DOI: 10.1097/JPN.0000000000000400

CE **Continuing Education**

J Perinat Neonat Nurs • Volume 33 Number 2, 126–135 •

Obesity and Socioeconomic Disparities

Rethinking Causes and Perinatal Care

Cecilia M. Jevitt, PhD, CNM, FACNM

ABSTRACT

Obesity affects more than 35% of women aged 20 to 39 years in the United States. This article summarizes recent research that reconceptualizes obesity as adipose disease associated with smoking; socio-economic disparities in employment, education, healthcare access, food quality, and availability; and environmental toxins, ultimately altering microbiomes and epigenetics. Individual prenatal care of women with obesity includes early testing for diabetes, counseling on epigenetic diets, advice supporting weight gain within national guidelines, and vigilance for signs of hypertensive disorders of pregnancy. Intrapartum care includes mechanical cervical ripening measures, patience with prolonged labor, and uterotonic medication readiness in the event of postpartum hemorrhage. Postpartum care includes thrombus risk amelioration through early ambulation, use of compression stockings, and anticoagulation. Delays in lactogenesis II can be offset by measures to support early breastfeeding. Sociopolitical action by nurses at national, state, and community levels to reduce population disparities in racism, education, and employment; reduce pollution from obesogenic chemicals; and improve food quality and distribution policies is likely to have the broadest impact in future obesity reductions and prevention.

Key Words: epigenetic diets, microbiome, obesity, perinatal weight gain, socio-economic disparities

Author Affiliation: The University of British Columbia, Faculty of Medicine, Midwifery Program, Vancouver, British Columbia, Canada.

Disclosure: The author has disclosed that she has no significant relationships with, or financial interest in, any commercial companies pertaining to this article.

Each author has indicated that he or she has met the journal's requirements for Authorship.

Corresponding Author: Cecilia M. Jevitt, PhD, CNM, FACNM, The University of British Columbia, Faculty of Medicine, Midwifery Program, Apt 505, 5988 Gray Ave, Vancouver, British Columbia, V6S 0K4, Canada (cecilia.jevitt@ubc.ca).

Submitted for publication: October 15, 2018; accepted for publication: January 28, 2019.

Historically, social deprivation and poverty have been associated with hunger and starvation. During the last 2 decades in the postindustrialized world, poverty has become more associated with obesity than starvation.[1] When obesity was initially recognized as increasing in 1990, no state in the United States (US) had an obesity prevalence of 15% or greater.[2] In 2016, the prevalence of obesity in women aged 20 to 39 years in the United States was 36.5%.[3] Worldwide, obesity has almost tripled since 1975, with obesity-related disease killing more people than undernutrition.[1]

Obesity affects some racial/ethnic groups more than others. Hispanic and non-Hispanic black women have the highest age-adjusted prevalence of obesity (50.6% and 54.8%), followed by non-Hispanic white women (38.0%) and non-Hispanic Asian women (14.8%).[3] Obesity is increasingly recognized as an adipose organ disease with health compromising effects across the life span including the perinatal period with risks to both mother and newborn (see Table 1).[3–5]

When conditions affect more than half a population, causation needs to be sought beyond personal choices by considering systemic social and environmental contributions to health. This review examines new concepts in obesity and associations between socioeconomic disparities in education, income, health, and obesity, concluding with individual and population-targeted nursing actions to ameliorate obesity-related perinatal morbidities.

OBESITY AS AN ORGAN DISEASE

Obesity originally was attributed to excess caloric intake without sufficient physical activity for energy use with white adipose tissue serving as the storage depot for excess energy.[3] Responsibility for excess weight was viewed as a personal lack of discipline in eating and physical activity. Newer research in metabolism,

Table 1. **Obesity-related reproductive morbidity and mortality**[a]

Preconception
Irregular menstruation
Polycystic ovary syndrome
Infertility
Pregnancy
Spontaneous abortion
Neural tube defects
Cardiac defects
Urinary tract infection
Excessive weight gain
Gestational diabetes
Hypertensive disorders of pregnancy
Placental abruption
Intrauterine growth restriction
Macrosomia
Prolonged pregnancy
Stillbirth
Intrapartum
Preeclampsia and eclampsia
Induction of labor
Dysfunctional labor
Prolonged stage 1 labor
Pitocin augmentation of labor
Epidural placement failure
Instrumental birth
Shoulder dystocia
Cesarean delivery
Increased use of general anesthesia
Low Apgar scores
Postpartum
Immediate postpartum hemorrhage
Endometritis
Urinary tract infection
Wound dehiscence
Wound infection
Deep vein thrombosis
Delayed lactogenesis II
Breastfeeding latch problems
Excessive weight retention

[a]From American College of Obstetricians and Gynecologists[4] and Anstey and Jevitt.[5]

psychoneuroimmunology, and adipose has moved the definition of obesity from a set of character faults to the definition promoted by the Obesity Society: "a chronic, relapsing, multifactorial, neurobehavioral disease, wherein an increase in body fat promotes adipose tissue dysfunction and abnormal fat mass physical forces, resulting in adverse metabolic, biomechanical, and psychosocial health consequences."[6] This newer definition allows for the inclusion of multiple potential contributors to obesity including social disparities in economics, education, nutrition, and the environment.

