Received: 13 November 2023 | Revised: 22 August 2024 | Accepted: 22 August 2024

DOI: 10.1111/obr.13828

REVIEW

Clinical Management

OBESITY Reviews WILEY

The vices and virtues of medical models of obesity

Jonathan Sholl[1] | Andreas De Block[2]

[1]Collège Sciences de la Santé, ImmunoConcept, Université de Bordeaux, CNRS UMR, Bordeaux, France

[2]Institute of Philosophy, Leuven, KU, Belgium

Correspondence
Jonathan Sholl, Université de Bordeaux, Collège Sciences de la Santé, ImmunoConcept, CNRS UMR 5164, Bordeaux, France.
Email: jonathan.sholl@u-bordeaux.fr

Summary

Despite numerous public health organizations supporting the pathologization of obesity and considering recent obesity rates a health crisis, many researchers in the humanities, social sciences, and even in the health sciences remain unconvinced. In this paper, we address a set of arguments coming from these academic fields that criticize medical models of obesity for their supposedly flawed diagnostic categories that shift focus onto individuals and support moralizing judgements. Clarifying some key claims in these models and explicating the view of obesity in terms of energy dysregulation, we aim to tease apart misunderstandings and argue that not only do these models not say what they are often accused of saying, but their apparent vices may actually be virtues in helping to combat stigma. Building on the social psychology of stigma and disease labeling, we then suggest that current medical models are largely supportive of many moral and political aims promoted by critics of these models.

KEYWORDS
disease categories, disease labeling, obesity models, stigma

1 | INTRODUCTION

Numerous public health organizations consider the unprecedented rise in obesity rates a health crisis.[1,2] In tandem, major health organizations such as the American Medical Association (AMA), the WHO, the European Commission, The Obesity Society, and the World Obesity Federation all largely converge on viewing obesity as a disease, but their definitions remain heterogeneous, some relying on body mass index (BMI), largely for epidemiological aims, and others focusing on the pathophysiology of abnormal or excessive adiposity.[3,4] As we discuss, this latter characterization has led to several *medical models of obesity* (hereafter MMOs) that go beyond BMI to include physiology, neuroscience, and evolutionary biology, as well as ecology, sociology, and economics, which all contribute to describing the pathophysiology and etiology of the systemic energy dysregulations now characterizing obesity. In what follows, we take explanations of obesity and its causes to be (among) the core aims of such models. These MMOs are thus the set of working hypotheses that are central to much of the current medical research on obesity. For that reason, our sketch of MMOs relies largely on recent reviews in high-impact obesity journals.

Yet, not all researchers are convinced that obesity constitutes a major public health problem or that it is clearly pathological. Medicalization critics have long argued that many medical categories (e.g., addiction, ADHD) are rationalizations of moral judgments: translating badness into sickness.[5,6] According to many of them, this is especially evident with obesity, where claims of importing moral judgments about otherwise "naturally occurring bodily diversity" abound.[7–10] Their concerns are reflected in academic and activist circles such as Fat Studies, Health at Every Size, National Association

to Advance Fat Acceptance, and Fat Underground, and have influenced the general public. These movements and fields argue that the pathologization of obesity is largely ideology masking shoddy science.

Within this debate, distinct issues have been explored by critical theorists, scientists, and philosophers, ranging from asking whether obesity is a disease[11,12] and evaluating the role of genetics and GWAS studies,[13] to questioning the reliance on BMI.[14] These debates center around both the empirical claims made by MMOs and their supposed links to perpetuating antifat bias and stigma.[15,16] This raises the challenge of determining how and *whether* MMOs have such harmful implications.

We take up this challenge as follows. First, we sketch some of the main arguments in the humanities and social sciences against MMOs and the moralizing assumptions often associated with them. Second, we engage with scientific literature about MMOs to provide a broad overview of how distinct models converge on explaining obesity and its causes. We use this to address misunderstandings and argue that not only do these models not say what critics often claim they are saying, but their apparent vices may actually be virtues in helping to combat stigma and increase empathy. Finally, we disentangle issues surrounding the moralization and stigmatization of disease labels by appealing to social psychology. We argue that some aspects of these models, for example, the clarification of etiology, can contribute to *counter*-moralizing efforts. Given the trade-offs with combatting weight-related stigma, such findings should be cautiously celebrated. Overall, we argue that the strong empirical and philosophical reasons to defend MMOs have complex, yet largely *supportive*, implications for the social–political aims of critical theorists of obesity, such as improving the body image, well-being, and treatment of individuals living with obesity.

2 | THE APPARENT VICES OF MEDICAL MODELS OF OBESITY

There is a shared interest among philosophers, social scientists, and critical theorists to analyze conceptual issues facing obesity diagnoses, and to evaluate whether the aims of obesity research align with those of improving public health.[11,12,14,15,17–19] The aims of these analyses are quite heterogeneous. Here, we focus on challenges specifically targeting MMOs and their implications.

There are several broad, and often intertwined, arguments used to challenge MMOs:

1. Some evidence seems to challenge viewing obesity as a disease, for example, that mortality appears *lowest* at moderate levels of overweight (the so-called obesity paradox), or the possibility of being metabolically healthy while having obesity. According to the critics, these findings (and others) show that *the diagnostic category is fundamentally flawed*.[9,16,19] A strong version claims that "[f]at is simply not the culprit in the etiology of chronic disease" (p. 298),[20] whereas a moderate version stresses that BMI cutoffs are "arbitrary" and not based on solid scientific evidence.[21]
2. By defining obesity as an individual, *bodily* problem, increasingly targeted by pharmaceutical agents, interventions tend to focus on biology, for exsmple, losing weight via drugs or surgery, thereby *obscuring or not addressing the (more) harmful social or political environment*.[22] This critique reflects a widespread argument in the medicalization literature that individualizing "can obscure the social forces that influence well-being" (p. 152).[6,23]
3. MMOs *carry implicit and explicit moral judgments*, for example, their language implies deviations from a socially acceptable "ideal" weight,[9,24] and, more importantly, they *carry attributions of individual responsibility*, for example, medical models translate the moralizing view that obesity is due to gluttony and sloth into over-eating and not exercising enough.[16] This critique is closely linked with the second one: individualizing health problems foregrounds personal responsibility because health is something to be self-regulated.[23] Similarly, failed interventions, for example, surgery or diet, may represent failures of self-discipline.[25] As one scholar puts it, "the medicalization of obesity has not served to limit the stigmatization and discriminatory treatment to which obese persons are subject because of their body size. Instead, by making obese persons individually responsible for their fatness, biomedical discourse fuels and even legitimizes lipophobia" (p. 1181).[26]
4. The *negative health effects of obesity are less than the negative (health) effects of the measures taken against it*—typically through label-induced stigma.[18,27] In this vein, Mehl[16] argues that it is an "unlikely assumption" that pathologizing obesity will improve patient outcomes given the high prevalence of antifat bias in healthcare. The claim is that stigma and other harms are (1) *caused by* the diagnosis and (2) are *worse for health* than the "supposed" disease.

Argument 1 largely raises empirical or evidential questions about how obesity is described and explained within MMOs. While argument 3 is partly about the importation of value judgments, together with argument 2, these arguments also question the etiology of obesity advanced by MMOs. Argument 4 targets the psychosocial effects of MMOs, which is largely an empirical issue and requires a careful analysis of the trade-offs when using disease labels.

While these critiques and their targets are quite diverse, there seems to be a shared feeling that MMOs imply that there is something "wrong" with having obesity that needs fixing and that this diagnosis assumes or entails a moral failure in self-control:

> Medicalizing diversity inspires a misplaced search for a "cure" for naturally occurring difference. Far from generating sympathy for fat people, medicalization of weight fuels anti-fat prejudice and discrimination in all areas of society. People think: If fat people need to be cured, there must be something wrong with them. (pxiii)[28]

Many critics retort that there is, in fact, nothing wrong with having obesity, that the problem and responsibility are largely social, and that pathologizing it only worsens bias and stigma.

Any analysis of what a medical model explicitly claims, or implicitly assumes, should aim to be as accurate and charitable as possible. Yet, many of the above concerns reflect outdated views on obesity, which are at odds with the teams of scientists around the world trying to better understand obesity because they believe that doing so will help improve the lives of those living with it.[29] So how do MMOs conceive of obesity?

3 | ENGAGING WITH MEDICAL MODELS OF OBESITY

Building on philosophical attempts to clarify scientific descriptions of obesity,[12–14] we take a broader perspective. In what follows, we explicate an apparent consensus in current MMOs that obesity is a disease characterized by systemic energy dysregulation that drives abnormal or excessive adiposity. We take the main aims of MMOs to be (1) precisely describing the dysregulations involved (pathophysiology) and (2) what causes them. To that end, we first set out an evolutionary argument as to why having excessive adiposity is harmful; we then discuss how current medical research nuances BMI-based definitions by clarifying the roles of appetite and adiposity dysregulations in driving excess adiposity, and we survey evidence for the etiology of these dysregulations, largely identifying environmental mismatches. Overall, we aim to provide a solid basis from which to address some misunderstandings and moral implications of MMOs in subsequent sections.

