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SCIENTIFIC CONTRIBUTION

The biopsychosocial model: Its use and abuse

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Abstract
The biopsychosocial model (BPSM) is increasingly influential in medical research and practice. Several philosophers and scholars of health have criticized the BPSM for lacking meaningful scientific content. This article extends those critiques by showing how the BPSM's epistemic weaknesses have led to certain problems in medical discourse. Despite its lack of content, many researchers have mistaken the BPSM for a scientific model with explanatory power. This misapprehension has placed researchers in an implicit bind. There is an expectation that applications of the BPSM will deliver insights about disease; yet the model offers no tools for producing valid (or probabilistically true) knowledge claims. I argue that many researchers have, unwittingly, responded to this predicament by developing certain patterns of specious argumentation I call "wayward BPSM discourse." The arguments of wayward discourse share a common form: They *appear* to deliver insights about disease gleaned through applications of the BPSM; on closer inspection, however, we find that the putative conclusions presented are actually *assertions* resting on question-begging arguments, appeals to authority, and conceptual errors. Through several case studies of BPSM articles and literatures, this article describes wayward discourse and its effects. Wayward discourse has introduced into medicine forms of conceptual instability that threaten to undermine various lines of research. It has also created a potentially potent vector of medicalization. Fixing these problems will likely require reimposing conceptual rigor on BPSM discourse.

Keywords Biopsychosocial model · Medicalization · Informal fallacies · Defining diseases · Medically unexplained illness · George Engel

Since its articulation by George Engel (1977), the biopsychosocial model (BPSM) has enjoyed growing acceptance and use in medicine. A recent major work on the BPSM described the model as having "become the orthodox overarching model for health, disease and healthcare" (Bolton and Gillett 2019, 5). Such an assessment of the BPSM's place in contemporary medicine is arguably overstated (Wade and Halligan 2017). But perhaps not by much. The BPSM literature has grown exponentially in recently decades, and prominent researchers in medical subfields such as psychiatry, chronic illness, spine care, and disability studies have used terms like "status quo," "overarching conceptual framework," and "dominant" to characterize the BPSM's status (Bolton and Gillett 2019; Edwards et al. 2016; Ghaemi 2011; McLaren 2021; Weiner 2008). The BPSM is also increasingly taught in medical schools and healthcare trainings (Barron et al. 2021; Bolton and Gillett 2019).

It is therefore surprising that the BPSM has received relatively little critical scrutiny from medical scholars and philosophers. To be sure, the BSPM is sometimes discussed in the health philosophy literature (see, e.g.: Berghmans et al. 2009; Kelly et al. 2014; Boisaubin and McCullough 2004; Brendel 2003; Solli and Da Silva 2012; Lindau et al. 2003). There have also been a few more extended philosophical and theoretical discussions of the BPSM's strengths and weaknesses (Bolton and Gillett 2019; Gask 2018; Gatchel and Turk 2008; Ghaemi 2009, 2010, 2011; McLaren 1998, 2021; Van Oudenhove and Cuypers 2014; Saraga et al. 2014; Weiner 2008). Nonetheless, as Weiner (2008, 211) notes, the model has received "remarkably little" close attention in light of its widespread influence.

The lack of attention is especially surprising given the serious questions raised by some existing criticisms of the BPSM. McLaren, Ghaemi, and others have argued that the BPSM is vague and/or devoid of meaningful scientific

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content (Bolton and Gillett 2019; Ghaemi 2009; McLaren 1998; Van Oudenhove and Cuypers 2014; Weiner 2008). Indeed, McLaren goes so far as to say that, as a scientific model, the BPSM "doesn't exist" (McLaren 2021, 644). These criticisms—which, we will see, are compelling—raise fundamental questions about the BPSM's place in medicine. How can the BPSM be playing the role it is now said to have in healthcare? What does it mean to have an arguably non-existent model guiding whole areas of medical research and practice? Ghaemi is one of the few scholars to have given a sustained answer to these questions (Ghaemi 2010). He argues that, in practice, "the biopsychosocial approach" often devolves into unprincipled eclecticism. The BPSM's all-inclusive nature has left its adherents free to select and mix and match different perspectives—including incompatible dogmatisms—in a haphazard way.

This article offers a different critical argument about the BPSM's impact. The main thesis I advance can be summarized as follows: As some scholars have attempted to use the BPSM as a guiding framework for medical research, they have inadvertently introduced a general explanatory gap into their work. There is an expectation that, by "applying the biopsychosocial model," they will be learning and demonstrating new things about disease; yet the BPSM does not actually offer tools for constructing (probabilistically) true knowledge claims. I contend that this gap is often bridged in practice with certain forms of specious argumentation. These arguments have a common form. They purport to offer scientific conclusions about disease. It might be claimed, for example, that BPSM-based research has established that temporomandibular disorder is a disease caused by various specific biological and psychosocial factors. When we examine such claims closely, however, we find that they lack compelling scientific bases and rest heavily on question-begging arguments, appeals to the BPSM's authority, and other fallacious rhetorical maneuvers. These maneuvers are, we might say, doing the work of the missing BPSM and producing the hoped-for knowledge claims. Using these specious arguments—which I call "wayward" BPSM discourse—researchers have, likely unwittingly, introduced into the health literature many unsubstantiated claims. These include that various ill-defined states of suffering are *diseases* with known etiologies, and that various phenomena correlated with patients' symptoms are the *causes* of those symptoms.

This article will show what wayward BPSM discourse is and why it is a problem. I begin by providing some needed background on the BPSM. Section one offers a brief overview of the model. In section two, I argue, consistent with others (Bolton and Gillett 2019; Ghaemi 2010, 2011; McLaren 1998; Quintner and Cohen 2019; Weiner 2008), that the BPSM is not a scientific or explanatory model. The BPSM cannot be used to distinguish disease from non-disease, define diseases, or identify genuine cause-effect relationships. (This is not to say the BPSM has no value. As I argue, it is still a useful tool for organizing and communicating information about the psychosocial determinants of health). In sections three through five, I develop my main argument. Drawing on Engel's seminal 1977 article and several BPSM illness literatures, I describe the patterns of specious argumentation that constitute wayward discourse. I then highlight the deleterious effects of wayward discourse. Among other things, wayward discourse has sown disruptive conceptual instability in certain lines of medical research and also created a potentially dangerous new vector of medicalization in society. I conclude by arguing that correcting these problems will require imposing conceptual rigor on BPSM discourse.

Overview of the BPSM

Although it has roots in the work of others (Ghaemi 2011), the BPSM was first formally introduced by George Engel in his 1977 article, "The Need for a New Medical Model: A Challenge for Biomedicine." Engel's goal in proposing the BPSM was to remedy certain perceived deficiencies of the biomedical model. Disease, Engel argued, involves important psychological and social factors in addition to biological ones. He noted, for example, that people who experience a mismatch between their social role and cultural resources are more apt to become ill (Engel 1977, 132). The problem with biomedicine is that it typically ignores such psychosocial health determinants because it is "basically mechanistic and reductionistic" in orientation (Engel 1977, 134). The result is an impoverished understanding of health and disease. The BPSM, Engel argued, would overcome the biomedical model's limitations by conceiving of disease as the outcome of interactions among biological, psychological, and social factors.

Figure 1 provides a visual representation of the BPSM that is broadly consistent the verbal and graphical depictions of the model offered in the literature (Bolton and Gillett 2019; Edwards et al. 2016; Engel 1977; Gatchel et al. 2014; Spurgeon 2002; Turk et al. 2011; Vogele 2015; Waddell 1993; Wade and Halligan 2017).

According to the model, illness is a product of dynamic interactions among the sorts of factors listed in Fig. 1, and must be understood as such. Thus a BPSM-based account of chronic pain, for example, might posit that the pain is not a product of bodily damage alone, but of perceptions of that damage modulated by the patient's attitudes or other psychological factors (which might, in turn, be shaped by other psychosocial or biological factors) (Clauw et al. 2019; Ohrbach and Dworkin 2016; Spurgeon 2002). BPSM researchers have also explored how social status and stressors can affect health outcomes (Bolton and Gillett 2019; Engel 1977).

