HHS Public Access

Author manuscript

Curr Diab Rep. Author manuscript; available in PMC 2016 November 01.

Published in final edited form as:

Curr Diab Rep. 2015 November ; 15(11): 95. doi:10.1007/s11892-015-0666-6.

Mind the Gap: Race\Ethnic and Socioeconomic Disparities in Obesity

Patrick M. Krueger, Ph.D.[1] and **Eric N. Reither, Ph.D.**[2]

Patrick M. Krueger: Patrick.Krueger@ucdenver.edu; Eric N. Reither: Eric.Reither@usu.edu

[1]Department of Health & Behavioral Sciences, University of Colorado Denver | Anschutz Medical Campus, Administration Building, 1201 5th Street, Denver, CO, 80217

[2]Department of Sociology and the Yun Kim Population Research Laboratory, Utah State University, 0730 Old Main Hill, Logan, UT, 84322

Abstract

Race/ethnic and socioeconomic status (SES) disparities in obesity are substantial and may widen in the future. We review seven potential mechanisms that recent research has used to explain obesity disparities. Those seven mechanisms fall into three broad groups—health behaviors, biological and developmental factors, and the social environment—which incorporate both proximate and upstream determinants of obesity disparities. Efforts to reduce the prevalence of obesity in the U.S. population and to close race/ethnic and SES disparities in obesity will likely require the use of multifaceted interventions that target multiple mechanisms simultaneously. Unfortunately, relatively few of the mechanisms reviewed herein have been tested in an intervention framework.

Keywords

Body Mass Index; Obesity; Overweight; Race/Ethnicity; Socioeconomic Status; Disparities

INTRODUCTION

Recent research suggests that the decades-long increase in body mass is reaching a plateau, at least for some age groups [1], although other data contradict that trend [2]. Nevertheless, disparities in body mass by race/ethnicity, sex, and socioeconomic status (SES) persist and may widen in the future if members of advantaged groups stop gaining weight or begin losing weight more quickly than members of disadvantaged groups [2, 3]. Disparities in body mass foreshadow important disparities in health outcomes including disability, diabetes, cardiovascular disease, some cancers, and premature mortality. Indeed, the high prevalence of obesity explains 30% or more of the shortfall in life expectancy in the United States, relative to other high income nations [4].

Correspondence to: Patrick M. Krueger, `Patrick.Krueger@ucdenver.edu`.

CONFLICT OF INTEREST: Patrick M. Krueger and Eric N. Reither declare that they have no conflict of interest.

HUMAN AND ANIMAL RIGHTS AND INFORMED CONSENT: This article does not include human or animal research subjects.

DISPARITIES IN BODY MASS

This section briefly describes current race/ethnic and SES disparities in body mass and obesity. Researchers often use body mass index (BMI), measured as kg/m^2, to define overweight ($25.0 \leq BMI < 30.0$) and obesity ($BMI \geq 30.0$) among adults. BMI is calculated the same way among children, but the thresholds for overweight (between the 85^{th} and 95^{th} percentiles) and obesity (the 95^{th} percentile or higher) derive from comparison to age and sex specific growth charts from the 2000 U.S. population [5]. BMI is an imperfect measure of body fatness, but it is widely used in population-based studies because it is easy to collect and is highly predictive of adverse outcomes.

Race/ethnic disparities are substantial. Among adults aged 20 or older in 2011–2012, 10.9% of Asians were obese, followed by 33.4% of whites, 42% of Hispanics, and 47.8% of blacks [1]. These overall figures obscure important disparities in body mass within Asian and Hispanic groups. Among U.S. born Asians in 2011, Chinese have an average BMI of 24.9 (just below the threshold for overweight), Asian Indians have an average BMI of 25.8, and Filipinos have an average BMI of 27.3 [6]. Among U.S. born Hispanics in 2011, Cubans have an average BMI of 29.5 (just below the threshold for obese), Puerto Ricans have an average BMI of 30.6, and Mexicans have an average BMI of 31.1 [6]. Although the prevalence of obesity is lower among children than adults, race/ethnic disparities among children are nevertheless substantial. Among children in 2011–2012, the prevalence of obesity was 8.6% among Asians, 14.1% among whites, 20.2% among blacks, and 22.4% among Hispanics.

There are considerable SES disparities in obesity, which are increasing over time. Adults with a college degree have nearly twice the prevalence of obesity as adults with less than a high school degree [7]. Among children, SES disparities are widening due to declining levels of obesity among children whose parents have a college degree or more education, even as the prevalence of obesity increases among children whose parents have a high school degree or less [3]. Among adults, SES disparities in obesity are greater among females than among males [8], but among children, sex differences in the association between SES and obesity are inconsistent and vary across age groups and by race/ethnicity [9].

MECHANISMS AND POTENTIAL INTERVENTIONS

Overweight and obesity can arise from an excess of calories consumed relative to the calories expended. However, as Keith and colleagues [10] note, an exclusive focus on diet and physical activity is neither justified by existing research nor a fruitful strategy for designing effective interventions to reduce or prevent obesity. By focusing on a wider array of social, biological, and behavioral determinants of obesity, deeper insights may be gained with respect to the development and persistence of race/ethnic and SES disparities in obesity [11, 12]. We consider seven potential mechanisms that may explain obesity disparities, which fall into three broad categories: health behaviors, biological and developmental factors, and social environment. Each mechanisms frames obesity disparities from a different perspective, offers unique insights into the origins of the obesity epidemic, or provides distinct points of leverage for designing interventions.

