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ABSTRACT (Maximum 250 words. The template will warn you if you exceed the limit. Section are automatically established based on the article type. Do not change them.)

Background: With the ever-increasing incidence of obesity in pediatric and adult populations nationwide, the need to find impactful and long-term solutions to the epidemic is paramount. Urban and rural populations both show climbing rates of obesity, with significantly more children and adults being affected in rural areas. Many of the most prominent factors leading to obesity are related to socioeconomic status, behavior, lack of access to quality food resources, and lack of knowledge of healthy food groups. Obesity increases the risk of disease in nearly every organ system in the body.

Methods: The authors conducted a narrative review by searching for relevant articles written in English in electronic databases such as PubMed/MEDLINE, Web of Science, Semantic Scholar, and Google Scholar. A total of 88 articles were studied, and finally, 47 articles were included.

Results: Environmental factors such as low access to fresh produce and living in a rural areas increase the risk of adults and children developing obesity. Early intervention in pediatric obesity cases have shown promising effectiveness on obesity prevention and it is suggested that stepwise, realistic goal-setting can lead to greater success in weight loss and management.

Conclusion: With the rates of obesity amongst children and adults continuing to climb in rural areas, addressing the causes is paramount. Biological, behavioral and social factors all contribute to obesity rates in rural areas, often due to the challenges of accessing healthy food and fewer spaces for physical activity.

Keywords (Keywords must be as many as possible and from the US NLM at https://www.ncbi.nlm.nih.gov/mesh)**:**

Obesity, physical activity, Obesity complications, Obstructive sleep apnea (OSA) , Rural and urban population, Leptin, Pro-opiomelanocortin (POMC)

INTRODUCTION

The climbing obesity rates in the United States and the resultant obesity epidemic were first identified among the adult population in the 1970s. Since then, the incidence of obesity has steadily risen, with over 72 million adults in the United States affected by the disease, according to recent studies.[1] However, the rise in pediatric obesity in the last 30 years is newer and equally concerning.[2] It is a risk factor diseases such as early-onset diabetes mellitus type 2 (DM2) and obesity in children are also more prone to significant problems with mental health and self-esteem. It is also a risk factor for Diabetes Mellitus Type 2, Non-alcoholic fatty liver disease (NAFLD), non-alcoholic steatohepatitis (NASH), hypertension, and obstructive sleep apnea. 34% of children in the U.S. are considered obese,[3] and these same children have a higher chance of becoming obese adults.[4] It has become clear that preventing pediatric obesity will also be an important part of improving the quality of life of the U.S. population.[4]

Obesity is connected to several leading causes of preventable deaths in the U.S., making the need for understanding and finding solutions to the multifactorial causes of obesity significant to ensure it does not soon outpace smoking as the leading cause of preventable premature death. One of the challenges of obesity prevention stems from its complex causes, ranging from biological and psychological causes to socioeconomic and geographical factors and beyond.[5]

The distribution of these afflicted communities is neither linear nor static. However, people living in rural areas were found to have higher occurrences of obesity.[6] These individuals, especially those living in a rural area of low socioeconomic status, are especially vulnerable. Various social determinants of health often afflict these areas, some of the most prominent being a lack of access and transportation to healthcare, a lack of affordable healthy food options, and low health literacy. These challenges make obesity prevention even more challenging because they often lead to the main causes of both pediatric and adult obesity.[6]

Calorie intake exceeding caloric expenditure is one of the leading causes of obesity.[3] High-calorie, sugar-sweetened drinks are especially related to both pediatric and adult obesity. In addition, the sedentary lifestyle promoted by the dawn of the new technological age is another large contributor to the increasing

rates of obesity.[7] However, while it is easy to assign blame to these behaviors, one cannot ignore the environmental and psychological factors that also play a role. Fresh produce, which is incredibly important for a healthy diet, is often too expensive for low-income families to afford. This can lead to cheaper food options such as frozen meals and fast food as affordable alternatives. In addition, personal conflicts such as mental health problems and stigmatization have been seen to correlate with obesity rates, especially in adult populations.[8] Such complexities have made finding effective prevention and education measures difficult despite past and current efforts. Weight loss programs, drugs for weight loss, and bariatric surgery are among a few of the methods by which public health and healthcare workers are attempting to mitigate the obesity epidemic.[9] However, with the varying or even low success of some of these programs and the financial and safety burdens implicated in using pharmacological or surgical options, the goal of finding and implementing programs or medical options to enhance long-term weight loss success is still an ongoing challenge.[9]

Epidemiology

As per WHO, since 1990, obesity in adults has doubled, and obesity in adolescents has increased by four times. In 2022, one in eight people in the world was living with obesity. Of 2.5 billion people, 43% were overweight and 16%, or 890 million, have obesity. 37 million children under the age of five and over 390 million children and adolescents aged five-nineteen years were overweight, and 60 million were living with obesity.[10]