3.1 | Explaining obesity pathophysiology

First, what evolutionary pressures may have led to the existence and regulation of fat tissue and when could its excess be (evolutionarily) harmful? Evolutionary models suggest that having dedicated tissues where fat can be stored as energy, and mechanisms to monitor and regulate fat levels, likely evolved to survive periods of illness, to protect against food insecurity, and to support energy intensive events, such as reproduction or migration.[30,31] While the adaptive ability to deposit fat likely helped avoid the costs of having *too little* fat (i.e., starvation), organisms likely also faced selection pressures from depositing *excess* fat.[30,32,33] An upper limit to fat storage may thus have evolved through fitness costs, for example, higher weight worsens mobility and increases energy demands, which requires more time foraging and thus increases the risk of predation.[34,35] As Matthewson and Griffiths[36] point out, phenotypic changes that leave an organism more likely to die from predation leave it worse off in any relevant biological sense. Biologically "optimal" levels of fat storage thus reflect a trade-off between having enough to avoid starvation, yet not so much as to increase predation risks. In line with this, animals experiencing higher predation tend to *reduce* fat stores.[33,37]

One often misunderstood implication is that while storage of *some* fat may reflect the adaptive benefit of surviving for roughly 3–6 weeks without food or a similar period of sickness, chronically abnormal or excessive adiposity reduces fitness *in natural environments*. Moreover, the extremely high levels of body fat observed in recent years do not correspond to any known disease process or period of food shortage for which such adiposity would be adaptive.[33]

Next, physiological models face the challenges of explaining energy regulation in a way that aligns with an evolutionary explanation of fat storage, while also explaining why so many people today are storing so much (i.e., the obesity epidemic). The central models focus on mechanisms involved in regulating energy intake and expenditure, in feedback with appetite control systems, as central points where dysregulations occur. Given the growing emphasis on the neurology of appetite and energy regulation,[38] with key factors being AGRP neurons involved in food seeking, the lateral hypothalamus involved in food consumption and reward, and CGRP neurons involved in satiety, various researchers have suggested that obesity is at least partly a neuroendocrine disease.[14,39,40]

To explain obesity's pathophysiology, some models thereby target disruptions in *energy balance or homeostasis* based on interactions between increasing fat levels, alterations in some of the above brain regions, and hormones regulating intake and satiety (e.g., leptin, ghrelin, insulin), which form feedback loops maladaptively defending higher body weight "set points,"[41–43] (for variations on these models, see[44]). By identifying pathways where adiposity and appetite systems are dysregulated, these models partly clarified how humans store (excess) fat, the physiological compensations to fat loss,[45] and the possible links between energy expenditure, the drive to eat, and satiety.[46] Yet, it remains unclear why such tight regulation of set points would have evolved and, more importantly, why environmental factors have been persistently disrupting these set points in so many people.[31,33]

Challenging the notion of a single set point or a fixed set range, others propose dynamic models.[31,44] One is the *dual intervention point model* in which there are genetically based lower and upper thresholds of body weight where active physiological regulation occurs to defend against excessive leanness and excessive weight gain, and between which there is no regulation, with weight varying due to environmental factors.[30,33] This model not only explains nuances in fat regulation but aligns with an evolutionary explanation and may help explain the obesity epidemic. While the lower limit likely reflects the evolutionary pressure of starvation, as we evolved the use of tools, fire, and weapons for hunting, and developed social defenses and protections, we largely removed the selection pressure of predation.[32,44] This may have contributed to relaxing any selection against the upper limit of fat storage, entailing that mutations could persist and drift in recent populations, leaving some with genetic resistance (narrower thresholds) and others genetically predisposed (wider thresholds) to weight gain and obesity,[47] thus explaining some interindividual variability. In this model, obesity is a disease largely afflicting those with genetic susceptibilities to excess weight gain, due to inheriting a higher upper

limit (including other genetic factors discussed below), and thus struggling to regulate appetite and fat deposition in modern environments, for example, where energy-dense food is abundant and designed to disrupt regulatory mechanisms. As nothing in this model requires that the limits correspond exactly to typical BMI cutoffs, it appears consistent with a "personal fat threshold," which can be at various levels of BMI, and beyond which excess energy is deposited as fat, largely visceral but also subcutaneous, which can damage pancreatic beta cells and induce metabolic dysfunctions.[48,49] In these models, weight gain reflects chronic positive energy imbalance, where overeating is a necessary but not sufficient cause, just as is lower energy expenditure.[45] As we discuss, these models align with etiological aspects targeting mismatches between apparently adaptive/genetic mechanisms for fat deposition or food intake and modern environments.

Critical approaches to MMOs rarely discuss the intricacies of these models. Still, one can imagine objections to these scientific explanations. For instance, one could ignore the evolutionary argument by pointing out that current individuals with obesity face little risk of predation and are thus safer than ever. Furthermore, what is bad for an individual's realized fitness (say celibacy) need not be bad for the individual. This would even be convincing, if it were not that MMOs also explain why obesity was not just bad for our ancestors' fitness, but also negatively affects individuals today. After all, central to these models is the notion that the health impairments are progressive and likely chronic,[3,50,51] which rests on research specifying metabolic and endocrinological alterations, various biomechanical and cardiometabolic complications,[52,53] mechanistic links with other diseases such as diabetes, cardiovascular disease, cancer, and proinflammatory cascades,[54–57] and obesity ultimately being an independent risk factor for all-cause mortality.[58–60]

From this "constellation" of anatomy and physiology[52] comes an apparent consensus of obesity characterized by *systemic dysregulations of energy homeostasis and appetite*, resulting in abnormal or excessive fat deposition, which form feedback loops driving subsequent complications, comorbidities, and mortality risks. One reason BMI remains useful *for establishing population level trends*, even if only providing a partial, and sometimes misleading, clinical picture,[61,62] is that it serves as an anthropometric proxy or "indicator"[63] of these dysregulations and health risks (see Figure 1 and further nuances below). Accordingly, MMOs are providing more precise explanations of epidemiological associations based on coarse-grained BMI measures.

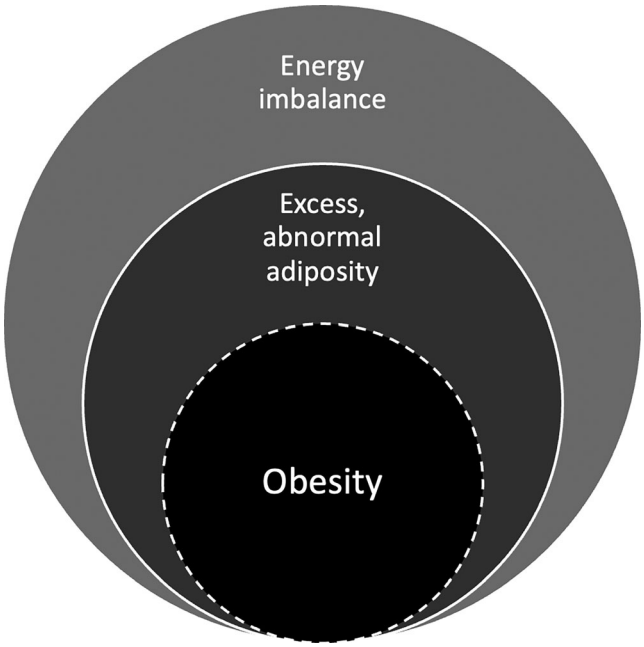

FIGURE 1 Overlapping circles delineating obesity. Not everyone in a positive energy imbalance will develop obesity, but some will. Not everyone with excess or abnormal adiposity has obesity, but some do. The first two groups are heterogeneous, reflecting larger populations. Obesity, a subset of these roughly correlating with BMI and related anthropometric measures, is a disease characterized by systemic energy dysregulation that drives excessive and abnormal adiposity. The exact cutoff or diagnostic line is still under debate, as with many medical conditions (hence the dotted lines), but the aim is to identify and group clusters of dysregulations to guide research and treatment.

3.2 | Explaining obesity pathogenesis

Now, what causes these dysregulations? Obesity is generally considered a multifactorial disease, but genetics play an important role in explaining body weight variability,[64] with the genetic heritability for BMI being between 40 and 70%.[65–67] GWAS studies have identified roughly 900 variants contributing to body weight variations.[44] This helps explain the strong correlations between parental and childhood obesity,[56,68,69] with high maternal BMI,[70] gestational diabetes,[71] and potentially high paternal BMI,[72] all predicting childhood obesity.

This in no way implies that obesity is "genetically determined" but that genetics help explain individual *susceptibility* (interindividual variability)—why not everyone develops obesity in the same environment. Hence, obesity etiology is often described as a "gene-by-environment" interaction*.[33,56] That said, there are important distinctions between rare monogenic forms of obesity and so-called common (polygenic) obesity. In the former, specific genetic mutations are gradually being identified,[66,74] some of which pertain to "syndromic" forms of obesity, whereas the latter mutations involve quantitative variations in susceptibility. Most of the latter influence energy regulation, many relating to neurology,[75] for example, responsiveness to food cues, degree of food reward, appetite/satiety signaling, ability to metabolize and absorb nutrients, degree of fat deposition following caloric surplus, basal metabolic rates, and nonexercise activity thermogenesis, each of which influences susceptibility to environmental changes.

