 2002).

Birth Weight: The attained birth weight is a result of the interactive effects of length of gestation, rate of fetal growth, adequacy of the intrauterine environment, uteroplacental function, and genetic potential (Parsons, 2001), with a higher prevalence of obesity seen for the lowest and highest birth weights, suggesting a more complex association between growth in utero and obesity. Lower birth weight, because of presumed intrauterine malnutrition and association with rapid weight gain, is associated with central adiposity, the metabolic syndrome (dyslipidaemia, insulin resistance, obesity and vascular disease), diabetes mellitus, and cardiovascular disease outcomes in adulthood. Infants large for gestational age, probably through consequences of maternal insulin resistance and glucose intolerance, are also at higher risk of future obesity (Gidding, 2006).

Early Life Feeding: After birth, influences within the early-life period are also linked to later overweight. In particular, the method of infant feeding, breast- or bottle-feeding, has been studied with variable results (Hediger, 2004 and Li, 2003). Some studies have found a dose-response effect, with an increased protective effect associated with increased duration of breast feeding. 35 Although breastfeeding is associated with higher blood cholesterol level at 1 year of age, it may also result in lower blood cholesterol levels in adults. Rapid weight gain during the first 4-6 months of life is associated with future risk of overweight (Li, 2003). Studies suggest that partially breastfed and formula-fed infants consume 20% more total calories perday than do exclusively breastfed infants.

Genetic syndromes associated with childhood obesity include the following:

- Prader-Willi syndrome
- Pseudohypoparathyroidism
- Laurence-Moon-Biedl (Bardet-Biedl) syndrome
- Cohen syndrome
- Down syndrome
- Turner syndrome

Hormonal disorders associated with childhood obesity include the following

- Growth hormone deficiency
- Growth hormone resistance
- Hypothyroidism
- Leptin deficiency or resistance to leptin action
- Glucocorticoid excess (Cushing syndrome)
- Precocious puberty
- Polycystic ovary syndrome (PCOS)
- Prolactin-secreting tumors

Medications that may cause weight gain in children and adolescents include the following:

- Cortisol and other glucocorticoids
- Megace
- Sulfonylureas

- Tricyclic antidepressants (TCAs)
- Monoamine oxidase inhibitors (MAOIs), such as phenelzine
- Oral contraceptives
- Insulin (in excessive doses)
- Thiazolidinediones
- Risperidone Clozapine

Prognosis: Overweight is associated with a number of comorbidities in children. Although the amount of information available about children is less than that about adults, it is clear that children experience many detrimental effects of overweight similar to adults. Table 1 presents comorbid conditions related to overweight that may present during childhood and adolescence.

Metabolic Syndrome: The National Cholesterol Education Program defined the metabolic syndrome (also known as the insulin-resistance syndrome) as a cluster of traits that include hyperinsulinemia, obesity, hypertension, and hyperlipidemia (DeFronzo, 1991). The prevalence of the metabolic syndrome in adolescents is 4% overall, but it is 30% to 50% in overweight children (Cook, 2003). The metabolic syndrome is believed to be triggered by combination of genetic factors with environmental factors such as excess calorie intake and reduced levels of physical activity. The primary cause of the syndrome appears to be obesity leading to excess insulin production, which is associated with an increase in blood pressure and dyslipidemia. The effects of increased insulin resistance are multiple and include increased hepatic synthesis of very-low-density lipoprotein, resistance of the action of insulin on lipoprotein lipase in peripheral tissues, enhanced cholesterol synthesis, increased high-density lipoprotein degradation, increased sympathetic activity, proliferation of vascular smooth muscle cells, and increased formation and decreased reduction of plaque (Berenson, 1998). Prospective data suggest that the most important risk factor for the metabolic syndrome is the rate of increase in BMI in youth (Pankow, 2004).

Type 2 Diabetes Mellitus (Pinhas-Hamiel, 1996): Concomitant with the rise in the prevalence of overweight and the metabolic syndrome has been a dramatic increase in type 2 diabetes mellitus in youth. Type 2 diabetes mellitus had been primarily a disease of adulthood; however, type 2 diabetes now occurs in adolescents typically with a BMI _30 kg/m2, a level that would be considered obese even by adult standards.
Accordingly, the dramatic increase in the prevalence of type 2 diabetes among adolescents with obesity is likely to be accompanied by a host of diabetic-related complications in adulthood and a reduction in life span. Although obesity, per se, is associated with a heightened risk of morbidity related to abnormalities in glucose homeostasis, recent data indicate that the rate of increase in BMI during adolescence may also represent a significant risk factor for diabetes (Tirosh, 2011).

Inflammation (Daneils, 2005): The association of obesity and inflammation is well recognized in adults. Data on this association in youth are also emerging. In general, inflammation occurs through the activation of the mononuclear phagocytes, which leads to the upregulation of interleukin-1, an upstream regulator with many downstream effects. In the liver, upregulation of interleukin-1 leads to an increase in acute-phase reactants, such as C-reactive protein. Inflammation also increases oxidant stresses, common in obesity, in which free radicals are generated in excess of the ability to detoxify them. This may lead to vascular damage over time. Insulin resistance is aproinflammatory condition, increasing tumor necrosis factor-and other cytokine production.

Cardiovascular Abnormalities: It is well recognized that CVD causes a substantial proportion of excess mortality in overweight individuals, studies have shown convincingly that obesity during childhood and adolescence is a determinant of anumber of cardiovascular risk factors, including atherogenic dyslipidemia (increased triglycerides, lowered high-density lipoprotein), hypertension, left ventricular hypertrophy, and atherosclerosis (Daniels, 1998), increased BMI consistently has been shown to be associated with higher blood pressure. Left ventricular hypertrophy has also been related to overweight in children. It has been shown that lean body mass, fat mass, and systolic blood pressure were independently associated with left ventricular mass in children and adolescents (Berenson, 1998).

Obstructive sleep apnea: Obstructive sleep apnea is also associated with obesity in children and adults. Increased BMI was related to an increased risk of obstructive sleep apnea in children and adolescents. They also showed that obstructive sleep apnea was associated with increased left ventricular mass index in a pediatric population (Amin, 2002).

Psychosocial Abnormalities: Psychosocial dysfunction in individuals who have obesity in childhood and adolescence is a serious concern

- The causal pathways by which psychological disturbances exert influence on body weight are unclear, although the association is likely to be complex rather than simple (Friedman, 1995).
- The best-studied area is depression.
- Many studies have confirmed the association between depression and subsequent obesity (Goodman, 2002).
- The development of overweight also may be related to subsequent psychosocial difficulties.

One of the primary mediators of the psychopathological relations with obesity is compromised peer relationships.49 Overweight children have fewer friends, and social network mapping suggests that normal-weight children have more relationships with a central network of children, whereas overweight children appear to have more isolated and peripheral relationships. In addition to having fewer friends, being teased about weight is another important mediator of psychosocial distress. Teasing over-weight youth has been shown to be associated with an increase in both their suicidal ideation and number of suicide attempts (Eisenberg, 2003).

Orthopedic disorders: Numerous orthopedic disorders, including genu valgum, slipped capital femoral epiphysis, and tibia vara, are observed more commonly in children with obesity. Excess weight in young children can cause bowing of the tibia and femur; the resulting overgrowth of the proximal tibial metaphysis is called Blount disease.

Liver and gallbladder dysfunction: Evidence of liver dysfunction, with elevated plasma concentrations of transaminases, is observed in 20% of children with obesity; the liver dysfunction most commonly reflects hepatic steatosis, but cirrhosis may develop in rare instances. Vitamin E supplements may be effective in reversing this so-called steatohepatitis, suggesting that the disorder reflects a relative state of vitamin E deficiency. Cholelithiasis is more common in adults with obesity than in adults with normal weight. Although gallstones are unusual in childhood, nearly one half of all cases of cholecystitis in adolescents are associated with obesity. Cholecystitis may be even more common during rapid weight loss, particularly with very controlled–energy diets (Di Sario, 2007).

