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REVIEW

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Philosophically, is obesity really a disease?

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Summary

The question of whether obesity should be regarded as a disease remains controversial. One source of controversy can be addressed by distinguishing between two ways in which the word "obesity" is used. In medicine, the word "obesity" now typically refers to some or all of a set of interrelated dysfunctions of metabolism, adipose tissue, and dietary intake regulation. In other contexts, such as government-funded public education programs, the word "obesity" refers to a body mass index (BMI) category taken to indicate excess body fat. The result is that when medical experts say, "Obesity is a disease," the majority of outside medicine inevitably takes this to mean "being fat is a disease." In order to address this ambiguity, we apply key philosophical accounts of disease to the two different senses of "obesity." We draw two major conclusions: First, although obesity as understood in clinical medicine meets the criteria to be considered a disease, obesity as defined by BMI does not. Second, adequately addressing this disease requires us to distinguish it clearly and unambiguously from high BMI. Making this distinction would help both the public and policymakers to better understand the disease of obesity, facilitating advances in both prevention and treatment.

KEYWORDS
adiposity-based chronic disease, dietary intake dysregulation, disease, philosophy

1 | INTRODUCTION

Within medicine, the debate on classifying obesity as a disease seems all but over and a clear winner has emerged: Obesity *is* a disease. The World Health Organization (WHO)[1] and, more recently, the European Commission[2] have recognized obesity as a disease, as have many other organizations.[3–6] Any attempt to relitigate this question might thus be seen as waking a sleeping dog, if not flogging a dead horse. Outside medical journals, symposia, and bariatric clinics, however, there remains widespread skepticism about the disease status of obesity. In the social sciences, the "medicalization" of obesity is widely claimed to have contributed to weight stigma and fatphobia, that is, the marginalization or oppression of people of higher weight.[7–11] In fact, in those circles, as well as in the fat liberation movement, the word "obese" is considered by many to be a slur,[12] and people-first language is said to make matters worse, not better.[13,14] For those researching or treating obesity, this may not seem like an urgent problem. After all, public opinion often deviates from the medical or scientific consensus, and few medical conditions are discussed in the same way across multiple academic disciplines. This is not usually taken to undermine the consensus within medicine, though it might be thought to indicate a need for more public education. However, in this case, the point at issue is, precisely, whether

Abbreviations: ABCD, adiposity-based chronic disease; BMI, body mass index; TOS, The Obesity Society; WHO, World Health Organization; WOF, World Obesity Federation.

doctors and scientists are best placed to do the educating. That is, the controversy is not over *how* obesity should be understood as a medical issue, but *whether* it should be understood as a medical issue. Medical experts may believe that the debate about the disease classification of obesity should be over, but they have no special authority to declare it to be over. The fact is that this debate is still going on, and as long as it does, it is important that medical experts remain at the forefront, communicating new findings and challenging misinformation.

Still, one might well wonder whether any useful discussion is even possible between those who research and treat obesity and those who regard "obese" as a slur. Philosophy of medicine offers a way forward, if not to resolution, at least to more fruitful interaction. A recent essay by Quill Kukla argues that the decision to classify a condition as a disease will always, necessarily, depend on one's goals and context; at bottom, Kukla claims, disease classifications are always made because they are useful in some way. This means that the same condition can be a disease in one context, and not a disease in another context. For example, they write that "… at one and the same time it is perfectly appropriate for Deaf parents to insist that their child's deafness is not a disease but rather an identity and a neutral or even positive variation on human capacities, and for insurance companies to classify it as a disease for purposes of coverage for people who do want treatment for it."[7] Kukla's point is not that we should use the concept of disease strategically until we uncover its true meaning, but that there is no true meaning outside of these pragmatic and strategic uses of the concept. "Disease" is an inherently pragmatic, strategic concept.

Within medicine, the classification of obesity as a disease has had many strategic benefits, especially in providing more and better treatment. But many would argue that these benefits have come at a cost, particularly to those who have a BMI in the "obese" category, but who either do not want or do not need medical treatment for this condition.[8,14–16] Following Kukla, we suggest that medical researchers and practitioners should take a pluralist view, defending the disease classification of obesity within clinical medicine while also acknowledging that defining obesity as a disease is not accurate or appropriate in all contexts outside of medicine. In this same spirit of pluralism, we suggest that, in defining obesity as a disease, our emphasis should be firmly on dysregulation of dietary intake, and on related dysfunctions of the metabolism and adipose tissue rather than on elevated BMI. This allows us to maintain and build on the pragmatic benefits of classifying obesity as a disease in the medical context, without committing us to the much more controversial and potentially harmful claim that everyone without a "normal" or "healthy" BMI has a disease.

