



Commentary

Exploring Obesity as a Gendered Contagion: Impact on Lifestyle Interventions to Improve Cardiovascular Health

Ynhi T. Thomas, MD, MPH, MSc¹; Connie B. Newman, MD, FACP, FAHA, FAMWA²; Nina G. Faynshtayn³; and Alyson J. McGregor, MD, MA, FACEP⁴

¹Henry J.N. Taub Department of Emergency Medicine, Baylor College of Medicine, Houston, TX, USA; ²Department of Medicine, Division of Endocrinology, Diabetes, and Metabolism, New York University School of Medicine, New York, NY, USA; ³Undergraduate College, Brown University, Providence, RI, USA; and ⁴Department of Emergency Medicine, Warren Alpert Medical School, Brown University, Providence, RI, USA

ABSTRACT

Purpose: Obesity increases the risk of cardiovascular disease. Lifestyle interventions such as physical activity and diet are important components for reducing the risk of obesity. Data suggest that lifestyle choices differ between men and women, as well as in groups. The purpose of this review was to explore whether obesity can be considered as a gendered social contagion, associated with differences in lifestyle and response to lifestyle interventions in men and women.

Findings: There are important sex-based differences of obesity to consider. There is evidence that peers have an influence on lifestyle preferences such as physical activity level and dietary habits, but the evidence is inconclusive if the differences exist between men and women. Similarly, data from lifestyle intervention studies are not conclusive whether there are differences between men and women. There is not enough evidence for the notion that obesity is a gendered social contagion.

Implications: More research is needed to understand differences in lifestyle and lifestyle interventions between men and women, especially across the life span, which could have profound public health implications. (*Clin Ther.* 2022;44:23–32.) © 2021 Elsevier Inc.

Key words: cardiovascular health, obesity, physical activity, sex and gender medicine, social contagion, women's health.

INTRODUCTION

Obesity significantly increases the risk of cardiovascular disease (CVD), both directly and indirectly by increasing insulin resistance, and is estimated by the National Health and Nutrition Examination Survey 2015 to 2016 data to occur in 40.6% of the population, with a prevalence of 41.1% of female subjects and 39.9% of male subjects.¹ Lifestyle interventions such as diet and physical activity affect body weight and body mass index, and therefore lifestyle interventions are critical components for reduction of the risk of obesity and CVD associated with obesity. Data suggest that lifestyle choices differ in women and men as well as in social groups, indicating that obesity may be a gendered social contagion, defined as the spread of thoughts, attitudes, or behaviors in a group through conformity or imitation.² The question of whether the effectiveness of lifestyle interventions for obesity differs by sex, gender, or other personal variables is not yet answered. A better understanding of these differences is important for the development of weight loss strategies for individual patients at risk for CVD.

The aims of the present report were to review the data on sex and gender differences in lifestyle choices that affect obesity, as well as the effects of lifestyle

Accepted for publication November 17, 2021
<https://doi.org/10.1016/j.clinthera.2021.11.011>
0149-2918/\$ - see front matter

© 2021 Elsevier Inc.

interventions on weight loss. Our hypotheses are that obesity is a gendered social contagion, associated with differences in lifestyle, and that weight loss due to lifestyle interventions for obesity differs in men and women.

SEX DIFFERENCES IN OBESITY

The development of excess adipose tissue is influenced by biological sex, as well as gender and the environment. Adult women compared with adult men, with and without obesity, have a higher percentage of body fat.³ In female subjects, circulating estrogen (which is markedly decreased in postmenopausal women) influences the amount and location of adipose tissue, the metabolism of fat, and possibly satiety, food cravings, and energy expenditure.⁴ When excess calories are consumed, triglycerides are stored in adipocytes, which accumulate to form adipose tissue (body fat). The amount, location, and metabolism of adipose tissue differ in men and women.⁵ This is mainly caused by biologically based sex differences in genetics and sex hormones, including estrogen, testosterone, and other androgens.⁶

In premenopausal women, estrogen increases sympathetic tone and lipoprotein lipase activity in adipocytes, causing lipid deposition in subcutaneous tissue and the gluteal and femoral areas, especially in women who do not have obesity.⁷ In men, body fat is commonly located in the visceral (intra-abdominal) region. In men, testosterone levels correlate inversely with the amount of visceral fat.⁸ In women with androgen excess, increased adipose tissue is generally located in the visceral region.⁹ The mechanism underlying the accumulation of visceral fat in women with chronic androgen excess is not understood.¹⁰

Visceral fat and overall fat mass are associated with an increased risk of CVD and diabetes.¹¹ Adipocytes in the subcutaneous area, compared with the visceral area, are larger in diameter, enabling storage of more triglycerides for energy needs. It has been hypothesized that subcutaneous fat is important in times of low food supply and helps women meet the caloric needs for gestation and breastfeeding.⁵ In contrast, visceral fat is more suited to meeting immediate energy needs, such as during hunting.