*For nuances in the scientific and philosophical debates about whether or in what sense heritable traits that are mediated by the environment (gene–environment covariances) are "genetic," see Lynch and Bourrat.[73]

Accordingly, environmental factors explain a significant portion of common, polygenic obesity,[76] with a growing role played by the notion that many changes in modern environments are exceeding our capacities to regulate metabolism and appetite. Some highlight changes early in life, such as the effects of poor parental nutrition, stress, and lifestyle on the fetus; macronutrient content of infant formula; and early life adversity.[56] Most, however, highlight the recent and drastic changes in our food environment that drive prolonged energy imbalances—an evolutionary mismatch due to an "obesogenic" environment.[69,77,78] While there is little debate as to whether this mismatch exists, much debate focuses on specific nutritional causes,[43,79,80] with many agreeing on an increased consumption of cheap, readily available energy-dense foods, such as ultraprocessed foods, which are designed by food manufacturers to bypass satiety mechanisms.[81–83] There is also considerable evidence that protein-diluted dietary patterns, for example, increases in consumption of fats and carbohydrates with a relative *decrease* in percent protein (often from ultraprocessed foods), work against our evolved nutrient-specific appetite systems that regulate food intake.[80,84–86] These changing eating patterns involve complex links to globalized food systems[87] and marketing practices.[88]

Other established etiological factors include sleep disturbances, ingesting more chemicals and endocrine disruptors ("obesogens"), less variability in ambient temperatures, medications with the side effect of weight gain, for example, some antibiotics and psychotropics, and specific alterations in our gut microbiota.[76,89] Moreover, there are robust links with socioeconomic inequalities, with higher obesity prevalence in upper-income populations of middle-income countries, and higher rates in lower social strata in high-income countries,[90–92] education levels,[93] and stress.[94] All these factors can exacerbate or be exacerbated by predispositions and mismatches with food environments, further complicating cause and effect.

Overall, while open questions remain (next section), when it comes to explaining what obesity is and what causes it, these models and the multiple lines of evidence adduced point more towards consilience than confusion; they suggest a working hypothesis that the distinct mechanisms and dysregulations cluster, despite their multifactorial etiology, around systemic energy dysregulation. These MMOs are also consistent with the notion that having obesity is "normal" in modern environments, but this does not make it any less maladaptive or harmful. Still, there are issues with interpreting some statistical anomalies and with refining measures of obesity for various purposes.

4 | NUANCED IMPLICATIONS AND SCIENTIFIC VIRTUES OF MMOS

First, it has long been known that some subpopulations living with obesity appear to have no concurrent negative health effects,[67] often deemed "metabolically healthy obesity".[95] In addition to their being no equivalent discussions about "healthy" Parkinson's disease or healthy cancer, such debates are relics of relying on BMI-based studies and, similar to the subpopulations of smokers who do not develop lung cancer, are nevertheless insufficient to undermine the robust associations between obesity and adverse health outcomes.[58,96] If we shift from health snapshots to longitudinal studies, we find more evidence for the adverse effects of living with obesity, even the "metabolically healthy" forms.[97–99] Relatedly, the "fit-but-fat" phenotype is often used to suggest that obesity is or can be harmless, whereas the nuanced interpretation in line with MMOs is that individuals can *mitigate* the harms of obesity by increasing their lean muscle mass and cardiorespiratory fitness.[100]

Second, there are many nuances surrounding the role of BMI in MMOs. Critics are correct that BMI remains sometimes too central to research about and some definitions of obesity and their operationalization, but the role it plays needs careful consideration. For instance, while the AMA cautions against an over-reliance on BMI, mainly in clinical settings, it does *not* recommend to jettison it, but to contextualize it as one tool among others.[101] As mentioned above, researchers have long acknowledged the use of BMI as a *population screening tool* that is important for establishing cross-country and temporal comparisons, especially in low-income settings,[102,103] despite its limitations for clinical diagnoses.[17,56,61,62,67] BMI thus remains a useful "proxy" that correlates with the disease and helps to cluster various dysregulations and symptoms. Moreover, some findings suggest that BMI classifications might even *under*estimate the effects of adiposity on health.[104]

Now, according to one critic, the "medical model of obesity" is built on three assumptions[16] (p. 3): "(1) Other things being equal and above a certain threshold, the more one weighs, the unhealthier one is (or vice versa); (2) an "overweight" or "obese" body is to some degree inherently unhealthy; and (3) people who are "overweight" or "obese" are so because they do not eat properly and/or do not exercise enough." We can now see why each of these is misleading.

First, most MMOs do postulate a threshold beyond which adipose tissue becomes harmful, for example, through reduced fitness or physiological dysregulation, or by crossing a "personal fat threshold".[49] The nuance is that, as with any continuous variable, the threshold for excessive fat deposition will vary, but there remains a persistent trend of increasing weight and worse health outcomes when looking at larger studies and the broader evidence base.[58,67,96] Such claims, however, neither require nor imply the assumption that less weight is *always* healthier (e.g., Mestre et al.[67] explicitly address this), nor that weight loss is always the optimal intervention,[56,105] especially when eating disorders are involved.[106,107]

Next, as MMOs focus on *obesity* as a disease process, they do not take *overweight* to be pathological any more than other models of continuous variables like blood pressure or cholesterol take elevations above "normal" to be so; in general, increasing weight is a potential risk factor to be monitored.[3] As Rubino et al.[61] claim, while overmedicalization is a real concern, the implication should not be to abandon MMOs, but to develop clinically relevant definitions in line with best evidence (e.g.,[53,108]). Moreover, for many patients or families living with obesity and facing lack of healthcare, social disadvantage, and stigma, *under*-medicalization, or unmet clinical need, is a serious

problem.[61,109] While there is much to be understood about the boundaries between overweight and obesity, these findings do not fundamentally challenge the core claims of MMOs.

Finally, while various etiological factors interact with physiology to drive chronic energy imbalances, *none* of these imply individual failures in willpower or self-control, and many obesity researchers are very explicit about this.[61,110–112] In other words, contra some critics,[16] nothing in the current MMOs supports the moralizing claim that obesity is caused by "sloth and gluttony," nor do these MMOs target an individual's failure to eat well or exercise as etiological factors.

Consider the etiology of common, childhood obesity. Some children may carry genetic and epigenetic predispositions to excess fat deposition, with body weight influenced by their prenatal environment; the children might inherit higher upper limits for storing fat; perhaps they metabolize or absorb nutrients less effectively, leaving them regularly hungry, and they might be more prone to weight gain due to taking medications for mental health or other conditions. Responsibility and blame make little sense, unless we are to blame children for their genes, upbringing, and physiological capabilities. As blame may shift onto parents, we can also consider environmental causes—while individuals face difficult choices when navigating their environment, they have little to no control over what foods are available, how processed foods are designed, the ingredients they contain, the fact that satiating nutrients like fiber and protein tend to be more expensive, or, importantly, how their evolved appetite systems leave them susceptible to overeat in energy-dense, protein-diluted food environments. Not to mention that some children may come from broken homes, experience trauma, economic adversity, and/or stigma, each of which can exacerbate the above factors. The phenomenon and etiology of obesity is no different in adults; it just involves increasingly complex exacerbating factors. When the relevant etiological drivers are considered, there is scant room left for blaming individuals, let alone populations, for their supposedly collective failures in willpower as a cause of modern rates of obesity. The underappreciated take-away should be that of *empathy* as we better understand the myriad challenges everyone faces in preventing obesity, let alone living with it.

It should be noted, however, that MMOs can challenge fatalism by proposing interventions on multiple levels. If modern obesity is largely due to mismatches with obesogenic environments and gene–environment interactions, then the most effective public health interventions are likely those targeting environmental causes[113] (p. 272). Accordingly, researchers increasingly cite socioecological or socioeconomic interventions,[92,114] such as improving school lunches, developing community-based activity programs, supporting governmental bans or taxes on specific foods, increasing high school completion rates, as well as addressing internalized weight biases and the stigmas fueling them.[56,111] As such, there is no *inherent* link between pathologizing and interventions at the level of the individual.

That said, individual interventions exist.[115,116] While in some cases weight loss efforts can be counter-productive, for example, if eating disorders are involved, in most cases there are significant health benefits from even moderate weight loss,[117] many pertaining to obesity-associated metabolic diseases.[118–120] This is complicated by the observation that maintaining long-term weight loss and adhering to dietary changes is difficult, largely due to physiological compensations to losing weight, such as increased appetite or energy expenditure,[43,45,121] as well as persisting obesogenic food systems. There is increasing promise in obesity drugs (e.g., GIP and GLP-1 agonists) that may rival the effectiveness of bariatric surgery, without the invasiveness.[122,123] Finally, while physical activity may play only a moderate role in weight loss,[124,125] the robust evidence that it can improve metabolic and cardiorespiratory health, increase lean muscle tissue, help regulate appetite, satiety, mood, and sleep, and even reduce all-cause mortality,[126–130] all make physical activity an indispensable adjunct regardless of its effects on weight. Consequently, we can and should promote health at every body size, but this does not mean that every body size is optimally healthy or that weight loss has no benefits.

In sum, not only do many obesity researchers explicitly *reject* the stigmatizing assumptions about what causes obesity and its links to individual responsibility,[110] but there is no obvious way in which current MMOs support these assumptions. Importantly, MMOs readily acknowledge the considerable weight stigma and prejudice that exists in many societies,[61] as well as the admittedly unhealthy obsessions with thinness and extreme diet cultures.[9,131]

Now, some might still object that pathologizing obesity, even if based on seemingly coherent scientific models, remains problematic because it exacerbates antifat stigma and discrimination by other means. However, studies in the social psychology of disease labelling further nuance the links between pathologizing and moralizing.