Universal Assessment of Obesity Risk (Sarah, 2007): These recommendations support a shift from simple identification of obesity, which often occurs when the condition is obvious and intractable, to universal assessment, universal preventive health messages, and early intervention. If primary care providers are to have an impact on the childhood obesity epidemic, then their best approach is assessment of obesity risk for all patients, with anticipatory guidance on healthy behaviors to minimize that risk. The work of the expert committee and writing groups addresses all stages of care, from normal-weight, low-risk children to severely obese children. Figure 1 presents an overview of the process to assess obesity risk. BMI is the initial screen that should be calculated at each well-child visit and should serve as the starting point for classification of health risks. Children in the healthy-weight category (BMI of 5th–84th percentile) have lower risks, although parental obesity, family medical history, and current diet and physical activity behaviors may alter that assessment. These children and their families should receive support in maintaining or establishing healthy lifestyle (prevention) behaviors. The likelihood of health risks increases in the 85th to 94th percentile (overweight) category and again is influenced by parental obesity, family medical history, and current lifestyle habits, as well as BMI trajectory and current cardiovascular risk factors. Some of these children should receive prevention counseling, whereas others should receive more-active intervention. Children with a BMI above the 95th percentile (obese) are most likely to have obesity-related health risks, and should be encouraged to focus on weight control practices. Providers must use clinical judgment in assessing health risks, because no formula exists that can integrate BMI pattern, family background, and health behaviors to determine future weight and health.

Chronic Care Model (Bodenheimer, 2002): The expert committee recommendations are comprehensive and ambitious.Health care-centered efforts alone cannot effect change, but they can complement and potentially enhance evolving public health efforts, such as school wellness policies, parks and recreation programs, and shifts in child-targeted food advertisements. In addition, health care provider offices and health care systems will need to change, in many cases, to implement these recommendations.These recommendations can serve as guides that will improve as new information becomes available. The chronic care model is a new structure that integrates community resources, health care, and patient self-management to provide more-comprehensive and more-useful care. This paradigm offices linked to community resources, such as exercise programs; support for self management, which requires educating patients and families about assessment and monitoring; an expanded practice team that supports patient self-management and monitors adherence to evidence-based care pathways; and clinical information systems that can remind the team of routine tests and treatments and can monitor the practice's adherence to goals. In this model, practices plan a change and a method to measure that change, implement the change, and then examine the measure of change. The plan is modified depending on how well the goals are met. The chronic care model has obvious applications in childhood obesity, and several large health maintenance organizations have initiated some of these approaches. Several programs have linked to community efforts, such as community-based exercise programs. The care model for obesity recognizes the importance of changes in the school, worksite, and community. Figure 2 shows how the environment and the medical system support the patient and the family in their management of the condition (Sarah, 2007).

Prevention

Given the difficulty of behavior-based weight loss and subsequent weight maintenance and the expense and potential harm of medication and surgery, obesity prevention should be a public health focus. Efforts must begin early in life, because obesity in childhood, especially among older children and those with more-severe obesity, is likely to persist into adulthood (Daneils, 2005). Therefore, childhood represents an important opportunity to establish healthy eating and activity behaviors that can protect children against future obesity. Pediatric providers are accustomed to provide guidance on nutrition in early childhood routinely. In addition, they know the family's medical history and social and behavioral interactions. They are well positioned to guide families in the areas of eating and activity. The targets of obesity prevention should be all children, starting at birth. Lifestyle behaviors to prevent obesity, rather than intervention to improve weight, should be aimed at children with healthy BMIs (5th–84th percentile) and some children with BMIs in the overweight category (85th–94th percentile), depending on their growth pattern and risk factors. Clinicians should be aware of the increased risk of obesity for children with obese parents and those whose mothers had diabetes mellitus during the child's gestation.

The complexity of obesity prevention lies less in the identification of target health behaviors and much more in the process of influencing families to change behaviors when habits, culture, and environment promote less physical activity and more energy intake. The prevention writing group has provided suggestions on how to interact with families to promote target behaviors and how to create office systems that support the clinician's ongoing commitment to obesity prevention (Daniels, 1998). Potential interventions for obesity in youth span a continuum from preventing the development of obesity to treating established obesity and its complications. Treatment of obese children, can be a strategy for preventing adult obesity. Here, obesity prevention refers to avoiding the occurrence of obesity during childhood and adolescence. Obesity prevention includes both population-oriented and individual-oriented approaches, with an emphasis on population-oriented approaches. Population approaches focus on environmental and policy change (upstream approaches) that have the broadest reach and the lowest intensity and cost and are critical for reaching the least-advantaged population segments (Figure). Treatment interventions(downstream approaches) are individually oriented, usually delivered in specialty care, primary care, and health systems, and are more familiar to health professionals than is population-oriented intervention. Treatment approaches are important and appropriate for children who are already overweight. Individually oriented prevention approaches that focus on children at high risk of becoming overweight resemble treatment approaches in their process and delivery setting. Individually oriented prevention strategies may be highly intensive, costly, and have low reach in terms of the numbers that can potentially be served. Whereas formal treatment for overweight children and adolescents is delivered almost exclusively in medical settings, the settings in which preventive interventions function extend from medical settings to families and communities. Settings for preventive interventions include schools and other group childcare settings such as day care and Head Start services, maternal and child healthcare clinics, and the Supplemental Nutrition Program for Women, Infants and Children (WIC) programs. A range of theories is relevant, from those addressing social and community change to those concerned with family functioning to individual cognitive and behavioral processes (Kumanyika, 2003). First, the interventions must be designed with specific knowledge of the target audience and the best way to engage them in the process of change, whether the audience is individuals, families, organizations, or governments.

This approach includes identifying the specific issues, social and cultural values, and incentives and disincentives that are most salient to the audience in question; factors that will increase the probability that the individual or group will pay attention to, participate in, and be motivated by the change process; exposure to models and practical experiences with regard to the actions relevant to change; and facilitators and barriers to change in the relevant structural and social environments and in the interactions among individuals, systems, and groups. Identification of relevant differences among subgroups within the population helps to guide the nature of the intervention. "Tailoring" is a deliberate attempt to account for important individual or subgroup variables when developing program messages or intervention strategies. Many elements of tailoring are related to cultural variables Needs assessment and process evaluation to identify elements of these focal points are critical in the development of effective intervention approaches. Defining an initial focal point is one stage of tailoring; the second stage involves further tailoring to individuals within this focal point on the basis of additional variables (eg, household composition for parents, and regulatory orientation policy makers). The definition of the focal points themselves may change as the heterogeneity within population groups becomes evident56.

Treatment: The primary goal of obesity treatment is improvement of long-term physical health through permanent healthy lifestyle habits. Implementation of these habits alone will lead to improved weight (weight loss or weight maintenance during linear growth) for some children, but other children and youths may need additional focused efforts to achieve negative energy balance. Others may need additional help with behavior modification strategies to develop and to sustain healthy habits. Emotional health (good self-esteem and appropriate attitudes toward food and body) is also an important outcome.The establishment of permanent healthy lifestyle habits is a good outcome, regardless of weight change, because of the long-term health benefits of these behaviors. Improvement in medical conditions is also an important sign of long-term health benefits.

The metric for improved weight is BMI percentile, generally to _85th percentile. Serial weight measurements can reflect energy balance in the short term. Weight maintenance leads to reductions in absolute BMI because of ongoing linear growth, and even slow weight gain can result in lower BMI percentiles because the BMI for a given percentile curve increases with age. In general, younger and more mildly obese children should change weight more gradually than older and severely obese youths.

