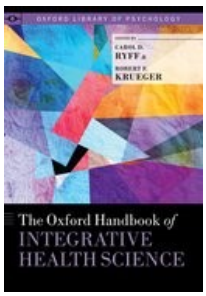

The Oxford Handbook of Integrative Health Science

Carol D. Ryff (ed.), Robert F. Krueger (ed.)

https://doi.org/10.1093/oxfordhb/9780190676384.001.0001

Published online: 09 October 2018 **Published in print:** 29 November 2018 **Online ISBN:** 9780190676407 **Print ISBN:** 9780190676384

CHAPTER

19 Weight Identity Among Older Adults in the United States: Genetic and Environmental Influences

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https://doi.org/10.1093/oxfordhb/9780190676384.013.16 Pages 263–274

Published: 09 October 2018

Abstract

This chapter uses twin pairs from the Midlife in the United States study to investigate the genetic and environmental influences on perceived weight status for midlife adults. The inquiry builds on previous work investigating the same phenomenon in adolescents, and it shows that perceived weight status is not only heritable, but also heritable beyond objective weight. Subjective assessment of physical weight is independent of one's physical weight and described as "weight identity." Importantly, significant differences are shown in the heritability of weight identity among men and women. The chapter ends by discussing the potential relevance of these findings for broader social identity research.

Keywords: twin pairs, Midlife in the United States Study, MIDUS, genetic influences, environmental influences, perceived weight status, midlife adults, BMI, physical weight, weight identity

Subject: Health Psychology, Psychology

Series: Oxford Library of Psychology

Collection: Oxford Handbooks Online

Introduction

The prevalence of obesity among U.S. adults has risen steadily in recent decades (Ogden, Carroll, Kit, & Flegal, 2014). However, a substantial fraction of adults who are classified as overweight or obese according to their body mass index (BMI) do not self-identify as overweight or obese. This mismatch between objective BMI and self-perceived weight status has been observed across all ages for both women and men. To date, explanations for the mismatch primarily focus on underlying demographics (age, sex, gender, race/ethnicity), personality traits, or the effects of social and contextual factors such as school environments or media (Mueller, 2015; Squiers, Renaud, McCormack, Tzeng, Bann, & Williams, 2014; Sutin & Terraciano, 2016). This pattern of mismatch has been observed for other aspects of physical health, and has led to the suggestion that self-reported measures of health may tap into a more complex "health identity" (McMullen & Luborsky, 2006), which could explain why people continue to report the same overall health despite the onset of a new disease, a visit to the hospital, or the worsening of a specific illness (Boardman, 2006). This concept of a "health identity" when applied to physical weight (Grover, Keel, & Mitchell, 2003) has helped to explain why some individuals adhere to a specific "weight identity" even when losing or gaining significant weight.

This is also important because health scientists have long relied on self-assessed health items as valid and reliable indicators of overall well-being (Idler & Benyamini, 1997). These measures have been shown to be at least as valid as physician-assessed morbidity (Ferraro & Farmer, 1999), and self-assessed health is strongly associated with overall mortality risk (Idler & Angel, 1990). In this chapter, we demonstrate that some of the inflexibility of self-assessed weight status is due to unobserved genetic variation in the population. In other words, the likelihood of two otherwise-comparable people change their assessment of their physical weight after gaining 20 pounds depends to some extent on each person's genetic
p. 264 composition. Thus, we suggest that genetic ↳ variation may contribute in some way to our understanding of why individuals who share much in common can perceive or respond differently to the "same" social environments.

Our chapter builds on recent work that links genetic factors with self-perceptions of weight (Wedow, Briley, Short, & Boardman, 2016), as well as to self-assessed health in general (Nes, Røysamb, Tambs, Harris, & Reichborn-Kjennerud, 2006; Okbay et al., 2016). Currently, no work exists that investigates the link between genetics and weight identity among older adults. This omission is important because of its connection to new and highly interdisciplinary approaches to health sciences that link theoretical concepts in the social and biological science, in this case, human agency and heritability, to understand life course health trajectories and late life health.

Our investigation of the heritability of weight identity is informed by a biosocial perspective that highlights that heredity always involves the integration of nature *and* nurture. Variation in heritability reflects both variation in genetic architecture and variation in environments broadly construed. Consider the following two examples: In the first study, using the Midlife in the United States (MIDUS) data, the authors compared the similarity of smoking onset among identical twins and fraternal twins (Boardman, Blalock, & Pampel, 2010). They then evaluated this same question across historical periods to describe the relative contribution of genes to smoking behaviors at different points in US history. They concluded that genes had little effect on smoking behaviors in the early 1960s because smoking was primarily a social phenomenon. However, following the release of the Surgeon General's Report in 1964, the characteristics of smokers changed dramatically, such that the pool of smokers was increasingly comprised of individuals whose smoking was affected by genetic more so than social forces.

