

Social explanation in psychiatry

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Psychiatry is widely viewed by its practitioners as a ‘biopsychosocial’ discipline, a conception that can be traced back to Engel’s influential biopsychosocial model (BPS). In this paper I want to unpack one of BPS’s most distinctive and yet nebulous features: its emphasis on the *social* aspects of psychiatry. Nobody today could be in doubt as to the explanatory significance of biological and psychological factors in psychiatry – the ‘B’ and ‘P’ in ‘BPS’. In contrast, the ‘S’ in BPS has always been obscure. In what sense is psychiatry a social discipline? What explanatory roles are assigned to social factors by researchers and clinicians in psychiatry? What is the nature of the interaction between social and bio-psycho factors in mental illness? These questions have received little philosophical attention, which is a shame, because the waters aren’t half muddy.

After providing a retrospective on the BPS model, I distinguish four differing explanatory roles for social factors in psychiatry: 1. Input-Side roles, 2. Output-Side roles, 3. Process roles, and 4. Kind roles. Each of these roles deserves a paper (or more) in its own right; many questions will remain unanswered (or even unasked) here. The aim of this paper is simply to flesh out the conceptual territory, and stimulate further discussion of these neglected issues.

1. A Retrospective on the BPS Model

Psychiatry uncomfortably incorporates biological and psychosocial perspectives on mental illness. As a branch of medicine, psychiatry is under pressure to conform to a biomedical model, according to which genuine mental disorders are classified as diseases, to be characterised primarily in biological terms. As Zachar (2000: 21) explains,

Biomedical materialists claim that psychiatric disorders are best conceptualized as brain diseases. Many of them think that psychological analyses are irrelevant for understanding the nature of psychiatric disorders. Thinking that psychiatry should approach its professional

problems as scientifically as possible, they usually downplay the distinction between psychiatry and neurology. Some would prefer that these two specialties be merged.

Psychiatry also draws heavily on psychotherapeutic approaches, which focus on the psychosocial factors involved in mental disorder. Here concepts of abnormal or impaired belief, experience, and social structure take priority over concepts of neural dysfunction in characterising mental illness, and targeting treatments. In research, the emergence of subfields such as cognitive neuropsychiatry (Halligan & David 2001; Broome & Bortolotti 2009), social psychiatry (Morgan & Bhugra 2010), and psychiatric epidemiology (Susser et al. 2006), alongside ongoing developments in pathophysiology, provides further indication of psychiatry's need for explanations at the psychological/cognitive, social, as well as biological level.

This heterogeneity continues to generate much uncertainty concerning the conceptual foundations for psychiatry. What exactly is psychiatry a science *of*? Mind or brain? Individual or society? Dysfunction or deviance? Similar issues played out in the last century concerning the foundations for psychology, pitting those seeking wholesale reduction – or even elimination – of mentalistic concepts to those of neuroscience, against anti-reductionists who have sought to protect the descriptive-explanatory autonomy of the cognitive sciences. The current climate in philosophy of mind, however, is much less fraught. To me, at least, people seem less concerned about competition between high- and low-level explanations, and are simply getting on with the task of understanding the mind, appealing to evidence from multiple explanatory levels as and when it seems relevant.

Faced with apparent competition between the aforementioned paradigms in psychiatry, then, a similarly deflationary response would be that psychiatry too must embrace and integrate different levels of explanation: biological, psychological, and social. Such holistic concepts of medicine date back to Hippocrates, but found memorable voice in Engel's (1977, 1980) biopsychosocial model of psychiatric illness. Engel had great hopes and lofty aspirations for the BPS model. The model was intended to provide an entire conceptual framework for psychiatry. Engel not only sketched a pluralist

scheme for clinicians' case formulation, classification, and treatment; he envisaged a broad anti-reductionist metaphysics for psychiatric categories.