Although obesity is defined as a body mass index (BMI) of 30 or greater (BMI = weight [kg]/[height (m)]^2), there is debate about BMI as an appropriate measure of adiposity because for any BMI unit, the composition of adipose and muscle varies between people of the same sex across race and ethnic groups.[1,2,7] Waist circumference is a better predictor of future obesity-related morbidity[7]; however, waist circumference changes dramatically in pregnancy. In addition, the definition of obesity using waist circumference varies by race with a waist circumference of 35 inches or greater in Caucasian females but 31 inches or greater in Asian females indicating obesity.[3,7] Therefore, BMI at the first prenatal visit is the measurement used to define obesity in perinatal research.[8]

White adipose tissue

White and brown adipose tissues are endocrine organs with multiple metabolic functions. Brown adipose, found mainly in newborns, produces heat when metabolized, a protective factor during cold exposure. Like the dermis, white adipose tissue is widely distributed throughout the body and might be the largest hormone-producing tissue secreting endocrine, paracrine, and autocrine signals.[9] White adipose is rich in macrophages that are the source of tumor necrosis factor α and interleukin-6. Production of proinflammatory adipocytokines is a major contributor to the chronic inflammatory state found in obesity and likely contributes to obesity-related diabetes, atherosclerosis, hypertension, and perinatal morbidities and mortality.[9,10] Table 2 outlines factors secreted by white adipose tissue, functions, and the main sites of production.

Epigenetics of obesity

Barker developed the fetal origins hypothesis in the early 1990s, using evidence from birth records linked to adult health records to demonstrate that adult diseases including diabetes, hypertension, heart disease, and stroke are the sequelae of poor maternal health and in utero undernourishment.[11] Fetal programming in utero has been hypothesized to start with maternal undernutrition and calorie restriction that reduce placental growth and function causing decreased glucose oxidation with increased amino acid and lactate oxidation.[12] Endocrine changes include increased cortisol production with decreased production of insulin, insulin-like growth factor-1, and growth hormone.[12] This undernutrition and decreased fetal growth programs the child for insulin resistance, metabolic syndrome, hypertension, type 2 diabetes, and heart disease. Counterintuitively, low birth weight newborns catch up on growth if exposed to excess calories and the programmed metabolic dysregulation pushes the child toward obesity.[11,12]

Research since Barker has shown that genes can be dysregulated by methylation producing novel

Table 2. **White adipose tissue secretions and functions**[a]

Molecule	Main site of production	Function
Leptin	Subcutaneous adipose tissue. Also produced by the placenta as a growth hormone	Increases appetite based on signals from adipose stores, decreases energy expenditure
Adiponectin	Secreted only from adipose tissue, mainly subcutaneous	Improves insulin sensitivity, stimulates fatty acid oxidation and glucose uptake in skeletal muscle, stimulates appetite, and reduces energy expenditure
Factor D (adipsin)	Adipose tissue	May be involved in the complement pathway and adipose metabolism
Resistin	Produced by many types of cells in addition to adipocytes, increased during infection	Function unknown, may be involved in insulin resistance
Angiotensin		Precursor of angiotensin II (blood pressure and electrolyte regulation, possible role in hemostasis)
Plasminogen activator inhibitor (PAI-1)	Vascular cells and monocytes within adipose, adipocytes	Changes the balance between fibrinogenesis and fibrinolysis, contributes to atherosclerosis
Acylation-stimulating protein	Subcutaneous adipose tissue	Increases lipogenesis, may contribute to dyslipidemia in diabetes and cardiovascular disease
Tumor necrosis factor (TNF-α)	Macrophages within adipose tissue, mainly visceral adipose tissue	Proinflammatory cytokine, insulin resistance, potential contributor to type 2 diabetes
Interleukin-6 (IL-6)	More produced in visceral adipose tissue, adipose produces 30% of circulating IL-6	Proinflammatory cytokine, insulin resistance, potential contributor to type 2 diabetes
Visfatin	Visceral adipose tissue	Proinflammatory cytokine, activates leukocytes, and stimulates production of TNF-α and IL-6

[a]From Coelho et al[9] and Cinti.[10]

phenotypes, suggesting that programming may occur at the cellular level.[13,14] Fetal programming in utero is now conceptualized as epigenetic changes, changes in gene expression that are independent of changes in deoxyribonucleic acid. The preconception use of folic acid supplementation to prevent neural tube defects is an example of a bioactive diet that provides beneficial epigenetic changes. Women with obesity have higher circulating levels of the adipose-secreted molecules in ovarian follicular fluid that may produce epigenetic changes in ova. The same processes may affect sperm and seminal fluid, producing the reduced fertility seen in men with obesity.[14]

Epigenetic changes may occur in ova, sperm, or the zygote, and then be replicated through cell division. Ova, sperm, and the developing blastocyst are adaptive and primed for success in the immediate prepregnancy, undernourished environment.[15] If the child enters a new environment rich in calories, the adaptive gene becomes maladaptive and may prime the child for obesity. Phenotypic changes related to deoxyribonucleic acid methylation have been shown to persist across generations and might be a partial explanation for the dramatic rise in obesity during the last 3 decades.[15]

