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The Presence of Childhood Obesity in Nebraska and the Physiological Repercussions of the Disease

Madison R. Bezousek
University of Nebraska-Lincoln

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By Madison Bezousek, BS

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Faculty Mentors:

Kim Hansen, Ph.D., Biological Sciences

Karin van Dijk, Ph.D., Biochemistry

Abstract

Childhood Obesity can cause lifelong repercussions in children and adults. There has been stigma around the causes of obesity and its relation to lifestyle choices, without consideration of the genetic and syndromic causes. In this literature review the causes of obesity were investigated, along with the effect on the physiological systems and the enviornmental factors that are continuing the obesity epidemic. Preventing and treating lifestyle obesity is something that is seen to have the greatest effect on youth, especially with intergenerational obesity. Nebraska schools have implemented programs to encourage healthy living, and ongoing research is being implemented to reduce the obesity population. With genetic and syndromic obesity, there is treatment that has been found to be effective when implemented with a healthy lifestyle before the other body systems are effected. Overall, obesity will persist without prevention and treatment and recognizing that the environment children and adults are placed in is a crucial aspect of reducing the rates of childhood obesity.

Key words: Obesity, Childhood obesity, genetics, lifestyle, syndromes, physiology

Introduction

Obesity is a disease that plagues many Americans and can have lifelong health repercussions. It is essential to investigate the ways in which obesity has become increasingly pertinent in American culture and what can be done to prevent the health problems that are associated with the disease, especially with the development occurring at a young age. In this review, the causes of obesity such as genetic predispositions, environmental factors, and syndromic relations are investigated as well as the physiological setbacks that result from obesity. Research for effective treatment and prevention is an important part of this review to navigate health misconceptions and understand that not all forms of obesity are able to be prevented or have the same treatment. When treating obesity, prevention is needed and in order to prevent it we first must know what causes obesity.

Obesity is a term broadly placed on those with an accumulation of adipose tissue. Obesity is commonly found to occur from an "imbalance between calories ingested versus how much energy is expended, results in excess adipose tissue, and sedentary behaviors combined with high-calorie diets, a characteristic of modern societies are considered the major environmental factors driving the pathogenesis of obesity" (*The Genetics of Pediatric Obesity*, 2015). According to the National Heart, Lung, and Blood Institute, 3 in 4 adults and 1 in 5 children ages 2-19 in the United States have obesity (*What Are Overweight and Obesity?*, 2022). An important factor to test obesity is completed by doctors throughout our lives using a Body Mass Index (BMI). For children, BMI is compared to the height and weight percentiles determined by the age and sex of the child. If a child ranks in the 95th percentile or above for their cohort they are medically deemed obese. For adults, obesity is determined by calculating BMI using height and weight and having a score of 30.0 or above (*Defining Adult Overweight and Obesity*, 2022).

When diagnosing obesity, providers try to investigate if the disease is a result of a genetic predisposition, syndrome-related, or environmental factors. Having this knowledge can bring clarity on the best way to prevent and treat obesity to not impact the body's physiological fprocesses.

Obesity Studies

With the increase in research and genetic testing available, obesity has been tied to genetics and epigenetics in combination with the environment and lifestyle choices. The first incidence of obesity's relation to genetics was studied by Carl von Noorden who conducted human twin studies and family studies in 1907 to see if twins that were reared together resembled a more similar BMI to their biological parents or their adopted family. He wanted to prove that there were two forms of obesity: exogenous and endogenous. With exogenous obesity, the environment is still a critical factor to consider because the cause of obesity can be as simple as overeating without energy expenditure. With endogenous obesity, it was due to a genetic or physiological factor that needed to be discovered in the patient (*The Biology and Genetics of Obesity*, 2014). Through many decades of science, von Noorden's research was dismissed during rat feeding experiments in which every patient that ate excessively gained weight despite animal studies such as the Hetherington and Ranson study where lesions in the hypothalamus and thus incomplete signaling caused weight gain in rats. It was not until the late 1980's that research returned to human twin and family studies to discover a tie to genetics when evidence showed that children had closer BMI's to their biological parents than to the adopted parents that raised them and shaped their food environment (*An Adoption Study of Human Obesity*, 1986). This research then shaped the ongoing research into mapping obesity-related genes to understand the epigenetics of the disease. Using previously dismissed animal studies, Leptin and its pathways

involving the hypothalamus were discovered and revolutionized how obesity is viewed medically and not only reduced stigma about obesity being based on choice but also is crucial in learning how to regulate these hormones and pathways to treat disease (*Leptin and Beyond: An Odyssey to the Central Control of Body Weight*, 2011).

