Obesity Stigma and Size Acceptance

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Introduction

Two thirds of adults in the US are now considered overweight or obese (Ogden & Carroll, 2010 adults). This has been described as a public health epidemic and physicians have been called upon to address the crisis (Surgeon General of the United States, 2001). Yet our interactions with obese patients have little long-term effect on their weight (Ockene et al., 1999), leave patients feeling dissatisfied (Wadden et al., 2000), and physicians feeling frustrated and powerless (Campbell, Engel, Timperio, Cooper, & Crawford, 2000).

Physicians' current approaches in treating obese patients are often ineffective (Mann et al., 2007) and detrimental to the doctor-patient relationship (Malterud & Ulrisen, 2011), in part because they are colored by obesity stigma. Obesity stigma is the collection of pervasive negative attitudes, stereotypes, and beliefs about overweight and obese people (Puhl & Brownell, 2006). The foundational premise of obesity stigma is the belief that obesity is primarily caused by the obese individual's choices (Puhl & Brownell, 2003). This belief and other beliefs informed by stigma are widely held by physicians and other healthcare providers (Puhl & Brownell, 2001), but are inconsistent with our current scientific understanding of obesity. The Size Acceptance movement, a social movement advocating for the rights and dignity of people of all sizes, has proposed a new medical approach to obesity, the Health At Every Size (HAES) model (Association for Size Diversity and Health [ASDAH], 2012). The Health At Every Size model is a more appropriate approach to working with obese patients; it acknowledges that weight is not primarily under individual control, de-emphasizes weight loss as a marker of success, supports positive body image, and encourages healthy eating and fitness habits for all patients regardless of weight (Bacon & Aphramor, 2011).

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Obesity Stigma

What is Obesity Stigma?

Physicians are accustomed to thinking about the word "stigma" in clinical terms. To doctors, a stigma is an outward sign of a disease, a clue that can be helpful in diagnosis. In sociology, the word stigma has a different meaning, one that medical professionals should also be aware of if they are to avoid perpetuating prejudice. Sociologist Erving Goffman (1963) defined stigma as "an attribute that is deeply discrediting;" such an attribute reduces a person "from a while and usual person to a tainted, discounted one," (p. 13). Goffman describes three types of stigmatized conditions: bodily disfigurements or differences, character flaws, and racial/tribal/religious affiliation. Examples of stigmatized persons in each of these groups include the physically handicapped, the mentally ill, and members of a minority religious group, respectively.

Today in the US, obesity is a highly stigmatized condition, and as a result, overweight and obese individuals face teasing, bullying, discrimination, and even violence based on their body size (Puhl & Heuer, 2009). Obesity stigma falls into two of Goffman's categories; it is an outwardly obvious body difference, but it is also perceived as a character flaw (Malterud & Ulriksen, 2011) because obese individuals are widely believed to be lazy and weak-willed (Puhl & Heuer, 2009).

Obesity Stigma in Healthcare

Obesity stigma is pervasive in most domains of life, but is especially concerning in the area of healthcare. Several types of studies help to frame the problem. Qualitative studies help to identify themes and concerns for both physicians and patients. Survey data tells us how

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prevalent self-reported anti-fat attitudes are among physicians (and other healthcare providers). Experimental studies aim to uncover any additional bias that providers are unlikely to self-report.

Several qualitative studies have explored the types of negative attitudes held by healthcare professionals and the experiences of obese patients. A review of these studies identified several patterns of beliefs (Malterud & Ulriksen, 2011). Physicians tend to believe that obesity is caused by patients' food and exercise choices, and that obesity management is primarily the responsibility of the patient (Epstein & Ogden, 2005). At the same time, physicians feel that patients want to place responsibility for their weight with the physician (Epstein & Ogden, 2005). This creates a perceived conflict between doctor and patient and makes interactions with obese patients distasteful for physicians. Obese patients, however, feel very responsible for their weight (Brown, Thompson, Tod, & Jones, 2006), experience a great deal of shame for failing to lose weight (Rogge, Greenwald, & Golden, 2004), and feel that their weight loss efforts are often ignored or discounted by healthcare providers (Merril & Grassley, 2008). These feelings of shame, guilt, and disrespect make interacting with physicians a negative experience for obese patients (Malterud & Ulriksen, 2011).

Numerous surveys of physicians have examined how pervasive negative beliefs are about the obese. As the prevalence of obesity in the US has increased, anti-fat attitudes among physicians seem to have increased as well. In 1969, a survey of 100 doctors and medical students found that obese patients were more likely to be viewed as "unintelligent, unsuccessful, inactive, and weak-willed" (Maddox & Liederman, 1969). A 1982 study of family practice physicians showed that one third of respondents reported that obesity was a condition that "aroused feelings of discomfort, reluctance, or dislike." Participants in this study also associated obesity with poor hygiene, noncompliance, hostility, and dishonesty (Klein, Najman, Kohrman,

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& Munro, 1982). In 1987, two-thirds of US family physicians surveyed believed that obese patients lack self-control and 39% agreed that obese patients are lazy (Price, Desmond, Krol, Snyder, & O'Connell, 1987). A 2003 study of over 620 primary care physicians reported that more than half viewed obese patients as awkward, unattractive, ugly, and noncompliant (Foster et al., 2003).

Similar anti-fat biases have been recorded in other countries. Thirty percent of General Practitioners (GPs) surveyed in France reported that they considered their overweight patients to be more lazy and self-indulgent than patients in the normal weight range (Bocquier et al., 2005). Another French study found that physicians are likely to acknowledge that obesity stigma is wide-spread; 73% of the GPs surveyed reported that doctors and other providers hold negative attitudes toward their obese patients (Thuan & Avignon, 2005). A survey of Israeli primary care physicians found that 31% agreed that overweight people tend to be lazier than normal-weight people and 25% agreed that overweight people lack willpower and motivation compared to normal weight people (Fogelman et al., 2002).

Experimental work on physician attitudes towards obese patients has corroborated the survey results. In one experiment, primary care physicians examined the chart of a patient presenting with a migraine. The charts were identical except for patient sex and BMI. As the patient's BMI increased, physicians indicated they would spend less time with the patient and would order more lab tests. Physicians' view of the patient also became more negative with increasing BMI. The negative attitudes extended beyond judgments about the patient; as patient BMI increased physicians were more likely to report lower job satisfaction, less patience, and less desire to help the patient. Furthermore, physician respondents reported that seeing obese

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patients was a waste of their time, and that heavier patients were more annoying than normal weight patients (Helb & Xu, 2001).

Other experimental work has sought to remove self-reporting bias from the research on obesity stigma. To study implicit attitudes, researchers have used the Implicit Association Test, a task which times participants as they sort words and images into categories to uncover hidden biases. Using this technique, Teachman and Brownell (2001) found that even physicians specializing in treating obesity exhibit significant implicit anti-fat bias, even in the absence of explicit bias. They did find however, that the level of bias in this subject group was lower than that in the general population. Schwartz et al. (2003) used the same method to look at anti-fat bias among attendees of a medical conference about obesity. They found that these health professionals exhibited significant anti-fat bias.