2 | OBESITY: EXCESS WEIGHT OR EXCESS DIETARY INTAKE?

From a philosophical point of view, the question "Is obesity a disease?" concerns the relationship between the two terms "obesity" and "disease." The complexity surrounding the term "disease" is widely acknowledged. An expert panel convened by The Obesity Society (TOS), for example, notes that there is no "clear, specific, widely accepted, and scientifically applicable definition of disease."[17] What has not been widely acknowledged, however, is the lack of conceptual clarity around the other term in the question, "obesity." In fact, the word "obesity" is used in two distinct senses, one referring to body size as measured by BMI and the other to a physiological dysfunction. Consider that WHO has described obesity and overweight as "abnormal or excessive fat accumulation that may impair health."[18] The use of "may" here implies that abnormal or excessive fat accumulation can exist *without* impairment to health. According to this account, one might have obesity in the sense of falling into the "obese" BMI category but not (or not yet) have a disease if the excess or abnormal fat does not (yet) impair health. Thus, in this particular definition, the term "obesity" is clearly *not* referring to a disease: It would make no sense to say that a disease "may impair health" since disease just *is* the term we use to refer to a certain kind of impairment to health.

By contrast, groups like the World Obesity Federation (WOF) and TOS clearly define obesity *itself* as a disease.[4,6] In this context, the term "obesity" is being used differently from how it was used by WHO in the example above. Here, the word "obesity" refers directly to the actual disease, that is, to an impairment that already exists and not to a BMI range that may *cause* impairment. How did this ambiguity arise? How can it be that scientists ostensibly drawing on the same evidence can generate different and even contradictory definitions of obesity? We think that they are actually describing two different phenomena. The primary concern of WHO is population health with a goal to reduce the prevalence of disease in populations, so their emphasis in discussing obesity has been on what is seen as a modifiable risk factor, namely, the individual's body fatness or BMI. In the more clinically focussed guidelines from TOS and WOF, the goal is to help those who are already sick, and so, here, there is less emphasis on BMI, since this is only one dimension of the disease.[19,20] Our goal in this paper is not to argue for one of these definitions of the word "obesity" over the other but to highlight the fact that the word is used in different ways. This ambiguity around the word "obesity" hinders us when it comes to explaining the disease of obesity to the wider public. Much confusion and controversy might be avoided if we were to clarify that when doctors say that obesity is a disease, they do not mean that being "fat" is a disease.

In medicine, significant progress has been made in understanding the pathophysiology of obesity: The archaic view that obesity arises from "gluttony or sloth"[21] has been largely superseded by the recognition that it is "a heritable neurobehavioral disorder that is highly sensitive to environmental conditions."[22] Different models have been proposed to describe the complex underlying pathophysiology of obesity. For example, the "energy balance model" proposes that "the brain is the primary organ responsible for body weight regulation operating mainly below our conscious awareness via complex endocrine, metabolic, and nervous system signals to control food intake in response to the body's dynamic energy needs as well as environmental influences."[23] Conversely, the "carbohydrate insulin model"

proposes that "hormonal responses to highly processed carbohydrates shift energy partitioning toward deposition in adipose tissue, leaving fewer calories available for the body's metabolic needs."[24] According to this model, increasing adiposity causes overeating through effects on appetite to compensate for the sequestered calories. Although these two "competing paradigms" of obesity pathogenesis highlight the extent of scientific disagreement and controversy in this area, they remain aligned in considering the problem of obesity as one of dietary intake dysregulation rather than excess body weight.

Whatever the cause underlying the "neurobehavioural disorder" of obesity, it is usually manifested as an excess accumulation of dysfunctional adipose tissue.[25] It has become increasingly clear that adipose tissue is not just a passive storage depot but also an active endocrine organ, producing hormones including leptin and adiponectin, and that, in people with obesity, the adipose tissue functions less well. This new knowledge has led to better treatments, including drugs like semaglutide and tirzepatide.[26,27] These drugs, like bariatric surgery,[28] typically lead to significant weight loss and to improvements in overall metabolic health. However, it does not necessarily follow that the therapeutic effects are caused by weight loss as such. For example, metabolic improvements after bariatric surgery occur quickly and precede any weight loss.[29] Rather, effective obesity treatments such as drug therapy and surgery act to alter the influence of gut- and fat-derived hormones on the parts of the brain which control appetite.