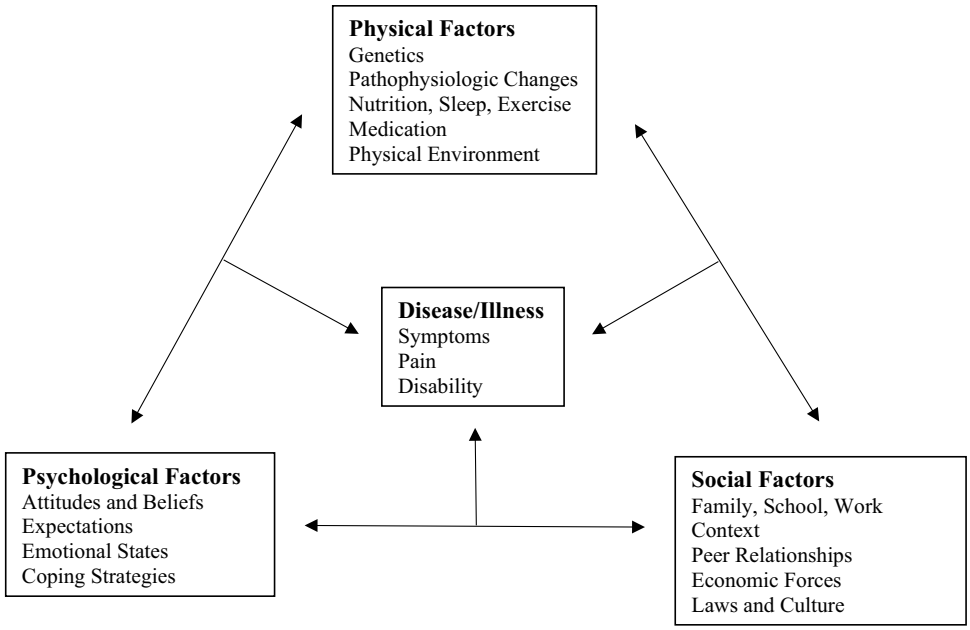

Fig. 1 The biopsychosocial model

Note that a number of more specific versions of the BPSM have been proposed over the years (Bolton and Gillett 2019; Lindau et al. 2003; Wade and Halligan 2017). I will not be dealing with these models in this article. My focus will be on references to, and applications of, the general version of the BPSM described above.

What the BPSM can and cannot do

As McLaren has argued (1998), for the BPSM to be a genuinely scientific model, it would have to go beyond merely positing that illness involves biological, psychological, and social factors. It would have to provide an integrating theory that explained exactly *how* these factors interact to cause illness in practice. The model could do this by, for example, defining its three domains clearly and explaining how social factors of type X cause biological events of type Y, which in turn produce symptoms of type Z, and so on. Engel hoped that general systems theory could be used to build this kind of scientific version of the BPSM (Engel 1977). Yet he never built such a model, and nor has anyone else—although work on this project remains ongoing (Bolton and Gillett 2019; Edwards et al. 2016; Kelly et al. 2014; McLaren 1998, 2021). What the BPSM is, then, is essentially the *general proposition* that illness involves biological, psychological, and social factors.

It is easy to see that the BPSM, as it stands (Fig. 1), offers an exhaustive description of all possible causal relationships surrounding illness. The model's three domains include more or less everything that impacts human life. The BPSM simply posits that when people fall ill, it is because some subset of all possible causal factors somehow interacted to make them ill. The model is thus vague, all-inclusive, and lacks meaningful scientific content (Bolton and Gillett 2019; Brendel 2003; Ghaemi 2010, 2011; McLaren 1998; Weiner 2008). Essentially the BPSM states a truism about illness.

The BPSM's lack of content means it is limited in two important ways. These limitations are perhaps obvious; but they nonetheless need emphasis for reasons that will become clear.

First, the BPSM does not provide criteria by which to distinguish disease from non-disease, or by which to define specific diseases. "Disease," according to most common medical and philosophical definitions, is a bodily disruption or abnormality that has negative consequences for the organism as a whole (see, e.g.: Boorse 1975; Kingma 2014; Murphy 2020; Roberts, *forthcoming*).[1] Engel himself called this our "dominant" concept of disease (1977, 130). (I will use this standard concept of disease in this article, as it has broad scientific, political, and social relevance, and also for normative reasons laid out elsewhere (Roberts, *forthcoming*)). To see why the BPSM cannot be used to distinguish disease from non-disease, consider the following states of suffering: Late-stage HIV infection, stress, people injuring themselves while playing football, anger concerning particular zoning rules, being repulsed by certain foods, sights, or smells. Each of these unpleasant phenomena will undoubtedly be preceded by complex chains of biological, psychological,

[1] And *a disease* would refer to a subset of this phenomenon defined by some characteristic abnormality, agent, or pathophysiological process or mechanism (Roberts, *forthcoming*; Weiner 2008).

and social factors (genetic predispositions, life experiences, cultural customs, legal decisions, etc.). Thus, for each, we could at least partially fill in the fields of the BPSM shown in Fig. 1. Yet completing this exercise would tell us nothing about whether the given state of suffering is a disease (or a potential disease, or best treated as a disease)—in the sense of being a state of suffering caused *primarily* by a known defect or dysfunction of the body. Because all suffering "fits" the BPSM, fitting per se does not establish diseasehood.[2]

It is important to note that what is at stake here is not just our usage of the term "disease" per se. Calling something "a disease" often has powerful effects. It implies that the cause of the problem is more or less known and that it is organic in nature. This, in turn, implies that the problem is not a case of malingering, primarily psychological in nature, or under the patient's direct control, and that, therefore, the patient is entitled to the sick role and its benefits. Calling a problem "a disease" also generally brings it under the jurisdiction of physicians, whose primary expertise is in the body and its defects, thereby encouraging pursuit of characteristically medical modes of treatment and management. These ways of handling human problems can have negative consequences, especially if the problem at hand is actually medically unexplained or a non-disease. For these and other reasons, it is important to avoid applying the appellation "disease" to phenomena that do not fit the definition of that term (Roberts, *forthcoming*). This means that there is also a strong normative case to be made that the BPSM *should not* be used to define disease(s), barring developments that would justify doing so.

Second, the BPSM itself does not provide intellectual tools for establishing causality. Unlike, say, the Henle-Koch postulates or Evans' criteria for causality (Evans 1976), the BPSM does not articulate epistemic principles that would allow researchers to distinguish true cause-effect relationships from spurious correlations. Furthermore, because the BPSM is really an atheoretical model (Brendel 2003; Ghaemi 2011; Skarmeta et al. 2019), it obviously cannot establish which explanations make theoretical sense. Thus, although the BPSM tells us we can list a huge array of factors as disease causes (see Fig. 1), the model itself does not tell us how to determine which factors play a causal role in any given case.

To sum up, the BPSM can appropriately be called a "conceptual framework," but it is not a scientific model or an explanatory model of disease (Bolton and Gillett 2019; Ghaemi 2011; McLaren 1998; Quintner and Cohen 2019). There is nothing in the model itself that would allow us to distinguish disease from non-disease, define specific diseases, or separate genuine cause-effect relationships from spurious correlations.

This is not to say the BPSM has no value, however. As a conceptual framework, it can still serve as a useful tool for organizing and communicating information on the determinants of health and illness. There is now a large body of research indicating that psychosocial factors often play important roles in shaping health outcomes (Bolton and Gillett 2019; Edwards et al. 2016; Gatchel et al. 2014; Vogele 2015). Although the BPSM itself is not a necessary or sufficient tool for uncovering these relationships, it can certainly focus attention on them in several useful ways. Consider the example of lower back pain (LBP). LBP has long been a vexing problem for medicine. In many cases, patients experience pain and disability that cannot be adequately accounted for in terms of anatomical or physiological abnormalities (Weiner 2008). This makes LBP often difficult to manage from a biomedical perspective. In recent decades, however, significant advances in understanding and treating LBP have been made. Working from a BPSM perspective, researchers have found, for example, that fear avoidance, bodily fixation, stress, and other psychosocial factors affect LBP severity; working from such insights, they have developed new treatment modalities (e.g., exercise, therapy, addressing social/workplace factors) that appear to be more effective than biomedical approaches in reducing LBP pain and disability (Gatchel and Turk 2008; Gatchel et al. 2014; Waddell 1993; Weiner 2008). Even Weiner (2008), a spine specialist critical of the BPSM's weaknesses as a scientific model, has acknowledged that it has been helpful in focusing attention on factors relevant to understanding and treating LBP, and now plays a prominent role in spinal care as a result.

These points can be extended beyond LBP. Important psychosocial determinants of health have been identified in many other areas as well, and the BPSM offers a schema for organizing this information and communicating it to medical and nursing students (Bolton and Gillett 2019), as Engel (1977) hoped the BPSM would.[3] In addition, the BPSM can help inform clinical epistemology in more general ways as well. For example, *McWhinney*'s *Textbook of Family Medicine* (McWhinney and Freeman 2009), which draws on the BPSM and similar frameworks, has helped practitioners develop a more holistic approach to medical care. McWhinney and Freeman recognize the value of biomedical approaches to disease. However, they also encourage physicians and other practitioners to move beyond considerations of organic pathology by understanding each

[2] Nor, as will be discussed further below, does the BPSM provide us with a workable alternative (i.e., non-biomedical) definition of disease.

[3] For another point of view, see Ghaemi (2011, 2012), who argues that the BPSM has been less effective as pedagogical tool than is commonly appreciated.

patient as a person whose being is fundamentally social and psychological, in addition to biological. Attending to these aspects of the patient can promote trust, bring to light additional information relevant to patient well-being, and expand opportunities for treatment (McWhinney and Freeman 2009). It is worth noting that, despite general awareness of their importance, psychosocial factors are sometimes deemphasized in everyday medical practice (Weiner 2008; Edwards et al. 2016). As a popular model, the BPSM may be able to help correct this imbalance.

In sum, the BPSM, as a conceptual framework, has expanded the parameters of medical research and practice in some helpful ways. So long as medical actors do not attempt to use BPSM itself for the purposes of defining disease(s) or establishing causal relationships, it can play a useful role in medicine.