As has been well documented (Ghaemi 2010; McLaren 1998), the BPS model *as such* never really took root. It lacked specificity and content, and therefore unsurprisingly failed to engender a clearly recognisable scientific program. And yet the model continues to exert significant influence in psychiatric research and practice. For example, the failure to identify specific genes or biomarkers for any psychiatric illness has led many researchers to downgrade expectations for wholly reductive accounts of the causes of mental disorder. Most now agree that psychiatric illnesses have extremely complex aetiologies, combining biological and wider environmental factors. Despite its well-known shortcomings, the BPS model remains one of the only reference points for those seeking to describe this heterogeneity.¹ In more practical terms, BPS also forms the basis for core components of psychiatric training, and many (perhaps most) psychiatrists today would describe their role in terms favourable to the BPS approach. Nevertheless, when it comes to the clinic, BPS often falls by the wayside, with decisions guided by a much narrower range of factors. Gabbard and Kay (2001: 1956) nicely capture this disparity,

Almost all psychiatrists... endorse the notion that psychiatrists are distinct from all other mental health professionals in that their training and expertise allow them to be the ultimate integrators of the biological and psychosocial perspectives underlying diagnostic understanding and treatment. However, the biopsychosocial model made famous by Engel has been relegated to political lip service in our managed care era.

In clinical practice, BPS is, in a sense, everywhere and yet nowhere.

¹ Kendler (2012: 383) references BPS in outlining his own 'empirically based pluralism'.

The term ‘BPS model’ thus continues to mean many different things for different people, from the regression analysts in epidemiology to the educators in our hospitals. This malleability, however, exposes the lack of detail, precision, and clarity that has beset this model. What specific predictions does the BPS model make? What practices does it advise? What philosophical commitments does it involve? Nowhere are these questions more pressing than in understanding the intended explanatory role of social factors in psychiatry. In the following Sections, I attempt to impose some conceptual order on this issue.

2. *Input-Side Roles: Social Causation in Psychiatry*

My first proposed explanatory role for social factors concerns the postulation of social causes of psychiatric illness. The literature on social causation has boomed since the 1980s, and the findings have been varied, robust, and compelling. Here I shall focus on a few key findings concerning schizophrenia. After doing so, I will discuss some philosophical issues arising from these findings.

Schizophrenia is a complex condition that shows a high degree of heritability, and hence one infers a significant genetic component. Despite major advances in genomic research, however, there has been no single gene, nor even a clear subset of genes, that predicts the onset of schizophrenia (Harrison & Owen 2003). As research has expanded to consider other risk factors, the importance of broadly social environmental factors has become well established.² Wahlberg et al. (1997) found that children at high biological risk of schizophrenia, who were reared in adoptive households in which there high levels of ‘communicative deviance’, were at significantly greater risk of developing the illness. Bhugra (2000) reported increased rates of schizophrenia in migrant groups, with incidence even higher in the second generation. This generational effect suggests a complex role for socio-economic factors that affect established migrant populations. Relatedly, Kopell and McGuffin (2000) found a strong correlation between psychiatric admission rates, particularly for schizophrenia, and socio-economic deprivation, as assessed by factors such as unemployment, overcrowding, and being

² Early environmental insults, such as prenatal infections and nutrition, maternal substance misuse, and obstetric complications, are also implicated in this aetiology.

unskilled. Selten & Cantor-Graae (2007) report that various types of ‘social defeat’ are implicated in the development of schizophrenia.

It is worth emphasising that none of these studies claims that social factors alone are responsible for the onset of schizophrenia. The picture is far more complicated, involving the interplay between social, psychological, and biological factors in ‘a cascade of increasingly deviant development’, to borrow a phrase from Broome et al. (2005: 24). It is also important to note that none of these social risk factors has much predictive power, if any, when considered in isolation. In combination, however, a gamut of social factors can have more predictive power for the development of psychiatric illness than biological factors alone.

Generalising from this case, a typical epidemiological picture of mental illness will involve a multitude of contributing factors, each playing some causal role across the course of development. Features of the subject’s social environment are severely implicated in this picture, earning their place alongside biological – i.e. genetic or physiological – and psychological factors. What are the implications for the philosophy of psychiatry? A first issue concerns the operative notion of ‘causation’ assumed in these discussions. In what sense is a subject’s socio-economic status, for example, a ‘cause’ of schizophrenia? Kendler (2012) suggests that we need to take a ‘dappled’ or multi-level view of psychiatric causation. A similar perspective is offered by Murphy (2015: Section 2.3),

In studying eating disorders, for example, we find that social factors may explain particular epidemiological patterns, like different levels of eating disorder across populations. But social factors don't tell us why only one girl in a family gets bulimia. To explain that we can look at a particular brain chemistry that puts her at risk... But that does not establish that neurobiology really is fundamental. Rather, nothing is fundamental.