Maternal smoking and obesity

Exposure to maternal smoking in utero has been associated with at least a 30% increase in risk for adult obesity.[16,17] Cigarette smoking has declined substantially in the United States; however, the prevalence of smoking remains higher in lower-income groups.[18] A study in which 26.1% of nurses reported smoking during pregnancy found a significant association between maternal smoking during pregnancy and overweight and obesity in adulthood with the risk of overweight and obesity increasing with the number of cigarettes smoked daily.[19] Children of women who stopped smoking at the start of pregnancy did not have an increased risk for adult overweight and obesity. Tobacco-exposed, low birth weight newborns may exhibit the growth catch-up phenomena described by Barker.[11]

Obesity and the microbiome

The newborn is colonized by the maternal vaginal microbiome during birth. Emerging evidence reveals that the gut microbiome has nutrient and satiety sensing and signaling properties, along with roles in energy

harvesting and inflammatory response modulation.[20–24] A cascade of gut reactions begins with a high-carbohydrate, high-fat diet that favors changes in the gut microbiome and initiates an inflammatory environment that allows for increased permeability between intestinal cells.[20,21] Adipose tissue cytokines increase this chronic, low-level inflammation changing gut-to-brain signaling that stimulates hyperphagia and increases obesity.[20,21] Human and animal studies have shown that those with obesity have gut microbiomes that are both different in bacterial composition and diversity compared with the microbiomes of those with normal weights.[22,23] Maternal prenatal nutrition altering the gut microbiome may cause histone modification or deoxyribonucleic acid methylation epigenetic changes or alter the fetal gut microbiome provoking obesity-stimulating epigenetic changes.[25]

CONTRIBUTION OF SOCIAL DISPARITIES TO OBESITY

Although individual dietary and activity choices are under personal agency, those choices are limited by social and environmental constraints. Disparities in available education, employment, income, healthcare access, the lived and built environment, and available nutrition shape individual choices and epigenetic inheritance (see Figure 1). Understanding the contributions of social disparities to obesity broadens opportunities for health-improving interventions, moving these from the individual to community level.

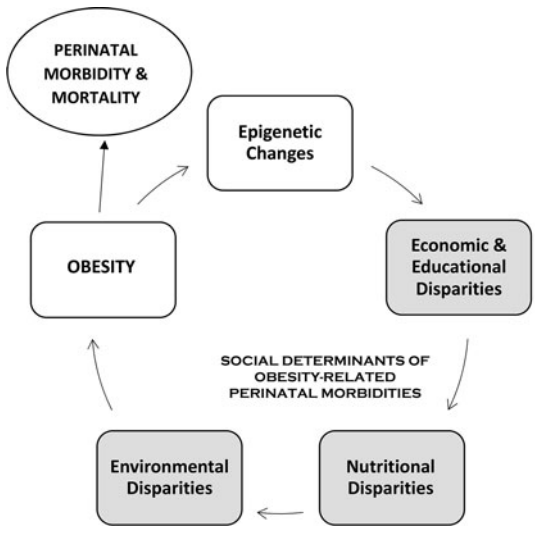

Figure 1. Social determinants of obesity-related perinatal morbidity and mortality.

Socioeconomic disparities

Racism

Race is a controversial sociopolitical construct that categorizes people by phenotype. Race as a proxy for genetic variation is investigated for its links to disease risk. Ethnicity is a broader category that combines racial phenotypes with cultural variables including shared history, language, religion, and dietary customs. Racism, the discrimination and unequal treatment of individuals of racial and ethnic groups in the United States in almost all aspects of daily life, is a chronic life stressor that can have health consequences.[26] The stress of racism erodes health through wear and tear according to the life-course perspective.[26] Other hypotheses implicate chronic activation of the hypothalamic-pituitary-adrenal axis by stress as a cause of disease. Regardless of explanatory model, the stress of racism may cause epigenetic changes.[26] The socioeconomic inequalities imposed by racism systematically expose groups to other obesity-linked inequalities, including lower education; reduced access to healthcare; lower health literacy; stressful, low-wage work; unsafe neighborhoods; environmental pollutants; and food deserts.

Using interview data from a probability sample of 1870 including non-Hispanic white, non-Hispanic black, and Hispanic women, investigators linked the concepts of racism and vigilance (chronic attention to potential prejudice, discrimination, and racism) with increased BMI and waist circumference.[27] Black women had higher vigilance scores than Hispanic or white women (30%, 16%, and 9%, respectively). Waist circumference patterns and BMI mirrored vigilance findings with black women and Hispanic women having wider mean waist circumferences than white women (9.0 cm and 6.4 cm larger). The authors proposed that consumption of obesogenic foods and the subsequent release of mood-elevating neurohormones may be used as a stress-coping mechanism that accounts for elevated BMI and larger waist circumferences.[27,28] Alternatively, the stress of chronic vigilance may activate the hypothalamic-pituitary-adrenal axis increasing adipose deposition.