Health Behaviors

Diet and Physical Activity—Diet and physical activity have received the most attention of the potential behavioral mechanisms that shape obesity, and have been most frequently been the subject of intervention studies. We consider diet and physical activity together as a single mechanism because, together, they define energy balance. Exercising more often or at more vigorous levels increases energy output, whereas consuming more calories increases energy intake. Increases in body mass occur when energy intake exceeds energy output, and the balance of calories is stored as body fat [13, 14]. Some research suggests that declining levels of physical activity might explain the swift rise in BMI and obesity in the U.S., at least among working age adults [15]. Further, between 1988 and 2010, leisure-time physical activity also declined among white, black, and Mexican American men and women, although there were no significant increases in caloric intake in any of those groups [16]. Prior research, however, shows that increased snacking could be primarily responsible for the U.S. obesity epidemic [17]. From our perspective, evidence suggests that both diet and physical activity play important roles in shaping current obesity disparities.

Understanding disparities in specific sports might allow interventions to be tailored to diverse preferences across groups, encourage sports that have higher caloric expenditures, or identify sports that are most likely to be maintained as individuals age [18]. Among adults, blacks and Mexican Americans are most likely to participate in team sports (e.g., football, soccer, basketball), whereas whites are most likely to participate in facilities based sports (e.g., weight lifting, swimming, golf)—disparities that are widest for those with the highest levels of education [18]. Among children, whites have higher rates of participation in most sports, with the exception of basketball, which is more common among blacks and Hispanics [19]. Participation in most sports increases with family income among children, except for basketball which is most common among children with the least income [19].

The types of food consumed also vary across race/ethnic and socioeconomic groups, and may have implications for total caloric intake [20]. Among children, discretionary calories (i.e., sugary drinks, salty snacks, and sweet snacks) declined between 2003 and 2010, although those declines occurred primarily among Hispanics and whites [21]. Black children are less likely than whites to consume sugary drinks at school, but are much more likely to consume sugary drinks at home [22]. Mexican American children are most likely to meet federal recommendations for fruits, vegetables, and dry beans and peas, relative to whites and blacks. Income, however, is inconsistently associated with consuming recommended amounts of specific food groups among children. Higher income children are more likely to consume adequate amounts of some fruits and vegetables, but there are few differences in other foods [23, 24]. Among adults, those with higher incomes are more likely to meet the minimum federal recommendations for consumption of fruits, vegetables, and whole grains [23, 24]. Further, Mexican American adults were most likely to meet recommendations for dry peas and beans and total grains, and blacks were least likely to meet recommendations for whole fruits, total vegetables, and milk [23, 24].

Recent population-based analyses suggest the need for significant reductions in energy balance to meet Healthy People 2020 recommendations [25], with greater reductions in

energy balance required with increasing age. Wang and colleagues [14] suggest that net reduction of 23 kcal/per day/per capita would be enough to meet the goals among children. A more aggressive net reduction of 166 kcal/per day/per capita would be necessary to meet the goals among adults aged 20–39, and 222 kcal/per day/per capita among adults aged 60 and older [13]. To close race/ethnic and socioeconomic disparities in obesity, net kcal reduction would have to be even greater among blacks, Mexican Americans, and persons with low incomes [13, 14]. Unfortunately, existing diet and physical activity interventions do not result in dramatic reductions in obesity or obesity disparities. Meta-analyses find no reductions or small reductions in body mass for interventions that promote healthy diets, reduced calorie diets, and encourage physical activity among adolescents [26, 27] or adults [28]. Further, diet and physical activity interventions are poorly suited to maintaining weight loss for longer durations, and rebound weight gain is common [13].

Sleep Duration—Short sleep duration may be linked to increased body mass through multiple mechanisms. First, short sleep has been linked to hormonal dysregulation, including decreased insulin sensitivity, impaired glucose tolerance, and altered levels of ghrelin and leptin—hormones linked to appetite regulation [29, 30]. Second, short sleep duration is linked to the consumption of poorer quality food (including sugary and fatty snacks) and a greater quantity of food [31, 32]. Finally, short sleep is associated with fatigue and reduced physical activity [33]. Notably, declines in average sleep duration in the U.S. occurred simultaneously with an increasing prevalence of obesity [34]. As a result, research identifies associations between short sleep, poor sleep quality, and elevated body masses among children and adults [35, 36].

Some evidence suggests that sleep duration varies across race/ethnic and socioeconomic groups. Among both children and adults, blacks, Hispanics and, in some samples, Asians, have shorter sleep durations than whites [37–39]. Children who live in families marked by low levels of income and parental education have shorter sleep durations and more sleep problems [40, 41]. Among adults, those who work longer hours, have lower levels of education, lower family incomes, or less diverse sources of income report shorter sleep hours [38]. However, sleep duration may not mediate disparities in obesity—two recent studies find no evidence that sleep duration mediates race/ethnic or SES differences in obesity [39, 42].

A more promising line of research suggests that the association between sleep duration and body mass varies across race/ethnicity, although results are sometimes inconsistent. Among adults, one study finds no race/ethnic differences in the association between sleep duration and body mass [43], but another finds that sleep duration is inversely associated with body mass among Mexican Americans, but not among Cuban Americans or Puerto Ricans [44]. A recent experiment finds that short sleep duration is associated with weight gain most strongly among black males, and more modestly among black females and white males [45]. Race/ethnic differences in the association between sleep duration and obesity vary by gender among adolescents. Among girls, longer sleep hours are associated with *higher* body masses for blacks, but have no significant association with body mass for whites, Asians, or Hispanics [39]. Among boys, sleep duration is unassociated with body mass for blacks, but is inversely associated with body mass for whites, Asians, and Hispanics [39]. Sleep

duration is a promising new area of study that may eventually lead to interventions that can reduce obesity disparities.