5 | WHY PATHOLOGIZING IS SOMETIMES COUNTER-MORALIZING—THE MORAL VIRTUES OF MMOS

Few people would deny that talk of "obesity" emphasizes the health risks of fatness. But does it also support the idea that a person is responsible for their weight? Building on the insights above from MMOs, social psychology sheds further light on this question.

In a series of studies, Crystal Hoyt and colleagues[132] presented subjects with different messages about obesity. In one study, they asked one group of participants to read a vignette claiming that obesity is a disease, and another group to read a vignette explicitly arguing that obesity is not a disease. In general, they found that the people with obesity who read the "obesity is a disease" vignette were afterwards less dissatisfied with their bodies and subsequently chose higher calorie meals than people with obesity given the "obesity is not a disease" article. Hoyt and colleagues argue that the effect of the disease and nondisease messages on calorie consumption is mediated by body satisfaction. The message that obesity is a disease makes people with obesity more satisfied with their bodies, which keeps them from making the lower calorie choices.

First, this research suggests that it is wrong to think that (a particular form of) pathologizing obesity will always result in healthier choices, which is why clinical treatments of obesity recommend a close provider–patient relationship. Secondly, the authors of the paper explicitly state that the disease message tends to have a positive influence on body image satisfaction, which seems to run counter to claims that medicalizing fatness leads to unhappiness among people with obesity (see also Monterosso et al.[133]). Interestingly, the reason Hoyt and colleagues give for the positive effect of the disease label on the well-being of people with obesity is that representing obesity as a disease reduces attributions of moral responsibility. In other words, it has the opposite effect of representing obesity as a product of weak will. Other studies corroborate this hypothesis. In one (small-scale) study, individuals with obesity who watched a narrative video in which obesity was described as a chronic but treatable medical condition reported less internalized weight bias and more positive views on patient–provider relations.[134] In another study, labeling obesity as a disease reduced blameworthiness and this led to more positive attitudes towards individuals with obesity.[135,136] These studies suggest that pathologizing need not be moralizing in the sense of implying culpability or personal responsibility for one's condition or one's treatment.

Although research on the exculpatory effects of the pathologization of obesity is still largely exploratory, its conclusions are in line with what research on other conditions shows: pathologizing often has a counter-moralizing effect.[137] Yet, what exactly is someone not responsible for and how can the disease label reduce the attribution of responsibility? Hoyt et al.[132] (p. 997) argue that the disease label "suggests that bodies, physiology, and genes are malfunctioning. By invoking physiological explanations for obesity, the disease label encourages the perception that weight is unchangeable." Indeed, other studies confirm that strong endorsement of a genetic etiology of fatness is predictive of a belief that people with obesity have no control over their weight.[138,139] This works, in part, by activating psychological biases concerning how people understand genetic explanations. These researchers also noted that a nongenetic physiological attribution led to a somewhat increased perception of control over one's weight, compared with a genetic etiology of obesity (see also Lebowitz et al.[140]). So, it seems clear that the disease label alone does not directly remove blame. *What removes blame is the etiology suggested by the disease label*. This matters for moralization because it shapes whether individuals are considered responsible for their actions: the more uncontrollable the cause, the less one is held responsible.[141]

Consequently, obesity may be perceived as beyond a person's control (1) if it is viewed as genetically determined, (2) when the specified etiology implicates uncontrollable external factors (e.g., food availability, costs), or (3) internal factors (e.g., the person cannot control their physiology or behavior). The first possibility entails that someone would become or stay obese regardless of their behavior because their obesity is viewed as determined by their genes.[138] Likewise, emphasizing external etiology may picture individuals as victims of (gene-) environment interactions, with little or no control over their life in general or their food choices in particular (e.g.,[142,143]). This may partly remove blame, but it may also undermine a sense of agency.

The third possibility is when an internal cause is seen as compulsive, or as hindering one's choices, similar to addictions.[144] While we are not necessarily supporting the notion of "food addiction," there is evidence that obesity involves neurological, hormonal, and behavioral changes that resemble other addictions.[137,145–147] These changes can exacerbate food-seeking behaviors by increasing hunger and fatigue,[43] which parallels the pathologization of alcoholism. Moreover, research shows that viewing obesity as involving a compulsion can reduce stigma. When individuals were educated about obesity and food addiction, there was less weight bias, individuals with obesity were seen as less at fault, and there was a reduction in the belief that obesity involves lack of willpower.[148] This counterfactual perspective suggests that seeing obesity as involving compulsions reduces stigma because people are not seen as responsible for their condition (but see Frank and Nagel[149]). Studies indicate that if weight is seen as controllable through diet and behavior, stigma increases.[150,151] Finally, considering how the body resists weight loss by, for example, overriding satiety signals, increasing appetite and food reward, such physiological counter-regulatory mechanisms show that obesity is not a lifestyle choice, but involves significant biological factors, akin to the compulsions found in other conditions.

If anything, these studies suggest that *depathologizing obesity may inadvertently support the view that it is a lifestyle choice reducible to gluttony and sloth*, and thus (more) under one's control, which has been shown to increase antifat prejudice.

Yet, the psychosocial impacts of disease labels remain complicated. First, negative prejudices and discrimination or bias, both crucial to the concept of stigma, seem only to partially respond to factual input. This may be in part because feelings of disgust drive negative perceptions independent of causal attributions.[152] A meta-analysis revealed that educational approaches to reducing stigma generally lead to improved attitudes, but these improvements are not always consistent and may sometimes be short-lived[153] (see also Teachman et al.[154]). Therefore, a comprehensive strategy that goes beyond factual education is required to reduce weight stigma.[155] Furthermore, labeling obesity as a disease may sometimes increase stigma by activating "essentialist" biases, which are more likely for conditions that are seen as genetically caused than for conditions that are associated with environmental causes.[156] If it is believed that your obesity is due to genetics, you are more likely to be seen as essentially different than when people believe that it has social/environmental causes. Such nonblame stigmatization can involve marginalization, therapeutic pessimism, and perceived dangerousness.[157] The study by Hoyt et al.[132] underscores that pathologizing obesity may have a similar effect, as that study documented how the disease label led to strengthened beliefs that weight is unchangeable, and that people with obesity are thus hopeless. Hence, although pathologizing obesity may take away blame (and part of the stigma), it may still lead to an increase in other forms of stigma, including pity. After all, pity involves a power differential between the person feeling pity and the person who is pitied, which can undermine the latter's self-esteem.

Despite these complications, the general implication from social psychology literature is not to reject MMOs, as their critics suggest. This literature instead highlights the need for a considerate weighing of the trade-offs involved. While emphasizing the gene-by-environment aspects of obesity might *decrease* stigma by supporting perceptions of determinism or the role of uncontrollable etiological factors, the same emphasis may *worsen* stigma by viewing individuals with obesity as a homogeneous (essentially different) group. Consequently, it is important to inform all stakeholders, and especially people with obesity, that MMOs do not conceive common obesity as a purely genetic disease. As we saw, MMOs tend to emphasize the environmental etiology of most forms of obesity. Doing so may help mitigate these essentializing effects of obesity's pathologization.[156] The emphasis on environmental causes may even increase the sense of agency of people with obesity, as environmental attributions lead to a somewhat increased perception of control over one's condition, at least compared with a genetic etiology.[142] However, whether emphasizing such environmental causes will change public perceptions of obesity remains unclear.[158] Nevertheless, informing people with obesity about how social and environmental factors constitute an obesogenic niche may help to identify what they can change.[87] This can involve awareness of the harmful aspects of one's food environment or of reliable strategies to improve health at any body size, or it could encourage collective action, for example, challenging the harmful practices of food companies or supporting community health efforts. These approaches thus come close to Hannah Pickard's "responsibility without blame" framework for effectively and caringly dealing with addictions by helping to identify where (individual) control exists.[159]

Admittedly, even if these mitigating measures are taken, it can be expected that some stigma and essentializing biases will remain. But given the health threat that constitutes obesity, some increases in stigma may be a necessary trade-off when trying to advance longer-term public understanding of and viable options for those with obesity.[61] Admittedly, as experienced stigma can induce less healthy eating behaviors,[160] the optimal way to deal with this trade-off may be hard to find.

6 | CONCLUSION

Body composition mirrors the intricate interplay of habits, pleasures, and cultural ideals. Across cultures, body size embodies personal and societal notions of beauty, goodness, and health. Individuals and groups may value larger bodies for diverse reasons, independent of any scientific explanation. Nothing in the above contradicts such choices and values. However, that people continue to drink alcohol, identify with being a drinker, and take great pleasure in alcohol should not give scientists pause as to whether to research the links between excessive alcohol consumption and fatty liver disease. We think obesity is not so different.

Of course, if MMOs would explicitly or unnecessarily stigmatize individuals with obesity, this would be reason for great concern. Yet, as we have argued, not only do MMOs *not* say what many critics claim they say, for example, ignoring social and environmental causes or blaming individuals, they provide a sound basis upon which to challenge prevailing prejudices about obesity, thereby aligning with many of the moral and political aims of fat activists. If this convergence is to be better understood and its implications developed, this will require an honest evaluation of the scientific evidence and models of obesity, as we attempted here. Going forward, we need multiple efforts clarifying what our best models say, and what they do not, and to further promote compassion and informed decision-making.

