

1 CHAPTER 6

2 THE PSYCHOLOGY
3 OF OBESITY4 ASHLEY MOSKOVICH, JEFFREY
5 HUNGER, AND TRACI MANN

6 The field of psychology traditionally focuses on the study of individuals, their
7 internal mental processes, and their behavior; as such, it has adopted this approach
8 in the study of obesity. Rather than examining larger groups, such as communities,
9 or distal factors, such as laws, policies, or environmental barriers, psychologists
10 have explored people's emotions, beliefs, goals, and behaviors as causes and conse-
11 quences of obesity. In recent years, however, and paralleling the direction of the
12 field as a whole, psychologists have broadened their focus to include the influence
13 of relationships and social networks on obesity, as well as the role of genetic
14 and neurological factors in the development of obesity. With regard to treatment,
15 psychologists have focused primarily on behavioral rather than medical interven-
16 tions, and recently have begun to explore whether weight loss is a reasonable or
17 even necessary goal in treatments for obesity.

18 CAUSES OF OBESITY

19 A substantial body of work in psychology has been dedicated to exploring the causes
20 of obesity. Psychology originally viewed obesity as a result of psychopathology or as
21 a response to a significant trauma in a person's past (Kolata 2007). However, sup-
22 port for these assumptions has been mixed, and these explanations are not thought

1 to account for most cases of obesity (Stunkard and Wadden 1992). Rather, the focus
 2 has been on individual factors such as thoughts, behaviors, and biology, as well as
 3 aspects of the individual's social environment such as familial and peer influences.
 4 Many early theories of obesity continue to be refined and incorporated into current
 5 etiological models.

6 The Individual

7 In 1957 it was proposed that obesity was the result of overeating—a behavioral fac-
 8 tor—and was not exclusively due to impaired metabolic functioning (Kaplan and
 9 Kaplan 1957). Much of the work within the field has since focused on behavioral,
 10 rather than biological, causes of obesity, and on overeating in particular. Early
 11 research focused on differences in eating between obese and non-obese individuals,
 12 but over time it became clear that many of the factors that promote overeating in
 13 obese individuals also promote overeating in some non-obese individuals. Interest
 14 has not only focused on determining *who* overeats, but also *when* and *why* overeating
 15 occurs. Four major individual factors have been explored as causes of overeating:
 16 interoceptive awareness (i.e., sensitivity to internal bodily states), response to
 17 emotional experience, cognition, and biology.

18 Schacter's internal-external theory of eating holds that different factors guide
 19 eating for obese individuals from those that guide eating for non-obese individuals
 20 (Schacter 1968; Schacter, Goldman, and Gordon 1968). According to this theory,
 21 non-obese individuals use the internal sensations of hunger and satiety to guide
 22 their eating, whereas obese individuals are less interoceptively aware and instead
 23 rely on external cues such as the time of day or the presence of appetizing foods to
 24 regulate eating. Although this theory is intuitive and parsimonious, original sup-
 25 port for it was mixed and its basic premises have since been questioned (Rodin
 26 1981). In particular, some have challenged the assertion that an overreliance on
 27 external cues by obese individuals necessarily indicates that they are less interocep-
 28 tively aware than non-obese individuals (Rodin 1981; Herman and Polivy 2008).
 29 Furthermore, it has also been argued that non-obese individuals are not better than
 30 obese people at using internal cues to regulate their eating, and that they are also
 31 influenced by external cues (Rodin 1981; Herman and Polivy 2008).

32 Although it has been challenged, the internal-external hypothesis has not been
 33 abandoned; rather, recent work has instead been expanding upon it. For example,
 34 Herman and Polivy (2008) posit that the conflicting findings from past research
 35 actually demonstrate a clear pattern of eating behavior. They argue that some types
 36 of external cues (e.g., portion size), which the authors refer to as *normative* cues,
 37 influence everyone, whereas other cues (e.g., palatability), called *sensory cues*, only
 38 impact the eating of obese and dieting individuals. Thus the internal-external
 39 hypothesis is still present in the way in which we view the causes of obesity.

40 Differences in the way individuals respond to emotional experience is another
 41 factor that is thought to play a role in the development of obesity. According to the
 42 psychosomatic hypothesis (Kaplan and Kaplan 1957), obese people overeat as a way

1 of coping with emotional distress. Obese individuals are thought to have learned
2 that eating reduces feelings of anxiety and therefore overeat as a way to self-soothe.
3 This results in a continuous cycle in which overeating aimed at reducing distress
4 actually promotes further distress and subsequent overeating (Kaplan and Kaplan
5 1957). Similarly, and foreshadowing the internal-external hypothesis, Bruch theo-
6 rized that obese individuals have difficulties with interoceptive awareness and
7 therefore misinterpret emotional states as hunger (Bruch 1961).

8 Although these early theories have received only mixed support (Greeno and
9 Wing 1994), eating in response to emotional experience is still viewed as an impor-
10 tant contributor to overeating and obesity (Canetti, Bachar, and Berry 2002).
11 Research shows that some individuals, not exclusively obese individuals, exhibit a
12 tendency to overeat when in the presence of strong emotion, particularly negative
13 affect (e.g., Van Strien and Ouwens 2003; Polivy, Herman, and McFarlane 1994).
14 While emotional eating is well documented, it is still unclear *why* such behavior
15 occurs. Many studies have been unable to demonstrate that emotional eating atten-
16 uates negative affect (i.e., negative emotion states such as anxiety) (e.g., Herman
17 and Polivy 1975). Recent work, however, found that eating palatable food improved
18 mood among individuals classified as emotional eaters (Macht and Mueller 2007).
19 Replication of this effect and clarification of moderators and mechanisms will be
20 essential to this growing body of work.

21 The third individual factor thought to contribute to overeating, cognitive con-
22 trol, was introduced in a study by Herman and Mack (1975). This study highlighted
23 what has been termed the *disinhibition effect*—the phenomenon that restrained
24 eaters, or dieters, engage in overeating after a perceived diet violation. This phe-
25 nomenon is considered a cognitive one (as opposed to a physiological one, for
26 example) because what matters is not whether restrained eaters actually violated
27 their diets, but whether they *think* that they did. Research supporting the role of
28 cognition shows that restrained eaters will overeat if they believe they have violated
29 their diet when in fact they did not, but they will not overeat after an actual diet
30 violation if they are led to believe that the food they consumed did not actually
31 violate their diet (Polivy and Herman 1985).

32 An individual's self-imposed restraint essentially functions as a form of self-
33 control and is therefore susceptible to a broad range of factors known to contribute
34 to regulatory failures. These factors, such as ego threats (Heatherton, Herman, and
35 Polivy 1991), stress (Greeno and Wing 1994), and distraction (Mann and Ward
36 2000), have been shown to lead to overeating among restrained eaters, but not
37 among non-restrained individuals. Although restraint status has proven to be a
38 reliable indicator of overeating and has generated a considerable amount of research,
39 there is controversy over how it is best conceptualized and measured (Ruderman
40 1986); many believe that restraint only leads to self-regulation failure among
41 individuals who are also disinhibited eaters.

42 The fourth factor that has been explored as a source of overeating is individual
43 differences in biology. In an effort to explain discrepant results of externally based
44 eating behavior among obese individuals, Nisbett proposed a biological explanation

1 known as set-point theory (Nisbett 1972). According to Nisbett, everyone has
2 a biologically determined and physiologically defended weight set-point that ulti-
3 mately guides eating behavior. Regardless of current weight status, obese and non-
4 obese individuals may at times be below their actual set-point, causing them to be
5 in a state of deprivation that promotes externally guided eating. When individuals
6 are at their set-point, deprivation ceases and externally guided eating diminishes.
7 Set-point theories suggest that biology is a key predictor of obesity, but they
8 are impossible to prove or disprove and cannot account for a variety of eating phe-
9 nomenon, ultimately limiting their utility (Pinel, Assanand, and Lehman 2000).