Wayward BPSM discourse

Do researchers appreciate the BPSM's limitations? To some extent, the answer is "yes." Spurgeon, for example, writes, "implicitly or explicitly, when we *adopt a biopsychosocial position* we are concerned primarily with the *understanding of illness* rather than the *explanation of disease*" (2002, 601).[4] This view, which has been echoed by other prominent BPSM researchers (Edwards et al. 2016; Gatchel et al. 2014; Gatchel and Turk 2008; Herman 2005; Schwartz 2007; Turk et al. 2011; Wade and Halligan 2017), reflects an accurate assessment of the BPSM's capabilities and limitations. It is not a model that can produce scientific explanations of phenomena. Rather, it is a general perspective one can take to research and treatment. Notably, BPSM-based studies often describe their objects of study specifically as illness, illness behaviors, the experience of disease, disability, and so on. This also suggests some awareness that the BPSM cannot properly be used for defining and explaining *disease*.

Unfortunately, many researchers—including some of those cited above—appear to have become confused about the BPSM's capabilities. For example, Gatchel and Turk (2008, 2832) write: "The data supporting the predictive power of psychosocial variables [in back pain] support and thus validate the biopsychosocial model" and also render concerns about its unfalsifiability "moot." This argument is mistaken. The empirical data "fit" the BPSM *because* it is all-inclusive and unfalsifiable; they do not demonstrate that the BPSM is a valid scientific model.[5] Nonetheless, the authors take this position and conclude that the BPSM offers, vis-à-vis the biomedical model, a more comprehensive "theor[y] of disease and causation" (Gatchel and Turk 2008, 2833). This mistaken idea that the BPSM has been validated and thus has the capacity to define diseases and establish their causes has been echoed by other prominent researchers (Edwards et al. 2016; Engel 1977; Gask 2018; Gatchel et al. 2014; Maltzman 1994; Wade and Halligan 2017).

Adopting this strong position on the BPSM's capabilities tends to place the researcher in an implicit bind. It creates an expectation that one can and will learn new things about disease by putting the BPSM to work; yet the BPSM itself offers no tools for generating new knowledge. I argue that, in practice, researchers have often bridged this gap between capacities and expectations with specious arguments that *seem* to deliver new insights about disease. I refer to these specious arguments, which follow certain common patterns, as "wayward" BPSM discourse.

Wayward BPSM discourse works something like this: A claim will be made that some poorly-understood state of suffering is a *disease* caused by various factors. This claim will be presented as a scientific conclusion that has been reached by the researchers "using" or "applying" the BPSM, or "taking a biopsychosocial perspective" on the ailment in question. On closer inspection, however, we find that what has *actually* happened is this: researchers have referenced or alluded to the BPSM in a *general, verbal way,* and used this discussion as an opportunity to *assert* the existence of a new disease and/or causal relationship by means of *fallacious rhetorical maneuvers*. The key rhetorical maneuvers of wayward BPSM discourse include the following:

- Concept shifting. While arguing for the BPSM's aptness or superiority as a medical model, researchers will sometimes inappropriately blur the conceptual distinction between disease and illness (or syndrome). The practical effect of this maneuver is often to lower the bar for calling problems "diseases," in ways that are unjustified.
- Question begging. Wayward BPSM discourse is characterized by various forms of the begging-the-question fallacy (using premises that contain, or presuppose the truth of, one's conclusion). A common example is declaring that some malady is a "biopsychosocial disease" based on arguments that assume this is the case.
- Appeals to authority. Wayward discourse includes many arguments that boil down to the following: D is a disease caused by factors X, Y, and Z because the BPSM says so.

By employing such maneuvers, researchers have been able to, so to speak, fill the intellectual vacuum created by miscasting the BPSM as an explanatory model, and to

[4] Italics added to quotations for emphasis throughout this article.

[5] Bolton and Gillet (2019, 4, 14) describe the significance of the psychosocial findings on disease correctly: They indicate that "we need" a BPSM, not that we have a valid one already.

construct seemingly-illuminating (but actually spurious) arguments about disease.[6]

The resort to fallacious arguments in wayward discourse is almost certainly unintentional—a result of misunderstanding or carelessness, mixed with excitement about the BPSM's perceived potential. Yet the effects of wayward discourse have been pernicious, nonetheless. I will develop these arguments over the remainder of this article.

Examples of wayward BPSM discourse

In this section I use three case studies to illustrate what wayward BPSM discourse is and how it works. These studies focus on Engel's 1977 article and the BPSM literatures on temporomandibular disorder and irritable bowel syndrome. I use each of these cases to highlight one of the three rhetorical maneuvers discussed above. In this article's Online Appendix, I demonstrate that these rhetorical maneuvers appear in other BPSM literatures as well. I discuss the negative effects of wayward discourse in the next section of this article.

Engel's 1977 article

In his seminal 1977 article, Engel claims to present a new biopsychosocial model that offers better criteria for defining disease and a "blueprint" for medical research and practice (Engel 1977, 131–32, 135). Yet Engel never presents a workable model (McLaren 1998). How, then, does he arrive at the aforementioned claims?

Here I argue that Engel's claims are best seen as expressions of an underlying concept-shifting maneuver. In his article, Engel repeatedly substitutes the terms "disease" and "illness" for one another at critical junctures in his text. He uses this maneuver to expand the boundaries of the concept *disease*. Engel then uses this expanded concept of disease as a premise for his key claims. He uses it to imply—without ever stating clearly—new "biopsychosocial" criteria for defining disease, and names two new diseases in the process. Engel also argues that a new "biopsychosocial" medical model is needed to handle his expanded concept of disease. This is essentially the extent to which Engel articulates the BPSM: As put forth in his article, "the biopsychosocial model" is not a worked-out model, but rather Engel's name for the thing that would, in principle, be suitable for the study of disease as he defines it. In this subsection, I show how Engel's key claims rest on concept-shifting arguments, and explain why those arguments are faulty.

Let us begin by considering Engel's discussion of schizophrenia, which occupies a prominent place in his article. Engel wants to argue that schizophrenia is a medical disease—that is, a problem falling under medicine's purview—and that, if we carefully consider this disease's properties (along with those of several other ailments) we will see that medicine ought to embrace his BPSM.

The claim that schizophrenia is a medical disease faces a hurdle, which Engel acknowledges. Whereas diseases are generally defined in terms of characteristic bodily dysfunctions ("specific pathogenesis and pathology"), schizophrenia is defined by "psychological… abnormalities" (Engel 1977, 131), and thus would not currently qualify as a disease by normal biomedical standards.[7] Why, then, is schizophrenia to be regarded as a medical disease? The way Engel arrives at his answer to this question is revealing, for it exemplifies the core concept-shifting maneuver at work in his article.

Engel does not formally present a new definition of disease and show that it is satisfied by schizophrenia. Instead, Engel's arguments work by forging an equivalence between schizophrenia and an ailment we already take for granted as a medical disease: diabetes mellitus. Engel notes that diabetes is well described in reductionist/biochemical terms; thus, there is little doubt it qualifies as a medical disease. Indeed, Engel introduces diabetes as a "paradigm of somatic disease" (Engel 1977, 131). However, Engel then changes his terminology in a subtle yet consequential way. He equates *diabetes itself* with "illness" and also calls it "a human experience" (Engel 1977, 131–32). Engel then argues that, as *an illness* and *human experience*, diabetes is shaped by psychosocial as well as biological factors. For example, psychosocial factors may affect how patients interpret their diabetes symptoms, thus altering their illness experience (Engel 1977, 132). Schizophrenia, Engel argues, is no different. It is also an illness and human experience that is shaped by psychological and social factors. As for the missing/unknown biological component in schizophrenia, Engel simply "mak[es] the assumption that a specific biochemical abnormality[…] exists in schizophrenia," while also suggesting that we do not necessarily need to emphasize biological factors when discussing *illness* (Engel 1977, 131–32).

After describing "the reality of diabetes and schizophrenia as human experiences" – by listing various known and conjectured biological, psychological, and social factors involved in diabetes and schizophrenia *qua* illnesses – Engel concludes:

[6] To be sure, researchers also present legitimate scientific arguments validated through other means under the heading of "applying the BPSM." These uses of the BPSM are simply not the focus of this article.

[7] Engel calls schizophrenia a "mental disease," but tacitly acknowledges it does not meet the criteria for disease (i.e., "somatic disease") used in medicine.

> This list surely is not complete but it should suffice to document that diabetes mellitus and schizophrenia…. are entirely analogous and… appropriately conceptualized within the framework of a medical model of disease. (Engel 1977, 131)

So, having shifted to a language of *illness* and *human experience* to frame schizophrenia and diabetes as equivalent, Engel then travels back in the other direction. He implicitly argues that, since diabetes and schizophrenia are "entirely analogous," then schizophrenia must be a *medical disease*, since that is what diabetes is. Insofar as Engel makes a case for *why* schizophrenia is a disease falling under medicine's purview, it depends on this underlying disease-to-illness-to-disease concept shift.[8]

Engel later makes a very similar argument with respect to grief. Grief, he argues, though it involves no serious bodily defect, can qualify as a disease partly because "as with classic diseases, ordinary grief constitutes a discrete syndrome with a relatively predictable symptomatology which includes, incidentally, both bodily and psychological disturbances" (Engel 1977, 133). Here we have another concept-shifting argument: Engel starts with "classic diseases" and then redescribes them as "syndromes" in order to expand the boundaries of "disease" to include grief. (Engel actually offers a version of this "syndrome" concept-shifting maneuver in connection with schizophrenia as well[9]).