The presence of estrogen in premenopausal women may have implications for satiety and for energy expenditure.⁴ During menopause, women accumulate

visceral fat and develop a greater risk of cardiometabolic disease. Postmenopausal women have less fat oxidation and energy expenditure compared with women before menopause.¹² Limited data suggest that estrogen replacement in postmenopausal women may prevent weight gain.¹³ More research is needed to better understand the effects of estrogen on adipose tissue and metabolism.

Sex chromosomes may also have a role in sex differences in fat mass and its distribution. Analysis of genome-wide association studies in >200,000 people identified ~49 loci related to the waist-to-hip ratio. Of these, sex differences were found in 20 loci, and 19 of the 20 loci were associated with greater effects in female subjects compared with male subjects.¹⁴ In addition, studies of adult mice with testes or ovaries removed after puberty suggest that two X chromosomes are associated with higher body weight and fat, compared with one X chromosome.¹⁵

There is evidence that women and men differ in their diets, cravings, and adherence to recommended macronutrient intake, with women consuming more sugar than men.^{16–18} Studies of food intake across the phases of the menstrual cycle show greater intake of some macronutrients, as well as increased appetite and food cravings, in the luteal phase, which suggest a role for sex steroid hormones.^{19,20} In a cross-sectional survey of the diet of 210,000 people (52.5% women, mean age of 55 years), in the United Kingdom Biobank population, calorie consumption adjusted for body weight was higher than recommended in 42% of women and 32% of men.²¹ In analyses adjusted for age, socioeconomic status, and ethnicity, women were more likely than men to consume more total sugar, total fat, and saturated fat than recommended. Men had a greater rate of nonadherence to recommendations for protein. Other research showed that cravings differ in women and men (ie, sweet foods for women, savory foods for men).²² Although biological sex has a significant impact on obesity outcomes, it is only one of the many important aspects underlying this disease.

IMPACT OF SEX DIFFERENCES IN OBESITY ON CARDIOVASCULAR HEALTH

The leading cause of death for women is CVD, which usually presents in women approximately 10 years later than in men.²³ This lag may be related to the presence of circulating estrogen, produced by the ovaries in premenopausal women, and the

reduction in circulating estrogen during menopause that is associated with a shift in body fat to the visceral area.^{4,12,24} Increase in visceral fat is associated with increased CVD and diabetes.^{25–29} Symptoms of atherosclerotic CVD differ in women and men, which could lead to delayed diagnosis and treatment of women.^{30,31}

Sex differences in obesity can affect cardiovascular health in significant ways. Obesity is both a disease and a risk factor for conditions such as diabetes, hypertension, and hyperlipidemia that contribute to CVD. Obesity is associated with increased risk of heart failure, atrial fibrillation, and atherosclerotic coronary artery disease.³² In the Framingham Heart Study cohort followed for up to 44 years, the risk of CVD was greater in women with obesity compared with men with obesity (64% vs 46%, respectively).³³

Obesity has been linked to sex differences in cardiac morphology. In a study based on clinical and echocardiographic data from 581 women and men without established CVD and body mass index >27.0 kg/m², the prevalence of subclinical cardiac disease was higher in women than in men (77% vs 62%; $P < 0.001$).³⁴ Women had a higher prevalence of left atrial dilatation than men, whereas men had a higher prevalence of abnormal left ventricular geometry. Similarly, in the Strong Heart Study cohort, left ventricular mass was greater in obese women than in obese men, and relative wall thickness was also greater in women than in men.³⁵

Obesity can lead to a significant risk of CVD in women. Given the impact of sex differences in obesity on cardiovascular health, it is important to understand any lifestyle differences and response to lifestyle interventions in men and women.

LIFESTYLE DIFFERENCES IN MEN AND WOMEN

Although obesity is complex, lifestyle choices such as inadequate diets and insufficient physical activity may lead to weight gain. Lifestyle preferences develop throughout a person's life through socialization.³⁶ The social environment, which comprises the social relationships and surroundings of an individual, plays an important role in lifestyle differences in men and women.

In a review of 13 studies by Sawka et al³⁷ on physical activity and sedentary behavior among youth (ages 6–18 years), higher physical activity levels among friends

were associated with higher levels of physical activity of the individual. The 3 longitudinal studies included in the review evaluated the change in the participants' physical activity levels over time and found that their level changed to reflect their friends' level. Six studies noted differences between the influence of friends on physical activity and sedentary behaviors of boys and girls. Boys were more active and more likely to be influenced by the physical activity behaviors of their friends compared with girls. However, the data supporting an association between friends and influence on an individual's sedentary behavior were inconsistent.