Screen Time & Sedentary Behaviors—Sedentary behaviors—including using the computer or watching television—is positively associated with obesity [46]. At first glance, physical activity and sedentary behaviors are simple inverses. However, individuals who participate in regular vigorous physical activity may also spend large portions of their day in sedentary activities. Thus, there is only a weak association between physical and sedentary activities [47, 48]. Sedentary behaviors may result in elevated body mass through the displacement of physical activity, by providing additional opportunities for snacking, or by disrupting sleep [49, 50]. There are important disparities in sedentary behaviors. Blacks, Hispanics, and those with few socioeconomic resources tend to be more sedentary than whites or those with more socioeconomic resources [46, 51]. Indeed, children in low income families have greater access to televisions, DVD players, and video games in their bedrooms than children in higher income families [52].

Few studies have examined whether sedentary behaviors account for disparities in obesity. Interestingly, the positive association between watching television and obesity is stronger for whites than for blacks or Hispanics, and for children in high income families than in low income families [53]. One meta-analysis finds that interventions that reduce sedentary behaviors are associated with modest reductions in body mass, and that combining sedentary behavior interventions with physical activity or diet interventions did not yield additional reductions in body mass [54]. Future research could specifically compare the impact of sedentary behavior interventions on obesity across race/ethnic or SES groups.

Biological and Developmental Factors

Thrifty Gene Hypothesis—A considerable body of scientific evidence has shown that obesity is a heritable condition [55, 56]. Several decades ago, this observation led to the development of the “thrifty gene” hypothesis (TGH) [57], which posits that human populations subjected to millennia of feast-or-famine conditions naturally select for genes that promote rapid weight gain in times of food surplus. According to TGH, elevated obesity risks in some populations (e.g., Native Americans from the U.S. Southwest) can be explained by interactions between thrifty genes and exposure to modern food-rich environments, thereby offering a straightforward and intuitively appealing evolutionary explanation for certain race/ethnic disparities in obesity.

Despite some genetic and anthropological evidence offering tentative support for the TGH [58, 59], it has been challenged on several fronts. First, human genes are only loosely tied to race/ethnic background; indeed, there is substantially more genetic variability within than between race/ethnic groups [60]. Second, while recent genome-wide association studies affirm that specific genes—including the fat mass and obesity associated gene (*FTO*) and melanocortin 4 receptor gene (*MC4R*)—are associated with obesity [55], these associations are not consistently observed across race/ethnic groups and explain only a small proportion of population variability in body weight [55, 56, 61]. This makes the notion of a thrifty gene (or genes) seem unlikely as a singular explanation for large race/ethnic disparities in obesity.

Third, new mathematical models show that the survival benefits conferred by thrifty genes should have led to ubiquitous genetic predispositions toward obesity over the course of human evolution, which has not occurred [61]. In sum, recent evidence suggests that the TGH provides, at best, an incomplete biological perspective on racial/ethnic disparities in obesity and provides no plausible explanation for large and widening SES disparities in obesity.

Developmental and Epigenetic Perspectives—Developmental and epigenetic explanations for obesity disparities provide compelling alternatives to traditional arguments about thrifty genes. Developmental perspectives emphasize that prenatal, perinatal and early childhood exposures to adverse environmental conditions tend to manifest in poor health outcomes later in life [62]. For example, maternal obesity and dietary practices can increase the odds of obesity at birth and throughout the remainder of the life course—which are subsequently transmitted to future generations through a "vicious cycle" of obesity and diabetes [63]. Consistent with this perspective, a recent study of 1,116 mother-child pairs found that parental BMI explained 37% of the difference in BMI between white and black children and 19% of the difference in BMI between white and Hispanic children, after adjusting for SES [64]. Of potentially great importance for future interventions, this study also found that factors in infancy (e.g., rapid weight gain between birth and 6 months of age) and early childhood (e.g., insufficient sleep from 6 months to 2 years of age) eliminated racial/ethnic differences in BMI, skinfold thickness, fat mass, and waist circumference at age 7 that were not explained by parental SES or BMI.

The importance of prenatal, perinatal and early childhood conditions may be explained by rapidly emerging knowledge about epigenetic mechanisms. Unlike traditional genetic explanations, the field of epigenetics emphasizes heritable and developmental alterations in gene function that are unrelated to DNA sequence [55, 61]. As Russo et al. [55] explain, "These non-genetic alterations are under the tight regulation of two major epigenetic mechanisms acting at the transcriptional level: methylation of cytosine residues of DNA and modification of the histone proteins associated with DNA (chromatine remodeling)" (p. 694). In other words, epigenetic "marks" on DNA affect genetic expression, without altering genetic code. A growing body of evidence indicates that epigenetic marks are related to obesity risks. For instance, research has linked paternal obesity to hypomethylation of the insulin-like growth factor 2 gene (*IGF2*) among newborns, which provides evidence supporting the intergenerational inheritance of obesity through epigenetic mechanisms [65]. Research has also linked perinatal nutrition in humans [66] and parental exposures to toxic chemicals in rats [67, 68] to epigenetic changes that promote obesity.

In summary, the field of epigenetics offers a compelling biological explanation for obesity disparities among minority and low-SES groups that is not tied to inherent genetic differences. Moreover, both developmental and epigenetic perspectives strongly emphasize the importance of interventions that target the health and wellbeing of parents before conception, during all phases of pregnancy, and throughout infancy and early childhood. As shown by Taveras et al. [46], improving early life conditions in disadvantaged populations has the potential to drastically reduce disparities in childhood obesity in the U.S. population.

Social Environment

Neighborhood Context—Neighborhood context can shape behaviors in myriad ways. Some neighborhoods are relatively safe and aesthetically pleasing environments that provide opportunities for exercise and healthy diets. Conversely, other neighborhoods are noisy, dangerous, offer few opportunities for healthy behaviors, or harbor norms that promote obesity [69, 70]. Neighborhoods are also marked by important race/ethnic and SES inequalities, with nonwhites and low income individuals often living in segregated and isolated communities [71].

