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Philosophy, Psychiatry, & Psychology, Volume 31, Number 3, September 2024, pp. 269-284 (Article)

Published by Johns Hopkins University Press



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AN EXTERNALIST
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Article submitted on March 10, 2023
Revision submitted on August 21, 2023
Accepted on September 9, 2023

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ABSTRACT: The biopsychosocial (BPS) model in psychiatry has come under fire for being too vague to be of any practical use in the clinic. For many, its central flaw consists in lack of scientific validity and philosophical coherence: the model never specified how biological, psychological and social factors causally integrate with one another. Recently, advances in the cognitive sciences have made great strides towards meeting this very ‘integration challenge.’ The paper begins by illustrating how enactivist and predictive processing frameworks propose converging accounts of biopsychosocial integration that are far superior to those of previous theories. It argues, however, that the main problem of implementing the BPS model has less to do with integration than with the lack of a social etiology. Psychiatric practice leans heavily towards ‘bio’ and ‘psycho’ approaches, without an equally developed set of explanatory and therapeutic resources for dealing with the ‘social’ dimension of illness. This leaves psychiatry essentially internalist in orientation. As illustrated most poignantly by conditions such as functional neurological disorders, internalism comes with the risks of stigma and the curtailment of therapeutic possibilities. The paper argues that the an-

swer to the failings of the BPS model lies in combining the integration challenge with the development of an ‘externalist psychiatry,’ which casts both causes and treatment of psychiatric illness onto the social environment. The following two papers explore the conditions that might make this idea a reality.

KEYWORDS: Biopsychosocial model, enactivism, predictive processing, social etiology, functional neurological disorders, externalist psychiatry

VARIABLY PRAISED FOR its theoretical soundness or dismissed as practically useless, the biopsychosocial (BPS) model remains highly debated in philosophy of psychiatry. Its main strength, it is widely acknowledged, is to serve as an aide-memoire against the perils of biologism; to remind practitioners of the existence of psychosocial dimensions to illness whose treatment cannot rely on a laser-like focus on the

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The author reports no conflict of interests.

brain. Its weak point is its vagueness. According to critics, its theoretical audacity has paled in comparison to its clinical utility. The model appealed to three dimensions of health without specifying the conditions whereby one dimension could be more significant than another in particular disorders. It also never clarified how exactly bio, psycho, and social forces causally interact with one another. Beyond its call for holistic care, in short, it has given practitioners little guidance to work with in the clinic. Still, its relevance holds on because the question of how bio, psycho and social forces integrate has since been taken up by the cognitive sciences. Popular frameworks in embodied cognition—enactivism and predictive processing (PP) above all—have in their own ways revolutionized how we think about biopsychosocial causal integration, leaving open the question of what clinical implications should follow.

After taking stock of the current state of the BPS model, this paper discusses the enactivist and PP contribution to the ‘integration problem.’ It lists their merits and their points of convergence. It argues, though, that tackling this integration problem will not be sufficient for realizing the BPS model in clinical practice. This is because these theories, along with much of the literature on the model, leave undefined the ontological domain of the ‘social.’ They tend to collapse the ‘social’ into the ‘psychosocial’ and gloss over, in so doing, a crucial distinction between patient-centered (bio and psycho) and externalist (social) approaches to illness. Taking the experience of patients with functional neurological disorders as a case in point, the paper highlights the drawbacks of relying solely on patient-centered frameworks, which come in the form of stigma and curtailment of therapeutic possibilities. Finally, introducing an idea that will be fully outlined in the following two papers, it argues that only by developing an ‘externalist psychiatry’—a psychiatry that casts both causes and treatment of psychiatric illness into the social world—will the BPS model live up to its original promise.

THE CURRENT STATE OF THE BPS MODEL

Contemporary philosophers of psychiatry largely agree on the indefensibility of the biomedical model. The biomedical model views symptoms of illness as the direct consequence of physiological disruption, and recovery the direct consequence of physiological reparation. Phenomena such as ‘nocebo’ and ‘placebo’ effects, along with a wider gamut of social effects on health, manifestly invalidate the model’s premises: they show that, at least at first view, the causal arrow can go from mind to body and not simply the other way around. The psychosocial context around the patient can play a significant role in the rise and exacerbation of illness. We know that the prevalence of disorders with psychosocial origins is high, and that patients suffering from such disorders bring formidable economic, practical, and ethical challenges to primary care (Van den Bergh et al., 2017). Clinicians who adhere strictly to a biomedical model are ill-equipped to explain these conditions to patients, let alone treat them.¹