Similarly, in a systematic review by Fitzgerald et al³⁸ of friends' influence on physical activity in adolescents, peer support was found to be associated with physical activity. For instance, having peers that promote physical activity involvement was linked with an adolescent's ability to overcome barriers in engaging in physical activities. Friendship quality and peer acceptance were also important for perceived self-competence, sport continuation, and enjoyment of physical activity. Most of the studies reviewed did not specify differences between boys and girls as a primary end point. Of the few that did, boys engaged in less intense physical activity in the presence of family than with friends, and the reverse was observed for girls. The perception of peer norms predicted the intention to engage in physical activity for boys but not for girls. A separate systematic review by Macdonald-Wallis et al³⁹ also noted that similarities in physical activity level were stronger for boys than for girls. Interestingly, a study of 375 youth (185 male subjects, 190 female subjects) tracking accelerometer usage over 7 days found that although boys were more active than girls, the magnitude of differences for overall physical activity was smaller than studies using self-reported data.⁴⁰

The lower level of physical activity in girls compared with boys seems to continue across the life span. In a questionnaire-based study of 344 men and 756 women, men reported higher levels of moderate-intensity, vigorous-intensity, and total leisure-time physical activity than women.⁴¹ In another questionnaire-based study of 4353 parents of school-aged children (58% women and 42% men), men were slightly more likely than women to engage in regular sporting activities, but, interestingly, men were also less likely to pursue regular daily walking.⁴² A cross-

sectional study from the National Health Interview Survey by Caspersen et al⁴³ noted that adult women (27%) had a higher prevalence of physical inactivity than men (21%), with greater differences between women and men at the younger and older age groups of 7 and 11 percentage points, respectively. Men were also more likely to report regular, sustained physical activity than women (27% vs 21%). Again, moderate or large differences were noted at the youngest and oldest age groups of 9 and 11 percentage points. The observed differences were smaller at ages 30 to 44 years and 45 to 64 years, but the rate for men increased in the group ages 65 to 74 years, whereas the rate for women was essentially unchanged. After age 74 years, the prevalence of regular, sustained activity began to decline substantially for both groups, but the decline was more significant for women. More recent data from the Centers for Disease Control and Prevention have also noted that physical inactivity increased with increasing age for adults ages 50 to 64 years (25.4%), 65 to 74 years (26.9%), and ≥ 75 years (35.3%), and it was significantly higher for women.⁴⁴ More studies are needed to understand the factors that influence physical activity differences between men and women across the life span.

In addition to physical activity, dietary habits are important components of an individual's lifestyle. In a review on the effects of friendship networks and dietary behavior in youth (ages 6–18 years), the authors included 7 articles of the 9041 articles retrieved and found that friends' unhealthy food consumption was associated with an individual's unhealthy food consumption, with a stronger association for boys than for girls.⁴⁵ Each of the included reports evaluated dietary behaviors through self-reported questionnaires. This review, however, has several limitations, including a small number of studies and with most focusing on school friendship networks, although the authors recognized the importance of peer networks outside of school. Most of the studies included were observational studies, which cannot determine causality. Other reviews have also provided evidence that a youth's or adolescent's eating behavior is influenced by his or her friends' eating behaviors, with an individual modeling the behavior of his or her peers.^{46–48}

In a robust review of 69 experimental studies with >5800 participants between 1974 and 2014, the authors considered the social phenomenon of modeling for food intake, in which people adapt

their food consumption behavior to that of their eating companion.⁴⁹ In general, social modeling seems to influence what and how much people consume. Studies that examined food intake mostly focused on the consumption of high-energy-dense foods, such as cookies, popcorn, and ice cream. These studies have found that people ate more, or less, when their eating companions ate more, or less, of these high-energy-dense foods. In studies that looked at modeling of food choices, the results were inconsistent, and the studies were noted to be underpowered.

It has been suggested that homophily, which is the similarity of social contacts, may influence health behaviors and weight similarities.^{50,51} One motive that has been postulated for modeling is that individuals might model to become more attractive or likable to another person. However, the review of experimental studies by Cruwys et al⁴⁹ found that participants adhered to social norms provided by remote models even when eating alone, when they believed their food intake was not observed by researchers, and when they did not expect further interaction with the model. It is questionable if participants model to purely elicit social approval. Overall, Cruwys et al concluded that an individual's sex, body weight, dieting status, personality factors, and hunger did not influence the strength of modeling. The authors also noted that most of the studies to date are on youth and adolescents (15 studies) or young adults (43 studies), and there is limited empirical evidence for adults ages >30 years.

Although studies have suggested that the social environment may influence certain lifestyle factors such as the level of physical activity or dietary intake, there are not enough studies that have primarily considered differences in men and women as a primary end point. More studies are also needed across the life span and to further explore the complexities of social relationships and environments.

EFFECTIVENESS OF LIFESTYLE INTERVENTIONS FOR WEIGHT LOSS IN MEN AND WOMEN

Studies evaluating differences in weight loss in men and women in response to various lifestyle interventions (including diet, physical activity, and a combination of both) have found inconsistent results, and in the few that reported differences, the percent change in weight loss was small. These trials generally had a small number of subjects, fewer men than women, and,

in many, the weight outcome was a change in weight in kilograms, which could be misleading because the absolute weight loss in men may be greater because of larger body size.⁵² Most studies did not specify weight outcomes in men compared with women as a primary end point.

