

Of Mind and Matter: Psychological Dimensions in Obesity

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Obesity is a physiological energy imbalance, a chronic disorder that results from an increase in caloric intake and/or a decrease in caloric expenditure. Other than the accumulation of excess adipose tissue, there are no signs or symptoms characteristic of all obese people. Obesity rates have increased exponentially worldwide in the past thirty years for reasons that we do not entirely understand. Multiple environmental, genetic, neuro-endocrinological, and psychosocial factors contribute to the development of obesity. Though there are many different, and even controversial, frameworks for obesity, most researchers acknowledge that it can lead to serious medical and psychological morbidity. This paper focuses on psychological dimensions in the study of obesity: the intricate human “minded brain” that promotes self-regulation, motivation, and self-efficacy; the complexities involved in considering obesity a psychiatric disorder, with the possibility of a so-called “obese personality”; the role of stigma, prejudice, and discrimination; and psychiatric symptomatology among the obese.

KEYWORDS: obesity; self-regulation; eating; the “minded brain”; personality; psychiatric symptoms

INTRODUCTION: THE NATURE OF OBESITY

Obesity is a chronic (though sometimes a remitting and relapsing) condition that *results in* an accumulation of adipose (fat) tissue, whether due to increased caloric intake and/or a decreased expenditure of calories. In other words, it *results from* a physiological energy imbalance. (Karasu & Karasu, 2010, p. 2) Other than the accumulation of fat, though, there are no other signs or symptoms characteristic of everyone who is obese or even overweight (Allison et al., 2008). Obesity is currently defined, somewhat arbitrarily, as a body mass index ([BMI] i.e., the weight in kilograms

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AMERICAN JOURNAL OF PSYCHOTHERAPY, Vol. 66, No. 2, 2012

divided by height in meters squared) of 30 kg/m² or greater, and it is subdivided further into Class I, Class II, and Class III, depending on increasing levels of BMI. Overweight is defined as having a BMI between 25 kg/m² and 29.9 kg/m² (Karasu & Karasu 2010, pp. 13–15.)

Obesity rates have increased exponentially during the past 30 years for reasons that are not completely apparent. For example, Flegal, Carroll, Ogden, and Curtin (2010) noted that in 2007 to 2008, more than 32% of adult men and 35.5% of adult women in the United States were obese as measured by BMI. It is not, however, just a U.S. problem: obesity is now considered a “global pandemic” (Swinburn et al., 2011, p. 894).

MODELS OF OBESITY

There are many, even controversial, models or frameworks for understanding obesity. Researchers cannot even agree on whether obesity itself is a disorder or disease to be “medicalised” (Chang & Christakis, 2002, p. 151). There is even a question about whether society should hold obese people personally responsible for their obesity—i.e., determining whether obesity is a “crime against society” and reflective of “failures of self-care” (Fitzgerald, 1994, p. 197). For example, even some health care professionals who work with obese patients have expressed a bias against the obese and believe that obesity may be prevented primarily by self-control of psychological problems or behavior (Maroney & Golub, 1992; Teachman & Brownell, 2001, Schwartz et al., 2003; Foster et al., 2003).

Others, particularly those in the National Association to Advance Fat Acceptance (NAAFA), believe obesity is an example of body diversity, analogous to sexual, ethnic, or racial diversity, to be celebrated, not treated (Saguy and Riley, 2005). Still others believe obesity is an example of inappropriate adaptation to an environment of abundance, namely, our exposure to infinite varieties of sugar, fat, and salt-laden processed foods, as well as to considerably less opportunity for physical activity in our technologically driven modern world (Power and Schulkin, 2009, p. 11). Along those lines, Devlin (2007) has even suggested that obesity is a “cultural disorder of sorts” (p. S84). Swinburn et al. (2011) have described obesity as the “result of people responding normally to the obesogenic environments they find themselves in” (p. 806). These researchers contend that the physiological energy imbalance that results in fat accumulation (obesity) is “proximally determined by behaviours and distally by environments” (p. 807).