Challenging Obesity Stigma

Physicians' negative attitudes towards obese patients are troubling for a number of reasons. Most importantly, these commonly held beliefs about obesity (that it is primarily caused by poor diet and exercise choices and that overweight and obese people could lose weight if they actually tried) are inconsistent with our current scientific understanding of obesity. These attitudes, potentially borne of professional frustration, have developed into an outright prejudice that interferes with the provision of good patient care. In this section, these beliefs about obesity will be challenged.

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MYTH: Obesity is primarily caused by poor individual choices about diet and exercise.

This belief is pervasive across healthcare, but it represents a lack of understanding of the biology, psychology, and sociology of obesity. Obesity is caused by a complex interaction of a multitude of factors, most of which are not modifiable.

The Energy In = Energy Out Model

Obesity is frequently described as a mismatch between calories consumed and calories expended, suggesting that preventing or reversing obesity is a simple process of reducing intake, increasing output, or both (Delaet & Schauer, 2010). Implicit in this theoretical framework is an assumption that energy intake and output are under conscious control, and that individuals who fail to regulate these factors are to blame for their weight.

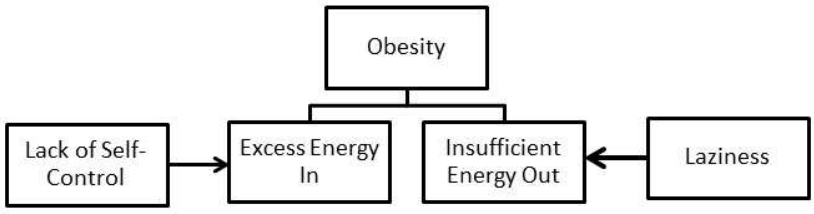

Figure 1. Concept model: the causes of obesity – an overly simplified view.

While this energy balance model is basically correct, it is simplistic, failing to account for a plethora of modifying conditions including genetic, microbiologic, neuroendocrine, environmental, and economic factors. Judgments about the character flaws of obese people are borne of this oversimplification. A more complete and complex concept model is necessary if we are to truly understand obesity. For the purposes of this discussion, each side of the obesity

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equation will be examined separately. Factors affecting the 'energy in' side will be divided into intrinsic and extrinsic or environmental factors. Discussion of the 'energy out' side of the equation will focus on the three types of energy expenditures – basal metabolism, adaptive thermogenesis, and physical activity.

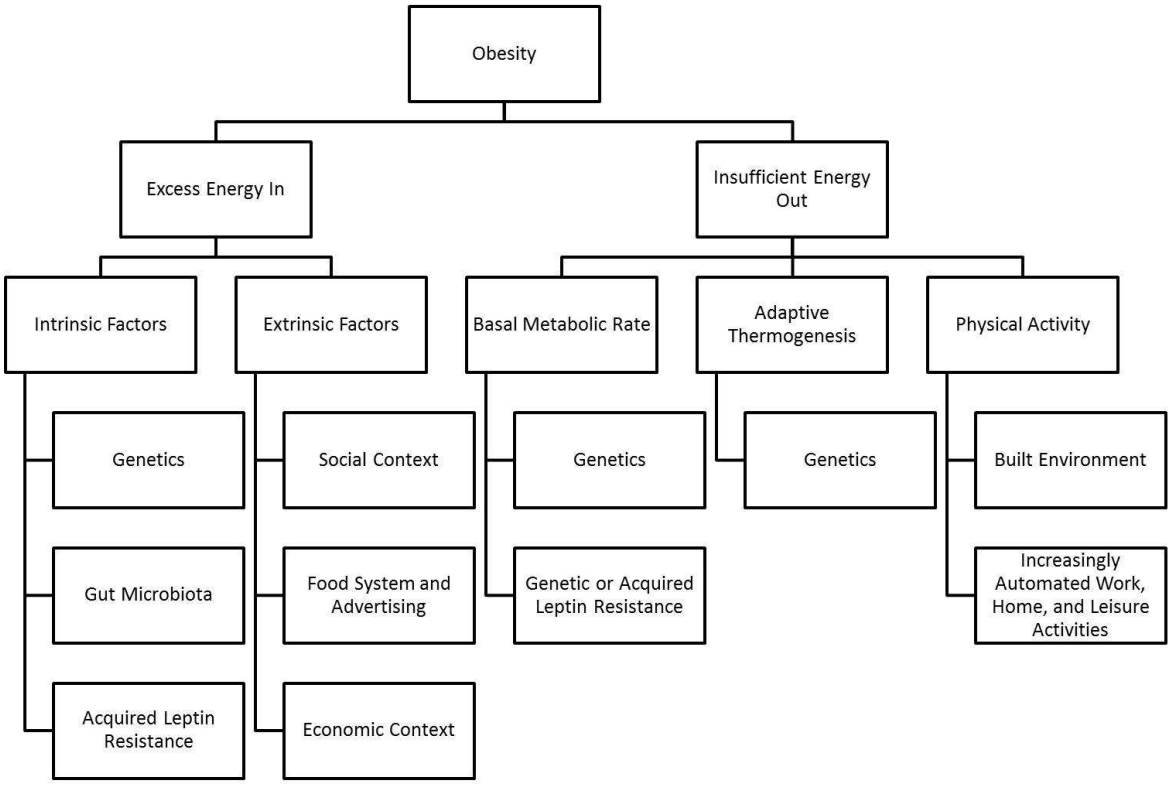

Figure 2. Concept model: the causes of obesity – the current scientific understanding.

Intrinsic Factors Affecting 'Energy In'

In the obesity equation, the "energy in" side refers to calories consumed and digested; what we eat. Certainly we have some control over this, but it is important to remember that making food choices is not solely an intellectual exercise, it is the answering of a fundamental biological drive: hunger.

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The hunger drive is regulated by the hypothalamus, the part of the brain also responsible for thirst, sex drive, and temperature regulation (Friedman, 2004). The neural pathways that control hunger in the hypothalamus are not entirely understood. However, studies of hypothalamic lesions have found that lesions in the medial hypothalamus lead to obesity and lesions in the lateral hypothalamus lead to thinness (Speigelman & Flier, 2001). The hypothalamus works to integrate a host of genetic and neuroendocrine factors aimed at maintaining a genetically determined body weight set point (Martinez, 2000).

Genes for leptin, neuropeptide Y pro-opiomelanocortin, cholecystokinin, and melanin-concentrating hormone all affect the homeostasis of food intake (Martinez, 2000). Leptin is an amino acid signaling molecule that acts in the weight regulation pathway. Normally, leptin is released by adipocytes in response to feeding and interacts with its receptors in the hypothalamus where it acts to decrease hunger (Friedman, 2004). A subset of obese humans and animals suffer from leptin gene mutations leading to low leptin levels. Homozygotes suffer from insatiable appetites and morbid obesity (Farooqi et al., 2002), while heterozygotes have a less severe phenotype (Farooqi et al., 2001).

It is estimated that a full 5-10% of obese humans suffer from low leptin levels. In these individuals, leptin is functioning normally to decrease appetite, but the low levels of circulating leptin mean that appetite remains high. When these individuals are treated with leptin, most respond with markedly decreased appetite (Friedman, 2004).

However, most obese humans actually have high leptin levels, which we would expect to substantially decrease the hunger drive. When these individuals are treated with exogenous leptin, only a subset of them respond with decreased appetite. This pattern indicates that leptin resistance may be to contributing to the increased hunger drive in this group of patients

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(Friedman, 2004). Several signaling molecules have been implicated in this pattern of leptin resistance, including absence of the melanocortin-4 receptor in hypothalamic neurons (Friedman, 2004). It is also possible that leptin resistance is acquired; evidence from rodent models suggests that early exposure to diets high in fat and/or fructose may be associated with later leptin resistance (Scarpace & Zhang, 2009).

Experiments in transgenic mice have shown that mice with high leptin levels remain very thin on a normal diet (as is expected) but on a palatable high-fat diet they no longer respond to leptin (leptin insensitivity) and become obese (Ogus, Ke, Qui, Wang, & Chelab, 2003). The wide availability of palatable and inexpensive high-fat food may be activating a similar gene response in humans.

Though leptin is a major target of current research into hunger and satiety, many other signaling molecules are also involved in the process. For example, cessation of eating behavior is partially mediated by satiety that is signaled by gut distention and release of cholecystokinin (CCK) from the mucosal epithelium of the small intestine, leading to cessation of eating. This is a short-term mechanism – mice injected with CCK do not lose weight over time because they eat more frequent, smaller meals (Speigelman & Flier, 2001).

After an individual has responded to a hunger cue and eaten food, the food must be absorbed before it can be used by the body. A large group of bacteria known as gut microbiota assist with this process by breaking down toxins, synthesizing some vitamins, producing fatty acids, modulating the immune response, and protecting the host from pathogenic bacteria.

Research indicates that the gut microbiota of obese individuals is very different from that of lean individuals, and is more efficient at breaking down fats for storage in the host's adipose tissue (DiBaise et al., 2008). In experiments with mice, researchers found that conventionally

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raised mice had 40% higher body fat content than sterile mice despite identical food consumption. Next, the microbiota from the conventional mice was transplanted into the sterile mice, leading to a 60% increase in body fat while dietary intake remained constant (DeBaise, 2008).

Evidence suggests a similar process is at work in humans, where analysis of gut micribiota has indicated that obese individuals have a higher proportion of bacteria from the division Firmicutes (DiBaise, 2008). Differences in the digestive efficiency of gut microbiota mean that an obese individual and a lean individual ingesting identical numbers of calories will actually absorb different numbers of calories.

Extrinsic Factors Affecting 'Energy In'

Biochemical regulation of hunger and the effects of gut microbiota are powerful intrinsic factors affecting the 'energy in' side of the obesity equation, and neither is under the control of will-power. Extrinsic factors also play a crucial role in this energy balance. It is impossible to thoughtfully examine obesity in the United States without examining the US food system. The food system does not only represent an infrastructure for producing food and transporting it to market, but also a multi-billion dollar commercial enterprise.

First, there are the concrete problems of access and affordability. Too many Americans live in food deserts, usually impoverished neighborhoods without places to buy healthy groceries. Without resources to access transportation, food desert residents are forced to buy less healthy prepared foods from convenience stores or fast food restaurants (Morris, Neuhauser, & Campbell, 1992). A study of African American adults found that those who lived within a census tract with a supermarket were more likely to get the daily recommended servings of both

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fruits and vegetables (Moreland, Wing, & Diez Roux, 2002). Another study found that opening a supermarket in a food desert leads to increased fruit and vegetable consumption by area residents (Wrigley, Warm, & Margetts, 2003). A study of US food stamp recipients found that greater than 90% of recipients did their primary food shopping at a grocery store, but even among those who went to the grocery store, living further away from the store was associated with decreased fruit consumption (Rose & Richards, 2004).

Food pricing is another huge issue. The economics of mass production (not to mention government subsidization of corn and other crops) has made many prepared foods far less expensive than fresh fruits or vegetables. As energy-density of food increases, energy-cost decreases, making energy-dense refined grains, fats, and sweets the cheapest food options (Drewnowski & Darmon, 2005).

Does the lower price of energy-dense food make people more likely to purchase it? While many continue to couch this debate in terms of personal choice and responsibility, economics provide a different theoretical framework. Price elasticity of demand is an economic measure showing how responsive demand is to changes in price. At high elasticities (greater than one), decreases in price dramatically increase demand. At low elasticities (less than one), decreases in price increase demand less dramatically and increases in price decrease demand less dramatically. In general, products necessary for survival have less price elasticity of demand than luxury goods. A meta-analysis of 160 economic studies on food price elasticity found that while price elasticity is on average less than one in all food categories, it is greatest for some of the least healthy food categories, including prepared food away from home and soft drinks, suggesting that the comparatively low price of these items is helping to drive increased consumption (Andreyeva, Long, & Brownell, 2010). Food prices in the US have been falling

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about 0.2% per year since World War II, with the exception of a small increase in the 1970's, and in response consumption has increased (Lakdawalla & Philipson, 2009).

The effects of the food system extend beyond concrete issues of access and affordability; the effects of marketing are profound and deserve to be examined. The food and beverage industry spent $7.3 billion on advertising in 1999, and in 2000 food marketing represented 7.7% of the US GDP (Harris, Kaufman, Martinez, & Price, 2002). By 2006, the food and beverage industry spent over $10 billion on advertising to children alone (McGinnis, Gootman, & Kraak, 2006).

The foods and beverages being promoted by these advertisements are not healthful. In one study of Saturday morning children's programming, 44% of food advertisements were for fats or sweets and 11% were for fast food restaurants. Sweetened breakfast cereal was the single most frequently advertised item, and there were zero advertisements for fruits or vegetables (Kotz & Story, 1994). Gamble and Cotunga's (1999) review of food advertising targeting children over from 1974 to 1999 found that the foods advertised were consistently high-fat and that the nutritional content of advertised foods has not improved over that time span despite increasing public awareness of the importance of healthy diets.

Studies show that all this marketing is effective at influencing several levels of consumer behavior, and these effects have been especially well-studied in children. A review of research on the effects of food advertising on pre-school and school age children concludes that advertising increases the number of brand-specific food requests that children make and that the frequency of these requests is directly related to the frequency of advertising (Coon & Tucker, 2002). In general, exposure to food advertisements has shown to foster "more favorable attitudes, preferences, and behaviors towards the advertised product," (Story & French, 2004).

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Gorn and Goldberg's experimental study in 1982 aimed to observe the effect of advertising on food choices in more controlled conditions. Five to eight year old children watched a half hour cartoon with five minutes of advertisements for candy and Kool-Aid, advertisements for fruits and fruit juice, public service announcements about healthy eating, or no advertisements. Afterwards, subjects were allowed to select among various snacks. Children in the candy and Kool-Aid group selected the most candy and Kool-Aid and 20% less fruit than the other children.

Several health organizations have recognized the link between advertising exposure and obesity. A report prepared for the UK's Food Standards Agency found that not only does advertising affect consumption and increase daily caloric intake, but that increased exposure to food advertisements is directly associated with increased body weight (Hastings et al., 2003). The World Health Organization (2003) has examined this evidence and considers the marketing of fast food and junk food to children to be a "probable" factor for increasing the risk of obesity (World Health Organization 2003).

Making Food Choices

Far from being entirely rational decisions, food choices are ultimately determined by the integration of hunger drive, higher cognitive input, options available in the environment, and other sensory and emotional cues, including economic pressures and marketing influences (Speigelman & Flier, 2001). These factors are wide-ranging and difficult for individuals to control. Differential digestion and absorption of food by gut microbiota further complicates the 'energy in' side of the obesity equation (DiBaise et al., 2008). The 'energy out' side of the obesity equation is equally complex.

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Factors Affecting 'Energy Out'

The 'energy out' side of the obesity equation represents how energy from food is used by the body. There are three main tasks for which energy is used in the body: basal metabolic rate, adaptive thermogenesis, and physical activity. The basal metabolic rate (BMR), or resting metabolic rate, is the energy used by the body to complete the collection of biochemical processes that sustain life. Stated another way, the BMR is the energy used by the body at complete (temperature-neutral) rest. Adaptive thermogenesis is energy used by the body to generate heat in response to colder environmental conditions. Lastly, physical activity includes all voluntary movement (Speigelman &Flier, 2001).

Basal Metabolic Rate

BMR is widely variable from one individual to the next, and this person-to-person variability is largely due to genetic differences (Friedman, 2004). Animal models help to demonstrate this variability. In one study, mice genetically predisposed to obesity and normal mice were fed the same number of calories. The genetically obese mice became obese and the normal mice did not. Environmental temperature and physical activity levels were identical in both groups, so the weight differences between the mice were attributable to differences in basal metabolic rate (Friedman, 2004).

In a study of identical human twins, several sets of identical twins were overfed by the same amount. Between twin pairs, there was a large variation in the amount of adipose tissue that developed over the course of the experiment, but within twin pairs, the amount of adipose tissue that developed was consistent, again suggesting that genetic effects on the BMR were responsible for the between-pair variation (Bouchard et al., 1990).

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A complex system of biochemical signaling determines the BMR, and many of the signaling molecules that function to stimulate hunger also act to decrease BMR. For example, neuropeptide Y, which is released in response to starvation and acts in the hypothalamus to stimulate hunger, also acts to decrease the BMR. Low levels of leptin stimulate hunger and also decrease BMR by decreasing reproductive functions, growth, and release of thyroid hormones (Speigelman & Flier, 2001).

Various therapies have been tested in humans with the goal of increasing BMR to induce weight loss. Both dinitrophenol and thyroid hormone successfully increased BMR and induced weight loss but both also had unacceptable side effects (Friedman 2004).

Adaptive Thermogenesis

The second part of the 'energy out' side of the obesity equation is adaptive thermogenesis, the energy used to create heat to maintain body temperature. On a biochemical level, heat is produced during oxidative phosphorylation that has become uncoupled from ATP production. This is a beneficial process in colder environmental conditions, and when excess fuel needs to be burned off (Speigelman & Flier, 2001). Clearly, the uncoupling of oxidative phosphorylation from ATP production cannot be willfully controlled, and is instead mediated by several genes. Two lines of mutant mice are unable to uncouple oxidative phosphorylation from ATP production: BAT mice and UCP-1 mice. A whole category of "energy out" is unavailable to these mice; BAT mice are typically obese while UCP-1 are cold intolerant (Speigelman & Flier, 2001).

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Physical Activity

Physical activity refers to any voluntary movement and is the third part of the 'energy out' side of the equation. Compared to basal metabolic rate and adaptive thermogenesis, physical activity represents a small percentage of total energy expenditure. Physical activity level is affected not only by personal choice, but also by myriad environmental factors.

For humans, the nature of both work and leisure time have undergone a major shift in the past fifty years. Work in the US has become far more sedentary as jobs move away from farm and factory labor and into the service sector. Energy expenditure in work outside the home has decreased an estimated 100kcal/day since 1960 (Church et al., 2011). Housework has also become more automated; clothes washers, dishwashers, and other automated devices have decreased daily energy expenditure by an additional estimated 111kcal/day (Lanningham-Foster, Nysse, & Levine, 2003). The rise of media such as the television and the personal computer means that our leisure time has also become more sedentary (Tucker & Friedman, 1989).

Second, the built environment has also changed in ways that discourage physical activity. The expansion of the suburbs and the decline of public transportation mean that Americans are using personal automobiles to do most of their traveling. Cities are oriented around cars rather than pedestrians in a way that makes it clear that automobile transportation is the default choice. Several studies have shown that individuals living in neighborhoods where the built environment discourages physical activity are indeed at higher risk for obesity (Papas et al., 2007).

In these ways, structural and economic changes have decreased the amount of physical activity we engage in overall. Instead of being incorporated into our work, leisure activities, or travel, physical activity has been sequestered in its own separate activity: exercise

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As exercise became its own separate activity, so too did our understanding that this activity would need a specific space. These spaces, in the form of parks, fitness centers, and pools, are disproportionately situated close to affluent Americans and far from low-income Americans (Papas et al., 2007).

Causes of Obesity

As we have seen, the idea that obesity is caused by poor personal choices is far too simplistic. Widespread failure of self-control is an unlikely cause of the obesity epidemic. Instead, internal factors such as genetics, gut flora, and neuroendocrine changes, as well as external factors such as the structure of work in our economy, the influence of the food manufacturing industry, and the built environment all play a role. Broadly defined, environmental rather than personal changes are most likely to explain why the prevalence of obesity has dramatically increased over the past fifty years. Meanwhile, genetic differences are the most likely explanation for differences in body weight between individuals exposed to similar environmental conditions.

MYTH: Obese and overweight people could lose weight if they actually tried.

As we have just seen, the causes of obesity are by and large not under personal control and therefore cannot be reversed simply by trying harder. However, the question of what causes obesity in the first place and what approaches would work to reverse it are different questions, and so they will be addressed separately. In other words, once people have become obese, can diet and exercise weight-loss programs work?

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It is important to realize that just as the food industry is a multi-billion dollar industry designed to sell people more food than they need, the diet industry is also a corporate interest with considerable advertising influence. Diet industry revenue in the US was $33.3 billion in 1994 and grew to over $55 billion in 2006 (Andreyeva, Puhl, & Brownell, 2008). Though commercial and non-commercial diets are often successful at producing short-term weight loss, evidence that these programs successfully produce long-term weight loss is lacking. Understanding the physiology of hunger and basal metabolic rate helps us understand why long-term weight loss is so rarely successful. After initially losing weight, the body's regulatory mechanisms respond by increasing subconscious hunger drives and decreasing energy expenditure (Friedman, 2004). Decreasing adipose tissue during initial weight loss leads to decreased levels of circulating leptin (Maffei et al., 1995), which increases the hunger drive and decreases energy expenditure in an attempt to return the body to its set-point weight (Friedman & Halaas, 1998). The end result is that after significant weight loss, an individual needs far fewer calories to maintain their new weight than someone who started out at that same weight (Friedman, 2004).