The BPS model arose in the late 1970s to offset these limitations. George Engel, the psychiatrist who devised it, insisted that illnesses are multifactorial, with several causes, and that the reductionism in medicine and psychiatry should give way to a more comprehensive approach (Engel, 1977, 1981). He was drawn to systems theory because a focus on systemic interactions at the information level could dispel metaphysical distinctions between ‘bio,’ ‘psycho,’ and ‘social’ domains. If every health condition emerges from a combination of all these three classes of factors, he argued, it should be treated accordingly. Farewell, then, to Kraepelian biologism as well as to Freudian psychoanalysis or other systems that are exclusively biological or exclusively psychodynamic in nature. The new biopsychosocial psychiatry would attend to bio, psycho, and social aspects all the same. The most important implication of this framework was a greater emphasis on the therapeutic alliance, which the BPS model shared with the patient-centered care and humanistic medicine movements growing at the time. It has been a duty

of clinicians working under this model to attend to the whole person: to consider how the disorder affects and is affected by the patient's agency, values, and achievement of personal goals. Engel hoped to bring psychological and social dimensions of illness within the scope of scientific inquiry, on par with the biological dimension.²

Fast forward to the present day and the BPS model is everywhere taught, having become the conceptual status quo in psychiatry. It has convinced large sections of the profession that there are several contextual factors at play in psychiatric disorders and that an exclusive focus on the brain is unlikely to be adequate. Yet, with psychiatry everywhere said to be in crisis (Di Nicola & Stoyanov, 2021), and with an ever-mounting philosophical literature dealing with this crisis, it is uncontroversial to say that the BPS model has failed to deliver. Many critiques have been leveled at it. Its claims might be right, most critics say, but insofar as they lack specific guidance for clinicians beyond guarding them against biologism, they are ultimately trivial. According to the most prominent of these critiques, the BPS model remains difficult to implement because it suffers from an 'integration problem' (Bolton & Gillett, 2019; Coninx & Stilwell, 2023; De Haan, 2020; Farre & Rapley, 2017; Gallagher, 2022; Ghaemi, 2010; Van Oudenhove & Cuypers, 2014). Beyond the groundwork provided by systems theory, the model has never offered an account of how 'bio,' 'psycho,' and 'social' factors causally interact with one another. Its scientific status thus remains dubious until it is refurbished with a set of theoretical constructs that yield a clearer picture of biopsychosocial causation.

As some of the same critics point out, the integration problem has gradually turned into a productive challenge. Over the past few decades, the cognitive sciences have made great strides in unraveling the causal relations between bio, psycho, and social forces affecting subjective experience. The main frameworks in contemporary embodied cognition—enactivism and PP—could well be seen as attempts to answer that very question of biopsychosocial causal integration. What's more, these theories take seriously the causal significance of the social environment, thereby

aligning with the anti-reductionist spirit of the BPS model (this is particularly so in the case of enactivism) (Aftab & Nielsen, 2021; Cormack et al., 2023). In what follows, I discuss these two theories, starting with the enactive one. I confine my discussion to their account of causality, before turning to their limitations, and, eventually, to my own suggestion that meeting the 'integration challenge,' although fundamental, has very little bearing on the practical challenge of making the BPS model work in the clinic.

THE ENACTIVIST MAKEOVER: CASTING ILLNESS IN CAUSAL TERMS

Enactivism is a non-representationalist framework for the study of the mind. It is premised on the idea that 'cognition' should be understood as 'sense-making,' namely, the embodied and embedded interaction of organisms with the environment, where the environment holds meaning for the organism depending on the latter's specific physiological and organizational structure (Thompson, 2007; Varela et al., 1991). For enactivists, consciousness emerges out of this interaction, rather than in brain activity alone. The enactivist take on biopsychosocial causation is to argue, emphatically, that 'mind' and 'body' are not two distinct ontological categories, but two aspects of this 'sense-making' process. Because 'mind' and 'body' are two sides of the same coin, it is deceptive to say that psychology affects physiology or vice versa. Where enactivists make an important distinction between the 'physiological' and the 'psychosocial' is at the level of *causes* to the whole organism.

The enactivist account of causality goes by the name of 'circular causality' (Fuchs, 2018, 2021), which differs from 'linear causality' because any effect on the system is assumed to depend on, and affect in turn, the overall global organization of that system. This account owes much to previous theories in systems biology and psychosomatics but finds, in my view, its clearest articulation in recent enactivist literature (De Haan, 2020, 2021). Proponents argue that physiological causes should be understood as 'local-to-global' (or 'upward') causes, whose effect on consciousness depends on the overall state the organism finds themselves

in. For example, the role played by MDMA in heightening the activity of serotonin and boosting the feeling of ecstasy is of such local-to-global type, but where the effects fundamentally depend on the state of the person as a whole, on their history and social setting. By contrast, the role that, say, winning a trophy might have in heightening the activity of serotonin and boosting the feeling of ecstasy is a global-to-local ('downward') cause, where the global sense-making conditions of the organism affects lower-level physiological structures. Psychosocial causes are of this type. Importantly, physiological and psychosocial causes are always engaged in a relation of circular causality, where, depending on the case, one cause might take the leading role in affecting experience, but cannot be taken in isolation from the overall organism-world system.