Police maltreatment, a frequent cause of vigilance, was examined in another study comparing personal or vicarious unfair treatment by police to waist circumference in a sample of black and white women.[29] Individuals experiencing vicarious unfair treatment by police had waist circumferences that were approximately 2 inches larger than those who did not, leading authors to conclude that discriminatory policing is a potential contributor to racial disparities in health.[29]

Education

Disparities exist in access to education and the quality of public education in the United States, with obesity rates increasing as years of completed education decrease across racial and ethnic groups.[30,31] An analysis of the relationship of obesity and education using 2011-2014 National Health and Nutrition Examination Survey data revealed a 39% rate in the lowest income group but a 31% obesity rate in the highest income group.[31] Educational disparities may decrease health literacy reducing attention and comprehension to weight management education. Researchers analyzed 2007-2014 National Health and Nutrition Examination Survey data for usage of calorie labeling in restaurant menus finding that usage in all groups increased with increasing education and income.[32] Limited or poor-quality education is linked with reduced employment opportunity and lower income.[30–32] The higher obesity rates of women may be associated with multiple disparities in education, employment, and income.

Employment

Low-wage employment opportunities are more often filled by individuals from racial and ethnic minorities. A study of US transit workers found that African American and Hispanic females had higher obesity rates than men or other female operators.[33] Work stresses, including 10- to 15-hour shifts and the constant vigilance required during driving, in addition to the intersecting social identities of lower social class and minority women, were thought to augment hypothalamic dysfunction contributing to the higher obesity rates of minority women.[33]

Shift work requiring changes in sleep/wake cycles alters the circadian rhythms of key metabolic hormones such as leptin and insulin and has been associated with obesity.[34] A systematic review of 16 cross-sectional studies investigating short sleep, obesity, and metabolic changes found consistent associations between short sleep and higher consumption of poor nutritional quality foods with high fat content and fewer fruits and vegetables.[34] Increased intake may result from increased waking hours for snacking and meals.[34]

Access to healthcare

Healthcare insurance, the key to healthcare access, is still tied to employment in the United States. Black and Asian individuals are more likely than Hispanic or white individuals to be unemployed longer than 27 weeks,[35] thus limiting access to healthcare, especially preventative healthcare education. A 2-decade increase in part-time work, stagnation in wages, and changes in types of employment has eroded employer-sponsored health coverage and access to healthcare.[36] Provisions in the Affordable Care Act were targeted at reducing the cost of providing health insurance for employers; however, recent legislative changes have limited the impact of the Affordable Care Act in increasing healthcare access.[36]

Nutritional disparities

Food quality and quantity

A major constraint on personal nutrition is the cost of food. Low-wage earners have decreased ability to purchase fruits, vegetables, and whole gain foods as those foods cost more per calorie than processed, calorie-dense foods and sugared beverages.[37,38] A multinational literature review of food prices and diet quality found that individuals with reduced incomes stretched food dollars by meeting energy needs at the lowest cost per calorie, processed foods regardless of the overall nutritional value. Processed foods were also viewed as easier to prepare and more palatable.[38] Living in a food desert (a residential neighborhood with few full-scale markets for affordable and nutritious foods and more convenience stores) has been linked to increased risk for obesity.[35] Convenience stores typically sell calorie-dense, more affordable, low-nutrition foods with long shelf lives, thus making an obesogenic diet more available.[37,38]

Another study of food purchases explored the concepts of food security (availability and access to sufficient, safe, and nutritious foods at all times) and food deserts related to income using the United States Department of Agriculture's 2012-2013 Food Acquisition and Purchase Survey.[39] The nationally representative sample found that having a supermarket less than 1 mile from home was not associated with food security but access to an automobile was. Most households used automobiles to travel to markets with the lowest cost foods, often bypassing closer, more nutritious but expensive food sources.

Infant feeding

Nutritional disparities may begin immediately after birth. Obesity has been linked to delayed lactogenesis II and latch difficulties, eroding maternal confidence in breastfeeding.[5,40] Infants of women with pregestational obesity are at risk for early formula supplementation or weaning with early introduction of juices and solids such as cereal.[41,42] The epigenetic potential of these feeding behaviors is unknown. Two studies have examined early infant feeding in Hispanic and African American families with low incomes, finding that rates of age-inappropriate feeding were high.[41,42] Early supplemental feeding may confuse infant gut and hormonal

signals for hunger and satiety, increasing the risk for childhood obesity. Research also found parenting attitudes about feeding that put children at risk for obesity including beliefs that parents knew when infants were hungry regardless of newborn cues.[41] Powerful barriers to following national infant feeding guidelines have also been identified including single parenting, low income, and depression.[42]

Environmental disparities

Unsafe neighborhoods

The built environment is considered a contributor to sedentary behavior and obesity. Other environmental factors such as neighborhood safety and pollutants may also contribute to obesity. One national study found that non-Hispanic black and Hispanic individuals reported neighborhood crime and animals as barriers to safe walking significantly more often than white individuals.[43] Women also consider the potential for sexual violence when perceiving safety in neighborhoods.[44] Those living in neighborhoods perceived as unsafe may spend more time indoors in sedentary activities such as television watching.