Brownell and colleagues (2010) emphasize that environmental factors can undermine personal responsibility and sabotage efforts when it comes

to obesity. They suggest that legislative and regulatory mandates, including caloric labeling on menus, healthier choices for student lunches, and even taxes on certain foods and beverages, may have a role in society by “bridging the divide between views based on individualistic versus collective responsibility” (p. 379).

According to the International Classification of Diseases, Edition 10, to be published officially in 2013, (ICD 10-CM) by the World Health Organization, though, obesity will be classified under the “Endocrine Nutritional and Metabolic Disease” category. Whatever model we use, and although there is a category of “metabolically benign” obesity (Stefan et al., 2008, p. 1614) most who work in this field acknowledge that obesity, for the majority of people, can result in substantial medical morbidity, including increased risk of certain cancers (e.g. breast, colon, prostate), sleep apnea, cardiovascular disease, hypertension, and metabolic disorders. For example, the so-called “metabolic syndrome” is a cluster of symptoms, including abdominal obesity, hypertension, lipid abnormalities, abnormal glucose regulation, insulin resistance, and even overt diabetes, that are considerably more common in the obese (see Karasu & Karasu, 2010, pp. 30–44). Many researchers contend that obesity can lead to increased mortality rates and, if current trends continue, life expectancy in the future will decrease for the first time in history (Olshansky et al., 2005; Finkelstein et al., 2010).

OBESITY RESULTING FROM COMPLEX INTERACTIONS

In all likelihood, fat accumulation is dependent on a complex interaction of multiple biological, environmental, and psychological factors. In other words, genetic, gender, perinatal, developmental, dietary, environmental, neural, and psychosocial factors contribute (Levin 2004, pp. R1297–1298). It is also highly reasonable to assume that there are really “the obesities.” A person who is more than 100 pounds (or even 50 pounds) overweight is probably very different biologically from someone who needs to lose only a few pounds to be of normal weight (Bouchard et al., 2004). One of the most important *biological factors*, for example, is the considerable genetic contribution, in some studies, reported to be up to 65% or higher; hundreds of gene loci are involved, in all aspects of fat accumulation and weight control. (Rankinen et al., 2006). Even the distribution of fat on our bodies—whether around the waist and internal abdominal organs (i.e., the so-called android, or “apple-shaped” distribution more commonly seen in men) or peripherally and around the hips and lower body (i.e., the so-called gynoid, or “pear-shaped” distribution more

commonly seen in women)—is most likely genetically determined (Karasu & Karasu, 2010, p. 17).

Genetically, though, some people are more apt to develop fat accumulation, given similar environmental conditions, than others (Friedman, 2009). Friedman (2003), in whose laboratory at Rockefeller University the fat regulating hormone leptin was isolated in the early 1990s, has asked the provocative question, “. . . why, in our current environment where almost everyone has essentially free access to calories, anyone is thin?” (p. 856). The answer lies in the way our genes interact with our environment. Neuro-endocrine and gastrointestinal factors play a biological role. There are literally hundreds of hormones, such as insulin, leptin, ghrelin, adiponectin, cortisol, neuropeptide Y, gastrin, cholecystokinin, amylin, and glucagon, that are produced selectively by different organs throughout the body, including the brain and even adipose tissue itself, that are involved in the maintenance of our weight (Karasu and Karasu 2010, pp. 153–174).

Environmental factors include not only prenatal and postnatal exposures to toxins in the infant and mother, but may also include sleep deprivation, circadian rhythms, increasing portion size, “portion distortion” (p. 1103) and failure to appreciate “consumption norms,” i.e. appropriate portion size (Wansink & Van Ittersum, 2007, p. 1104), and exposure to medications and even possibly viruses (Karasu & Karasu 2010, pp. 239–240; p. 335).

Psychological factors include the relationship of mind to brain, particularly as it relates to eating and food choice, cognitive factors involved in self-regulation, motivation, and self-efficacy, perceptions of prejudice and discrimination, as well as increased prevalence of psychiatric symptoms, such as anxiety and depression, among the obese.