In another article using the MIDUS twin sample, Boardman, Blalock, and Button (2008) evaluated genetic contributions to overall positive affect. Using multiple indicators of self-reported affect over the past 30

days, they constructed an indicator of positive affect and estimated its heritability at 0.60 for both men and women. They then evaluated the number of stressful life events and chronic stressors to which the respondents were exposed and calculated the same heritability estimate after considering stress exposure. In this way, they did not assess positive affect per se, but rather psychological resilience. They demonstrated genetic influences on resilience but calculated a heritability estimate of 0.52 for men but only 0.38 for women. The difference in heritability for men and women suggests that social experiences and identities connected to gender are critical to understanding how and when genes might affect individual characteristics. Further, the heritability of self-reported positive affect, though shaped by social environment, provides evidence that subjective health assessments or identities may well reflect genetic contribution.

In this chapter, we utilize data from a large study of adult twin pairs to extend research on health identity in two important ways. First, we focus explicitly on physical weight rather than overall health per se. In doing so, we build on complementary work that has detailed the contours of "weight identity" (Grover et al., 2003) rather than the broader notion of "health identity." Second, we decompose the variance of weight identity into environmental and genetic components in order to better describe the sources of weight identity in the population. Our study replicates previous work in this area (Wedow, Briley, Short, & Boardman, 2016) but extends this work by illustrating the important role that genetic variance plays with respect to health identity among older adults.

Background Literature on Genetics and Identity

The results of recent meta-analyses suggest that the heritability of subjective well-being is between 30% and 50% (Nes, Røysamb, Tambs, Harris, & Reichborn-Kjennerud, 2006; Okbay et al., 2016). That is, roughly one third to one half of the variance of subjective well-being in the population may be due to genetic variation. Yet, the idea that health identity reflects a genetic component is rarely considered in the social sciences. Our study addresses this limitation by ascertaining the proportion of variance in weight identity that may be genetically influenced. Researchers have made gains in their efforts to assess the genetic origins of environmental sensitivity (Belsky & Pluess, 2009; Boyce & Ellis, 2005; Shanahan & Hofer, 2005), but very little work has considered health identities, such as weight identity, within this overall framework. This is a significant omission because those who are less flexible in their health or weight identity may be less likely than others to make significant changes in order to improve or maintain their health (Boardman et al., 2011, 2012). By studying both objective BMI and one's own perceived weight ↳ status, we examine the p. 265 possibilities that the covariation between these traits is due in part to common genetic influences, and more importantly, that unique genetic influences contribute to subjective weight identity. Stated more simply, we explore the idea that some of the tendency to be open to change in weight self-assessment may have roots in observed or unobserved genotype.

Social scientists have long shown that one's current health, one's health lifestyle, and one's access to healthcare interact to shape self-rated health assessments (Kelleher & Leavey, 2004). These relatively proximate factors give rise to the understanding that self-rated health captures a "spontaneous assessment" (Bailis, Segall, & Chipperfield, 2003) of one's health. That is, a large component of health self-assessment simply taps into how an individual feels at a particular time, how that individual describes their health to those around them and that individual's recent interactions with physicians (Altman, Van Hook, & Hillemeier, 2016; McMullen & Luborsky, 2006). But, as others have shown (Boardman, 2006), regardless of current health status, changes in health, or new information from doctors, some individuals continue to report the same level of overall health. Bailis et al. (2003) referred to this aspect of self-rated health as an "enduring self-concept." It is this understanding of self-rated health that is particularly important to social epidemiologists because previous work has shown that people who are relatively pessimistic about their

health are also less likely to adhere to physicians' recommendations or to follow-up with physicians despite explicit requests to do so (Idler & Benyamini, 1997).