Genetic Predispositions & Obesity

With the ability to look closely at nucleotide changes through replication processes using genetic technology, it has been able to be determined that genetic obesity factors are related to monogenic causes, syndromic obesity, and polygenic obesity (Genetic and Epigenetic Causes of Obesity, 2018). Monogenic mutations can involve a homozygous or compound heterozygous inheritance of many different genes most commonly Leptin-related genes such as LEP and LEPR which are both autosomal recessive. The monogenic cause is from a single gene mutation based in the leptin-melanocortin pathway. Leptin is important in hunger regulation and ultimately plays a role in obesity and obesity prevention. Leptin is a hormone secreted by the fat cells (adipocytes) that sends a signal to the hypothalamus signaling satiety. Mutations can occur in which a gene associated with leptin release is not able to carry out its function and overeating occurs due to the inability to receive the signal that hunger is satisfied. Evidence of this was found with a frameshift mutation of c.398delG where normal weight is found at birth with excessive weight gain starting in early childhood. Besides LEP and LEPR mutations there are also mutations involving melanocortin and growth factor receptors. Propiomelanocortin (POMC) mutation is associated with adrenal gland insufficiency in releasing ACTH and controlling appetite regulation. Melanocortin receptor (MC4R) is a receptor that is found in abundance in the hypothalamus and lack of receptor activation is associated with the onset of early obesity and excessive hunger and food-seeking behavior in children. There have also been many deletion

mutations involving SIM1, NTRK2, and SH2B1 where deletion of appetite receptors or insufficient binding occurs where hunger is not able to be regulated properly (Profound Obesity Associated with a Balanced Translocation that Disrupts the SIM1 gene, 2013). It has also been found that micro-RNAs have a role in the proliferation and differentiation of adipocytes that can persist with obesity. According to the Nutrition Journal in the National Library of Medicine, "A significant association with increased levels of certain miRNA (miR-486-5p, miR-486-3p, miR-142-3p, miR-130 b, and miR-423-5p) was seen with BMI in children with obesity, with a significant change in the profile of 10 miRNAs with weight change. This knowledge of the impact of miRNA on obesity has helped with treatment and targeting these biomarkers to treat obesity" (*The Role of MicroRNAs on Adipogenesis, Chronic Low Grade inflammation and Insulin Resistance in Obesity,* 2017). There is also significant evidence that intrauterine health can have an effect on the genetics related to metabolism and appetite regulation in children. Infants that are in-utero when the mother has malnutrition but then are exposed to high fats or infants in-utero whose mothers are obese or have a hunger hormone deficiency are not able to have basic metabolic function (*DNA methylation differences after exposure to prenatal famine are common and timing- and sex-specific*, 2009). There is also evidence that infant exposure to endocrine-disrupting chemicals or obesogens from the mother will result in methylation in metabolic pathways and will increase the likelihood of obesity and excessive weight gain (*Genetic and Epigenetic Causes of Obesity,* 2018).

Obesity is often coupled with other diagnoses in which obesity is a dominating symptom. Syndromic obesity is most commonly associated with the early-onset of obesity in children or pre-development. Two of the most common syndromes associated with obesity from a single gene mutation or a chromosomal disorder are Bardet-Biedl Syndrome (BBS), 16p11.32

microdeletion syndrome, and Prader Willi Syndrome (PWS). These syndromes are most commonly involved in different genetic mutations that have specific comorbidities or symptoms that can be used to diagnose the syndrome for specific treatment. Renavigating hormone pathways is not an option for syndromic obesity so surgery is usually implemented at a young age most commonly using a gastric sleeve. Because of the association with the inability to regulate hunger hormones in the hypothalamus, there is typically a connection to syndromes with decreased cognitive function which is continuously being researched through brain mapping (*An Overview of Monogenic and Syndromic Obesities in Humans*, 2013).