THE HUMAN “MINDED BRAIN”

It is significant that unlike the brains of animals that function on a kind of metabolic “autopilot,” our higher-functioning human “minded brain” (Damasio 1997, p. 169) can sabotage our efforts at weight control, despite our best intentions. Our rational (cognitive) minds enable us to contemplate and plan our food intake, remember what we have eaten, even remember where our favorite restaurants are, and allow us to have insight into our behavior. In other words, we can appreciate the importance of health-conscious eating and exercise. We can even be “restrained eaters” (Lowe & Butryn, 2007, page 436) as, for example, when we choose the less appealing food alternative because of its health value or make choices based on extrinsic factors, such as cost, brand, convenience, etc. (Mela,

2006). We can also choose to eat less than we want rather than less than we need (Lowe & Butryn 2007).

We can, however, also throw caution to the winds and choose not to exercise or eat sensibly, despite our cognitive knowledge. We can indulge our “inner saboteurs,” as cognitive therapist Judith Beck (2008, p. 16) describes our rebellious inner voice. In other words, we can eat *reflectively*, with cognitive awareness and consideration of long-term goals about health and fitness and even social expectations, or *reflexively*, the more primitive type process that involves reward and motivation (Alonso-Alonso & Pascual-Leone, 2007). Ancel Keys, one of the major pioneers in studying obesity, wrote years ago, “Plenty of patients insist they want to reduce, know that calorie imbalance is the problem, understand the rudiments of caloric values, of food and exercise, and still cannot, or at least do not, reduce. Obviously, psychological as well as dietetic problems must be solved” (1965, p. 1329). In that context, Friedman (2009) acknowledged while it is generally appreciated the obese eat more and exercise less, we *really still do not know why this is so*, though he felt it is “less about conscious choices . . . and more about their biological makeup” (p. 977S). It is likely, though, that psychological dimensions, at least in a supporting role, factor into any understanding of obesity as well.

SELF-REGULATION, MOTIVATION, SELF-EFFICACY

Psychologist Roy Baumeister has focused along those lines on our human ability for self-regulation or self-control. For Baumeister, self-regulation, though, is finite and subject to fatigue, stress, time of the day, substances such as alcohol, distractions in the environment, and even how often we use it (2002). For example, diminished self-control from a kind of “overuse” may explain why it is often so difficult to avoid the dessert tray in front of us when we have ordered so sensibly (and made so many healthy choices) for the earlier part of a meal. It may even explain why we are less able to resist temptation when there is a beautiful display in a store or soothing, pleasant music in the background.

One of the major distinctions between humans and animals is our ability to stop what we are doing in the middle of doing it. In other words, the ability to self-regulate our behavior enables us to be flexible. We can foresee possibilities, and we can be motivated by a future goal (Baumeister, 2005, p. 310). This, of course, becomes important in weight control and any discussion of obesity: we are theoretically capable of avoiding temptations by anticipating the consequences of our behavior and envisioning future satisfaction (e.g., of being slimmer eventually). Further, Baumeister

distinguishes *resistible impulses* from truly *irresistible*, physiological ones such as breathing, sleeping, or urinating, and he believes that people sometimes use “rationalization” to explain their conscious lack of self-control with so-called “irresistible” impulses (Baumeister, 2002). Vohs and Baumeister (2004, p. 3) maintain “nearly every major personal and social problem . . . involves some kind of failure of self-regulation,” including alcohol and drug addiction, and even “some obesity and eating disorders.” Herman and Polivy (2004, p. 498), likewise, note that sometimes dieters experience what they call the “what-the-hell effect” or “all-or-none-effect.” This is the “perverse logic” of dieters, when their motivation for self-regulation fails and once they yield to a temptation, they forgo their diet completely, having had one failure in self-regulation.

We have both *extrinsic motivation*, in which we do something as a means to something else (e.g. avoiding punishment or gaining some reward), and *intrinsic motivation*, in which we do something for its own sake (Baumeister, 2005, p. 163). Those who have higher levels of intrinsic motivation are considered to be “self-motivated” (Grant, 2012, p. 113). Those who operate more from extrinsic motivation require praise or reward, what we might call “incentives” (Grant, p. 113).

For some, though, extrinsic motivation can be an important stimulus for changing behavior, including for losing weight. This is the premise behind the television show, *The Biggest Loser*. Not only does the winner gain fame and fortune (and a much thinner, healthier body), but avoids the failure and humiliation of losing the contest or failing to lose weight. This kind of motivation is an incentive—it encourages a person to act in a way he or she might not otherwise. The problem with incentives, though, says Grant (2012), is that they can backfire: “Once the incentive stops, so does the motivation” unless the particular behavior “can be transformed into habitual behavior and become part of accepted expectations” (pg. 69). Otherwise, “. . . the impact of incentives could be short-lived and even corrosive” (p. 69). Grant, though, emphasizes “incentives *cannot* teach self-discipline (p. 118) and since they do not address “the root causes of the problem, are often limited as a tool for long-term improvements” (Grant, 2012, p. 133). This view may be of particular importance when it comes to motivation for weight control, especially since obesity is a chronic disorder.

An important aspect of maintaining self-control is the monitoring of behavior. For example, we are more inclined to limit our food intake if dirty dishes are not quickly removed from the table. For example, Brian Wansink (2006, pp. 37–40) has written of the differences in food con-

sumption when his subjects could see how many half-eaten chicken wings they had consumed. Using a “diet buddy” (Beck, 2008, pp. 35–37), weighing ourselves daily, keeping a record of food intake, and even having a diet coach for consultation, make us accountable and more able to monitor our behavior successfully. We are social creatures and as such, often influenced by those who are important in our lives. We may develop what is called “goal contagion” (Fitzsimons & Bargh, 2004, p. 158), i.e., adopting the goals of other people or the goals other people want us to have.

Another important human component involved in weight control is self-efficacy: the optimistic belief that we can actually bring something about. (Rothman et al., 2004). In that context, we do better when we can meet some of our goals more immediately, such as experiencing the reinforcement we feel when we lose those first few pounds. Conversely, a sense of self-efficacy can be undermined by a partner who sabotages efforts at weight control or even by our own pessimistic, self-defeating personality.

OBESITY AS A PSYCHIATRIC DISORDER?

In the current version of the Diagnostic and Statistical Manual (DSM IV-TR), though, obesity is not a psychiatric diagnosis. In preparation for the eventual release of Diagnostic and Statistical Manual-V (DSM-V) there has been considerable discussion during the past few years about the inclusion of obesity as a mental disorder. At present, though, obesity may be classified on Axis III, under “General Medical Conditions.” Alternatively, a patient may be given a psychiatric diagnosis (e.g. depression, anxiety, etc.) with the qualifying statement, “due to the medical condition, obesity.” This causal relationship is harder to prove, of course, as it is not often possible to know whether a particular psychiatric condition is a cause or consequence of a patient’s obesity.

Berthoud (2007) has suggested our higher cognitive functions are a major element responsible for the increasing rates of obesity throughout the world. For Berthoud (2007), “neurophysiology is no less physiology than adipocyte (fat cell) or liver physiology” (p. 489). Spiegel et al. (2005, p. 553) have even suggested that brain imaging will eventually play an important role and become “a cornerstone” in our efforts to understand the mechanisms behind obesity.

Volkow and O’Brien (2007) have argued that obesity is not only a metabolic disorder but also a brain disorder. These authors maintain “some forms of obesity are driven by an excessive motivational drive for food”

and they consider obesity a “food addiction” analogous to substance abuse and dependence (p. 708). For Volkow and O’Brien (2007), obesity is “characterized by compulsive consumption of food and the inability to refrain from eating despite the desire to do so” (p. 708). Kalivas and Volkow (2005) emphasize, as well, that the “cardinal feature” of an addiction is the “continued vulnerability to relapse,” even years later (p. 1403). Certainly, this model can apply to those who have been obese and lost weight, only to relapse eventually and gain back much or even more of their original weight. This pattern has been described as “weight cycling” or more commonly, “yo-yo” dieting (see Karasu & Karasu, 2010, pp. 197–198).