This same understanding can be applied to an individual's self-assessed weight status (e.g., "overweight," "normal weight," or "underweight"), the most important component of which is physical weight. However, physical weight is not the only determining factor. For example, two similar size individuals, both of whom consider themselves to be "normal weight," may gain the same amount of weight over the same period of time. Yet, the result may very well be that one individual changes their assessment of their own weight from normal weight to "overweight," while the other individual may continue to self-assess as normal weight. The tendency to adhere to a particular weight identity in the United States is likely related to the unusually strong social forces that frame what a "healthy" body should look like and promote fat-stigmatizing beliefs. These forces, which are internalized by many, may contribute to why individuals report that those who are overweight are less attractive (Cross et al., 2017), less intelligent (Robinson & Christiansen, 2014), and of lower social and economic status (Vartanian & Silverstein, 2013) compared to individuals who are "normal" weight. That is, this work suggests that variation in the social environmental factors connected to morally laden stereotypes about "good" and "bad" weights accounts for most of the variation in weight identity.

This overall focus on weight identity is particularly important during midlife because many overweight and obese adults perceive that they are discriminated against because of their physical weight. Carr and Friedman (2005) showed that individuals with a BMI exceeding 35 (e.g., Obese II/III classes) are more likely than others to have experienced discrimination at work, by healthcare providers, and in their day-to-day activities. Specifically, 31% of Obese II/III adults experienced discrimination at the workplace, compared to 21% of normal weight adults. They argued that these multiple forms of discrimination have a strong effect on obese adult's self-acceptance that may independently contribute to other physical and mental health problems in the future. Yet, discrimination and hostility in the environment may be a key mechanism through which weight identity is shaped and reinforced over time. They also showed that the link between BMI and discrimination is not evident among upper white-collar workers, which demonstrates that the social context in which one experiences obesity is an important determinant of the way in which one is treated by others. In sum, it is important to consider the multiple environmental factors that shape weight identity at different stages of the life course.

However, it is also possible that some of the variation in weight identity reflects genetic variation. This understanding builds on the work of Nes, Røysamb, Tambs, Harris, & Reichborn-Kjennerud (2006), who showed that roughly one third to one half of variation in overall subjective well-being may be due to genetic variation. Further, more recent work has identified specific genetic polymorphisms that predict self-rated health (Harris et al., 2017). Global descriptions of overall genetic influence are referred to as *heritability estimates*, and they provide important clues about specific health outcomes that may be strongly (or weakly) influenced by genes. For instance, Boardman, Domingue, and Daw (2015) used genome-wide data from the
p. 266 Health and ↳ Retirement Study to calculate a heritability estimate of 0.20 for self-rated health. Taken together, these results suggest, regardless of the sample or the statistical method, consistent evidence for genetic contributions to health identity.

In a recent article (Wedow, Briley, Short, & Boardman, 2016), it was shown that BMI *and* perceived weight status were both influenced significantly by genes. Specifically, they estimated heritability values for BMI of roughly 0.80 and roughly 0.45 for perceived weight status. This suggests that nearly 80% and 45% of the variation in BMI and perceived weight, respectively, are influenced by genetic variation in the population. The discrepancy of these estimates is worth noting because it is clear that the genetic influences of physical weight, which have many well-established physiological pathways, far exceed those of perceived weight. This does not mean that the genetics underlying perceived weight are not important; rather, it highlights that this phenotype comprises very different determinants than those linked to physical health.

Importantly, Wedow et al., (2016) also assessed the contribution of genetic variation to perceived weight status that is above and beyond BMI and showed that roughly 25% of weight identity is influenced by unmeasured genotype. Further, evaluations of this association among adolescents, young adults, and adults yielded very similar estimates across groups. Others have shown that teenagers are more likely than adults to hold a rigid health self-assessment despite changes in objective health because of the significance of maintaining a constant identity in light of the massive physical, environmental, and social changes that occur during adolescence (Boardman, 2006). However, that the genetic contribution to weight identity is constant across adolescence through adulthood is a strong indication that this process cannot be accounted for by the typical developmental trajectories of teenagers entering adulthood. The goal of this article was to evaluate the same models using different data among a significantly older group of adults (mean age = 46.39 in Wave 1).

Focusing on older adults is important because recent findings suggested that the effects of weight stigma and discrimination based on one's weight are stronger at younger ages (Frisco, Houle, & Martin, 2010; Van Hook & Baker, 2010). Patterns of weight discrimination show that adolescents and young adults are more likely to be discriminated against for their weight than are older persons (Puhl, Andreyeva, & Brownell, 2008). In many ways, these findings are intuitive because young people's normative reference groups are likely to change as they age and experience new and more diverse social contexts (Moen & Hernandez, 2009; Neugarten, Moore, & Lowe, 1965; Settersten, 2003). Yet, as discussed previously, research has shown strong evidence that weight-related discrimination continues throughout the life course and remains an important risk for health and well-being of obese adults (Carr & Friedman, 2005).