With little in the way of additional clarification, Engel then refers to "the proposed biopsychosocial concept of disease" (Engel 1977, 134), as though one had been presented. Thus, Engel does not explicitly articulate and defend a new biopsychosocial definition of disease. At best he implies one. Using concept-shifting arguments, he expands the boundaries of the concept "disease" and takes this as an opportunity to apply the appellation "disease" to new ailments. Insofar as Engel implies a new definition of disease, it is something like the following: A disease is a "symptom cluster" precipitated by the "complex interaction" of known or conjectured biological, psychological, and social factors (Engel 1977, 131, 133).[10]

The ultimate purpose of Engel's discussions of schizophrenia, diabetes, and grief is to make a case for his BPSM. After arguing that schizophrenia is a disease that belongs in a medical frame, he adds: "But the existing biomedical model does not suffice. To provide a basis for understanding the determinants of disease [and devising adequate treatments]… requires a biopsychosocial model" (Engel 1977, 131–32). That is, since disease, according to Engel, is caused/constituted by psychosocial factors in addition to biological ones, it can only be adequately understood with "a biopsychosocial model" (Engel 1977, 132–34). Unfortunately, Engel never explains what the BPSM is or how it could account for the psychosocial aspects of schizophrenia or grief. He only says that it "would" (Engel 1977, 133). Thus, although Engel later writes of "the proposed biopsychosocial model" (Engel 1977, 134–35), he does not, in fact, propose a model. "The biopsychosocial model" is mostly a placeholder. It is Engel's name for the thing that would, hypothetically, explain the version of "disease" he constructs by conflating disease with illness.

In sum, Engel's key claims in his article stem, in one way or another, from an underlying disease-illness concept-shifting maneuver. As Engel notes, several of his key conclusions hinge on "obliging ourselves" to think of diabetes and schizophrenia "in exactly the same terms" (Engel 1977, 131), and this is accomplished by running *disease* into *illness*. Indeed, the thesis statement he offers at the opening and close of his main argument bears witness to this strategy: "The dominant model of *disease* today is biomedical, and it leaves no room within its framework for the social, psychological, and behavioral dimensions of *illness*" (Engel 1977, 130, 135). Engel's arguments for the superiority of the BPSM over the biomedical model work by substituting *illness* for *disease*.

The concept-shifting arguments that Engel employs while advancing his key claims are fundamentally flawed because, as Engel himself acknowledges (Engel 1977, 130), "disease" for our society generally means something like objectively-verifiable disruption of the body, whereas "illness" refers to subjective malaise and impairment of the person. Disease and illness (and human experience, syndrome,[11] etc.) are *not*

[8] Had Engel stayed on the terrain of disease as commonly understood, he would not have been able to define schizophrenia as a medical disease (because it lacks a known defining biological abnormality). Likewise, had Engel stayed on the subject of illness, he would only have been able to establish that diabetes and schizophrenia are alike as human experiences, and not that the latter is a medical disease.

[9] Referencing the work of Kety, Engel argues that both schizophrenia and diabetes belong under "the medical model" because "both are symptom clusters or syndromes," and share certain broad similarities *qua* syndromes (Engel 1977, 131).

[10] Engel also appears to argue that a person's not knowing why they are suffering or what to do about it is a necessary condition for classifying that suffering a disease (Engel 1977, 133). This seems clearly wrong. For example, if I get food poisoning or catch a cold, then I may be convinced I know why I am suffering and what to do about it. Yet this does mean that E. coli and rhinovirus infections are not diseases.

[11] To be sure, diseases can *involve* or *produce* syndromes. However, this is not what Engel argues. He argues that "diseases" *are* "syndrome[s]" (Engel 1977, 131, 133). This argument seems clearly wrong. Disease labels are generally supposed to refer (explicitly or implicitly) to causes rather than symptoms or syndromes. Thus physicians say that one patient has rotavirus, another norovirus, another cholera, etc., and not that all have diarrhea-vomiting disease. Moreover, the same disease can sometimes produce quite variable patterns of symptoms. Physicians do not regard every distinctive manifestation

the same thing. This means that Engel's core argument is a *non sequitur*. The standard biomedical model is a model of *disease*. The fact that it cannot explain all aspects of *illness* proves nothing in particular.

Moreover, Engel fails to recognize that redefining disease as illness imposes an enormous burden on him, which he fails to meet. Disease so-defined—essentially, all human suffering involving known or presumptive biological, psychological, and social factors—is clearly a vast phenomenon. It would arguably fall within the purview of *all* the physical and social sciences, including biology, chemistry, psychology, sociology, economics, and so on. Engel tacitly acknowledges this when he writes that the "psychobiological unity of man requires that the physician accept the responsibility to evaluate *whatever problems* the patient presents" and that "the physician's basic professional knowledge and skills must span the social, psychological, and biological" (Engel 1977, 133). But from these points it follows that, if Engel wanted rightfully to claim the phenomenon of *illness* for medicine (relabeled as "medical disease"), he would have to show that he had truly produced a new, expansive medical science capable of handling it—i.e., one built on the sort of integrative theory discussed previously. Yet he does not do that. Instead, Engel appears to proceed by folding "illness" back under "disease" and taking it for granted that "diseases" belong to medicine, and by this means brings ailments like schizophrenia and grief into medicine's ambit—while saying, essentially, that he *hopes* to build a medicine capable of handling them in the future.

In the end, then, Engel's arguments about the nature of disease and putative benefits of the BPSM seem uncompelling. They rest on unjustified conceptual maneuvers. They also, if accepted, would assign a potentially vast portion of human suffering to medicine, but without improving medicine's ability to treat that suffering.

In addition to yielding a problematically expansive definition of disease, Engel's concept-shifting maneuvers also open the door to serious problems in causal inference-making. For example, Engel argues at one point that, in schizophrenia, "conditions of life and living… [and] psychophysiologic responses to life change may[12] interact with existing somatic factors" to shape the onset and severity of "the manifest disease" (Engel 1977, 132). This argument has significant flaws. The presumptive somatic and physiologic factors in schizophrenia are unknown according to Engel, and "life" is an all-encompassing category. A sound causal explanation cannot invoke unknown/conjectured factors and all-inclusive categories. However, since Engel makes these claims while still in the epistemically-uncharted territory of "illness" and "human experience," there is nothing internal to the discussion itself that clearly rules them out. Engel's concept-shifting maneuvers thus create a discursive space in which there appear to be few checks on the causal claims one can make about disease and illness.

Despite their flaws, Engel's concept-shifting arguments have become a part of the wider BPSM discourse. For example, as discussed in this article's online Appendix, Maltzman argues that, due in part to "developments in biopsychosocial medicine," a disease can be defined as a syndrome or cluster of biological and psychosocial problems; on this basis, "alcoholism is a disease" (Maltzman 1994, 13–15). The Appendix's discussions of alcoholism, chronic pain, and chronic fatigue syndrome provide further examples of BPSM researchers using concept-shifting arguments to frame these maladies as diseases or disease equivalents. Echoing Engel, they also advance questionable causal claims in the process.

Temporomandibular disorder(s)

This subsection focuses on temporomandibular disorder (TMD). I argue that TMD has become the subject of unjustified claims and that these claims are at least partly products of the question-begging strand of wayward BPSM discourse. To keep the detail presented to a minimum, I have provided a full version of the TMD case study in the online Appendix, and offered an abridged version here.

TMD is an illness construct that was first formally defined in 1992 (Dworkin and LeResche 1992). TMD, by definition, refers to a pool of diverse jaw-related signs and symptoms. These include muscular pain and tenderness, clicking in the jaw joint, reduced jaw mobility, and osteoarthritis. An individual manifesting one or more of these problems *ipso facto* qualifies for a diagnosis of TMD. The way TMD has been depicted in the literature is somewhat confusing. TMD is often described as a "disease" and a "disorder," and treated as though it were one: "TMD" is said, for example, to "cause," and "manifest" in, patients' jaw symptoms, which are also sometimes called "phenotypes" of TMD (Li and Leung 2021, 459; Ohrbach and Dworkin 2016, 1093–94, Slade et al. 2016, 1091). However, TMD, properly described, is an unvalidated research construct. We know that TMD has not been validated in part because there are proposals on the table to radically redefine the TMD construct (Ohrbach 2021; Ohrbach and Dworkin 2016; Schiffman et al. 2014), which would not be the case if researchers were satisfied it was valid. Thus the "disease" depictions of "TMD" found in the literature appear to be problematic. As an unvalidated construct

Footnote 11 (continued)

of, say, tuberculosis or COVID 19 as a separate disease that gets its own label.

[12] The context makes it clear that, by "may," Engel means "can" or "do in fact sometimes"; he is not stating a mere possibility here, in other words.

defined by mandible symptoms, TMD cannot cause or explain those symptoms. How, then, can researchers claim that TMD is a *disease* that causes/explains patients' suffering?