In public health, on the other hand, there has been a huge emphasis on BMI, rather than on the mechanisms that trigger overeating and weight gain. In fact, especially since the 1990s, public health education has often presented "obesity" simply as the label for a BMI above 30 $kg{\cdot}m^{-2}$.[30] In short, most people have been taught to understand "obesity" as referring to a BMI category. In this context, the claim that obesity is a disease will naturally often be seen as confusing, if not downright laughable.[31–33] Public understanding of the disease of obesity could thus be improved significantly if it was made clear that although the disease usually causes increased BMI, increased BMI (or increased adiposity however we measure it) is not itself the disease; any more than tar staining in smokers is a disease. This distinction is especially important given the relative crudeness and variability of BMI as a predictor of other obesity-related disease states. For example, the equivalent incidence of type 2 diabetes at a BMI of 30 $kg{\cdot}m^{-2}$ in White British adults occurs at 23.9 $kg{\cdot}m^{-2}$ in South Asian British adults.[34] The threshold at which BMI reflects excess adiposity differs according to several confounding factors, as well as the underlying obesity-related disorder under consideration. Machine learning approaches, using metabolomic, proteomic, and gut microbiome data in large cohort studies are beginning to unravel the complex molecular basis for the heterogeneity in the relationship between BMI and adverse metabolic outcomes.[35] These observations have prompted organizations such as the American Association of Clinical Endocrinologists (AACE) and the American College of Endocrinology (ACE) to move away from a sole reliance on the BMI and to "emphasise a complications centric approach" to therapeutic strategies and outcome prioritization and also "alludes to a precise pathophysiologic basis," proposing "adiposity-based chronic disease" (ABCD) as a new diagnostic term for obesity.[36] So, the movement away from treating an elevated BMI as a disease in its own right by some clinicians has begun, but a wider appreciation of the philosophical basis for seeing the disease of obesity as a neurobehavioural disorder rather than merely as excess weight or fat has yet to emerge.

3 | PHILOSOPHICAL ACCOUNTS OF DISEASE

In this section, we outline some of the most important recent philosophical accounts of disease. Our goal here is not to provide a once-and-for-all, definitive answer to the question of whether obesity is a disease. Indeed, if Kukla is right, such a definitive answer may be neither possible nor desirable. Nonetheless, this does not preclude discussion of how best to understand a particular disease given the goals and assumptions of medicine.[7] There may not be one, true, universal account of disease, but there are better and worse accounts of particular diseases. Some accounts of obesity will do a better job than others of helping us to achieve our goals, especially treatment and prevention. In our view, there are at least two reasons why it is more effective to define the disease of obesity in terms of its impact on dietary intake, metabolism, and adipose tissue rather than in terms of body size. First, it avoids creating needless controversy with, or harm to, those who have a high BMI but do not see this as a medical issue. Second, it accords better with popular and medical usage of the term "disease," thus making for a better public understanding of the condition. To demonstrate this second point, we turn to the philosophy of medicine because, if there is one thing that field has done better than any other, it is to gather, analyze and summarize the different ways in which the term "disease" is used, particularly in medicine but also in the wider culture.[37] In short, the philosophy of medicine provides a ready-made, comprehensive list of plausible definitions of disease. On any of these definitions, we argue, obesity is best understood as a disease of dietary energy intake dysregulation, not one of body size.

3.1 | Biology or culture?

A key question for the philosophy of medicine is how best to define the concepts of health and disease. Possible answers have often been categorized as either naturalist or normativist. Naturalists claim that disease and related concepts such as health can be defined without reference to values, whereas normativists reject this claim.[38,39] Thus, for example, the naturalist Christopher Boorse argues that disease can be defined in purely biostatistical terms, as quantitative deviation from normal functioning.[40] The normativist Rebecca Cooper, by contrast, compares the difference between diseases and other conditions to that between flowers and weeds.[41] No matter how carefully we examine some plant or how accurately we trace the evolution of its species, this could never reveal whether it is a weed, because weeds are not what philosophers call a "natural kind."[42] Instead, a weed is a

plant that humans value in a certain way, as undesirable and intrusive, for example. A crucial question for philosophers of medicine is whether "disease" is like "weed" in this respect.

Recently, however, philosophers have argued that so-called naturalists and normativists disagree on more than just this question. And their disagreements are not always consistent: It is by no means unusual to take a seemingly "naturalist" position on one issue and a seemingly "normativist" position on another.[38,43] In fact, few philosophers now defend an entirely value-free concept of disease (though a recent paper by Veit[44] provides a notable exception), while many who endorse a normative account of disease are nonetheless keen to show that their theories are still compatible with natural science.[45,46] In short, it is no longer possible to neatly categorize philosophical theories of disease as either naturalist or normativist. Instead, we categorize theories of disease into those that prioritize biology and those that prioritize culture.