10 Current research is examining how underlying differences in neurobiology
11 guide eating behavior. For example, there are promising results in neuroimaging
12 research on impulsivity, craving, binge eating, and restraint (Lowe et al. 2009). Such
13 work may help explain conflicting results found in behavioral research and may
14 provide necessary insight into the transactional relationship between internal and
15 external variables.

16 The Environment

17 In addition to individual variables that contribute to overeating and the development
18 of obesity, psychologists also focus on environmental causes, including familial and
19 social influences.

20 Because obese children are likely to become obese adults (Epstein, Wing, and
21 Valoski 1985), factors associated with the development of pediatric obesity, includ-
22 ing the family environment, are the focus of much research. Parents affect the eating
23 of their children both directly and indirectly. Parental influence, whether by model-
24 ing eating behavior, directly controlling their children's eating behavior, or provid-
25 ing feedback regarding the child's weight and shape, has been shown to foster
26 restrained and disinhibited eating in children (e.g., Cutting et al. 1999; Pike and
27 Rodin 1991; Carper, Fisher, and Birch 2000; Francis and Birch 2005). A restrained
28 eating style, as mentioned above, promotes vulnerability to disinhibition and over-
29 eating, increasing the likelihood that obesity will result.

30 Preliminary evidence suggests that chronic levels of psychological stress in the
31 family are related to obesity in children (Koch, Sepa, and Ludvigsson 2008).
32 Psychological stress encountered in childhood may lead to the development of an
33 emotional eating style. A recent study conducted by Greenfield and Marks (2009)
34 found that individuals who reported experiencing physical and psychological vio-
35 lence from their parents when they were children had a greater risk for adult obesity
36 than those who had not experienced such violence. Moreover, the association
37 between a past history of physical and psychological violence and obesity was par-
38 tially mediated by the use of food as a coping response to stress. Family stress levels,
39 like parental influences over eating, appear to contribute to obesity by fostering
40 individual behaviors that are associated with overeating.

41 In addition to familial influences on overeating and obesity, researchers have
42 also looked at other social influences. A recent study by Christakis and Fowler (2007)

1 investigated the “social spread” of obesity; that is, they examined whether weight
 2 gain in one individual was correlated with weight gain in members of their social
 3 networks. Their results suggest that obesity can spread through social ties. While
 4 the findings are not able to speak to mechanisms through which this may occur, the
 5 authors suggest that social influences may promote obesity by changing norms for
 6 acceptability of being overweight and also by directly influencing individual behav-
 7 iors such as eating. A study exploring social influences of obesity in adolescents
 8 found similar results as overweight adolescents were more likely to have overweight
 9 friends than their normal weight peers (Valente et al. 2009). Recent work, however,
 10 has critiqued the methodology used to investigate social network effects and has
 11 argued that the studies should be interpreted with caution (Cohen-Cole and
 12 Fletcher 2008).

13 The past 50 years of research on the psychological causes of obesity have helped
 14 to isolate many important predictive factors and to clarify that obesity results from
 15 a variety of intra- and inter-individual factors. We hope that these factors (and
 16 other newly isolated factors) can ultimately be combined into an integrated model
 17 that can predict who will become obese and why.

18 CORRELATES OR CONSEQUENCES OF OBESITY

19 Because research on the causes and consequences of obesity is correlational by
 20 nature, any variable that is found to be associated with obesity can technically only
 21 be considered a correlate, as opposed to a predictor or a consequence. However, the
 22 literature does divide the correlates of obesity into those that have been shown to
 23 precede obesity in time and are thought to be causes, and those that follow obesity
 24 and are thus considered to be consequences. In this section, we focus on factors that
 25 may be consequences of obesity, or perhaps only correlates, but that we are reason-
 26 ably certain are not causes.

