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Using Physiologic, Genetic, and Epigenetic Information to Provide Care to Clients Who Are Obese

ABSTRACT

The pathology of obesity is a complex process involving interactions among behavioral, environmental, immunologic, genetic, and epigenetic factors. This article gives a broad overview of obesity. The physiology of fat storage, influence of eating behaviors on obesity, and the genetic relationship between eating and food sources are discussed. Specific genes that have been associated with obesity are introduced, with information on leptin and genes such as *FTO*, *GLUT4*, and others. This synopsis of obesity expands into environmental influences and epigenetic factors. These include food selection, gut microbiota, pregnancy, and exercise. The nurse will gain specific knowledge to assist in tailoring therapies specific to clients who are working to overcome the long-term effects of this disorder

In 2015, President Obama announced the Precision Medicine Initiative, focusing on prevention and treatment strategies that take individual variability into account. Individualizing care employs the use of genetics and biological databases to identify methods for characterizing patients and to encourage healthcare providers to build evidence for individualized clinical practice (Collins & Varmus, 2015). The goal of this initiative is to precisely tailor therapies that specifically meet the genetics and physiology of a patient (Ashley, 2015). One area of health that is in need of this implementation is the prevention and treatment of obesity and obesity-related disorders. Scientific understanding of obesity has great potential for improving the health of individuals, communities, and the global population.

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Obesity

The pathology of obesity is complex and involves multiple interactions among behavioral, environmental, immunologic, genetic, and epigenetic factors. The dramatic rise in the rates of obesity and associated pathologies such as insulin resistance, metabolic syndrome, and diabetes mellitus type 2 (DM2) has been well documented (Hoelscher, Kirk, Ritchie, Cunningham-Sabo, & Committee, 2013).

Metabolic syndrome is characterized by conditions including elevated blood glucose, hypertension, excess body fat, and abnormally high lipid levels. DM2 is characterized by high blood glucose levels as a result of reduced systemic sensitivity to insulin. These conditions are highly associated with other comorbidities such as cardiovascular disease, nonalcoholic liver disease, and cancer (Lallukka & Yki-Järvinen, 2016).

Each of these interrelated comorbid conditions has been associated with both genetic and environmental factors that contribute to a chronic low-grade systemic inflammatory response. This inflammatory response is thought to originate predominantly in adipose tissue (Wensveen, Valentić, Šestan, Turk Wensveen, & Polić, 2015).

Pathophysiology of Adipose Tissue Formation

Adipose tissue functions as a major energy storage depot and has a critical role in metabolic and

thermoregulatory processes. The human body is composed of predominantly two types of adipose tissue, white and brown fat. White fat stores energy in the form of triglycerides and is the major source of chronic systemic inflammation (Wensveen et al., 2015). White adipose tissue can be classified into two major areas: subcutaneous and visceral. Subcutaneous fat lies beneath the skin. Visceral fat connects inner organs and accumulates within the abdominal cavity and mediastinum (Petrovic et al., 2015).

Brown fat is found primarily in infants and in small amounts in adults. It has the ability to dissipate energy through thermogenesis. Recently, a third type of fat has been found to play a major role in obesity. It is often referred to as "beige" or "brite" fat. These cells, interspersed in the white fat, can be "cold induced" or "exercise induced" to generate heat and reduce the chronic inflammatory response that leads to comorbidities (Petrovic et al., 2015; Rao et al., 2014).

The activities of different fat cell types adapt to conditions of metabolic stress. During periods of overnutrition, brite or beige fat decreases thermogenic activity, becomes resistant to insulin, and activates "obesity-generated inflammation." Key regulators of this immune response are innate lymphoid cells, which recently have been linked to the regulation of host metabolism. The key cells involved in this response are Treg cells (T-regulatory cells).

In recent studies on rats, the cytokine (interleukin-33) produced by the Treg cells was found to be reduced when the rats were fed a high-fat diet. However, when cytokine was given to the rats, the obese condition was reversed even when the rats were fed a high-sucrose/high-fat diet. This also led to reduced inflammation and reversal of insulin resistance (Han et al., 2015). Although interleukin-33 has been shown to reduce adiposity, reduce fasting glucose, and improve glucose and insulin tolerance in mice, further studies are needed to understand the role of the cytokine in humans (Miller et al., 2010).