While suggestive, however, appeals to ‘dappling’ and denials of fundamentality only get us so far: more work is still needed to clarify the sense of social causation at issue.

Kendler and Campbell (2009) have appealed to *interventionist* models of causation in this connection.³ Interventionism is a close kin to *variable-based* models of causation more familiar from the social sciences. In a variable-based model, numerous possible causal factors are inputted into a statistical model, subjected to regression analysis, whereupon highly correlated variables are interpreted as bearing causal relations (modulo various background conditions). This very much fits with the statistical style of causal inference pursued by Kendler in his epidemiological studies; the appeal of interventionism is therefore understandable.

Although interesting, models of this sort fail to tell us how a putative causal factor does its causal work. That is, they don’t illuminate the *mechanisms* by which the social environment interacts with the mind/brain. Interventionists might claim this as an advantage of their view, allowing the identification of targets for the prevention or treatment of mental illness – for *interventions*, in the clinical sense – even given ignorance about the underlying mechanisms. That may well be right, but demands for mechanistic explanation are never far behind. As Lindberg & Tost (2012: 663) remark,

[S]o far, there is strong epidemiological evidence supporting a causal role for social environmental risk factors in neuropsychiatric disease, but very little empirical or theoretical accounts of how these factors may impact the brain.

Whereas philosophers have dedicated significant time developing frameworks for mechanistic explanation of bio-psycho phenomena, there is little clarity on the mechanisms underlying social influences on cognition in general, and mental illness in particular. How should we conceive of such mechanisms? The terrain once again remains to be mapped out.

³ Woodward (2003).

One initial distinction is that between mechanisms of *social-biological* causation and those of *social-psychological* causation. As an example of the former, we might consider mechanisms by which early social stressors, such as the deprivation of maternal care and peer-rejection, engage the sympathetic-adrenomedullary system (Frankenhaeuser 1986) and the hypothalamic-pituitary-adrenocortical system (Stratakis & Chrousos 1995). Such events can have lasting effects on the regulation of stress responses, implicated in the development of numerous behavioural and emotional disorders.⁴ As an example of the latter, these very same molecular processes will be modulated by psychological/cognitive mechanisms of stressor recognition or threat appraisal, which can be fruitfully described using perceptual and evaluative models. This highlights two broadly differing ways in which social stressors can ‘get under the skin’. Each type of explanation may require differing characterisations of the relevant social phenomena. It remains unclear, however, whether such phenomena themselves can be subjected to mechanistic analysis. Do the interactions involved in maternal feeding and grooming, say, constitute sufficiently regular causal systems to merit mechanistic characterisations? What about the interactions involved in a child’s entry into – and subsequent acceptance or rejection from – a peer group? What would such characterisations look like? Bechtel (2007: 240 fn. 1) offers the following brief remarks,

Just how appropriate and fruitful it is to view social systems as mechanisms is a topic beyond the focus of this book. But clearly some social systems have characteristics of mechanisms; they are coordinated systems constituted of individuals that perform a variety of operations. Of special importance is the fact that they are organized and, as a result, feed back upon their constituents and constrain their behaviour.

⁴ Gunnar & Quevedo (2007).

Nobody knows, then, quite what form a mechanistic framework for social causation will take. Given the importance of social causation in psychiatry, however, it presumably should not be long before we begin to find out.