According to the CDC, during August 2021–August 2023, the prevalence of obesity in the US was 40.3%, in adults, approximately the same in males and females. For age-specific data, prevalence was higher in the age group of 40–59 than in ages 20–39 and 60 and older. The prevalence of obesity was lower in adults with higher education levels. The prevalence of severe obesity in adults was 9.4% and was higher in females than males for each age group. From 2013–2014 through August 2021–August 2023, the age-adjusted prevalence of obesity did not change significantly, while severe obesity prevalence increased from 7.7% to 9.7%.[11] Approximately 1 in 5 U.S. children and adolescents have obesity. Obesity affects some groups more than others, including adolescents, Hispanic and non-Hispanic Black children, and children in families with lower incomes. Health care for obesity is expensive for patients and the health care system. Between 2017-2020, 19.7% children and adolescents in the US were found to have obesity. Predictions estimate that by 2030, nearly half of the U.S. adult population will become obese, significantly, including severe obesity.[12]

Obesity is a serious and common chronic disease affecting some groups more than others, including, non-Hispanic Black adults and adults with less education.[13] To determine whether an individual is obese, body mass index (BMI) is a quick and reliable measure. For adults, the following formula and ranges have been established:.[13]

[BMI = weight(kg) / Height $(m)^2$] [13]

For adults, BMI categories are underweight, healthy weight, overweight, and obesity which is further classified into three categories. The following values are referenced for adults age 20 and older.[14] For children and teens who are growing, BMI is compared to other children and teens of the same sex and age. This is accomplished using BMI for age percentiles or growth charts. Percentiles are used to rank an individual by indicating what percent of the reference population the individual equals or exceeds and are the best indicator to evaluate growth patterns in children.[13] Please refer to table 1 for BMI classifications of adults and Children.[13]

Table 1. BMI Classifications for Weight in Adults and Children

BMI category	BMI Range (kg/m^2) in adults	BMI Range in children
Underweight	Less than 18.5	Less than 5^{th} percentile
Healthy Weight	18.5 to less than 25	5^{th} percentile to less than 85^{th} percentile
Overweight	25 to less than 30	85^{th} percentile to less than 95^{th} percentile
Obesity	30 or greater	95^{th} percentile or greater
Class 1 Obesity	30 to less than 35	120% of the 95^{th} percentile or greater, or 35 kg/m^2 or greater
Class 2 Obesity	35 to less than 40	120% to less than 140% of the 95^{th} percentile, or BMI 35 kg/m^2 to less than 40 kg/m^2
Class 3 (Severe Obesity)	40 or greater	140% of the 95^{th} percentile or greater, or BMI 40 kg/m^2 or greater

Table 1: Body Mass Index (BMI) has slightly different classifications based on whether the individual is an adult or a child.

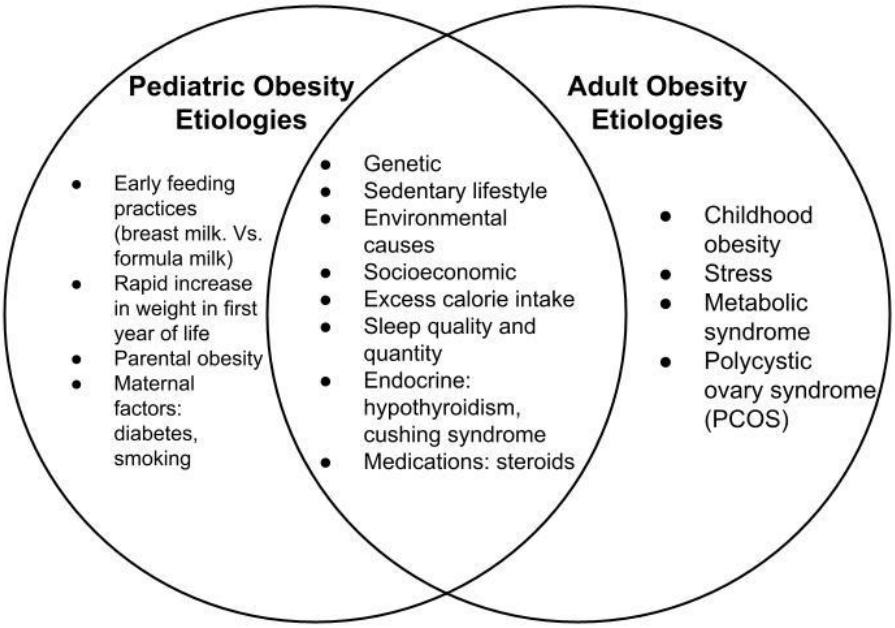
Etiology of Pediatric and Adult Obesity

Obesity is a multifactorial condition that is greatly affected by environmental and social factors, as well as some genetic predispositions. Specific genes, such as leptin, pro-opiomelanocortin, and melanocortin 4 receptor genes, have all been linked to obesity.[15] Although their occurrence is rare, the resulting association with obesity of the mutations within these genes has been key to better understanding obesity's pathogenesis.[16]