Physiology of Eating and Metabolizing Food

Food metabolism and food consumption also play an essential role in the development of obesity and its comorbidities. Ghrelin, a hormone produced in the stomach, is pivotal in whole-body energy metabolism. Levels of ghrelin peak immediately before routine mealtimes. Once food is eaten, glucagon-like peptide-1 gastric hormone is released into the bloodstream and produces a glucose-stimulated insulin secretion from the pancreas. Because ghrelin enhances insulin response for the disposition of ingested nutrients, it also has been linked to promoting synthesis of fatty acids and triglycerides in the liver (Gagnon, Baggio, Drucker, & Brubaker, 2015).

Eating behavior is a complex process that involves physiologic, psychological, social, and genetic factors. Taste and taste sensitivity are under genetic control. Genetically-derived perceptions of taste have a significant impact on food choices, amount of food consumed, and our physiologic response to individual food items. Individuals who are sensitive to bitter taste demonstrate avoidance of specific foods including vegetables.

Some individuals have a high genetic preference for sweet and high-fat food. These genetic variations are linked to height variations among children with the same genetic predisposition for height (Grimm & Steinle, 2011). Gene polymorphisms for the bitter taste receptor T2R38 have been shown to influence taste for brassica vegetables, such as broccoli, cauliflower, or kale. Individuals with a specific polymorphism may avoid vegetable consumption and compensate by increasing their consumption of sweet and fatty foods (Chamoun et al., 2018).

Unami is a savory taste that is found in tomatoes, soy sauce, and is commonly added to foods as monosodium glutamate (MSG). It is perceived by the glutamate receptors. Individuals with specific variations of this gene exhibit a strong preference for unami. The preference for these foods is so strong that individuals will tend to overconsume foods with this flavoring (Grimm & Steinle, 2011).

There is a statistical relationship between the cluster of differentiation 36 (*CD36*) gene and a taste preference for high-fat foods. One allele (variant form of the gene) more common in African Americans leads to craving of high fat in the diet. The results of studies suggest that African Americans with the specific variant allele may be genetically predisposed to crave fat-rich foods. In this study, individuals with the A/A genotype preferred creaminess regardless of fat content in salad dressings, whereas those with C/T or T/T were more influenced by fat content (Keller et al., 2012).

Genetic Predisposition to Obesity

The association of obesity with high caloric intake and sedentary lifestyles only partially explains the development of obesity. There is strong evidence that genetics plays an important role in the risk for becoming obese. Studies on twins have placed the heritability of obesity in a range from 41% to 85%, with differences based upon culture and environment (Feng, 2016). Subsequently, several single-gene studies have led to discoveries in the obesity pathways. These single-gene mutations are most often associated with severe and early-onset obesity.

Leptin

In the 1950s, mice homozygous for the leptin receptor gene (*ob*: *ob*/*ob*) developed excess adipose tissue. These mice typically weighed more than three times the weight of normal mice. During the 1990s, a mutation in the leptin gene was discovered in severely obese mice. Mice who were given leptin, a peptide-regulating appetite and metabolism, lost weight (Kebede & Attie, 2014).

It has subsequently been discovered that humans who cannot produce leptin can benefit from being given leptin, but those who produce some leptin do not lose weight when given leptin. The exact mechanisms for leptin resistance are unclear at this time, but multiple factors including inflammation and stress responses are involved with leptin resistance. Because leptin stimulates secretion of anti- and pro-inflammatory cytokines, there is the suggestion that leptin is involved in regulation of energy metabolism and may activate obesity-induced inflammation (Sáinz, Barrenetxe, Moreno-Aliaga, & Martínez, 2015).

Apolipoprotein E

The human apolipoprotein E (*APOE*) gene has three major alleles (i.e., 2, 3, and 4). *APOE4* is the ancestral form and evolved more than 80,000 years ago. The *APOE* gene functions in lipid metabolism and deposition of adipose tissue and is associated with regulation of food intake. The *APOE3* isoform of the gene is the most common in human populations. In a recent study, *APOE3* was associated with more efficient use of dietary energy, increased fat storage, and higher body mass index (BMI) and body weight in children.

APOE4 carriers are associated with increased fatty acid mobilization and utilization as a fuel. Function of the 4 allele declines with age, which has been associated with age-related disease risks for cardiovascular and endocrine systems. This was not a problem until approximately 200 years ago because most individuals died from infectious disease instead of chronic disease (Huebbe et al., 2015).