ACKNOWLEDGMENTS

JS would like to warmly thank everyone in the 2023 *Theory & Method in Biosciences* team (University of Sydney), as well as Louise Baur and Timothy Gill, for their kind support, thoughtful discussions, and detailed feedback on earlier drafts. Both authors thank the three anonymous reviewers for their useful suggestions.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

ORCID

Jonathan Sholl https://orcid.org/0000-0003-3786-2061
Andreas De Block https://orcid.org/0000-0002-7927-8210

REFERENCES

1. Kim S, Popkin BM. Commentary: Understanding the epidemiology of overweight and obesity—a real global public health concern. *Int J Epidemiol*. 2006;35(1):60-67. doi:10.1093/ije/dyi255
2. Blüher M. Obesity: Global epidemiology and pathogenesis. *Nat Rev Endocrinol*. 2019;15(5):288-298. doi:10.1038/s41574-019-0176-8
3. Bray GA, Kim KK, Wilding JPH, World Obesity Federation. Federation on behalf of the WO. Obesity: a chronic relapsing progressive disease process. A position statement of the world obesity federation. *Obes Rev*. 2017;18(7):715-723. doi:10.1111/obr.12551
4. Wharton S, Lau DCW, Vallis M, et al. Obesity in adults: A clinical practice guideline. *CMAJ*. 2020;192(31):E875-E891. doi:10.1503/cmaj.191707
5. Conrad P, Schneider JW. *Deviance and medicalization: From badness to sickness*. Temple University Press; 1992.
6. Conrad P. *The medicalization of society: On the transformation of human conditions into treatable disorders*. Johns Hopkins University Press; 2007. doi:10.56021/9780801885846
7. Campos PF. *The obesity myth: why America's obsession with weight is hazardous to your health*. Gotham Books; 2004.
8. Gard M, Wright J. *The obesity epidemic: science, morality and ideology*. 0th ed. Routledge; 2005. doi:10.4324/9780203619308
9. Brown H. *Body of truth: How science, history, and culture drive our obsession with weight–and what we Can do about it*. Da Capo Press; 2015.
10. Monaghan L, Colls R, Evans B (Eds). *Obesity discourse and fat politics: Research, critique and interventions*. Routledge; 2016. doi:10.4324/9781315795645
11. Hofmann B. Obesity as a socially defined disease: Philosophical considerations and implications for policy and care. *Health Care Anal*. 2016;24(1):86-100. doi:10.1007/s10728-015-0291-1
12. Kilov D, Kilov G. Philosophical determinants of obesity as a disease. *Obes Rev*. 2018;19(1):41-48. doi:10.1111/obr.12597

13. Serpico D, Borghini A. From obesity to energy metabolism: Ontological perspectives on the metrics of human bodies. *Topoi*. 2021;40(3):577-586. doi:10.1007/s11245-020-09722-1
14. Steele M, Finucane FM. Philosophically, is obesity really a disease? *Obes Rev*. 2023;24(8):e13590. doi:10.1111/obr.13590
15. Darren TMG, Powell J (Eds). *Routledge handbook of critical obesity studies*. Routledge; 2021. doi:10.4324/9780429344824
16. Mehl KR. The medical model of "obesity" and the values behind the guise of health. *Synthese*. 2023;201(6):215. doi:10.1007/s11229-023-04209-z
17. Kyle TK, Dhurandhar EJ, Allison DB. Regarding obesity as a disease: Evolving policies and their implications. *Endocrinol Metab Clin North am*. 2016;45(3):511-520. doi:10.1016/j.ecl.2016.04.004
18. Pausé C. Borderline: The ethics of fat stigma in public health. *J Law Med Ethics*. 2017;45(4):510-517. doi:10.1177/1073110517750585
19. Flegal KM. Use and misuse of BMI categories. *AMA J Ethics*. 2023;25(7):550-558. doi:10.1001/amajethics.2023.550
20. Stewart RS, Korol SA. De-signing fat: Re-constructing the global obesity epidemic. *Int J Appl Philos*. 2009;23(2):285-304. doi:10.5840/ijap200923221
21. Flegal KM. How body size became a disease. In: *Routledge handbook of critical obesity studies*. 1st ed. Routledge; 2021:23-39. doi:10.4324/9780429344824-5
22. Reiheld A. With all due caution: Global anti-obesity campaigns and the individualization of responsibility. *Int J Fem Approach Bioeth*. 2015;8(2):226-249. doi:10.3138/ijfab.8.2.226
23. Clarke AE, Mamo L, Fosket JR, Fishman JR, Shim JK. *Biomedicalization: Technoscience, health and illness in the U.S*. Duke University Press; 2010. doi:10.1515/9780822391258
24. Monaghan L. Extending the obesity debate, repudiating misrecognition: Politicising fatness and health (practice). *Soc Theory Health*. 2013;11(1):81-105. doi:10.1057/sth.2012.10
25. Throsby K. The war on obesity as a moral project: Weight loss drugs, obesity surgery and negotiating failure. *Sci Cult*. 2009;18(2):201-216. doi:10.1080/09505430902885581
26. Gracia-Arnaiz M. Thou shalt not get fat: Medical representations and self-images of obesity in a Mediterranean society. *Health*. 2013;5(7):1180-1189. doi:10.4236/health.2013.57159
27. Chrisler JC, Barney A. Sizeism is a health hazard. *Fat Stud*. 2017;6(1):38-53. doi:10.1080/21604851.2016.1213066
28. Wann M. Fat studies: An invitation to revolution. In: Rothblum ED, Solovay S, eds. *The fat studies reader*. New York University Press; 2009:ix-xxv.
29. Baur LA. Changing perceptions of obesity—recollections of a paediatrician. *Lancet*. 2011;378(9793):762-763. doi:10.1016/S0140-6736(11)61365-2
30. Speakman JR. The evolution of body fatness: Trading off disease and predation risk. *J Exp Biol*. 2018;221(Suppl_1):jeb167254. doi:10.1242/jeb.167254
31. Speakman JR, Hall KD. Models of body weight and fatness regulation. *Philos Trans R Soc B*. 1888;2023(378):20220231. doi:10.1098/rstb.2022.0231
32. Speakman JR. A nonadaptive scenario explaining the genetic predisposition to obesity: The "predation release" hypothesis. *Cell Metab*. 2007;6(1):5-12. doi:10.1016/j.cmet.2007.06.004
33. Speakman JR, Elmquist JK. Obesity: An evolutionary context. *Life Metabolism*. 2022;1(1):10-24. doi:10.1093/lifemeta/loac002
34. Heldstab SA, van Schaik CP, Isler K. Being fat and smart: A comparative analysis of the fat-brain trade-off in mammals. *J Hum Evol*. 2016;100:25-34. doi:10.1016/j.jhevol.2016.09.001
35. Pontzer H, Yamada Y, Sagayama H, et al. Daily energy expenditure through the human life course. *Science*. 2021;373(6556):808-812. doi:10.1126/science.abe5017
36. Matthewson J, Griffiths PE. Biological criteria of disease: Four ways of going wrong. *J Med Philos: Forum Bioeth Philoso Med*. 2017;42(4):447-466. doi:10.1093/jmp/jhx004
37. Gosler AG, Greenwood JJD, Perrins C. Predation risk and the cost of being fat. *Nature*. 1995;377(6550):621-623. doi:10.1038/377621a0
38. Sternson SM, Eiselt AK. Three pillars for the neural control of appetite. *Annu Rev Physiol*. 2017;79(1):401-423. doi:10.1146/annurev-physiol-021115-104948
39. O'Rahilly S, Farooqi IS. Human obesity: A heritable neurobehavioral disorder that is highly sensitive to environmental conditions. *Diabetes*. 2008;57(11):2905-2910. doi:10.2337/db08-0210
40. Purnell JQ, le Roux CW. Hypothalamic control of body fat mass by food intake: The key to understanding why obesity should be treated as a disease. *Diabetes Obes Metab*. 2024;26(S2):3-12. doi:10.1111/dom.15478
41. Keesey RE, Hirvonen MD. Body weight set-points: Determination and Adjustment1. *J Nutr*. 1997;127(9):1875S-1883S. doi:10.1093/jn/127.9.1875S
42. Schwartz MW, Woods SC, Porte D, Seeley RJ, Baskin DG. Central nervous system control of food intake. *Nature*. 2000;404(6778):661-671. doi:10.1038/35007534
43. Hall K, Farooqi IS, Friedman JM, et al. The energy balance model of obesity: Beyond calories in, calories out. *Am J Clin Nutr*. 2022;115(5):1243-1254. doi:10.1093/ajcn/nqac031
44. Speakman JR, Levitsky DA, Allison DB, et al. Set points, settling points and some alternative models: Theoretical options to understand how genes and environments combine to regulate body adiposity. *Dis Model Mech*. 2011;4(6):733-745. doi:10.1242/dmm.008698
45. Hall K, Guo J. Obesity energetics: Body weight regulation and the effects of diet composition. *Gastroenterology*. 2017;152(7):1718-1727.e3. doi:10.1053/j.gastro.2017.01.052
46. Blundell JE, Gibbons C, Beaulieu K, et al. The drive to eat in *homo sapiens*: Energy expenditure drives energy intake. *Physiol Behav*. 2020;219:112846. doi:10.1016/j.physbeh.2020.112846
47. Speakman JR. Thrifty genes for obesity, an attractive but flawed idea, and an alternative perspective: The 'drifty gene' hypothesis. *Int J Obes (Lond)*. 2008;32(11):1611-1617. doi:10.1038/ijo.2008.161
48. Taylor R, Holman RR. Normal weight individuals who develop type 2 diabetes: The personal fat threshold. *Clin Sci*. 2014;128(7):405-410. doi:10.1042/CS20140553
49. Cuthbertson DJ, Steele T, Wilding JP, et al. What have human experimental overfeeding studies taught us about adipose tissue expansion and susceptibility to obesity and metabolic complications? *Int J Obes (Lond)*. 2017;41(6):853-865. doi:10.1038/ijo.2017.4
50. Jastreboff AM, Kotz CM, Kahan S, Kelly AS, Heymsfield SB. Obesity as a disease: The Obesity Society 2018 position statement. *Obesity*. 2019;27(1):7-9. doi:10.1002/oby.22378
51. Jepsen CH, Bowman-Busato J, Allvin T, et al. Achieving consensus on the language of obesity: A modified Delphi study. *eClinicalMed*. 2023;62:102061. doi:10.1016/j.eclinm.2023.102061
52. Heymsfield SB, Wadden TA. Mechanisms, pathophysiology, and management of obesity. *N Engl J Med*. 2017;376(3):254-266. doi:10.1056/NEJMra1514009
53. Garvey WT, Mechanick JI. Proposal for a scientifically correct and medically actionable disease classification system (ICD) for obesity. *Obesity*. 2020;28(3):484-492. doi:10.1002/oby.22727
54. Kelsey MM, Zaepfel A, Bjornstad P, Nadeau KJ. Age-related consequences of childhood obesity. *Gerontology*. 2014;60(3):222-228. doi:10.1159/000356023
55. Kawai T, Autieri MV, Scalia R. Adipose tissue inflammation and metabolic dysfunction in obesity. *Am J Physiol Cell Physiol*. 2021;320(3):C375-C391. doi:10.1152/ajpcell.00379.2020
56. Lister NB, Baur LA, Felix JF, et al. Child and adolescent obesity. *Nat Rev Dis Primers*. 2023;9(1):24. doi:10.1038/s41572-023-00435-4
57. Pati S, Irfan W, Jameel A, Ahmed S, Shahid RK. Obesity and cancer: A current overview of epidemiology, pathogenesis, outcomes, and management. *Cancers (Basel)*. 2023;15(2):485. doi:10.3390/cancers15020485

