The conundrum of the psychological interface: On the problems of bridging the biological and the social

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Abstract
In this article, we consider how certain types of contemporary biosocial psychiatric research conceptualise and explicate biology-social relations. We compare the historic biopsychosocial model to recent examples of social defeat research on schizophrenia and cultural neuroscience work on affective disorders. This comparison reveals how the contemporary turn towards the ‘biosocial’ within psychiatric research relies upon ideas of the psychological as an interface. This is problematic because psychological notions of ‘experience’ are used as the central mechanics of biosocial processes, but lack any meaningful engagement with considerable debates within psychology and cognitive science about what the mind, and indeed the psychological, actually is, its relationship to social life, and how we should study it. The psychological interface is therefore vital to these biosocial hypotheses but is remarkably underdeveloped in comparison to its biological and sociological components. We argue that biosocial psychiatric research could gain a great deal from engaging with contemporary theorisations of experience and being more critical of vague appeals to psychological phenomena.

Keywords
biopsychosocial, biosocial, cultural neuroscience, schizophrenia, social defeat

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Introduction

In this article, we consider how certain types of contemporary biosocial psychiatric research conceptualise and explicate biology-social relations. Our main argument is that the contemporary turn towards the biosocial within psychiatric research reduces the psychological to a (superficial) interface, which mediates – in unspecified ways – between the biological and the social. This, we will argue, is problematic because it appeals to lay psychological notions of experience as the central mechanics of biosocial processes. At the same time, however, this concept of experience becomes a black box. It is rarely explicated, and there is little meaningful engagement with the considerable debate within psychology and cognitive science about what the mind, and indeed the psychological, actually is, its relationship to social life, and how we should study it. In the following, we will explore how contemporary psychiatric biosocial research conceptualises the biological and the social as distinct categories that interact through a poorly defined psychological interface. Hence, while psychological phenomena (i.e. mental illnesses) are often the outcomes of biosocial psychiatric models, the psychological (in the guise of ‘experience’, ‘uncertainty’, ‘internalisation’, etc.) also performs a central function within those models, bridging disparate conceptual components, and even translating them into one another. This approach to the biosocial, using the psychological to interweave biological and social components, entails epistemological and empirical questions regarding the statuses of, and relations between, traditional human science disciplines and their engagements (or lack thereof) with imaginings of one another’s intellectual categories.

In this article, we are particularly interested in exploring how the psychological is used within biosocial psychiatric research as an interface, because, as we will show, it seems to be integral to much of what we call biosocial psychiatric research, yet it is simultaneously under-explicated. To explore this issue, we analyse specific examples of composite biosocial psychiatric research – social defeat and cultural neuroscience – before discussing, in the final part of the article, some contemporary psychological attempts at re-theorising experience. We analyse these examples in the spirit of facet methodology, whereby dissimilar representations of a central research problem are explored to highlight salient issues within that underlying problem (Mason, 2011). A facet approach allows us to elaborate a critical appraisal by attending to ‘different ways of seeing’ biosocial psychiatric research (ibid.: 77). In this spirit, we bring together ‘combinations and constellations of facets’ (ibid.: 76) that singularly reveal ‘facets’ of our research problem, and in sum delineate something of its complex nature. For Mason, research problems resemble cut gemstones in that they have several distinct faces, each uniquely reflecting and refracting light, so that our appreciation of the full entity is enhanced by adopting several perspectives from various angles. Therefore, this article attends to research enterprises that are rarely collated, the collation of which serves to reveal something interesting about our core concern – the psychological interface. Our purpose here is not to analyse all biosocial psychiatric research, but rather to explore some emblematic examples hereof so as to elucidate some of the ways in which the psychological becomes posited as a bridging interface between biology and the social. Before commencing with this analysis, however, it is necessary to specify what we mean by ‘biosocial psychiatric research’.
Biosocial psychiatric research

We use the term biosocial psychiatric research as a useful, but admittedly broad, way of denoting the large body of work that, today, pursues causal explanations of mental disorders by integrating explicit sociological and biological concerns. In recent years, much scholarship has attended to the wider concept of the biosocial, with social theorist Meloni (2014) arguing that biology has ‘become social’. Meloni takes his point of departure in the idea that the brain is social, that is, profoundly connected to and shaped by the organism’s encounters with the world, and the much discussed (and controversial) findings within epigenetics, which suggest that the genome itself is reactive to the environment. To simplify his argument, where once biologists rejected the social, and sociologists rejected the biological, the social and biological as epistemic fields are becoming, in some parts, more intertwined. We cannot, in this article, give space to discuss the large and growing literature both discussing and examining these developments, and how they came about (see Fitzgerald, Rose, and Singh, 2016), and so we shall confine ourselves to the notion of biosocial psychiatric research. Much as social theory has had a tenuous relationship with biology, so has the role of social phenomena in the genesis of mental illness long been a contested terrain in psychiatry, with sociological explanations repeatedly falling in and out of favour (Brown and Harris, 1978; Fitzgerald, Rose, and Singh, 2016). Over the previous two decades, much epidemiological psychiatric research has rejected absolute biological accounts in favour of more biosocial sympathies (e.g. Kirkbride et al., 2007; March et al., 2008; Morgan, 2008; Murray, 2017).

Biosocial psychiatric research is not held together by dedicated disciplinary trappings and is not a self-assigned label. Instead, we use the term to indicate (albeit imperfectly) research enterprises relying on a set of assumptions regarding the combined (and, importantly, co-dependent) social and biological causation of psychiatric disorder. We focus on this particular intellectual commitment as a unifying theme, rather than one particular institution/research group per se, because biosocial psychiatric research is an amalgam manifest in several eclectic forms. These forms are not necessarily led by medical psychiatrists, with researcher backgrounds ranging from anthropology to neuroscience, but rather are psychiatric because they are fundamentally concerned with the biosocial causation of psychiatric disorder. Ultimately, our demarcation of biosocial psychiatric research is a classificatory abstraction that, like innumerable others, encompasses and partly squashes heterogeneity as a means of artificially emphasising commonalities (e.g. ‘biological research’, ‘psychiatric research’, etc.).

As well as work on social defeat and cultural neuroscience, discussed at length in this article, examples of contemporary biosocial psychiatric research include: (a) ‘syndemics’ work seeking to explain the epidemiological clustering of certain diseases and social circumstances (e.g. depression, diabetes and poverty), each causing and caused by the others (Singer et al., 2017); (b) early life exposure research exploring how early social circumstances contribute to molecular pathways that cause psychiatric disorder later in life (Van Hazebroek et al., 2019); (c) biosocial criminology, which researches co-dependent social (e.g. family structure) and biological (e.g. genetic predisposition) causes of criminal behaviour (Rocque and Posick, 2017); (d) geographic ‘exposomic’ research on how
place (eco-socially conceived) is embodied in ways that are hypothesised to cause psychiatric disorder (e.g. allostatic load reflecting social interactions; Prior, Manley, and Sabel, 2019); and (e) dementia prevention research investigating the longitudinal interplay of genetics and social context in determining susceptibility to conditions such as Alzheimer’s disease in later life (Livingston et al., 2020). These examples highlight the eclectic breadth of contemporary biosocial psychiatric research (and this, it should be said, does not include the even broader explorations of the epistemological and ontological terrain of the biosocial, e.g. Meloni, 2014). We could expand this list tenfold, but suffice to say that the early 21st century has witnessed an increase in diverse biosocial approaches to the causation of psychiatric disorder. Interestingly, this heterogeneity indicates the convergent evolution of biosocial thought within several disciplinary siloes rather than the branching-out of one particular progenitor.