Some have shown important biological processes that are implicated in this process. Consider for example the work of Tsenkova, Carr, Schoeller, & Ryff (2011), who used the biomarker samples from the MIDUS 2 study to show how the experience of discrimination exacerbates the health consequences of waist-to-hip ratio. Specifically, they showed a strong link between waist-to-hip ratio and an indicator of glycemic control (with HbA_{1C}); this effect was nearly twice as high among those who perceived that they were discriminated against because of their weight. It is therefore important to consider that sensitivity to perceptions of weight-related discrimination may also be linked to fairly static foundations in weight identity. If genetic variants are associated with weight and weight identity, which may change across the life course, then the interaction of genotype and environment needs to be considered in this important line of research. Our chapter is specifically designed to explore this question.

Weight Identity in the MIDUS Study: Findings for Midlife Adults

To study the genetic contributions to weight identity, we used the twin subsample from the MIDUS study. The first national survey of MIDUS was conducted in 1995–1996 by the MacArthur Foundation Research Network on Successful Midlife Development. The study was the result of a large collaboration of researchers across numerous disciplines, including psychology, sociology, epidemiology, demography, anthropology, medicine, and healthcare policy. The goal was to evaluate the interactive roles of behavioral, psychological, and social factors as determinants of health for adults between the ages of 35 and 85. The study included a national probability sample (N = 3,487) and oversamples for a small number of cities, a sample of siblings (N = 950) from the main respondents, and most importantly for our purposes, a national sample of twin pairs (N = 1,914). ↳

Twins in the Present Analyses

We use monozygotic (MZ) and dizygotic (DZ) twin pairs in our analyses. In our final univariate and bivariate twin analyses, we used a total of 356 MZ pairs, 331 same-sex DZ pairs, and 249 opposite-sex DZ pairs. Weight, controlling for height, was measured in pounds and was assessed at all three waves of MIDUS. Perceived weight status was assessed at each wave with the following response categories using the question, "Which of the following do you consider yourself?" (1) "very underweight," (2) "somewhat underweight," (3) "about the right weight," (4) "somewhat overweight," or (5) "very overweight."

Using twin modeling techniques in the structural equation modeling programs M*plus* 7.4 (Muthén & Muthén, 1998–2015) and *OpenMx* (Boker et al., 2011) with the R 3.2.0 statistical program (R Core Team, 2016), we provided estimates for the genetic component (additive A) and two components of environmental influence (shared C and nonshared E) of our objective weight and perceived weight status phenotypes. Our models controlled for the main effects of age, sex, age^2, and an age × sex interaction, as is standard in twin models (McGue & Bouchard, 1984). We also controlled for height. We compared nested models using χ^2 difference tests to assess fit and arrive at the most parsimonious models. We estimated univariate models for each of the three waves of MIDUS and provided standard errors for our parameter estimates.

We next fit bivariate twin models, or Cholesky triangular decomposition models, to 4 × 4 covariance matrices. Here, we used the most parsimonious models from our univariate analyses to inform the models we fit in our bivariate approach. In these models, we decomposed the variance–covariance matrix with regression of observed measures on latent factors in which we modelled a variance–covariance path that approximates the variance shared between our two phenotypes: objective and perceived weight (Purcell, 2008). As shown by diagramming the bivariate path model in Figure 19.1, in these models objective weight and perceived weight status were observed. A, C, and E are the latent factors of additive genetic variation, shared environmental variation, and nonshared environmental variation, respectively. The a_{11} and a_{22} paths represent the additive genetic variation associated with objective weight and *residualized* perceived weight status, respectively. Likewise, the c_{11}, c_{22}, e_{11}, and e_{22} paths represent the environmental variation associated with objective weight and *residualized* perceived weight status, respectively. Put differently, the pathways specific to perceived weight status represent unique genetic and environmental influences that are not shared with objective weight. Unique to the bivariate models, the covariance paths (a_{21}, c_{21}, and e_{21}) represent genetic and environmental variation *shared* between objective weight and perceived weight status, and these paths can be interpreted similarly to regression coefficients. When squared, the coefficients represent the proportion of variance in perceived weight status that is shared with objective weight.