Dietary Management: Age-specific dietary modification is the cornerstone of treatment. The major goals in dietary management are to provide appropriate caloric intake, provide optimum nutrition for the maintenance of health and normal growth, and help the child to develop and sustain healthful eating habits. The most recent Dietary Reference Intakes recommend a fat intake of 30% to 40% kcal in children 1 to 3 years old, with a reduction to 25% to 35% in children 4 to 18 years old (compared with 20% to 35% in adults); a carbohydrate intake of 45% to 65% kcal in all children and adults; and protein intakes of 5% to20% kcal in children 1 to 3 years old with gradual increase to10% to 30% kcal in children 4 to 18 years old (compared with 10% to 35% kcal in adults) (Panel on Macronutrients, 2002). Because it is difficult for parents to judge calorie intake and energy expenditure on a regular basis, it is necessary to guide parents towards the diet and physical activity patterns of their children. Counseling and recommendations must be made within the context of the family's culture, living environment, and socioeconomic status. Most dietary strategies for weight loss emphasize balance, variety, and adequacy of the overall eating pattern. Dietary recommendations also emphasize reducing the number of meals eaten outside the home, planning for healthy snacks, offering healthier, low-calorie food choices (especially fruit and vegetables), and structuring eating times and places for family meals. Involving children in meal planning, shopping, gardening, and preparation of food has been promoted, along with including all caregivers (including grandparents) in helping the child to adhere to recommended consumption patterns and healthier food choices (US, 2005).

Physical Activity (Carnethon, 2003): Most reports of successful weight loss and maintenance emphasize the importance of incorporating regular physical activity into treatment programs. Children are similar to adults in that regular exercise provides additional health benefits for overweight individuals, including prevention of future risk acquisition, improved insulin sensitivity, blood pressure reduction, and improved socialization through group participation in activities. Regular physical activity is critical for the prevention of abnormal weight gain and weight maintenance. The current recommendation for the amount of physical activity is 30 to 60 minutes of regular exercise daily.
"Working up a sweat" during the activity suggests adequate effort expended. These recommendations apply to children of normal weight as well as to children who are overweight. Recommended activities must be enjoyable and congruent with the child's and family's lifestyle and be rewarding independent of the health benefit. A complementary approach is to restrict sedentary free-time activities to _2 h/d. Fitness levels vary significantly among overweight individuals. Whereas one child may not be able to walk several blocks without becoming short of breath, another may be adoept at playing sports. Other variables also influence the recommendation for a child's physical activity and exercise

program. Some may have easy access to recreational areas and play and exercise equipment, whereas others may not be allowed out of the house for safety reasons. Parental supervision and availability for participation vary greatly and must be considered.

Pharmacological Treatment: Data supporting the use of pharmacological therapy for pediatric overweight are limited and inconclusive (Yanovski, 2004). Sibutramine has been studied in a randomized controlled trial of severe obesity. It has been shown to be efficacious as compared with behavior therapy alone, but it may be associated with side effects including increases in heart rate and blood pressure (Berkowitz, 2003). Orlistat is approved for use in adolescence. The efficacy of orlistat has not been tested extensively in young patients. Orlistat is associated with gastrointestinal side effects and requires fat-soluble vitamin supplementation and monitoring (McDuffie, 2002). For rare genetic and metabolic disorders, pharmacological treatment may be useful. For example, recombinant leptin is useful in hereditary leptin deficiency. Octreotide may be useful in hypothalamic obesity (Lustig, 2002). Metformin, used to treat type 2 diabetes mellitus, has been used in insulin-resistant children and adolescents who are overweight, but long-term efficacy and safety are unknown (Freemark, 2001).

Surgical Treatment: Surgical approaches to treat severe adolescent obesity are being undertaken by several centers. Indications used include a BMI>40 kg/m2 and severe associated comorbidities, such as obstructive sleep apnea, type 2 diabetes mellitus, and pseudotumor cerebri (Inge, 2004). More severe elevation of BMI (>50 kg/m2) may be an indication for surgical treatment in the presence of less severe comorbidities such as hypertension and dyslipidemia, particularly if the degree of overweight hinders performing the activities of daily living. An experienced team approach including comprehensive medical and psychological evaluation is critical both for selection of appropriate candidates and for postoperative care that is sophisticated and often intense (Inge, 2004). Weight loss goals and reduction of morbidity are often achieved with gastric bypass surgery. The rates of short-term mortality appear to be low, but significant complications can occur. Intermediate and long-term outcomes, including information on malabsorption of critical nutrients, are unknown. Overall, surgical therapy should be reserved for full-grown adolescents with the severest obesity-related morbidity, offered only by experienced multidisciplinary teams, and presented to families with appropriate informed consent procedures (Inge, 2004).

Healthcare Delivery Systems (Sarah, 2007): Obesity treatment and prevention require a long-term care model. Substantial changes in the current healthcare delivery system are needed to accommodate the needs of long-term weight management for children as they grow. Children are at risk for not receiving appropriate intervention when physical growth and maturation occur simultaneously and when important lifelong nutrition and physical activity habits are formed. Emphasis should be placed on self-management, in which the child and his or her family (rather than the healthcare provider) set the goal. It is important that children and patients in treatment understand the implications of their choices through a problem-solving approach and that strategies be tailored to individual needs. The effectiveness of this long-term care model is also dependent on a comprehensive team approach that targets the individual, the family, and the many environmental influences affecting the child's behaviors.

Aims

- To raise awareness of the importance of undertaking population to the prevention of excess weight gain in children and adolescents;
- To describe considerations for undertaking obesity prevention overall and
- in key risk subgroups;
- To differentiate environmental and policy approaches to obesity prevention from those used in clinical prevention and obesity treatment;
- To identify potential targets of environmental and policy change using an
- ecological model that includes multiple layers of influences on eating and physical activity across multiple societal sectors;
- To highlight the spectrum of potentially relevant interventions and the nature of evidence needed to inform the population

MATERIALS AND METHODS

Study Design: The study was a case-control study.

Sampling technique: The sampling technique was convenient sampling.

Setting: The study was conducted in different localities in Iraq.

Baghdad: Endocrine clinic in central child teaching hospital and from al Eskan primary health care.

Diyala: Al mustafa primary health care, Al saray primary health care.

Al sulaimaniah: Ali kamal primary health care.

Al anbar: Al gatanah primary health care.

Al naseriah: Al fadhliah primary health care.

Time of data collection: Data collection was carried out for four hours a day, four days a week over the period between February 2010 till the end of November 2011.

Patients: After clarifying the purpose behind the study, assuring high confidentiality and having verbal consents from parents, the sample constituted of 200 children aged from 2-15 years who were patients visiting a health facility included in the study. 100 was obese BMI >95th centile for age percentiles charts for both males and females were considered by using WHO standards (the source of the charts used in this study CDC growth charts: national center for chronic disease prevention and health promotion.2000) Other 100 child were selected from children consulting the outpatient clinics Of ENT, DERMATOLOGY and DENTIST in central child teaching hospital regarding that they are not obese (BMI lie between the 5th and 85th centile for age and sex) and have no other illness.

Tool of Data Collection (questionnaire): The tool of data collection was a structured questionnaire form that was administered and filled by the research team members through direct interviews & examination to mothers or/and included children. This questionnaire was developed by the researcher and the supervisor and public health specialist. In addition to general demographic information (age and sex);

The following information were taken from mothers attending health centers with their children:-

Name, age, sex, residency, weight at birth (low, average, large), history of prematurity, maternal smoking during pregnancy, family history of obesity, family housing (owned or rented), number of rooms, number of people living in the house, crowding index (≤3 person/room: not crowded, >3person/room: crowded) (Ghosh, 2005). Family status (divorce, separated, live together, widow). Parental educational level (not educated, primary, secondary school and higher education). Mother and father education was coded as low (primary school or less) and high (at least 1 year of secondary schooling or higher) (Martorell, 2000). Parental employment , type of feeding (breast, mixed or formula fed), feeding habbits(number of meals per day(1, 2, 3), number of snacks per day (none,1, ≥ 2), Frequency and amount of consumption of soft drinks, were assessed by number of servings, number of 6-ounce servings of 100% fruit juices in excess of 1 serving per day (Kranz, 2004).