58. Aune D, Sen A, Prasad M, et al. BMI and all cause mortality: Systematic review and non-linear dose-response meta-analysis of 230 cohort studies with 3.74 million deaths among 30.3 million participants. *BMJ*. 2016;353:i2156. doi:10.1136/bmj.i2156
59. Di Angelantonio E, Bhupathiraju SN, Wormser D, et al. Body-mass index and all-cause mortality: Individual-participant-data meta-analysis of 239 prospective studies in four continents. *Lancet*. 2016;388(10046):776-786. doi:10.1016/S0140-6736(16)30175-1
60. Li S, Fu Z, Zhang W. Association of anthropometric measures with all-cause and cause-specific mortality in US adults: Revisiting the obesity paradox. *BMC Public Health*. 2024;24(1):929. doi:10.1186/s12889-024-18418-9
61. Rubino F, Puhl RM, Cummings DE, et al. Joint international consensus statement for ending stigma of obesity. *Nat Med*. 2020;26(4):485-497. doi:10.1038/s41591-020-0803-x
62. Rubino F, Batterham RL, Koch M, et al. Lancet Diabetes & Endocrinology Commission on the definition and diagnosis of clinical obesity. *Lancet Diabetes Endocrinol*. 2023;11(4):226-228. doi:10.1016/S2213-8587(23)00058-X
63. Hebebrand J, Holm JC, Woodward E, et al. A proposal of the European Association for the study of obesity to improve the ICD-11 diagnostic criteria for obesity based on the three dimensions etiology, degree of adiposity and health risk. *Obes Facts*. 2017;10(4):284-307. doi:10.1159/000479208
64. Bouchard C. Genetics of obesity: What we have learned over decades of research. *Obesity*. 2021;29(5):802-820. doi:10.1002/oby.23116
65. Segal NL, Feng R, McGuire SA, Allison DB, Miller S. Genetic and environmental contributions to body mass index: Comparative analysis of monozygotic twins, dizygotic twins and same-age unrelated siblings. *Int J Obes (Lond)*. 2009;33(1):37-41. doi:10.1038/ijo.2008.228
66. Loos RJF, Yeo GSH. The genetics of obesity: From discovery to biology. *Nat Rev Genet*. 2022;23(2):120-133. doi:10.1038/s41576-021-00414-z
67. Mestre LM, Lartey ST, Ejima K, et al. Body mass index, obesity, and mortality—part I: Associations and limitations. *Nutr Today*. 2023;58(3):92-99. doi:10.1097/NT.0000000000000609
68. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med*. 1997;337(13):869-873. doi:10.1056/NEJM199709253371301
69. Tyrrell J, Wood AR, Ames RM, et al. Gene–obesogenic environment interactions in the UK biobank study. *Int J Epidemiol*. 2017;46(2):559-575. doi:10.1093/ije/dyw337
70. Oken E, Gillman MW. Fetal origins of obesity. *Obes Res*. 2003;11(4):496-506. doi:10.1038/oby.2003.69
71. McIntyre HD, Catalano P, Zhang C, Desoye G, Mathiesen ER, Damm P. Gestational diabetes mellitus. *Nat Rev Dis Primers*. 2019;5(1):1-19. doi:10.1038/s41572-019-0098-8
72. Larqué E, Labayen I, Flodmark CE, et al. From conception to infancy—early risk factors for childhood obesity. *Nat Rev Endocrinol*. 2019;15(8):456-478. doi:10.1038/s41574-019-0219-1
73. Lynch KE, Bourrat P. Interpreting heritability causally. *Philos Sci*. 2017;84(1):14-34. doi:10.1086/688933
74. Ang MY, Takeuchi F, Kato N. Deciphering the genetic landscape of obesity: A data-driven approach to identifying plausible causal genes and therapeutic targets. *J Hum Genet*. 2023;24(12):1-11. doi:10.1038/s10038-023-01189-3
75. Locke AE, Kahali B, Berndt SI, et al. Genetic studies of body mass index yield new insights for obesity biology. *Nature*. 2015;518(7538):197-206. doi:10.1038/nature14177
76. Masood B, Moorthy M. Causes of obesity: A review. *Clin Med*. 2023;23(4):284-291. doi:10.7861/clinmed.2023-0168
77. Swinburn B, Egger G, Raza F. Dissecting obesogenic environments: The development and application of a framework for identifying and prioritizing environmental interventions for obesity. *Prev Med*. 1999;29(6):563-570. doi:10.1006/pmed.1999.0585
78. Raubenheimer D, Simpson SJ, Tait AH. Match and mismatch: Conservation physiology, nutritional ecology and the timescales of biological adaptation. *Philos Trans R Soc B*. 2012;367(1596):1628-1646. doi:10.1098/rstb.2012.0007
79. Ludwig DS, Aronne LJ, Astrup A, et al. The carbohydrate-insulin model: A physiological perspective on the obesity pandemic. *Am J Clin Nutr*. 2021;114(6):1873-1885. doi:10.1093/ajcn/nqab270
80. Raubenheimer D, Simpson SJ. Protein appetite as an integrator in the obesity system: The protein leverage hypothesis. *Philos Trans Royal Soc B Biol Sci*. 1888;2023(378):20220212. doi:10.1098/rstb.2022.0212
81. Martínez Steele E, Raubenheimer D, Simpson SJ, Baraldi LG, Monteiro CA. Ultra-processed foods, protein leverage and energy intake in the USA. *Public Health Nutr*. 2018;21(1):114-124. doi:10.1017/S1368980017001574
82. Hall K, Ayuketah A, Brychta R, et al. Ultra-processed diets cause excess calorie intake and weight gain: An inpatient randomized controlled trial of ad libitum food intake. *Cell Metab*. 2019;30(1):67-77.e3. doi:10.1016/j.cmet.2019.05.008
83. Harb AA, Shechter A, Koch PA, St-Onge MP. Ultra-processed foods and the development of obesity in adults. *Eur J Clin Nutr*. 2023;77(6):619-627. doi:10.1038/s41430-022-01225-z
84. Gosby AK, Conigrave AD, Lau NS, et al. Testing protein leverage in lean humans: A randomised controlled experimental study. *PLoS ONE*. 2011;6(10):e25929. doi:10.1371/journal.pone.0025929
85. Gosby AK, Conigrave AD, Raubenheimer D, Simpson SJ. Protein leverage and energy intake. *Obes Rev*. 2014;15(3):183-191. doi:10.1111/obr.12131
86. Martinez-Cordero C, Kuzawa CW, Sloboda DM, Stewart J, Simpson SJ, Raubenheimer D. Testing the protein leverage hypothesis in a free-living human population. *Appetite*. 2012;59(2):312-315. doi:10.1016/j.appet.2012.05.013
87. Wells JCK, Marphatia AA, Amable G, et al. The future of human malnutrition: Rebalancing agency for better nutritional health. *Glob Health*. 2021;17(1):119. doi:10.1186/s12992-021-00767-4
88. Kroker-Lobos MF, Mazariegos M, Guamuch M, Ramirez-Zea M. Ultraprocessed products as food fortification alternatives: A critical appraisal from Latin America. *Nutrients*. 2022;14(7):1413. doi:10.3390/nu14071413
89. Keith SW, Redden DT, Katzmarzyk PT, et al. Putative contributors to the secular increase in obesity: Exploring the roads less traveled. *Int J Obes (Lond)*. 2006;30(11):1585-1594. doi:10.1038/sj.ijo.0803326
90. Ballon M, Botton J, Charles MA, et al. Socioeconomic inequalities in weight, height and body mass index from birth to 5 years. *Int J Obes (Lond)*. 2018;42(9):1671-1679. doi:10.1038/s41366-018-0180-4
91. Buoncristiano M, Williams J, Simmonds P, et al. Socioeconomic inequalities in overweight and obesity among 6- to 9-year-old children in 24 countries from the World Health Organization European region. *Obes Rev*. 2021;22(S6):e13213. doi:10.1111/obr.13213
92. Zhu W, Marchant R, Morris RW, Baur LA, Simpson SJ, Cripps S. Bayesian network modelling to identify on-ramps to childhood obesity. *BMC Med*. 2023;21(1):105. doi:10.1186/s12916-023-02789-8
93. Inoue K, Seeman TE, Nianogo R, Okubo Y. The effect of poverty on the relationship between household education levels and obesity in U.S. children and adolescents: An observational study. *Lancet Reg Health am*. 2023;25:100565. doi:10.1016/j.lana.2023.100565
94. Tomiyama AJ. Stress and obesity. *Annu Rev Psychol*. 2019;70(1):703-718. doi:10.1146/annurev-psych-010418-102936
95. Hinnouhu GM, Czernichow S, Dugravot A, Batty GD, Kivimaki M, Singh-Manoux A. Metabolically healthy obesity and risk of mortality:

Does the definition of metabolic health matter? *Diabetes Care*. 2013; 36(8):2294-2300. doi:10.2337/dc12-1654

96. Prospective Studies Collaboration, Whitlock G, Lewington S, et al. Body-mass index and cause-specific mortality in 900 000 adults: Collaborative analyses of 57 prospective studies. *Lancet*. 2009; 373(9669):1083-1096. doi:10.1016/S0140-6736(09)60318-4
97. Chang Y, Kim BK, Yun KE, et al. Metabolically-healthy obesity and coronary artery calcification. *J am Coll Cardiol*. 2014;63(24):2679-2686. doi:10.1016/j.jacc.2014.03.042
98. Opio J, Croker E, Odongo GS, Attia J, Wynne K, McEvoy M. Metabolically healthy overweight/obesity are associated with increased risk of cardiovascular disease in adults, even in the absence of metabolic risk factors: A systematic review and meta-analysis of prospective cohort studies. *Obes Rev*. 2020;21(12):e13127. doi:10.1111/obr.13127
99. Marcus Y, Segev E, Shefer G, et al. Metabolically healthy obesity is a misnomer: components of the metabolic syndrome linearly increase with BMI as a function of age and gender. *Biology*. 2023;12(5):719. doi:10.3390/biology12050719
100. Valenzuela PL, Santos-Lozano A, Barrán AT, et al. Joint association of physical activity and body mass index with cardiovascular risk: A nationwide population-based cross-sectional study. *Eur J Prev Cardiol*. 2022;29(2):e50-e52. doi:10.1093/eurjpc/zwaa151
101. Berg S. *AMA: Use of BMI alone is an imperfect clinical measure*. American Medical Association, 2023. Accessed July 31, 2023. https://www.ama-assn.org/delivering-care/public-health/ama-use-bmi-alone-imperfect-clinical-measure
102. Finucane MM, Stevens GA, Cowan MJ, et al. National, regional, and global trends in body-mass index since 1980: Systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9·1 million participants. *Lancet*. 2011; 377(9765):557-567. doi:10.1016/S0140-6736(10)62037-5
103. Abarca-Gómez L, Abdeen ZA, Hamid ZA, et al. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: A pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *Lancet*. 2017;390(10113):2627-2642. doi:10.1016/S0140-6736(17)32129-3
104. Zapata JK, Azcona-Sanjulian MC, Catalán V, et al. BMI-based obesity classification misses children and adolescents with raised cardiometabolic risk due to increased adiposity. *Cardiovasc Diabetol*. 2023; 22(1):240. doi:10.1186/s12933-023-01972-8
105. Si K, Hu Y, Wang M, Apovian CM, Chavarro JE, Sun Q. Weight loss strategies, weight change, and type 2 diabetes in US health professionals: a cohort study. *PLoS Med*. 2022;19(9):e1004094. doi:10.1371/journal.pmed.1004094
106. Jebeile H, Gow ML, Baur LA, Garnett SP, Paxton SJ, Lister NB. Treatment of obesity, with a dietary component, and eating disorder risk in children and adolescents: A systematic review with meta-analysis. *Obes Rev*. 2019;20(9):1287-1298. doi:10.1111/obr.12866
107. Jebeile H, McMaster CM, Johnson BJ, et al. Identifying factors which influence eating disorder risk during behavioral weight management: A consensus study. *Nutrients*. 2023;15(5):1085. doi:10.3390/nu15051085
108. Kuk JL, Ardern CI, Church TS, et al. Edmonton obesity staging system: Association with weight history and mortality risk. *Appl Physiol Nutr Metab*. 2011;36(4):570-576. doi:10.1139/h11-058
109. Ayala-Marín AM, Iguacel I, Miguel-Etayo PD, Moreno LA. Consideration of social disadvantages for understanding and preventing obesity in children. *Frontiers in Public Health*. 2020;8:423. doi:10.3389/fpubh.2020.00423
110. Grannell A, Fallon F, Al-Najim W, le Roux C. Obesity and responsibility: Is it time to rethink agency? *Obes Rev*. 2021;22(8):e13270. doi:10.1111/obr.13270
111. Heeren FAN, Darcey VL, Deemer SE, Menon S, Tobias D, Cardel MI. Breaking down silos: The multifaceted nature of obesity and the future of weight management. *Philos Trans Royal Soc B Biol Sci*. 1885;2023(378):20220215. doi:10.1098/rstb.2022.0215
112. Nadolsky K, Addison B, Agarwal M, et al. American Association of Clinical Endocrinology Consensus Statement: Addressing stigma and bias in the diagnosis and Management of Patients with obesity/adiposity-based chronic disease and assessing bias and stigmatization as determinants of disease severity. *Endocr Pract*. 2023;29(6):417-427. doi:10.1016/j.eprac.2023.03.272
113. Wells JCK. Obesity as malnutrition: The role of capitalism in the obesity global epidemic. *Am J Hum Biol*. 2012;24(3):261-276. doi:10.1002/ajhb.22253
114. Lobstein T, Baur L, Uauy R. Obesity in children and young people: A crisis in public health. *Obes Rev*. 2004;5(s1):4-85. doi:10.1111/j.1467-789X.2004.00133.x
115. Dietz WH, Baur LA, Hall K, et al. Management of obesity: Improvement of health-care training and systems for prevention and care. *Lancet*. 2015;385(9986):2521-2533. doi:10.1016/S0140-6736(14)61748-7
116. Bray GA, Ryan DH. Evidence-based weight loss interventions: Individualized treatment options to maximize patient outcomes. *Diabetes Obes Metab*. 2021;23(S1):50-62. doi:10.1111/dom.14200
117. Magkos F, Fraterrigo G, Yoshino J, et al. Effects of moderate and subsequent progressive weight loss on metabolic function and adipose tissue biology in humans with obesity. *Cell Metab*. 2016;23(4): 591-601. doi:10.1016/j.cmet.2016.02.005
118. Sjöström L. Bariatric surgery and reduction in morbidity and mortality: Experiences from the SOS study. *Int J Obes (Lond)*. 2008;32(7): S93-S97. doi:10.1038/ijo.2008.244
119. Jansen SCP, Hoorweg BBN, Hoeks SE, et al. A systematic review and meta-analysis of the effects of supervised exercise therapy on modifiable cardiovascular risk factors in intermittent claudication. *J Vasc Surg*. 2019;69(4):1293-1308.e2. doi:10.1016/j.jvs.2018.10.069
120. Legaard GE, Lyngbæk MPP, Almdal TP, et al. Effects of different doses of exercise and diet-induced weight loss on beta-cell function in type 2 diabetes (DOSE-EX): A randomized clinical trial. *Nat Metab*. 2023;5(5):880-895. doi:10.1038/s42255-023-00799-7
121. Hall K, Heymsfield SB, Kemnitz JW, Klein S, Schoeller DA, Speakman JR. Energy balance and its components: Implications for body weight regulation. *Am J Clin Nutr*. 2012;95(4):989-994. doi:10.3945/ajcn.112.036350
122. Michos ED, Lopez-Jimenez F, Gulati M. Role of glucagon-like Peptide-1 receptor agonists in achieving weight loss and improving cardiovascular outcomes in people with overweight and obesity. *J am Heart Assoc*. 2023;12(11):e029282. doi:10.1161/JAHA.122.029282
123. Nogueiras R, Nauck MA, Tschöp MH. Gut hormone co-agonists for the treatment of obesity: from bench to bedside. *Nat Metab*. 2023; 5(6):933-944. doi:10.1038/s42255-023-00812-z
124. Allison DB, Bier DM, Locher JL. Measurement rigor is not a substitute for design rigor in causal inference: Increased physical activity does cause (modest) weight loss. *Int J Obes (Lond)*. 2023;47(1):3-4. doi:10.1038/s41366-022-01234-9
125. Pontzer H. Exercise is essential for health but a poor tool for weight loss: A reply to Allison and colleagues. *Int J Obes (Lond)*. 2023;47(2): 98-99. doi:10.1038/s41366-022-01248-3
126. Ekelund U, Ward HA, Norat T, et al. Physical activity and all-cause mortality across levels of overall and abdominal adiposity in European men and women: The European prospective investigation into cancer and nutrition study (EPIC). *Am J Clin Nutr*. 2015;101(3): 613-621. doi:10.3945/ajcn.114.100065
127. Martin CK, Johnson WD, Myers CA, et al. Effect of different doses of supervised exercise on food intake, metabolism, and non-exercise