3. *Output-Side Roles: Social Effects of Mental Illness*

An obvious counterpoint to input-side explanatory roles is *output-side* roles, concerning the social effects of psychiatric disorders. Psychiatric illnesses obviously have social effects, given the often profound changes in behaviour associated with such conditions. One might therefore question the explanatory gains of considering such effects. It is useful to distinguish, however, between what we can call *concomitant* and *characteristic* social effects. By a concomitant social effect I mean, roughly, any change in a subject's social environment that can be attributed, even in part, to their change in mental state. This could include anything from changes in attitude on the part of the subject's family or friends, to increased contact with the criminal justice system, to institutionalisation, to altered legal classification, and so on. A clear understanding of the concomitant social effects of psychiatric illness falls to sociology. By a characteristic social effect I mean any change in the subject's social environment that is a regular, predictable, feature of the course of development and presentation of a given psychiatric illness. Characteristic social effects are a subset of the concomitant social effects. What differentiates characteristic from mere concomitant effects is the frequency or reliability with which they associate with a given illness. Clearly there will be vagueness at the borders here: how reliable, regular, or predictable does a social effect have to be in order to class as characteristic? We do not have to answer that question. The value in the distinction comes in seeing the role of characteristic social effects in psychiatric theorising.

The explanatory role of characteristic social effects is clearly evident in psychiatric classification and symptomatology. Such effects are among the core diagnostic features for many disorders. I appeal here to the DSM fifth edition (2013). In schizoid personality disorder, for example, there is typically 'a pervasive pattern of detachment from social relationships and a restricted range of expression of emotions in interpersonal settings... [The individual] lacks close friends or confidants other than first-degree relatives' (2013: 652-3). Similarly in antisocial personality disorder there is

typically ‘disregard for and violation of the rights of others... Failure to conform to social norms with respect to lawful behaviours, as indicated by repeatedly performing acts that are grounds for arrest’ (2013: 659). In avoidant personality disorder (APD) one also typically finds ‘a pervasive pattern of social inhibition... [The individual] avoids occupational activities that involve significant interpersonal contact because of fears of criticism, disapproval, or rejection... Offers of job promotions may be declined because the new responsibilities might result in criticism from co-workers. These individuals avoid making new friends unless they are certain they will be liked and accepted without criticism’ (2013: 672-673). Beyond personality disorders, selective mutism is ‘characterised by a consistent failure to speak in social situations in which there is an expectation to speak (e.g., school) even though the individual speaks in other situations. The failure to speak has significant consequences on achievement in academic or occupational settings or otherwise interferes with normal social communication’ (2013: 189).

These examples highlight the importance of characteristic social effects in the classification and diagnosis of psychiatric illness. Another key explanatory role for such social effects in mental illness concerns the creation of *causal loops*. From the previous section, I assume that many social factors causally contribute to the development and onset of psychiatric illness. Insofar as such illnesses typically also have clear social effects, such effects might serve to increase the ‘toxicity’ or deleterious nature of the subject’s social environment, thereby creating a toxic cycle of causal feedback on her mental health. We can distinguish three ways in which such causal loops could operate:

First, during the prodromal stage, during which the individual presents early signs of an illness, these early symptoms might contribute to creating a social environment that increases the risk of the subject developing an acute form of the illness. Call this a *prodromal causal loop*.

Second, when the individual has developed an acute form of the illness and is symptomatic, these full-blown psychiatric symptoms might contribute to creating a social environment that

increases the risk of either a) a greater duration of illness, or b) greater severity of illness. Call this a *symptomatic causal loop*.

Third, when the individual is in remission, the lasting social effects of their prior psychiatric illness might contribute to a social environment that increases the risk of the individual either a) having a recurrence of the illness, or b) developing another psychiatric illness. Call this a *remissive causal loop*.

Although conceptually distinct, in practice we should expect significant overlap between each of these looping processes. For example, the early social effects contributing to a prodromal causal loop might persist or worsen, thus contributing to a symptomatic causal loop once the subject has developed a full-blown disorder. Nevertheless, we gain clearer explanations of certain phenomena by considering these categories independently.