Transposed onto the field of health and illness, the enactivist account of circular causality yields a clearer picture of how 'bio,' 'psycho,' and 'social' dimensions integrate. In illnesses with a clear organic pathophysiology like a tumor, the leading cause of experience is of the upward type, though the experience of illness (e.g., whether we experience cancer with dread or acceptance) fundamentally depends on our global sense-making conditions (Stilwell & Harman, 2019). By contrast, psychiatric disorders have primarily downward causes, although, even here, there might be upward influences (e.g., genetic polymorphisms, deeply seated bodily dispositions) that affect the onset and character of the disorder. Events like traumas can have a downward effect that can lead to lasting neurological changes. Once the disorder develops, diagnostic labels applied by medical personnel and social discourse also have downward regulatory influence on illness experience.

Similarly, drugs have an 'upward' influence that affects neurological structures and therefore experience (at least beyond a certain threshold, which is determined by the system as a whole), but it would be wrong to say that a drug treats a disorder in isolation. The enactivist account here finds support in studies showing that patients who are administered drugs unknowingly (e.g., through a computer-programed infusion pump) experience a much weaker relief than when they

receive the same drug consciously in full view (Benedetti et al., 2011). What misleadingly passes for the 'placebo effect' is the downward effect of sense-making in the therapeutic context (Arandia & Di Paolo, 2021; Hutchinson & Moerman, 2018; Ongaro & Ward, 2017), except for when it is elicited through conditioning, in which case it is partly the result of 'upward' forces. Psychotherapy, which in enactivist language is an example of 'participatory sense-making' (Fuchs & De Jaegher, 2009; Nielsen, 2023), should be seen as exerting a downward effect, one that with time can seep deep enough to lead to lasting neurological changes. As enactivists point out, changes in one's social environment—for example, finding meaningful employment—also have a downward, global-to-local effect (De Haan, 2020).

Downward and upward causes on a health condition can temporally crisscross and overlay each other. The enactivist picture of causality accords here with empirical findings in psychosomatics and social epidemiology (McEwen, 2012) and with cultural-ecosocial systems theories (Gómez-Carrillo & Kirmayer, 2023). For instance, diabetes might have an ultimate downward cause (eating habits) but a proximate upward cause (insulin deficiency) and, accordingly, a proximate upward-type treatment in the form of insulin injections (psychotherapy, which exerts a downward regulatory effect, is not very effective here). Conditions such as anxiety or depression tend to have mostly proximate downward causes and call for similar treatment, that is, non-pharmacological therapy (even when pharmacological, the therapy is mostly effective via 'placebo effects,' namely downward causes). Evidence shows that psychotherapy and therapeutic rituals are most effective on conditions that have a largely proximate downward cause, whereas medication is most effective on conditions with a largely proximate upward cause, while complex, chronic conditions fare best with a combination of both types of therapy (Van der Kolk, 2015). An enactivist-based BPS model thus recognizes that some conditions might be more 'biological' in origin and others more 'psychosocial'—thereby superseding the critique that the traditional BPS model treats 'bio,' 'psycho,' and 'social' aspects indiscriminately (Ghaemi, 2010)

—but it does so through a conceptual overhauling of the nature of causation that radically upgrades Engel’s framework.

PREDICTIVE PROCESSING

An oft-remarked limitation of enactivism is that it has yet to provide an account of the neuronal processes that enable the kind of dynamic and value-laden interaction with the environment that it emphatically supports. At least according to many of its supporters, PP fills this gap, for, as Ramstead et al. (2021:59) put it, it “provides an implementation of enactivism” at the neural level. The theory is complex and heavily debated (see Clark [2016] for an excellent primer). At its core, it posits that the brain helps us making sense of the world by continuously predicting the source of the (otherwise meaningless) barrage of inputs that hit the senses. It does so not by passively receiving and processing the input, as earlier models would have it, but by actively anticipating it, generating a cascade of downward flowing probabilistic predictions about the world. This neurally encoded and hierarchically structured stream of top-down predictions meets bottom-up sensory signal. Any mismatch between prediction and actual sensory signal is converted into ‘prediction error’ that propagates upward in the hierarchy and updates the prediction, so that the brain gets progressively better and better at aiding our perception of the world. Some of these ‘priors’ act as stable background knowledge, accounting for the most temporally extended and spatially diffuse features of the world; others are forever refined through learning. The main goal of the brain according to PP—what typifies the latter a unifying theory of brain function—is the minimization of prediction error. This is something that happens in bodily action as well, which for PP is simply the other side of perception: you predict something and act in a way that fulfills the prediction, a process going by the name of ‘active inference.’