Food deserts are geographic areas (urban neighborhoods or rural towns) that have limited access to supermarkets with whole grain foods and fresh fruits and vegetables, but where fast food restaurants and convenience stores offering fatty, salty, or highly processed foods may be readily available [72]. Areas where both minority and low income groups are concentrated are most likely to contain food deserts [73], and access to supermarkets is even more limited than either race or poverty alone would predict [74]. However, evidence that links food deserts and obesity is mixed. In a study based in Pittsburgh, Pennsylvania, researchers find that prices for healthy food is positively associated with obesity, but distance to the nearest supermarket is not [75]. In a nationally representative cohort study, Lee [76] finds that residents of poor and minority neighborhoods are more likely to have access to fast food restaurants and convenience stores, although they also had greater access to other food establishments, including supermarkets. She also finds that variation in food outlet availability is not associated with obesity among young children, after adjusting for confounders. Shier and colleagues [77] find that greater access to supermarkets is associated with *increased* levels of obesity.

Neighborhoods also vary in their access to built environments that may foster physical activity (e.g., sidewalks, bike paths, recreational facilities, and parks) and help reduce obesity [78, 79]. Neighborhoods with concentrated poverty or numerous minority residents are often marked by the poorest quality built environments [80]. In some areas, high levels of crime or perceptions of crime mean that residents are afraid to use neighborhood amenities even if they are available [80]. One study finds that residents of low SES and high minority neighborhoods have diminished access to physical activity facilities, and that greater access to such facilities is associated with reduced odds of being overweight [81]. In contrast, high income neighborhoods are generally more aesthetically pleasing, have more access to walking and biking opportunities, and feel safer than lower income neighborhoods [82, 83]. Some research shows that the association between access to physical activity facilities and physical activity is stronger for blacks than for whites [84]. Among Hispanics, access to parks is negatively associated with body mass, although that association only holds for girls [85]. Built environment variables are usually more weakly associated with obesity than are individual-level variables [86]. Nevertheless, infrastructural investments may pay important dividends for obesity reduction because built environments impact many individuals simultaneously.

Neighborhoods also vary in their ability to support sufficient sleep. Low income or minority neighborhoods are often clustered around airports or highways, which increase nighttime

noise [87]. Several studies have affirmed associations between neighborhood characteristics and sleep duration. Living in cities, especially larger cities, is associated with shorter sleep durations [88]. Further, residents of distressed neighborhoods—marked by high levels of crime, noise, and racist attitudes, and low levels of cleanliness—tend to report shorter sleep durations [89–91]. Indeed, neighborhood economic disadvantage explains about half of racial disparities in sleep problems [92]. Tests of whether sleep duration mediates the association between neighborhood context and obesity are lacking, but this is a promising avenue for future research.

Several studies find that neighborhoods marked by socioeconomic disadvantage or high levels of poverty are persistently associated with individual-level obesity [93, 94], even after adjusting for neighborhood-level measures of racial segregation and concentrated obesity [70]. In addition, a recent experiment that randomly assigned some residents who received housing vouchers to move to low-poverty neighborhoods found that living in a higher income community was associated with lower odds of being obese or having diabetes over the follow-up period [95]. These studies provide compelling evidence that impoverished communities increase the risk of obesity, but more research is needed to understand which mechanisms are most important.

Social Networks—A seminal paper by Christakis and Fowler [96] suggests that networks of classmates, friends, co-workers, or family may inform health behaviors and shape individual risks of obesity. Subsequent research suggests that Christakis and Fowler's findings reflect reverse-causality [97], wherein obesity plays an important role in friend selection [98], especially among whites and females [99].

Nevertheless, social networks have potential for understanding obesity disparities. Simulation studies suggest that dieting efforts are more successful when undertaken with friends, and even greater benefits may accrue to those who diet with friends of friends [100]. A review of friendships and food behaviors among adolescents finds that fast food consumption behaviors are often shared among groups of boys, whereas dieting, body image concerns, and eating disorders are often shared among groups of girls [101]. Network-based interventions have also shown promise. One study of black women [102], and a second study with a more diverse sample [103], found that subjects who enrolled in a weight loss program with a partner lost more weight than subjects who enrolled without a partner, but only if their partner also lost weight. Simulation studies suggest that improving the quality of schools may reduce race disparities in obesity, but only when social networks support those interventions [104].

CONCLUSION

At first glance, the solution to the obesity epidemic may involve nothing more than rebalancing caloric consumption with energy expenditure [13, 14]. Current evidence, however, suggests that disparities in obesity result from a multifaceted array of social, behavioral, developmental, and biological mechanisms. This wide range of potential mechanisms is daunting from a policy perspective, given limited evidence on the relative importance of each pathway for obesity and the difficulty in designing, funding and

evaluating programs that address multiple mechanisms simultaneously. Given limited success in closing obesity disparities through programs that narrowly target diet and physical activity, public health researchers and policy makers might have greater success if they (1) *consider the context* where obesity occurs most frequently, such as low-income neighborhoods, (2) develop programs that *enhance flexible resources* such as knowledge and beneficial social connections, and (3) devote special attention to *early life interventions* that have shown tremendous promise in eradicating obesity disparities in the United States.

One caveat remains. Efforts to close disparities may be even more difficult than simply reducing the prevalence of obesity. High status individuals may have resources (e.g., greater knowledge, stronger social connections, more economic resources) that leave them better positioned to capitalize on emerging interventions than minorities or those with low-SES, resulting in growing disparities [11, 12]. Thus, efforts to close disparities might emphasize interventions that are low cost, that can be implemented widely, and that can target low-SES and minority populations.

Acknowledgments

The authors acknowledge support from the National Institute of Diabetes and Digestive and Kidney Diseases of the National Institutes of Health (award number R21DK089414) and the Eunice Kennedy Shriver National Institute of Child Health and Human Development funded University of Colorado Population Center (award number R24HD066613). The National Institutes of Health played no role in the preparation of this manuscript, or the decision to submit it for publication. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

References

• Of importance

•• Of major importance

1. Ogden CL, Carroll MD, Kit BK, et al. Prevalence of childhood and adult obesity in the United States, 2011–2012. Journal of the American Medical Association. 2014; 311(8):806–14. [PubMed: 24570244]
2. Datar A, Chung PJ. Changes in socioeconomic, racial/ethnic, and sex disparities in childhood obesity at school entry in the United States. JAMA Pediatrics. 2015:E1–E2.
3. Frederick CB, Snellman K, Putnam RD. Increasing socioeconomic disparities in adolescent obesity. Proceedings of the National Academy of Sciences. 2014; 11(4):1338–42.
4. Preston SH, Stokes A. Contribution of obesity to international differences in life expectancy. American Journal of Public Health. 2011; 101(11):2137–43. [PubMed: 21940912]
5. Ogden CL, Flegal KM. Changes in terminology for childhood overweight and obesity. National Health Statistics Reports. 2010; 25:1–5. [PubMed: 20939253]
6. Krueger PM, Coleman-Minahan K, Rooks RN. Race/ethnicity, nativity, and trends in BMI among U.S. adults. Obesity. 2014; 22(7):1739–46. [PubMed: 24634406]
7. Reither EN, Hauser RM, Yang Y. Do birth cohorts matter? Age-period-cohort analyses of the obesity epidemic in the United States. Social Science & Medicine. 2009; 69:1439–48. [PubMed: 19773107]
8. Wang Y, Beydoun MA. The obesity epidemic in the United States--gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. Epidemiologic Reviews. 2007; 29:6–28. [PubMed: 17510091]
9. Wang Y. Disparities in pediatric obesity in the United States. Advances in Nutrition. 2011; 2:23–31. [PubMed: 22211187]

10. Keith SW, Redden DT, Katzmarzyk PT, et al. Putative contributors to the secular increase in obesity: exploring the road less traveled. International Journal of Obesity. 2006; 30:1585–94. [PubMed: 16801930]

11. Link BG, Phelan J. Social conditions as fundamental causes of disease. Journal of Health and Social Behavior. 1995:80–94. Extra Issue. [PubMed: 7560851]

12. Pampel FC, Krueger PM, Denney JT. Socioeconomic disparities in health behaviors. Annual Review of Sociology. 2010; 36:349–70.

13•. Basu S, Seligman H, Winkleby MA. A metabolic-epidemiological microsimulation model to estimate the changes in energy intake and physical activity necessary to meet the Healthy People 2020 obesity objective. American Journal of Public Health. 2014; 104(7):1209–16. This paper uses advanced simulation methods to estimate the necessary change in caloric balance to achieve Healthy People 2020 goals. The paper also discusses implications for disparities in obesity. [PubMed: 24832140]

14. Wang YC, Orleans T, Gortmaker SL. Reaching the Healthy People goals for reducing childhood obesity: closing the energy gap. American Journal of Preventive Medicine. 2012; 42(5):437–44. [PubMed: 22516482]

15. Church TS, Thomas DM, Tudor-Locke C, et al. Trends over 5 Decades in U.S. Occupation-Related Physical Activity and Their Associations with Obesity. PLoS ONE. 2011; 6(5):e19657. [PubMed: 21647427]

16. Ladabaum U, Mannalithara A, Myer PA, et al. Obesity, abdominal obesity, physical activity, and caloric intake in US adults: 1988 to 2010. The American Journal of Medicine. 2014; 127(8):717–27. [PubMed: 24631411]

17. Cutler DM, Glaeser EL, Shapiro JM. Why Have Americans Become More Obese? Journal of Economic Perspectives. 2003; 117(3):93–118.

18. Saint Onge JM, Krueger PM. Education and race/ethnic differences in types of exercise in the United States. Journal of Health and Social Behavior. 2011; 52(2):197–211. [PubMed: 21673147]

19. Turner RW, Perrin EM, Coyne-Beasley T, et al. Reported sports participation, race, sex, ethnicity, and obesity in U.S. adolescents from NHANES physical activity (PAQ_D). Global Pediatric Health. 2015; 2:1–9.

20. Austin GL, Krueger PM. Increasing the percentage of energy from dietary sugar, fats, and alcohol in adults is associated with increased energy intake but has minimal association with biomarkers of cardiovascular risk. Journal of Nutrition. 2013; 14(10):1651–8. [PubMed: 23946345]

21. Bleich SN, Wolfson JA. Trends in SSBs and snack consumption among children by age, body weight, and race/ethnicity. Obesity. 2015; 23(5):1039–46. [PubMed: 25919923]

22. Dodd AH, Briefel R, Cabili C, et al. Disparities in consumption of sugar-sweetened and other beverages by race/ethnicity and obesity status among United States schoolchildren. Journal of Nutrition Education and Behavior. 2013; 45(3):240–9. [PubMed: 23414783]

23. Kirkpatrick SI, Dodd KW, Reedy J, et al. Income and race/ethnicity are associated with adherence to food-based dietary guidance among US adults and children. Journal of the Academy of Nutrition and Dietetics. 2012; 112(5):624–35. [PubMed: 22709767]

24. Hiza HA, Casavale KO, Guenther PM, et al. Diet quality of Americans differs by age, sex, race/ethnicity, income, and education level. Journal of the Academy of Nutrition and Dietetics. 2013; 113(2):297–306. [PubMed: 23168270]

25. U.S. Department of Health and Human Services. Office of Disese Prevention and Health Promotion. Healthy People 2020. Washington, DC: 2013. http://www.healthypeople.gov/2020/

26. Metcalf B, Henley W, Wilkin T. Effectiveness of intervention on physical activity of children: systematic review and meta-analysis of controlled trials with objectively measured outcomes (EarlyBird 54). British Medical Journal. 2012; 345(e5888):1–11.

27. Ho M, Garnett SP, Baur LA, et al. Impact of dietary and exercise interventions on weight change and metabolic outcomes in obese children and adolescents: A systematic review and meta-analysis of randomized trials. JAMA Pediatrics. 2013; 167(8):759–68. [PubMed: 23778747]

28. Dombrowski SU, Knittle K, Avenell A, et al. Long term maintenance of weight loss with non-surgical interventions in obese adults: systematic review and meta-analyses of randomised controlled trials. British Medical Journal. 2014; 348(g2646):1–12.

29. Spiegel K, Leproult R, L'hermite-Balériaux M, et al. Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin. The Journal of Clinical Endocrinology and Metabolism. 2004; 89(11):5762–71. [PubMed: 15531540]
30. Morselli L, Leproult R, Balbo M, et al. Role of sleep duration in the regulation of glucose metabolism and appetite. Best Practice and research Clinical Encocrinology and Metabolism. 2010; 24(5):687–702.
31. Beebe DW, Simon S, Summer S, et al. Dietary intake following experimentally restricted sleep in adolescents. Sleep. 2013; 36(6):827–34. [PubMed: 23729925]
32. Kruger AK, Reither EN, Peppard PE, et al. Do sleep-deprived adolescents make less-healthy food choices? British Journal of Nutrition. 2014; 111:1898–904. [PubMed: 24524288]
33. McKnight-Eily LR, Eaton DK, Lowry R, et al. Relationship between hours of sleep and health-risk behaviors in US adolescent students. Preventive Medicine. 2011; 54(4–5):271–3. [PubMed: 21843548]
34. Matricciani LA, Olds TS, Blunden S, et al. Never enough sleep: a brief history of sleep recommendations for children. Pediatrics. 2012; 129(3):548–56. [PubMed: 22331340]
35. Vgontzas AN, Fernandez-Mendoza J, Miksiewicz T, et al. Unveiling the longitudinal association between short sleep duration and the incidence of obesity: the Penn State Cohort. International Journal of Obesity. 2014; 38:825–32. [PubMed: 24100421]
36. Mitchell JA, Rodriquez D, Schmitz KH, et al. Sleep duration and adolescent obesity. Pediatrics. 2013; 131:e1428–e34. [PubMed: 23569090]
37. Marczyk Organek KD, Taylor DJ, Petrie T, et al. Adolescent sleep disparities: sex and racial/ethnic differences. Sleep Health. 2015; 1(1):36–9.
38. Krueger PM, Friedman EM. Sleep duration in the United States: a cross-sectional population based study. American Journal of Epidemiology. 2009; 169(9):1052–63. [PubMed: 19299406]
39. Reither EN, Krueger PM, Hale L, et al. Ethnic variation in the association between sleep and body mass among U.S. adolescents. International Journal of Obesity. 2014; 38:944–9. [PubMed: 24480862]
40. El-Sheikh M, Bagley EJ, Keiley M, et al. Economic adversity and children's sleep problems: multiple indicators and moderation of effects. Health Psychology. 2013; 32(8):849–59. [PubMed: 23148451]
41. Marco CA, Wolfson AR, Sparling M, et al. Family socioeconomic status and sleep patterns of young adolescents. Behavioral Sleep Medicine. 2012; 10(1):70–80. [PubMed: 22250780]
42. Piccolo RS, Yang M, Bliwise DL, et al. Racial and socioeconomic disparities in sleep and chronic disease: results of a longitudinal investigation. Ethnicity & disease. 2013; 23(4):499–507. [PubMed: 24392615]
43. Ford ES, Li C, Wheaton AG, et al. Sleep duration and body mass index and waist circumference among U.S. adults. Obesity. 2014; 22(2):598–607. [PubMed: 23836704]
44. Knutson KL. Association between sleep duration and body size differs among three Hispanic groups. American Journal of Human Biology. 2010; 23:138–41. [PubMed: 21080442]
45••. Spaeth AM, Dinges DF, Goel N. Effects of Experimental Sleep Restriction on Weight Gain, Caloric Intake, and Meal Timing in Healthy Adults. Sleep. 2013; 36(7):981–90. This study randomly assigns exposure to short sleep to adults, and demonstrates that the association between short sleep and weight gain varies across race/ethnicity. [PubMed: 23814334]
46. Andersen RE, Crespo CJ, Bartlett SJ, et al. Relationship of physical activity and television watching with body weight and level of fatness among children: Results from the third national health and nutrition examination survey. JAMA. 1998; 279(12):938–42. [PubMed: 9544768]
47. Brodersen NH, Steptoe A, Williamson S, et al. Sociodemographic, developmental, environmental, and psychological correlates of physical activity and sedentary behavior at age 11 to 12. Annals of Behavioral Medicine. 2005; 29(1):2–11. [PubMed: 15677295]
48. Fakhouri TI, Hughes JP, Brody DJ, et al. PHysical activity and screen-time viewing among elementary school–aged children in the united states from 2009 to 2010. JAMA Pediatrics. 2013; 167(3):223–9. [PubMed: 23303439]