Although more commonly associated with reduced risk for Alzheimer's disease, the *APOE2* allele has increased risk for obesity. Mice with the *APOE2* allele have increased adiposity when consuming a high-fat, high-cholesterol diet. They also had higher levels of inflammation that are associated with metabolic disease (Kuhel et al., 2013).

Brain-Derived Neurotropic Factor

Early-onset severe obesity is related to a number of rare genetic defects. These include rare leptin deficiencies, adrenocorticotropic hormone insufficiency, and other rare disorders. Brain-derived neurotropic factor is associated with Prader–Willi syndrome. With Prader–Willi syndrome, individuals have a significant reduction in the number of oxytocin-producing neurons. This leads to hyperphagia, obesity, and behavioral anomalies (Huvenne, Dubern, Clément, & Poitou, 2016).

Pro-opiomelanocortin

The normal protein produced by the pro-opiomelanocortin (*POMC*) gene is produced in the hypothalamus, plays a role in feeding behavior, and is positively regulated by leptin. A rare recessive single gene defect in the *POMC* gene leads to pro-opiomelanocortin deficiency, which is associated with early-onset obesity, adrenal insufficiency, and red hair pigmentation. Affected infants are normal weight at birth, but are constantly hungry, which leads to hyperphagia. This is accompanied by adrenal insufficiency, with severely low blood sugars, an inability to produce bile, and excess bilirubin levels (Aldemir, Ozen, Sanlialp, & Ceylaner, 2013).

Olfactomedin 4 and Homeobox B5

Early-onset severe obesity in both children and adults has been linked to the regulation of two genes in an area near the olfactomedin 4 (*OLFM4*) gene and within the homeobox (*HOXB5*) gene. The *OLFM4* gene is potentially linked to gut flora and there are studies that show the gene product downregulates innate immunity to the gut microbiome and infection to gut microflora. However, the DNA area that demonstrated the link to severe, early obesity in children was located near but outside of this gene, and the relationship is not known at this time (Early Growth Genetics Consortium, 2012). The *HOXB5* gene is associated with lung and gut development. Although it is associated with early-onset severe obesity in children, the precise role it plays in the development of the disorder is not fully understood (Xia & Grant, 2013).

Fat Mass and Obesity-Associated

Single gene defects that lead to severe obesity are usually rare mutations and not associated with more common forms of obesity. The development of Genome Wide Association Studies (GWAS) allows researchers to identify more prevalent forms of obesity. Of the single gene disorders, the *FTO* (fat mass and obesity-associated) gene is frequently found in the global population and has shown the greatest promise in identifying mechanisms associated with the risk of obesity. One single nucleotide polymorphism (SNP) shows the strongest association in Caucasians. Another, more recently discovered variant, has a high association with European and African ancestry individuals. *FTO* gene expression is highest in the brain in hypothalamic nuclei where energy balance and feeding patterns are regulated (Xia & Grant, 2013).

Although the exact role the *FTO* gene has in obesity is not completely understood, there are differences in postprandial appetites for the three phenotypes. The two alleles, A & T respond differently to food and satiety. The homozygous AA persons are "obesity-risk" as opposed to the TT low-risk obesity phenotype. In preschool children, the AA group tends to demonstrate obesity-prone eating behaviors such as increased food responsiveness and a tendency to eat in response to external clues (Karra et al., 2013). Another study indicated that being a carrier of the A allele (AT or AA) was associated with greater energy intake at meals. Although overall weight of the food was not increased, fat intake was 30% higher in those with the A allele (Cecil, Tavendale, Watt, Hetherington, & Palmer, 2008). More recently, it was found that if the overall fat content of the diet of adolescents was below 30%, the A allele of the *FTO* gene was not associated with increased body fat (Labayen et al., 2016).