In a systematic review of 21 trials of various lifestyle interventions, with trial duration ranging from 6 weeks to 30 months, only 5 trials evaluated percent body weight loss.⁵² In the 3 trials that evaluated diet plus exercise, percent body weight loss was significantly greater in men although the mean difference was small (1.6%). In a subsequent meta-analysis by Robertson et al⁵³ of weight loss in 13 clinical trials of lifestyle interventions, mean percent weight change did not differ between women and men. The authors acknowledged the limitations of these studies, which in addition to having a small number of subjects and a gender imbalance in participants, did not assess adherence or whether management in men and women was comparable. The Keep It Off Trial⁵⁴ and the Weight Loss Maintenance Study⁵⁵ evaluated maintenance of lost weight and did not find sex differences in weight regain.

Despite the lack of robust data showing differences in percent weight loss in men and women, some studies suggest that men and women prefer different types of lifestyle interventions. In the Keep It Off trial, women were more likely to report using commercial weight loss programs.⁵⁴ In the Lighten Up Trial, 740 overweight men and women randomized to 1 of 8 weight loss programs for 12 weeks, including commercial programs, found <50% of men chose programs of commercial companies.⁵⁶ Weight loss did not differ in men and women at the program end or at 1 year. It is hypothesized that men prefer simple, fact-based language with individual feedback.⁵⁷⁻⁵⁹ The hypothesis that gender-tailored interventions improve weight loss should continue to be studied.

Large, randomized trials of >1000 participants using lifestyle interventions to achieve weight loss have found either no difference between men and women in percent body weight loss, or a small difference that was statistically significant. However, these trials were not designed or powered to detect sex or gender differences, and weight loss was not a primary outcome. In the Diabetes Prevention Program, in adults with prediabetes, men and women had similar socioeconomic status at randomization; however, men (mean age, 54 years) were older than women (mean

age, 48 years), had a larger waist circumference and higher caloric intake, and were more physically active.^{60,61} In participants (734 women, 345 men) randomized to intensive lifestyle intervention (ILI), which consisted of reduced calorie diet and moderate physical activity, the percent reduction in body weight at 1 year was significantly greater in men versus women (8% vs 7%; $P = 0.02$), and men had a greater reduction in percent waist circumference (5.2% vs 4.4%). Men reported higher levels of physical activity compared with women but a similar percent reduction in calories consumed. In the placebo group (747 women, 335 men), weight loss and the reduction in waist circumference and reported caloric intake did not differ according to sex. Despite the greater weight loss and waist circumference in men, progression to diabetes did not differ in men and women.

In Look AHEAD (Action for Health in Diabetes), a randomized controlled trial of 5145 adults with diabetes and obesity or overweight (mean age, 59 years), the ILI group lost 4.7% of body weight compared with 2.1% in the usual care group at year 8, and 50% and 35.7%, respectively, lost 5% or more body weight.^{62,63} Examination of all years of the study found that men and women in the ILI group had comparable weight loss.⁶³ At year 8, women in the ILI group had a significantly greater mean percent weight loss compared with men, although the difference (0.9%) was small. In the usual care group, which received counseling and diabetes education, significantly greater percent weight loss was observed at years 4 and 8 in women compared with men, with a difference of 1.0% and 1.3%. As in the Diabetes Prevention Program,^{60,61} disproportionately more women were enrolled in Look AHEAD, and the analyses were not powered for sex differences.

Given the importance of obesity as a risk factor for diabetes and CVD, and the difficulty in losing weight and maintaining weight loss, more randomized controlled trial studies are needed to inform lifestyle interventions for men and women. These trials should be balanced between men and women and powered to detect potential differences in percent weight loss.

OBESITY: IS IT A GENDERED SOCIAL CONTAGION?

The idea that obesity can be socially transmitted was recognized in studies that found a tendency for obesity to spread and cluster among peer groups. One study

found that an adolescent's weight was correlated with his or her friends' weight, in which an individual who had friends with obesity were more likely to have obesity.⁶⁴ Furthermore, the impact of peer weight was greater among female subjects and in those with higher body mass index. In a more robust study, adolescents were followed up into early adulthood to investigate the effects of peer influences on weight outcomes.⁶⁵ Body mass index and overweight status of friends who were overweight were associated with increased risk of being overweight, with this influence persisting into young adulthood. However, peer effects on weight gain were stronger for male subjects than for female subjects in this instance.

Perhaps the most significant study for the notion of obesity as socially "contagious" is the one by Christakis and Fowler.⁶⁶ In this study, the authors used data from the Framingham Heart Study, which followed participants for 32 years. The main subject was referred to as the ego, while those he or she interacted with were called alters. Together, egos and alters composed a social network. The study found that between friends, the risk of obesity for the ego was increased by 57% if an alter developed obesity. More specifically, in same-sex friends, a man had a 100% chance of developing obesity if his male friends did, whereas the female-to-female spread of obesity was not significant. For opposite-sex friends, the difference was not significant. Among siblings, one sibling's chance of developing obesity increased by 40% if the other sibling also developed obesity. In particular, the risk of obesity increased by 67% among sisters, whereas this risk was only 44% in brothers. It is important to note that these differences could be related to gendered as well as biological factors. In married couples, husbands of wives with obesity had a 44% risk of obesity, compared with a slightly lower value of 37% for wives of husbands with obesity. Geographic distance did not change the effects between friends and siblings. These findings suggest that common exposure to environmental factors may not be responsible for the phenomenon observed. Similarly, a study by Hrushka et al⁶⁷ was able to replicate the findings of Christakis and Fowler⁶⁶; that is, a person's risk of obesity increased if they had alters with obesity in their social network.

Why obesity seems to cluster and spread differently within various social networks may be due to the different norms that each group embraces. In general,

norms may encourage an ego's ideas about acceptable weight, influencing him or her to internalize these norms and to pursue the group's ideal.^{66,67} The tendency for individuals to conform to norms may be due to a desire to gain or maintain approval in a social group.⁶⁸ This suggests that ideals, beliefs, and norms affect weight outcome. The data suggest that obesity is socially contagious, but it is less clear if the contagion is gendered.

Our hypotheses that obesity can be considered a gendered social contagion, associated with differences in lifestyle among men and women, and differences in weight loss in response to lifestyle interventions, are not supported by the available data. Whether this is indeed the case, or whether the findings are due to the quality of the trials, is not known. Differences in lifestyle between men and women may be influenced by social relationships, with some evidence that the level of physical activity and dietary habits are affected by an individual's peers. The implication of these differences among men and women is not conclusive, with the evidence to date showing inconsistencies in findings.

Studies that evaluated lifestyle interventions had limitations: unequal numbers of men and women, limited data for percent change in body weight, and lack of studies that defined sex differences in weight loss as a primary end point. Other gaps in the existing lifestyle intervention research include the lack of accurate assessment of caloric intake, improper evaluation of adherence, and sparsity of data on weight regain and on different durations of an intervention. More studies are also needed to compare different types of interventions, as well as interventions that are tailored for men compared with women. To address these research gaps, future randomized controlled studies should be powered to detect significant differences between men and women in weight loss, with a primary outcome of assessing mean percent body weight loss according to sex. Study durations should also extend for a minimum of 1 year with a 6-month follow-up assessment. Other ideas include the evaluation of a variety of interventions such as commercial weight loss programs, intensive lifestyle behavioral counseling, group versus individual behavioral counseling, diet versus diet plus exercise, and programs tailored for men compared with women. In addition, future studies should focus on the assessment of effects

of geography (rural versus urban), social networks, access to healthy foods, and educational levels. Clearly, more research is needed to determine whether sex differences exist with regard to reduction in percent body weight due to lifestyle interventions, and whether preferences for lifestyle interventions differ in men and women.

CONCLUSIONS

Obesity is a complex disease with sex differences. The amount, location, and metabolism of adipose tissue differ in men and women due to differences in genetics and sex hormones. Sex differences in obesity can affect cardiovascular health. Obesity increases risk of heart disease in women and has been linked to sex differences in cardiac morphology. Understanding differences in lifestyles and response to lifestyle interventions in men and women is important. Lifestyle preferences are influenced by the social relationships and surroundings of an individual and may lead to differences in physical activity level or dietary habits. However, the evidence is not conclusive regarding whether there are particular differences between men and women. Similarly, data on men's and women's responses to lifestyle interventions are inconsistent. The evidence to date does not support obesity as a gendered social contagion, and more research is needed.

ACKNOWLEDGMENTS

Dr. Thomas is the lead author who conceptualized the idea, appraised the literature, wrote the manuscript, and edited it. Dr. Newman provided expertise on sex differences in obesity, appraised the literature on this topic, and edited the manuscript. Ms. Faynshtayn completed the literature review and wrote the Introduction section. Dr. McGregor served as the senior author who helped conceptualize the idea, provided her expertise on sex and gender medicine, appraised the literature, and edited the manuscript.

DISCLOSURES

The authors have indicated that they have no conflicts of interest regarding the content of this article.