Studies have shown that a very prominent etiology for pediatric and adult obesity is related to environmental components.[16] Sedentary lifestyles and dietary intake both profoundly impact the risks of obesity. Children who have one or both parents who are overweight in the house have a much higher chance of developing obesity. And consequently, overweight children are far more likely to become overweight adults.[17]

There are also risks associated with developing obesity depending on the nutritional and lifestyle environments pregnant women and children experience at specific times. Epigenetic changes play the role of intergenerational transmission of obesity along with genetic factors. Exposure to factors in the environment makes children susceptible to obesity even without inheriting genetic factors.[17] Please refer to Figure 1 for causes of obesity in the pediatric and adult population.[15-17]

Figure 1. Pediatric vs. Adult Obesity Etiology [15-17]



Pathophysiology

The pathophysiology of obesity is related to energy expenditure in calories being less than the amount of energy intake.[18] Fat, also called adipose tissue, is our main energy storage source. White fat, the main component of adipose tissue, plays a major role in storage of energy and physiology. The triglycerides stored in this white fat store the body's available energy and are broken down and circulated throughout the body as free fatty acids during fasting or starvation.[18]

As an endocrine organ, adipose tissue also plays essential roles in energy balance and reproduction within organisms.[19] It is stored most prominently in the cephalad regions of the trunk for men and caudal regions of the trunk and subcutaneously for women. Increased weight eventually leads to the hypertrophy of the adipose tissue.[20] This change in their structure leads to changes and effects in their supporting tissue and blood supply, resulting in further complications as weight gain continues.[20]

Specific to energy storage is a hormone called leptin, secreted by adipose tissue. Leptin is secreted in specific amounts to help maintain ideal energy balance within organisms.[20] Increased body weight increases plasma leptin concentrations, often leading to a negative energy imbalance and can perpetuate weight gain. The rate at which leptin is produced is proportional to the amount of adipose tissue in the body, and its role in increasing hunger signaling in the brain can lead to increased appetite. This and its concentration in plasma play a role in adipocyte production and, causatively, weight gain. Leptin plays an important role in monogenic obesity, especially in congenital leptin deficiency.[21]

The central and peripheral pathways that control hunger and, consequently, play a role in energy intake, expenditure, and weight loss play significant roles in obesity. Within the arcuate nucleus of the hypothalamus, pathways and signals regulating hunger and energy homeostasis are integrated and regulated. Two pathways, the agouti-related peptide(AgRP)/neuropeptide Y (NPY)-expressing neurons and the pro-opiomelanocortin (POMC)/cocaine-and amphetamine-regulated transcript (CART)-expressing neurons play essential and opposite roles in regulating energy intake.[22] The AgRP/NPY pathway is orexigenic, which promotes hunger and food consumption while down-regulating energy expenditure during periods of low energy intake. The hormones insulin and leptin, which facilitate glucose uptake into cells and send "hunger signals" to the brain, respectively, play a large part in regulating this pathway.[22]

The POMC/CART pathway performs the opposite function. As an anorexigenic signaling pathway, it promotes energy expenditure and encourages decreased food consumption.[23] These neurons are stimulated by leptin to release a neuropeptide called alpha-melanocyte-stimulating hormone (α-MSH), which leads to decreased food consumption. Opposite to the AgRP/NPY pathway, the POMC/CART pathway is inhibited during low energy, so its appetite-suppressing actions are inhibited, and greater energy uptake is promoted. The AgTP/NPY pathway is also inhibited by activating 1B receptors for the neurotransmitter serotonin 5-hydroxytryptamine (5-HT).[22] A decrease in the signaling for 5-HT is highly correlated with increased appetite and weight gain, while increased signaling for 5-HT has the opposite effect. Norepinephrine (NE) is another neurotransmitter that could potentially affect hunger and weight gain. It increases food uptake via the stimulation of the dorsal pathway and inhibits food uptake by activating the ventral pathway in the hypothalamus.[24] Please refer to Figure 2 below for a diagram of the effects of the POMC/CART and AgRP/NPY pathways on food intake.[22-24]

Figure 2. The POMC/CART and AgRP/NPY Pathways' Effects on Food Intake [22-24]