Fitzgerald, Rose, and Singh (2016) have argued that the trajectory of psychiatric thought towards the biosocial – mirroring a concurrent sociological turn to the biosocial (Meloni, 2014; Meloni, Williams, and Martin, 2016; Niewöhner and Lock, 2018) – is revitalising the relations between psychiatry and sociology in a way that was last evident in the early 20th century. This is telling, because as with much academic thought, the contemporary psychiatric turn to the biosocial has historical precedent. Indeed, Meloni (2019) has argued that the biosocial turn is more of a complex continuation of historic scholarships rather than a radical break with the past. For example, in the 1920s, the American behavioural psychologist Albert Paul Weiss (1929, 1930), incidentally also a keen physiologist and sociologist, famously advocated for biosocial understandings of mental illness (Elliott, 1931). For Weiss, the biosocial approach was a materialist means of dealing with the psychic – mental and psychological – concepts, which he deemed too ethereal to be of use to psychiatry. He reclassified this psychological interiority as behaviour to make it more conducive to conventional scientific study. He attributed psychiatric conditions to biosocial responses, mediated through the sensory organs, whereby external stimuli interacted with, shaped, and were shaped by internal biology to produce physiological and behavioural outcomes. In this account, separate biological and social realms are clearly distinguished, and their interaction via the sensory organs produces psychiatric phenomena. This is the stimulus-response mechanism of classic behaviourism, positing two different types of ‘stuff’ (biology and behaviour) and a mechanism through which they influence each other. While it may seem like an outmoded way of thinking, we argue that similar styles of thought are used in contemporary psychiatric research today.

What we will show in this article is that there is still a tendency, in some biosocial psychiatric research, to consider the social and the biological as two distinct categories of phenomena, and to subsequently bring those separate things together within one overarching account of life (Ingold, 2013). Much work has attended to questions of how these two components of being relate to one another, but our focus in this article is more specifically on the ways that nondescript notions of the psychological can come to occupy that question of relating. The psychological, we will argue, commonly takes on an ambiguous role here as both outcome and core mechanism. First, and most obviously, it is the traditional outcome within biosocial psychiatric research, articulated in terms of mental disorder or illness. Second, and more importantly for this article, it is
also a bridging interface that connects between sociality and neurobiology, a sort of *arbiter* or mediator of the world and the molecule. This second role is rarely scrutinised within biosocial psychiatric research, and it is this role that we therefore focus on here. For example, in Cacioppo’s famous work on social isolation and loneliness in human beings, loneliness is *perceived social isolation* (Cacioppo et al., 2015; Cacioppo and Hawkley, 2009). Here, perception (as a psychological phenomenon) comes to bridge between a social phenomenon (social isolation) and a series of deleterious neurological effects in the individual, with Cacioppo et al. (2015: 761) concluding that

social isolation has been recognized as a major risk factor for morbidity and mortality in humans for more than a quarter of a century…. The extant evidence indicates that the perception of social isolation (i.e., loneliness) is also a risk factor for broad-based morbidity (both physical and psychological) and mortality.

In this example, which we will attend to in more detail later in the article, the role of the psychological interface is two-fold: it functions as both an outcome of a biosocial process and the key bridging mechanism between the social world and the neurobiology of human beings – as the mediator of our worlds. As a result, it is not just the social circumstance in and of itself that has deleterious effects upon human beings – it is the *perception* thereof.

We are particularly interested in exploring how the psychological is used within biosocial psychiatric research as an interface, because in examples such as this it seems to be integral to the model, and yet under-explicated. To explore this issue, we analyse two specific examples of composite biosocial psychiatric research – social defeat and cultural neuroscience, before discussing, in the final part of the article, some contemporary psychological attempts at re-theorising experience. We analyse these examples in the spirit of facet methodology, whereby dissimilar representations of a central research problem are explored to highlight salient issues within that underlying problem (Mason, 2011). We do not seek to assess biosocial psychiatric research in its entirety. Instead, we analyse a few illustrative instances of work in the field to explicate how biological and social components are conceptually combined via appeals to psychology, which hence acts as a sort of bridging interface. We do not suggest that the practice is ubiquitous, solely that it is widely evident in biosocial traditions. We consider the examples of social defeat research on schizophrenia and cultural neuroscience research on affective disorders. These examples represent influential and dissimilar approaches to biosocial psychiatric research, and in doing so underline the important similarities that we seek to draw out. However, before proceeding to examine these traditions, it is first helpful to return to a historical and somewhat similar composite psychiatric model that utilised a notion of the psychological at its core: the biopsychosocial model.

**The biopsychosocial model**

George Engel (1977, 1980) introduced the biopsychosocial model into psychiatry in the late 1970s. Engel saw psychiatry as splitting into two opposed positions in relation to medicine, the ‘exclusionist’ and the ‘reductionist’. The former aimed to break away
from medicine and its physiological concerns, while the latter aimed to adopt an entirely physiological focus. In response, Engel dismissed contemporary biomedical psychiatry as outdated and dogmatic, and instead advocated a middle-ground position encompassing biological, psychological, and social phenomena in equal measure. He termed this the ‘biopsychosocial’ model (Benning, 2015). The biopsychosocial model became mainstream in late 20th-century psychiatry (Ghaemi, 2009, 2010).

Engel (1980) grounded his biopsychosocial approach in a systems theory drawn from early 20th-century biology. Systems theory proponents argued that clinicians mistakenly attempted to emulate reductionist laboratory practices of isolating distinct entities for study because they believed such methods to be uniquely scientific. Engel deemed such an approach problematic because human patients could only be understood as individual entities themselves, at a different existential level than the phenomena studied in laboratory science. Each patient is comprised of a multitude of complex parts, but is not intelligibly reducible to composite parts, because the clinical significance of those parts is lost when they are stripped of their context. At the same time, each person is also a part, occupying a mid-range position within a ‘continuum of natural systems’ encompassing several levels of biological, psychological, and social phenomena (see Figure 2 in Engel, 1980). The key to mental health is the maintenance of these separate levels within a dynamic but functionally stable configuration.

While Engel does not explicitly categorise biological, psychological, and social parts of the continuum, he does identify the position of the person within the nested continuum of natural systems as ‘the highest level of the organismic hierarchy and the lowest level of the social hierarchy’ (1980: 536). Hence there is an immediate separation of distinct types of biological and social entities, and a degree of uncertainty regarding where and how the psychological fits in, though the person level seems to be a mostly psychological entity in Engel’s writing. Of course, the keen observer could also add that all of the separation is unclear in some respects: where do we draw the distinction between ‘person’ and ‘culture’? – given that who counts as a person is a profoundly cultural question – and how do we distinguish, particularly in an age of climate change, between ‘biosphere’ and ‘nation-society’? However, for the purposes of our argument, it is helpful to focus on the observation that the psychological is here perhaps most aptly defined as an overlap of biological and social levels – a tellingly vague operationalisation.

Engel’s reliance on systems theory as a basis for biopsychosocial psychiatry was an attempt to develop a unique methodological approach dedicated to understanding the patient and their position within a complex milieu of biological, psychological, and social phenomena. This methodology was intended to avoid unidirectional approaches in which clinicians moved from biological to psychological to social concerns in a set order, separating out discreet composite phenomena. Instead, he advocated a circular approach to biopsychosocial psychiatric practice, iteratively and simultaneously. However, Ghaemi (2009) has noted that the model gives little guidance on how to move between levels and draw them together, and in practice biopsychosocial psychiatrists approach this issue in whatever manner they choose. He claims that ‘there is no rationale why one heads in one direction or the other: by going to a restaurant and getting a list of ingredients, rather than a recipe, one can put it all together however one likes’ (ibid.: 3). While Engel articulated bidirectional links between biological,
psychological, and social phenomena, he did not prescribe any specific approach to usefully uniting them as a basis for intervention. At best, he provided complex diagrams for individual patients, listing relevant phenomena operating at each level. In this manner, the biopsychosocial model failed to integrate its component levels.

The story of the biopsychosocial model offers insights into the problems faced by composite conceptual schemas that seek to integrate biological and social phenomena through an overlapping area of psychological material lying at the centre. In this article, we argue that similar issues affect the contemporary turn to the biosocial. The biopsychosocial raises questions regarding how composite biosocial models employ notions of the psychological to delineate mechanisms of action (see Manning, 2019). To explore these questions, we will draw on examples of contemporary biosocial psychiatric research: social defeat research on schizophrenia and cultural neuroscience research on affective disorders.

Social defeat

The first area of biosocial psychiatric research that we will examine is social defeat research on schizophrenia. We do so because this tradition exemplifies the problems that emerge from those composite biosocial conceptualisations that distinguish the biological from the social and seek to move between the two in some way. Social defeat is a biosocial psychiatric research tradition that is rooted in the long-standing practice of using animal-models to investigate human psychology, with an emphasis on social stress (Björkqvist, 2001; Ramsden, 2012). In the case of social defeat, the animal used is the rat. Social defeat emerged from the resident-intruder paradigm, whereby a small ‘intruder’ rat is introduced into a large ‘resident’ rat’s cage, the resident having previously co-resided with a female rat to encourage territoriality (Koolhaas et al. [2013] provides a good overview of the experiment). The intruder and resident rats are typically video recorded displaying a range of aggressive social behaviours for several minutes. The resulting footage is analysed to classify component behaviours in terms of duration, frequency, and type. These metrics offer a means of operationalising the social behaviours of the rats. The intended result is that the intruder is socially defeated by the resident, allowing researchers to examine the effects of social defeat.

While resident-intruder experiments are employed to emulate social interaction in a laboratory setting, the major concern of such research is typically the extrapolation of physiological implications (Björkqvist, 2001). During the 1990s, a substantial body of work developed outlining a wide range of pathological molecular processes stemming from the rats’ interactions. In comparison with controls, defeated intruder rats have been found to have impaired dopaminergic and corticosterone response (Albeck et al., 1997; Tidey and Miczek, 1997), immunological function (Stefanski and Engler, 1999), and neurological development (Alleva and Aloe, 1989). They also display reduced testosterone, heightened norepinephrine and epinephrine (influencing heartrate; Stefanski, 2000), hardening of the arteries, and an increased probability of cardiac tachyarrhythmia (Sgoifo et al., 1999). Overall, the evidence suggests that social defeat in rats entails a range of harmful physiological consequences.
Psychiatric research frequently applies rat-model research findings to human phenomena (Rose and Abi-Rached, 2013). In the case of social defeat, such extrapolation was largely implicit until the early 2000s, when scholars began to explicitly advocate for directly using social defeat as an animal model for human mental health (see Fletcher and Birk [2020] for a fuller analysis of this history). Such appeals were especially concerned with the potential to develop parallel biological understandings (Björkqvist, 2001). The promise of a biosocial social defeat approach to human mental health was realised by Selten and Cantor-Graae in 2005, when they proposed a social defeat hypothesis of schizophrenia (Selten and Cantor-Graae, 2005). The paper was the first dedicated attempt to apply social defeat as an explanation of human mental illness, specifically epidemiological findings regarding schizophrenia incidence.

The authors took as their point of departure a meta-analysis of epidemiological research on schizophrenia, migration, and urbanicity, which found that migrants (and descendants of migrants) and people living in cities have a higher risk of developing schizophrenia (Cantor-Graae and Selten, 2005; see also Morgan, Knowles, and Hutchinson, 2019; Vassos et al., 2012). They argued that these associations are explained by social defeat because urban-residents and migrants face elevated social competition and a greater chance of social defeat, which they define as ‘a subordinate position or as “outsider status”’ (Selten and Cantor-Graae, 2005: 101). Having implicated the experience of social defeat in schizophrenia, they progress to the biological, suggesting that ‘a chronic and long-term experience of social defeat may lead to sensitisation of the mesolimbic dopamine system (and/or to increased baseline activity of this system) and thereby increase the risk of schizophrenia’ (ibid.). This hypothesis is further supported by the observation that the mesolimbic dopamine system is sensitised in both socially defeated rats and humans living with schizophrenia, which could suggest the existence of a shared underlying mechanism whereby social experience is translated into biological processes (Cantor-Graae and Selten, 2005; Selten, Cantor-Graae, and Kahn, 2007).

A social defeat account of schizophrenia seeks to integrate certain phenomena that are discretely categorised as either biological or social, with each influencing the other and neither working without the other. Overall, Selten and colleagues broadly move from the social (admittedly crudely conceptualised from the perspective of a social scientist) to the biological. The process that is described is mostly unilinear, beginning with social considerations (i.e. some meaningful interaction between two organisms) as the initiators of various biological cascades associated with the model. From this point onwards, the mechanics of schizophrenia are devoid of the social, the initial interaction having been transformed into molecular phenomena.

In social defeat, then, we can read a relatively unidirectional composite biosocial account in which the social encounter initiates biological processes that ultimately result in psychoses. Tellingly in this respect, Selten co-authored a paper entitled ‘Defeat Stress in Rodents: From Behavior to Molecules’ (Hammels et al., 2015). Social defeat could thus be characterised as presenting a linear socio-bio-psycho chronology. However, what we are especially interested in here is not the ordering of distinct components, but rather the translational mechanics that are used to move between those components. How is it that the social initiates the biological, that being socially defeated
becomes manifested in the mesolimbic system? It is here that we discover the psychological bridges of ‘uncertainty’ and later ‘experience’.

Over the years, various explanations of the social-biological transition have been offered in publications regarding the social defeat hypothesis. Beginning in 2004, Selten and Cantor-Graae (2004) published a chapter in which they did not yet name ‘social defeat’, but in which they outlined the foundations of the hypothesis. In this chapter, the authors suggest of associations between migration and schizophrenia, that ‘the common mechanism is uncertainty about social rank or status’, and that this mechanism ‘contributes to the etiology of schizophrenia by disturbing brain dopaminergic function’ (Selten and Cantor-Graae, 2004: 16–17). Thus, at its genesis, social defeat was a process whereby social circumstances translated into neurophysiological processes through a psychological phenomenon – an individual’s sense of ‘uncertainty’. Despite its core function as the socio-to-bio translator, what exactly constitutes ‘uncertainty’ remains unclear.

A year later, the authors outlined the hypothesis explicitly and in greater detail as outsider status sensitising the mesolimbic system, ‘whereby exposure to a given stimulus results in an enhanced response at subsequent exposures, in this example excess release of dopamine’ (Selten and Cantor-Graae, 2005: 102). In this case, the stimulus is exclusion from membership of a majority group. This depiction of the translational mechanics of social defeat has broadly continued. In 2016, the authors wrote, ‘The long-term experience of being excluded from the majority group leads to an increased baseline activity and/or sensitization of the mesolimbic dopamine system’ (Selten, Van Os, and Cantor-Graae, 2016: 294). Note here that an undefined notion of ‘experience’ is preferred to uncertainty. Other additions have sometimes been offered, for example: ‘The psychosocial environment, and chronic stress in particular, can mediate changes in gene expression, the hypothalamic-pituitary-adrenal (HPA) axis, mesolimbic DA neurotransmission, and behaviour’ (Selten et al., 2013: 1183). In this example we see a broader range of potential causes and consequences, and a bringing together of the social and the psychological and the ‘psychosocial’.

In sum, one might point to a range of imaginings of the translation of the social into the biological, via nondescript psychological terms, within the broad body of work on social defeat. However, some ideas are repeated more frequently and with more fervour than others. Experiences of stress, defeat, and/or exclusion, sensitising the mesolimbic system, are predominant. This approach can be narrowed down to experience causes sensitisation. As a consequence, the psychological issue of ‘experience’ (or ‘uncertainty’ in earlier work) is employed as a bridge between social exclusion and biological sensitisation. This interface of experience is not explicated further, and indeed, its deconstruction is not necessarily the authors’ focus, which is instead dedicated to the resulting schizophrenia. However, in the intellectual sense, it is notable that such a pivotal moment in the proposed process remains so ethereal. Experience provides a somewhat puzzling psychological interface between the social and the biological. What it means to experience or perceive something, and what experience is, is (as we will return to), far from settled territory within the human and social sciences. This ethereal idea of experience lies at the centre of social defeat and performs a great deal of explanatory labour; it is only ever glimpsed fleetingly and is never engaged with directly, let alone with any psychological
writings on the topic. We will return to this observation in the discussion below, but before doing so, we will highlight another similar psychological interface in a dissimilar composite biosocial approach to mental illness – that of cultural neuroscience.

**Cultural neuroscience**

We preface this section by saying that many of the findings within cultural neuroscience, and many of the biological mechanisms posited as central – such as plasticity – are controversial and subject to ongoing scientific debate (though, as we argue, this is not particularly evident in cultural neuroscience scholarship). In this section, we are not interested in adjudicating between the various truth claims that underlie this field. Instead, we are interested solely in the ways in which the social, biological, and psychological become defined within these fields. Put somewhat broadly, questions of the social and the cultural have gradually moved into the neurosciences since the 1990s, often influenced by fields such as social, cognitive, and cultural psychology (Fitzgerald and Callard, 2015; Rose, 2013; Rose and Abi-Rached, 2013).

Combining cross-cultural psychology with neuroscience, the field of cultural neuroscience emerged during the past decade (Han et al., 2013). It differs from social neuroscience in its explicit focus on relations between neurophysiological and cultural traits (Chiao et al., 2010), and is chiefly concerned with how the brain, genes, and culture interact and shape each other, as well as ‘understanding the biosocial nature of the human brain’ (Han et al., 2013: 336). It was initially defined by Chiao and Ambady (2007: 238) as ‘a theoretical and empirical approach to investigate and characterize the mechanisms by which [the] hypothesized bidirectional, mutual constitution of culture, brain, and genes occurs’. Intellecutally, cultural neuroscience is composed of three major concerns: cultures, brains, and genes. It uses the cultural variations documented by anthropologists as its starting point and conceptualises culture as being comprised of material, social, and subjective culture. Material culture describes the material objects created and used by members of a culture; social culture denotes their rules and institutions; and subjective culture encompasses their values, beliefs, and behaviours. Ultimately, culture is conceptualised as ‘a complex and dynamic external social environment in which the human brain is fostered and shaped’ (Han et al., 2013: 340).

The transition from culture to the mind is made via appeals to cultural psychology, which has traditionally been interested in how cultural variations translate into unique patterns of cognition and emotion (e.g. Shweder and Sullivan, 1993). A key uncertainty here is how said translation occurs. Having progressed from the sociocultural to the psychological, the concept of plasticity is then central to moving from cultural psychology to cultural neuroscience – from the psychological to the biological. Plasticity posits that the brain exists in a highly malleable state and is continually reconfigured, both structurally and functionally, in relation to extrinsic factors. It follows that brain structure and function adapt according to culture. Cultural neuroscientists hypothesise that the socialisation process of internalising the culture into which a person is born is echoed in neurophysiological adaptation. They suggest that the brain is moulded in response to extrinsic cultural stimuli experienced by the person (Han et al., 2013). Again, the invocation of experience, in the guise of internalisation, is notable, to which we will return.
Cultural neuroscience has also included a strong genetic focus since its initiation (see Chiao and Ambady, 2007). Genes are typically employed as a biological influence working alongside the social influence of culture, with the brain being shaped by these two forces (Han et al., 2013). The introduction of genes is grounded in the theoretical traditional of gene-culture co-evolution, whereby biological and social evolutionary processes are conceived as guiding the trajectories of each other (Lumsden and Wilson, 1981). The classic example of this theory in practice is the high prevalence of lactose-tolerance in cultures traditionally dependent on dairy agriculture (Kim and Sasaki, 2014). Cultural neuroscience builds on this tradition to suggest that a combination of one’s genes and culture shapes one’s brain. It is in the domain of genetics that cultural neuroscience is found most wanting, having so far failed to accrue robust evidence that culture moderates the effects of gene expression on the brain (Han et al., 2013).

The traditional methodological approach of cultural neuroscience is to measure neurological differences between different cultural groups and to associate those with cultural traits. This research draws on a long-standing body of work stratifying psychological characteristics by culture, and seeks to establish corresponding genetic and neurological stratification, being particularly bound up with the hypothesis that psychological phenomena are rooted in neurophysiology (again, a seemingly contentious claim that is largely naturalised within cultural neuroscience). The field is hence characterised by the drawing together of diverse strands of existing research, and by the development of neurological and/or genetic hypotheses based on past cultural psychological observations (Han et al., 2013).

An interesting example of cultural neuroscience in action is Chiao and Blizinsky’s (2009) study of associations between cultural collectivism/individualism and the allelic frequency of the serotonin transporter functional polymorphism (5-HTTLPR) in relation to affective disorders. They collected representative population-level data on allelic frequency of the 5-HTTLPR for 29 countries and on mental health disorders (anxiety, mood disorder, impulse control, substance abuse) for 12 of those countries. They compared this against Fincher et al.’s (2008) comparison of cultural collectivism-individualism and geographic pathogenic load, which found a positive correlation between the general prevalence of pathogens in a region and cultural collectivism, and concluded that collectivism was a cultural-evolutionary response to disease pressures.

Building on Fincher et al. (2008) study, Chiao and Blizinsky (2009) conducted several analyses. First, they found a strong association between short allele 5-HTTLPR and collectivism, operationalised at the national, regional, and ‘cultural cluster’ levels (see Gupta, Hanges, and Dorfman, 2002). Second, they found that the frequency of short allele 5-HTTLPR in a population was significantly negatively associated with incidence of anxiety and mood disorders. Finally, they conducted a mediation analysis between frequency of short allele 5-HTTLPR, individualism-collectivism, and prevalence of anxiety and mood disorders, finding that the inclusion of cultural individualism-collectivism dramatically decreased the effect of 5-HTTLPR. The authors interpret these results as revealing a causative pathway whereby short allele 5-HTTLPR begets neural information-processing biases that facilitate cultural collectivism, which is protective against affective disorders. To support this hypothesis, they appeal to neuroimaging results revealing associations between short allele 5-HTTLPR and amygdala response
to emotional stimuli (e.g. Munafò, Brown, and Hariri, 2008). They suggest that their findings provide the first robust evidence of a link between cultural values, genetic expression, and mental illness, and that the influence of culture may help to explain the inconsistent results of similar studies regarding associations between genes and mental illness.

Cultural neuroscience shares the composite nature of social defeat. Cultures, brains, and genes are generally conceptualised as three distinct components, representing a strongly composite biosocial approach. This is partially a legacy of the intellectual foundations of cultural neuroscience, emerging from the separate traditions of cultural psychology, neuroscience, and gene-culture co-evolution. The distinctness of those founding disciplines is sustained in the repeated identification of cultures, brains, and genes in the cultural neuroscience literature. At a broader scale, there is also an evident distinction between the biological and the social. Culture is deemed a social factor, albeit biologically influenced, while the brain and genes are biological factors, albeit socially influenced. Where cultural neuroscience differs from social defeat is in its bidirectional approach to the relations between its components. Chiao and Ambady (2007: 238) note that ‘humans may come into the world with a set of possible developmental trajectories, but once on a certain trajectory, plasticity may alter both the path and the end state’. In this sense, the mechanisms described by cultural neuroscience are sincerely multidirectional. There is also a much greater equality of attention and conceptual depth in relation to biological and social considerations. Cultural neuroscience’s reliance on cultural psychology and appeals to anthropology mean that, while certainly open to the usual reductionism critiques, it generally presents reasonably sophisticated conceptualisations of social phenomena, even if those conceptualisations are questionably operationalised for the purposes of analysis. However, despite significant co-dependent toing and froing between considered concepts, a neat bio/social distinction is sustained.

Turning to the issues of how distinct components interact in cultural neuroscience, we can see an interesting similarity with the biopsychosocial model. This is evident in appeals to the psychological as a sort of overlap of distinct social and biological phenomena, in as much as the psychological is a co-manifestation of a person’s neurological and cultural characteristics. However, it also differs in important respects, simultaneously relying on an anthropological notion of ‘internalisation’ to explain how culture is inscribed on the brain (albeit affecting the biological via plasticity rather than sensitisation) and a neuroscientific notion of psychology as a product of neurophysiology. The ambiguity in this tripartite psychology is seemingly born of the amalgamation of distinct disciplines that do not readily align with one another, without explicitly addressing their discordances. Cultural neuroscience’s psychological interface (comprised of internalised culture and externalised neurons) is much grander than, and yet equally as puzzling as, the ‘experience’ of social defeat.

Before unpacking this psychological interface more particularly, it is worth briefly exploring what links our aforementioned disparate examples of biosocial psychiatric research. Echoing social defeat research, syndemics accounts for high rates of childhood asthma in violent neighbourhoods via ‘stress-promoted immune-system deregulation caused by living in a pervasive atmosphere of fear and the perceived threat of ever-present violence’ (Singer et al., 2017: 945). In a similar manner, the diathesis–stress
theory in early life exposure research suggests that ‘when biologically vulnerable individuals are confronted with adverse life experiences, the combination of the biological predisposition and stress associated with these experiences may exceed a certain threshold and catalyze the development of [anti-social behaviour]’ (Van Hazebroek et al., 2019: 170). This diathesis–stress theory is also influential in biosocial criminology, albeit in relation to criminal behaviour more broadly, whereby social environment interacts with biological predispositions via experiences of ‘stress’ (Rocque and Posick, 2017). Though similarly under-explicated, contemporary dementia prevention research hypothesizes that social engagement is negatively associated with dementia via ‘emotional and intellectual stimulation’ (Chern and Golub, 2019: 288). This body of work is notable in appealing to stimulating experiences rather than the stressful experiences that are prevalent in biosocial psychiatric research. That said, ‘stress’ has been proposed as a mediator between social circumstances and cerebral dysfunction in dementia (Fratiglioni, Paillard-Borg, and Winblad, 2004). Across these diverse examples, associations between measures of social stuff and biological stuff are bridged via brief appeals to experiential notions that are ill-defined, and yet which comprise substantive components of the biosocial mechanics proposed. Specifically, each appears to depict a sort of psychological interface through which the social gets under the skin and sometimes through which the biological gets out of the skin. It is to this psychological interface that we now turn our attention.

The psychological interface

Social defeat and cultural neuroscience differ considerably. In drawing on these traditions, we are purposefully using disparate examples to make our case, drawing out similar facets (Mason, 2011) between these different approaches. While dissimilar in several respects, it is evident that both the social defeat and cultural neuroscience traditions of biosocial psychiatric research share a foundational assumption that humans are made up of distinct types of biological and social stuff. The result is that there is no challenge to traditional biological/social binaries at an ontological level, but rather a claim that the two can coexist and complement each other, or indeed be translated into one another. Social defeat and cultural neuroscience can hence both be considered to be composite (see Ingold, 1998). It is important to note that these ontological commitments are not acknowledged explicitly in biosocial research, and are likely of little concern to the researchers, but they are nonetheless inherent to the models. This is perhaps significantly influenced by positivistic approaches to reality as being that which can be measured, though this is difficult in relation to some of the social phenomena (e.g. culture, marginalisation, etc.).

Each tradition approaches its task as one of uniting fundamentally different entities within a single overarching causative schema. This is typically enacted through bridging mechanisms, such as stress or information-processing biases in the cases of Selten’s and Chiao’s respective works. The apparent explanatory centrality and psychological aesthetic of these bridges leads us to question the status of the psychological as an unresolved concept that seems vital to these biosocial accounts without ever being explicated. Indeed, what is most deserving of critical reflection, we argue, when
reading the works of Selten and Chiao, is that the psychological is the most important, yet least explained, component of the biosocial processes they posit. In other words, these two examples, despite their many differences, conceptually rest upon an idea of a psychological interface, a bridge that links the complexities of social life to the complexities of (neuro)biology. For both, the psychological, or the experiential, performs a crucial role. For both, we are never told how this happens, nor what the psychological is.

Considering the biopsychosocial model as a comparator suggests that the term ‘biopsocial’ is simply the former title stripped of the ‘psycho’ caveat. However, as discussed in relation to the biopsychosocial, Engel’s model always struggled to define its psychological subject matter, and seemed to be chiefly concerned with biological and social concerns. Similarly, as discussed in the introduction, Weiss’ early 20th-century conceptualisation of the biosocial was an explicit attempt to make psychological phenomena more amenable to the scientific method by reclassifying them as either biological or social concerns. There has hence always been something of a notable psychological deficit within complementary psychiatric research, with scholars seemingly struggling, or simply not attempting, to address the nature and role of interiorised psychological phenomena such as experience, while simultaneously being unable to do away with their models’ indebtedness to that very same phenomena.

To think about why psychological phenomena have tended to be the crucial and yet weaker party in biosocial research, it is helpful to consider their status within social defeat and cultural neuroscience specifically. For each, psychological phenomena are most evident in terms of outcomes, as manifest in mental illness. Selten’s work on social defeat focuses on schizophrenia while Chiao’s cultural neuroscience is chiefly concerned with affective disorders. Both are cast in partially psychological terms in relation to their final expression. In this sense, the psychological emerges as a palpable indication of deeper biosocial processes. Besides this role as an outcome, the psychological is also invoked as a type of bridge. In the case of Selten’s social defeat, this psychological bridge is evident in the conceptualisation of stress, whereby discrimination is ‘experienced’ or generates a sense of ‘uncertainty’, which is transformed into molecular changes. Within Chiao’s cultural neuroscience, genetically constrained neural development engenders cognitive information-processing biases, rendering the person more receptive to certain types of sociocultural stimuli. Hence, this conceptualisation of the psychological interface is crucial to the respective research, as it enables conceptual movement back and forth between the biological and the social.

Noting these same traits, Ingold (1998) has characterised the role of psychological phenomena in biosocial research as a sort of bridging infrastructure. The mind is typically invoked as an ill-defined exchange through which the biological and the social interact. Ingold argues that it has been used in this way because it is partly reconcilable with the biological, being rooted in neurophysiology, and the social, being the vehicle of human experience. Psychology thus has one foot in each component half of the biosocial binary, but is not necessarily a distinct third entity in its own right.

Ingold’s account of the psychological as an ill-conceived entity that bridges the biological and the social does appear to explain its puzzling status in contemporary biosocial research. This observation is certainly in line with the argument that the psychological is often inserted out of necessity, rather than being carefully crafted with any intellectual
depth. It may therefore be the case that the supposed tying together of the component phenomena of biosocial accounts is reliant on remarkably underdeveloped imaginings of psychology, which are paid scant attention while simultaneously being made to do most of the explanatory labour.

There is also some statistical support for psychology’s role as an interface between the biological and the social. Based on a bibliometric cluster analysis (see Boyack, Klavans, and Börner, 2005), social neuroscientist Cacioppo (2007) has argued that psychology is a ‘hub science’ through which other scientific specialisms interact. Interestingly, in the resulting map (see Figure 3 in Boyack, Klavans, and Börner, 2005: 364) psychology lies at the centre of biomedicine, psychiatry, neuroscience, and sociology, as well as being positioned between the other hub disciplines of medicine and social science. Cacioppo concludes that this position at the intersection of medicine and social science is indicative of the importance of psychology to the sciences generally. We offer a slightly different interpretation, contending that psychology is a go-to means of nominally reconciling difficult-to-reconcile ideas. While the psychological is repeatedly employed as an interface between the biological and the social, this is often done in a fleeting and ethereal manner. It appears that a substantive body of biosocial research continues to get away with these brief invocations of underdeveloped psychological concepts, as models are repeatedly cited, reiterated, and built upon without any critical comment on those concepts.

It is also well known that despite psychology’s various disciplinary and scientific crises, since the 1930s, the discipline has consistently become more popular, more ingrained in societal practices and imaginaries. Rose (1996) has argued that the reason for the popularity of psychology as a discipline, and indeed psychological concepts, lies less in their scientific veracity, and more in the discipline’s practical applicability (through tests, measurements, and so forth) for ordering the lives of people and for intervening the practical, societal issues (Rose, 1996, 2008). In this sense, we might argue that what we have called the psychological interface and its appeals to notions of experience, perception, and so forth arises exactly because it is practically useful. The appeals to the psychological interface are practically useful because this is a form of ‘ontological gerrymandering’ (Woolgar and Pawluch, 1985), wherein conceptual boundaries are redrawn and one can shift away from troublesome concepts such as experience. In other words, appeals to experience, for instance, are useful practically, because everybody knows what experience/experiencing is, but nobody really knows – or agrees – what this is.

More broadly, the status and role of experience – especially ‘inner’ experience, consciousness, introspection – has been a central sticking point for the discipline (Overgaard, 2006). It is, then, not surprising that appeals to the psychological are frequently undertheorised. From the banishment of introspection and consciousness by the behaviourists in the early 20th century, to its gradual reintroduction via cognitive science in the 1960s, and later the embrace of phenomenology in some quarters of neuroscience (Costall, 2006; Overgaard, 2006), the theorisation and operationalisation of experience has been far from straightforward. Indeed, as theoretical psychologists such as Stenner have argued, we must ‘separate the psychic from the social, but also we cannot and ought not to separate...
them’ (Stenner, 2018: 13). For Stenner, there is a ‘foundational paradox’ to scientific psychology:

If the power of modern natural science lay in its ability to exclude the ‘inner’ or mentality from consideration, and to observe things purely from the outside, then a paradox is confronted by those who wish to study subjectivity objectively. This paradox was managed (and in fact mismanaged) by practically excluding questions of subjectivity and experience from the remit of psychology. (ibid.: 10)

If we accept Stenner’s arguments here, then what our analysis of the psychological interface reveals is a larger paradox of psychological science: that the objective study of the subject by necessity precludes the latter. In this article, we have argued that the implicit assumptions about the psychological interface, coupled with a notable lack of attention to those assumptions, recall old ideas of stimulus-response models, which are a problematic point of departure for understanding human life and behaviour. Using psychological phenomena as a shorthand – ‘experience’, for example – elides the difficult (but, importantly, the substantial and ongoing) debates about what experience is, how it may vary under different circumstances, and indeed how we ought to research it. The ‘experience’ of a rat that is lowered into the cage of a larger, more aggressive rat, and which is then attacked, is profoundly different from the ‘experience’ of the urban dweller. If biosocial accounts are to incorporate forms of psychological phenomena, especially as their core mechanics, then they must surely do so in a manner that engages with the complexities of those phenomena and the readily available disciplinary knowledges that are dedicated to those complexities.

The current approach to biosocial psychiatric research effectively sells psychology short in a manner that is equivalent to reducing the social to context or the biological to genes. Consider the argument (e.g. Rose, 1996, 2008) that psychology’s success is grounded in practical usefulness more than scientific veracity and the argument (e.g. Stenner, 2018) that the discipline of psychology is fundamentally paradoxical insofar as it seeks to study the ‘inner’, even as the ontological status of this ‘inner’ is perpetually in doubt. When these two arguments are taken into account, then the tendency to appeal to the psychological as an interface between ethereal sociality and some sort of neurological imprint is perhaps unsurprising. In this light, the interfacing of the psychological, its ontological bracketing, is a productive and useful move as it exactly removes difficult and persistent (and perhaps unanswerable) philosophical and methodological questions from the equation.

Ultimately, one might reasonably ask, ‘So what?’ Even if our analysis is correct, and that these instances of biosocial psychiatric research have ‘philosophical’ problems, then what does that actually matter to scientific practices? This is a good question. We would argue that perhaps the perennial uncertainty around areas of experience and of the psychological interface can actually be made productive. Rather than being considered philosophically ‘incorrect’, but to the scientific enterprise practically meaningless, we might consider how (potential) psychological bridges between the social and the biological raise new questions, new avenues for investigation. This is not to say that the research is ‘wrong’, but rather that there is a potential blind spot here, one that might perhaps
be productive to experiment with, to target, to explore further, difficult as it may be. The long-standing psychological problem of figuring out experience is perhaps an unsolvable philosophical problem, but it might simultaneously be an opportunity to identify problems that are often not even fully recognised as meaningful problems, as a first step towards better theorising, in this instance, the mechanics of psychiatric disorder.

Of course, this justification could fairly be criticised as somewhat idealist or naïve. In practice, a great deal of research explicitly black-boxes phenomena filed under ‘experience’ in order for the experiments to work. The reduction of complex phenomena for experimental purposes is widely documented in STS and is not inherently problematic. Indeed, scientific experimentation via reduction can be incredibly fruitful. From this perspective, the observations in this article are perhaps (hopefully) interesting but largely inconsequential. However, we offer a few caveats. First, reduction might become problematic in instances where an area of biosocial psychiatric research has so far failed to translate experimentation into effective real-world interventions. Assuming that the underlying rationale is alleviating suffering, then practical applicability is desirable, and by this measure much of the research discussed above has not yet succeeded. Greater psychological engagement might aid this. Of course, it might be entirely unhelpful, but it is an additional option that is at least worth pursuing. Second, to our reading of the literature, some of what we have described above moves beyond reduction towards eradication, whereby experience largely operates as a linking word with little behind it at all. This is certainly not true of all, or most, but of some. Third, we need not leap from a reduced category of ‘experience’ to a comprehensive phenomenological account of mental life. Some relatively minor progression of biosocial psychiatric research in the direction of the psychological might unlock new insights into a range of phenomena such as schizophrenia, affective disorders, dementia, and so on. Ultimately, if nothing more, we hope that this article provokes some critical reflection on the strange status of the psychological interface, operating as simultaneously vague but integral mechanics underpinning biosocial imaginings of psychiatric disorder.

Conclusion

In this article, we have shown how composite biosocial psychiatric research can attempt to bridge discrete biological and social categories of phenomena through appeals to a poorly defined psychological interface. Using the contemporary examples of social defeat research on schizophrenia and cultural neuroscience research on affective disorders, in comparison with the older biopsychosocial model, we have drawn much-needed critical attention to the often unremarked upon tendency for biosocial psychiatric research to invoke psychological notions as the vital mechanics of hypothetical processes in mental disorder, and yet to pay little attention to explaining or developing those psychic mechanics. We have sought to draw attention to this practice because it is potentially undermining important research in the human sciences, and is too often overlooked, unchallenged, or worse still, unreflectively replicated. We have argued that in some ways this tendency is a reflection of a longer trend in the history of psychology, wherein tricky questions around ‘experience’ and subjectivity have long been bracketed.
Naturally, much science and many scientists are unlikely to be concerned with the need to do justice to psychology and its heritage. Instead, our concern here is that a specific flaw is evident across several biosocial models and has become concealed in its own ubiquity – overlooked and recreated so readily that it has become something of a standard for the field, potentially impeding future research in a similar vein. Drawing attention to it opens the possibility that it can be addressed, at least partly. As noted, the bracketing out of complexities to facilitate experimentation is not inherently bad and is often very useful. However, in the instances discussed in this article, they have not yet proved useful. This matters for the same reasons that the research traditions themselves matter, namely that they attend to solving distressing human problems. Their improvement could hence feasibly result in the amelioration of forms of suffering. It could be that, for those approaches that already offer reasonable sociological and biological sophistication, an equally sophisticated appreciation of the psychological mechanics of their models represents a missing piece that enables the full realisation of a biosocial understanding of psychiatric disorders and subsequently its translation into practicable solutions. However, it could also be that a relatively minor engagement with psychological phenomena offers important new understandings. It is in everyone’s interest that biosocial psychiatric research should be as robust as is necessary for developing effective interventions.

Moving forwards, we offer some concluding suggestions for addressing this biosocial problem of weak psychological bridges. One option is that biosocial research might be improved by rejecting interiorised psychological phenomena such as experience altogether, as championed by Weiss, and instead doubling down on models that neatly integrate the biological and social components upon which they principally focus (a compartmentalisation of phenomena that perhaps warrants dedicated critical exploration in its own right). This would certainly deal with the problem of unsophisticated appeals to vague psychological notions. However, we are not aware of a convincing model of biosocial exchange that does not resort in some manner, however fleeting, to concepts such as experience, perception, internalisation or similar, and we are not convinced that the absolute rejection of the psychological would be a fruitful avenue for understanding human life. As a preferable option, such research might instead benefit from a more substantial engagement with the psychological as a category with equivalent intellectual prowess as the biological and the social, seeking to develop it beyond operationalisation as a sort of mysterious interface between two more important issues. To this end, the tools for the immediate advancement of biosocial psychiatric research are already at hand. While cultural neuroscience in particular busies itself with sophisticated sociological and biological scholarship, its psychological interface is rather wanting. Hence, robust engagement with contemporary psychological theory could potentially inspire major progress in the field. As we have highlighted towards the end of this article, psychology is alive to the puzzling nature of matters such as perception, generating vigorous debate, and an abundance of conceptualisations. It is in such scholarships that biosocial psychiatry may discover the intellectual resources to facilitate its progression into a new terrain of shared biological, sociological, and psychological understanding. Of course, more sophisticated theorisation carries no guarantee of consequent improvements in addressing psychiatric disorder, but given the ubiquity of the shortcoming and how readily available a potential solution is, we argue that it is certainly
a worthwhile consideration. Ultimately, if the psychological bridge is to remain at the heart of contemporary biosocial psychiatric research, then it surely warrants more sophisticated engagements with its core concepts and the considerable expertise that exists regarding those concepts.

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Note

1. Proponents of cultural neuroscience generally appeal to ‘cultural psychology’, but the tradition to which they refer is typically better characterised as ‘cross-cultural psychology’. Cultural psychology focuses on the emergence of minds within given cultural contexts, whereas cross-cultural psychology is interested in comparing psychological variations across nations, borders, or ‘cultures’. The former is rather anthropological in nature, while the latter is more reminiscent of mainstream quantitative psychological research (Shweder and Sullivan, 1993).

References

Albeck, D. S., McKittrick, C. R., Blanchard, D. C., Blanchard, R. J., Nikulina, J., McEwen, B. S., and Sakai, R. R. (1997) ‘Chronic Social Stress Alters Levels of Corticotropin-Releasing Factor and Arginine Vasopressin Mrna in Rat Brain’, Journal of Neuroscience 17(12): 4895–903.
Alleva, E. and Aloe, L. (1989) ‘Physiological Roles of Nerve Growth Factor in Adult Rodents: A Biobehavioral Perspective’, International Journal of Comparative Psychology 2(4): 213–30.
Benning, T. B. (2015) ‘Limitations of the Biopsychosocial Model in Psychiatry’, Advances in Medical Education & Practice 6: 347–52.
Björkqvist, K. (2001) ‘Social Defeat as a Stressor in Humans’, Physiology & Behavior 73(3): 435–42.
Boyack, K. W., Klavans, R., and Börner, K. (2005) ‘Mapping the Backbone of Science’, Scientometrics 64: 351–74.
Brown, G. W. and Harris, T. (1978) Social Origins of Depression: A Study of Psychiatric Disorder in Women. London: Tavistock.
Cacioppo, J. T. (2007, 1 September) ‘Psychology Is a Hub Science’, Observer, available at: https://www.psychologicalscience.org/observer/psychology-is-a-hub-science.
Cacioppo, J. T., Cacioppo, S., Capitanio, J. P., and Cole, S. W. (2015) ‘The Neuroendocrinology of Social Isolation’, Annual Review of Psychology 66: 733–67.
Cacioppo, J. T. and Hawkley, L. C. (2009) ‘Perceived Social Isolation and Cognition’, Trends in Cognitive Sciences 13(10): 447–54.
Cantor-Graae, E. and Selten, J. P. (2005) ‘Schizophrenia and Migration: A Meta-analysis and Review’, American Journal of Psychiatry 162(1): 12–24.
Chern, A. and Golub, J. S. (2019) ‘Age-Related Hearing Loss and Dementia’, *Alzheimer Disease and Associated Disorders* 33(3): 285–90.

Chiao, J. Y. and Ambady, N. (2007) ‘Cultural Neuroscience: Parsing Universality and Diversity Across Levels of Analysis’, in S. Kitayama and D. Cohen (eds) *Handbook of Cultural Psychology*. New York, NY: Guilford Press, pp. 237–54.

Chiao, J. Y. and Blizinsky, K. D. (2009) ‘Culture–Gene Coevolution of Individualism–Collectivism and the Serotonin Transporter Gene’, *Proceedings of the Royal Society B: Biological Sciences* 277(1681): 529–37.

Chiao, J. Y., Hariri, A. R., Harada, T., Mano, Y., Sadato, N., Parrish, T. B., and Iidaka, T. (2010) ‘Theory and Methods in Cultural Neuroscience’, *Social Cognitive and Affective Neuroscience* 5(2–3): 356–61.

Costall, A. (2006) ‘“Introspectionism” and the Mythical Origins of Scientific Psychology’, *Consciousness and Cognition* 15(4): 634–54.

Elliott, R. M. (1931) ‘Albert Paul Weiss: 1879–1931’, *American Journal of Psychology* 43: 707–9.

Engel, G. L. (1977) ‘The Need for a New Medical Model: A Challenge for Biomedicine’, *Science* 196(4286): 129–36.

Engel, G. L. (1980) ‘The Clinical Application of the Biopsychosocial Model’, *American Journal of Psychiatry* 137(5): 535–44.

Fincher, C. L., Thornhill, R., Murray, D. R., and Schaller, M. (2008) ‘Pathogen Prevalence Predicts Human Cross-cultural Variability in Individualism/Collectivism’, *Proceedings of the Royal Society B: Biological Sciences* 275(1640): 1279–85.

Fitzgerald, D. and Callard, F. (2015) ‘Social Science and Neuroscience Beyond Interdisciplinarity: Experimental Entanglements’, *Theory, Culture & Society* 32(1): 3–32.

Fitzgerald, D., Rose, N., and Singh, I. (2016) ‘Revitalizing Sociology: Urban Life and Mental Illness Between History and the Present’, *British Journal of Sociology* 67(1): 138–60.

Fletcher, J. R. and Birk, R. H. (2020) ‘From Fighting Animals to the Biosocial Mechanisms of the Human Mind: A Comparison of Selten’s Social Defeat and Mead’s Symbolic Interaction’, *Sociological Review* 68(6): 1273–89.

Fratiglioni, L., Paillard-Borg, S., and Winblad, B. (2004) ‘An Active and Socially Integrated Lifestyle in Late Life Might Protect Against Dementia’, *The Lancet Neurology* 3(6): 343–53.

Ghaemi, S. N. (2009) ‘The Rise and Fall of the Biopsychosocial Model’, *British Journal of Psychiatry* 195(1): 3–4.

Ghaemi, S. N. (2010) *The Rise and Fall of the Biopsychosocial Model: Reconciling Art and Science in Psychiatry*. Baltimore, MD: John Hopkins University Press.

Gupta, V., Hanges, P. J., and Dorfman, P. (2002) ‘Cultural Clusters: Methodology and Findings’, *Journal of World Business* 37(1): 11–15.

Hammels, C., Pishva, E., De Vry, J., van den Hove, D. L., Prickaerts, J., van Winkel, R., Selten, J.-P., Lesch, K.-P., Daskalakis, N. P., Steinbusch, H. W. M., Van Os, J., Kenis, G., and Rutten, B. P. F. (2015) ‘Defeat Stress in Rodents: From Behavior to Molecules’, *Neuroscience & Biobehavioral Reviews* 59: 111–40.

Han, S., Northoff, G., Vogele, K., Wexler, B. E., Kitayama, S., and Varnum, M. E. (2013) ‘A Cultural Neuroscience Approach to the Biosocial Nature of the Human Brain’, *Annual Review of Psychology* 64: 335–59.
Ingold, T. (1998) ‘From Complementarity to Obviation: On Dissolving the Boundaries Between Social and Biological Anthropology, Archaeology and Psychology’, Zeitschrift für Ethnologie 123(1): 21–52.

Ingold, T. (2013) ‘Prospect’, in T. Ingold and G. Palsson (eds) Biosocial Becomings: Integrating Social and Biological Anthropology. Cambridge: Cambridge University Press, pp. 1–21.

Kim, H. S. and Sasaki, J. Y. (2014) ‘Cultural Neuroscience: Biology of the Mind in Cultural Contexts’, Annual Review of Psychology 65: 487–514.

Kirkbride, J. B., Morgan, C., Fearon, P., Dazzan, P., Murray, R. M., and Jones, P. B. (2007) ‘Neighbourhood-Level Effects on Psychoses: Re-examining the Role of Context’, Psychological Medicine 37(10): 1413–25.

Koolhaas, J. M., Coppens, C. M., de Boer, S. F., Buwalda, B., Meerlo, P., and Timmermans, P. J. (2013) ‘The Resident-Intruder Paradigm: A Standardized Test for Aggression, Violence and Social Stress’, JoVE 77: e4367.

Livingston, G., Huntley, J., Sommerlad, A., Ames, D., Ballard, C., Banerjee, S., Brayne, C., Burns, A., Cohen-Mansfield, J., Cooper, C., Costafreda, S. G., Dias, A., Fox, N., Gitlin, L. N., Howard, R., Kales, H. C., Kimigaki, M., Larson, E. B., and Ogunkiiyi, A. …, Mukadam, N. (2020) ‘Dementia Prevention, Intervention, and Care: 2020 Report of the Lancet Commission’, The Lancet 396(10248): 413–46.

Lumsden, C. J. and Wilson, E. O. (1981) Genes, Mind and Culture: The Coevolutionary Process. Cambridge, MA: Harvard University Press.

Manning, N. (2019) ‘Sociology, Biology and Mechanisms in Urban Mental Health’, Social Theory & Health 17(1): 1–22.

March, D., Morgan, C., Bresnahan, M., and Susser, E. (2008) ‘Conceptualising the Social World’, in K. McKenzie, C. Morgan, and P. Fearon (eds) Society and Psychosis. Cambridge: Cambridge University Press, pp. 41–57.

Mason, J. (2011) ‘Facet Methodology: The Case for an Inventive Research Orientation’, Methodological Innovations Online 6(3): 75–92.

Meloni, M. (2014) ‘How Biology Became Social, and What It Means for Social Theory’, Sociological Review 62(3): 593–614.

Meloni, M. (2019) Impressionable Biologies: From the Archaeology of Plasticity to the Sociology of Epigenetics. New York, NY: Routledge.

Meloni, M., Williams, S., and Martin, P. (2016) ‘The Biosocial: Sociological Themes and Issues’, Sociological Review Monograph 64(1): 7–25.

Morgan, C. (2008) ‘Social Science, Psychiatry and Psychosis’, in K. McKenzie, C. Morgan, and P. