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

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Abstract Race/ethnic and socioeconomic status (SES) disparities in obesity are substantial and may widen in the future. We review nine potential mechanisms that recent research has used to explain obesity disparities. Those nine mechanisms fall into three broad groups—health behaviors, biological factors, and the social environment—which incorporate both proximate and upstream determinants of obesity disparities. Efforts to reduce the prevalence of obesity in the US 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 show a continued increase in obesity prevalence [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 USA, relative to other high-income nations [4].

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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≤BMI<30.0) and obesity (BMI≥30.0) among adults. BMI is calculated the same way among children, but the thresholds for overweight (between the 85th and 95th percentiles) and obesity (the 95th percentile or higher) are derived from age- and sex-specific growth charts from the 2000 US 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 US-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 US-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.

SES disparities in obesity are significant and increasing over time. Adults with a college degree have just half 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. Interventions that target diet and physical activity have received substantial attention in the research literature, although Keith and colleagues [10] encourage researchers to explore a broader array of mechanisms. Considering a wider array of social, biological, and behavioral determinants of obesity may offer additional insights into the development and persistence of race/ethnic and SES disparities in obesity [11, 12]. We review nine potential mechanisms that may explain obesity disparities, which fall into three broad categories: health behaviors, biological factors, and social environment. Each mechanism 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 most frequently been the subject of intervention studies. We consider diet and physical activity together as a single mechanism because, along with metabolism, 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 USA, at least among working age adults [15]. 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]. Other research, however, shows that increased snacking could be primarily responsible for the US obesity epidemic [17]. Regardless of the drivers of historical trends in obesity, 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); these race/ethnic disparities 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 are most likely to meet recommendations for dry peas and beans and total grains, and blacks are least likely to meet recommendations for whole fruits, total vegetables, and milk [23, 24].

Food insecurity—marked by limited or uncertain access to safe and nutritious foods—is associated with overweight and obesity in cross-sectional studies. Food insecurity is hypothesized to influence overweight and obesity through

mechanisms including the consumption of inexpensive but calorically dense foods, eating to excess when foods are available, or metabolic changes that encourage the storage of excess calories [25]. Notably, food insecurity is more common among blacks, Hispanics, and those with less income, than among whites and those with more income [26]. Although food insecurity has been linked to insulin resistance [27], longitudinal studies do not find consistent evidence that food insecurity predicts future obesity [26, 28].

Recent population-based analyses suggest the need for significant reductions in caloric intake, or increases in energy expenditure, to meet Healthy People 2020 recommendations [29], with greater reductions in energy balance required with increasing age. Wang and colleagues [14] suggest that a net reduction of 23 kcal/day/capita would be enough to meet the recommendations among children. A more aggressive net reduction of 166 kcal/day/capita would be necessary to meet the recommendations among adults aged 20–39 and 222 kcal/day/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]. Existing diet and physical activity interventions promise modest reductions in obesity or obesity disparities. Meta-analyses find no reductions or small reductions in body mass for interventions that promote healthy diets, reduced calories consumed, and encourage physical activity among adolescents [30, 31]. Among adults, meta-analyses find modest benefits of dietary and physical activity interventions for weight loss [32], although those interventions are generally more effective among whites and Hispanics than among blacks [33, 34].

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 that affect appetite regulation [35, 36]. Second, short sleep duration is linked to the consumption of poorer quality food (including sugary and fatty snacks) and a greater quantity of food [37, 38]. Finally, short sleep is associated with fatigue and reduced physical activity [39]. Notably, declines in average sleep duration in the USA occurred simultaneously with an increasing prevalence of obesity [40]. Indeed, research has identified associations between short sleep, poor sleep quality, and elevated body masses among both children and adults [41, 42].

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 [43–45]. Children who live in families marked by low levels of income and parental education have shorter sleep durations and more sleep problems [46, 47]. Among adults, those who work longer hours or who have lower levels of education, lower family incomes, or less diverse sources of income report shorter sleep hours [44]. However, two recent studies find no evidence that sleep duration mediates race/ethnic or SES differences in obesity [45, 48].