Of course, the notion of a “food addiction” is a complex one since, unlike substance abuse for drugs or alcohol, we can never be “food free” and we are, as well, always obviously “food dependent.” We have, as Heber (2010) describes, a “natural human tendency to eat” (p. 282S), and Moreno and Tandon (2011) have emphasized the “significant neurobiological and psychological underpinnings” (p. 1128) involved in overeating. Barry et al. (2009) suggest that a putative diagnosis of “overeating disorder,” analogous to a substance dependence disorder, might apply only to a subset of those who are overweight or obese and who chronically experience a loss of control over their eating. These researchers also emphasize: “An addiction model of obesity assumes that overeating is the primary cause of obesity” (p. 445). They assert that weight control is much more complicated because of human differences in caloric needs as well as the vast complexity of human metabolism in maintaining body weight. Devlin and his colleagues (2000), for example, had expressed a similar thought, namely that even though, for example, overweight people generally consume more calories than those who weigh less, they may not necessarily be *overeating* when considering their larger size. They say: “This distinction is important, as the idea that obese individuals bring about or maintain their obesity by inappropriate overeating underlies many of our culture’s negative stereotypes about obesity” (p. 857). Recent studies, though, such as one by Gearhardt and colleagues (2011, and a concept predicted by Spiegel et al., 2005) demonstrated by neuro-imaging (MRI) that there are similar patterns of neural activation in both substance and “addictive-like eating behavior” in group of almost 50 young women (p. 809).

Years ago, and from a more psychological perspective, Stunkard (1959, p. 286) explored three variables to be of significance regarding eating patterns. These included “the presence (or absence) of expressions of

self-condemnation,” in the context of a “deviant” eating pattern; the “degree of personal meaning or symbolic representation” that someone attaches to a specific eating pattern; and a person’s own assessment of the level of stress he or she experiences. These three variables apply today in any exploration of *disordered* eating patterns, such as binge eating or the night eating syndrome.

Occhiogrosso (2008, pp. 265–276) explored the highly complex relationship that psychiatry has had with eating, and particularly overeating, by reviewing all the articles published in the *American Journal of Psychiatry* since its first publication in the mid-1800s. She found that overeating (gluttony) was seen as sinful and immoral initially. Somewhat later, overindulgence was linked to criminality. In the early 20th century, Freud influenced psychiatric thinking with his focus on oral fixations and the oral character. It was, though, not until the 1970s and even 1980s that separate eating disorders, including anorexia nervosa and bulimia nervosa, were first delineated in the psychiatric *Diagnostic and Statistical Manual*. Today, there are still questions and controversies about including night eating syndrome and binge eating disorders in DSM-V, though both syndromes were described by Stunkard more than fifty years ago (1959).

THE EATING PARADOX

There are, though, two kinds of eating—homeostatic for maintenance of internal bodily functioning (i.e., “regulatory” eating) and nonhomeostatic or hedonic eating (i.e., “nonregulatory” eating) for pleasure or associated with external factors, such as cravings, availability, variety, palatability, etc. (Friedman, 2008, pp. 12–13). Power and Schulkin (2009, p. 173) have noted the “paradox of eating” because eating is “both necessary for homeostasis and a threat to homeostasis” simultaneously. Further, Devlin (2007) has noted that hedonic overeating may be related to an eating disorder model, a substance use disorder model (e.g. a dysfunction of our reward systems), or even a model based on affect regulation of our stress response, but he does not believe obesity should be classified as a psychiatric disorder. Bachman and Histon (2007) also take issue with obesity as a brain disorder, adding they would like to see more evidence “before adding the stigma of ‘brain-damaged’ to the high physical and social burden obese persons already bear” (p. 738). Recently, though, the American Psychiatric Association’s DSM-V Task Force has rejected the idea of obesity as a mental disorder (Gever, 2010).

AN OBESE PERSONALITY?

More than 50 years ago, in their conceptualization of obesity as a psychosomatic disorder, Kaplan and Kaplan (1957) *could not* find any “specific personality picture that characterizes obese patients” (p. 195). Nor could psychiatrist and researcher Stunkard and his colleagues (Weinberg, Mendelson, & Stunkard, 1961) find there was any single personality typical of all who have obesity. Further, Stunkard (1959) could not find any distinguishing psychological characteristics that were seen in all obese people, whether he evaluated basic personality structure, psychodynamic conflicts, or even the intensity of their basic biological drives. Likewise, Rodin and her colleagues (1989) could not find any common etiological factors, such as a lack of impulse control, inability to delay gratification, or even a maladaptive eating style in all the obese subjects they studied. Even in a study of the “severely obese” who underwent bariatric surgery, Stunkard and Wadden (1992) found there is “no single personality type” (p. 524S) that is characteristic, though they may be likely to suffer from body image disparagement and disordered eating such as binge eating. Friedman and Brownell (1995) also found that obesity is “strikingly heterogeneous with respect to etiology, effects of excess weight on medical variables, and response to various treatments” (p. 3). In other words, these researchers concluded “. . . the effects of being obese vary across individuals” (p. 3). They did find that those obese individuals who presented for treatment were more likely to have a higher prevalence of psychopathology (e.g. anxiety and depression), and that women seemed to suffer more from their obesity, particularly because of the stigma of obesity, than did men. Wadden et al. (2002) also found “personality is as diverse in the obese as it is in those of normal weight” (p. 148).

More recently, Sutin et al. (2011) reported on a longitudinal study of almost 2000 people (from the Baltimore Longitudinal Study of Aging) that spanned more than 50 years and investigated the relationship of personality traits with changes in BMI across adulthood. Initially, the sample, begun in 1958, consisted of men only, most of whom were white and college educated. By the late 1970s, women and minorities became part of the sample. The current sample included individuals of which 45% of were of normal weight, 38% were overweight, and 17% were obese. The researchers used the five-factor model of personality (Neuroticism, Extraversion, Openness to Experience, Agreeableness, and Conscientiousness) as they assessed weight fluctuations over time. There were up to 32 assessments (i.e., actual staff measurements, rather than self-report) of

height and weight and up to 16 assessments of personality over the 50-year period. Not surprisingly, over time, those who rated highest in impulsivity (i.e., difficulty with impulse control) and neuroticism and lowest in conscientiousness were more apt to be more than twenty pounds heavier (11 kilograms) than those in the bottom 10% of the same categories. The researchers also found that those subjects “with a dispositional proneness to experience negative emotions” (p. 585) especially those who were highly impulsive, antagonistic, and subject to depression, were less apt to have maintained a steady weight over time.

THE STIGMA OF OBESITY

The stigma of obesity, and particularly the prejudice and overt discrimination that result from it, have been the focus of considerable research. Puhl and Brownell (2001), for example, have reported that the obese have been discriminated against in employment, health care, education, salary, and even jury selection. Stunkard and his colleagues (1998) called attention to the long history of such prejudice, recorded as early as 12th century in Japan, and even much earlier in Biblical references. Gluttony, of course, was considered one of the Seven Deadly Sins (as was Sloth) that led to eternal damnation in Christian theology. Wadden and Stunkard (1985) emphasized not only did obesity raise moral objections, but it is also was considered an “aesthetic crime”—namely “it is ugly” (p. 1062). Ancel Keys (1965) expressed a similar thought when he said, “. . . in both sexes and at all ages obesity is disgusting as well as a hazard to health” (p. 1331). As previously noted, even health care professionals who work with the obese have negative stereotypes about these patients (Maroney & Golub, 1992; Teachman & Brownell, 2001, Schwartz et al., 2003; Foster et al., 2003). For example, Foster et al. (2003) reported that more than 50% of their sample of more than 600 physicians from diverse geographical areas described the obese as “awkward, unattractive, ugly, and noncompliant” (p. 1173), and these physicians were generally pessimistic about treating their obese patients. Further, their data “suggest” that obesity is seen primarily “as a matter of behavioral management . . .” (p. 1174). Schwartz et al., 2003, also surveyed clinicians and researchers at an international obesity conference and found a clear anti-fat bias among the attendees.