I argue that the claims surrounding TMD appear to be, at least in part, products of a loop of question-begging argumentation that has become common in the literature. The loop generally looks something like this: During the first step, researchers will invoke the BPSM to *define* or *construct* TMD as a "complex disease"—that is, one caused/constituted by a "complex interaction" of various biological and psychosocial factors. (The precise nature of the "complex interaction" is generally not specified). During the second step, the previous step is forgotten. The idea that TMD is a "complex disease" is now treated as though it were a fact of nature being *revealed by* ongoing applications of the BPSM. Sometimes, researchers will further claim that the "complex" nature of TMD validates a BPSM-based approach; they will then invoke the BPSM to affirm claims made about TMD previously, as well as to advance new claims (at which point one is back at the start of the loop). These sorts of arguments beg the question because the conclusions they present are, in one way or another, assumptions in disguise. Partly by deploying such arguments, researchers have *reified* TMD as an objectively-existing disease with its own causes and effects, in the absence of evidence to support such claims.

We can see a small but nonetheless significant example of the question-begging loop in a recent article by Ohrbach, a prominent TMD researcher. In that article, Ohrbach introduces TMD as a "complex index disease" and writes that "the biopsychosocial model–based [TMD diagnostic system]… was perhaps the first diagnostic system to *formally recognize* TMDs as a complex disease not limited to the masticatory system" (Ohrbach 2021, 89). This account is problematic. As I explain in more detail in the Appendix and below, researchers have, if anything, invoked the BPSM to *define* TMD as a "complex disease" caused/constituted by diverse elements. In the above-quoted passage, however, Ohrbach implies that researchers instead independently discovered this "complex disease" and then merely acknowledged its existence with the TMD diagnostic system. Although it does not offer an explicit argument, the above-quoted statement subtly begs the question with respect to the status of TMD. What is presented as a conclusion or inference of sorts—namely, that TMD is a complex biopsychosocial disorder – is really the foundational assumption of TMD research (Dworkin and LeResche 1992; Ohrbach and Dworkin 2016, 1095), mischaracterized as a discovery or inference. In other words, Ohrbach's statement appears to present an assumption as a fact. Note its effect: The statement implies that "TMD" is a preexisting disease rather than a construct.

During the mid-2000's the U.S. National Institutes of Health (NIH) funded a major TMD study known as "OPPERA." The OPPERA study has been highly significant in the field of TMD research. It is referenced frequently in the literature, and has provided the data underlying many claims made about TMD and its causes. In several descriptions of the OPPERA project offered by field leaders, we find additional question-begging transformations of TMD.

Consider a highly-cited article on OPPERA written by several of the project's key researchers (Slade et al. 2016).

In their article, Slade et al. explain how researchers went about the OPPERA study and describe some of its key findings. Since this information will help us understand the claims made about OPPERA, I will briefly summarize it in this paragraph and the next. According to Slade et al., the NIH "funding opportunity was effectively a rallying call to apply the full expanse of the biopsychosocial model (Engel 1977) to an epidemiologic study of painful TMD" (Slade et al. 2016, 1085). As described by the authors, this meant researchers started from the "premise" that "TMD is a complex disorder resulting from an interplay of causes from multiple genetic and environmental domains" (Slade et al. 2016, 1091). Consistent with this conception of the problem, the researchers involved in the OPPERA project opted to "mov[e] beyond prevailing biomechanical explanations of TMD" and proceeded by analyzing various types of patient and environmental data for "putative [TMD] risk factors" and "vulnerability alleles" (Slade et al. 2016, 1085).

Importantly, as Slade et al. note, the OPPERA study used a new definition of TMD. Researchers defined "TMD" as jaw-related pain occurring more than four days per month (Bair et al. 2013). After evaluating hundreds of potential correlates and antecedents of such jaw pain, researchers identified a number of statistically significant associations. They found, for example, that jaw pain onset was associated with various other nonspecific symptoms (especially those listed on the somatization subscale of the widely-used SCL-90R), several indicators of general physical and psychosocial well-being, and also several genetic markers (Bair et al. 2013; Slade et al. 2016). These associations varied in strength. Another finding was that stress was associated with jaw pain onset in patients with a particular genetic marker, but not others (Slade et al. 2016).

Now consider how Slade et al. describe the significance of the OPPERA findings: "The decade of research discoveries summarized above endorses the premise [of the OPPERA study] namely, that TMD is a complex disorder resulting from an interplay of causes from multiple genetic and environmental domains" (Slade et al. 2016, 1091). They also refer to the factors identified in the OPPERA project as elements of the "complex etiology" of TMD. These claims seem unwarranted. Strictly speaking, the OPPERA results show only that some patients with jaw pain are more likely

than controls to have certain genetic variants and to experience various nonspecific pains and psychosocial symptoms before jaw pain onset. These are certainly interesting findings worth exploring further. However, they do not clearly establish that patients have a particular *"complex disorder" existing over and above their jaw pain, or that the aforementioned factors are etiological elements of this disorder.*[13] One reaches this conclusion only by *assuming* the factors identified in the OPPERA study are indeed causal/constitutive elements of a disorder called "TMD." But this essentially involves reading the conclusion into the data.

Slade et al.'s arguments thus appear to beg the question. The evidence they adduce to "endorse the premise" of the OPPERA study—i.e., that TMD is a complex disorder with a particular etiology—appears to have been produced by *superimposing that premise on the empirical data*, in the context of "apply[ing] the full expanse of the biopsychosocial model" to jaw pain.[14] Again, note the effect of the question-begging arguments: They imply "TMD" is a confirmed disease with a known etiology.

In a highly-cited review article, Ohrbach and Dworkin (2016) also appear to place the OPPERA findings in a loop of question-begging argumentation. For example, they write:

> Findings from OPPERA and other published studies have supported identification of TMDs as a complex disorder within a biopsychosocial illness model, confirming that for almost all cases, TMDs are not a condition localized to pathology in orofacial structures. (Ohrbach and Dworkin 2016, 1097)

Here we can see at least three question-begging arguments. First, the authors claim that the BPSM was used to "identify" TMD as a "complex disorder," when the BPSM was actually used to define it as such. The authors' claim portrays an assumption as a demonstrated fact. Second, the authors claim that the OPPERA findings support the proposition that TMD is a "complex disorder." However, as discussed, this argument only works if we read the proposition into the empirical findings. The argument thus masks a hidden question-begging maneuver. Third, the authors argue that the apparent resonance between the OPPERA findings and the biopsychosocial approach to jaw pain "confirm[s]" that TMDs have a non-local etiology. However, this claim also begs the question in that the evidence supports the conclusion only if the conclusion is read into the evidence.[15] As shown in the Appendix, Ohrbach and Dworkin (2016) extend this loop of question-begging argumentation further in their article, and the loop appears in other TMD articles as well.

In sum, we can see the question-begging variety of wayward BPSM discourse—and its power—at work in the TMD literature. While "applying the biopsychosocial model" to jaw symptoms, researchers have used question-begging maneuvers to *define* TMD as a "complex disease" caused by a vast web of biological and psychosocial factors, and then represented this construction as a fact revealed through empirical research. This reification of TMD helps explain why it seems plausible to say that "TMD," despite never having been properly validated, is a *disease* that causes the symptoms by which it is actually defined.

For a non-TMD-related example of question-begging argumentation, see the discussion of chronic pain in the Appendix.

Irritable bowel syndrome

As the BPSM's status has grown, researchers have increasingly begun to define diseases by appealing directly to its authority. The appeal-to-authority maneuver in wayward BPSM discourse sometimes look like this: According to the BPSM, disease "involves" a "complex interaction" of biological, psychological, and social forces; some problem involves just such a "complex interaction"; therefore, that problem is a disease. Typically, however, no explicit argument will be offered. Instead, the author(s) will take the BPSM as authoritative and then assert that some state of ill health is a disease caused by factors X,Y, and Z, using idiomatic terms like "complex interaction," "complex disease," and "biopsychosocial disease" as literary tropes—as though a phrase such as "complex interaction" itself were sufficient for establishing causality.

For an example of the appeal-to-authority argument, consider an article on irritable bowel syndrome (IBS) by Camilleri and Choi (1997). As the authors note, IBS is a diagnosis of exclusion. To be diagnosed with IBS, a patient must report bowel troubles and also show no signs of "organic disease" (Camilleri and Choi 1997, 3, 8, 9, 11). Yet Camilleri and Choi classify IBS itself as "a disease." In fact, they call it "the most common disease diagnosed by gastroenterologists" and say that "it" "affects about 20%

[13] To justify the claims made by Slade et al. in the quoted passage, the OPPERA project would have had to identify a genuine disease or disorder in participants (e.g., a common, characteristic abnormality or pathological process) and then show that the illness correlates identified in the study actually play a meaningful causal role in that disease. Nothing in the relevant literature indicates this occurred (Bair et al. 2013; Slade et al. 2016).

[14] Slade et al.'s arguments may also indicate that the vagueness of the BPSM and "complex disorder" idea effectively grant researchers wide discretion in deciding what observations would be validating of a construct like TMD. I address this aspect of wayward discourse further in the next subsection.

[15] See Appendix for additional details on this point.

of all people at any one time" and "has a large economic impact" (Camilleri and Choi 1997, 3).