To consider some examples, let's start with the prodromal causal loop.⁵ This concept is discussed by Kendler and colleagues (2003: 1193), who note that 'while psychiatric epidemiology often focuses on the causal relationship between environmental adversity and the individual (e.g. environment to person), individuals probably make important contributions to the quality of their environments (person to environment).' For example, individuals with certain traits known to increase risk of major depression are more likely to create or seek out social environments that further increase their risk of developing the illness. As Kendler (2008: 698) reports, 'an individual with high levels of the personality trait of Neuroticism – strongly associated with risk for major depression – is more prone to conflictual interpersonal relationships, reduced levels of social support, and increased rates of stressful life events, all of which increase risk for depression.'

Another example involves patients in the prodromal stage of schizophrenia. Schizophrenic symptoms are classified either as positive, such as unusual thought contents or hallucinations, or negative, such as blunted affect, emotional withdrawal, and social isolation. A study by Ventura and

⁵ I discuss these issues in connection with Hacking's (1986) famous discussion of the looping effects at length in other work.

colleagues (2004) suggests that negative symptoms such as social isolation can be worsened by the onset of positive symptoms in an 8-week prodromal phase prior to a major psychotic event. Hoffman (2007) has also argued that social isolation is a highly significant risk factor for the development of worsening positive symptoms. Hoffman (2007: 1068) suggests that this confluence of factors therefore might result in a ‘feedforward or autocatalytic process...—with worsening social withdrawal driving positive symptoms and vice-versa in a cascading process—which could account for the relatively rapid decompensations so often exhibited by patients with schizophrenia.’

Let’s now turn to the symptomatic causal loop. One extremely common feature of mental illness is that patients are often disinclined, for reasons related to their core psychiatric symptoms, to seek or accept medical help. Such aversion will often have the predictable effect of prolonging or exacerbating their illness. To consider a hypothetical example, someone suffering from persecutory delusions may find it extremely hard to believe that medical staff don't bear malevolent wishes towards them. Such patients will be unlikely to accept assistance if offered, and presumably would be less likely to seek such help of their own volition. Patients with psychosis may also lack a kind of ‘insight’: the symptomatic patient may not believe that they are unwell and hence that they need help. A patient with depression might lose the insight that they deserve help, with similar consequences. Patients with anxiety disorders such as agoraphobia, social anxiety, or panic disorder simply might be unable to endure the stress of seeing a GP. In all such cases, then, core symptoms of mental disorder can produce social effects – the distancing of the individual from medical assistance – that worsen or prolong their condition.

Turning finally to the remissive causal loop, undergoing a period of mental illness is known to increase risk of poverty, unemployment, educational failure, and loss of social capital. Such losses will typically persist long beyond the symptomatic phase, sometimes resulting in life-long disadvantage (Bartel & Taubman 1986). As we’ve seen in discussing the social causes of psychiatric illness, conditions such as social isolation and socio-economic deprivation are known risks for the initial onset of mental disorder. Such factors will be equally critical in determining the subject’s likelihood of relapse.

The preceding distinctions should prove useful in categorising the complex interactions between psychiatric symptoms and causation. If the notion of social causation in psychiatry is under-theorised, then concepts of social looping are even less developed. Understanding and modelling these notions will be crucial to unravelling the aetiological complexity of mental illness.

4. Process Roles: Social Factors in the Patient-Practitioner Relationship

Social factors are heavily implicated in the *processes* of clinical engagement, in particular those involved in the patient-practitioner relationship (PPR). Engel himself placed considerable emphasis on such process roles. Indeed it is arguable that Engel's primary concern in presenting the BPS model was to provide a framework for a socially-informed approach to clinical engagement. As Engel (1980: 543) describes it, the model 'provides a conceptual framework and a way of thinking that enables the physician to act rationally in areas now excluded from a rational approach. Further, it motivates the physician to become more informed and skilful in the psychosocial areas, disciplines now seen as alien and remote even by those who intuitively recognize their importance.' As noted above, however, Engel's proposals were vague and unfocused. How in practice should clinicians take account of a patient's social context? And to what end?