physical activity: The E-MECHANIC randomized controlled trial. *Am J Clin Nutr*. 2019;110(3):583-592. doi:10.1093/ajcn/nqz054
128. Posadzki P, Pieper D, Bajpai R, et al. Exercise/physical activity and health outcomes: An overview of Cochrane systematic reviews. *BMC Public Health*. 2020;20(1):1724. doi:10.1186/s12889-020-09855-3
129. Zhao M, Veeranki SP, Magnussen CG, Xi B. Recommended physical activity and all cause and cause specific mortality in US adults: Prospective cohort study. *BMJ*. 2020;370:m2031. doi:10.1136/bmj.m2031
130. Wang Y, Nie J, Ferrari G, Rey-Lopez JP, Rezende LFM. Association of physical activity intensity with mortality: A national cohort study of 403 681 US adults. *JAMA Intern Med*. 2021;181(2):203-211. doi:10.1001/jamainternmed.2020.6331
131. Rasmussen N. *Fat in the fifties*. Johns Hopkins University Press; 2019. doi:10.1353/book.66181
132. Hoyt CL, Burnette JL, Auster-Gussman L. "Obesity is a disease": Examining the self-regulatory impact of this public-health message. *Psychol Sci*. 2014;25(4):997-1002. doi:10.1177/0956797613516981
133. Monterosso J, Royzman EB, Schwartz B. Explaining away responsibility: Effects of scientific explanation on perceived culpability. *Ethics Behav*. 2005;15(2):139-158. doi:10.1207/s15327019eb1502_4
134. English S, Vallis M. Moving beyond eat less, move more using willpower: Reframing obesity as a chronic disease impact of the 2020 Canadian obesity guidelines reframed narrative on perceptions of self and the patient-provider relationship. *Clin Obes*. 2023;13(6):e12615. doi:10.1111/cob.12615
135. Mulder LB, Rupp DE, Dijkstra A. Making snacking less sinful: (Counter-)moralising obesity in the public discourse differentially affects food choices of individuals with high and low perceived body mass. *Psychol Health*. 2015;30(2):233-251. doi:10.1080/08870446.2014.969730
136. Nutter S, Alberga AS, MacInnis C, Ellard JH, Russell-Mayhew S. Framing obesity a disease: indirect effects of affect and controllability beliefs on weight bias. *Int J Obes (Lond)*. 2018;42(10):1804-1811. doi:10.1038/s41366-018-0110-5
137. Hurt RT, Varayil JE, Mundi MS, Martindale RG, Ebbert JO. Designation of obesity as a disease: lessons learned from alcohol and tobacco. *Curr Gastroenterol Rep*. 2014;16(11):1-7. doi:10.1007/s11894-014-0415-z
138. Dar-Nimrod I, Cheung BY, Ruby MB, Heine SJ. Can merely learning about obesity genes affect eating behavior? *Appetite*. 2014;81:269-276. doi:10.1016/j.appet.2014.06.109
139. Smith J, Ayre J, Jansen J, et al. Impact of diagnostic labels and causal explanations for weight gain on diet intentions, cognitions and emotions: an experimental online study. *Appetite*. 2021;167:105612. doi:10.1016/j.appet.2021.105612
140. Lebowitz MS, Pyun JJ, Ahn W-K. Biological explanations of generalized anxiety disorder: Effects on beliefs about prognosis and responsibility. *Psychiatr Serv*. 2014;65(4):498-503. doi:10.1176/appi.ps.201300011
141. Ringel MM, Ditto PH. The moralization of obesity. *Soc Sci Med*. 2019;237:112399. doi:10.1016/j.socscimed.2019.112399
142. Pearl RL, Lebowitz MS. Beyond personal responsibility: Effects of causal attributions for overweight and obesity on weight-related beliefs, stigma, and policy support. *Psychol Health*. 2014;29(10):1176-1191. doi:10.1080/08870446.2014.916807
143. Ahn WK, Lebowitz MS. An experiment assessing effects of personalized feedback about genetic susceptibility to obesity on attitudes towards diet and exercise. *Appetite*. 2018;120:23-31. doi:10.1016/j.appet.2017.08.021
144. Koob GF, Volkow ND. Neurocircuitry of addiction. *Neuropsychopharmacol*. 2010;35(1):217-238. doi:10.1038/npp.2009.110
145. Berthoud HR, Münzberg H, Morrison CD. Blaming the brain for obesity: Integration of hedonic and homeostatic mechanisms. *Gastroenterology*. 2017;152(7):1728-1738. doi:10.1053/j.gastro.2016.12.050
146. Devoto F, Zapparoli L, Bonandrini R, et al. Hungry brains: A meta-analytical review of brain activation imaging studies on food perception and appetite in obese individuals. *Neurosci Biobehav Rev*. 2018;94:271-285. doi:10.1016/j.neubiorev.2018.07.017
147. Gearhardt AN, DiFeliceantonio AG. Highly processed foods can be considered addictive substances based on established scientific criteria. *Addiction*. 2023;118(4):589-598. doi:10.1111/add.16065
148. Latner JD, Puhl RM, Murakami JM, O'Brien KS. Food addiction as a causal model of obesity: effects on stigma, blame, and perceived psychopathology. *Appetite*. 2014;77:79-84. doi:10.1016/j.appet.2014.03.004
149. Frank LE, Nagel SK. Addiction and moralization: The role of the underlying model of addiction. *Neuroethics*. 2017;10(1):129-139. doi:10.1007/s12152-017-9307-x
150. Latner JD, Ebneter DS, O'Brien KS. Residual obesity stigma: An experimental investigation of bias against obese and lean targets differing in weight-loss history. *Obesity*. 2012;20(10):2035-2038. doi:10.1038/oby.2012.55
151. O'Brien KS, Puhl RM, Latner JD, Mir AS, Hunter JA. Reducing anti-fat prejudice in preservice health students: A randomized trial. *Obesity*. 2010;18(11):2138-2144. doi:10.1038/oby.2010.79
152. Vartanian LR. Disgust and perceived control in attitudes toward obese people. *Int J Obes (Lond)*. 2010;34(8):1302-1307. doi:10.1038/ijo.2010.45
153. Hadlaczky G, Hökby S, Mkrtchian A, Carli V, Wasserman D. Mental health first aid is an effective public health intervention for improving knowledge, attitudes, and behaviour: A meta-analysis. *Int Rev Psychiatry*. 2014;26(4):467-475. doi:10.3109/09540261.2014.924910
154. Teachman BA, Gapinski KD, Brownell KD, Rawlins M, Jeyaram S. Demonstrations of implicit anti-fat bias: The impact of providing causal information and evoking empathy. *Health Psychol*. 2003;22(1):68-78. doi:10.1037/0278-6133.22.1.68
155. Puhl R, Suh Y. Health consequences of weight stigma: Implications for obesity prevention and treatment. *Curr Obes Rep*. 2015;4(2):182-190. doi:10.1007/s13679-015-0153-z
156. Heine SJ, Cheung BY, Schmalor A. Making sense of genetics: The problem of essentialism. *Hastings Cent Rep*. 2019;49(S1):S19-S26. doi:10.1002/hast.1013
157. Haslam N, Kvaale EP. Biogenetic explanations of mental disorder: The mixed-blessings model. *Curr Dir Psychol Sci*. 2015;24(5):399-404. doi:10.1177/0963721415588082
158. Reynolds JP, Vasiljevic M, Pilling M, Marteau TM. Communicating evidence about the environment's role in obesity and support for government policies to tackle obesity: A systematic review with meta-analysis. *Health Psychol Rev*. 2022;16(1):67-80. doi:10.1080/17437199.2020.1829980
159. Pickard H. Responsibility without blame for addiction. *Neuroethics*. 2017;10(1):169-180. doi:10.1007/s12152-016-9295-2
160. Vartanian LR, Porter AM. Weight stigma and eating behavior: A review of the literature. *Appetite*. 2016;102:3-14. doi:10.1016/j.appet.2016.01.034

How to cite this article: Sholl J, De Block A. The vices and virtues of medical models of obesity. *Obesity Reviews*. 2024;25(12):e13828. doi:10.1111/obr.13828