49. Thorp AA, Owen N, Neuhaus M, et al. Sedentary Behaviors and Subsequent Health Outcomes in Adults: A Systematic Review of Longitudinal Studies, 1996–2011. American Journal of Preventive Medicine. 2011; 41(2):207–15. [PubMed: 21767729]
50. Drescher AA, Goodwin JL, Silva GE, et al. Caffeine and Screen Time in Adolescence: Associations with Short Sleep and Obesity. Journal of Clinical Sleep Medicine: JCSM: Official Publication of the American Academy of Sleep Medicine. 2011; 7(4):337–42. [PubMed: 21897768]
51. Hanson MD, Chen E. Socioeconomic status, race, and body mass index: the mediating role of physical activity and sedentary behaviors during adolescence. Journal of Pediatric Psychology. 2007; 32(3):250–9. [PubMed: 16896193]
52. Tandon P, Zhou C, Sallis JF, et al. Home environment relationships with children's physical activity, sedentary time, and screen time by socioecnomic status. International Journal of Behavioral Nutrition and Physical Activity. 2012; 9(88):1–9. [PubMed: 22233712]
53. Singh GK, Kogan MD, Van Dyck PC, et al. Racial/Ethnic, Socioeconomic, and Behavioral Determinants of Childhood and Adolescent Obesity in the United States: Analyzing Independent and Joint Associations. Annals of Epidemiology. 2008; 18(9):682–95. [PubMed: 18794009]
54. Liao Y, Liao J, Durand CP, et al. Which type of sedentary behaviour intervention is more effective at reducing body mass index in children? A meta-analytic review. Obesity Reviews. 2014; 15(3): 159–68. [PubMed: 24588966]
55. Russo P, Lauria F, Siani A. Heritability of body weight: Moving beyond genetics. Nutrition, Metabolism and Cardiovascular Diseases. 2010; 20(10):691–7.
56. Speakman JR, Westerterp KR. A mathematical model of weight loss under total starvation: evidence against the thrifty-gene hypothesis. Disease Models & Mechanisms. 2013; 6(1):236–51. [PubMed: 22864023]
57. Neel JV. Diabetes mellitus: a "thrifty" genotype rendered detrimental by "progress"? Am J Hum Genet. 1962; 14:353–62. [PubMed: 13937884]
58. Arnaiz-Villena A, Fernández-Honrado M, Rey D, et al. Amerindians show association to obesity with adiponectin gene SNP45 and SNP276: population genetics of a food intake control and "thrifty" gene. Mol Biol Rep. 2013; 40(2):1819–26. [PubMed: 23108996]
59. Reinhard KJ, Johnson KL, LeRoy-Toren S, et al. Understanding the Pathoecological Relationship between Ancient Diet and Modern Diabetes through Coprolite Analysis: A Case Example from Antelope Cave, Mojave County, Arizona. Current Anthropology. 2012; 53(4):506–12.
60. Kuzawa CW, Sweet E. Epigenetics and the embodiment of race: Developmental origins of US racial disparities in cardiovascular health. American Journal of Human Biology. 2009; 21(1):2–15. [PubMed: 18925573]
61. Herrera BM, Keildson S, Lindgren CM. Genetics and epigenetics of obesity. Maturitas. 2011; 69(1):41–9. [PubMed: 21466928]
62. Shonkoff JP, Boyce W, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. JAMA. 2009; 301(21):2252–9. [PubMed: 19491187]
63. Dabelea D, Crume T. Maternal Environment and the Transgenerational Cycle of Obesity and Diabetes. Diabetes. 2011; 60(7):1849–55. [PubMed: 21709280]
64••. Taveras EM, Gillman MW, Kleinman KP, et al. Reducing racial/ethnic disparities in childhood obesity: The role of early life risk factors. JAMA Pediatrics. 2013; 167(8):731–8. This study uses early life factors, including parental body mass, to explain a substantial share of race/ethnic disparities in BMI. [PubMed: 23733179]
65. Soubry A, Schildkraut JM, Murtha A, et al. Paternal obesity is associated with IGF2 hypomethylation in newborns: results from a Newborn Epigenetics Study (NEST) cohort. BMC Med. 2013; 11:29. [PubMed: 23388414]
66. Milagro FI, Mansego ML, De Miguel C, et al. Dietary factors, epigenetic modifications and obesity outcomes: Progresses and perspectives. Molecular Aspects of Medicine. 2013; 34(4):782–812. [PubMed: 22771541]