The same principle holds true for interoceptive states such as pain (Barrett & Simmons, 2015). PP is credited with offering persuasive explanations of phenomena that are difficult to accommodate theoretically, like chronic pain. But it has done

so, in the first instance, by upending the understanding of the phenomenon that we thought was easiest to grasp: *acute* pain. The latter, for PP, is not a direct readout of sensory signal, but of a process that is invariably mediated by top-down predictions. When we are not in pain, the brain is continuously predicting our state of well-being. When the sensory signal goes beyond the predicted range of what is defined by higher priors as ‘well-being,’ predictions are very quickly updated to the perception of pain. Of course, in the context of acute pain there is a tight correlation between lesion and pain perception because the latter, though determined by priors, is dominated by the sensory signal. In chronic pain, the situation reverses: here, it is highly precise top-down prediction of pain, even in the absence of lesion, that is responsible for generating the experience (Kaptchuk et al., 2020; Ongaro & Kaptchuk, 2019).³ Psychiatric disorders are similarly explained within the PP framework as disorders of prediction. A major strength of PP as a unifying framework is to explain a vast constellation of such disorders with reference to different aberrances in the workings of the same underlying process (e.g., Downey, 2020; Edwards et al., 2012; Gadsby & Hohwy, 2020; Gilbert et al., 2022; Kube, Berg, et al., 2020; Kube, Schwarting, et al., Liddle & Liddle, 2022; 2020; Neemeh & Gallagher, 2020; Popkirov et al., 2019; Smith et al., 2021; Sterzer et al., 2018; Wilkinson et al., 2017).

A similar story, if in reverse, applies to the experience of recovery. The latter should not be seen as the direct effect of organic restoration but as the process of predicting that certain interoceptive changes are signals that recovery is underway. The ongoing prediction that we are deviating from well-being must be revised on facing evidence that the body is going back to a healthy state. This process of updating, however, takes longer (or might not take place at all) if the subject does not receive any signal that healing is taking place. Without this information, the brain might explain away the variation in interoceptive input that follows an effective medical intervention as mere “noise” and might stick to a prediction of ongoing pain. The awareness of receiving treatment leads us to infer from even small interoceptive changes in the body

the consequence of healing, and to experience relief accordingly in the form of ‘placebo effects.’ Psychotherapy, likewise, should be understood as a process of learning that works on changing priors over time.

Albeit through a different route, pharmacological treatment acts on the same process. This challenges conventional understandings of drug specificity. Consider, for example, Flaten et al. (1999) experiment on the effects of carisoprodol, which is typically used as centrally acting skeletal muscle relaxant. Flaten et al. found that its effect substantially depends on the contextual information given to the subject. People who receive the drug under the information that it is a stimulant (rather than a relaxant) perceive a higher *stimulant* effect than people who are given a placebo while being told it is a stimulant. PP theory would explain this counterintuitive finding by suggesting that people interpret drug-caused interoceptive change—whatever its exact nature—as the proof of the drug is having the expected stimulant effect, therefore heightening forward predictions. The effects of antidepressant medications may, to a substantial degree, be viewed similarly: a relatively general physiological change caused by the substance may prompt the organism to interpret that change in the direction of the received contextual cue. Obviously, drugs differ greatly in terms of their degrees of specificity. The effect depends on how exactly drugs tinker with the neural workings of the predictive process. Mechanisms might also vary depending on whether drugs affect interoceptive input or neurotransmitters that encode for the strength of top-down predictions directly (Büchel et al., 2014). Be as it may, PP shows that their efficacy should always be considered biosychosocially.

POINTS OF CONVERGENCE

The compatibility of enactivism and PP is subject of ongoing debate. As a theory of brain function, PP has been charged with neglecting the dynamic interaction between organism and the environment (Arandia & Di Paolo, 2021). In response, enactivist-minded PP advocates insist that the pro-

cess of prediction is realized across brain, body and world, in a flexible and context dependent way. For example, in the context of pain, what travels up the neural axis should not be thought simply as nociceptive signal to be processed in the brain but also a prediction error that has already been processed at the level of the peripheral nervous, autonomic, neuroendocrine, and immune systems (Kiverstein et al., 2022). Outside the body, cultural practices can play a central role in balancing the relative influence of bottom-up and top-down streams of information, so the social environment a person is immersed in should be seen as constitutive of prediction error minimization (Kirchhoff & Kiverstein, 2019; Ongaro & Kaptchuk, 2022; Veissière et al., 2019). According to this radical version, PP makes externalist claims that align with enactivism as well as ‘extended cognition’ takes on the mind (Clark, 2015, 2022). The jury is still out on whether the two approaches diverge in substantial ways or at what level they do so (Korbak, 2021; Di Paolo et al., 2022; Ramstead et al., 2021).