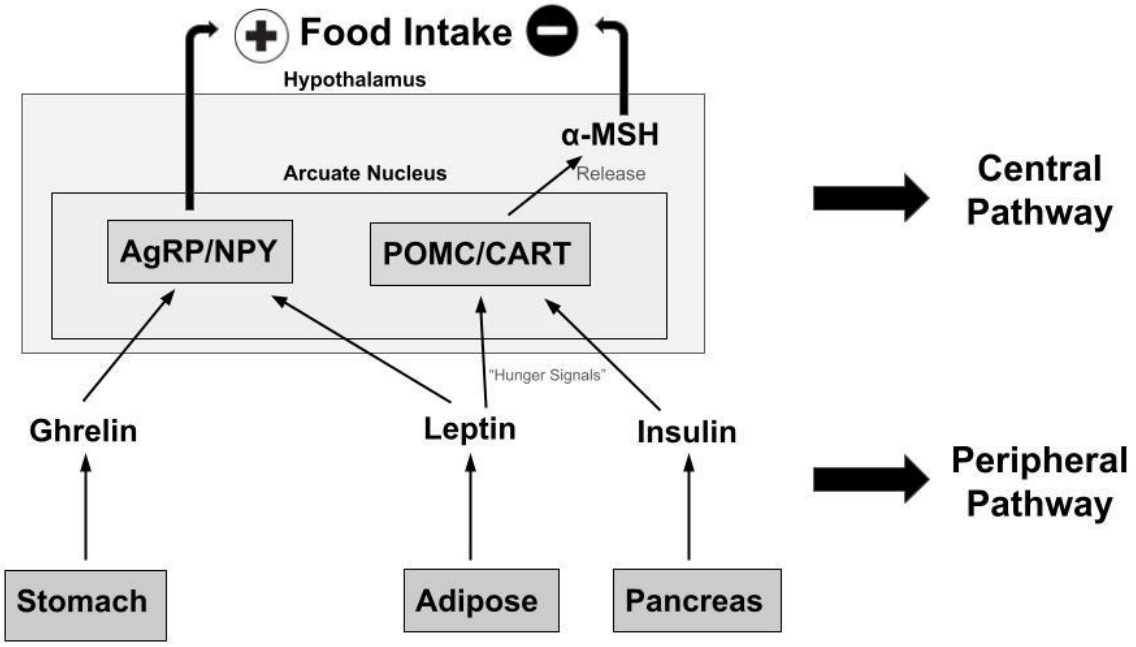


Figure 2: The agouti-related peptide(AgRP)/neuropeptide Y (NPY)-expressing neurons and the pro-opiomelanocortin (POMC)/cocaine-and amphetamine-regulated transcript (CART)-expressing neurons have opposite roles in regulating energy intake.

However, integration and signaling in the hypothalamus are not the only locations where obesity-related hormones are made. Hormones secreted in the gastrointestinal system play a role in the peripheral pathway of hunger and energy regulation.[22] Intestinal hormones glucagon-like peptide-1 (GLP-1), cholecystokinin (CCK), and peptide YY (PYY) are secreted when there is food intake and play an anorexigenic role by promoting satiety. Ghrelin, a hormone secreted by the stomach, is the single known orexigenic hormone, also called the hunger hormone, and it promotes increased appetite and energy consumption. Low levels of this hormone during fasting have an inverse correlation with BMI, and it has been seen that altered suppression of this hormone has been correlated to obesity. These central and peripheral pathways play a role in energy homeostasis and, as a result, play a key role in obesity.[22]

Complications:

People who are overweight or obese have an increased risk for many severe diseases, including early death, hypertension, high or low LDL cholesterol and high triglyceride levels, type 2 diabetes, coronary heart disease, stroke, gallbladder disease, osteoarthritis, sleep apnea and breathing problems, many types of cancers, lower quality of life, mental illness, and body pain.[25]

According to the WHO, being overweight in childhood and adolescence affects immediate health and increases risk and earlier onset of non-communicable diseases in them. Childhood and adolescent obesity also have adverse psychosocial consequences. It affects school performance and quality of life, compounded by stigma, discrimination and bullying.[26]

There are additional findings in pediatric patients who are obese. Obese adolescents have advanced puberty with advanced menarche, although this advance is not so constant in males. Boys with obesity frequently present with gynecomastia and it also increases risk of pediatric obstructive sleep apnea (OSA).[27] Psychosocial problems develop as a result of weight-based stigma. One aspect of stigma is poor integration of the network of friends in a social environment, which initially leads to passive marginalization due to diminished attractiveness as a friend.[27] Similar to weight-based stigma, bullying occurs in boys or girls who are obese, specifically in places where no preventative measures are in place.[27] Another common finding is low self-esteem consisting of an emotional self-evaluation of one's worth - it contributes to a certain state of happiness or unhappiness.[27] Please refer to Table 2 for summary of complications of obesity.[25-27]