REFERENCES

1. Hales CM, Carroll MD, Fryar CD, Ogden CL. Prevalence of obesity and severe obesity among adults: United States, 2017–2018. *NCHS Data Brief*. 2020 no 360. Hyattsville, MD: National Center for Health Statistics.
2. Colman AM. *A Dictionary of Psychology (3 ed.)*. Social Contagion. Oxford University 358 Press; 2014.
3. Jensen MD. Adipose tissue and fatty acid metabolism in humans. *J R Soc Med*. 2002;95(Suppl 42):3–7 PMID: 11133069.
4. Mauvais-Jarvis F, Clegg DJ, Hevener AL. The role of estrogens in control of energy balance and glucose homeostasis. *Endocr Rev*. 2013;34:309–338.
5. Shi H, Clegg DJ. Sex differences in the regulation of body weight. *Physiol Behav*. 2009;97:199–204 Epub 2009 Feb 27. PMID: 19250944; PMCID: PMC4507503.
6. Link JC, Reue K. Genetic basis for sex differences in obesity and lipid metabolism. *Annu Rev Nutr*. 2017;37:225–245 Epub 2017 Jun 19. PMID: 28628359; PMCID: PMC5759759.
7. Palmer BF, Clegg DJ. The sexual dimorphism of obesity. *Mol Cell Endocrinol*. 2015;402:113–119.
8. Khaw KT, Barrett-Connor E. Lower endogenous androgens predict central adiposity in men. *Ann Epidemiol*. 1992;2:675–682.
9. Evans DJ, Barth JH, Burke CW. Body fat topography in women with androgen excess. *Int J Obes*. 1988;12:157–162.
10. Navarro G, Allard C, Xu W, Mauvais-Jarvis F. The role of androgens in metabolism, obesity, and diabetes in males and females. *Obesity (Silver Spring)*. 2015;23:713–719.
11. Wajchenberg BL. Subcutaneous and visceral adipose tissue: their relation to the metabolic syndrome. *Endocr Rev*. 2000;21:697–738.
12. Lovejoy JC, Champagne CM, de Jonge L, Xie H, Smith SR. Increased visceral fat and decreased energy expenditure during the menopausal transition. *Int J Obes (Lond)*. 2008;32:949–958 Epub 2008 Mar 11. PMID: 18332882; PMCID: PMC2748330.
13. Gambacciani M, Ciaponi M, Cappagli B, et al. Body weight, body fat distribution, and hormonal replacement therapy in early postmenopausal women. *J Clin Endocrinol Metab*. 1997;82:414–417 Erratum in: *J Clin Endocrinol Metab*. 1997;82:4074. PMID: 9024228.
14. Shungin D, Winkler TW, Croteau-Chonka DC, et al. New genetic loci link adipose and insulin biology to body fat distribution. *Nature*. 2015;518:187–196 PMID: 25673412; PMCID: PMC4338562.
15. Chen X, McClusky R, Chen J, et al. The number of x chromosomes causes sex differences in adiposity in mice. *PLoS Genet*. 2012;8:e1002709.
16. Vaideloo M, Scott M, Quatromoni P, Jacques P, Parekh N. Trends in dietary fat and high-fat food intakes from 1991 to 2008 in the Framingham Heart Study participants. *Br J Nutr*. 2014;111:724–734 Epub 2013 Sep 19. PMID: 24047827; PMCID: PMC4103899.