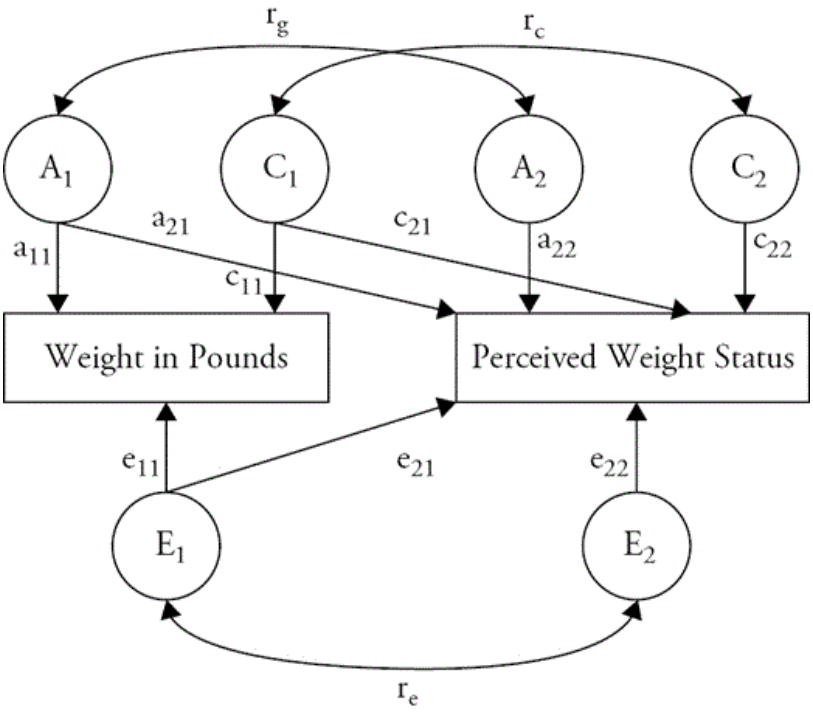

Figure 19.1 ACE bivariate path model for objective weight and perceived weight status.

These shared paths also allow us to estimate a genetic correlation coefficient r_g that indicates the extent to which latent factor variation (e.g., genetic influence) is shared between our two phenotypes. The paths described in Figure 19.1 can be used with Equation 19.1 to estimate the genetic correlation coefficient as

$$r_g = \frac{a_{21}}{\sqrt{(a_{21}^2 + a_{22}^2)}} \tag{19.1}$$

Thus, in these models we can get a sense of the amount of genetic variation that is influencing both objective weight and perceived weight status at the same time. We estimated bivariate models across all three waves of MIDUS and provided standard errors for our r_g estimates.

Results

p. 268 In Table 19.1, we report the twin pair descriptive statistics for our outcome phenotypes, objective ↳ weight and perceived weight status. Weight increased as twins aged from Waves 1 to 2, but mean levels changed only slightly from Waves 2 to 3. Mean weight in pounds ranged from 171.13 to 178.57 across waves. Mean perceived weight status (on a 5-point Likert scale) was quite consistent across waves and ranged from 3.72 to 3.37, or from *about the right weight* to *slightly overweight* (concentrated toward *slightly overweight*). Table 19.1 also reports the number of MZ, same-sex DZ, and opposite-sex DZ twin pairs used for each phenotype by wave. Finally, we report MZ, same-sex DZ, and opposite-sex DZ phenotypic correlations for each trait by wave. For objective weight, phenotypic correlations across waves were roughly .7 for MZ pairs and about .4 for same-sex DZ pairs. For perceived weight status, phenotypic correlations ranged from .29 to .58 across waves for MZ pairs and from .12 to .29 for same-sex DZ twin pairs. That the same-sex DZ correlations were on average about half the MZ correlations across waves for each of our phenotypes suggests that genetic influences are additive (Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990). To check this observation, we procedurally fit a model for nonadditive (dominance) genetic effects, and we found estimates of zero for the dominance genetic parameter, providing further evidence that an additive genetic model is the most appropriate.

Table 19.1 The MIDUS Waves 1–3 Twin Pair Descriptive Statistics for Objective Weight and Perceived Weight Status

Twin Wave	Objective Weight							
	Mean	***SD***	N_{MZ}	$N_{SS\ DZ}$	$N_{OS\ DZ}$	r_{MZ}	$r_{SS\ DZ}$	$r_{OS\ DZ}$
Wave 1	171.13	39.49	323	299	218	.77	.41	.20
Wave 2	178.43	42.16	217	218	141	.67	.36	.21
Wave 3	178.57	42.79	144	153	97	0.69	.37	.18

Twin Wave	Perceived Weight Status							
	Mean	***SD***	N_{MZ}	$N_{SS\ DZ}$	$N_{OS\ DZ}$	r_{MZ}	$r_{SS\ DZ}$	$r_{OS\ DZ}$
Wave 1	3.72	0.75	315	295	218	.47	.29	.13
Wave 2	3.79	0.73	218	217	142	.29	.12	.06
Wave 3	3.75	0.76	146	154	98	.58	.27	.17

Note: SD = Standard Deviation; N_{MZ} = Number of MZ twins; $N_{SS\ DZ}$ = Number of Same-Sex DZ twins; $N_{OS\ DZ}$ = Number of Opposite-Sex DZ twins; r_{MZ} = MZ twins correlations; $r_{SS\ DZ}$ = Same-Sex DZ twin correlations; $r_{OS\ DZ}$ = Opposite-Sex DZ twin correlations.