We can impose structure here by distinguishing between *epistemic*, *practical*, and *ethical* roles for social factors in the clinical process. Once again, these categories will inevitably overlap and interact in various ways. Their explanatory value becomes apparent, however, when considered in isolation. Regarding the epistemic role, social knowledge about a patient's past and present social environment is required to construct a full case history and provide an accurate diagnosis. Indeed standard case formulation in psychiatry explicitly calls for the listing of social factors that may be relevant to understanding the patient's condition. As noted above, diagnosing a patient as having, say, avoidant personality disorder clearly requires detailed knowledge of the types of social interaction that the patient is expected or required to engage in, and whether she has been fulfilling these expectations. Indeed it would be difficult if not impossible to diagnose a patient's behaviour as *avoidant* without such information about the social context in which that behaviour is embedded. This epistemic process role also relates back to our discussion of the causal roles for social factors.

Knowledge of the patient's social environment provides valuable evidence of the specific risk factors encountered by the individual during development and in recent life history, which may aid diagnosis of their disorder.

The second process role has a more *practical* dimension. On a caricatured biomedical model, the patient-practitioner exchange would be reduced to a cursory assessment of the patient's behavioural symptoms, pointing them in the direction of a brain scanner, detecting the underlying functional abnormality, and sending them home with a suitable pharmaceutical prescription. In many actual clinical contexts, in contrast, the nature of the social interaction between patient and practitioner is crucial in facilitating certain kinds of treatment. This is evident in the 'behavioural experiments' used in Cognitive Behavioural Therapy (CBT) to generate changes in the patient's mental state. Beck and colleagues (1979) emphasise the importance of interpersonal rapport, warmth, and empathy in allowing for effective therapy. Others have stressed the importance of so-called 'socialisation to the model' as an essential precursor to CBT. Wells (1997: 45) describes such socialisation as 'selling the cognitive model and providing a basic mental set for understanding the nature of treatment.' This is admittedly a rather narrow sense of 'social', pertaining solely to the communication and rapport between patient and practitioner. The role is nonetheless crucial to understanding the practical importance of interpersonal factors in the clinic.

The third process role concerns the *ethical* significance of social factors in clinical practice. This ethical role overlaps significantly with the epistemic and practical roles just considered. I've argued that acquiring knowledge of a patient's social environment can aid diagnosis, and that certain types of social interaction between patient and practitioner may enable some kinds of treatment. As such, these epistemic and practical roles may also have significant ethical consequences, in terms of improving outcomes for the patient, and improving the experience of care by reducing stress levels, for example. One could also argue that there is some inherent value in such socially-engaged practice, insofar as the patient is to be approached as a person embedded within a particular socio-cultural context, with idiosyncratic hopes and fears regarding their own treatment and outcomes.

5. Kind Roles: The Nature and Constitution of Psychiatric Illnesses

The discussion so far has avoided a fundamental question: are psychiatric illnesses themselves inherently social? This question relates to philosophical theorising about the metaphysics of psychiatric kinds, a topic so vast that I cannot cover much ground here. Instead I will provide two new arguments for the view that social factors place constitutive (as opposed to merely causal) conditions on some psychiatric kinds. By a ‘constitutive condition’, I mean roughly any condition that specifies – in part or whole – the nature of a given kind or entity. One can cash this out modally as follows: condition *C* is constitutive of kind *K* just in case, necessarily, if *K* is instantiated, then *C* obtains. For example, perhaps being disposed to assent to *p* is partly constitutive of believing that *p*; if so, then necessarily, if *S* believes that *p*, then *S* is disposed to assent to *p*. Likewise, it seems that being caused by the indentation of a foot is partly constitutive of being a footprint: necessarily, if *x* is a footprint, then *x* was caused by the indentation of a foot. Another way to cash this out is that constitutive conditions are relevant to the *individuation* of kinds: what distinguishes belief from desire is, in part, that belief – but not desire – satisfies the dispositional condition noted above.

The claim is that some mental illnesses are partly individuated by features of the subject’s social environment. More precisely: necessarily, for some psychiatric kinds *K*, if an individual *S* instantiates *K*, then *S* stands in certain relations *R* to individuals or institutions in her family, community, or wider society. This schema crucially is not trivially satisfied by all social *causal* conditions, because not all social causes of mental illness provide *necessary* conditions on instantiating a given psychiatric kind. For example, clearly it is not a necessary constitutive condition on having schizophrenia that one belong to a migrant community, or that one comes from a household with high levels of communicative deviance. These social causes are merely contingent risk factors for developing this condition.