Concerning juice intake was classified as:

None
≤1 serving/day
>1 serving/day (excess juice)

Physical activity was assessed by Was he exercised daily, which type of sports, and how many hours per day? Less than 30 minutes of exercise considered physical inactivity, TV watching is assessed by Was he interested in computer and video games and how many hours he played? the number of hours spent watching TV (watching TV for ≤2 hours was considered usual and watching TV for >2 hours was considered a risk factor for overweight and obesity (Weaver, 2003).

Examination: For each child, height, weight and BMI were taken. Height was measured with child in socks and stand erectly with occipital area, buttock and the heel touching the wall and the height was measured by microtoise height measures. Weight was measured by UNISCLAE weight measures. BMI was calculated by using the equation:

$$BMI = \frac{Weight\ \ in\, kg}{\left(Height\ \ in\ \ meters\right)^2}$$

BMI for age percentiles for both males and females were considered by using WHO standards (the source of the charts used in this study CDC growth charts: national center for chronic disease prevention and health promotion, 2000). Obese children were identified when BMI is more than 95th percentile. While those with BMI between 85-95th percentiles considered to be overweight.

Limitations of the study:

- Convenient sampling was an obligatory alternative for random sampling, in the same time it was a constraint for the study too.
- Inquiring about items that practitioners educate for were not deeply explored during interviews, so this study did not expose the real defects concerning this issue.
- We could not take the accurate prevalence of obesity in our country because of lack of time and thus number of data collected for this purpose.
- We could not know the significance of specific risk factors such as Psychological, Enviromental and Genetics because it needs many associated specialties in these fields.

Statistical analysis: Statistical Package for Social Sciences version 18 (SPSS 18) was used for data input and analysis. Discrete variables presented as numbers and percentages. Continuous variables presented as mean and standard deviation (SD). Chi square test for independence used to test the significance of association between two discrete variables. t test used to test the significance of difference in mean between two independent samples. Chi square test for goodness of fit used to test the significance of observed distribution. Findings with P value less than 0.05 were considered significant. Odds ratio (OR) is used to estimate the risk of exposure. OR is produced using the binary logistic model as follow:

Sex was adjusted for age groups. Age groups were adjusted for sex. Other variables were adjusted for age groups and sex. Comparison/background levels for variables were as follow: 2-5 years for age groups, female for sex, normal birth weight for birth weight, non-premature for prematurity, negative for family history of childhood obesity, up to 3 for crowding index, parents living together for marital status, non-obese for parental body habit, non-smoker for maternal smoking, higher than secondary for both maternal and paternal education, less than ID 500,000 for family income, exclusive breast milk for feeding in infancy, negative for overeating, up to 250cc a day for sweet drinks, negative for frequent eating of sweets, up to two hours for TV watch, and active for low physical activity.

Notes: P value is the probability of error/chance (sampling error, error because of sampling). The finding is not considered significant if the probability of error more than 5% (P>0.05), so you cannot say the finding is wrong but say not significant because still the probability of trueness is more than 90% in some instances.

OR (Odds Ratio): OR indirectly estimates the relative risk (RR), since only cohort studies give RR (direct estimation), and case control studies give OR. If the OR > 1 then the exposure will increase the risk to develop the outcome (risk factor). If OR = 1 (nil value or no effect value), the there is no effect of exposure on developing the outcome in concern. If OR < 1 then the exposure will decrease the probability of developing the outcome (protective factor). Usually we do not use P value in determining the significance of OR but we look to the95% confidence interval, if the nil value is included in the range then the OR is not significant. The study sample composed of 100 obese child with mean age 9.5±3.6 years (minimum 2 years and maximum 15 years) and 100 control (non-obese) child with mean age of 8.7± 3.7 years (minimum 3 years and maximum 15 years), and there was no significant difference in mean age between the two study groups (P > 0.05, table 1A) There was no significant association between being obese and each of age group, gender, birth weight, history of prematurity (P > 0.05, table 1A). It is significant to find obese children more to have a family history of childhood obesity and having obese parent(s) (P < 0.05, table 1A).It is estimated that the risk to have obesity doubles on age 13-15 year compared to ages 2-5 years (Table 1B) Being a male brings double risk to have obesity. (Table 1B). Obese children are 1.8 more likely to have positive family history of childhood obesity (table 1B), in the same time those obese children are 1.7 times more to be associated with parental obesity (Table 1B). The increase in educational level in any of parents is significantly associated with child obesity, in the same time obese children are found to be of families

Table 1(A). Demographic variables and family history for study participants

	Obese		Control		Total		Test	
	N	%	N	%	N	%	Statistic	P
Age(year); M±SD	9.5±3.6		8.7±3.7		9.1±3.7		1.618[A]	0.107
Age Group (year)								
2-5	22	22.0	34	34.0	56	28.0		
6-12	44	44.0	38	38.0	82	41.0	3.591[B]	0.166
13-15	34	34.0	28	28.0	62	31.0		
Sex								
• Male	58	58.0	50	50.0	108	54.0	1.288[B]	0.256
• Female	42	42.0	50	50.0	92	46.0		
Birth Weight								
• > 3.5	20	20.0	26	26.0	46	23.0		
• 2.5-3.5	56	56.0	52	52.0	108	54.0	1.018[B]	0.601
• <2.5	24	24.0	22	22.0	46	23.0		
Prematurity	24	24.0	22	22.0	46	23.0	0.113[B]	0.737
Family History of Childhood Obesity	58	58.0	44	44.0	102	51.0	3.922[B]	0.048
Obese Parent (any)	60	60.0	42	42.0	102	51.0	3.885[B]	0.049

N; number, %; percent, P; P value
[A] t test
[B] Chi square test

Variables	OR [95%CI]
Age Group (year)	
• 2-5	---[A]
• 6-12	1.8[0.9;3.5]
• 13-15	2.0[1.0;4.3]
Sex	
• Male	2.0[1.4;4.6]
• Female	---[A]
Birth Weight	
• > 3.5	0.7[0.3;1.4]
• <2.5	0.9[0.5;1.9]
• 2.5-3.5	---[A]
History of Prematurity	1.0[0.5;2.1]
Family History of Childhood Obesity	1.8[1.0;3.2]
Obese Parent (any)	1.7[1.0;3.1]

[A] Background/comparison level

Table 2(A). Odds ratio for social and for environmental factors

	Obese		Control		Total			
	N	%	N	%	N	%	X^2	P
Marital Status								
• Living Together	74	74.0	68	68.0	142	71.0		
• Divorced	8	8.0	16	16.0	24	12.0	3.038	0.219
• Widow	18	18.0	16	16.0	34	17.0		
• Separated	0	0.0	0	0.0	0	0.0		
Maternal Smoking	4	4.0	4	4.0	8	4.0	0.000	1.000
Maternal Education								
• Not Educated	8	8.0	24	24.0	32	16.0		
• Primary	22	22.0	26	26.0	48	24.0	14.216	0.003
• Secondary	26	26.0	26	26.0	52	26.0		
• Higher than Secondary	44	44.0	24	24.0	68	34.0		
Paternal Education								
• Not Educated	6	6.0	16	16.0	22	11.0		
• Primary	8	8.0	28	28.0	36	18.0	23.134	0.000
• Secondary	32	32.0	26	26.0	58	29.0		
• Higher than Secondary	54	54.0	30	30.0	84	42.0		
Crowding Index >3	42	42.0	44	44.0	86	43.0	0.082	0.775
Family Income (ID 1000)								
• < 500	10	10.0	18	18.0	28	14.0		
• 500-2500	28	28.0	42	42.0	70	35.0	9.831	0.007
• > 2500	62	62.0	40	40.0	102	51.0		

N; number, %; percent, X^2; chi square test statistic, P; P value

Table 2 (B). Odds ratio for social and for environmental factors

Variables	OR [95%CI]
	N
Marital Status	
• Living Together	---[A]
• Divorced	1.0[0.5;2.2]
• Widow	0.5[0.2;1.4]
• Separated	---
Maternal Smoking	0.8[0.2;3.7]
Maternal Education	
• Not Educated	0.2[0.1;0.4]
• Primary	0.5[0.2;1.0]
• Secondary	0.5[0.3;1.2]
• Higher than Secondary	---[A]
Paternal Education	
• Not Educated	0.2[0.1;0.6]
• Primary	0.2[0.1;0.4]
• Secondary	0.7[0.3;1.4]
• Higher than Secondary	---[A]
Crowding Index >3	0.9[0.5;1.6]
Family Income (ID 1000)	
• < 500	---[A]
• 500-1500	1.2[0.5;3.1]
• > 1500	2.9[1.2;7.0]

[A] Background/comparison level

Table 3 (A). Eating history and physical activity of study sample

	Obese		Control		Total			
	N	%	N	%	N	%	X^2	P
Feeding in Infancy								
• Breast Milk	24	24.0	38	38.0	62	31.0		
• Mixed	30	30.0	38	38.0	68	34.0	11.017	0.004
• Bottle Milk	46	46.0	24	24.0	70	35.0		
Over Eating	88	88.0	50	50.0	138	69.0	33.754	0.000
Sweet Drinks (any) (> 250 cc a day)	90	90.0	50	50.0	140	70.0	38.095	0.000
Frequent Eating of Sweets	86	86.0	50	50.0	136	68.0	29.779	0.000
TV Watch> 2 Hours	82	82.0	56	56.0	138	69.0	15.802	0.000
Low Physical Activity (< 2 hours a day)	96	96.0	24	24.0	120	60.0	108.000	0.000

N; number, %; percent, X^2; chi square test statistic, P; P value

Table 3 (B). Odds ratio for eating history and for physical activity of study sample

Variables	OR [95%CI]
	N
Feeding in Infancy	
• Breast Milk	---[A]
• Mixed	1.2[0.6;2.4]
• Bottle Milk	2.9[1.4;6.1]
Over Eating	7.3[3.5;15.0]
Sweet Drinks (any) (> 250 cc a day)	9.6[4.4;21.1]
Frequent Eating of Sweets	6.4[3.2;13.0]
TV Watch> 2 Hours	3.4[1.7;6.6]
Low Physical Activity (< 2 hours a day)	76.0[25.3;228.4]

[A] Background/comparison level

Table 4. Effects of body habit on bone age and cholesterol level of the study sample

	Obese		Control		Total			
	N	%	N	%	N	%	X^2	P
Bone Age								
• Normal	16	16.0	90	90.0	106	53.0		
• Advanced	84	84.0	10	10.0	94	47.0	109.916	0.000
• Delayed	0	0.0	0	0.0	0	0.0		
Hypercholesterolemia	84	84.0	10	10.0	94	47.0	109.916	0.000

N; number, %; percent, X^2; chi square test statistic, P; P value

Table 5. Proportions of abnormal lipoprotein levels among obese children

Serum Lipoproteins	N	%	X^2	P
Decreased HDL	42	84.0	23.120	0.000
Increased LDL	42	84.0	23.120	0.000
Hypercholesterolemia	84	84.0	46.240	0.000
Increased TG	28	56.0	0.720	0.396

N; number, %; percent, X^2; chi square test statistic, P; P value, HDL; high density lipoprotein level, LDL; low density lipoprotein level, TG; triglycerides.

with higher income (P<0.05, Table 2 A). There was no significant association between marital status, maternal smoking, crowding index with childhood obesity (P > 0.05, table 2 A).Compared to parents of education higher than secondary, it is found that children having parents of low education level (uneducated or primary education) are protected against obesity (OR < 1, table 2 B). The increase in income is significantly associated with obesity in children (P < 0.05, Table 2 A) that obesity is 2.9 times more to be associated with incomes more than ID 1500,000 (Table 2 B). Regarding feeding history of early childhood; it is found that more dependence on bottle milk is associated with obesity (P < 0.05, Table 3 A) that those exclusively bottle fed are 2.9 times more likely to have obesity (Table 3 B). The association between each of overeating ingesting sweet drinks, frequent sweets eating, prolonged TV watch (over two hours) and low physical activity are significantly associated with obesity (P < 0.05, Table 3 A) that the risk to have obesity is 2.9 in bottle fed, 7.3in overeating child (Table 3 B), 9.6 in sweet drinks, 6.4 in frequent sweets eating, 3.4 in TV watch more than two hours, 76.0 in low physical activity (Table 3 B). Concerning the effects of obesity: It is found that there is a significant association between each of advanced bone age and hypercholesterolemia with obesity in children (P < 0.05, Table 4). All of decreased HDL, increased LDL, and hypercholesterolemia are significantly found in obese children (P < 0.05, Table 5).

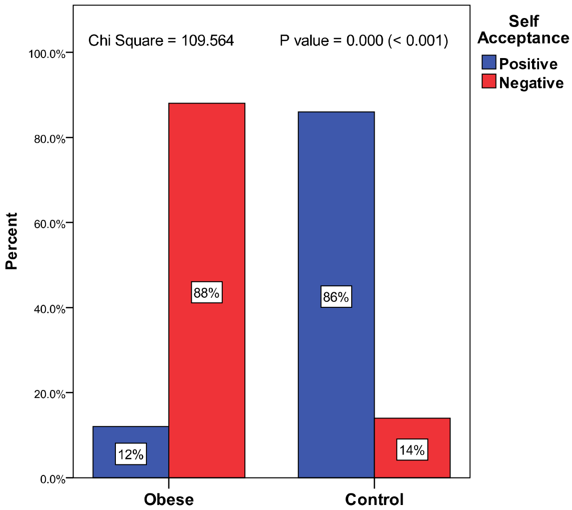

Figure 1. Distribution of study sample according to study group and to self acceptance of body habit

DISCUSSION

Studies that have used BMI to identify overweight and obese children based on percentage of body fat have revealed high specificity (95–100%), but low sensitivity (36–66%) for this system of classification (Dehghan, 2005). In this study BMI percentile was defined for age and sex using 85th and 95th percentiles as a cut off point for children > 24 months old age to define overweight and obesity respectively The rate of childhood overweight is increasing with age. Currently, over 10% of children aged 2-5, 15% of children aged 6-11, and 16% of adolescents aged 12-19 years are classified as overweight (Gyovai, 2003) Although overweight tended to be more common in girls (Martorell, 2000), in this study overweight and obesity although were more among males than females but this did not reach a statistical significance, probably because of the small sample size. In younger children, parental obesity is a more potent risk factor than the child's own weight status in predicting whether the child will become an obese adult, (Aa) If 1 or both parents are overweight. Younger children may be as much as 2 times more likely than their leaner peers to be obese. which is compatible to our study in which positive family history have 1.8 risk for a child to be obese Although there is positive relationship between birth weight and subsequent weight gain in most studies (Power, 2002 and Parsons, 2001), a recent study, based on the 1958 birth cohort, showed that there was a weak positive association between birth weight and adult BMI which was abolished after adjustment for maternal weight. However, studies relying on birth weight as a proxy for fetal growth may obscure a distinct effect of poor intrauterine environment on obesity. This is because there are strong associations between parental size, offspring birth weight and subsequent size through the adulthood (Power, 2002). In this study no significant positive correlation of birth weight with overweight/obesity was found, calculated by BMI percentile.Which is consistant with Kumanyika *et al* (Aa) Martorell *et al* found that obesity levels increased over time but at varying rates, and concluded that rising incomes in developing countries and increased 'westernization' will most likely lead to increased levels of obesity (Martorell, 2000). Many changes are taking place in developing countries, which are of concern. These include the adoption of 'Western' diets, which are high in saturated fats, sugar and refined foods, and changes in lifestyle which include reduced levels of physical activity, and increased stress, particularly in urban areas. This phenomenon has been labeled the 'Nutrition Transition' by Popkin (Popkin, 1994) and is the probable cause of the emerging problem of obesity among adults in developing countries. Undoubtedly, children are exposed to these influences as well, but perhaps the impact of this exposure increases with age. T