Hunger is controlled through the central nervous system through the leptin-melanocortin pathway in the hypothalamus which involves signals from many different hormones such as ghrelin, cholecystokinin, peptide YY, and glucagon-like peptide. Mechanoreceptors in the stomach and intestines also determine hunger based on distention which is all sensed by the arcuate nucleus. It is commonly a combination of dysfunction between these different receptors that lead to polygenic obesity or the involvement of multiple gene dysfunctions that involve hormones in the brain as well as receptors throughout the body in which genome-wide research can reveal the best-targeted treatment (*Polygenic Obesity in Humans,* 2008).

Nebraska Obesity Statistics

The CDC's Division of Nutrition and Physical, and Obesity (DNPAO) researches state nutrition, physical activity, and obesity. Of those that were represented in the survey, 44.1% of adults engaged in at least 5 hours of physical activity a week, with 24.2% responding that they had not participated in any physical activity in the last month. Based on a BMI of 25 or more, 64.1% of adults in Nebraska were deemed overweight and of this 64.1%, 26.9% were deemed obese with a BMI of 30 or more which has ranked Nebraska as the 15th highest obesity rate

(*Nebraska Obesity Rates Moderate Compared to U.S., Still a Concern,* 2018). This data was pre-pandemic and nationwide obesity has increased since 2020 with 42% of Americans saying they gained weight at an average of 30 pounds from 2020-2021 (*Has the Pandemic Affected Obesity Rates*, 2022). This is most closely related to environmental factors that caused caloric intake without energy expenditure. Without a regular routine, many people were sedentary, stressed about the unknown, and increased consumption of unhealthy foods and alcohol. While this obesity trend is impactful for many current generations, obesity existed before the pandemic and efforts to prevent its prevalence in society ongoing research is occurring for early-onset obesity in children.

One of the CDC's recommendations to decrease childhood obesity from infancy is increasing the duration and frequency of breastfeeding for which the state gathered statistics. In Nebraska, 72.8% of infants were breastfed with 44.4% lasting at least 6 months (*Breastfeeding Report Card*, 2011). This statistic is expected to increase with more remote and work-from-home opportunities as a result of the pandemic. Preschool age children aged 2-5 years old consist of 16.7% overweight with their weight falling into the 85th-95th percentile and 13.7% obese with their weight falling into the 95% percentile or above. Preschool age obesity is especially concerning for healthcare because of the exponential growth that occurs during this age where bone development is crucial. There is an increase in the occurrence of obesity of school age children ages 10-17 due to what is believed a change in lifestyle due to the school-related exposure to junk food and unhealthy options. In Nebraska, 16% of school-age children are obese using BMI measurements. When surveying schools in Nebraska, 24.2% of schools did not provide nutritious options outside of the food service program and 5.3% offered fruits or healthy snacks in vending machines and school stores (*Nebraska Pediatric Nutrition Surveillance*

System, 2010). Nebraska chose to respond to the obesity epidemic starting in early childhood by making the following programs: Foster Healthy Weight in Youth Nebraska's Clinical Childhood Obesity Model, "Whatcha doin?" Campaign, The Great Park Pursuit Outdoor Adventure, and Healthy Communities Intervention Grants Program. These programs focused on developing resources and tools to kids and parents from pediatric clinics, promoting healthy fruits and veggies and making physical activity fun, and increasing state funding to focus on nutrition and physical activity promotion. It was important to implement education to parents to decrease intergenerational obesity due to environmental factors when parents are not encouraging or educating their children on living a healthy lifestyle. In additional demographic research, it is found that in Nebraska African American youth have higher obesity rates than white youth and Hispanic youth have significantly higher obesity rates than African American youth (2016 Nebraska assessment). This has a correlation to the data found by the state of childhood obesity organization where it is found that the

> "food, beverage, and restaurant companies spend almost $14 billion per year on advertising, more than 80% of which promotes fast food, sugary drinks, candy, and unhealthy snacks…because the beverage industry spends millions of dollars every year marketing to communities of color, African American children and teens see more than twice as many ads for sugary drinks than their white peers" (*Nebraska MHC/Youth Needs Assessment,* 2015).