27 Stigma and Discrimination

28 Overweight and obese individuals face stigmatization and unfair treatment simply
 29 because of their weight. It is often assumed that denigration and derision of over-
 30 weight individuals can only serve to help motivate weight loss, justifying prejudice
 31 and discrimination as “for their own good” (Brownell 2005). In actuality, perceived
 32 weight discrimination and stigmatization likely have negative effects on an indi-
 33 viduals’ psychological well-being (Hatzenbuehler, Keyes, and Hasin 2009) and may
 34 contribute to the physical health problems (Maclean et al. 2009) that are usually
 35 directly attributed to obesity.

36 Implicit bias against overweight is particularly strong (Teachman et al. 2003),
 37 equivalent—if not greater than—bias due to age, race, and gender (Nosek, Banaji,

1 and Greenwald 2002). Moreover, Puhl, Andreyeva, and Brownell (2008) demon-
2 strated that approximately 5 percent of overweight (BMI between 25 and 30) men
3 and 10 percent of overweight women experienced weight discrimination on a
4 daily basis. For individuals with a BMI greater than 35, these numbers jumped to
5 28 percent and 45 percent for men and women, respectively. Among this segment
6 of society, daily interpersonal discrimination—through decreased civility and
7 increased harassment and rejection—is the agonizing norm.

8 Physicians and other health care professionals routinely endorse negative ste-
9 reotypes about their overweight and obese patients. In a recent survey involving
10 primary care physicians, more than half of the physicians in the sample believed
11 that their obese patients were awkward, unattractive, and non-compliant, and a
12 third believed that they were weak-willed, lazy, and sloppy (Foster et al. 2003).
13 Given the sensitive nature of explicitly admitting such beliefs, this number is
14 undoubtedly an underestimation. Similar stigmatizing attitudes are also expressed
15 by the next generation of doctors, medical students (Wear et al. 2006). Particularly
16 concerning is how this biased perception translates into diminished care for
17 overweight patients. Nearly 70 percent of overweight and obese women reported
18 weight stigmatization from their physician (e.g., inappropriate comments about
19 their weight), and 46 percent reported similar experiences from nurses (Puhl
20 and Brownell 2006). Given how likely it is for obese patients to view the medical
21 setting as stigmatizing or threatening, it is not surprising that many patients avoid
22 or delay treatment (Drury and Louis 2002). This under-utilization of health care
23 services may lead to some of the physical health problems that are attributed
24 to obesity.

25 Overweight individuals also face noticeable discrimination in employment set-
26 tings, where they tend to be stereotyped as less conscientious and agreeable than
27 non-overweight employees (Polinko and Popovich 2001). A meta-analytic review
28 of weight discrimination in employment concluded that overweight applicants
29 were at a particular disadvantage when applying for jobs with considerable contact
30 with the public, and that they were rated less favorably when being evaluated as a
31 potential coworker (Roehling et al. 2008). In a survey of over 2,000 overweight
32 employees, 54 percent reported experiences of weight stigma by their fellow employ-
33 ees, and 43 percent reported such experiences by their employer (Puhl and Brownell
34 2006). This type of workplace discrimination ranges from inappropriate comments
35 and abusive joking to denied promotions and termination (Puhl and Heuer 2009).

36 Weight bias can also have a negative effect on romantic relationships (Chen
37 and Brown 2005), customer service interactions (King et al. 2006), and the educa-
38 tional environment (e.g., Puhl and Latner 2007). Given the unmistakably harmful
39 impact that weight bias has on the lives of millions of overweight and obese indi-
40 viduals, and the lack of success in reducing obesity itself, social and behavioral
41 scientists should consider it a priority to develop and test effective strategies
42 for eliminating this pervasive bias. Attempts to reduce weight bias have involved
43 providing information about the etiology of obesity or trying to create empathy
44 for the daily struggles of obese people, but these efforts have had mixed results

1 (e.g., Teachman et al. 2003). It may be the case that using multiple stigma-reduction
2 approaches in concert will be necessary to combat such a strong bias.