Differences aside, let me highlight here the more obvious parallels between the two, which lie at the level of biopsychosocial integration. Like enactivism, an upshot of PP is to cast disorders in causal terms, rather than in essentialist terms such as ‘mental’ or ‘physical.’ This is the consequence of viewing all symptoms as the product of the same inferential process, never as strictly reducible to physiological dysfunction and sometimes only loosely related or unrelated to it. PP thus shatters the artificial but pervasive distinction between somatic and psychiatric symptoms, showing that these lie on a continuum, differing only in the extent to which they are coupled to an organic disorder. This conclusion accords with the enactivist emphasis that psychosocial or physiological causes cannot be taken in isolation. PP’s understanding of drug action also falls in line with enactivism’s, which views drugs as ‘incentives’ for the organism’s holistic response to the environment (*Fuchs, 2018, p. 270). It would be a fallacy to think of a psychiatric condition as caused by the lack of a drug’s substance, like the pop theory of ‘chemical imbalance’ has it. Within the causal frameworks

presented here, bio, psycho and social factors might be at play at different degrees depending on the case, but never on their own.

On the whole, through furnishing the BPS model with a solid set of theoretical tools, and by legitimizing the causal significance of the external social dimension of illness, enactivism and (radical) PP have gone some way towards meeting the ‘integration challenge’ to the BPS model. According to Bolton and Gillett (2019), such frameworks should ultimately provide the sufficient theoretical basis for delving into the biopsychosocial composition of specific health conditions, as well as particular stages of particular health conditions (e.g., see for leads Sass et al., 2018 on schizophrenia; *Gallagher, 2022 on Autism Spectrum Disorder).

THE PRACTICAL CHALLENGE TO THE BPS MODEL

The argument of this section and the leitmotif of the paper is that meeting the integration challenge is unlikely to affect the practical challenge of implementing the BPS model. It has not so far, and it probably will not in the future. Evidently, in a world where enactivism and (radical) PP were taken seriously, psychiatry as it is currently practiced would have to be radically different. It is hard to imagine, however, what it would look like. One wonders: what are these developments going to add to the clinician’s toolkit beyond what has already been advocated by person-centered medicine for over 50 years: broadly, care, empathy, and attention to the patient’s lifeworld? Referring to the contributions of the enactivist framework, De Haan says:

An explicit [enactivist] integrative framework can help communication. In particular, it can provide 1) orientation, 2) treatment rationale, 3) a shared language for communication with all those involved, and 4) the means to explain treatment decisions to health insurers and society at large. (2020, p. 7).

But isn’t this precisely what an enactivism-powered BPS model, perhaps unlike many other approaches, does *not* do?

Let’s consider, by way of conjecture, how such an approach would fare on the psychiatric treat-

ment of patients with functional neurological disorders (FNDs). Formerly known as ‘psychogenic,’ ‘conversion,’ or ‘medically unexplained,’ these are conditions that exhibit no structural physiological disruption but still occasion a variety of chronic, somatically experienced symptoms. These disorders represent an anomaly for biomedicine and despite a high diagnostic prevalence they have been relatively marginalized and under-researched (McLoughlin et al., 2023). The typical story of FNDs patients is one of an exasperating journeying through the medical system as batteries of medical tests turn negative. Options for treatment quickly run dry and it is common that the therapeutic relationship deteriorates as a result. Patients crave for an explanation of their disorders; psychiatry generally fails to provide a meaningful one (O’Sullivan, 2016).

Explanations of a *psychological* type add insult to injury: anything suggesting that FNDs are ‘all in the head’ comes with the threat of stigma and mutual distrust (Burke, 2019; Miresco & Kirmayer, 2006; Ongaro et al., 2022). As many scholars have noted, this stigma has culturally specific origins. It has to do with a deeply ingrained distinction between the materiality of the ‘body’ and the immateriality of ‘mind’ that historically emerged with the Enlightenment, along with corresponding dichotomies between ‘reality’ and ‘imagination’ and between ‘instinctual’ and ‘intentional’ action (Goldberg, 2017; Kirmayer, 1988). Under such dualistic framings, the experience of pain is only legitimated by the concreteness of a bodily lesion. When lesions are nowhere to be found, pain stops being a symptom and becomes a disease in its own right, a disease of the mind. And where ‘mind’ is understood as the seat of agency, the patient is ultimately to blame. Unsurprisingly, many patient activist groups have revolted against the widespread use of psychological explanations of FNDs because of their implied dismissal of genuine experience. They also resist them because a diagnosis of ‘psychogenic disorder’ tends to divert research away from the potential discovery of biological upward causes of symptoms that would make the disorder ‘legitimate’ (as it happens, many cases of neurological disorders or autoimmune conditions do get misdiagnosed as FNDs) (Walzl et al., 2019).