Glucose Transporter 4

Glucose transporter type 4 (GLUT4) is an insulin-regulated glucose transporter protein that is primarily found in adipose tissues and muscle (cardiac and skeletal). This protein lowers plasma glucose, facilitates glucose uptake, and has an anti-inflammatory effect on the body. During fat accumulation or even high intake of lipids, the action of GLUT4 is inhibited. Individuals who are heterozygous for the *GLUT4* gene (GLUT4+/–) have reduced GLUT4 expression in adipose tissue and skeletal muscle. They also have some insulin resistance and are predisposed to diabetes. High levels of GLUT4 protein in these tissues increase insulin sensitivity and glucose tolerance (Richter & Hargreaves, 2013). Both diet and exercise can enhance the expression of GLUT4. It was found that quercetin, a flavonoid found in onions and capers, increased expression of GLUT4 (Mehta et al., 2017). Exercise training also has an epigenetic effect of acetylation of the GLUT4 promoter and increased transcription of GLUT4 (Richter & Hargreaves, 2013).

Genome-Wide Association Techniques

Several other gene patterns have been found using GWAS techniques. In a meta-analysis on nearly 250,000 individuals, a total of 32 BMI-associated loci were found (Speliotes et al., 2010). These studies have led to the discovery of multiple SNPs in the *FTO* gene. Several of these are associated with obesity, have been found to confuse the functions of *FTO*, and obscure the functions of the gene, making it difficult to draw conclusions (Hess & Brüning, 2014).

In a more recent genome-wide analysis of 339,224 individuals, 97 loci were associated with obesity. These genes were associated with synaptic function within the hypothalamus, glutamate signaling, insulin secretion/action, energy metabolism, lipid biology, and adipogenesis. This provides support that the central nervous system is involved in obesity susceptibility (Locke et al., 2015).

Synthetic Chemicals and Obesity

One common misinterpretation is that calories in equals calories out. Although this is primarily true and many adults consume significantly more calories than are needed to maintain health, many factors play a role in reducing or preventing obesity. Although decreased caloric intake can lead to short-term weight loss, this may not meet the needs of clients either behaviorally or biologically (Mozaffarian, 2016).

Sugar and Artificial Sweeteners

It has been widely documented that added sugars increase the risk for obesity. In a study on 32 genetic variants associated with higher BMI, genetic association with adiposity increased with higher intakes of sugar-sweetened beverages. This was also true for individuals on high-fat diets and those who have increased hours of watching television. The genetic association with BMI was weakened with increased levels of physical activity. This gives rise to the concept that the environmental factors associated with obesity not only have a physiologic interaction with weight but also have a long-term influence that changes the ability of the body to reverse the development of obesity (epigenetic effects) (Huang & Hu, 2015).

The global market for artificial sweeteners now exceeds $1 billion per year. Over the past few years, the use of these sweeteners has more than doubled in the United States in an effort to reduce caloric content without sacrificing sweetness in the diet (Schiffman, 2012). Artificial sweeteners have yielded mixed results in regard to their relationship to obesity. For example, aspartame, in combination with MSG, promoted fat accumulation and increased prediabetic symptoms. Saccharin binds directly to receptor cells on the beta cells of the pancreas, stimulating insulin release. This may have the effect of lowering blood sugar and stimulating increased appetite. Conversely, stevia has been found to treat insulin resistance in mice (Simmons, Schlezinger, & Corkey, 2014).

Food Additives

Food additives have been found to be a source for epigenetic influences on how foods are processed in the body and how the body responds to the foods that are eaten. Food additives are so predominant that it is

difficult to eat a "modern diet" and maintain a healthy weight. Over 4,000 nonfood ingredients have been added to the food supply. Some of these are intentional to enhance palatability or promote safety, and some are unintentional such as pesticides or other persistent organic pollutants. Many of these chemicals are endocrine disrupting and can accumulate in the tissues of top consumers like humans. The use of artificial colors increased fivefold from 1950 to 2010.

Although identification of which chemicals are leading to obesity may seem overwhelming, studies on zebrafish may hold the key. Advantages to studying zebrafish include rapid reproductive rate and low cost in vivo studies on the whole organism (Simmons, Schlezinger, & Corkey, 2014). Organs of the zebrafish are similar to humans in structure and function. They also share common pathophysiologic pathways with obesity models in mammals. Studies on zebrafish are especially important in understanding regulation of lipid metabolism (Landgraf et al., 2017).