3 **Mental Health Consequences**

4 Given the myriad ways in which obese people are discriminated against and stigma-
5 tized in our society, it would not be surprising if mental health problems resulted.
6 And because it was thought that obesity was *caused by* psychopathology, many
7 studies explored whether obesity and a variety of mental disorders co-occurred.
8 Thus resulted several decades of cross-sectional studies that were similar in that
9 they included a measure of obesity and a measure of at least one emotional prob-
10 lem, but that varied according to how they defined obesity, and how they defined
11 and measured emotional problems (Friedman and Brownell 1995). In that genera-
12 tion of studies, individuals usually self-reported their weights, and trained clini-
13 cians almost never used diagnostic criteria to assess mental disorders, which were
14 often measured with a single self-reported item. Findings from these studies rarely
15 found evidence for the relationship between obesity and psychopathology, but
16 because of the overall inconsistency of the results, it was possible to use them to
17 support whatever conclusion one desired. The majority of reports concluded that
18 obesity was not related to mental illness (see Stunkard and Wadden 1992 for a
19 review), but some suggested that obesity was associated with increases in the preva-
20 lence of mental illness, while still others argued, in what became known (rather
21 condescendingly, in our view) as the “jolly fat hypothesis,” that obesity was actually
22 related to improvements in emotional health (Crisp and McGuinness 1976).

23 In more recent years, a number of studies (Hasler et al. 2004; Mather et al.
24 2009; Onyike et al. 2003; Pickering et al. 2007; Scott et al. 2007; Simon et al. 2006)
25 have addressed this question with carefully attained nationally representative sam-
26 ples and clinical measurement of mental illness. Like the prior generation of
27 research, the results of these studies were inconsistent, with some finding evidence
28 for associations between obesity and increased risk for depression or anxiety,
29 and others finding no significant relationships (or in one case, the opposite: that
30 obesity was associated with a lower risk of anxiety; Hasler et al. 2004). Even when
31 significant associations are found, they tend to be small effects, with odds ratios
32 around 1.3.

33 The most rigorous tests of the link between obesity and mental illness come
34 from prospective longitudinal studies, and these have focused primarily on depres-
35 sion (Bjerkeset et al. 2008; Herva et al. 2006; Roberts et al. 2000, 2003). These stud-
36 ies measured BMI at baseline and then depression from one to 17 years later. All
37 three found a significant relationship between baseline obesity and increased
38 depression at follow-up, although in the study with the 17-year follow-up, the rela-
39 tionship was found for men, but not women. Importantly, the one prospective
40 study that controlled for baseline depression still found a significant relationship
41 between obesity and later depression (Roberts et al. 2003). In addition, that study
42 also tested the reverse path, and found that baseline depression did not predict later

1 obesity (controlling for baseline obesity), suggesting, in what appears to be the most
 2 rigorous test to date, that the causal direction of the relationship may be obesity
 3 preceding depression rather than the reverse.

4 Considering the findings from the nationally representative cross-sectional
 5 studies and the prospective longitudinal studies, it seems that a reasonable conclu-
 6 sion is that obesity does seem associated with later depression, but that this rela-
 7 tionship is not particularly strong. Only one prospective study looked at anxiety,
 8 and it did not find evidence for a link between obesity and anxiety (Bjerkeset et al.
 9 2008). In general, the evidence for a link between obesity and anxiety seems weaker
 10 and more tenuous than that between obesity and depression.

11 What does seem clear, however, is that regardless of whether obesity causes
 12 diagnosable mental disorders, it does have psychological consequences. Research
 13 with children and adolescents generally finds that obesity is related to body dissat-
 14 isfaction (Ricciardelli and McCabe 2001; Wardle and Cooke 2005) and low self-es-
 15 teem (French, Story, and Perry 1995; Wardle and Cooke 2005). When individuals of
 16 all ages and genders are studied, it appears that the relationship between obesity
 17 and low self-esteem is stronger in women than in men, gets stronger from child-
 18 hood through college age, and then starts to weaken (but remains significant)
 19 among adults (Miller and Downey 1999).

20 The relationship between obesity and self-esteem may be mediated by the stig-
 21 matizing behaviors of others, such as, among children, weight-related teasing
 22 (Davison and Birch 2002; Thompson et al. 1995). It may also be mediated by indi-
 23 viduals' own internalization of stigma, such as believing their obesity is entirely
 24 under their control (Pierce and Wardle 1997), or by the emotional toll of chronic
 25 and repeated diet failures (Miller and Downey 1999).