Table 2. Summary of Complications of Obesity [25-27]

Complications of obesity
Greater height and growth rate
Advanced puberty, menarche, gynecomastia
Polycystic Ovarian Syndrome begins at puberty
Musculoskeletal Disorders
Sleep Disorders, Obstructive Sleep Apnea
Respiratory complications, asthma, breathing issues
Increased risk of diabetes, hypertension, heart issues
Psychosocial Problems: Stigma, bullying, low self esteem
Non alcoholic fatty liver, gallstones

METHODS

Search strategy and selection criteria

The authors conducted a narrative review by searching for relevant articles written in English in electronic databases such as PubMed/MEDLINE, Web of Science, Semantic Scholar, and Google Scholar pertaining to the topics of obesity in pediatric patients and in adults in rural as well as urban areas. We selected keywords identified through the literature, and for each keyword, we undertook a search of the existing literature. Keywords used are Obesity, physical activity, Obesity complications, Obstructive sleep apnea (OSA), Rural and urban population, Leptin, and Pro-opiomelanocortin (POMC). All three authors performed the search separately and included it in the manuscript after agreeing on the existing literature. Authors JY and PM did the initial search, and SK addressed the differences of opinion based on scientific data and evidence-based medicine. The purpose of this review is not to perform a systematic review but to identify available evidence that can be useful for informing providers about obesity. The included articles met all the following inclusion criteria: literature published in English from 2001 onwards, evidence for specific causes, pathophysiology for children with obesity, and evidence targeting children under the age of 18 and adults. Additionally, thirteen articles are included from the past five years, especially for World Health Organization (WHO) and Centers for Disease Control and Prevention (CDC) guidelines inclusion. One author specifically examined and evaluated the titles as well as the abstracts of each of the retrieved records to determine which ones were appropriate and should be selected per the eligibility criteria. Articles not written in English and articles which are not related to obesity topics were excluded. A total of 88 articles were studied, and finally, 47 articles were included to write this piece of narrative review.

RESULTS

Factors Most Affecting Pediatric and Adult Obesity in Rural vs. Urban Areas

Several factors affect the likelihood of pediatric obesity, regardless of whether the child grows up in a rural or urban area. Maternal obesity and diabetes are correlated to a higher birth weight in newborns.[28]. While the mechanism behind this is unclear, it cannot be stated definitively whether the infant's early exposure to glucose or changes in mechanisms of energy balance is the cause of this higher birth weight. However, this correlation is strong enough to warrant further investigation. Another impact that affects the likelihood of childhood and adult obesity is environmental factors. If the child and their guardian(s) live in

areas with ethnically-targeted food marketing, low access to fresh produce, and high access to fast food, these aspects can also lead to a greater risk of pediatric obesity in these populations.[28] However, despite some similarities, there is a clear disparity between the risk of obesity in pediatric and adult populations in rural vs. urban areas.

Children living in rural areas have a 26% greater risk of becoming obese than children in urban areas, with an odds ratio of 1.26 (95% confidence interval, 1.21–1.32).[29] One of the factors that has affected this statistic is access to areas where children can play safely. Recreational areas such as parks close to the city to increase relative safety and give more opportunities for children to play and exercise are more available in city areas but not in rural communities.[30] Children in rural areas have also been found to watch more television and play more video games that promote a sedentary lifestyle.[30] Because children are also more likely to consume high-fat or high-calorie foods while watching television may also lead to an increased obesity rate in rural pediatric populations. A lack of resources for weight management in smaller schools is another barrier faced by children in rural areas.[30]

Adults in rural populations also face specific barriers that increase their risks for obesity more than their urban counterparts and can also affect their children as a result. One of the significant barriers is a lack of nutritional education and access to resources such as nutritionists.[30] Many are unaware of nutritional recommendations and portion sizes, and are further challenged by the high costs of healthier food options and time limitations. This lack of nutritional literacy and socioeconomic challenges are further exemplified by adults in rural areas consuming, on average, fewer portions of fruits and vegetables and consuming more high-sugar soft drinks.[32] The lack of access to nutritionists trained explicitly in advising and coaching on nutrition is also a benefit that more urban residents have that is very limited to rural communities. To make matters more complicated, it has also been found that nutritionists are more unlikely to work in underserved areas than other health professionals.[33] Challenges such as this have led to a prevalence of obesity amongst adult rural populations to be 6.2% greater than the prevalence seen in adult urban communities (P= .006).[34]

Table 3. Statistics of Rural vs. Urban Obesity Prevalence[32, 34-35, 37]