Stigma and discrimination toward the obese continue to this day and may even be one of the last bastions of acceptable and tolerated behavior. Comedians on television feel no compunction about denigrating the

weight of an obese public person, as for example, when the obese governor of New Jersey, Chris Christie, was considering a run for higher public office and became the subject of insensitive late-night humor.

Devlin et al. (2000) describe obesity as “the most easy to recognize and the most difficult to treat of medical conditions” (p. 854). Obesity, unlike certain other stigmatized diseases that are not necessarily always apparent, is not a condition that a person is able to conceal. Lewis et al. (2011, p. 1349) explain that the “physical visibility of the fat body” is publicly obvious to everyone. They note that the interplay of the weight loss industry, the fashion industry, the media, and the health profession, among others, reinforce and intensify the stigma of obesity. They describe three distinct categories of stigma obese individuals may experience (p. 1351): *direct stigma*, such as being subject to taunts publicly, *environmental stigma*, such as being unable to fit into airplane or bus seats, and *indirect stigma*, such as having their supermarket carts scrutinized by other shoppers.

In their study of 141 (105 female, 36 male) Australian subjects, with BMI ranges from 30kg/m² to over 70 kg/m², Lewis et al. (2011, p. 1349) found shaming stigma experiences (even subtle ones) did not motivate their subjects to lose weight, but rather had the opposite effect and “created a barrier” to participating in “health-promoting activities.”

Volger et al. (2012, p. 149), in replicating an earlier study by Wadden and Didie (2003), found that the obese are very sensitive regarding use of terms to describe their obesity. For example, they found that the most undesirable terms were “fatness,” “excess fat,” “obesity,” and “large size,” whereas “weight,” “heaviness,” “unhealthy body weight,” “weight problem,” and “unhealthy BMI” were more acceptable. Even a physician’s own weight may have an impact on care of the obese patient. For example, Bleich et al. (2012) found, in a cross-sectional survey of 500 primary care physicians, normal weight physicians were more apt to confront and to feel more comfortable with issues of obesity, including discussions regarding diet and exercise counseling with their obese patients, than were obese physicians. These researchers also found that there was a higher probability that physicians would initiate conversations regarding weight when they perceived their patients’ weights as greater than their own.

PSYCHIATRIC SYMPTOMS AMONG THE OBESE

It is no wonder that researchers might expect the obese to have a greater incidence of psychiatric symptoms (Stunkard et al., 1998) and

might even experience the *double stigma* of obesity and mental illness. Wadden and Stunkard (1985) acknowledged reports of increased psychiatric symptoms in the obese, especially those “severely obese,” i.e., more than 75% or more overweight, but they took issue with the methodology of some of those earlier studies. They also emphasized that many studies did not take into account the considerably high levels of anxiety and depression seen in patients seeking general medical care, “regardless of their weight” (p. 1064). Furthermore, Wadden and Stunkard (1985), and later Friedman and Brownell (1995), suggested obese individuals who seek treatment may be different from those who do not because the former may suffer greater levels of anxiety and depression and are more likely to experience body image concerns, e.g. thinking of their body as “grotesque” or “loathsome” (Wadden & Stunkard, 1985, p. 1064), and binge eat. They concluded that obese patients may have “mild levels of psychopathology” (Wadden & Stunkard, 1985, p. 1064) but not much greater than those who present for medical or surgical evaluation.

Bruch (1952) and Stunkard (1957) have called attention to the stress that dieting itself can cause for the obese individual. For example, Bruch (1952) reported psychotic states among patients in her small sample; she noted some became “delusionally” preoccupied with their weight (p. 340). Stunkard (1957) reported that more than half of 72 subjects noted symptoms of anxiety, restlessness, irritability, and nervousness. Stunkard found symptoms of depression, with intense anxiety, crying spells, sleep disturbances, difficulty working, and even thoughts of suicide in 25 of the dieting patients in his study. He referred to this cluster of symptoms as “dieting depression” (p. 82).