26 Physical Health Consequences

27 It has long been thought that obesity leads to physical health consequences, and the
 28 particular ailments attributed to obesity have included cardiovascular disease,
 29 strokes, hypertension, many cancers, diabetes, gallstones, chronic renal failure, fatty
 30 liver disease, gout, osteoarthritis, migraines, dementia, carpal tunnel syndrome,
 31 asthma, infertility, pregnancy complications, polycystic ovaries, erectile dysfunc-
 32 tion, hirsutism, sleep apnea, and incontinence (NHLBI Expert Panel 1998). It has
 33 not been shown, however, that obesity per se actually causes these ailments, because
 34 the primary type of study that can show such causal links cannot be conducted for
 35 obesity. That type of study, the randomized controlled trial, is not possible because
 36 it requires randomly assigning individuals to either be obese or not obese, and then
 37 watching those individuals over the next several decades to see which diseases they
 38 contract.

39 Researchers must settle for the next best form of evidence, prospective longitu-
 40 dinal studies, in which individuals who happen to be obese (or not obese) are
 41 observed over many decades. Health differences found between obese and non-
 42 obese people in these studies may appear to have been *caused* by obesity, but in fact

1 may have actually been caused by some additional factor that also varies between
2 obese and non-obese people. It is all too easy for people not versed in research
3 methods to mistakenly assume that such studies are convincing evidence of obesity's
4 causal role in many diseases.

5 In the last 20 years, scientists (as well as activists and journalists) have begun to
6 explore a variety of confounding factors that may account for the relationship
7 between obesity and physical health problems (Campos et al. 2005). This has led to
8 a contentious and politicized debate (described in Kolata 2007) that is unlikely to be
9 easily resolved. The confounding factors that may play an important role in this
10 relationship are obese people's greater likelihood (than non-obese people) of lead-
11 ing sedentary lifestyles, repeatedly gaining and losing weight (known as "weight
12 cycling"), avoiding health care, and being in lower socioeconomic groups (Campos
13 2004; Ernsberger and Koletsky 1999). These factors have been shown to be associ-
14 ated with poor health and, to the extent that they are more likely to occur among
15 obese people than non-obese people, may account for the relationship between
16 obesity and disease.

17 Another factor that muddies the evidence for obesity as a cause of health prob-
18 lems is data suggesting that obesity does not shorten an individual's lifespan, or at
19 least not until extremely high levels of obesity are reached (Flegal et al. 2005). It is
20 difficult (although not impossible) to convincingly argue that obesity causes such a
21 long list of ailments but somehow does not shorten one's life. The evidence is also
22 called into question because studies supporting the relationship between obesity
23 and health outcomes tend to be conducted by researchers with significant conflicts
24 of interest (Fraser 1998).

25 As researchers with no vested interests on either side of this debate (as we study
26 psychological outcomes of diets and obesity rather than physical ones), we have
27 observed this debate from the outside. It is our conclusion that the links between
28 obesity and some diseases are quite convincing, such as that between obesity and
29 type 2 diabetes (Colditz et al. 1995; Ford, Williamson, and Liu 1997) and osteoarthri-
30 tis (Hochberg et al. 1995). However, it is equally clear to us that the strength of the
31 links between obesity and many other ailments has been overstated (Olshansky
32 et al. 2005). Even if these relationships cannot be entirely explained away by con-
33 founding variables, they do not seem strong enough to warrant the amount of alarm
34 they receive from the media, scientists, and the government.

35 TREATMENT OF OBESITY

36 Despite imperfect models regarding its etiology, strong beliefs in the physical health
37 consequences of obesity have led researchers to develop treatment interventions.
38 Most of these interventions have been diets designed to promote weight loss.
39 More recent efforts, however, have included interventions aimed at treating the

- 1 self-esteem and body dissatisfaction that result from obesity, without necessarily
- 2 focusing on weight loss.