The biology/culture distinction can also be seen in the various debates about obesity that are currently going on in society. Although, as noted above, medicine and public health may sometimes describe obesity in different ways, their descriptions are both fundamentally scientific, and thus, both disciplines fall on the biological side. They acknowledge the role of culture in shaping how we understand and respond to obesity, but for them, questions about obesity are, first and foremost, empirical questions. By contrast, for those who reject the claim that obesity is a disease, the starting point is often cultural, not biological. Their primary concern is to make sense of how the concept of obesity has been constructed in and by different societies and cultures. Just as scientists do not ignore cultural questions, this culture-centered approach likewise does not necessarily deny biology, though it often involves critical analysis of how biology has been interpreted.

In the past few years, two significant articles have compared obesity with philosophical concepts of disease.[47,48] They both conclude that obesity is a disease in the culture-centered sense but disagree on the biological aspect. Hofmann argues that obesity does not meet the biological criteria for disease. However, his account of obesity centers largely on body weight and BMI rather than on dysfunctions of food intake regulation or adipose tissue. Kilov and Kilov, by contrast, give a fuller account of the pathophysiology of obesity, on the basis of which they conclude that it meets both biology- and culture-centered definitions of disease. This conclusion is well supported by both evidence and argument, but it can be made even more robust by distinguishing between the two different senses of "obesity" outlined above. In short, in our view, the case that obesity should be seen as a disease in the medical sense and for medical purposes is made all the stronger when we focus on the underlying pathophysiological basis for obesity and not on BMI.

3.2 | Culture-based theories: Disease as an evaluative concept

In the humanities and social sciences, there is broad agreement that, if obesity is a disease at all, it is a socially constructed one.[10,49–52] How should we understand this claim? For those who prioritize such culture-based theories of disease, "disease" is indeed comparable to "weed," to use Cooper's analogy. That is, on this account, "disease" is a term that some society or group has applied to some condition it considers abnormal or undesirable.[53] Those who are classified as having a disease then respond to that classification, and their response in turn shapes how the classification is understood. Social construction thus, in Hacking's term, loops.[54] For defenders of this culture-based view, then, the diseases recognized as such in a given culture or society will often reflect the values and structures of that culture or society. Consider the example of homosexuality, widely discussed by philosophers of medicine,[37,55–58] which was listed in the first two editions of the DSM but not in the third or subsequent editions.[59] Or consider the proposal, in 1851, of drapetomania, a disease that supposedly caused enslaved people to try to escape from their "masters."[7] By contrast, Kilov and Kilov give the example of the Hmong culture, in which epilepsy is not seen as a disease but as a spiritual gift conferring special status.[47] Such cases seem to support the claim that "disease" is a normative, culture-driven category that is used and applied differently in different times and places.

This is not to say that diseases are not real, in the everyday sense of that word. Even from within a culture-centered perspective, one can make a strong case that drapetomania and homosexuality are not diseases but that, say, COVID-19 infection and pancreatic cancer are. Moreover, one can endorse a culture-driven concept of disease without taking a relativist position or denying biology. Glackin, for example, has argued that, although it may be a matter of interpretation (and thus a conceptual, and cultural, question) whether a given condition ought to be seen as a disease, whether a particular individual has that condition is a matter for empirical observation, over which there is no room for interpretation.[45,46]

Both Kilov and Kilov and Hofmann consider obesity to meet the criteria for disease in this culture-centered sense.[47,48] Hofmann's account, however, does not fully engage with the emerging understanding of the physiology of obesity. This is a perfect example of the confusion that arises from the conflation of the two senses of the term "obesity." Hofmann and those who defend the disease classification of obesity are effectively talking past each other because they simply mean different things by "obesity." Obesity as understood in the medical context is much more than a BMI category. The existence of people who are of normal weight but "metabolically obese" has been discussed for decades.[60,61] It is also well known that conditions like insulin resistance, dyslipidemia, and non-alcoholic fatty liver can occur in lean people: Patients with lipodystrophy, for example, typically display the metabolic complications associated with obesity but are usually lean with a "normal" BMI, because of an absence of an ability to store "fuel" correctly in fat cells.[62] Meanwhile, surgical removal of adipose tissue does nothing to reverse the metabolic disorders like insulin resistance that are associated with excess body fat.[63] By contrast, metabolic/bariatric surgery causes significant and sustained improvement in metabolic health, even when it does not result in "normal" or "healthy" weight or BMI. Indeed, these effects are so marked that some have argued that this surgery should be seen

primarily as a treatment for metabolic illness and not for excess weight, at least in those patients with obesity-related metabolic dysfunction.[64] In other words, metabolic/bariatric surgery has a profound effect on the "gut–brain axis" and works to reduce dietary intake by changing hormonal influences on the regulation of food intake rather than by an independent effect on fat mass.[65] Hofmann rightly argues that excess weight is not a disease. But, as these observations show, it has been quite some time since medicine (as opposed to public health) has defined obesity merely as high BMI or excess weight. It is thus necessary to reconsider whether and how obesity meets the criteria of biology-based theories of disease.