To combat the ads for sugary drink consumption that can increase the risk for obesity, Nebraska worked to increase water consumption and accessibility in schools. It was found that "Replacing sugary drinks and caloric beverages with water has been shown to result in weight loss of 2 percent to 2.5 percent and a reduction of up to 235 calories per day from the average American

diet" (*School Water Access Facts*, 2019). A state bill was implemented that mandated a minimum of two water bottle filling stations, minimum one water bottle filling station for each floor or wing, and minimum one water bottle filling station for every 75 students (*Drinking Water Access in Schools*, 2010). Improvements in promoting healthy living at schools, especially in places where at-home nutrition is not prioritized, is going to hopefully result in less environmentally-related obesity and less adults with obesity and other lifelong physiological setbacks.

In 2018, Nebraska residents were found to consist of 32.8% of adults having a BMI of 30 or more indicating obesity. The data from Nebraska Public Media finds that 1 in 3 Nebraska adults has obesity and 1in 10 Nebraska adults have been diagnosed with diabetes ((*Nebraska Obesity Rates Moderate Compared to U.S., Still a Concern*, 2018). Of these statistics, individuals with low income and those that live in rural communities are the most susceptible to obesity and other comorbidities that impact other physiological systems.

Obesity's Effects on Other Systems

Looking at the physiological effects that obesity implicates on children is an important category to research in order to motivate prevention and to help understand the repercussions. There are many known comorbidities with obesity such as diabetes and cardiovascular issues. In this review it was important to acknowledge the less known repercussions of the disease. From a young age, parents and caretakers assume a majority of the responsibility of the nutrition of growing children. The most important aspects of growth and development are nutrition, energy, and hormones. Obese children are seen to develop earlier and are also taller than non-obese children. This is due to an increase in both leptin and sex hormone levels which accelerate puberty and cause epiphyseal growth plates to close faster than other children at their age

(*Growth and Puberty in Obese Children and Implications of Body Composition*, 2017). Menarche, a marker for puberty in girls, is found to occur at a younger age in girls when childhood obesity is present (Growth and Puberty in Obese Children). It has also been found that an early menarche results in more obesity in adults with women who experienced menarche before the age of 12 weighing about 3kg/m2 more than women who underwent menarche after 13.5 years old (*The relation of menarcheal age to obesity in childhood and adulthood*, 2003).

The development of bones, joints, and muscles is adversely affected by childhood obesity. According to the American Academy of Orthopaedic Surgeons, "excess weight can cause vitamin deficiencies, hormonal imbalances, and increased stress and tension that can affect bone growth and overall musculoskeletal health, causing deformity, pain, and, potentially, a lifetime of limited mobility and diminished life quality" (*The Impact of Childhood Obesity on Bone, Joint, and Muscle Health*). During childhood bones are continuously growing where both size and strength is increased and additional pressure on the growth plates where development occurs can impair this growth and strength. Common problems that arise with the musculoskeletal system combined with childhood obesity are early arthritis, broken bones, blount's disease, impaired mobility and slipped capital femoral epiphysis (SCFE) (*Obesity and the Musculoskeletal System: Age and Impact,* 2022). Many of the injuries that result from these impairments are harder to treat for obese children and adults compared to those that have a lower BMI. Metal implants and crutches may not be able to help improve realignment or mobility for obese patients and typically surgery is the only option for partial or full recovery.

Obesity can not only affect children and adults physical ability, but can also impair children's growing brains. According to the American Psychological Association, "excess weight in children is associated with a range of impairments in executive function, including weaker

working memory, attention, mental flexibility, and decision-making" (*Obesity and the growing brain*, 2016). More research has been implemented in the development and function of children's brains and imaging techniques have been able to find structural changes such as a small orbitofrontal and anterior cingulate cortices and a reduced volume of the hippocampus.

Obesity can have adverse effects on fertility from both the male and female perspectives. Obesity even from a young age can affect fertility by causing the sex hormones in the body, estrone in women and androstenedione in men to not be processed in the brain properly which regulates ovarian and testicular function. In men, obesity can cause sperm count to depreciate or cause the motility in sperm to be insufficient due to increased temperatures from insufficient hormone regulation. Most women with PCOS which occurs in women as young as 15 also have obesity which is the most common hormone disorder that can cause difficulty in getting pregnant. However while pregnant, women with obesity are more susceptible to gestational diabetes, hypertension, preeclampsia, and sleep apnea (*What's the Link Between Obesity and Infertility*, 2014).