he acquisition of adiposity is also cumulative and overweight would be expected to rise with age Increase in parental education was associated with a significant improvement in frequency of different diet administration with more effect of that of mothers over that of fathers. This last effect is expected as mothers are directly involved in food preparation more than fathers and their knowledge would exert such a positive effect (Saleem, 2006). On the other hand, this would also explain partly why high educated parents have more overweight or obese children. In this study, a positive correlation found of overweight and obesity with father and mother education (P-value <0.05). This is in agreement with the results of another study done by WHO in 1986 including 71 national nutrition surveys representing 50 developing countries (Martorell, 2000). Maternal smoking during pregnancy is one potentially modifiable factor that appears to increase the risk for obesity Although there is significant dose response relationship between maternal smoking and obesity prevalence in later childhood (Parsons, 2001), 8 mother only has been recorded to use tobacco during or before pregnancy in the present study. There are several potential mechanisms through which children's eating patterns and level of physical activity may be affected by having parents who work outside the home. Children may eat differently if child care providers are more likely to give them food that is highly caloric and of poor nutritional value. Further, parents who work outside the home may serve more high-calorie prepared or fast foods, and unsupervised children may make poor nutritional choices when preparing their own after-kindergarten snacks. Similarly, unsupervised children may spend a great deal of time indoors, perhaps due to their parents' safety concerns, watching television or playing video games rather than engaging in more active indoor or outdoor activities (Anderson, 2003). Many studies have found a strong correlation between parent employment and child overweight problems (Anderson, 2003 and Dietz, 1991). The present study has confirmed a significant negative correlation between the breastfeeding and overweight and obesity. While a significant correlation was observed between overweight/ obesity and the formula feeding. A dose-response, protective relationship of breastfeeding with the risk of overweight was suggested by other study depending on analysis of data from the Centers For Disease Control and Prevention (CDC) Pediatric Nutrition Surveillance System in the United States where a total of 246371 children were examined from 1988 to 1997 (Mei, 2004). The link between long-term breastfeeding and lower rates of overweight could operate through several possible biological mechanisms. Birch *et al* (Birch, 1998), showed that children who were breastfed are better able to adjust intake at a meal in response to a high calorie preload; thus, breastfed children may learn to self-regulate caloric intake better than non breastfed infants do. Also, breastfed and formula-fed infants have a different hormonal response to feeding: formula feeding provokes a greater insulin response, possibly resulting in earlier fat deposition (Lucas, 1980). Another possibility is that the higher protein intake in formula-fed infants has a programming effect on glucose metabolism. Finally, breastfed infants adapt more readily to new foods such as vegetables, thus influencing the subsequent caloric density of their diet (Mei, 2004). At the age of 2-6 years old, the recommendations for diet contents are similar to those for older individuals. Challenges here relate to providing quality nutrient intake and avoiding excess caloric intake. Dairy products are the major source of saturated fat and cholesterol in this age group, and therefore a transition to low-fat milk and other dairy products is important. Sweetened beverages and other sugar-containing snacks are a major source of caloric intake (Freemark, 2001). Higher longitudinal intakes of calcium, monounsaturated fat, and servings of dairy products were associated with lower body fat (Carruth, 2001).

Another study about dietary composition and weight changes among low income preschool children (1379 children aged 2-5 years in North Dakota with special supplemental nutritional program for women, infant, and children; (WIC program) did not reveal a significant relation between total intake of fat, fiber, fruits, vegetables and weight changes (Newby, 2003). This study is not like our study where there was a significant correlation for body weight with frequency and amount of consumption of dairy products, fruits/vegetables, grains and excess juice. Also a significant correlation was found with number of meals or snackes consumed per day. Asking about the frequency and amount of food consumed in the last 24 hours to determine diet quality among preschool children to determine the amount of consumption of sweety diet, fruits/vegetables, dairy products, grains and juice is prone to limitations. One of the limitations of this method was that dietary intakes for children were collected by proxy in all survey waves. Children's diets were reported by an adult who may have lacked complete knowledge of everything the child ate. Another potential limitation to this method is the possibility of a bias in unknown direction owing to the change in dietary intake assessment methods between surveys. In this study, although we have asked about participation of the child in an indoor activity was, but physical activity was difficult to be assessed. Physical activity was assessed by the number of hours of TV watching, and there was a highly significant correlation between watching TV for >2 hours and overweight /obesity (P-value < 0.005). This result is similar to the results of other study reported by Tremblay *et al*. (Tremblay, 2003),in Canada. obesity is significantly more common than expected in young children and strongly associated with social deprivation. obesity have adverse and long term health effects. In conclusion, the coexistence of overweight/obesity with psychosocial deprivation has recently emerged from epidemiological studies in developing countries (Armstrong, 2003). But in our study we could not asses that precisely.

Conclusions

- Overweight/obesity were more common among males, no significant correlation was found with sex.
- There was highly significant protective effect of breastfeeding against development of obesity .
- Limitation of physical activity and watching TV for > 2 hours duration is a significant risk factor for overweight/obesity.
- There was no significant positive correlation between birth weight and/or history of prematurity and overweight/obesity.
- There was a significant correlation observed among paternal education, formula feeding, number of meals or snacks/day, and frequency and amount of consumption of saturated fat, dairy products, fruits/vegetables and grains with overweight/obesity.
- There was a significant correlation between high income and overweight/obesity.
- **A**lthough psychological effect was obvious on children in the study, this could not be assessed statistically.

Recommendations

- Encouraging breastfeeding, exclusive breastfeeding for the initial 6 months and then to continue on breastfeeding as long as possible together with complementary foods.

Improving nutritional quality after weaning:

- Delay the introduction of 100% juice until at least 6 months of age and limit to no more than 4–6 oz/day; juice should only be fed from a cup.
- Respond to satiety clues and do not overfeed; infants and young children can usually self-regulate total caloric intake; do not force children to finish meals if not hungry, because they often change caloric intake from meal to meal.
- Introduce healthy foods and continue offering it, if initially refused. Parents should not introduce foods without overall nutritional value simply to provide calories.

For children aged > 2 year, parents and caregivers should follow special dietary recommendation which include:

- Eating vegetables and fruits daily, limit juice intake.

- Eating whole-grain breads and cereals rather than refined-grain products.
- Reducing the intake of sugar-sweetened beverages and foods.
- Using nonfat (skim) or low-fat milk and dairy products daily, use vegetable oils which is low in saturated fat instead of butter or most other animal fats in the diet.
- Reducing salt intake, including salt from processed foods.
- Balancing dietary calories with physical activity to maintain normal growth, 60 minute of moderate to vigorous play or physical activity daily.
- For treatment of obesity, intervention should begin early with gradual changes, and the clinician should involve the family and all caregivers in the treatment program.
- Clinician should educate families about medical complications of obesity and long term risk of obesity.
- Design special nutritional programmes for women, infant, and children.
- A large reduction in caloric intake of kcal per day, along with increased physical activity, can produce a loss of body weight over the relatively short period of about 6 months . behaviors for weight loss are focused on caloric reduction: decreasing overall food intake, reducing portion sizes, substituting lower-calorie for higher calorie foods, and increasing physical activity.