On the other hand, *neurobiological* explanations, when something aberrant *is* found in the brain (Perez et al., 2021), go some way towards softening stigma only to disappoint in illuminating the condition to patients or to lay out a clear rationale for treatment. Generally, they are also known to induce pessimism and helplessness (Haslam & Kvaale, 2015; Loughman & Haslam, 2018; Myers, 2016; Schroder et al., 2020). To be sure, fighting stigma in the context of FNDs has been so crucial that most clinical efforts have gone in the direction of highlighting neurobiological mechanisms, with some reported success (Stone, 2023). But it is usually hard to discern whether neurobiological aberrations point at a biological cause to the illness or are neurobiological *manifestations* of the illness (most illnesses manifest themselves in the brain in one way or another). Besides, defining the disorder in neurobiological terms, as enactivists never tire to point out, risks isolating the patient from their lifeworld and from the therapeutic possibilities that might arise from it.

So, a biopsychosocially-trained practitioner of enactivist bent will certainly avoid biologizing or psychologizing the disorder. They might instead consider the latter as a disruption in the relationship between the organism and the environment, where biological and psychological factors play a role that is only relative to that of social ones, and they might, following the ‘integration challenge’ and hypothetical future scientific breakthroughs, even grasp the relative significance of each of these causal factors. But it is unclear how one should proceed from here.

The central problem seems to be that we lack the means and language to account for the social causes of FNDs. In a recent paper titled “Is ‘another’ psychiatry possible?” Rose and Rose (2023) echo this concern when writing that “In these examples of ‘alternatives’ there seems to be an unresolved problem of the ‘social’—lurking in the shadows but never explicated in a satisfactory way.” “A developed concept of the social is missing”. FNDs throw this problem into sharp relief. What we know about the social dimension surrounding these conditions, and the way we know about it, simply does not translate into effective

treatment. Evidence shows that, to a degree, these disorders are precipitated by a range of ‘social stressors’ (Ludwig et al., 2018; Morsy et al., 2022) and that they reveal correlations with ‘social determinants of illness’ such as socioeconomic status and educational attainment (Binzer et al., 1997; Deka et al., 2007; Osman et al., 2020). But current biopsychosocial frameworks do not specify how these determinants can be addressed from the clinic and, most importantly, it is difficult to see how explanations that invoke to social determinants can resonate to the patient’s struggle in any meaningful and therapeutic way.

To boot, a great proportion of FNDs cannot be traced to any unequivocal social trigger, or triggers might not take the form of threatening or traumatic events that can be easily identified (House, 2023). This suggests that just as the disorder might have undetected upward biological causes, there is a possibility that it might also have undetected downward social causes. The latter might be too subtle to be captured—reflecting the messy, unpredictable and spontaneous character of these disorders—but might still arise from interactions with the social world. The fact that such conditions show high responsiveness to ‘life events’ (Nicholson et al., 2016) supports the idea that there could be a significant but elusive social etiology behind FNDs. For all the focus on the ‘social determinants of mental illness,’ there are causes that might be *socially indeterminate*.

These shortcomings are even more significant when considering the potentially tremendous power of explanatory narratives in inflecting the experience of illness, shaping its course, and creating the conditions for recovery. We know that diagnoses of neuropsychological disease can dramatically shift patients’ position within their social world. Beside carrying legal implications and potentially inducing stigma, labels are known to induce ‘looping effects’ (Hacking, 1995) that can exacerbate the problem. Noticing a symptom while being aware of having a disease can lead to its amplification and to vicious attentional patterns that turn the problem into a chronic, self-sustaining one (Kirmayer & Bhugra, 2009; Kirmayer & Sartorius, 2007). But as explanatory narratives can trap, or even spell a demise, they also have the

power to heal. Medico-anthropological research has long shown that a fitting explanation can help the sick make sense of their condition; it can frame the disorder within the patient's culturally specific understanding of their world and chart a path to healing (Kirmayer, 1993; Kleinman, 1981; specifically on FNDs see observations by Canna & Seligman, 2020; Lagrand et al., 2023; Stone et al., 2016). It makes amorphous pain intelligible and treatment feel actionable. The efficacy of therapy significantly depends on the semantic groundwork that precedes it, which makes therapy meaningful to the patient and their community. Explanatory narratives build anticipation towards treatment that, when the latter comes, can lead to powerful—if misleadingly termed—‘placebo effects’ (Kirmayer, 2015; Moerman, 2002).