Here we have the same problem seen with TMD. Since IBS is a construct defined entirely by patients' symptoms, how can the authors claim it is a *disease* that causes/explains patients' symptoms? Further, since IBS diagnosis requires ruling out "organic disease," what type of "disease" is IBS? The closest Camilleri and Choi appear to come to an explanation is as follows: IBS is "a biopsychosocial disorder in which three major mechanisms interact: psychosocial factors; altered motility; and/or sensory function of the intestine" (1997, 3, 6). (Altered motility and sensory functions are the symptoms used to define IBS, while "psychosocial factors" refers to higher levels of somatization, paranoia, and other psychiatric maladies among patients (Camilleri and Choi 1997, 7)). Camilleri and Choi thus appear to arrive at the "disease" classification by describing patients' signs and symptoms as parts of a causal "interaction" (the nature of which is not defined precisely), then treating that interaction as constitutive of a "biopsychosocial disorder," and then treating biopsychosocial disorder as constitutive of "disease." The authors thus appear to use the terms "interaction" and "biopsychosocial disorder" as tropes by which to frame patients' symptoms and illness correlates as "a disease."

A virtually identical argument about IBS is offered in an article by Sandhu and Paul (2014). After acknowledging that IBS is a diagnosis of exclusion that entails "no serious underlying disease," the authors nonetheless call IBS "a disorder" and state that it is "the commonest *cause* of recurrent abdominal pain[…] in children" (Sandhu and Paul 2014, 613). To construct IBS as a disease with causal power, the authors assert that "IBS can be considered to be a brain-gut disorder possibly due to complex interaction between environmental and hereditary factors" (Sandhu and Paul 2014, 6013). While acknowledging that the etiology of IBS remains in question, the authors name infection, inflammation, genetic factors, allergy, and the symptoms of IBS itself as being among "the complex interplay of biopsychosocial factors considered to be involved in the development of IBS in children" (Sandhu and Paul 2014, 6014, 6017). Thus, again, BPSM terminology and the "complex interaction" trope are used to recast the IBS construct as something like a disease with known causes and effects. For another article making similar arguments about IBS, see: Mach (2004).

These examples (along with those discussed in previous subsections) illustrate that there is typically no specific standard that must be met for a factor to be deemed part of the "complex interaction" that causes/constitutes a "biopsychosocial disease." The specification of the "complex interaction" is often ultimately a discretionary move in which patient characteristics of uncertain significance are assigned etiological roles via the use of causal language. Such moves are, no doubt, enabled by the BPSM's lack of scientific content, which makes it a poor tool for vetting knowledge claims.

The appeal-to-authority argument is very common in wayward discourse. It is often intermingled with the question-begging arguments found in the TMD literature. For example, the articles discussed in the previous section each in some way referenced the BPSM's authority in constructing TMD as a "complex disease" (Ohrbach 2021; Ohrbach and Dworkin 2016; Slade et al. 2016). Examples of the appeal-to-authority argument can also be seen throughout this article's Appendix, including in the discussions of alcoholism, chronic fatigue syndrome, chronic pain, and the numerous ailments listed in the "Other Illnesses" section. The discussion of "gun violence disease" offered in the next section also constitutes a notable use of the appeal-to-authority maneuver.

The deleterious effects of wayward BPSM discourse

As demonstrated in the previous section and Appendix, wayward BPSM discourse creates a space in which ambiguous illness phenomena (i.e., poorly-understood behaviors, symptoms, and experiences) can be transformed into putative "diseases."[16] It also allows this transformation to occur in a relatively unconstrained way. Because wayward discourse is not governed by clear epistemic or theoretical principles, it imposes few restrictions concerning which factors can be regarded as constitutive or causative of a particular disease.

This section argues that the rise of wayward BPSM discourse has had significant negative consequences for medicine and society. In particular, wayward discourse has created certain disease construct dysfunctions that may have helped undermine certain lines of medical research. It has also created a potentially potent vector of medicalization in society.

Research construct dysfunction

When confronted with poorly-understood illnesses, it is common for medical experts to create special research constructs that define those illnesses for research purposes. Such constructs can help seed the knowledge creation process. To play this role properly, though, research constructs must be viewed as tentative and carefully updated in light of subsequent research findings. Updating is crucial, because the

[16] In some cases (e.g., TMD, chronic pain, and violence, discussed below), wayward discourse has played a leading role in the reification of illness constructs as diseases. In other cases (e.g., CFS, IBS, fibromyalgia, and alcoholism), it has played a supporting role.

ultimate goal is *validation*: Reaching a point where the constructs correspond to distinct causal structures (i.e., diseases) that can be studied and targeted with effective treatments. This process of matching construct to disease can be seen, for example, in the history of the AIDS epidemic. In this subsection, I argue that wayward discourse can undermine the construct updating process in two ways.

First, it can make research constructs *difficult to revise*. Consider, for example, the cases of TMD and CFS (discussed in the Online Appendix). "TMD" are "CFS" are research constructs. They are essentially labels that identify pools of unexplained symptoms for further study. After these constructs were originally developed, researchers were (as just discussed) supposed to revise them in light of incoming empirical findings in an attempt to validate them, or, alternatively, abandon the constructs if validation failed (Dworkin and LeResche 1992; Fukuda et al. 1994; Holmes et al. 1988). That is not what has happened, however. Although redefinitions of CFS and TMD have been proposed, both constructs have for decades remained relatively unchanged, in the face of little evidence for their validity (Institute of Medicine 2015; Ohrbach 2021; Ohrbach and Dworkin 2016; Schiffman et al. 2014).

Almost certainly, wayward BPSM discourse has contributed to this inertia. As we have seen, in wayward discourse, the "CFS," "IBS," and "TMD" labels have been equated with *diseases* and advanced as the *causes* patients' symptoms. This framing inherently discourages construct revision. After all, calling IBS or TMD "a disease" implies that the construct has been validated already. This can only reduce the apparent need to revise that construct. In fact, revision may become relatively difficult: Constructs may be revisable, but who can revise *a disease*? Thus it seems reasonable to suppose that the reification of illness constructs seen in the wayward BPSM literature has helped ossify these constructs at least to some degree.

Second, and the preceding points notwithstanding, wayward discourse can also yield *unstable* illness constructs that place research on a fundamentally chaotic path, especially over the longer term. To illustrate this point, let us return to the example of TMD. As noted above and in the Appendix, people meeting the diagnostic criteria for TMD manifest quite varied symptoms and problems (high patient heterogeneity) and also often qualify for other diagnoses (high comorbidity). Because such observations tend to make it less likely that a construct corresponds to a distinct disease, they are normally interpreted as a mark against validity and a sign that a construct may need to be revised. Yet Ohrbach and Dworkin (2016) seem unsure of what to make of comorbidity and heterogeneity in the case of TMD. At times they appear to argue that the diverse problems manifested by patients ("abundant variables," "appreciable variability") mean that the TMD construct[17] is *good*. These findings are said to show that the TMD construct is "accurate" and "a sufficient marker for underlying complexity"—i.e., the "complexity" ascribed to TMD as a "complex disease." Elsewhere, however, the authors appear to adopt the more typical position on heterogeneity and comorbidity. They suggest that the variability observed among patients means that the TMD construct should be modified in some way (perhaps decomposed into more homogenous sub-diagnoses) to allow for more "refined assessment" of patient subgroups (Ohrbach and Dworkin 2016, 1096–97). So, should researchers aggregate disparate presentations to capture the fundamental "complexity" of TMD or disaggregate them to produce groupings that are more scientifically and clinically meaningful (i.e., valid in the normal sense of the term)? The authors appear to take both positions.

This sort of uncertainty is characteristic of the TMD literature in general. Some researchers recommend "lumping" different mandible symptoms into aggregative TMD constructs, or even merging TMD with comorbid disorders; others favor "splitting" TMD into separate constructs; and, even within each position, the various proposals differ significantly in terms of their recommendations and rationales (Ohrbach 2021; Ohrbach and Dworkin 2016; Schiffman et al. 2014; Slade et al. 2016). Here we see how wayward discourse can produce constructs that set research on an unstable path. Because it is unclear what constitutes a "biopsychosocial disease" or the "complex disease" of TMD in the first place, it is not clear what observed heterogeneity and comorbidity mean for the TMD construct. Their meaning is, as Ohrbach (2021, 90) puts it, "within the eyes of the beholder" in TMD research. But if key empirical observations have no clear theoretical significance because one's framework and core concepts are vague, then the viability of one's research program is open to question.

The instability of the TMD construct is actually significantly greater than preceding discussion implies. As described in this article's Appendix, the current TMD diagnostic system includes two axes: Axis I lists the jaw symptoms that define TMD, while Axis II lists various psychosocial problems that are thought to play some role patients' illness states. According to Ohrbach and Dworkin, however, the existing TMD diagnostic system does not fully realize a biopsychosocial approach to mandible symptoms. The current Axis I, they argue, is just "a special case of the 'bio' in biopsychosocial" (Ohrbach and Dworkin 2016, 1098). Thus

[17] The authors are actually here discussing a simplified version of the TMD construct used in the OPPERA studies. This is not a crucial distinction for my purposes.

they have proposed adding a third axis to the TMD diagnostic system, which would include "findings from such diverse biologic considerations as genetics, epigenetics, and neuroscience." Exactly how the new axis would play a role in the definition and diagnosis of TMD is not fully clear. At one point, the authors suggest it would be used to collect information on the mechanisms of TMD as currently defined. At another, they suggest it would be used to redefine TMD from the ground up. And still other proposals are offered in the text (Ohrbach and Dworkin 2016, 1098). To complicate matters further, Ohrbach, Dworkin, and other field leaders have also proposed adding a *fourth* Axis to the TMD diagnostic system. This effort apparently would involve trying to produce yet another set of TMD diagnostic categories through statistical analysis of large pools of "biopsychosocial and molecular" data (Schiffman et al. 2014).