Table 19.2 presents the results of our univariate models. The final three columns in Table 19.2 indicate model fit statistics for the ACE versus the AE model. Because we were able to drop C from all of our univariate models without loss of model fit (last column in Table 19.2), we only present the results of the AE models in Table 19.2. Heritability for objective weight remained quite stable across all waves at about 0.73 with the results for adolescents reported previously (Wedow, Briley, Short, & Boardman, 2016; Haberstick et al., 2010). Even with our somewhat small twin sample, standard errors remain small. For perceived weight

status, heritability values show slightly more movement across waves, from about 0.31 to 0.54 (mean is 0.44), suggesting that perceived weight status is more heavily influenced by environmental factors than is objective weight. In both phenotypes, we see significant genetic influence across all three waves of MIDUS.

We present our bivariate results in Table 19.3 for all respondents. In the first several columns, we present standard errors and path coefficients for both the additive genetic [A] and nonshared environmental [E] components of our model.

Next, using the path coefficient-based Equations 19.2 and 19.3, we calculated heritabilities for objective weight and perceived weight status (coefficients found in Table 19.3):

$$h^2_{Objective\,Weight} = \frac{a^2_{11}}{a^2_{11}+e^2_{11}} \tag{19.2}$$

$$h^2_{Perceived\,Weight\,Status} = \frac{a^2_{21}+a^2_{22}}{a^2_{21}+e^2_{21}+a^2_{22}+e^2_{22}} \tag{19.3}$$

p. 269 **Table 19.2** Univariate Twin AE Parameter Estimates for Objective Weight and Perceived Weight Status

	Objective Weight				
	Variance Components		**Fit Statistics (χ^2)**		
	A	**E**	**AE**	**ACE**	**pr. <**
Wave 1	0.78	0.22	18.77	18.77	1.00
	(0.02)	(0.02)			
Wave 2	0.70	0.30	15.15	14.83	0.57
	(0.03)	(0.03)			
Wave 3	0.71	0.29	14.41	14.05	0.55
	(0.04)	(0.04)			

	Perceived Weight Status				
	Variance Components		**Fit Statistics (χ^2)**		
	A	**E**	**AE**	**ACE**	**pr. <**
Wave 1	0.48	0.52	3.87	3.87	1.00
	(0.04)	(0.04)			
Wave 2	0.31	0.69	4.44	4.44	1.00
	(0.06)	(0.06)			
Wave 3	0.54	0.46	13.74	13.74	1.00
	(0.07)	(0.07)			

Note: Standard errors for variance estimates provided in parentheses.

We found that these estimates were directly in line with the estimates from our univariate models in Table 19.2, which served as a robustness check for our twin methods. With the Cholesky bivariate method, the shared variance–covariance paths (a_{21} and e_{21}) allow for the determination of variance that is unique to perceived weight status, *independent* of objective weight. We used Equation 19.4 to calculate this residual heritability estimate:

$$h^2_{Unique\ Perceived\ Weight\ Status} = \frac{a^2_{22}}{a^2_{22}+e^2_{22}} \tag{19.4}$$

As reported in Table 19.3, we found heritability estimates for this residual phenotype of 0.30, 0.11, and 0.37 across each of our three waves, respectively. Importantly, these estimates indicated that perceived weight status contains a genetic component that is above and beyond either objective weight or environmental influences. Though Wave 2 results indicated a slightly decreased heritability estimate when compared to

Waves 1 and 3, overall the results indicated relatively consistent estimates of heritability for weight identity, and these results are highly comparable to results found for adolescents and young adults (Wedow, Briley, Short, & Boardman, 2016; about 0.25 across waves). In Figure 19.2, we graphically decompose our bivariate results, splitting genetic and environmental variance into variance shared between objective weight and perceived weight and variance unique to each of these phenotypes. The light gray portion of the bars in this figure correspond to genetic variance unique to perceived weight status.