To sum up, the real problem of the BPS model, which the integration challenge does not solve, is that it lacks a social explanatory framework and a set of social therapies that can be channeled by a practitioner. Modern psychiatry comes with heaps of theory, guidelines and training about bio and psycho causes and treatment of disorders—often in mutual exclusion (Harrington, 2019; Luhrmann, 2001)—but does not harbor an equally developed set of resources for dealing with the social dimension of illness (Kirmayer & Gold, 2011; Mescouto et al., 2020). The acknowledgement of the causal significance of social forces by the BPS model hardly translates into treatment rationale (see *Aftab and Nielsen 2021; Maung, 2021; Russell 2023 for similar concerns). Patient-centered approaches that are mindful of the sick person's sociocultural background are essential, but even these cannot offer *social explanations* and *social treatments* in the same way as there are biological and psychological explanations and treatments, which, though flawed, at least afford a dose of professional confidence. In short, casting illness in as a disruption of organism-social environment relations the enactive way seems to leave practitioners *without* “a shared language for communication for all those involved” (De *Haan, 2020:7). And if this semantic void is felt most poignantly in the case of FNDs, it also affects the whole range of (partly) sociogenic conditions, such as ‘depression,’ ‘psychosis,’ or ‘anxiety disorders,’ that routinely put a strain on modern healthcare.

LOOKING OUT FOR AN EXTERNALIST PSYCHIATRY

Something that arguably does not help addressing the problem just outlined—may this be the key suggestion of the paper—is the use of the term ‘psychosocial’ in monolithic fashion. As we have seen, using this term is justified by the fact that psycho and social forces are both downward causal forces, opposed to the upward effects of biological causes. But by employing the term monolithically one neglects that ‘psycho’ and ‘social’ entail radically different approaches to illness. If there is any point in making a distinction between the two is that the first is internalizing—placing causes and mechanisms of illness in the mind and using psychological language and therapies—while the second is externalizing, taking explanation and treatment of psychiatric disorder out of the head and into the social world. Both in terms of diagnosis and treatment rationale, these two attitudes entail starkly different therapeutic scenarios. The case of FNDs shows that the absence of externalist explanatory frameworks leaves a semantic vacuum filled by internalist bio-psycho language that comes with the risk of stigma and therapeutic cul-de-sacs.

This observation does not imply that internalist explanations and treatments are harmful in themselves (in many cases, they clearly are not). Nor that externalist approaches, whatever their nature, are inherently good. What seems to be harmful is the absence of any externalist alternative that complements the exclusively bio-psychological orientation of psychiatry, in line with the integration challenge. There are reasons to believe that an externalist framework that offers patients other ways of articulating their experience—finding an explanatory language that appeals not to biological or psychological but to a meaningful social dimension—would bypass the internalism's side effects while ushering in novel forms of treatment.

With all this in mind, the prospect of developing an ‘externalist psychiatry,’ an idea that has gathered momentum in analytic philosophy (Amoretti, 2023; Cooper, 2017; Davies, 2016; Davies & Roache, 2017; Drayson, 2009; Glackin, 2017; Glackin et al., 2021; Hoffman, 2016; Krueger, 2021; Krueger & Maiese, 2018; Laval-

lee, 2023; Levy, 2013; Maung, 2023; Miyahara, 2021; Roberts et al., 2019; Sprevak, 2011; Tate, 2019; Wilkinson, 2023; Zachar & Kendler, 2007), seems, at least in principle, extremely promising. The idea is viewed as an offshoot of the ‘extended mind’ thesis (Clark & Chalmers, 1998), which states that the mind is not “all in the head” but spills over into the world. If a notebook replaces the function of biological memory, the argument goes, there is no reason to exclude it from the domain of the mental. Like neural mechanisms, our epistemic tools can be a constitutive part of our cognitive system. The thesis spawned a huge philosophical literature that has been grappling with its metaphysical and scientific implications (Colombo et al., 2019; Gallagher, 2018). Some of these writings have attempted to square the thesis with the above-mentioned theories of PP and enactivism (Kirchhoff & Kiverstein, 2019), since, particularly in the latter case, the affinities are obvious. Others have expanded on Clark and Chalmers’ initial suggestion that the mind can also extend *socially*. They argue that the thesis is at its most interesting when it concerns other humans: whether in acts of shared intentionality or through engagement with large-scale institutions, other people could also be part and parcel of one’s mind (Kirchhoff & Kiverstein, 2019; León et al., 2019; Lyre, 2018; Ongaro et al., 2022). We would be speaking, here, of the ‘socially extended mind’ (Gallagher, 2013).