It is difficult to see how these proposed initiatives could add up to a coherent research program since they would prioritize and organize information in quite different ways. The probability that they would turn out to be complementary or converge on the same endpoint seems extremely small. The TMD literature illustrates how wayward discourse can set research on a chaotic path. Wayward discourse has helped cement the idea that there exists a "complex disease" called TMD that can only be adequately studied from a BPSM perspective. And yet the vagueness of the "complex biopsychosocial disease" concept at the center of TMD research has apparently left researchers without a clear sense of what it is they are looking for, or how to find it. The new axis proposals appear to try to pursue all hypotheses on mandible symptoms at once. (Ghaemi (2010) has previously noted the BPSM's tendency towards eclecticism and insufficiently systematized data collection).

This problem of construct instability is not limited to TMD. As discussed in the Appendix, Clauw et al. (2019) have proposed treating chronic pain as a "biopsychosocial disease." Yet how is this disease to be defined given the BPSM's lack of epistemic rules? Clauw et al. define chronic pain disease to include a diverse array of conditions—among them TMD, CFS, fibromyalgia, interstitial cystitis, endometriosis, migraine, low back pain, and rheumatoid arthritis—on the premise that these conditions share, or might share, some common mechanisms. But as Quintner and Cohen (2019) ask, does this mean that the etiologies of, say, endometriosis and rheumatoid arthritis are effectively the same? Should these maladies be lumped together for research and treatment purposes? Arguably not. And indeed Clauw et al. (2019) suggest that at least some conditions included in chronic pain disease should be kept distinct based on etiological and treatment differences. But this raises the question of what the value of the "chronic pain disease" super-category is. When should conditions be aggregated as opposed to disaggregated, and on what principle? Since there are many factors associated with pain conditions, which should define "chronic pain disease"? These questions are not adequately resolved in the text, and it is not clear how they ought to be answered given the vague nature of the BPSM and "biopsychosocial disease" concept.

The problems of construct ossification and instability—both of which are symptoms of wayward discourse's absence of clear theoretical and epistemic principles—matter. When it comes to the sorts of poorly-understood symptoms discussed above, researchers rely heavily on illness constructs to constitute objects of study and direct inquiry (Roberts, *forthcoming*). Thousands of studies have cited the TMD criteria, for example, and they are "the dominant if not required diagnostic system for NIH-funded research applications and most TMD peer-reviewed scientific publications" (Ohrbach and Dworkin 2016, 1096; Skarmeta et al. 2019). The TMD construct is the organizing core of the field of TMD research. Thus when a construct like TMD (or IBS, CFS, chronic pain disease, etc.) becomes *frozen through reification and/or linked to an illness concept such as "biopsychosocial disease" that does not readily support systematic inquiry, it can easily undermine scientific research into patients' symptoms*. These wayward discourse-related problems could help explain the relative lack of progress in explaining and treating the symptoms associated with TMD, IBS, and CFS.

The medicalizing power of wayward discourse

Wayward BPSM discourse is also a potent and potentially dangerous vehicle of medicalization. In particular, it has the capacity to [1] prematurely represent ambiguous states of suffering as organic problems falling under medicine's purview, and [2] expand the domain of "disease" in ways that unjustifiably increase the power of medicine and the state.

As a lead in to point [1], consider the following statement from Frederick Wolfe, a leading rheumatologist who helped define the "fibromyalgia" construct: "Perhaps [selecting] tender points, as the essential criterion [for defining 'fibromyalgia'], was a mistake. By ignoring the central psychosocial and distress features of the syndrome and choosing instead a physical examination item, we allowed FM to be seen as mostly a physical illness. More than that, we removed all traces of the most central features of the illness" (Wolfe 2003, 1671). Wolfe's statement illustrates a broader point: By choosing which information to foreground and which to deemphasize when creating an illness construct, one can represent the underlying problem in potentially arbitrary and misleading ways.

Wayward BPSM discourse's lack of clear epistemic standards makes it prone to this problem. Let us briefly return to the case of TMD. It is well known that TMD patients often exhibit many other nonspecific bodily symptoms and high levels of psychosocial distress (Bair et al. 2013; Li and

Leung 2021; Slade et al. 2016). On the basis of these sorts of observations, some have argued that mandible pain might, in cases, be a primarily psychogenic illness or an aspect of somatization or some other psychological disturbance (Dimitroulis 1998; Kumar and Brennan 2013). However, the TMD diagnostic system in some ways discourages such interpretations. It isolates patients' mandible symptoms and makes them the defining diagnostic features of the disorder "TMD," while relegating various somatization and psychosocial symptoms reported by patients to a secondary axis meant for supplemental information gathering (Dworkin and LeResche 1992, 303, 330). By partitioning patients' symptoms in this way, the TMD construct makes it easier to portray patients as suffering from a distinctive disorder centering on the jaw that causes or coexists with psychological troubles, rather than a primarily psychological problem of which their jaw pain is a symptom. And indeed, we see this line of interpretation being pursued quite often in the TMD literature (see, e.g.: Dworkin and LeResche 1992, 303–4, 330, 332; Kumar and Brennan 2013, 426–7; Li and Leung 2021, 6–7). The point here is not that any one position on jaw pain is right or wrong. The etiology of much TMD jaw pain has yet to be explained definitively. The point is that wayward discourse allows information to be partitioned and prioritized in ways that are at least potentially arbitrary and capable of misleading us about the nature of patients' suffering.

Although wayward discourse could be used to psychologize what are really best understood as organic diseases (Weiner 2008), medicalization appears to be the greater threat. The texts discussed throughout this article often acknowledge that "biopsychosocial diseases" can involve major psychological, moral, or attitudinal elements; that their physical aspects may be unknown, trivial, or secondary to other problems; and that they may best be treated with nonmedical or multidisciplinary approaches (Camilleri and Choi 1997; Gatchel et al. 2014; Maltzman 1994; Sandhu and Paul 2014; Wallace 1990). Notably, however, such acknowledgments rarely seem to lead to the conclusion that the ailments in question are *not* medical diseases, or that they should be relinquished to other epistemic communities for primary study and treatment. Instead, from Engel on, discussions of the "complex" nature of human suffering have shown a remarkable tendency to collapse back into the language of "medical disease." This framing has important consequences. It tends to perpetuate a focus on biological factors (see, especially the discussion of alcoholism in the Appendix) and edge out existential, spiritual, philosophical, depth psychological, and other nonmedical approaches to suffering (Ghaemi 2011).

This suggests we ought to be skeptical of claims that the BPSM is a humanizing or *de*-medicalizing force (see, e.g., Engel (1977) and Gatchel and Turk (2008)). Yes—it does seek to incorporate non-biological factors into accounts of suffering. This, in principle, seems like a welcome development. However, as argued in this article, BPSM-based discourse has also produced an operative concept of disease so vague that potentially any instance of human suffering can be counted a "medical disease."

Giving patients labels that selectively emphasize certain aspects of their suffering and imply diseasehood without due justification is problematic. In addition to having the potential to undermine research in the ways discussed above, it creates ethical problems. Patients have a right to know the true state of medical knowledge on their ailments. If one's malady consists of poorly-understood symptoms, sensations, and behaviors, then one should be told that, and not that one has a "complex biopsychosocial disease." Although some patients may *want* to be told they have a disease, this is no basis for offering such a diagnosis, especially since proffering disease labels can actually increase stigma and worsen patient prognosis (Hadler 1997; Speerforck et al. 2014).

There is another significant problem associated with the medicalizing power of wayward discourse. The word "disease" has powerful social ordering effects. It implies that the problem at hand is one that can only or best be understood by medical experts, and, therefore, that those experts ought to be granted authority over the problem. This authority can extend into the legal, political, and social domains. When, for example, a disease is declared a public health concern, health experts and agencies may be granted expanded powers to regulate commercial activities, constrain the movement of individuals, and shape the policies of public and private organizations in profound ways. This brings us to point [2] mentioned at this section's outset. By loosening the criteria for declaring problems "diseases," wayward discourse can be used to expand the reach and power of medicine and the state in ways that are not necessarily justified.

We can see a relatively transparent attempt to harness this power of wayward discourse in the violence-as-a-disease literature. In recent years, a group of health researchers have been invoking the BPSM to argue that gun violence is "a complex biopsychosocial disease" encompassing a vast amount of human activity, which health professionals should regulate as a form of "disease control and prevention" (Barron et al. 2021, 1–3; Grossman and Choucair 2019; Hargarten et al. 2018, 1024; Kohlbeck and Nelson 2020, 4–5).