3.3 | Biology-based theories: Disease as (harmful) dysfunction

Those theorists who prioritize biology generally say that, to say some condition is a disease, is, first and foremost, to state a set of biological facts: Fundamentally, on this account, to say "obesity is a disease" would be to say that when a person has obesity, some part or process of their body is not functioning correctly. The best-known example of a strictly biological account of disease is probably that of Boorse, who continues to defend his biostatistical theory despite widespread criticism.[40,66,67] This theory attempts to define disease, and related concepts such as health, in purely statistical terms. Boorse argues that it is possible to identify and describe diseases without referring to any human interests, preferences, or values, instead of relying only on statistical comparison between a given individual and others of their age, sex, and species. There are, though, a number of criticisms of Boorse's account that are widely taken to be fatal. (A recent summary of these criticisms is offered by Smart.[68]) A more promising biology-based account, one that resists at least some of these criticisms, can be found in Wakefield's harmful dysfunction analysis.[69–71] Wakefield agrees with Boorse that diseases are biological dysfunctions, but his account of dysfunction is based on the idea of selected effect, not on statistical comparison. For Wakefield, dysfunction is "… a failure of some physiological or psychological mechanism to perform a natural function that it was biologically designed to perform."[72] This selected effect view of functioning is now seen by many as the firmest ground on which to base a biological account of disease.[44,73,74] For Wakefield, however, to be a disease, a condition must be not only dysfunctional but also harmful. His discussion of the case of Mary Mallon, also known as Typhoid Mary, illustrates this point. Citing contemporary medical reports, Wakefield writes, "… there could not be a clearer case of internal dysfunction than Mary's, with bacteria utilizing her gall bladder as a spawning ground … ." [71] Yet, since Mallon had no symptoms, she herself was not harmed by the dysfunction, and so, for Wakefield, we cannot say she herself had a disease.

Kilov and Kilov argue that obesity meets both the biological dysfunction and the harm requirements. The biological dysfunction in question is, they write, a set of "… complex pathological adaptations of the arcuate nucleus in response to an obesogenic environment."[47] However, their inclusion of stigma as a harm of obesity is at odds with biology-centered philosophical accounts of disease such as that of Wakefield. This is because stigma and discrimination arise from the social and cultural meanings attached to bodies that are considered "fat" not from the dysfunction of obesity itself. A person without obesity wearing a sufficiently convincing "fat suit" would be treated the same (stigmatizing) way as a person who really has the disease of obesity. Conversely, a person who has the disease of obesity would likely experience less stigma if they lost weight, but this person would still have the disease, since the disease is chronic. In other words, the stigma is a response to the person's appearance and not to their physiological (dys)functioning. To make the point clearer, consider the case of homosexuality. There is now almost universal agreement in medicine, psychology, and other relevant disciplines that homosexuality is not a disease. Nonetheless, homosexuality remains profoundly stigmatized in many places. Should the widespread existence of homophobia cause us to revisit the question of whether homosexuality is a disease? Surely not. There is no doubt that weight stigma is pernicious and harmful and must be urgently and thoroughly addressed. But, just as homophobia tells us nothing about whether homosexuality is a disease, weight stigma is irrelevant to the question of whether obesity is a disease.

Another point about harm and obesity also deserves further clarification. A common objection to the claim that obesity is a disease is the observation that some people with a high BMI do not seem to experience any harm as a result.[10,75,76] Defenders of the idea that excess weight is itself pathological often assert that even if it is not currently harmful, excess body fat eventually causes harm. The WOF, for example, emphasizes that obesity is a "chronic, relapsing disease *process*."[4] Similarly, Kilov and Kilov point out that even metabolically healthy obesity is predictive of increased mortality, cardiovascular disease, type 2 diabetes, and non-alcoholic fatty liver disease.[47] They take this as evidence that obesity meets naturalist criteria for disease. But this very much depends on *which* naturalist—or, in our terms, biology-centered—account of disease one has in mind. Certainly, risk is not sufficient to meet Wakefield's harm requirement. It is indeed well known that people with higher levels of adiposity are at a significantly increased risk of morbidity and mortality, even if they currently are or seem metabolically healthy. But, unless and until they are harmed—actually, not potentially, harmed—we cannot say that they *have* a disease as defined by Wakefield.