Environmental pollutants

Endocrine-disrupting chemical pollutants are pervasive in outdoor and indoor environments and can be ingested orally, inhaled, or absorbed through the skin. Endocrine-disrupting chemicals (EDCs) with known obesogenic potential include persistent organochlorines, bisphenol A (BPA), phthalates, polybrominated diphenylethers, parabens, and phytoestrogens. Obesogenic EDCs contained in plastics, can liners of canned foods, flame retardants, detergents, dental sealants, pesticides, and herbicides affect adipose control through interfering with steroid and thyroid hormone function, altering insulin sensitivity, increasing adipose cell number and size, and favoring adipose deposition.[45,46] Adipose stores of EDCs can be mobilized into circulation during weight loss and lactation.[46] The summative effect of endocrine disruption may be alterations in the hormonal control of satiety and hunger.[45]

Disparities in available housing concentrate individuals with low incomes and racial/ethnic minority groups in neighborhoods with higher levels of pollutants including EDCs.[46] Sources include industrial waste and illegal dumping worsened by governmental failure to enforce environmental regulations.[46] A study using National Health and Nutrition Examination Survey 2003-2006 data found that income was inversely related to BPA levels with low income more related to BPA levels than race or ethnicity.[47] Individuals with high food insecurity, particularly those using emergency food sources such as food pantries, where foods are packaged for long shelf lives in EDC-containing cans or plastic containers, had higher BPA levels than food secure participants.[47]

DISCUSSION

Although obesity places women at risk for numerous morbidities, most women with obese BMIs complete pregnancy and birth without complications.[4,40,48,49] Recognition of the multiple contributors to obesity, including numerous social disparities that limit individual nutrition and activity choices, is the first step to improved care for women with obesity. Table 3 summarizes perinatal care measures for women with obesity.

The guidelines from the American College of Obstetricians and Gynecologist urge women to normalize BMI before attempting pregnancy[4]; however, weight loss programs of any kind have limited effectiveness.[52] Black and Hispanic women have lower access to health insurance that would allow primary healthcare support for prenatal weight loss guidance.[36] Calorie restriction alone is more effective than programs including a diet, psychological counseling, and physical activity.[52] With many women perceiving neighborhoods to be unsafe for physical activity,[43,44] the most effective preconception therapies may be epigenetic (those that create a better environment for ova and sperm), reduced calorie diets with folic acid, vitamin B, and essential micronutrients supplementation that avoid sources of toxic EDCs such as those in plastic containers and cans.[53,54] Fresh fruits, vegetables, whole grain carbohydrates, and other low glycemic index foods are examples of foods for epigenetic diets. Epigenetic diets may optimize the follicular and intrauterine environments limiting epigenetic changes that favor childhood and later adult obesity.[53] Assisting women to plan for nutritious meals and appropriate weight gain is an essential component of prenatal care. Gestational weight gain limited to 11 to 20 lb for women with prepregnancy obesity is associated with improved perinatal outcomes.[4] Nurses must be politically active in supporting federal, state, and community food distribution policies. Examples of these include the Women's, Infants' and Children's Supplemental Nutrition Program, laws requiring nutrition information on menus, and zoning laws that favor supermarkets over convenience stores.

Care measures for obesity-related morbidities do not differ between women of normal or obese BMIs. Diabetes and hypertension management remain the same regardless of BMI; however, obesity-related perinatal morbidities that fall into 2 categories, those caused by

Table 3. **Perinatal care measures for women with obesity**[a]

All stages of care
Use person-centered language (women with obesity instead of obese women)
Use appropriately large equipment such as blood pressure cuffs, gowns, chairs, and bariatric beds
Use weight-based drug dose calculations in place of standard doses
Avoid sources of endocrine-disrupting chemicals
Preconception
Normalize weight as much as possible
Screen for obesity-related disease (diabetes, hypertension)
Supplement with folic acid 4 mg daily
Epigenetic diets: low glycemic index foods, adequate micronutrients
Pregnancy
Measure height and weight at first prenatal visit, calculate BMI
Date pregnancy accurately
Screen early for gestational diabetes at first prenatal visit
Offer education in nutritious meal planning
Assist women with smoking cessation
Have women self-weigh at each prenatal visit
Support weight gain within National Academies of Science guidelines (11-20 lb, 5-9 kg)
Use ultrasonography as needed to measure adequacy of fetal growth
Screen for hypertensive disorders of pregnancy
Discuss safety related to place of birth
Recommend active management of the third stage of labor
Encourage measures to limit prolonged pregnancy through enhanced cervical ripening: nipple stimulation, membrane sweeping, and vaginal intercourse
Intrapartum
Support prolonged labor with ambulation, position changes, hydration
Be patient with prolonged phases of labor
Be prepared for shoulder dystocia
Have IV access and uterotonics ready in case of postpartum hemorrhage
Postpartum
Consider active management of the third stage with delayed cord clamping
Assess postpartum blood loss and uterine contraction frequently
Facilitate immediate skin-to-skin contact and early breastfeeding
Encourage frequent breastfeeding or nipple stimulation to hasten lactogenesis II
Use appropriately large nipple shield if pumping milk
Encourage early ambulation
Consider postpartum anticoagulation with obesity classes II and III or postcesarean
Assist women to plan postpartum weight loss if needed
Diabetes screening at 4-12 wk following gestational diabetes
Screen for postpartum depression
Policy promotion
Support laws that reduce disparities in employment, education, and access to healthcare
Support laws that increase neighborhood safety and the potential for recreation
Support laws that reduce workplace stress
Support laws that reduce environmental pollution