Years later, Stunkard and Rush (1974) revisited “dieting depression” and found those most vulnerable to the stress of dieting were those who had been obese since childhood. They concluded that, at least for some who were “biologically vulnerable,” dropping out of a weight loss program “was a highly adaptive method for coping with impending complications” (p. 531). More recently, Lowe and Levine (2005) reviewed the literature on the stress of dieting for the obese and found evidence for a “vulnerability to emotional eating” (p. 797). Sometimes, especially for women, dieting “promotes unrealistic expectations” regarding their ultimate shape and actual amount of weight that they might lose (p. 797). They also noted that some people became so preoccupied with food that they develop cognitive difficulties in processing information.

Hatzenbuehler et al. (2008) used data from Wave 2 of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC),

a nationally representative sample of adults in the United States, to investigate the relationship of perceived weight discrimination to psychiatric symptoms among overweight and obese adults. These researchers found that perceived weight discrimination is associated with “substantial psychiatric morbidity and comorbidity” (p. 2033) that was not even mitigated by a social support system and was “potentially harmful” to patients regardless of their actual weight by BMI (p. 2037). Furthermore, they found in their sample of over 22,000 people, that “results showed a stark, step-wise gradient” (p. 2037) in their subjects’ perceived weight discrimination with increasing levels of obesity in both men and women. As is typical of other studies, women reported more perceived weight discrimination. In their study, over half of those who responded met criteria for an Axis I diagnosis, including all the mood and anxiety disorders, as well as nicotine, drug, and alcohol dependence (p. 2036).

Many other studies have also confirmed the relationship of psychiatric symptomatology (and particularly mood disorders) to obesity (McElroy et al., 2004; Luppino et al., 2010; Megna et al., 2011; Dave et al., 2011). The relationship, though, is a complicated one, and as mentioned above, it is often difficult to ascertain whether obesity causes psychiatric symptoms or psychiatric symptoms cause obesity, i.e., which is causal and which is consequence (Karasu & Karasu, 2010, pp. 181–183). Further, many of psychotropic medications cause substantial weight gain in patients (Karasu and Karasu, 2010, pp. 241–248). Overweight and obesity are found in some subtypes of mood disorders, whereas decreased weight is more common in other subtypes (McElroy et al. 2004). In general, the “most methodologically sound clinical studies” indicate mood disorders are common in those who are *seeking treatment* for obesity (especially severe obesity), and being overweight or obese is common in those *seeking treatment* for certain mood disorders, but most overweight or obese people in large *community studies* do not have mood disorders (McElroy et al., 2004, p. 644).

CONCLUSION

Obesity is a complex, chronic disorder with potentially serious medical and psychological morbidity. In essence, obesity is the physiological *result* of an energy imbalance, whether from an increased intake and/or decreased expenditure of calories, and it *results in* the accumulation of adipose (fat) tissue. Environmental, neuro-endocrinological, perinatal, genetic, psychosocial, and dietary factors contribute to the development of

obesity. During the past thirty years the prevalence of obesity has reached epidemic proportions throughout the world.

There are numerous frameworks for studying obesity. At present we classify it as a medical disorder, but not a psychiatric disorder, though the symptom of overeating, which can result in increased adipose tissue, shares many similarities with a substance abuse disorder. The human behavior of eating involves a complex interaction of mind, brain, and body, i.e., in Damasio's term, the human "minded brain." Psychiatric symptoms, particularly anxiety and depression, may be more common in obese individuals, but it is often difficult to ascertain which is cause and which is consequence. Though some studies have reported common traits, such as higher levels of impulsivity and lower levels of conscientiousness, most have found that personality is generally as diverse in this population as it is in everyone else. Despite the high prevalence of obesity, there is considerable stigma against obese persons, even among health professionals who work with these patients. Prejudice and overt discrimination have a long history and remain significant today.

In our attempt to understand obesity in all its dimensions, we cannot focus *exclusively* on the psychological, as that approach *alone* will never cure or even control obesity. We must always consider the fundamental and complex interaction of genetics and the environment. However, obesity will never be eradicated if we fail to appreciate the enormous supporting role that psychological dimensions contribute to its study.

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