Tsai and Wadden (2005) reviewed studies of commercial and non-profit weight loss programs found that data supporting the efficacy of these programs is lacking. Studies of Weight Watchers, Jenny Craig, LA Weight Loss, Health Management Resources, OPTIFAST, Medifast, eDiets, Take Off Pounds Sensibly, and Overeaters Anonymous were reviewed. Attrition rates in the reviewed studies were high and were not controlled for. Only Weight Watchers had a large, randomized, controlled trial indicating that at two years, participants had lost 3.2% of their starting weight. The marketing for these programs frequently promises much more striking results, a pattern which prompted intervention by the Federal Trade Commission in the 1990's.

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It is not just commercial diet programs that are unlikely to be successful long-term. Study after study of dieting in general has found that long-term weight loss is rare. In one randomized controlled trial, researchers compared four different diet programs to a control group and followed up over two and a half years. They found that the control group did not have statistically significant weight gain, and that the dieters had lost an average of only 3.7lbs (1.7kg) (Jeffery & Wing, 1995). Unfortunately, few other randomized controlled trials of weight loss programs have been completed.

Prospective non-randomized studies follow dieters and non-dieters but allow participants to self-select into those groups. In a review of ten such studies (Mann et al., 2007), only one reported that the dieting group lost weight relative to the non-dieting group after four years of follow-up. Two studies found that dieting led to no change in weight after six months and two and a half years of follow-up. Finally, seven of the studies reviewed found that dieting led to weight gain relative to the non-dieting group.

Observational studies typically follow patients after a diet program but they have no control group and therefore can only demonstrate correlation, not causation. A review of several observational studies found that the average short-term weight loss was 30.8lbs (14kg) immediately after the diet period ended. However, at four year follow up, participants had regained an average of 24.2lbs (11kg). In fact, 41% of participants weighed more four years after dieting than they did at baseline. The actual results may be even less compelling, as the average follow-up rate in the reviewed studies was only 33%. Another methodological problem with these studies is that they used self-reported weights at follow-up, a weighing method which has been associated with weight under-reporting of up to 8.2lbs (3.7kg) (Mann et al., 2007).

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Aside from strict diets which may not be sustainable, even ongoing lifestyle modification programs have not proven effective. One controlled randomized trial comparing an ongoing lifestyle modification group with a control group over three years found that participants and controls both gained an average of 3.5lbs (1.6kg) (Jeffery & French, 1999).

A number of studies indicate that not only is dieting not successful, it may increase the risk of weight gain. A study of 3,553 adults over two years found that women who dieted gained an average of 1kg more than women who did not diet (French & Jeffery, 1994). A Finnish study of 7,729 adults found that dieting was predictive of weight gain (Korkeila et al., 1999). A study of 10,554 white and African American adults over six years found that dieters gained about 0.5kg more per year than non-dieters (Juhaeri et al., 2001). In a one year study of 287 young adults, "dietary restraint" was predictive of weight gain for women (Klesges, Isbell, & Klesges, 1992). In 1999, a study comparing adolescent girls who did not diet, practiced dietary restraint, or dieted found that those who practiced dietary restraint had two times the risk of obesity as the non-dieting group and those who dieted had three times higher risk (Stice et al., 1999).

MYTH: Obesity is a serious medical problem.

So far we have seen that obesity is not caused by personal choices and cannot reliably be reversed by dieting. This leaves us in a rather hopeless place if obesity is a serious health problem. In the healthcare world, obesity has been described as a risk factor (Hubert, Feinleib, McNamara, & Castelli, 1983), a behavior (Epstein et al., 1980), a disease (Jung, 1997), and even an epidemic (Surgeon General, 2001). However, few if any randomized controlled trials of the effects of obesity have been conducted on humans; such a study would involve inducing obesity in a group of study participants. As such, the current body of research on the effects of obesity

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cannot claim that obesity causes any particular outcome; it can only claim that obesity is correlated with the outcome in question. Are these correlative relationships as concerning as we have been led to believe?

BMI and Mortality

Most strikingly, obesity has been correlated with an increased risk of death. The National Institutes of Health (NIH, 1998) publication, "Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults" claims that "mortality begins to increase with BMIs above 25kg/m2…the increase in mortality generally tends to be modest until a BMI of 30kg/m2 is reached," (p. 1). The report states that persons with Body Mass Index (BMI) values over 30 have all-cause mortality rates 50 to 100% higher than those in the normal weight range. These conclusions are problematic for several reasons, starting with the use of the BMI as the diagnostic metric for obesity.

The History of the Body Mass Index

The Body Mass Index, originally called the Quetelet Index, was developed by Belgian mathematician and social scientist Adolphe Quetelet in 1832. Quetelet's interest in developing this ratio was not to describe a diagnostic parameter for obesity, but to prove that characteristics of human bodies followed a Gaussian distribution. He developed the ratio by studying published height-weight data from mainly Anglo-Saxon populations. His work helped to popularize the idea that mathematical principles could be used to describe both the natural world and social phenomena (Eknoyan 2007).

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In 1937, the Metropolitan Life Insurance Company published height-weight tables in an attempt to quantify obesity without the benefit of the BMI. Statistician and company vice-president Louis I. Dublin had noted an increase in life insurance claims associated with excess weight and wanted to stratify policy holders in terms of their weight-related risk. He did not want to inappropriately assign all tall people to higher risk levels, and so he recorded weight for height. He defined those who weighed 20-25% more than average as "undesirable" and those who weighed 70-100% more than average "morbidly obese" (Eknoyan, 2007). This was not an optimal metric for use by healthcare professionals because it represented data from a relatively restricted pool of white upper middle class policy-holders.

The BMI re-emerged to solve this problem in 1972. By then, increased body fat percentage had been associated with cardiovascular disease. Ancel Keys published a paper comparing several indices of height and weight to see which one correlated best with body fat percentage. He found that the Quetelet Index, which he renamed the Body Mass Index, was the best correlated with body fat percentage and the least dependent on height. He did note that skin caliper measurements might be more accurate on an individual basis, but that the BMI was an acceptable alternative that did not require special measurement equipment. The BMI was also an internal ratio; it did not depend on the weight for height distribution of a reference population (Keys et al., 1972).

In 1985, the National Institutes of Health released a consensus statement urging clinicians and researchers to adopt BMI as a diagnostic standard for obesity. At the time, a BMI of ≥ 27.8 for men and ≥ 27.3 for women was suggested as the diagnostic criterion for obesity, given that BMI relates to body fat percentage differently in men and women. The authors recognized that since BMI was estimating body fat percentage and body fat percentage was a continuous

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variable, "all quantitative definitions of obesity must be arbitrary," (p. 3). The BMI cutoffs suggested were based on expert consensus that body weight 20% higher than ideal represented a real health risk (NIH, 1985).

The present BMI cutoffs of 25 and 30 were adopted by the National Institutes of Health in 1998. The cutoffs for men and women were consolidated and the "overweight" category was added (NIH, 1998). For children and adolescents, the cutoff of ≥ the 95th percentile for BMI-for-age growth charts was suggested by the US Preventive Services Task Force in 2005. At the time, children in this group were termed "overweight" rather than "obese," in part to avoid the stigma of the "obese" label (Whitlock et al., 2005). The designation was changed to "obese" in the 2007-2008 National Health And Nutrition Examination Survey (NHANES) reports (Ogden & Carroll, 2010 children). The rationale for this cutoff is not mortality-driven, but is correlated with obesity in adulthood (Whitlock et al., 2005).

Minimizing Mortality

Since the adoption of the cutoffs of 25 and 30 for the diagnosis of overweight and obesity, respectively, further studies of the relationship between BMI and mortality have not replicated those results. In their own report, the NIH admits that the mortality vs. BMI curve is J-shaped – that there is increased mortality at lower BMI, usually below 20 but sometimes within the normal-weight range (NIH, 1998).

Several studies have found that the lowest mortality rates often fall in the BMI range currently labeled 'overweight,' BMI values of 25-30. A large 2005 (Flegal, Graubard, Williamson, & Gail) study using NHANES data found that rather than being associated with excess mortality, overweight was associated with a decrease of 86,000 deaths in the US. The

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NIH's claim that overweight and obesity represent increased mortality risk is especially problematic for groups other than young, white, males.

A study of 55-75 year olds that was cited in the NIH's own report found the lowest mortality rate for this age group exists in the 25-30 BMI range (Durazo-Arvizu et al., 1998). In African-Americans the bottom of the J-shaped mortality vs. BMI curve is 1-3 kg/m2 higher than it is for whites. The optimal (meaning lowest associated mortality) BMI for black men is 27.1, while the optimal BMI for black women is 26.8, both of which would be categorized as 'overweight' by well-meaning physicians following the current guidelines (Wienpahl, Ragland, & Sidney, 1990). The effect is even more staggering in the Pima people (Native Americans indigenous to southern Arizona); the optimal BMI for Pima men is 35-40 (considered obese by current guidelines) and no relationship could be found between mortality and BMI for Pima women (Hanson et al., 1995).

Despite these findings in their own published report, the NIH (1998) still concludes that "there are no studies that would support the exclusion of any racial/ethnic group from the current definitions of obesity," (p. 25). This conclusion suggests that a doctor caring for an African American man with a BMI of 27 should encourage him to lose weight, even though such efforts are unlikely to be successful and his weight-related mortality rate is already minimized.

Several studies have suggested that physical fitness (defined as time spent exercising or physical endurance) is a more important predictor of mortality than BMI. A review of these studies concludes that obese individuals who exercise actually have lower mortality risk than sedentary thin people (Blair & Brodney, 1999).

A recent study of more than 11,000 US adults examined the mortality effects of adopting four healthy lifestyle habits: eating five or more fruits and vegetables daily, exercising regularly,

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consuming alcohol in moderation, and not smoking. Researchers adjusted for age, sex, race, education, and marital status, and stratified the results based on weight category. They found that the obese group had a markedly increased risk of death only amongst people who had adopted none of the four studied lifestyle habits. In the groups that had adopted more healthy habits, increased mortality associated with obesity was less dramatic, and in the group that had adopted all four healthy habits, there was no mortality difference between obese individuals and normal weight individuals. This indicates that healthy lifestyle behaviors decrease mortality risk independent of weight loss (Matheson, King, & Everett, 2012).

BMI and Morbidity

The NIH publication "Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults" reports that overweight and obesity are associated with increased risk of hypertension, hyperlipidemia, type 2 diabetes, coronary heart disease (CHD), stroke, gallbladder disease, osteoarthritis, sleep apnea and respiratory problems, some types of cancer, pregnancy complications, menstrual irregularities, stress incontinence, and depression (NIH, 1998). However, again, these are associations, and the studies cited by the NIH largely do not control for many potential confounders such as dietary quality, fitness level, education level, or socioeconomic status.

The NIH suggests that all overweight and obese individuals are inherently unhealthy. However, a 2008 study (Wildman et al.) of obese, overweight, and normal weight adults in the NHANES examined cardiometabolic health markers such as blood pressure, triglycerides, fasting plasma glucose, C-reactive protein, and LDL cholesterol. This study found that 51.3% of overweight adults and 31.7% of obese adults were metabolically healthy. Meanwhile, the

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converse belief that thin people must be healthy was also challenged – 23.5% of normal weight participants were metabolically unhealthy. Unsurprisingly, older age and lower physical activity levels were independent correlates of unhealthy metabolic markers.

Conclusions: The Current Scientific Understanding of Obesity

The preceding sections have presented an understanding of obesity that challenges conventional wisdom. We have seen that the causes of obesity are multifactor, including genetic, biochemical, microbiological, and environmental factors not under an individual's control. Likewise, we have seen that while a few people are able to lose weight and maintain that weight loss long term, substantial weight loss is not a realistic goal for the majority of overweight and obese Americans. As far as the effects of obesity, we have seen that the BMI is not a concrete predictor of morbidity and mortality, but rather an imperfect estimator of body fat percentage. Furthermore, though there are health effects associated with excess body fat, these represent correlation rather than causation, and stronger predictive relationships exist between morbidity and mortality and other factors, such as cardiorespiratory fitness.

Where Does Obesity Stigma Come From?

Physicians as a group generally pride themselves on their scientific discipline, on using evidence-based approaches to diseases. How then, has this group of professionals come to adopt so many faulty conclusions about obesity and obese people?

Sociological research by Crandall (1994) uses Attribution Theory to explain the origin of obesity stigma. Rather than attempting to explain stigma through the actions and goals of social groups, Attribution Theory focuses on individuals maintaining their own sense of ego and safety.

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Attribution Theory holds that people see outcomes and try to link them to causes in order to psychologically distance themselves from the possibility of poor outcomes for themselves. The idea that poor personal choices lead to undesirable outcomes is supported by conservative American social values. These values, based on the Protestant work ethic, support individualism, self-determination, and self-discipline and are part of a world view that asserts that good deeds are rewarded and bad deeds are punished (Crandall, 1994). Interestingly, physicians reportedly value the Protestant work ethic even more highly than the general US population, perhaps due in part to their own experience with rigorous medical training (Klein, 1982). Studies of those who hold anti-fat attitudes have found that they are associated with political conservatism, authoritarianism, and favoring traditional sex roles (Crandall, 1994).

How do individuals use Attribution Theory to distance themselves from poor outcomes? For example, person A is diagnosed with lung cancer, person B may attribute this to person A's cigarette smoking. Since person B does not smoke cigarettes, he feels less vulnerable to lung cancer. He is spared psychological stress of accepting that he could also be diagnosed with cancer.

In the case of obesity, excess body fat is the poor outcome, not because of associated ill health effects, but simply because it deviates from the thin body ideal. Attribution theory requires an explanation for obesity that is under personal control. The popular consensus is that obese and overweight individuals are responsible for their own condition represents what Crandall terms a 'justification ideology' (Crandall, 1994).

From this justification ideology, many negative character traits are extrapolated. As we have seen, overweight people are likely to be viewed as lazy, incompetent, greedy, and lacking in self-control (Puhl & Heuer, 2009). In this way, obesity stigma is similar to symbolic racism.

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However, while racism is not socially acceptable, obesity stigma is justifiable, specifically because so many people believe that obesity is a choice (Crandall, 1994).

There are many health conditions that can be attributed to poor personal choices (such as the lung cancer example above). Among healthcare providers, obesity may be the most stigmatized of these conditions precisely because obesity treatments are so ineffective. Providers who feel frustrated that they aren't able to offer a cure are more likely to blame the victim.

Why is Obesity Stigma a Problem?

Obesity stigma represents a wide-spread prejudice directed against a group that includes two-thirds of Americans. This prejudice is analogous to racism, sexism, and homophobia and is damaging to our society in many of the same ways (Crandall, 1994). The societal costs of obesity stigma are high and include psychological effects such as depression and anxiety (Puhl & Latner, 2007), as well as size-based discrimination in education, employment, housing, parental rights, and other areas (Puhl & Heuer, 2009).

Obesity stigma in healthcare is problematic because it causes us to violate the ethical principles of beneficence and non-maleficence (Bacon & Aphramor, 2011). Obesity stigma informs an approach to treating obese patients that is not working. Physicians and health policy makers are focused on promoting weight loss (Surgeon General, 2001), but diet and exercise are rarely successful in achieving long-term weight loss (Mann et al., 2007). Meanwhile, we are ignoring other interventions that may actually help the fight against diabetes, heart disease, and other chronic conditions. More than just being ineffective, the traditional weight loss approach is harmful to patients' overall well-being, damaging self-esteem and promoting disordered eating and body dissatisfaction (Puhl & Brownell, 2006). Negative experiences with physicians are

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prompting many overweight patients to avoid medical care, which makes them less likely to receive important preventive services such as colonoscopies, mammograms, and pap smears (Olson, Schumaker, & Yawn, 1994).

Size Acceptance and a New Approach to Obese Patients

Now that physicians are aware of the problem of obesity stigma, how should we change our practice? The current medical approach to obesity has changed little since Hippocrates suggested that fat men "eat only once a day and…sleep on a hard bed and walk naked as long as possible," (Friedman, 2004). In his commentary on the stigma of obesity, Jeffery Friedman writes, "progress in this area will require that we move beyond this 2,000 year-old prescription and instead develop strategies that are based on 21st century science," (Friedman, 2004).

A sound scientific understanding of obesity is definitely required for appropriate and compassionate care of obese patients. However, given that obesity stigma is such a pervasive and damaging phenomenon, something more than this is also required: a philosophical shift in the way we understand obesity and obese people. Physicians have a great deal to learn from the Size Acceptance movement.

The Size Acceptance Movement and Health At Every Size

The Size Acceptance movement, also called the Fat Acceptance movement, is a social movement advocating for the rights and respect of all people regardless of body size. Several Size Acceptance advocacy groups exist, including the Council on Size and Weight Discrimination (CSWD) and the National Association to Advance Fat Acceptance (NAAFA) (Bacon and Aphramor 2011). NAAFA was founded in 1969, and its mission is to "help build a

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society in which people of every size are accepted with dignity and equality in all aspects of life" (NAAFA, 2011). NAAFA currently focuses on advocating for the rights and dignity of fat people in the areas of healthcare, the workplace, and education, but the organization's overarching goal is to create a society where obesity stigma is not tolerated (NAAFA, 2011).

Another Size Acceptance organization, the Association for Size Diversity and Health, was formed specifically around issues of size advocacy in healthcare and promotes an approach to weight called the Health At Every Size (HAES) model (Bacon and Aphramor 2011). While the traditional medical model holds that obese patients must lose weight in order to be healthy, the HAES approach proposes that health can be achieved through the adoption of healthy behaviors, with or without subsequent weight loss (Miller & Jacob, 2001).

The core principles of HAES include, "accepting and respecting the diversity of body shapes and sizes; recognizing that health and well-being are multi-dimensional and that they include physical, social, spiritual, occupational, emotional, and intellectual aspects; promoting all aspects of health and well-being for people of all sizes; promoting eating in a manner which balances individual nutrition needs, hunger, satiety, appetite, and pleasure; and promoting individually appropriate, enjoyable, life-enhancing physical activity, rather than exercise that is focused on a goal of weight loss" (ASDAH, 2012).

The HAES approach acknowledges that obese patients often suffer more from the psychological sequelae of dealing with both internal and external size prejudice than from the physiologic effects of obesity itself. As such, HAES uses a cognitive-behavioral approach to develop positive cognitions, identify body needs, and cultivate positive experiences with physical activity (Miller & Jacob, 2001).

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Physicians can and should adopt the principles of Size Acceptance and HAES in the treatment of our patients, as managers or policy-makers in our practices, and as advocates for the health of our communities.

Using Health At Every Size with Patients

Within the doctor-patient relationship, physicians should avoid perpetuating obesity stigma by treating our overweight patients with respect. Open, honest communication is important. We should acknowledge that obesity stigma exists and is common in healthcare and tell our patients that we are making efforts to make all patients feel respected and supported. Acknowledging that, "degrading attitudes and behavior creating dignity violation are not always recognized by the person in power," (Malterud & Ulriksen, 2011) we should invite feedback on our efforts by encouraging patients to let us know how we are doing.

Specifically, physicians can demonstrate understanding of obesity stigma by acknowledging that weight is not entirely under individual control. Discussing with patients the genetic, microbiological, neuroendocrine, and environmental factors that contribute to weight may help patients feel more supported and improve their self-esteem. This is likely to be contrary to the patient's own conceptual model of obesity; many obese patients blame themselves for their weight and experience a great deal of shame when they are unable to lose weight (Merrill & Grassley, 2008). A physician challenging this model may help the patient make steps towards accepting his or her own body.

Adopting HAES means focusing on overall well-being instead of on weight and weight loss (Miller & Jacob, 2001). Instead of measuring BMI, physicians should take a detailed diet and exercise history for every patient regardless of weight. Actively seeking to listen and

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understand the struggles of obese patients is important. Studies have shown that even well-intentioned advice can feel condescending when it does not seem to be delivered in a context of understanding the patient's experience (Malterud & Ulriksen, 2011). For example, a woman attempting to lose weight in preparation for bariatric surgery reported that her primary care physician suggested that she "just drink more water," and "push away from the table." The patient experienced this advice to be simplistic and condescending, responding, "Wow; if only I had thought of that before!" (Reed, 2003).

Another common complaint of obese patients is that their physicians relate all of their medical problems back to obesity, sometimes even refusing to treat these problems unless the patient first loses weight (Brown, 2006, Merril & Grassley, 2008). Using the HAES model, physicians should treat weight as a potentially related but ultimately un-modifiable risk factor for other conditions, much the way we understand that arthritis is related to age but still treat arthritis without demanding that our patients first get younger. Instead of recommending weight loss, the HAES intervention involves encouraging body acceptance, supporting intuitive eating, and advocating pleasurable physical activity (Bacon & Aphramor, 2011).

Physicians can encourage body acceptance and self-acceptance by providing brief cognitive-behavioral interventions, such as helping patients foster positive cognitions about their bodies. For example, when a patient feels frustrated that she can't fit into the clothes she likes, she can counter this negative cognition with a positive one, such as thanking her body for allowing her to participate in an activity she enjoys, like dancing. The approach of fostering positive body cognitions has come out of eating disorder treatment. Studies have shown that self-acceptance leads to more self-care behaviors and lower reported stress (Leary et al., 2007, Gross & Allen, 2010).

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Supporting intuitive eating is the second arm of the HAES intervention. Eating intuitively as opposed to restrictively means allowing oneself to eat any food. Rather than attempting to cognitively control their food choices, intuitive eaters pay attention to hunger cues and note how various foods make them feel across various categories, including "mood, concentration, energy levels, fullness, ease of bowel movements, comfort eating, appetite, satiety, hunger, and pleasure," (Bacon & Aphramor 2011).

The third part of the HAES intervention is supporting pleasurable physical activity. Rather than prescribing a set amount of physical activity with the goal of calorie expenditure, HAES promotes identifying and participating in physical activities that are intrinsically enjoyable. These enjoyable physical activities have cardiovascular benefits but also foster a positive body relationship and promote self-care (Bacon & Aphramor, 2011).

Efficacy of the Health At Every Size Program

Practitioners who are used to the traditional weight-loss model of obesity treatment often worry that the HAES approach will be ineffective or may lead to runaway weight gain. The evidence supports the opposite. Though the implementation and study of the HAES program is still in its infancy, several high quality randomized controlled trials have demonstrated its efficacy (Bacon & Aphramor, 2011).

A randomized controlled trial studying obese women compared two non-dieting interventions (an educational program and a psychoeducational intervention similar to HAES) with a control group. There was no significant change in weight or blood pressure in any of the groups, but the psychoeducational group improved in measures of mental well-being including self-esteem, restraint, and body dissatisfaction (Ciliska, 1998).

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A study of morbidly obese women (Tanco, 1998) compared a cognitive treatment program similar to HAES to a behavioral therapy weight loss program and a wait list control. The cognitive treatment group saw decreases in depression and anxiety as well as disordered eating behaviors and increases in self-control. The weight loss group saw none of these psychological improvements. Though weight loss was not a goal of the cognitive treatment, both treatment groups reported significant weight loss.

A British study (Rapoport, 2000) of overweight women compared a modified cognitive-behavioral approach similar to HAES to a standard cognitive-behavioral intervention in which weight loss was still the stated goal. Both programs increased physical activity and fitness, improved nutritional content of dietary intake, improved markers of cardiovascular risk including total cholesterol, fasting glucose, and blood pressure.

A 2005 study (Bacon) of female chronic dieters compared a six month diet program to a six month HAES intervention. Attrition rate was 42% in the diet group and 8% in the HAES group. Body weight and BMI remained stable in the HAES group throughout treatment and follow-up; the diet group initially lost weight but regained it by the two year follow-up. At follow-up, the HAES group demonstrated improvements in total cholesterol, LDL cholesterol, and systolic blood pressure. The diet group had initial improvements in LDL cholesterol and systolic blood pressure, but these were not sustained. Cognitive restraint/restricted eating scores were low in both groups at baseline, but increased in the diet group and decreased in the HAES group. The study also looked at scores on the Eating Disorder Inventory-2 questionnaire; the HAES group improved significantly in drive for thinness, binge eating behavior, body dissatisfaction, and interoceptive awareness. Meanwhile, the diet group showed improvement in three scales at six months, but had returned to baseline scores by two year follow-up. Mental

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health metrics at the end of the two year follow-up showed that the HAES group improved in depression, self-esteem, and body-image avoidance behavior. The diet group showed no long-term depression improvement, significantly lower self-esteem, and no significant improvement in body-image avoidance behavior. In participant evaluations, 100% of the HAES respondents and 47% of the dieting respondents agreed or strongly agreed that participation "helped me feel better about myself" (Bacon, 2005).

These studies demonstrate that HAES is likely to be effective in improving health and wellbeing and is not likely to cause additional weight gain. More research on HAES interventions is needed, especially research including men as participants. In addition to implementing HAES interventions in the treatment of patients, physicians should also use their understanding of obesity stigma and size acceptance to inform the way they manage their own practices and advocate for the health of their communities.

Size Acceptance in Practice Management

As the managers of medical practices, there are a number of changes physicians can make to take better care of their obese patients. Educating our co-workers and staff about obesity stigma and size acceptance is an important first step.

Practice policies should be examined and reassessed. For example, many patients find being weighed at the doctor's office to be a humiliating experience. One study found that more than 12% of obese women have delayed treatment or cancelled a medical appointment to avoid this experience (Olson et al., 1994). Making weighing optional, rather than a default procedure can go a long way towards making the healthcare experience a more positive one.

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The physical infrastructure of the office should also be examined. Waiting room seats should be large enough to accommodate all bodies. Large sized gowns, exam tables, and blood pressure cuffs should be readily available. Patients in one study described the lack of these accommodations as making healthcare access "a battle," (Merrill & Grassley, 2008). Patient education information about BMI or weight loss should be removed.

Advocating for Public Health

Physicians should resist public health messages that participate in fat-shaming. For example, a recent Georgia ad campaign by Strong4Life featured pictures of obese children with messages such as, "Chubby kids may not outlive their parents," and "Big bones didn't make him this way, big meals did," (Kotz, 2012). These public health messages purport to raise awareness of obesity as an issue, but in fact provide no information and are damaging to the psychological health of overweight children and adults.

Instead of focusing on blaming individuals, physicians should act as advocates for the health of their community by supporting efforts that address the systemic and environmental causes of the obesity epidemic. Keeping in mind that health involves overall well-being, physicians should support programs that aim to minimize economic disparity, reform the food system, and create an environment conducive to physical activity. The language of such public health efforts should stress the need to make the environment healthier for everyone, rather than utilizing the language of the "war on obesity," which is itself stigmatizing (Lewis et al., 2010).

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Conclusion

As we have seen, obesity stigma is a prejudice against overweight and obese people that has long been justified by a faulty understanding of the physiologic causes and consequences of body weight. However, our current scientific understanding of obesity indicates that obesity is not caused by personal choices and cannot be overcome with willpower alone. Furthermore, the health outcomes associated with obesity are only associations and do not represent a causal relationship. Evidence suggests that obese individuals who practice healthy lifestyle habits can be just as healthy as their thinner counterparts.

The medical community has played a powerful role in both justifying obesity stigma and perpetuating it. The current NIH treatment guidelines for obesity are not only ineffective, they are damaging to the physical and mental health of obese patients. Though prejudice against overweight and obese people is widely socially accepted, physicians are uniquely positioned and ethically obligated to counter this prejudice. Adopting the Health At Every Size model of medicine and joining the Fat Acceptance movement are concrete steps that physicians can take toward addressing this prejudice.

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