Abbreviations: BMI, body mass index; IV, intravenous.
[a]From American College of Obstetricians and Gynecologists,[4] Anstey and Jevitt,[5] Carlson et al,[48] Carlson et al,[49] Beckwith et al,[50] and American College of Obstetricians and Gynecologists.[51]

inflammation and those caused by hormone resistance, require special consideration. Leptin sensitivity, like insulin sensitivity, is reduced in obesity.[55] High leptin and visfatin levels limit the effectiveness of oxytocin, reducing myometrial contractions.[55] This is a likely factor in the higher incidence of prolonged pregnancies and the longer labors in obesity.[55] Because of the tocolytic effect of leptin, mechanical cervical ripening may be more effective than use of prostaglandins.[50] Most women will benefit from vigilant patience while awaiting the onset of labor and during prolonged labors.[48,49] Patience may include use of a longer labor curve; however, no partograms adjusted for BMI have been developed. Because of the high rate of uterine atony with postpartum hemorrhage associated with obesity,[48] active management of the third stage with delayed cord clamping can be discussed with women during the third trimester and recommended. High leptin levels may also reduce the effectiveness of oxytocin on myoepithelial cells involved in the milk ejection reflex contributing to

delayed lactogenesis II.[5] All measures that facilitate early breastfeeding support women with obese BMIs while lactation is established.[5]

The chronic, systemic inflammation associated with adipose dysfunction increases the risk for postpartum deep vein thrombosis.[4] As with others at risk for deep vein thrombosis, early postpartum ambulation is a key element of care.[4] Alternating compression stockings and low-dose anticoagulation for 24 to 48 hours postpartum are also care considerations particularly postcesarean surgery.[4] Blood glucose levels normalize immediately postpartum in women with obesity and gestational diabetes, but women remain at increased risk for future type 2 diabetes.[51] Therefore, postpartum diabetes screening at 4 to 12 weeks is offered to women with obesity complicated by gestational diabetes.[51]

Future obesity research will need to examine increasing global experience with obesity-related morbidities at the individual and population levels to capture the effects of socioeconomic and environmental disparities on obesity. Epigenetic changes will need to be studied over generations to reveal stronger causal links with obesity. Finally, clinical practices meant to ameliorate obesity-related morbidities will need to be studied for effectiveness, safety, and patient satisfaction.

CONCLUSION

The research cited in this article involving socioeconomic disparities, the microbiome, EDCs, epigenetics, and links to obesity moves the focus of obesity prevention from the individual level to the community and national health policy levels. Other factors are yet to be discovered and may limit the applicability of this review. Black and Hispanic women coping with obesity and social disparities in education, employment, and the environment also suffer from the dual discriminations of obesity and racism. The most effective measures for reducing future population obesity will not be accomplished at the individual patient level but will involve reducing the socioeconomic disparities that underpin obesity. Environmental activism to reduce obesogenic pollutants seems far removed from nursing care but may be an important strategy in future weight optimization. Attempts to reduce the stress of living with racism, improve educational equity, and promote fair employment practices are long-range strategies for obesity reduction. As the most numerous healthcare providers in the United States, nurses can affect changes in obesity at the individual and population levels through targeted patient care and sustained sociopolitical action.