That said, our goal here is not, ultimately, to endorse a particular philosophical account of disease. It may be that Wakefield, or any other philosopher, is simply wrong about disease. Or it may be that, as Kukla argues, disease designation is inevitably and irreducibly pragmatic. If so, what matters is that we define and describe the disease of obesity in such a way as to justify treatment for those who need it, without imposing any burden—moral, emotional, financial, or otherwise—on those who do not. One way to do this might be to follow Walker and Rogers, who suggest that "disease" is an inherently vague term and that it is simply impossible to say categorically whether a certain condition is or is not a disease.[77] In such cases, the preferences of the patient may then be the deciding factor in whether and how a condition should be treated.[77] On Walker's and Rogers' theory of disease, we could say that metabolically healthy obesity sits

on the borderline between disease and non-disease states and then take a case-by-case approach, assessing the benefits and disbenefits of treatment for each person given their particular goals and circumstances. However, in the case of obesity, the majority of borderline cases can be avoided altogether if we simply define the disease as distinct from—though often co-occurring with—the condition of excess BMI or body weight (however "excess" might be defined given current best medical evidence). The genetic predisposition to take in more energy than is needed over a prolonged period is both dysfunctional and harmful. High BMI may be a sign of that dysfunction and may bring its own harms (which may themselves require treatment), but it is neither necessary nor sufficient for disease.

Thinking about obesity from the selected effects point of view provides further clarity: We can infer that certain physiological functions have evolved to regulate dietary intake, in order to maintain energy homeostasis at a level that would best contribute to survival or reproduction. The disease of obesity can be seen as a heterogenous disorder of dietary energy homeostasis, with significant interindividual variation in the underlying causes.[78] This disorder is certainly dysfunctional. As a result of this dysfunction, excess energy is stored in adipose tissue, perhaps to the point where a person's body weight becomes harmful (for example due to obstructive sleep apnoea or pressure on the joints). But in this scenario, it is not the adipose tissue that is failing to perform the function for which it was selected in evolution. Indeed, we can plausibly infer that adipose tissue was selected in part for its ability to fulfill just this function.[25] Once a certain amount of weight gain has occurred, adipose tissue itself may *become* dysfunctional, behaving in ways that run counter to the functions for which it was selected, and triggering a vicious cycle of further dysregulation of dietary intake.[25] But the initial "lesion" is that physiological failure to regulate dietary intake, not the amount of adipose tissue. Irrespective of an individual's biological predisposition to dysregulation of dietary intake, which is strongly genetically influenced, the increases in population levels of this dysregulation over time have been driven by changes in the food environment. These proposals are consistent with the so-called "set point" theory of eating behavior and body weight regulation[79] and also with observations that effective obesity treatments such as drug therapy[80] and surgery[81] influence hunger and satiety at the hormonal level in a way that ineffective treatments such as calorie restriction in isolation cannot.[82] Most of the changes in concentrations of appetite-mediating hormones that influence this set point occur early; by that time, about 5% weight loss is achieved.[83]

Another crucial issue for biology-centered accounts of disease in general, but especially when applied to obesity, is that of environment. To give a satisfactory account of dysfunction, it is necessary to assess an individual's functioning relative to prevailing conditions and environment.[84] When assessing a person's arterial oxygen saturation via pulse oximetry, for example, it would be important to know if they have been acclimatized to a high-altitude environment like those found in the Andes.[85] In the case of obesity, it is similarly important to take account of environment, since a dysfunction that causes excess dietary intake is only harmful in circumstances where food—especially energy-dense food—is easily and consistently available. Smart's dispositional account of disease causation provides a useful way to explain this interaction between individuals and environment.[68,86] On this account, to say an environment causes a disease is to say that a certain feature (or disposition) of that environment interacts with a disposition or tendency in the individual in such a way as to bring about the condition in question. Thus, the obesogenic environment[87] can be said to cause obesity in the sense that the ubiquitous availability of energy-dense food triggers the tendency of some humans toward dysregulated dietary intake. Many humans, those without the disposition to obesity, have a physiological ability to adjust their food intake to maintain energy balance despite the abundance of energy available to them. But people with the disposition to obesity lack the physiological ability to make those adjustments to compensate for a hyper-abundant environment. This causes them to eat more than they otherwise would, which, in turn, leads to the vicious cycle of obesity. Some have attributed dysregulated dietary intake to food addiction,[88] though this proposal has always garnered controversy,[89] especially as the brain regions (such as the amygdala) and neurotransmitters (such as dopamine) associated with addiction are distinct from those in the hypothalamus associated with regulation of appetite and satiety. Either way, however, the disposition exists within the individual—as evidenced by the fact that not everyone is affected by the environment in the same way—but this disposition only becomes a disease when it is triggered by a facilitating disposition of the environment. In the case of obesity, it is the environment that offers the best hope for prevention. We cannot easily eliminate either the disposition to overeat when food is easily available or the putative dopaminergic effects of certain foods (though developments in pharmacotherapy mean that now, the impact can sometimes be counteracted). But it is possible to change at least some features of the environment to make it less obesogenic.