Across each of the three waves, our estimates of genetic correlation r_g remained stable between .77 and .90, with small and consistent standard errors. These r_g values provide strong evidence that there is a substantial
p. 270 overlap between the genetic variance that is associated with weight and the genetic ↳ variance that is associated with perceived weight status. These results, in conjunction with the heritability results for the residualized weight identity phenotype, support the notion that the heritability of one component of weight identity operates above and beyond simple changes in physical weight.

Table 19.3 Bivariate AE Estimates for Objective Weight and Perceived Weight Status Among All Pairs

	Path Coefficients						**Heritability Estimates**			**Genetic Correlation Estimates**	
	$\mathbf{a}_{11}$	$\mathbf{e}_{11}$	$\mathbf{a}_{21}$	$\mathbf{e}_{21}$	$\mathbf{a}_{22}$	$\mathbf{e}_{22}$	**Weight**	**Perceived Weight**	**Unique Perceived Weight**	$\mathbf{r}_g$	$\mathbf{r}_e$
Wave 1	0.89	0.46	0.56	0.35	0.41	0.63	0.79	0.48	0.3	.81	.48
	(0.01)	(0.02)	(0.03)	(0.04)	(0.04)	(0.02)				(0.03)	(0.04)
Wave 2	0.84	0.55	0.5	0.44	0.25	0.7	0.7	0.31	0.11	.9	.53
	(0.02)	(0.03)	(0.04)	(0.05)	(0.08)	(0.03)				(0.06)	(0.05)
Wave 3	0.84	0.54	0.53	0.43	0.44	0.58	0.71	0.48	0.37	.77	.60
	(0.02)	(0.04)	(0.05)	(0.07)	(0.06)	(0.04)				(0.05)	(0.07)

Note: Standard errors provided in parentheses below parameter estimates.

Discussion and Future Directions

To date, little research has conceptualized genetics as contributing to identities and even less has extended this investigation to health identities. A place for genetics in health identity formation is not at odds with the reality that identities can and do change. Indeed, social science paradigms are built around the idea that not all individuals respond to the same environments in the same way (Mead, 1934). We propose that genetic factors may impact subjective health assessments in general and weight and health identities in particular. In weight-related health research, our work is of particular importance because how one feels about one's body is determined in part by one's social context (Qvortrup, 2010), and these factors ultimately play a role in how individuals manage their own weight (Levine, ↳ Smolak, & Hayden, 1994). This work is critical because it suggests that genetic contributions to weight identity may be contingent on other social identities or social contexts.

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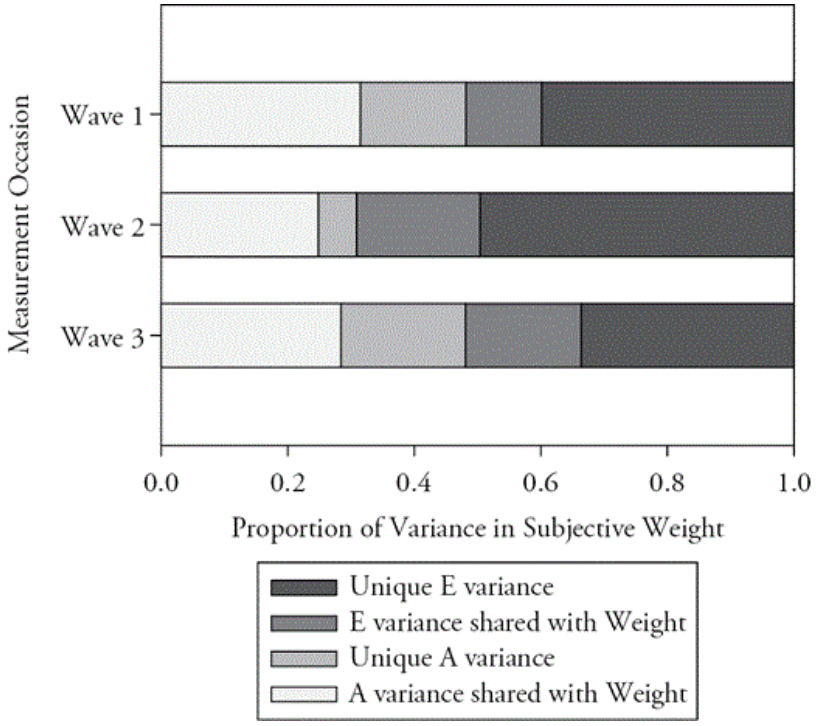

Figure 19.2 Variance decomposition of bivariate models for all respondents by wave. Genetic and nonshared environmental variance is split into variance shared between objective weight and perceived weight status and variance unique to perceived weight status.