Rural vs. Urban Obesity Prevalence				
Study	State (if applicable)	Rural Obesity (children or adults)	Urban Obesity (children or adults)	Statistical Significance
Johnson & Johnson, 201532	N/A	26% greater odds than urban children	26% lesser odds than rural children	odds ratio=1.26 95% confidence interval (1.21–1.32)
Tai-Seale & Chandler, 201034	Michigan	3-9% greater prevalence than urban children	3-9% less prevalence than rural children	N/A
Tai-Seale & Chandler, 201034	North Carolina	50% greater risk of obesity than urban children	50% decreased risk of obesity than rural children	p=0.0001
Trivedi et al., 201535	N/A	35.6% prevalence	30.4% prevalence	p<0.01
Befort et al., 201237	N/A	39.6% prevalence	33.4% prevalence	p<0.001

Management

Management includes the prevention of occurrence and treatment of obesity and its complications. WHO recommends starting both preventive and treatment of complication measures to be implied as soon as possible. Several programs and existing initiatives are already in place to promote healthier living habits and decrease the occurrence of obesity. Schaap et al in 2018 studied the fidelity of obesity prevention programs in the school. The study recommended better reporting of methods used as preventive measures against obesity.[35] Lombard et al.[36] reviewed papers from five different databases written for the prevention of weight gain, discussing nine methods applied for the prevention of weight gain in adult patients over a period of one to three years of duration. Some studies showed a difference in weight of 1-3.5 kg between the study and control groups. The study concluded that they could not find a successful formula for the prevention of weight gain, and high-intensity ways are not more successful, but a mixture of mail and in-person intervention was useful.[36]

Management starts with the very basic and most important step in history. The history of onset, progress, and associated complications of obesity is equally important as medication-induced obesity. Providers should ask about all over-the-counter and prescription medications, and weight-gain-causing medications should be used in the minimum dose and for the shortest duration possible. Lab tests should be ordered based on specific history as well as routine labs. Asking in detail about the strategies that have helped in weight loss can be a starting point for restarting the weight loss journey. The Obesity Medicine Association suggests bariatric surgery and medications for eligible patients along with healthy eating habits, physical activity, and behavioral counseling.[37]

The position statement of Italian society also emphasizes lifestyle intervention, medication use, and surgery for weight loss as the first, second, and third steps. As per the position statement, the availability of new safe and efficacious medications and medications under trials is increasing hopes for weight management in pediatric patients, especially adolescents.[38]

According to the American Academy of Pediatrics, clinical practice guidelines (CPG), obesity in pediatric patients is a treatable condition, and despite gaps and complexities, available evidence provides hope that increasing obesity can be managed by pediatricians. Instead of watchful waiting or a stage-wise approach, CPG recommends obesity management at a higher intensity level as per availability by collaboration of

pediatricians, other primary care physicians, dietitians, nutritionists, exercise specialists, policy makers, and community partners.[39]

DISCUSSION

Prevention

Despite a lack of standardized or structured obesity prevention, some promise has been seen with targeted prevention.[40] A two-year study done in Finland found that their intervention program on 13-15 year old patients resulted in promising improvement in known comorbidities of obesity, such as improved food consumption habits and decreased cholesterol. However, there was no significant decrease in obesity itself. Even without this decrease, the results of this study indicate that this intervention strategy could be potentially effective.

Based on these results and experience from individual patient cases, it is recommended that obesity prevention initiatives should be focused on the following: (1) early prevention strategies in the pediatric population, (2) increased education of patients and guardians about the comorbidity risks that come with pediatric and adult obesity, and a (3) stepwise, tailored prevention and management of obesity plans for patients.[41] To implement all preventive and management strategies effectively, it is recommended to use the methods of motivational interviewing (MI). Providers can start the conversation by using the 5As technique, which consists of "Asking" about a specific health-related behavior," Advising" the patient about the behavior, "Assessing" readiness to change, "Assisting" with goal setting, and "Arranging" follow-up.[40] Four communication skills such as "OARS", asking open-ended questions, providing affirmations, emphasizing points which the patient had said by reflective listening and putting everything to summarize can help to build the motivation for the change in the patients with obesity. Patients can be encouraged to come up with a SMART goal, which stands for specific, measurable, attainable, result-focused, and timed.[42]

More specifically, according to the CDC, there are various ways families can help avoid obesity.[37] Healthy eating style adopted by the entire family helps children maintain a healthy weight, beginning with modeling a healthy eating pattern - parents can offer a variety of fruits and vegetables throughout the day, such as frozen and canned fruits and vegetables, as they are often less expensive than fresh and are still a great nutritional source.[43]