REFERENCES

Amin RS, Kimball TR, Bean JA, Jeffries JL, Willging JP, Cotton RT, Witt SA, Glascock BJ, Daniels SR. 2002. Left ventricular hypertrophy and abnormal ventricular geometry in children and adolescents with obstructive sleep apnea. *Am J Respir Crit Care Med*., 165: 1395–1399.

Anderson PM, Butcher KF, Levine PB. 2003. Maternal Employment and Overweight Children. Dartmouth College Department of Economics*, J Health Econ.*, 22 (3):477-504.

Armstrong J, Dorosty AR, Reilly JJ. 2003. Coexistence of social inequalities in undernutrition and obesity in preschool children: population based cross sectional study. *Archives of Disease in Childhood.,* 88:671-675.

Baskin ML, Ard J, Franklin F, Allison DB. 2005. Prevalence of obesity in the United States. *Obes Rev.,* 6:5–7.

Bass J.Takahashi Js. Circadian integration of metabolism and energitics. USA science 2010; 330:1349-54.

Batterham RL, Cohen MA, Ellis SM, Le Roux CW, Withers DJ, Frost GS, Ghatei MA, Bloom SR. Inhibition of food intake in obese subjects (2003).

Bender R, Trautner C, Spraul M, Berger M. 1998. Assessment of excess mortality in obesity. *Am J Epidemiol.,* 147: 42-48.

Berenson GS, Srinivasan SR, Bao W, Newman WP III, Tracy RE, Wattigney WA. 1998. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. *N Engl J Med.,* 338:1650 –1656.

Berenson GS, Srinivasan SR, Bao W, Newman WP III, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk

Berkowitz RI, Wadden TA, Tershakovec AM, Cronquist JL. 2003. Behavior therapy and sibutramine for the treatment of adolescent obesity: a randomized controlled trial. JAMA., 289:1805–1812.

Biddle, S.J., Gorely. T., Marshall, S. J., Murday, I.,Cameron, N. 2004. Physical activity and sedentary behaviour in youth: issues and controversies, *J. R. Soc. Health. Jan.,* 124 (1): 29 -33.

Birch LL, Fisher JO. 1998. Development of eating behaviors among children and adolescents. *Pediatrics.,* 101 :539 – 548.

Bodenheimer T, Wagner EH, Grumbach K. 2002. Improving primary care for patients with chronic illness. JAMA., 288: 1775–1779 57.

Carnethon MR, Gidding SS, Nehgme R, Sidney S, Jacobs DR Jr, Liu K. Cardiorespiratory fitness in young adulthood and the development of cardiovascular disease risk factors. JAMA. 2003; 290:3092–3100.

Carruth BR, Skinner JD. 2001. The role of dietary calcium and other nutrients in moderating body fat in preschool children. *Int J Obes*., 25(4):559-566.

Carruth BR, Skinner JD. 2001. The role of dietary calcium and other nutrients in moderating body fat in preschool children. *Int J Obes* (Lond)., 25(4):559 - 566.

Cole TJ, Mary CB, Flegal KM, Dietz WH. 2000. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ,* 320:1240.

Cook S, Weitzman M, Auinger P, Nguyen M, Dietz, WH. 2003. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition ExaminationSurvey, 1988–1994. *Arch. Pediatr. Adolesc. Med.,* 157:821– 827.

Cummings DE, Clement K, Purnell JQ, Vaisse C, Foster KE, Frayo RS, Schwartz MW, Basdevant A, Weigle DS. 2002. Elevated plasma ghrelin levels in Prader Willi syndrome. *Nat Med.* 8:643– 644.

Cummings DE, Weigle DS, Frayo RS, Breen PA, Ma MK, Dellinger EP, Purnell JQ. 2002. Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. *N Engl J Med.,* 346:1623–1630.

Daneils *et al*, 2005. Overweight in children and adolescents. Pathophysiology, consequences, prevention and treatment; AHA scientific statement, 111:1999-2012.

Daniels SR, Loggie JMH, Khoury P, Kimball TR. 1998. Left ventricular geometry and severe left ventricular hypertrophy in children and adolescents with essential hypertension. *Circulation.,* 97:1907–1911.

Davis MM, Gance-Cleveland B, Hassink S, Johnson R, Paradis G, Resnicow G. 2007. Recommendations for prevention of childhood obesity. *Pediatrics.,* 120(suppl 4):228–252

DeFronzo RA, Ferrannini E. 1991. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. Diabetes Care., 14:173–194.

Dehghan M, Danesh NK, Merchant AT. 2005. Childhood obesity, prevalence and prevention. *Nutr J*., 4:74-82.

Di Sario A, Candelaresi C, Omenetti A, Benedetti A. 2007. Vitamin E in chronic liver diseases and liver fibrosis. *Vitam Horm.,* 76:55173.

Dietz, William. 1991. Factors Associated with Childhood Obesity. Nutrition., 7(4): 290-291

Donohoue, P.A. 2004. Obesity. In Behrman RE, Kliegman RM, Jenson HB,(eds.). Nelson Textbook of Pediatrics, 17 edition, Elsevier Saunders, Philadelphia, 173-177.

Eckel, RH. 2003. Obesity: a disease or a physiologic adaptation for survival?In: Eckel RH, ed. Obesity Mechanisms and Clinical Management. Lippincott, Williams & Wilkins, Philadelphia, Pa; 3–30.

Eisenberg ME, Neumark-Sztainer D, Story M. 2003. Associations of weight-based teasing and emotional well-being among adolescents. *Arch Pediatr Adolesc Med.,* 157:733–738.

Eneli I, Dele Davis H. 2008. Epidemiology of childhood obesity. In: Dele Davis H, ed. Obesity in Childhood & Adolescence. Vol 1. Westport, Conn: Praeger Perspectives; 3-19.

Farooqi IS, Jebb SA, Langmack G, Lawrence E, Cheetham CH, Prentice AM, Hughes IA, McCamish MA, O'Rahilly S. 1999. Effects of recombinant leptin therapy in a child with congenital leptin deficiency. *N Engl J Med.* 341:879–884.

Fiore H, Travis S, Whalen A, Auinger P, Ryan S. 2006. Potentially protective factors associated with healthful body mass index in adolescents with obese and nonobese parents: a secondary data analysis of the third national health and nutrition examination survey, 1988-1994. *J Am Diet Assoc.,* 106(1):55-64.

Freemark M, Bursey D. 2001. The effects of metformin on body mass index and glucose tolerance in obese adolescents with fasting hyperinsulinemia and a family history of type 2 diabetes. *Pediatrics.,* 107:E55.

Friedman MA, Brownell KD. 1995. Psychological correlates of obesity:moving to the next research generation. *Psychol Bull.*, 117:3–20.

Ghosh S. 2005. Does economic inequality matter in cases of infectious disease? An Analysis for India. *Asia –Pacific Population J,* 20(1):40-44.

Gidding SS, Dennison BA, Birch LL, Daniel SR, Gilman MW, *et al*. 2006. Dietary Recommendations For Children and Adolescents: A Guide for Practitioners. Pediatrics., 117(2):544-559.

Goodman, E., Whitaker, R.C. 2002. A prospective study of the role of depression in the development and persistence of adolescent obesity. *Pediatrics.,* 110:497–504.

Goran, M.I., Toth, M.J., Poehlman, E.T. 1998. Assessment of research-based body composition techniques in healthy elderly men and women using the 4-compartment model as a criterion method. *Int J Obes Relat Metab Disord*., 22:135–42.

Gyovai V, Gonzales J, Ferran k, Wolff C. 2003. Family dietary and activity Behaviors associated with overweight risk among low-income preschool age children. *Californian Journal of Health Promotion.,* 1(2):66-77.

Hediger ML, Overpeck MD, Kuczmarski RJ, Ruan WJ. 2004. Association between infant breastfeeding and overweight in young children. JAMA., 285:2453–2460.