For example, Wedow, Briley, Short, & Boardman (2016) showed that the genetic contributions to weight identity are significantly higher for women compared to men. They argued that the undue focus on women's bodies gives rise to heightened awareness of body size among women, and that such an environment effectively makes genetic variation related to weight identity more relevant. This explanation highlights the importance of gendered environments for theorizing genetic contributions to health (Short, Yang, & Jenkin, 2013) and is more generally in line with the "social trigger" gene–environment interaction hypothesis, in which genotype may provide insights about environmental sensitivity (Belsky & Pluess, 2009), though the primary driving force remains the social and cultural context that defines the environment.

Beyond gender, it is important to consider the broad social historical context in which individuals were socialized about a "healthy body size." We did not focus on age in our article. Age would be an indicator for

the historical period in which people attended schools and learned to model healthy lifestyles, including nutritional intake and physical activities, the two key components of physical size. It is also possible that the relative contribution of genetics to weight identity changes as a function of historical period. As such, we encourage future researchers to consider environmental triggers or suppressors across the different social contexts in which individuals live, work, and socialize. These contexts should be characterized as multidimensional, multilevel, and longitudinal (Boardman, Daw, & Freese, 2013) in order to describe environmental *and* genetic contributions to weight identity.

Methodologically, we used a behavioral genetics approach in our investigation of weight identity. However, we encourage future researchers to use genome-wide techniques to decompose this trait into genetic and environmental components. These new methods have emerged because of criticisms of the twin-based methods described in this chapter. These criticisms have emerged because the traits that have been analyzed have become increasingly distal from the genes that may ultimately be responsible for the trait. For example, the statement that "height depends on genes" is straightforward and one that most people would not dispute. However, the extension of the logic "heritability" to other individual characteristics, such as political ideology, happiness, or popularity, is less so. For these traits, many studies have relied on twin and sibling samples to derive heritability estimates, and while the underlying model's assumptions largely hold (Conley, Rauscher, Dawes, Magnusson, & Siegal, 2013), the results from twin studies have been called into question.

As a result, researchers have increasingly turned to molecular genetic data about genetic similarity among unrelated individuals (Yang, Lee, Goddard, & Visscher, 2011) to reduce the challenges tied to the assumptions regarding average relatedness among twins and siblings. This specific method (genome-wide complex trait analysis, GCTA) provides estimates of genetic contribution to traits that are substantively consistent across the behavioral genetic and molecular genetic approaches. Even though conclusions about the significance of genetic contribution to traits are largely consistent across behavioral genetic and molecular genetic approaches, patterned differences do emerge. The GCTA estimates are consistently lower than twin-based results. This has been discussed at great length elsewhere (Plomin & Deary, 2015). The difference is that GCTA relies on measured polymorphisms with a frequency of at least 1% in the population so that rare alleles, which may contribute importantly to a trait's value, are not included. These models specify an additive effect of each genetic polymorphism. In other words, they do not allow the effect of a polymorphism in one gene to depend on the genotype in another gene (which is called *epistasis*). They also do not incorporate gene–environment interaction effects, which are subsumed within the heritability estimate of twin studies because of the equal environment assumption. As Plomin and Deary (2015) made clear: "GCTA heritability represents the upper limit for detection of SNP [single-nucleotide polymorphism] associations in GWAS [genome-wide association] studies, which, like GCTA, are limited to detecting additive effects of common SNPs. Conversely, GCTA heritability represents the lower limit for heritability estimated in twin studies because twin studies can detect genetic influence due to DNA variants of any kind." We encourage researchers to utilize these same techniques to reproduce our estimates for the heritability of weight identity.

Finally, while not the central focus of this chapter, we consider the potential significance of our findings beyond weight identity. Identities in general denote a foundational concept in the social sciences because of
p. 272 the notion of *social* identities (Tajfel 2010). That is, an individual's overall concept ↳ of self guides their perceptions of membership in multiple social identities. These social identities may be defined by characteristics associated with gender, race, social class, religious organizations, neighborhood participation, and so on. The most important aspect of social identities is that they are imbued with a sense of status and power that is critical for understanding not only how groups interact internally, but also how different social groups interact with one another. Thus, a better understanding of how and why individuals

claim the identities that they do as they move through the social world is central to many social scientific questions, and may have implications well beyond those associated with health or weight.

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