Families can maintain physical activity levels together. Activities such as walking the family pet before and after school, walking to the school and back home from the school, riding a bike, washing vehicles, or raking leaves count as physical activity.[43] Lack of sleep is another contributing factor that is usually neglected. Setting a consistent sleep routine to good quality and quantity sleep also helps to prevent type 2 diabetes, injuries, and problems with attention and behavior, along with obesity.[43] CDC recommends 14-17 hours of sleep for newborns, 12-16 hours for infants, 11-14 hours for toddlers, 10-13 hours for preschoolers, 9-12 hours for school-going children, 8-10 hours for adolescents, and more than 7 hours for adults.[44]

Wills-Ibarra et al. 2024 studied Family Systems Theory (FST), which included various factors related to family intervention in obesity management, including interaction, support, and skills of parents and their effects on body mass index, changes in lifestyle, and effects on mental health. The study found improvement in BMI. The review discussed the role of existing FST and provided recommendations for future practice and research.[45]

In adults, recommendations to prevent obesity include tracking food intake. Eating five to nine servings of fruits and vegetables daily (One vegetable serving includes 1 cup of raw vegetables or ½ cup of cooked vegetables or vegetable juice while one fruit serving includes one small to medium fresh fruit, ½ cup of canned or fresh fruit or fruit juice, or ¼ cup of dried fruit).[45] Adults are also recommended to select whole-grain foods (brown rice, whole wheat bread) while avoiding highly processed foods. Additionally, adults are recommended to engage in at least five days of moderate physical activity combined with resistance and strengthening exercises.[45] Not only calculating the calories, but also education for reading the nutrition labels and calculating the portion size should be made a part of the prevention plan.[46]

Future directions:

A multi-faceted approach by an individual, family, and community is needed to combat obesity. Prioritizing healthy and appropriate portion size eating, following a daily routine of physical activity, getting age-recommended sleep hours, managing stress, tension, and seeking professional health care on time should be considered for every patient for the effective management of obesity.[47]

Conclusion:

Obesity is a chronic disease that afflicts children, teens, and adults alike. Childhood obesity can lead to obesity in adulthood and will increase the risk of developing chronic conditions such as diabetes, hypertension, and heart disease. With the rates of obesity amongst children and adults continuing to climb in rural areas, addressing the causes is paramount. Biological, behavioral, and social factors all contribute to obesity rates in rural areas, often due to the challenges of accessing healthy food and fewer spaces for physical activity. By targeting treatments and interventions at these social determinants of health, we can improve individual health and foster healthier communities. By providing more comprehensive weight-loss plans, we hope to help reverse the current obesity trends in pediatric and adult populations in rural America.

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Tables

Table 1. BMI Classifications for Weight in Adults and Children

BMI category	BMI Range (kg/m^2) in adults	BMI Range in children
Underweight	Less than 18.5	Less than 5th percentile
Healthy Weight	18.5 to less than 25	5th percentile to less than 85th percentile
Overweight	25 to less than 30	85th percentile to less than 95th percentile
Obesity	30 or greater	95th percentile or greater
Class 1 Obesity	30 to less than 35	120% of the 95th percentile or greater, or 35 kg/m^2 or greater
Class 2 Obesity	35 to less than 40	120% to less than 140% of the 95th percentile, or BMI 35 kg/m^2 to less than 40 kg/m^2
Class 3 (Severe Obesity)	40 or greater	140% of the 95th percentile or greater, or BMI 40 kg/m^2 or greater

Table 2: Body Mass Index (BMI) has slightly different classifications based on whether the individual is an adult or a child.

Table 2. Summary of Complications of Obesity [25-27]

Complications of obesity
Greater height and growth rate
Advanced puberty, menarche, gynecomastia
Polycystic Ovarian Syndrome begins at puberty
Musculoskeletal Disorders
Sleep Disorders, Obstructive Sleep Apnea
Respiratory complications, asthma, breathing issues
Increased risk of diabetes, hypertension, heart issues
Psychosocial Problems: Stigma, bullying, low self esteem
Non alcoholic fatty liver, gallstones

Table 3. Statistics of Rural vs. Urban Obesity Prevalence [32, 34-35, 37]

Rural vs. Urban Obesity Prevalence

Study	State (if applicable)	Rural Obesity (children or adults)	Urban Obesity (children or adults)	Statistical Significance
Johnson & Johnson, 201532	N/A	26% greater odds than urban children	26% lesser odds than rural children	odds ratio=1.26 95% confidence interval (1.21–1.32)
Tai-Seale & Chandler, 201034	Michigan	3-9% greater prevalence than urban children	3-9% less prevalence than rural children	N/A
Tai-Seale & Chandler, 201034	North Carolina	50% greater risk of obesity than urban children	50% decreased risk of obesity than rural children	p=0.0001
Trivedi et al., 201535	N/A	35.6% prevalence	30.4% prevalence	p<0.01
Befort et al., 201237	N/A	39.6% prevalence	33.4% prevalence	p<0.001

Figures

Figure 1. Pediatric vs. Adult Obesity Etiology [15-17]