For example, several researchers have argued that "fram[ing] gun violence as a biopsychosocial disease" allows us to assert that "the vector of disease [is] the gun itself, as it 'transmits' the agent to the host" (Kohlbeck and Nelson 2020, 3). For Hargarten et al. (2018, 1025), this makes "the gun… a necessary focus of intervention." Health professionals, they argue, should be involved in "specific examination of the gun and its design/safety characteristics" and also given *de jure* or *de facto* regulatory powers of some

kind (Hargarten et al. 2018, 1025–26). Options to be pursued include lowering magazine capacity, "banning" bump stocks, and "requiring background checks on all gun sales."

The biopsychosocial disease of gun violence is said to include far more than just the firearm, however. Other "aspects of the disease" include, literally, "high-risk youth; adults and elderly; […] and the environment." Culture and attitudes can play roles in "'spreading' the risk of the disease" as well. Therefore, it is claimed, these factors must also be "treated from [a] biopsychosocial perspective" (Hargarten et al. 2018, 1025–26).

Consistent with this view, researchers have begun recommending that physicians scan patients' genetic profiles, medical histories, psychological attributes, behaviors, and cultural and familial backgrounds in an attempt to gauge their propensity to commit violence; patients who "screen positive" can be given "appropriate behavioral interventions" to prevent violence *before* it occurs (Barron et al. 2021; Grossman and Choucair 2019, 1641). When a patient is hospitalized for a violent injury and the "perpetrator" is present, the latter can be given behavioral modification therapy to help them avoid "recidivism," thereby helping to "prevent and control" gun violence disease (Grossman and Choucair 2019, 1641; Hargarten et al. 2018, 1025–26) (note the blending of public health and criminal justice discourse). Other options for disease control include controlling firearm access for "at-risk" individuals, sharing hospital data with law enforcement, and creating behavior modification interventions that prevent violence by "addressing biopsychosocial aspects of students' lives" (Barron et al. 2021, 4; Grossman and Choucair 2019, 1641–43; Kohlbeck and Nelson 2020, 4).

Kohlbeck and Nelson carry these lines of argument further. Invoking the BPSM and writings of Paolo Freire, they argue that gun violence disease can be attributed to an underlying "disease of oppression" embedded in "our violent society." "Public health," they write, "has a role to address the disease of oppression" (Kohlbeck and Nelson 2020, 3). Instead of merely providing public education, health professionals should engage directly in the "dismantling of violent structures of power" and in fostering "liberation" (Kohlbeck and Nelson 2020, 4–5). This effort would entail helping to redistribute resources in society to eradicate the perceived root causes of violence and steering public discourse on violence to align with the authors' own views (Kohlbeck and Nelson 2020, 4–5). Thus, Kohlbeck and Nelson would have health professionals working to restructure society and manipulate the parameters of public debate as forms of disease control and prevention.

It is worth noting that the wayward BPSM discourse on gun violence is almost transparently political rather than scientific. The literature in this area does not provide a meaningful definition of "biopsychosocial disease" and then demonstrate that gun violence qualifies. Instead, it uses that construct in an explicitly opportunistic way. The articles on the topic consistently argue that gun violence "*can*" and "*should*" be "*framed* as a biopsychosocial disease" to expand medical jurisdiction over the problem (Barron et al. 2021, 1; Grossman and Choucair 2019, 1640; Hargarten et al. 2018, 1024–26; Kohlbeck and Nelson 2020). "Without this framing," Hargarten et al. warn, "we limit progress… [and] will be limited to education of our patients" (2018, 1025). Despite its almost conspicuously contrived nature, "gun violence disease" is treated as though it were a disease like any other. Medical and health professionals are said to have a right and a responsibility to "prevent and manage gun violence, just as they… prevent and treat other diseases," like HIV infection and tuberculosis (Barron et al. 2021, 2; Hargarten et al. 2018). (These arguments, it is important to note, also rely on the appeal-to-authority maneuver described above).

The "gun violence disease" literature is concerning for several reasons. One problem is that physicians do not possess the epistemic competence needed to treat gun violence disease as it is defined.[18] Their training does not qualify them to redesign society's power structures or to accurately identify and treat personal or cultural propensities for violence. Another problem is that the etiological factors of the "biopsychosocial disease" of violence include people's attitudes, values, customs, thoughts, and volitions. Although people are not entitled to commit violence, they are entitled to a level freedom in thought and action that may result in violence. Efforts to prevent violence must therefore be balanced against the need to respect people's civil liberties and autonomy. What the appropriate balance in this regard is and how it shall be achieved are political questions that deserve public debate. In wayward BPSM discourse, however, people's beliefs, etc., are treated as disease "risk factors" to be altered by medical and public health actors (Barron et al. 2021; Hargarten et al. 2018). Thus we find physicians taking up and exercising criminal justice functions (e.g., identifying "perpetrators" and giving them counseling to "prevent recidivism," sharing data with law enforcement, potentially controlling access to firearms) and harnessing the regulative powers of schools and the state (school-based behavior modification programs, firearm regulation) under the banner of "disease control." The fusing of medical authority and state power seen in BPSM violence interventions is troubling. Along with "national security," "public health" is one of the few imperatives that readily justifies state abrogation of

[18] Barron, Hargarten, and Webb (2021) tacitly acknowledge this when they note that "gun violence disease" would not fit well into existing medical school curricula, and recommend working discussion of the "disease" into classes on medical ethics.

individual rights. Thus, the production of a new and expansive public health problem in the "gun violence disease" discourse has the potential to significantly increase the power of the state, and not just that of the medical field per se.

It is important to note that the wayward BPSM argument on gun violence has been set forth in the leading health policy journal *Health Affairs* (Grossman and Choucair 2019). It also received considerable attention at at least one conference put on by prestigious health institutions (National Academies of Science 2019) and is echoed in the "Social-Ecological Model" of violence used by the U.S. Centers for Disease Control and Prevention (Centers for Disease Control and Prevention 2022). These points suggests that "gun violence disease" is not necessarily a fringe argument, and that its potential to shape medical and political practices should be taken seriously.

The medicalizing dimension of wayward BPSM discourse discussed in this subsection can be seen as a culmination of changes initiated by Engel himself. From one point of view, biomedicine's physical reductionism has certain virtues. It places limits on what can rightfully be called "a disease"; this, in turn, places boundaries on the domain of medicine. Engel explicitly rejected this approach to defining disease and medicine. He wanted medicine to have a vastly expanded role in managing human suffering (Engel 1977, 133). He believed the definition of disease should be modified to *fit* that role. It should be broad and flexible enough to facilitate and justify the treatment of conditions assigned to medicine through political, cultural, and social processes (see especially Engel (1977, 129–30, 132)). Thus we have Engel's very expansive concept of disease, the constitutive elements of which include such broad categories as "social behavior," "alterations… in feelings," and "behavioral aberrations" (Engel 1977, 130). This definition would allow physicians to bring many problems into medicine's ambit as "medical diseases."

Constructing a "gun violence disease" to promote medical intervention into society is therefore quite consistent with Engel's vision. Yet it is also concerning. For here we see a concept of "disease" that, instead of constraining scientific and political forms of authority, becomes a mechanism for their amplification.

Conclusion

In sum, the BPSM can serve as a useful tool for highlighting psychosocial factors important to health outcomes. It is not, however, a valid, authoritative, or superior explanatory model of disease. Treating it as such has created an epistemic void that has produced the wayward form of BPSM discourse described here. Participants in wayward discourse typically suggest they are presenting insights about disease gleaned through applications of the BPSM. Upon closer inspection, however, we find that key claims advanced often rest on flawed arguments and rhetorical maneuvers.

This article has detailed how wayward BPSM discourse has served as a wellspring of questionable claims in medicine. Its participants have argued that various poorly-understood states of suffering and undesirable behaviors are "diseases." These "diseases" are often asserted to be caused by various factors that have no proven etiological significance. In some cases, the "diseases" are said to be caused by hypothetical factors (as in the case of schizophrenia), or to cause themselves (e.g., IBS, TMD). I have also shown that several disease constructs created and reinforced via wayward discourse may have mispresented the nature of patients' suffering and set scientific research on epistemically unstable paths. Furthermore, wayward discourse has created a potentially potent and dangerous vector of medicalization in society.

In highlighting these problems, this study provides further evidence that the sorts of fallacious arguments in medicine noted by Binney (2019) are relatively widespread, consequential, and in need of remediation.

Fixing the problems associated with wayward discourse will likely require reimposing conceptual rigor on BPSM discourse where it has been lost. It is reasonable enough to "take a biopsychosocial perspective" on some illness, or to use the BPSM as a tool for presenting existing research findings. However, the limitations of the BPSM should be kept at the forefront of the discussion. It should be remembered that the standard BPSM, as it stands, offers no tools for generating valid claims about diseases and their causes. It should also be appreciated that there are no such things as "biopsychosocial diseases" or "complex diseases" in the sense implied in wayward discourse. Disease states can indeed involve "complex interactions" of factors. But "complex interactions" of factors per se do not constitute disease, and the "complex interaction" trope ought not be used to construct new diseases. Given that such guidelines for the correct use of the BPSM are at least to some extent acknowledged in the literature already, enforcing them should be possible. Doing so would, however, likely mean some significant scaling back of the claims currently being made within wayward BPSM discourse.

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Declarations

Competing interests The author has no competing interests to declare.

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