17. Vitale M, Masulli M, Coccozza S, et al. Sex differences in food choices, adherence to dietary recommendations and plasma lipid profile in type 2 diabetes—the TOSCA.IT study. *Nutr Metab Cardiovasc Dis*. 2016;26:879–885 Epub 2016 Apr 16. PMID: 27212622.
18. Millen BE, Quatromoni PA, Franz MM, Epstein BE, Cupples LA, Copenhafer DL. Population nutrient intake approaches dietary recommendations: 1991 to 1995 Framingham Nutrition Studies. *J Am Diet Assoc*. 1997;97:742–749 PMID: 9216550.
19. Krishnan S, Tryon RR, Horn WF, Welch L, Keim NL. Estradiol, SHBG and leptin interplay with food craving and intake across the menstrual cycle. *Physiol Behav*. 2016;165:304–312.
20. Gorczyca AM, Sjaarda LA, Mitchell EM, et al. Changes in macronutrient, micronutrient, and food group intakes throughout the menstrual cycle in healthy, premenopausal women. *Eur J Nutr*. 2016;55:1181–1188 Epub 2015 Jun 5. PMID: 26043860; PMCID: PMC6257992.
21. Bennett E, Peters SAE, Woodward M. Sex differences in macronutrient intake and adherence to dietary recommendations: findings from the UK Biobank. *BMJ Open*. 2018;8:e020017.
22. Hallam J, Boswell RG, DeVito EE, Kober H. Gender-related differences in food craving and obesity. *Yale J Biol Med*. 2016;89:161–173 PMID: 27354843; PMCID: PMC4918881.
23. Centers for Disease Control and Prevention. Leading Causes of Death—Females—All races and origins—United States. 2017. Accessed October 10, 2021. <https://www.cdc.gov/women/lcod/2017/all-races-origins/index.htm>.
24. Guthrie JR, Dennerstein L, Taffe JR, Leher P, Burger HG. The menopausal transition: a 9-year prospective population-based study. The Melbourne Women’s Midlife Health Project. *Climacteric*. 2004;7:375–389 PMID: 15799609.
25. Rosenquist KJ, Pedley A, Massaro JM, Therkelsen KE, Murabito JM, Hoffmann U, Fox CS. Visceral and subcutaneous fat quality and cardiometabolic risk. *JACC Cardiovasc Imaging*. 2013;6:762–771 Epub 2013 May 8. PMID: 23664720; PMCID: PMC3745280.
26. Abraham TM, Pedley A, Massaro JM, Hoffmann U, Fox CS. Association between visceral and subcutaneous adipose depots and incident cardiovascular disease risk factors. *Circulation*. 2015;132:1639–1647 Epub 2015 Aug 20. PMID: 26294660; PMCID: PMC4779497.
27. McLaughlin T, Lamendola C, Liu A, Abbasi F. Preferential fat deposition in subcutaneous versus visceral depots is associated with insulin sensitivity. *J Clin Endocrinol Metab*. 2011;96:E1756–E1760 Epub 2011 Aug 24. PMID: 21865361; PMCID: PMC3205890.
28. Neeland IJ, Turer AT, Ayers CR, Powell-Wiley TM, Vega GL, Farzaneh-Far R, Grundy SM, Khera A, McGuire DK, de Lemos JA. Dysfunctional adiposity and the risk of prediabetes and type 2 diabetes in obese adults. *JAMA*. 2012;308:1150–1159 PMID: 22990274; PMCID: PMC3556508.
29. Fuster JJ, Ouchi N, Gokce N, Walsh K. Obesity-induced changes in adipose tissue microenvironment and their impact on cardiovascular disease. *Circ Res*. 2016;118:1786–1807 PMID: 27230642; PMCID: PMC4887147.
30. Khan NA, Daskalopoulou SS, Karp I, Eisenberg MJ, Pelletier R, Tsadok MA, Dasgupta K, Norris CM, Pilote LGENESIS PRAXY Team. Sex differences in acute coronary syndrome symptom presentation in young patients. *JAMA Intern Med*. 2013;173:1863–1871 PMID: 24043208.
31. Ladwig KH, Fang X, Wolf K, Hoschar S, Albarqouni L, Ronel J, Meinertz T, Spieler D, Laugwitz KL, Schunkert H. Comparison of delay times between symptom onset of an acute ST-elevation myocardial infarction and hospital arrival in men and women <65 years versus ≥65 years of age: findings from the Multicenter Munich Examination of Delay in Patients Experiencing Acute Myocardial Infarction (MEDEA) Study. *Am J Cardiol*. 2017;120:2128–2134 Epub 2017 Sep 18. PMID: 29122276.
32. Powell-Wiley TM, Poirier P, Burke LE, Després JP, Gordon-Larsen P, Lavie CJ, Lear SA, Ndumele CE, Neeland IJ, Sanders P, St-Onge MP American Heart Association Council on Lifestyle and Cardiometabolic Health; Council on Cardiovascular and Stroke Nursing; Council on Clinical Cardiology; Council on Epidemiology and Prevention; and Stroke Council. Obesity and Cardiovascular Disease: a Scientific Statement from the American Heart Association. *Circulation*. 2021;143:e984–e1010.
33. Wilson PW, D’Agostino RB, Sullivan L, Parise H, Kannel WB. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. *Arch Intern Med*. 2002;162:1867–1872 PMID: 12196085.
34. Halland H, Lønnebakken MT, Pristaj N, Saeed S, Midtbø H, Einarsen E, Gerdtts E. Sex differences in subclinical cardiac disease in overweight and obesity (the FATCOR study). *Nutr Metab Cardiovasc Dis*. 2018;28:1054–1060 Epub 2018 Jun 21. PMID: 30177273.
35. De Simone G, Devereux RB, Chinali M, et al. Sex differences in obesity-related changes in left ventricular morphology: the Strong Heart Study. *J Hypertens*. 2011;29:1431–1438.
36. Mollborn S, Lawrence EM, Hummer RA. A gender framework for understanding health lifestyles. *Soc Sci Med*. 2020;265:113182.
37. Sawka KJ, McCormack GR, Nettel-Aguirre A, Hawe P, Doyle-Baker PK. Friendship networks