Nevertheless, I argue that some social causal conditions provide constitutive conditions on psychiatric kinds, just as being caused by an indentation of a foot provides a necessary condition on being a footprint. Consider posttraumatic stress disorder (PTSD). It is plausible that, necessarily, if *S* has PTSD, then there has been some traumatic (perhaps life-threatening) event *e* such that *S* directly

experienced *e*, eye-witnessed *e*, or learned about *e*'s occurrence to a close family member or friend.⁶ It seems to be not merely causal but also partly constitutive of having PTSD that some traumatic event has occurred in one's wider environment during one's life, and that one is related to this event in a particular way. To contrapose, were one *not* to have encountered such a traumatic event, then regardless of one's psychological or behavioural symptoms, the classification of PTSD would not apply.

Other psychiatric conditions follow a similar pattern. Consider the case of adjustment disorders, which according to the DSM (2013: 286-287) involve marked distress following an 'identifiable stressor' in one's social environment, such as the 'termination of a romantic relationship' or 'marked business difficulties'. The very concept of 'adjustment' implies that there has been some event or change to which the patient has failed to adapt; which has been found stressful or distressing, but where the response is in some sense disproportionate, impairing important areas of functioning far more than could normally be expected.

Another route to the conclusion that at least some psychiatric kinds are constitutively social goes via externalist views of mental content. The argument proceeds as follows. First, many psychiatric illnesses are partly constituted by the presence of *contentful states*: disordered or abnormal perceptions, beliefs, or desires. These are states that represent the world as being a certain way. Second, differing types of representational content help to individuate differing kinds of mental state. What differentiates a belief that cheddar is tasty different from a belief that stilton is smelly is that these two beliefs represent different propositions. Likewise, what differentiates a perception of redness from a perception of greenness is that these two perceptual states represent different properties. Third, from the first and second premises, it follows that many psychiatric illnesses are partly constituted by states that are kind-individuated by their representational contents. If having psychiatric illness *K* in part involves having contentful states of types *A*, *B*, and *C*, then having illness *K* involves having states that are individuated by their representational contents. Fourth, the

⁶ I here closely follow the diagnostic criteria for PTSD provided in DSM-V (2013: 271).

representational contents of a subject's beliefs, desires, and perceptions do not supervene on narrow, purely neural, facts about the subject's brain. These contents are determined by facts that includes the subject's relations to her natural and social environment. This is a familiar *social externalist* claim about mental content.⁷ Fifth, from premises three and four, it follows that many psychiatric illnesses are partly constituted by states that are kind-individuated in part with respect to features of the subject's social environment. Insofar as psychiatric illness *K* is partly constituted by having contentful state *A*, then if *A* is partly individuated by features of the subject's social environment, it follows that *K* is partly constituted by states that are themselves individuated by social features.

Some may dispute the first premise of this argument, that some mental disorders are constitutively representational. Others may dispute the fourth, social externalist, premise. I think that both premises are extremely plausible, although a full-dress defence of these claims must await another occasion. If sound, the argument provides a powerful, general, (if somewhat derivative), route from the social nature of content, to the social nature of mental illness. The argument thus carves out another important explanatory role for social factors within psychiatry, a role that will inform further theorising on the foundations of the discipline.

6. Conclusions

For half a century, the biopsychosocial model has beguiled and frustrated researchers and clinicians across psychiatry. Its continued relevance is best explained by its distinctive emphasis on the social aspects of psychiatry. To my knowledge, no other conceptual framework for psychiatry places the social on an equal footing with the biological and psychological in explaining mental illness. Whereas philosophy is replete with discussions of the bio-psycho relation, however, the interaction between bio-psycho and social factors remains woefully under-theorised. In addressing this dearth, I have distinguished a number of explanatory roles for social factors in psychiatry. Although numerous questions have been left open, I hope that the conceptual mapping I have provided will prompt and facilitate more sustained, detailed, reflection on these issues.

⁷ Burge (1979).

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