67. Manikkam M, Tracey R, Guerrero-Bosagna C, et al. Plastics Derived Endocrine Disruptors (BPA, DEHP and DBP) Induce Epigenetic Transgenerational Inheritance of Obesity, Reproductive Disease and Sperm Epimutations. PLoS ONE. 2013; 8(1):e55387. [PubMed: 23359474]
68. Skinner MK, Manikkam M, Tracey R, et al. Ancestral dichlorodiphenyltrichloroethane (DDT) exposure promotes epigenetic transgenerational inheritance of obesity. BMC Med. 2013; 11:228. [PubMed: 24228800]
69. Black JL, Mancinko J. Neighborhoods and obesity. Nutrition Reviews. 2008; 66(1):2–20. [PubMed: 18254880]
70. Boardman JD, Saint Onge JM, Rogers RG, et al. Race Differentials in Obesity: The Impact of Place. Journal of Health and Social Behavior. 2005; 46:229–43. [PubMed: 16259146]
71. Iceland J, Wilkes R. Does socioeconomic status matter? race, class, and residential segregation. Social Problems. 2006; 53(2):248–73.
72. Karpyn A, Young C, Weiss S. Reestablishing healthy food retail: changing the landscape of food deserts. Childhood Obesity. 2012; 8(1):28–30. [PubMed: 22799475]
73. Dutko, P.; Ver Ploeg, M.; Farrigan, T. Characteristics and infleuntial factors of food deserts, ERR-140. U.S. Department of Agriculture: Economic Research Service; 2012.
74. Bower KM, Thorpe RJ Jr, Rohde C, et al. The intersection of neighborhood racial segregation, poverty, and urbanicity and its impact on food store availability in the United States. Preventive Medicine. 2014; 58(0):33–9. [PubMed: 24161713]
75. Ghosh-Dastidar B, Cohen D, Hunter G, et al. Distance to Store, Food Prices, and Obesity in Urban Food Deserts. American Journal of Preventive Medicine. 2014; 47(5):587–95. [PubMed: 25217097]
76. Lee H. The role of local food availability in explaining obesity risk among young school-aged children. Social Science & Medicine. 2012; 74(8):1193–203. [PubMed: 22381683]
77. Shier V, An R, Sturm R. Is there a robust relationship between neighbourhood food environment and childhood obesity in the USA? Public Health. 2012; 126(9):723–30. [PubMed: 22898435]
78. Feng J, Glass TA, Curriero FC, et al. The built environment and obesity: A systematic review of the epidemiologic evidence. Health & Place. 2010; 16(2):175–90. [PubMed: 19880341]
79. Papas MA, Alberg AJ, Ewing R, et al. The Built Environment and Obesity. Epidemiologic Reviews. 2007; 29(1):129–43. [PubMed: 17533172]
80. Hood E. Dwelling Disparities: How Poor Housing Leads to Poor Health. Environmental Health Perspectives. 2005; 113(5):A310–A7. [PubMed: 15866753]
81. Gordon-Larsen P, nelson MC, Page P, et al. Inequality in the built environment underlies key health disparities in physical activity and obesity. Pediatrics. 2006; 117(2):417–24. [PubMed: 16452361]
82. Oreskovic NM, Kuhlthau KA, Romm D, et al. Built Environment and Weight Disparities Among Children in High- and Low-Income Towns. Academic Pediatrics. 2009; 9(5):315–21. [PubMed: 19477705]
83. Sallis JF, Slymen DJ, Conway TL, et al. Income disparities in perceived neighborhood built and social environment attributes. Health & Place. 2011; 17(6):1274–83. [PubMed: 21885324]
84. Lightfoot K, Blanchard C. Does Race or Sex Moderate the Perceived Built Environment/Physical Activity Relationship in College Students? Behavioral Medicine. 2011; 37(2):54–9. [PubMed: 21660773]
85. Hsieh S, Klassen AC, Curriero FC, et al. Built Environment Associations with Adiposity Parameters among Overweight and Obese Hispanic Youth. Preventive Medicine Reports. 2:406–12. [PubMed: 26339570]
86. Bodea TD, Garrow LA, Meyer MD, et al. Socio-demographic and built environment influences on the odds of being overweight or obese: The Atlanta experience. Transportation Research Part A: Policy and Practice. 2009; 43(4):430–44.
87. Muzet A. Environmental noise, sleep and health. Sleep Medicine Reviews. 2007; 11(2):135–42. [PubMed: 17317241]
88. Hale L, Do DP. Racial Differences in Self-Reports of Sleep Duration in a Population-Based Study. Sleep. 2007; 30(9):1096–103. [PubMed: 17910381]

89. Hill TD, Burdette AM, Hale L. Neighborhood disorder, sleep quality, and psychological distress: Testing a model of structural amplification. Health & Place. 2009; 15(4):1006–13. [PubMed: 19447667]

90. Steffen PR, Bowden M. Sleep disturbance mediates the relationship between perceived racism and depressive symptoms. Ethnicity and Disease. 2006; 16:16–21. [PubMed: 16599343]

91. Hicken M, Lee H, Ailshire J, et al. "Every Shut Eye, Ain't Sleep": The Role of Racism-Related Vigilance in Racial/Ethnic Disparities in Sleep Difficulty. Race Soc Probl. 2013; 5(2):100–12. [PubMed: 23894254]

92. Spilsbury JC, Storfer-Isser A, Kirchner HL, et al. Neighborhood disadvantage as a risk factor for pediatric obstructive sleep apnea. The Journal of Pediatrics. 2006; 149(3):342–7. [PubMed: 16939744]

93. Ruel E, Reither EN, Robert SA, et al. Neighborhood effects on BMI trends: Examining BMI trajectories for Black and White women. Health & Place. 2010; 16(2):191–8. [PubMed: 19879795]

94. Robert SA, Reither EN. A multilevel analysis of race, community disadvantage, and body mass index among adults in the U.S. Social Science & Medicine. 2004; 59:2421–34. [PubMed: 15474198]

95. Ludwig J, Sanbonmatsu L, Gennetian L, et al. Neighborhoods, Obesity, and Diabetes — A Randomized Social Experiment. New England Journal of Medicine. 2011; 365(16):1509–19. [PubMed: 22010917]

96. Christakis NA, Fowler JH. The Spread of Obesity in a Large Social Network over 32 Years. New England Journal of Medicine. 2007; 357(4):370–9. [PubMed: 17652652]

97. Cohen-Cole E, Fletcher JM. Is obesity contagious? Social networks vs. environmental factors in the obesity epidemic. Journal of Health Economics. 2008; 27(5):1382–7. [PubMed: 18571258]

98. Schaefer DR, Simpkins SD. Using Social Network Analysis to Clarify the Role of Obesity in Selection of Adolescent Friends. American Journal of Public Health. 2014; 104(7):1223–9. [PubMed: 24832139]

99. Ali MM, Amialchuk A, Rizzo JA. The influence of body weight on social network ties among adolescents. Economics & Human Biology. 2012; 10(1):20–34. [PubMed: 22056235]

100. Bahr DB, Browning RC, Wyatt HR, et al. Exploiting Social Networks to Mitigate the Obesity Epidemic. Obesity. 2009; 17(4):723–8. [PubMed: 19148124]

101. 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. Journal of Epidemiology and Community Health. 2011

102. Kumanyika SK, Wadden TA, Shults J, et al. TRial of family and friend support for weight loss in african american adults. Archives of Internal Medicine. 2009; 169(19):1795–804. [PubMed: 19858438]

103. Gorin A, Phelan S, Tate D, et al. Involving Support Partners in Obesity Treatment. Journal of Consulting and Clinical Psychology. 2005; 73(2):341–3. [PubMed: 15796642]

104•. Orr MG, Galea S, Riddle M, et al. Reducing racial disparities in obesity: simulating the effects of improved education and social network influence on diet behavior. Annals of Epidemiology. 2014; 24(8):563–9. This paper uses simulation methods to document the combined importance of social networks and school context for shaping race/ethnic disparities in obesity. [PubMed: 25084700]