Inge TH, Garcia V, Daniels S, Langford L, Kirk S, Roehrig H, Amin R, Zeller M, Higa K. 2004. A multidisciplinary approach to the adolescent bariatric surgical patient. *J Pediatr Surg.,* 39:442– 447.

Inge TH, Krebs NF, Garcia VF, Skelton JA, Guice KS, Strauss RS, Albanese CT, Brandt ML, Hammer LD, Harmon CM, Kane TD, Klish

Ivkovic-Lazar T. 2003. Development and differentiation of adipose tissue [in Croatian]. *Med Pregl.,* 56:142–145.

Keys A, Fidanza F, Karvonen MJ, Kimura N, Taylor HL. 1972. Indices of relative weight and overweight. *J Chronic Dis.,* 25:329–43.

Kimm SY and Obarzanek E. (2015). Childhood obesity: a new pandemic of the new millennium. Pediatrics 110: 1003–1007.

Kranz, S., Siega-Riz, A.M., Herring, A.H. 2004. Changes in Diet Quality of American Preschoolers Between 1977 and 1998. *Am J Public Health.,* 94(9):1525-1530.

Kuczmarski, R.J., Ogden, C.L., Grummer-Strawn, L.M., Flegal, K.M., Guo, S.S.,Wei, R., Mei, Z., Curtin, L.R., Roche, A.F., Johnson CL. CDC growth charts:United States. *Adv Data.,* 314:1–27.

Kumanyika, S.K, Obarzanek, E. 2003. Pathways to obesity prevention: report of a National Institutes of Health workshop. *Obes Res.,* 11: 1263–1274.

Li L, Parsons TJ, Power C. 2003. Breast feeding and obesity in childhood: cross- sectional study. BMJ., 327:327–328.

Lucas A, Sarson DL, Blackburn AM, Adrian TE, 1980. Aynsley-Green A, *et al.* Breast vs. bottle: endocrine responses are different with formula feeding. Lancet 1980; 1 :1267 –1269.

Lustig RH, Hinds PS, Ringwald-Smith K, Christensen RK, Kaste SC, Schreiber RE, Rai SN, Lensing SY, Wu S, Xiong X. 2003. Octreotide therapy of pediatric hypothalamic obesity: a double-blind, placebo-controlled trial. *J Clin Endocrinol Metab.,* 88:2586 –2592.

Martorell, R., Khan, L.K., Hughes, M.L., Strawn, L.M. 2000. Overweight and obesity in preschool children from developing countries. 2000 August;24(8):959-967 .

Masuzaki, H, Paterson J, Shinyama H, Morton NM, Mullins JJ, Seckl JR, Flier JS. 2001. A transgenic model of visceral obesity and the metabolic syndrome. *Science.,* 294:2166 –2170.

McDuffie JR, Calis KA, Uwaifo GI, Sebring NG, Fallon EM, Hubbard VS, Yanovski JA. Three-month tolerability of orlistat in adolescents with obesity-related comorbid conditions. *Obes Res.*, 10:642– 650.

McMinn JE, Baskin DG, Schwartz MW. Neuroendocrine mechanisms regulating food intake and body weight. *Obes. Rev.*, 1:37– 46.

Mei Z, Grummer-Srawn LM. 2004. Does Breastfeeding protect against pediatric overweight ? Analysis of longitudinal data from the Centers for Disease Control and Prevention Pediatric Nutrition Surveillance System. Pediatrics, 13 (2):81-86.

Montgomery SM, Ekbom A. 2002. Smoking during pregnancy and diabetes mellitus in a British longitudinal birth cohort. BMJ., 324:26-27.

Newby PK, Peterson KE, Berkey CS, Leppert J, Willet WC, *et al*. 2003. Dietary composition and weight change among low-income preschool children. *Arch Pediatr. Adolesc Med*, 157(8):759-764.

Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States,1999–2004. JAMA. 2006;295:1549 –1555.

Ouchi N, Kihara S, Funahashi T, Matsuzawa Y, Walsh K. Obesity, adiponectin and vascular inflammatory disease. *Curr. Opin. Lipidol.* 2003;14:561–566.

Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients). Washington, DC: National Academies Press; 2002.

Pankow JS, Jacobs DR Jr, Steinberger J, Moran A, Sinaiko AR. Insulin resistance and cardiovascular disease risk factors in children of parents with the insulin resistance (metabolic) syndrome. Diabetes Care. 2004;27:775–780.

Parsons TJ, Power C, Manor O. Fetal and early life growth and Body Mass Index from birth to early adulthood in 1958 British cohort: longitudinal study. BMJ 2001;323:1331–1335.

Pinhas-Hamiel O, Dolan LM, Daniels SR, Standiford D, Khoury PR, Zeitler P. 1996. Increased incidence of non-insulin-dependent diabetes mellitus among adolescents. *J Pediatr.,* 128:608–615.

Popkin BM. 1994. The nutrition transition in low income countries: an emerging crisis. *Nutr Rev,* 52: 285.

Power C., Jefferis B. 2002. Fetal environment and subsequent obesity: a study of maternal smoking. *International Journal of Epidemiology.,* 31:413-419 .

Rakowski W. 1999. The potential variances of tailoring in health behavior interventions. *Ann Behav Med.,* 21:284 – 289.

Saleem MB. 2006. Complementary foods for children under two years of age and its relation to nutritional status and selected socio-demographic factors in Basrah. *The Medical Journal of Basrah University*., 24(1,2):33-44.

Salsberry PJ, Reagan PB. 2005. Dynamics of Early Childhood Overweight. *Pediatrics.,* 116(6):1329-1338.

Sarah E. Barlow, 2007. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity summary. *Pediatrics.,* 120:5164.

Solomon CG, Manson JE. 1987. Obesity and mortality: a review of the epidemiologic data. *Am J Clin Nutr*., 66: 1044-1050.

Speliotes et-al. 2010. Metabolism initiative program in medical and population Genetics, Board institute, Cambridge, Mass.USA. Nat Genetics., 42:37-48.

Strauss RS, Pollack HA. Social marginalization of overweight children. 2003. *Arch Pediatr Adolesc Med*. 157:746 –752.

Strauss RS, Pollack HA. Social marginalization of overweight children. 2003. *Arch Pediatr Adolesc Med*. 157:746 –752.

Tirosh A, Shai I, Afek A, Dubnov-Raz G, *et al*. Adolescent BMI trajectory and risk of diabetes versus coronary disease. *N Engl J Med.* Apr 7, 364(14):1315-25.

Tremblay MS, Willms JD. Is the Canadian childhood obesity epidemic related to physical inactivity? *International Journal Obesity Related Metabolic Disorder* 2003; 27:1100–1105.

Troiano RP, Briefel RR, Carroll MD, Bialostosky K. Energy ad fat intakes of children and adolescents in the United States: data from the national health and nutrition examination surveys. *Am J Clin Nutr.,* 1343S–1353S.

US Department of Agriculture. The Food Guide Pyramid for Young Children. Accessed at March 10, 2005.

Weaver L, Edward C, Golden B, Reilly J. Obesity. In McIntosh N, Helms P, Smyth R (eds.): Forfar & Arneil's Textbook of Pediatrics. 6th edition. Churchill Livingstone, Philadelphia 2003:574-576.

Woods SC. 2004. Gastrointestinal satiety signals I. An overview of gastrointestinal signals that influence food intake. *Am J Physiol. Gastrointest*. *Liver Physiol.,* 286:G7–G13.

World Health Organization. 2009. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. *World Health Organ Tech Rep Ser* 854:1–452.

Yanovski JA. Intensive therapies for pediatric obesity. *Pediatr Clin North Am.* 2001;48:1041–1053.

Yost TJ, Rodgers CM, Eckel RH. Suction lipectomy: outcome relates to region-specific lipoprotein lipase activity and interval weight change. *Plast Reconstr. Surg.* 1993;

****