In animal studies and human epidemiologic research, MSG use has been associated with obesity and altered fat metabolism. Recent controlled intervention studies have indicated that MSG does not have an effect on overall body weight. Some foods naturally contain MSG such as ripe tomatoes, cheeses, and cooked meats. However, high doses of glutamate intake have led to altered body morphology, producing animals that are short and obese. In addition, MSG has a strong flavor-enhancing property that affects palatability of food, thus increasing intake. The average intake from natural sources of glutamate results in approximately 130–180 mg/kg/day. Daily intake of added glutamate adds an additional 5–10 mg/kg/day. In some population studies, persons who add high levels of glutamate to their diet significantly increased their risk for obesity when adjusting for age, diet, physical activity, and other lifestyle factors (Brosnan, Drewnowski, & Friedman, 2014).

Although organic or natural foods have lower pesticide and cadmium concentrations and higher omega-3 fatty acids in the meat and dairy, the information on the benefits of organic foods as they relate to obesity is limited and based upon small studies. There are almost no data from long-term cohort studies that focus on chronic diseases associated with obesity such as cardiovascular, cancer, or diabetes. Controlled human studies comparing organic and conventional diets are nearly nonexistent (Baran´ski, Rempelos, Iversen, & Leifert, 2017).

The higher levels of omega-3 fatty acids found in some organic foods as well as the higher levels of polyphenols, found in red and blue berries, are believed to work as antioxidants, which can help protect the body from the inflammatory processes associated with obesity. Limited studies on polyphenols in diets indicate a pronounced difference in body weight, fat mass, and triglyceride levels (Wang et al., 2014).

Toxins

As noted earlier, an individual who has the *APOE3* allele is at increased risk for obesity. But from an epigenetic perspective, the use of organophosphates, commonly used in agriculture, can alter the impact of the allele. Chlorpyrifos is widely used in agriculture as an insecticide. In mice, chlorpyrifos exposure is linked to increased food ingestion, increased blood glucose, and higher insulin resistance for those with the *APOE3* allele. Although individuals who have the same allele have an increased risk, the environmental exposure increases the risk for health disturbances (Peris-Sampedro et al., 2015).

Epigenetics and Obesity

Whereas genetic modifications are related to a change or changes in the DNA sequence, epigenetic changes are chemical modifications in the DNA-associated processes. These changes occur through adding or removing methyl groups to the DNA. This methylation process occurs predominantly at sites in the DNA sequence where cytosine is adjacent to guanines (CpG sites). Adding enough methyl groups to the DNA sequence in a specific area has the effect of turning the gene off in that area of the DNA (Van Dijk et al., 2015). The methylation processes in the body involve over 45 genes related to obesity. Physiologic responses to food and food intake alter the methylation patterns of these genes.

MicroRNA (miRNA) molecules are small noncoding RNAs that silence or regulate gene expression. To date, 10 of these miRNA molecules have been found that are associated with obesity or regulation of genes associated with obesity. These molecules are highly conserved in both plants and animals and are believed to be vital as biological regulators (Burgio, Lopomo, & Migliore, 2015). Many factors including gender, ethnic background, age, smoking, and exposure to chemicals influence the association between the methylation process and obesity. However, most studies indicate that reducing risk factors such as smoking and increasing exercise have a positive effect on the epigenetic processes involved with obesity (Van Dijk et al., 2015).

Pregnancy

The prenatal period is recognized as critical in establishing the epigenome of the individual. Several studies have indicated the relationship of prenatal and early postnatal periods in increasing the risk for obesity later in life (Van Dijk et al., 2015). Preliminary studies indicate that maternal obesity alters the DNA methylation processes, which have a direct impact on the health of the infant (Godfrey et al., 2017). Epidemiologic studies have

shown that maternal intake, either a deficient or an excess of calories and micronutrients, is associated with an increased risk of chronic disease including obesity.

In a study by Guénard et al. (2013), siblings born before and after maternal weight loss surgeries had methylation profiles compared. They reported differences in obesity characteristics and actions of genes involved in glucose homeostasis and immune function regulation between the siblings. This suggested that significant maternal weight loss, with improved metabolic health profiles, is associated with a distinct epigenome, which demonstrated lower weight and waist profiles in children. Ideally, maternal weight loss would be established prior to pregnancy to make obesity and chronic health issues associated with obesity later in life more scarce.

Gut Microbiota

Information about the relationship of gut microbiota and obesity has been widely disseminated. Higher intakes of dietary fiber have been linked to lower body weights. Oligosaccharides, inulin, and oligofructose are natural constituents of many plants including wheat, barley, onions, legumes, and asparagus. These compounds are known as "prebiotics" and resist digestion. Once they reach the small intestine and colon, they are fermented by the gut microflora. There is evidence that the fermentation has the potential to slow the glucose absorption rate, prevent weight gain, and increase beneficial nutrients and antioxidants. Additional studies indicate obesity is associated with changes in the microbiota, reduced bacterial diversity, and alterations of the genes associated with the metabolic pathways (Slavin, 2013).