References

1. World Health Organization. Obesity and overweight fact sheet. http://www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight. Accessed January 12, 2019.
2. Centers for Disease Control and Prevention. Obesity trends among U.S. adults between 1985 and 2010. https://www.cdc.gov/obesity/downloads/obesity_trends_2010.pdf. Accessed January 12, 2019.
3. Hales CM, Carroll MD, Fryar CD, Ogden CL. *Prevalence of Obesity Among Adults and Youth: United States, 2015-2016*. NCHS data brief, no. 288. Hyattsville, MD: National Center for Health Statistics; 2017.
4. American College of Obstetricians and Gynecologists. Practice bulletin no. 156: obesity in pregnancy. *Obstet Gynecol*. 2015;126(6):e112–e126.
5. Anstey E, Jevitt C. Maternal obesity and breastfeeding: a review of the evidence and implications for practice. *Clin Lact*. 2011;2–3:11–16.
6. Obesity Medicine Association. Definition of obesity. https://obesitymedicine.org/definition-of-obesity/. Accessed September 15, 2018.
7. Heymsfield SB, Peterson CM, Thomas DM, Heo M, Schuna JM. Why are there race/ethnic differences in adult body mass index-adiposity relationships? A quantitative critical review. *Obes Rev*. 2016;17(3):262–275.
8. Fitzsimons K, Modder J, Greer I. Obesity in pregnancy: risks and management. *Obstet Med*. 2009;2(2):52–62.
9. Coelho M, Oliveira T, Fernandes R. Biochemistry of adipose tissue: an endocrine organ. *Arch Med Sci*. 2013;9;2:191–200.
10. Cinti S. Adipose organ development and remodeling. *Comp Physiol*. 2018;8(4):1357–1431.
11. Barker DJP. *Mothers, Babies and Health in Later Life*. Edinburgh: Churchill Livingstone; 1994.
12. Kwon EJ, Kim YJ. What is fetal programming? A lifetime of health is under the control of in utero health. *Obstet Gynecol Sci*. 2017;60(6):506–519.
13. Waterland RA, Kellermayer R, Laritsky E, et al. Season of conception in rural Gambia affects DNA methylation at putative human metablastic epialleles. *PLOS Genet*. 2010;6:e1001252.
14. Fleming TP, Watkins AJ, Velasquez MA, et al. Origins of lifetime health around the time of conception: causes and consequences. *Lancet*. 2018;391:1842–1852.
15. Vickers M. Developmental programming and transgenerational transmission of obesity. *Ann Nutr Metab*. 2014;64:26–34.
16. Power C, Jefferis BJ. Fetal environment and subsequent obesity: a study of maternal smoking. *Int J Epidemiol*. 2002;31(2):413–419.
17. Rayfield S, Plugge E. Systematic review and meta-analysis of the association between maternal smoking in pregnancy and childhood overweight and obesity. *J Epidemiol Community Health*. 2017;71(2):162–173.
18. Vijayaraghaven M, Benmarnhia T, Pierce J, et al. Income disparities in smoking cessation and the diffusion of smoke-free homes among U.S. smokers: results from two longitudinal surveys. *PLoS One*. 2018;13(7):e0201467.
19. Harris H, Willett W, Michels K. Parental smoking during pregnancy and risk of overweight and obesity in the daughter. *Int J Obes*. 2103;37(10):1356–1363.
20. Bliss E, Whitesie E. The gut-brain axis, the human gut microbiota and their integration in the development of obesity. *Front Physiol*. 2018;9:900.
21. Sanmiguel C, Gupta A, Mayer E. Gut microbiome and obesity: a plausible explanation for obesity. *Curr Obes*. 2015;4(2):250–261.