3 **Treating the Physical Health Consequences of Obesity**

4 Given the field of psychology's long history of research on behavior change, it is not
 5 surprising that its approach to the treatment of obesity is to try to alter people's
 6 eating habits by teaching them diet strategies. Over the past 60 years, psychologists
 7 have studied dozens of behavioral weight loss techniques to see which are effective
 8 (Leon 1976; Ayyad and Andersen 2000). They have taught dieters how to select
 9 appropriate foods to eat, count calories, resist temptation, monitor how much food
 10 they eat, and to reward (or punish) themselves for eating the right (or wrong)
 11 amount of food (see Leon 1976 for a review). In some treatments they gave the diet-
 12 ers all the food that they were allowed to eat, and in some cases the dieters were
 13 required to do all their eating in a researcher's laboratory or clinic (e.g., Wing and
 14 Jeffrey 2001; Musante 1976). Psychologists have also explored the effectiveness of
 15 having dieters give some of their own money to researchers and then trying to earn
 16 it back by losing certain amounts of weight (e.g., Harris and Bruner 1971). They also
 17 tested whether using social pressure could get obese people to reduce their con-
 18 sumption (e.g., Wollersheim 1970). Finally, in efforts to create negative associations
 19 with desired foods, researchers have paired dieters' favorite foods with horrible
 20 odors, and in other cases have actually jolted obese patients with electric shock
 21 whenever they tried to eat a food they craved (e.g., Foreyt and Kennedy 1971; Meyer
 22 and Crisp 1964).

23 As silly (or cruel) as some of these treatments may sound, by and large, what-
 24 ever psychologists attempted generally led to at least *some* weight loss during the
 25 early months of the diet (Jeffery et al. 2000; Perri and Fuller 1995). Across over 200
 26 diet studies conducted from 1966 up until 2000, participants lost from 8 to 22
 27 pounds in as many weeks—about a pound a week (Wing 2002). For a diet to truly
 28 be considered successful, however, individuals must not just lose weight in the short
 29 term, but they also must keep it off. The majority of diet studies do not follow par-
 30 ticipants long enough to see if that happened. In fact, only 6 percent of over 800 diet
 31 studies found by researchers in the year 2000 had follow-ups of three years or more,
 32 and two-thirds of those had such serious flaws that they offered little useful infor-
 33 mation (Ayyad and Andersen 2000).

34 Across 14 studies with long-term follow-ups, participants initially lost an aver-
 35 age of 30 pounds, but by four or five years later, they had gained back all but 7 of
 36 those pounds (Mann et al. 2007). These results likely *inflate* the success of these
 37 diets because the studies have multiple sources of systematic bias—all of which
 38 make the diets look more successful than they were. For example, most of the people
 39 who start these diet studies do not finish them, and the people who do finish are the
 40 ones who do the best on the diets. In addition, many of the people in these studies
 41 are not actually weighed by the researchers, but rather just tell the researchers their
 42 weight over the phone or by mail. Since most people lie about their weight—and

1 say they weigh less than they actually do—this flaw causes the diets to look like they
2 led to more weight loss than they really did. Another source of systematic bias in
3 these studies is that many of the participants went on additional diets during the
4 long follow-up period. This makes it appear as if the original diet had led to sus-
5 tained weight loss, when, in fact, these dieters likely gained back a lot—if not all—of
6 the weight before they started a new diet.

7 If the goal of obesity treatment is to have obese people lose enough weight to
8 make them non-obese, and to maintain that weight loss in the long term, then it
9 seems clear to us that behavioral treatments for obesity are unsuccessful. However,
10 it is the association between obesity and physical health problems that makes effec-
11 tive obesity treatment a high priority in our country's research agenda. Given that,
12 we suggest that it doesn't make sense to use weight loss—an indirect and imperfect
13 measure of health—as a marker of successful obesity treatment when we could
14 be measuring improvements in physical health more directly (Ernsberger and
15 Koletsky 1999). The next generation of obesity treatment research should focus on
16 physical health outcomes such as heart rate, blood pressure, and cholesterol levels,
17 rather than weight change. Indeed, research on exercise interventions show positive
18 health benefits on all of these outcomes, even in the absence of weight loss (Caudwell
19 et al. 2009).