The body's largest organ and defense from the outside world reflects obesity in many ways. The skin and integumentary system has comorbidities with obesity in more than 50% of obese patients such as psoriasis, dermatitis, and melanoma (*The Link Between Obesity and Skin*, 2022). Psoriasis has a correlation to obesity due to the involvement of pro-inflammatory mechanisms in which Th1 cytokines are a risk factor for both diseases. Dermatitis and melanoma is correlated with a BMI >30 because of inflammation from unregulated hormones such as leptin, resistin, and ghrelin. More adipose tissue results in a greater skin surface area where there is a decrease in skin resilience and a reduction in ceramides which can lead to a development of striae distensae or commonly known stretch marks. Stretch marks are the result of the breaking

of dermal elastic fibers from excess stretching. These marks are seen as longitudinal lines on places in the body that tend to hold onto adipose tissue (*Characterization of skin function associated with obesity and specific correlation to local/systemic parameters in American women*, 2017). It has also been found that the body's natural defense to heal wounds is impaired in obese patients due to an increased area and decreased lymphatic drainage. A common part of the health of obese patients is the presence of insulin-resistance which has an effect on the skin. Various skin conditions can occur due to these factors but most commonly obese patients find the presence of Acanthosis nigricans and Keratosis pilaris on their skin. Acanthosis nigricans is a skin condition found in folds in the skin that is a result of stimulation of fibroblasts and keratinocytes by insulin and epidermal growth factors that result in dark raised plaques. Keratosis pilaris is a skin condition that is common on follicles around the body that has been found to have a higher association with increased BMI and increased skin surface (*Skin Disorders in Overweight and Obese Patients*, 2014). Skin surface can also lead to intertrigo in which skin folds are subject to friction and bacteria growth which can lead to other medical complications.

Environmental Factors of Obesity and Lifestyle Suggestions

There are many economic factors that help shape the environmental cause of obesity. A large part of this economic setback is healthy food being affordable and accessible to the entire population. Some research has placed the blame of obesity-inducing eating habits on government policies such as the farm bill which for decades has provided subsidies for farm products which are common products put into fattening food such as oils and sweeteners and fewer subsidies to fruits and vegetable growth (*Agricultural Subsidies and the American Obesity Epidemic*, 2013). The foods sponsored the most in the food bill are "commodity-rich foods" or livestock that

consume the commodity-rich foods which include corn, soybeans, wheat, rice, and sorghum. According to the USDA, there is a direct correlation between those that eat commodity-rich foods and being overweight or becoming obese. There is frustration from dieticians in regard to dietary recommendations for fruits and vegetables but not enough acreage is subsidized for this food group compared to the commodity-rich foods. With the US population and current dietary guidelines for vegetables at 2.5 cups per day, there would need to be 15 million more acres of vegetables grown in the US when it currently is at about 4 million acres. In comparison, corn, soybeans, and wheat occupy about 219 million acres which is encouraged by the government subsidies from the food bill which in turn makes these ingredients cheap for processed and unhealthy foods (*Finding the fat: The US Farm Bill and Health*, 2018).

Including information about environmental factors that lead to obesity would not be complete without including information about the ways in which screens and digital media have impacted health and childhood to adult obesity. With a change in lifestyle in children due to technology over the last few decades and the lifestyle effects that have carried these children into adulthood, there has been research targeting the connection between technology abuse and obesity. Spending time on screens does not have a direct effect on physical activity as those not interested in physical activity do not replace this activity when screens are taken away. However, there is a positive connection between energy intake and spending time on the screen. One study found that “up to a third of daily intake and half of children’s meals were consumed in front of a screen” (*Social Media Exposure and Obesity in Children and Adolescents*, 2017). Being in front of a screen is often associated with high-calorie foods and beverages which could prompt the need to eat when sitting in front of a screen or overeating because of the presence of a screen. Eating should be its own task or activity and eating while multitasking can distract from our

brain's processing of satiety hormones where we can understand if we are full or need additional food to satisfy our needs.