4 | DISCUSSION

We draw two main conclusions from this application of recent philosophical literature to obesity. First, the disease of obesity centers on the mechanisms by which dietary intake is regulated, and not on body weight or BMI. Addressing this disease requires both medical treatment for affected individuals and environmental measures to reduce the risk to the population. Second, this understanding of the disease is inconsistent with how the word "obesity" is typically understood by the general public. If the goal is to increase public understanding of this disease and to improve prevention and treatment, then perhaps it is time to reconsider whether the name "obesity" conveys the reality that this is a complex disease, manifesting in dysregulated dietary intake, dysfunctional adipose tissue, and dysfunctional metabolism, or whether it just creates needless confusion and controversy.

4.1 | Prevention and treatment

In "Sick Individuals and Sick Populations," Rose distinguishes between what he calls the causes of cases and the causes of incidence.[90] That

is, he distinguishes between two different questions one can ask, namely, "Why did this patient get this disease at this time?" and "Why is this disease so common in this population?" All too often, efforts to educate the public about obesity have ignored this latter question, treating individual behavior change as both cause and solution. Having a high BMI has been framed as an adverse behavior rather than as an outcome and as something that might be amenable to reversal with education and motivation. We have been aware of the shift in the population distribution of body weight since its first description by Kuczmarski in 1992.[91] Yet, it seems implausible that between 1980 and 2020, millions of individuals of different ages, sexes, and cultural backgrounds had a collective lapse of self-discipline, work ethic, or moral integrity.[92] Rather, the radical change in this period occurred in the food environment. To put it in the terms of Smart's dispositional account of disease causality, there were likely always people who had the disposition or tendency toward dysregulated dietary intake, but it was only when large groups gained access to unlimited amounts of food that this disposition could go into effect.[68] To paraphrase an anonymous COVID-era poem, it was "same storm, different boats."

For those in whom the disease is already in full effect, medical treatment tailored to the individual is essential. However, public health has a crucial role to play too, especially in prevention. Rose distinguishes "population" strategies from "high-risk" strategies. Population strategies aim to reduce the overall prevalence of exposure to some risk factors. The workplace smoking ban in Ireland[93] is an example of a population strategy, as is the sugar-sweetened drinks levy introduced in 2018.[94] The focus on weight and BMI rather than on dysregulation of dietary intake has led us down the wrong path when it comes to obesity prevention. If environmental changes have driven the so-called "obesity epidemic," then the modifiable risk factor to which individuals are exposed is *not* high BMI or even excessive body fat as such but the food environment, which triggers, and then facilitates, continued overeating. High-risk strategies ought not to focus on those with the highest BMI, just as Rose's high-risk intervention did not just target those who smoke the most. Rather, they should be aimed at those who not only have high BMI but also display other signs of risk such as family history. As for population strategies, anti-obesity measures should not be primarily concerned with body weight itself, just like tobacco control measures are not concerned with tar staining on smokers' fingers. Often, current public health obesity prevention strategies do the equivalent of stigmatizing people with tar-stained fingers rather than restricting access to tobacco products and providing adequate treatment for those with tobacco addiction or tobacco-related disease. Although there is good evidence that purposeful weight loss improves health in people who have a high body mass index, it is likely that these benefits arise from a reduction in calorie intake rather than as a result of the reduction in the mass of fat tissue directly. The former is necessary for the latter, but it more directly addresses the underlying pathophysiological perturbation associated with the complications of obesity. As the philosopher Alex Broadbent noted, the health benefits associated with weight loss depend on how that weight loss was achieved.[95]

