



Obesity Bias, Medical Technology, and the Hormonal Hypothesis: Should We Stop Demonizing Fat People?

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ABSTRACT

There is adequate evidence to demonstrate that bias toward obese individuals by health professionals is common. Bias predisposes to errors in medical judgment and care. There is also evidence to show that the pathophysiology of obesity is more complex than eating too much and moving too little. Widespread obesity is a new phenomenon in the United States and reflects changes in culture, including food, at many levels. The modern abundance of low-cost, available, palatable, energy-dense processed foods and the ability of these foods to activate central nervous system centers that drive food preference and overeating appear to play an important role in the obesity epidemic. The usual hormonal systems that promote body weight homeostasis appear to have been counterbalanced by pleasurable (hedonic) influences these foods generate in higher neurologic networks, including the limbic system. The use of medical technology, such as functional magnetic resonance imaging, to quantitate hedonic responses to food, enhance taste, and effectively develop and market commercial food products has produced new areas of ethical concern and opportunities to better understand eating and satiety. These developments further demonstrate the urgency to address the bias that exists toward obese patients.

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AN OLD PROBLEM

In his book, *Seeing Patients: Unconscious Bias in Healthcare*, Augustus A. White, III, MD, notes, “Researchers looking into the effect of prejudice and stereotyping on the delivery of healthcare have noted an influence on reasoning and decision making.”¹ He goes on to say, “Doctors are as guilty of stereotyping and prejudice that flows from it as are other people. Those prejudices show up in the hospital and office where patients are exposed to them and in the medical school where students are present.”¹ The bias of health

professionals toward obese patients has been both recognized and questioned for years.² Advances in neuroscience suggest that questioning is well taken.

EPIDEMIC OBESITY

Approximately 35% of Americans aged more than 20 years are obese.³ Recent estimates suggest that by 2030, 42% of Americans will be obese, and 11% will be extremely obese.⁴ Even more startling are estimates that approximately 17% (or 12.7 million) of children and adolescents aged 2 to 19 years are obese now.⁵ The immediate health consequences of this epidemic have already affected military readiness, and the projected downstream detriment of obesity to the health and the quality of life of Americans and the American economy is staggering. For instance, the \$313 million estimated cost of caring for patients with cardiovascular disease in 2009 is projected to increase to \$1.48 trillion by 2030.⁶

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A RECENT PROBLEM WITH NO GOOD SOLUTION

Widespread obesity in the United States is a recent phenomenon. Less than 20% of American adults were obese in 1960, and the prevalence of obesity remained relatively stable until 1980. Between 1980 and 1990, the prevalence of obesity jumped 8 percentage points and took off from there to the present levels.⁷ Attempts at diet and exercise to reverse obesity once it has occurred are often futile, and drug treatment of obesity is in its infancy and is relatively ineffective.⁸ Bariatric surgery, the more effective approach, is not without risks, is costly, and requires life-long follow-up for complications. Thus, physicians are left with few simple approaches to prevent or treat the well-known consequences of being overweight or obese. Trying to deal with obesity in the clinic under these circumstances is disheartening for patients and physicians.

CLINICAL SIGNIFICANCE

- Bias results in poor decision making and medical errors.
- Medical professionals are known to have bias against obese individuals.
- A better understanding of the pathophysiology of obesity may assist health professionals in moving beyond the bias that exists.

BIAS AND THE SET POINT

Perhaps the limited treatment options for obesity, predictable complications and comorbidities, recidivism, requirement for chronic care, and a sense of futility explain the documented implied negative bias and avoidance behaviors of health professionals toward obese patients.⁹ Limited knowledge and training in nutrition, limited time and support for nutritional counseling, the antiquated but still popular “set-point theory” of body weight control, and the consensus that obese people lack self-control are among likely factors contributing to this problem.¹⁰

The set-point theory suggests that weight is regulated around a stable set point and deviations from that weight normally result in innate biologic corrective measures.¹¹ Moreover, gluttony and disease are the factors most likely to upset this innate homeostasis. Recently, much has been learned about hunger, satiety, and eating to suggest that the control mechanisms of weight gain and loss are more complex than either factor.¹²

THE CEREBRUM TRUMPS THE HYPOTHALAMUS

Eating behaviors appear subject to both central nervous system and peripheral organ (gut, pancreas, and fat) inputs (Figure 1). To greatly simplify, body weight homeostasis, the central concept of the set-point theory, appears to be regulated by hormonal signals from the gastrointestinal tract (ghrelin, cholecystokinin, peptide YY) and adipocytes (leptin) to several regions of the hypothalamus (arcuate, lateral, and ventromedial nuclei), as well as the brainstem.¹³ These loci in the brain are responsible for hunger and energy balance and the release of signaling

proteins for both increasing (neuropeptide y and agouti y-related peptide) and decreasing (pro-opiomelanocortin and serotonin) food intake. Now we know that homeostatic mechanisms may be trumped by the human cerebral sensory/reward system of which calorie-dense food, rich in sugar, salt, and fat, is a stimulant.

This scenario appears to have developed as a process of evolution. The “thrifty gene” hypothesis suggests that those individuals best able to survive seasonal variations in food availability were those best able to store energy as fat.¹⁴ Archeological studies show that modern hunter-gathers changed preferences from the lean meat they obtained from small game to higher fat meat they obtained from larger animals and began to actively pursue them.¹⁵

This transition in food preference is supported by analysis of bone marrow specimens from prehistoric hunter-gatherers that show higher levels of fat than older species. An extreme example is today’s Alaskan Arctic Eskimos who have a diet of 50% fat.¹⁶ Modern humans also evolved intestinal adaptations to facilitate higher intake of meat and other nutritionally dense foods.¹⁷

While the brain was doubling in size over the last 2 million years, the energy requirement associated with that development also appears to have promoted the evolution of the brain corticolimbic pathways responsive to “hedonic” influences to some foods. Those influences can override normal homeostatic processes for weight control. As noted by one author, “The non-homeostatic brain reward circuitry that was acquired during evolution to seek out and eat as much high nutrient, calorie dense food as possible is able to overrule the physiological inhibitory

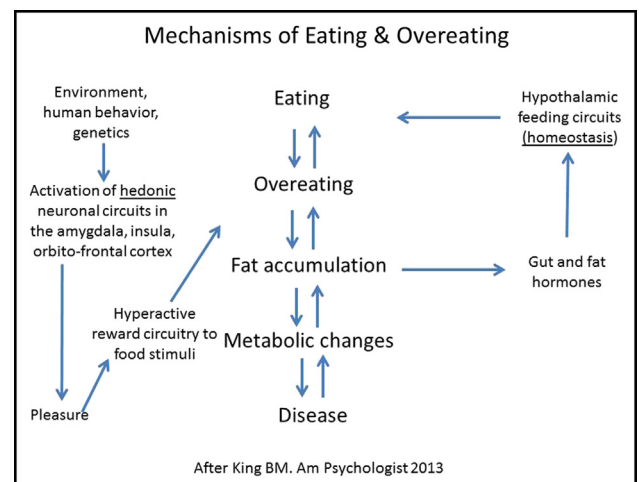


Figure 1 Mechanisms of eating and overeating.

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