A more promising line of research suggests that the association between sleep duration and body mass varies by race/ethnicity, although results are sometimes inconsistent. Among adults, whereas one study finds no race/ethnic differences in the association between sleep duration and body mass [49], another finds that sleep duration is inversely associated with body mass among Mexican Americans, but not among Cuban Americans or Puerto Ricans [50]. 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 [51•]. Race/ethnic differences in the association between sleep duration and obesity may also 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 [45]. Among boys, sleep duration is unassociated with body mass for blacks but is inversely associated with body mass for whites, Asians, and Hispanics [45]. Sleep duration is a promising new area of study that may eventually lead to interventions that can reduce obesity disparities.

Screen Time and Sedentary Behaviors Sedentary behaviors—including using the computer or watching television—are positively associated with obesity [52]. At first glance, physical activity and sedentary behaviors are simply opposites. 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 [53, 54]. Sedentary behaviors may result in elevated body mass through the displacement of physical activity, by providing additional opportunities for snacking or by disrupting sleep [55, 56]. 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 [52, 57]. 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 [58].

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 [59]. One meta-analysis finds that interventions that reduce sedentary

behaviors are associated with modest reductions in body mass, although combining sedentary behavior interventions with physical activity or diet interventions did not yield additional reductions in body mass [60]. Future research could specifically compare the impact of sedentary behavior interventions on obesity across race/ethnic or SES groups.

Biological Factors

Developmental Perspectives Developmental perspectives emphasize that prenatal, perinatal, and early childhood exposures to adverse environmental conditions tend to manifest in poor health outcomes later in life [61]. For example, obesity and poor dietary practices among pregnant women can increase the odds of obesity among offspring, not just at birth, but across the entire life course. Elevated obesity risks among the children of obese mothers are subsequently transmitted to future generations through a "vicious cycle" of obesity and diabetes [62]. Consistent with this perspective, a recent study of 1116 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 [63•]. Of potentially great importance for future interventions, that 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) fully explained race/ethnic differences in BMI, skinfold thickness, fat mass, and waist circumference at age 7 that were not explained by parental SES or BMI.

Human Genetics A considerable body of scientific evidence has shown that obesity is a heritable condition [64, 65]. Several decades ago, this observation led to the development of the "thrifty gene" hypothesis (TGH) [66], 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., women of West-African biogeographic ancestry [67]) 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. In support of this perspective, research has shown that African American women have lower resting metabolic rates than white women, even after controlling for factors such as fat mass and cardiorespiratory fitness [68].

Despite this and other genetic and anthropological evidence supporting the TGH [69, 70], it has been challenged on several fronts. First, there is substantially more genetic variability within than between race/ethnic groups [71]. 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 [64], these associations are not consistently observed across race/ethnic groups and explain only a small proportion of population variability in body weight [64, 65, 72]. 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 [72]. Thus, to date, it appears that DNA variation provides, at best, an incomplete biological perspective on race/ethnic disparities in obesity and provides little plausible explanation for large and widening SES disparities in obesity.

Epigenetics The importance of prenatal, perinatal, and early childhood conditions that are highlighted by developmental perspectives may be explained by rapidly emerging knowledge about epigenetic mechanisms. The field of epigenetics emphasizes heritable and developmental alterations primarily to the structure of the DNA molecule without altering the DNA code [64, 72]. As Russo et al. [64] 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 (chromatin remodeling). At the post-transcriptional level, a family of small, non-coding RNAs (microRNAs or siRNAs) completes the regulation of gene activity and expression during development or in response to environmental changes" (p. 694). In other words, epigenetic "marks" on DNA can 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 [73]. Presuming that IGF2 hypomethylation affects obesity risks over the life course of these infants, this study provides compelling evidence supporting intergenerational inheritance of obesity through epigenetic mechanisms. Research has also linked perinatal nutrition in humans [74] and parental exposures to toxic chemicals in rats [75, 76] to epigenetic changes that promote obesity.