The philosophical discussion around ‘externalist psychiatry’ goes hand in hand with these latest developments. It runs on the suggestion that psychiatric illness might also be constituted by relevant aspects of the social world. As Wilkinson (2023, p. 301) explains, the real promise of ‘externalist psychiatry’ lies not only in a new categorization of illness, but in its treatment too. This would consist in acting on relevant aspects of the world, rather than on the individual, conscious of the fact that illness is in part externally constituted. The implementation of an externalist psychiatry would be a way to make the BPS model truly biopsychosocial, for much of the interaction with the external world is social in kind. By employing explanatory narratives that resort to social factors, this type of psychiatry would potentially avoid

the pitfalls of internalism—for example, stigma, medicalization, negative looping effects—because it casts the possibility of healing on changes in the environment rather than on the individual. It would also imply radical novelty in treatment. The only problem would be to move this idea beyond the ideation stage. In the philosophical writings on the subject, one looks in vain for examples that add flesh to the analytical bones of the proposal. All we have is “suggestions that are promissory and in need of significant development” (Davies & Roache, 2017, p. 4).

The purpose of the following two papers is to make progress on this very front. To begin the discussion, I note that there is an important but underexplored question that logically follows from externalist commitments, which pertains to ‘cognitive ontology.’ The debate on ‘cognitive ontology’ has traditionally been a debate about the kind of entities that make up the mind (Janssen et al., 2017). Philosophers have long been arguing about whether we should adopt neuroscientific or psychological language in our descriptions and explanations of psychiatric disorders (Broome & Bortolotti, 2009; Murphy, 2017). So far, the discussion has been grounded on internalist premises. But if one endorses an externalist position, it follows that the debate about cognitive ontology should be extended to the social domain. It becomes a question of ‘social ontology.’ Specifically, it becomes a question of the ontology of social causes. What are social causes of psychiatric illness made of? Ultimately, how can these be conveyed in the clinic to therapeutic effect? Having satisfactorily dealt with the theoretical basis of biopsychosocial integration, I argue that philosophers of psychiatry should address the nature of social causes as the next main goal.

CONCLUSION

For all the critiques leveled at it, new paradigms in cognitive science are making up for what has long been considered the BPS model’s most fundamental flaw: the lack of a coherent account of how biological, psychological, and social factors causally integrate with one another. Reading textbooks on symptom perception from the 1980s to

the 1990s will reveal the progress that has been made since. The reason why these advancements fail to make a dent in clinical practice, however, is that, while acknowledging the causal significance of the social, they stop short of defining it. In the absence of a shared social etiology (without, that is, the same kind of rich explanatory framework and therapies we have about biological and psychological causes of illness), the ‘social’ remains an empty signifier. For this reason the BPS literature has had relatively little impact on the psychiatric profession, which stays bio-psychological in orientation. Stigma about mental health and a narrow range of therapeutic possibilities are the main consequences arising from the lack of any kind of developed externalist framework. FNDs are limit cases that lay this problem bare.

What’s interesting about ‘externalist psychiatry’ as an idea is that, if realized, it would come with the right answers to this problem. The challenge is, of course, realizing it. At present, it’s simply hard to imagine how externalism might work in practice and philosophical arguments go as far as they get. I have suggested that one way to make progress would be to extend the debate on ‘cognitive ontology’ into that of ‘social ontology’ and to look into the ontology of social causes of psychiatry illness. But as even this proposal might leave a theorist scratching their head, I will end this paper suggesting another source of inspiration that can aid the quest. For this, we need to look beyond the Western analytic tradition, and turn our attention to less familiar philosophies of psychiatry.

Doing so, one finds in the anthropological record an assortment of actually existing psychiatric systems that tick all the boxes for what philosophers would define as ‘externalist psychiatry.’ Anthropologists have long used the term ‘externalizing’ to classify them (Young, 1976). They observed that, in these contexts, a great portion of medical strategies do not revolve around the examination of the body, brain, or internal psychic states. These are systems that cast illness and treatment onto the external social environment; systems where, as a result, conditions such as FNDs are treated differently and where stigma around them does not arise; and where psychiatric

treatment is potentially more effective for all these reasons. It is by looking closely at how one such system works that the following papers seek to illuminate the conditions that might make a modern externalist psychiatry possible.

ACKNOWLEDGEMENT

Giulio Ongaro wishes to thank Doug Hardman, Nikita Simpson and two very helpful anonymous reviewers for their comments on a previous draft of these papers.

NOTES

1. Even contemporary philosophers of psychiatry who defend a version of the biomedical model do not discount the idea that psychosocial factors might be causally important. They simply tend to view the effects of these factors through their intermediate biological instantiation in the brain (Huda, 2019).

2. Importantly, Engel imported into psychiatry a tripartite division that had been widely accepted in social theory before. Already early in the twentieth century, anthropologist Marcel Mauss (1985[1938]) called for the recognition of a “triple man,” in which the psychological dimension figured as a mediating factor between the social and the biological.

3. See Pagnini et al. (2023) for a more comprehensive account of ‘predictive brain’ theory in relation to pain, which considers the role of attention and precision in generating symptoms.

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