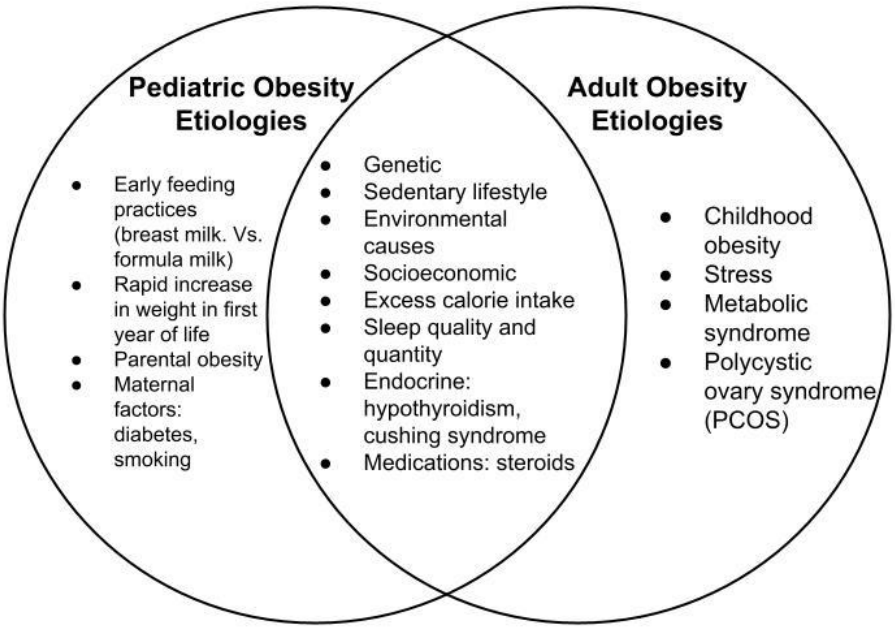

Figure 2. The POMC/CART and AgRP/NPY Pathways' Effects on Food Intake [22-24]

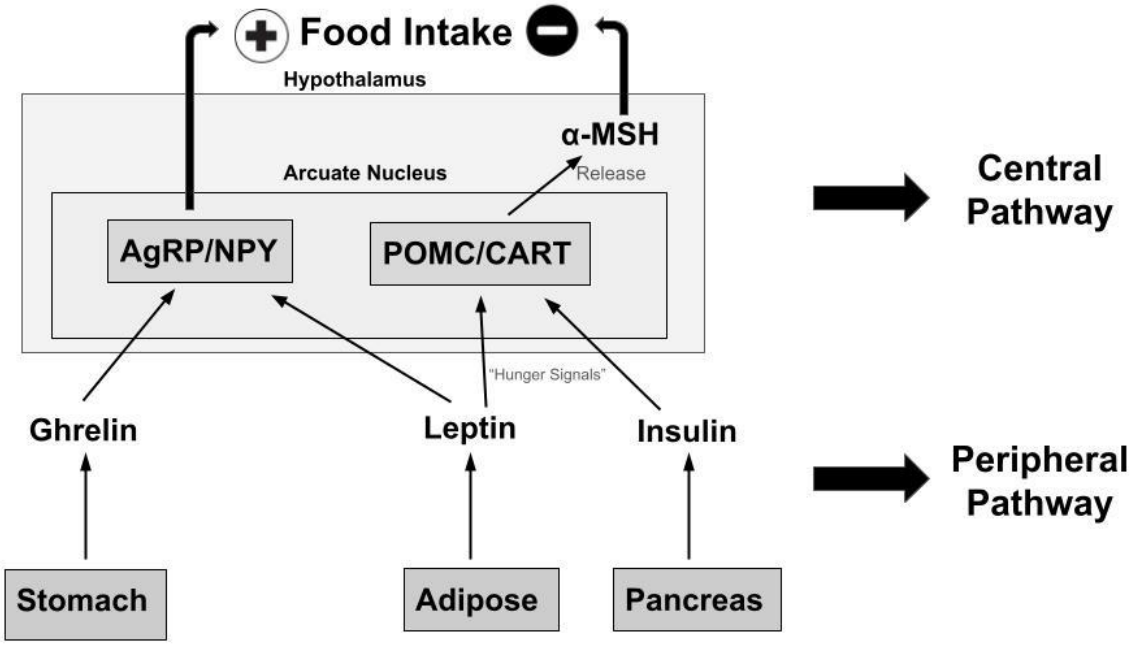

Figure 2: The agouti-related peptide(AgRP)/neuropeptide Y (NPY)-expressing neurons and the pro-opiomelanocortin (POMC)/cocaine-and amphetamine-regulated transcript (CART)-expressing neurons have opposite roles in regulating energy intake.