In summary, the field of epigenetics offers an intriguing biological explanation for obesity disparities among minority and low-SES groups. Moreover, both developmental and epigenetic perspectives strongly emphasize the importance of interventions that target the health and well-being of parents before conception, mothers during all phases of pregnancy, and offspring throughout infancy and early childhood. As shown by Taveras et al. [63•], improving early life conditions

in disadvantaged populations has the potential to drastically reduce disparities in childhood obesity in the US 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 [77, 78]. Neighborhoods are also marked by important race/ethnic and SES inequalities, with non-whites and low-income individuals often living in segregated and isolated communities [79].

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 [80]. Areas where both minority and low-income groups are concentrated are most likely to contain food deserts [81] and have even more limited access to supermarkets than either race or poverty alone would predict [82]. However, evidence that links food deserts and obesity is mixed. In a study based in Pittsburgh, Pennsylvania, researchers find that prices for healthy food are positively associated with obesity, but distance to the nearest supermarket is not [83]. In a nationally representative cohort study, Lee [84] 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 [85] find that greater access to supermarkets is associated with *increased* levels of obesity.

Neighborhoods also vary in their access to built environments (e.g., sidewalks, bike paths, recreational facilities, and parks) that may foster physical activity and help reduce obesity [86, 87]. Neighborhoods with concentrated poverty or numerous minority residents are often marked by the poorest quality built environments [88]. 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 [88]. 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 [89]. 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 [90, 91]. Some research shows that the association between access to physical activity facilities and physical activity is stronger for blacks than for whites [92]. Among Hispanics, access to parks is negatively associated with body mass, although that association only holds for girls [93]. Built environment variables are usually more weakly associated with obesity than are individual-level variables [94]. 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 [95]. Several studies have affirmed associations between neighborhood characteristics and sleep duration. Living in cities, especially larger cities, is associated with shorter sleep durations [96]. 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 [97–99]. Indeed, neighborhood economic disadvantage explains about half of racial disparities in sleep problems [100]. 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 [101, 102], even after adjusting for neighborhood-level measures of racial segregation and obesity prevalence [78]. 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 reduced the odds of obesity and diabetes over the follow-up period [103]. These studies provide compelling evidence that impoverished communities increase the risk of obesity, but more research is needed to clarify the relevant mechanisms.

Social Networks A seminal paper by Christakis and Fowler [104] suggests that networks of classmates, friends, coworkers, or family may inform health behaviors and shape individual risks of obesity. Subsequent research suggests that Christakis and Fowler's findings reflect reverse causality [105], wherein obesity plays an important role in friend selection [106], especially among whites and females [107].

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 [108]. 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 [109]. Network-based interventions have also shown promise. One study of black women [110], and a second study with a more diverse sample [111], 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. Perhaps because parents model behaviors for children, researchers also found that child and parent weight loss is correlated after exposure to a dietary and physical activity intervention [112]. Simulation studies suggest that improving the quality of schools may reduce race disparities in obesity, but only when social networks support those interventions [113••].

Access to Medical Care Race/ethnic minorities and low-income individuals are less likely than whites and those with more income to have access to private or otherwise high-quality health insurance [114]; only time will tell if those disparities close with the implementation of the Affordable Care Act. Disparities in access to medical care may result in disparate access to medical efforts to prevent or treat obesity. Bariatric surgery is an effective technique for reducing obesity, morbid obesity, and conditions that are linked to obesity including cardiovascular disease, metabolic disorders, and sleep disturbances [115]. Population-based studies show that adults who are black, low income, or under-insured are more likely than whites or those with more income or better health insurance to meet the eligibility criteria for bariatric surgery [115]. Nevertheless, adults who are white, have higher incomes, or have private insurance are most likely to *receive* bariatric surgery [116, 117]. Thus, unequal access to bariatric surgery may exacerbate disparities in obesity and the resulting medical conditions [114].

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. 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 USA.

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.

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Compliance with Ethics Guidelines

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.

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