- and physical activity and sedentary behavior among youth: a systematized review. *Int J Behav Nutr Phys Act.* 2013;10:130 Published 2013 Dec 1.
38. Fitzgerald A, Fitzgerald N, Aherne C. Do peers matter? A review of peer and/or friends' influence on physical activity among American adolescents. *J Adolesc.* 2012;35:941–958.
 39. Macdonald-Wallis K, Jago R, Sterne JA. Social network analysis of childhood and youth physical activity: a systematic review. *Am J Prev Med.* 2012;43:636–642.
 40. Trost SG, Pate RR, Sallis JF, et al. Age and gender differences in objectively measured physical activity in youth. *Med Sci Sports Exerc.* 2002;34:350–355.
 41. Azevedo MR, Araújo CL, Reichert FF, Siqueira FV, da Silva MC, Hallal PC. Gender differences in leisure-time physical activity. *Int J Public Health.* 2007;52:8–15.
 42. Fiala J, Brázdová Z. A comparison between the lifestyles of men and women—parents of school age children. *Cent Eur J Public Health.* 2000;8:94–100.
 43. Caspersen CJ, Pereira MA, Curran KM. Changes in physical activity patterns in the United States, by sex and cross-sectional age. *Med Sci Sports Exerc.* 2000;32:1601–1609.
 44. Physical Inactivity Among Adults 50 Years and Older: MMWR Data Highlights. Centers for Disease Control and Prevention. Accessed October 10, 2021. <https://www.cdc.gov/physicalactivity/inactivity-among-adults-50plus/mmwr-data-highlights.pdf>.
 45. Sawka KJ, McCormack GR, Nettel-Aguirre A, Swanson K. Associations between aspects of friendship networks and dietary behavior in youth: findings from a systematized review. *Eat Behav.* 2015;18:7–15.
 46. Fletcher A, Bonell C, Sorhaindo A. You are what your friends eat: systematic review of social network analyses of young people's eating behaviours and bodyweight. *J Epidemiol Community Health.* 2011;65:548–555.
 47. Salvy SJ, Howard M, Read M, Mele E. The presence of friends increases food intake in youth. *Am J Clin Nutr.* 2009;90:282–287.
 48. Salvy SJ, de la Haye K, Bowker JC, Hermans RC. Influence of peers and friends on children's and adolescents' eating and activity behaviors. *Physiol Behav.* 2012;106:369–378.
 49. Cruwys T, Bevelander KE, Hermans RC. Social modeling of eating: a review of when and why social influence affects food intake and choice. *Appetite.* 2015;86:3–18.
 50. Centola D. An experimental study of homophily in the adoption of health behavior. *Science.* 2011;334:1269–1272.
 51. de la Haye K, Robins G, Mohr P, Wilson C. Homophily and contagion as explanations for weight similarities among adolescent friends. *J Adolesc Health.* 2011;49:421–427.
 52. Williams RL, Wood LG, Collins CE, Callister R. Effectiveness of weight loss interventions—is there a difference between men and women: a systematic review. *Obes Rev.* 2015;16:171–186.
 53. Robertson C, Avenell A, Boachie C, et al. Should weight loss and maintenance programmes be designed differently for men? A systematic review of long-term randomised controlled trials presenting data for men and women: the ROMEO project. *Obes Res Clin Pract.* 2016;10:70–84.
 54. Crane MM, Jeffery RW, Sherwood NE. Exploring gender differences in a randomized trial of weight loss maintenance. *Am J Mens Health.* 2017;11:369–375.
 55. Svetkey LP, Stevens VJ, Brantley PJ, et al. Comparison of strategies for sustaining weight loss: the weight loss maintenance randomized controlled trial. *JAMA.* 2008;299:1139–1148.
 56. Jolly K, Lewis A, Beach J, et al. Comparison of range of commercial or primary care led weight reduction programmes with minimal intervention control for weight loss in obesity: Lighten Up randomised controlled trial. *BMJ.* 2011;343:d6500 Published 2011 Nov 3.
 57. Hunt K, Wyke S, Gray CM, et al. A gender-sensitised weight loss and healthy living programme for overweight and obese men delivered by Scottish Premier League football clubs (FFIT): a pragmatic randomised controlled trial. *Lancet.* 2014;383:1211–1221.
 58. Patrick K, Calfas KJ, Norman CJ, et al. Outcomes of a 12-month web-based intervention for overweight and obese men. *Ann Behav Med.* 2011;42:391–401.
 59. Morgan PJ, Lubans DR, Collins CE, Warren JM, Callister R. 12-Month outcomes and process evaluation of the SHED-IT RCT: an internet-based weight loss program targeting men. *Obesity (Silver Spring).* 2011;19:142–151.
 60. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med.* 2002;346:393–403.
 61. Perreault L, Ma Y, Dagogo-Jack S, et al. Sex differences in diabetes risk and the effect of intensive lifestyle modification in the Diabetes Prevention Program. *Diabetes Care.* 2008;31:1416–1421.
 62. Wing RR, Bolin P, et al. Look AHEAD Research Group Cardiovascular effects of intensive lifestyle intervention in type 2 diabetes. *N Engl J Med.* 2013;369:145–154 Erratum in: *N Engl J Med.* 2014;370:1866. doi:10.1056/NEJMoa1212914.
 63. Look AHEAD Research Group Eight-year weight losses with an intensive lifestyle intervention: the Look AHEAD study. *Obesity (Silver Spring).* 2014;22:5–13.
 64. Trogdon JG, Nonnemaker J, Pais J. Peer effects in adolescent overweight. *J Health Econ.* 2008;27:1388–1399 Epub 2008 May 13. PMID: 18565605.

65. Ali MM, Amialchuk A, Gao S, Heiland F. Adolescent weight gain and social networks: is there a contagion effect? *Appl Econ*. 2012;44:2969–2983.
66. Christakis NA, Fowler JH. The spread of obesity in a large social network over 32 years. *N Eng J Med*. 2007;357:370–379.
67. Hrushka DJ, Brewis AA, Wutich A, et al. Shared norms and their explanation for the social clustering of obesity. *Am J Public Health*. 2011;101(S1):S295–S300 Epub 2011 May 9. PMID: 21555656; PMCID: PMC3222514.
68. Deutsch M, Gerard HB. A study of normative and informational influences upon individual judgement. *J Abnorm Psychol*. 1955;51:629–636 PMID: 13286010.

Address correspondence to: Ynhi T. Thomas, MD, MPH, MSc, Department of Emergency Medicine, Baylor College of Medicine, 1504 Ben Taub Loop, Houston, Texas 77030. E-mail: ythomas@bcm.edu.