4.2 | Communicating obesity science

Philosophical and medical analysis both point to the conclusion that the disease of obesity centers on the physiological causes and effects of excess dietary intake, not on BMI. However, the fact is that, thanks to sustained and widespread public health education campaigns around BMI, the general public understands the term "obesity" as referring to excess weight as measured by BMI. Even medical researchers use terms like "overweight," "obese," and "heavy" interchangeably.[96] BMI categories are typically labeled in terms of weight, not risk, as in this UK NHS explainer[97] that lists the BMI categories as under*weight*, normal *weight*, over*weight*, and obese. This emphasis on weight is perhaps, in part, a vestige of an earlier era in which insurance companies published tables of ideal or desirable weight ranges, based on their observation that "excess weight" was associated with an increased risk of mortality. But whatever the historical roots, the result is that, at least outside of medicine, the claim that "obesity is a disease" will almost inevitably be misconstrued as the claim that "*excess weight* is a disease" or, more colloquially, "being fat is a disease." This, in turn, confirms a long-held prejudice that those with higher BMI have, by definition, failed to protect their health, a claim which is often used to justify weight stigma.[98] Perhaps part of the reason that people express confusion about obesity as a disease is that they have been taught to think of obesity as a risk factor, and we do not usually call diseases after their best-known risk factors. For example, as Broadbent points out, smokers are much more likely to get lung cancer but lung cancer is not referred to as "smoking-it is" or "smoking disease."[95] Nor, we can add, is smoking referred to as "pre-lung cancer." There are, of course, important ways in which smoking is *not* analogous to obesity; in particular, having obesity is not a behavior. But the comparison reminds us that calling this disease "obesity" in a context where "obesity" is usually taken as synonymous with "excess weight" is inevitably confusing. If the public at large is to understand, let alone endorse, the medical conception of the disease of obesity, it must be made clear that having a high BMI does not mean having the disease of obesity, and vice versa.

One possibility is to give the disease a name other than "obesity." There are precedents for changing medical terminology when it has outlived its usefulness or even become harmful. Over the course of the 20th century, terms like "feeble-minded" and "moron" were dropped from medical usage because they had started to be used in a pejorative way in the wider culture. Although "obese" may not be thrown around as a casual insult in the way "imbecile" or "moron" came to be, there is evidence that it carries negative connotations[99] and, as we have seen, some people consider it a slur.[12] Giving the disease a different name could help us to pursue the strategic goals of clinical medicine without causing needless controversy with those who, given their own goals and contexts, understand BMI or body weight in a radically different way. There is an intuitive appeal in the idea of a disease that affects the appetite and makes it difficult to regulate one's intake. But much of this intuitive appeal gets lost in translation when we call this disease "obesity." Most people already think

they know what obesity means—and, to them, it does not mean a heritable neurobehavioural disorder of regulation of dietary intake, it means having a high BMI or just being fat. But of course, this is a misunderstanding of the current state of obesity science: As is now widely acknowledged, BMI tells us nothing about the proportion of lean and fat mass, nor the distribution of body fat, or how well it serves as a storage depot for excess "fuel." The proposal for ABCD[36] is a huge step in the right direction, as this terminology represents a much more nuanced and clinically-focussed understanding of the disease. However, the term ABCD still emphasizes adiposity, which may encourage rather than challenge the popular misconception that the disease should be defined by body size. An advantage of the ABCD concept is that it acknowledges the role of adipocyte dysfunction (which often, but not always, arises from enlargement of the adipocyte due to excess dietary intake, and which can be quantified by measuring adipocyte-derived hormones such as leptin and adiponectin)[100] rather than fat mass per se. The term "corpulence"[101] has been proposed previously, but we think it is similarly unlikely to break the association between body size and disease, an association that is central to weight stigma. Further discussion is needed on what terminology might better distinguish the disease from the BMI category, but there can be little doubt that this is a distinction we must make clear to the wider public. Such clarity could significantly improve public understanding not only of the disease but also, crucially, of the medical, surgical, and other treatments now available to address it. This increased clarity would likely make the term "overweight" obsolete, and it would mean we no longer refer to "weight management" when describing specialist clinical services for patients with obesity complications. In short, it would serve to emphasize the reality that obesity medicine is about getting sick people well, not making "fat" people thin. This distinction—between the disease, on the one hand, and the cultural desire for thinness on the other—is more important than ever right now, as clinicians and patients around the world seek publicly funded access to new treatments. We have come so far in our understanding of obesity, but the ambiguity surrounding the word "obesity" itself remains a barrier to effective communication of that progress and, thus, to much-needed improvements in treatment and prevention. If, as Kukla argues, disease classifications are always strategic, why not be strategic in how we name the disease currently known as obesity?

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CONFLICT OF INTEREST STATEMENT

No conflict of interest statement.

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