Screen time also has an effect on inadequate sleep which increases the risk for obesity and weight gain, especially for children. Lack of sleep causes the hormones that regulate appetite to not work properly in which hormones such as ghrelin and leptin increase the feeling of hunger and inhibit satiety to be reached at a normal level. Decreased sleep can also cause eating habits outside of normal meal times which impact the body's processing of foods and increase unhealthy cravings (*Sleep Deprivation: Effects on Weight Loss and Weight Loss Maintenance,* 2022). This is a problem plaguing high school, college, and upper education level students due to study habits that impact sleep and eating while multitasking.

In a study specifically looking at the environmental impact on obesity, college students are especially impacted. According to the Obesity Medicine Association, "A significant number of college students gain weight during their college years. Almost 1 in 3 college American students have obesity presently. According to a survey in 2021, 44% of college students in the US described their weight as more than normal, i.e. either in the overweight or obese category" (Obesity Medicine Association, 2022). Not only does obesity impose negative health impacts, it can also impact academic performance and well-being. While drinking and binge-drinking is a common assumption for the weight-gain of college students there is not a positive correlation between alcohol consumption and obesity and it is found to be more correlated to diet, stress, and sleep. Many students are put into an environment where they are now responsible for their meals and with healthy meals usually being more costly, it is frequently inexpensive and quick foods that the college student consumes. Many college students may have played sports or were involved in physical activity and now with hectic school and potentially work schedules, they do

not find the time to exercise like they once did and their body cannot support the calorie consumption without exercise like it was capable of before. Suggestions for college students are to eat three balanced meals a day at normal meal times and to avoid snacking and all-nighters as those inhibit the proper digestion process by not allowing proper hormone regulation. University of Nebraska-Lincoln has made it a goal to promote healthy living and houses the Nebraska Center for the Prevention of Obesity Diseases Through Dietary Molecules (NPOD). Many on-campus students have noticed healthier food options through the dining halls to help promote healthy food intake.

Medical Treatments

While changes to environmental factors are able to be implemented to help reduce the rates of obesity, medical treatments are often needed for severe cases. For severe obesity, one of the most effective treatments has been bariatric surgery. The treatment has been found to be effective for genetic obesity causes in both adolescents and adults. This bariatric surgery most commonly involves a laparoscopic sleeve gastronomy procedure. This procedure has been shown to make improvements in ghrelin response and a reduction in food-seeking behavior when not needing to satisfy basic hunger needs (Matson et,al. 2012). Due to obesity being tied to a genetic component, there has been specific drug research for obesity-related genetic disorders where medication intervention is needed. The FDA recommends the prescription Imcivree for obesity in patients over the age of 6 that have tested positive for genetic conditions of pro-opiomelanocortin deficiency (POMC), proprotein subtilisin/kexin type 1 (PCSK1) deficiency, and leptin receptor deficiency (LEPR). Children and adults that are found to have this genetic deficiency are typically a normal weight at birth but quickly gain weight due to insufficient hunger signaling due to their respective conditions (*Treatment for weight*

management for people with certain genetic conditions, 2020). The medication Imcivree does not make genetic changes that affect weight gain but activates parts of the brain where hunger is regulated and also increases metabolism. For children with non-proven genetic obesity, there are non-invasive recommendations suggested first before prescriptions or surgical treatment is implemented. Most suggestions are in regard to lifestyle changes such as healthy eating, physical activity, limiting sedentary activity, and household support for healthy living. Medication for childhood obesity is only recommended to be implemented when there are severe comorbidities and lifestyle changes have been implemented and found ineffective. Family history is also an important factor when determining treatment such as cardiovascular risk or diabetes. Medication recommendations from the FDA include orlistat and metformin for non-genetic obesity causes. Orlistat is a gastric and pancreatic lipase inhibitor that helps limit the absorption of cholesterol from foods. This can cause problems with growth when implemented early in adolescents so other medications and vitamins may need to be used in combination. Metformin is a commonly-known treatment for type-2 diabetes mellitus for patients at least 10 years of age. When patients have an accumulation of adipose tissue, there is typically an association with insulin resistance. Metformin works by activating adenosine monophosphate-activated protein kinase to reduce glucose absorption, reduce glucose production, and increase glucose sensitivity (*Pharmacological Treatment of Overweight and Obesity in Adults*, 2021). The presence of medical treatments should not reduce the importance of implementing healthy living choices to reduce obesity. In the case of obesity, medicine can help treat but to prevent obesity it is up to the health consciousness of the person and the resources around them.

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