22. Li Y. Epigenetic mechanisms link maternal diets and gut microbiome to obesity in the offspring. *Front Genet*. 2018; 9:342.
23. Chu D, Meyer M, Prince A, Aagaard K. Impact of maternal nutrition in pregnancy and lactation on offspring gut microbial composition and function. *Gut Microbes*. 2016;7(6): 459–470.
24. Anthony K, Ma J, Mitchell K, Racusin D, Versalovic J, Aagaard K. The preterm placental microbiome varies in association with excess maternal gestational weight gain. *Am J Obstet Gynecol*. 2015;212(5):653. e1-e16.
25. Indrio F, Martini S, Francavilla R, et al. Epigenetic matters: the link between early nutrition, microbiome, and long-term health development. *Front Pediatr*. 2017;22;5:178.
26. Owen C, Goldstein E, Clayton J, Segars J. Racial and ethnic health disparities in reproductive medicine: an evidence-based overview. *Semin Reprod Med*. 2013;31(5):317–324.
27. Hicken M, Lee H, Hing A. The weight of racism: vigilance and racial inequalities in weight-related measures. *Soc Sci Med*. 2018;199:157–166.
28. Dallman MF, Pecoraro N, Akana SF, et al. Chronic stress and obesity: a new view of "comfort food". *Proc Natl Acad Sci U S A*. 2003;100:11696–11701.
29. McFarland M, Taylor J, McFarland C. Weighted down by discriminatory policing: perceived unfair treatment and black-white disparities in waist circumference. *SSM Popul Health*. 2018;5:210–217.
30. Centers for Disease Control and Prevention. Obesity and socioeconomic status. https://www.cdc.gov/obesity/data/adult.html. Accessed January 12, 2019.
31. Rubin R. Obesity tied to income, education but not in all populations. *JAMA*. 2018;319(6):540.
32. Feng W, Fox A. Menu labels, for better, and worse? Exploring socio-economic and race-ethnic differences in menu label use in a national sample. *Appetite*. 2018;128:223–232.
33. Choi B, Schnall P, Dobson M, Yang H, Baker D, Seo Y. A socioecological framework for research on work and obesity in diverse urban transit operators based on gender, race and ethnicity. *Ann Occup Environ Med*. 2017;29:15.
34. Dashti HS, Scheer FA, Jacques PF, Lamon-Fava S, Ordovas JM. Short sleep duration and dietary intake: epidemiologic evidence, mechanisms, and health implications. *Adv Nutr*. 2015;13(6):648–659.
35. Kosanovich K, Sherman E. Trends in long-term unemployment. US Bureau of Labor Statistics. https://www.bls.gov/spotlight/2015/long-term-unemployment/pdf/long-term-unemployment.pdf. Published March 2015. Accessed January 12, 2019.
36. Buchmueller T, Valleta R. Work, health, and insurance: a shifting landscape for employers and workers alike. *Health Aff*. 2017;36(2):214–221.
37. Gordon-Larsen P. Food availability/convenience and obesity. *Adv Nutr*. 2014;5:809–817.
38. Darmon N, Drewnowski A. Contribution of food prices and diet cost to socioeconomic disparities in diet quality and health: a systematic review and analysis. *Nutr Rev*. 2015; 73(10):643–660.
39. Wilde P, Steiner A, Ver Ploeg M. For low-income Americans, living ≤ 1 mile (≤1.6 km) from the nearest supermarket is not associated with self-reported household food security. *Curr Dev Nutr*. 2017;1:e001446.
40. Preusting I, Brumley J, Odibo L, Spatz DL, Louis JM. Obesity as a predictor of delayed lactogenesis II. *J Hum Lact*. 2017; 33(4):684–691.
41. Gross R, Mendelsohn A, Fierman A, Hauser N, Messito MJ. Maternal infant feeding behaviors and disparities in early child obesity. *Childhood Obes*. 2014;10(2):145–151.
42. Thompson AL, Bentley ME. The critical period of infant feeding for the development of early disparities in obesity. *Soc Sci Med*. 2013;97:288–296. doi:10.1016/j.socscimend.2012.12.007.
43. Whitfield GP, Carlson SA, Ussery EN, et al. Racial and ethnic differences in perceived safety barriers to walking, United States National Health Interview Survey—2015. *Prev Med*. 2018;114:57–63.
44. Hoffman EE, Mair TTM, Hunter BA, Prince DM, Tebes JK. Neighborhood sexual violence moderates women's perceived safety in urban neighborhoods. *J Community Psychol*. 2018;46(1):79–94.
45. Darbe P. Endocrine disrupters and obesity. *Curr Obes Rep*. 2017;6:18–27.
46. Ranjit N, Siefert K, Padmanabhan V. Bisphenol-A and disparities in birth outcomes: a review and directions for future research. *J Perinatol*. 2010;30(1):2–9.
47. Nelson JW, Scammell M, Hatch EE, Webster TF. Social disparities in exposure to bisphenol A and polyfluoroalkyl chemicals: a cross-sectional study within NHANES 2003-2006. *Environ Health*. 2012;11:10.
48. Carlson NS, Corwin EJ, Lowe NK. Oxytocin augmentation in spontaneously laboring nulliparous women: multilevel assessment of maternal BMI and oxytocin dose. *Biol Res Nurs*. 2017;19(4):382–392.
49. Carlson NS, Corwin EJ, Lowe NK. Labor intervention and outcomes in women who are nulliparous and obese: a comparison of nurse-midwife to obstetrician intrapartum care. *J Midwifery Womens Health*. 2017;62(1):29–39.
50. Beckwith L, Magner K, Kritzer S, Warshak CR. Prostaglandin versus mechanical dilation and the effect of maternal obesity on failure to achieve active labor: a cohort study. *J Matern Fetal Neonatal Med*. 2017;30(13):1621–1626.
51. American College of Obstetricians and Gynecologists. Gestational diabetes mellitus. ACOG Practice Bulletin No. 190. *Obstet Gynecol*. 2018;131:e49–e64.
52. Leung AW, Chan RS, Sea MM, Woo J. An overview of factors associated with adherence to lifestyle modification programs for weight management in adults. *Int J Environ Res Public Health*. 2017;14(8):922. doi:10.3390/ijerph14080922.
53. Vanhees K, Vonhogen IG, van Scooten FJ, Godschalk RW. You are what you eat, and so are your children: the impact of micronutrients on the epigenetic programming of offspring. *Cell Mol Like Sci*. 2014;71(2):217–285.
54. Thomas D, Elliott EJ, Baur L. Low glycaemic index or low glycaemic load diets for overweight and obesity. *Cochrane Database Syst Rev*. 2007 (3):CD005105. doi:10.1002/1461858.CD005105.pub2.
55. Mumtaz S, AlSaif S, Wray S, Noble D. Inhibitory effect of visfatin and leptin on human and rat myometrial contractility. *Life Sci*. 2015;125:57–62.

The CE test for this article is available online only. Log onto the journal website, www.jpnnjournal.com, or to www.NursingCenter.com/CE/JPNN to access the test. For more than 32 additional continuing education articles related to the topic of obesity, go to NursingCenter.com\CE.

Instructions:

- Read the article. The test for this CE activity is to be taken online at www.NursingCenter.com/CE/JPNN.
- You will need to create (its free!) and login to your personal CE Planner account before taking online tests. Your planner will keep track of all your Lippincott Professional Development online CE activities for you.
- There is only one correct answer for each question.
- A passing score for this test is 12 correct answers. If you pass, you can print your certificate of earned contact hours and access the answer key. If you fail, you have the option of taking the test again at no additional cost.
- For questions, contact Lippincott Professional Development: 1-800-787-8985.

Registration Deadline:
June 4, 2021

Provider Accreditation:

Lippincott Professional Development will award 1.5 contact hours for this continuing nursing education activity.

Lippincott Professional Development is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749. Lippincott Professional Development is also an approved provider of continuing nursing education by the District of Columbia Board of Nursing, #50-1223, Florida Board of Nursing, #50-1223, and Georgia Board of Nursing, CE Broker #50-1223.

Disclosure Statement:

The authors and planners have disclosed that they have no financial relationships related to this article.

Payment:

- The registration fee for this test is $17.95.