The microflora have recently been identified as having a role in epigenetic changes in the body. High-fat diets promote the growth of species within the phyla *Firmicutes* and *Bacteroidetes*, which are associated with obesity. These species have been found to affect the acetylation and methylation patterns of DNA, specifically for genes involved in cardiovascular diseases, lipid metabolism, obesity, and inflammatory response, increasing the risk for health problems. Pathogenic bacteria such as *Helicobacter pylori* and *Klebsiella* were associated with differential modification of over 200 regions of the DNA (Ye, Wu, Li, & Li, 2017). Conversely, the species associated with probiotics and consumption of low-fat, high-fiber diets enhance the action of metabolites such as folate and riboflavin, which exert beneficial effects on epigenetic regulation (Lee, Song, & Nam, 2017).

It was demonstrated in a study that depleting the gut microbiota in obese mice through antibiotic therapy improved sensitivity to insulin, increased glucose tolerance, decreased white fat, and increased brown fat markers. The conversion to brown or beige fat may be beneficial in understanding how to help individuals reduce obesity and limit insulin resistance (Suárez-Zamorano et al., 2015).

Exercise

Human differences in responsiveness to regular exercise were recognized more than 30 years ago. Large differences have been found in exercise capacity, skeletal muscle oxidative potential, and adipose tissue lipid mobilization. Strongest associations for variability in exercise response were found in the kinesin family of genes. The gene kinesin family member 5B (*KIF5B*), when inhibited, diminishes the number of mitochondria within muscle cells, whereas overexpression enhances mitochondrial reproduction.

In addition, multiple genes are now being studied that relate to a variety of physiologic responses to exercise. These include genes regulating heart rate (cyclic AMP responsive element binding protein 1), genes associated with mitochondrial function (tumor protein 53), and several genes related to vascular wall compliance and tone. Although these studies are in their infancy, regular routine exercise is still found to be associated with decreased chronic disease and prevention of many health-related problems for both the young and the elderly (Bouchard et al., 2015).

Nursing Implications

The study of genetic and epigenetic influences on the body is still developing. Currently, individuals rarely have genetic testing to identify genes affecting their obesity and chronic disease status although this practice may be more prevalent in the near future. Lifestyle modifications continue to be the best interventions in the prevention of obesity. Ideally, those modifications are introduced before obesity occurs.

In addition to encouraging exercise and healthy diets, guidance can be provided about more specific dietary changes. An example of this includes encouraging the use of stevia as the artificial sweetener of choice. When food choices are discussed, including more fiber in the diet would be beneficial as would decreased use of food additives such as MSG and encouraging the addition of micronutrients through foods or supplements such as folic acid for childbearing women, vitamin D, and omega-3 fatty acids. These provide added support to establishing and maintaining healthy metabolic profiles. Increasing consumption of active-culture yogurts will help maintain balanced gut flora.

When providing care for women during their childbearing years, encouraging them to attain a healthy weight prior to pregnancy is a preventative measure for the fetus. If the individual is obese at conception, guiding her to maintain glucose homeostasis during pregnancy is important.

While counseling individuals who are obese, it is imperative to develop plans of care with long-term goals. This means weeks to months of work before real progress can be obtained. Epigenetic changes do not occur within a few days; thus, an individual must make a long-term commitment to changes in lifestyle.

Conclusion

The increasing rate of obesity and its associated comorbidities make an understanding of the influence of genetics and epigenetics on weight gain an important tool for nurses. Genetic factors linked to adiposity are expressed in a variety of ways, including perceptions of taste, which impacts eating behaviors, appetite, and genetic defects related to early-onset obesity. Other factors such as the use of artificial sweeteners and exposure to chemicals used in agriculture can lead to altered fat metabolism. Knowledge of the behavioral, environmental, genetic, and epigenetic effects on weight gain is needed for the prevention and treatment of obesity. Counseling clients who struggle with obesity or who are at risk for becoming overweight requires the development of personalized interventions that take into account the complex nature of obesity. ✪

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