20 **Treating the Mental Health Consequences of Obesity**

21 Early psychological interventions focused on altering eating behavior to the neglect
22 of mental health sequelae. It has even been argued that some mental health conse-
23 quences of obesity, especially body distress, are important motivators for behavior
24 change and that addressing them would be detrimental to weight-loss efforts.
25 While there has been some support for this, the negative social and psychological
26 effects of body distress and obesity are important areas of concern (Heinberg,
27 Thompson, and Matzon 2001; Schwartz and Brownell 2004; Wilson 1996). Body
28 image and self-acceptance themselves became primary aims of treatment due to the
29 combination of these negative consequences and ineffective long-term weight loss
30 treatments (Wilson 1996; Schwartz and Brownell 2004; Rosen et al. 1995).

31 It has been assumed that weight loss is a necessary prerequisite for improve-
32 ment in body image. However, research findings have produced mixed results (see
33 Schwartz and Brownell 2004). A randomized control trial comparing a cognitive
34 behavioral body image therapy (CBT) or a no-treatment condition found that
35 although weight status remained unchanged, individuals who received CBT showed
36 significant improvement in body image as well as improvement in psychological
37 symptoms, self-esteem, overeating, and eating guilt (Rosen et al. 1995). Another
38 study found that the promotion of body and self-acceptance in a non-diet wellness
39 intervention improved not only body image, but also physical health parameters,
40 without changes in weight (Bacon et al. 2002). These positive psychological and health
41 outcomes were sustained at a two-year follow-up, whereas initial improvements in
42 the diet group were generally not maintained (Bacon et al. 2005). In contrast,

1 a study comparing a combined body image treatment and weight control program
 2 to a weight control program alone did not find any additional improvement in
 3 body image (Ramirez and Rosen 2001).

4 In addition to being targets of treatment, body image and weight acceptance
 5 may also promote successful weight maintenance after weight loss occurs. A cog-
 6 nitive behavioral therapy intervention for obesity created by Cooper and Fairburn
 7 (2001) encourages a shift from weight loss to acceptance of weight stability follow-
 8 ing a pre-set period of time. Such promotion of weight- and self-acceptance is
 9 thought to prevent psychological barriers, including focusing on unattainable
 10 weight loss, from interfering with weight maintenance. In support of this, satisfac-
 11 tion with body weight has been associated with improved maintenance of weight
 12 loss, but findings have been inconsistent and more research is needed (Foster et al.
 13 2004; Byrne, Cooper, and Fairburn 2004; Ames et al. 2005; Gorin et al. 2007).

14 Targeting body image dissatisfaction and self-acceptance either in isolation or
 15 in conjunction with behavior change therapy aimed at weight loss appears to be a
 16 promising component of treatment. While continued research is needed to explore
 17 the effects of these treatments, it is clear that the mental health consequences of
 18 obesity should not take a back seat to treatments aimed solely at the physical health
 19 consequences.

20 CONCLUSION

21 In 1999, psychologists, nutritionists, and other experts proposed a paradigm shift in
 22 research and policy on dieting and obesity (Cogan and Ernsberger. 1999). They sug-
 23 gested a health-centered, rather than a weight-centered approach to obesity. This
 24 approach would encourage obesity researchers to design their interventions with
 25 the goal of promoting fitness rather than weight loss, and to use health markers as
 26 their outcome variables, rather than relying on weight as the primary and critical
 27 measure of success. Work consistent with this approach is accumulating, and
 28 although the proposed shift has not occurred, we believe that it would be a positive
 29 direction for the field. It seems to us that even though interventions based on this
 30 new paradigm may not reduce the prevalence of obesity, they are likely to improve
 31 the nation's health, and by helping to unlink the association between obesity and
 32 illness, may even reduce the stigma of obesity.

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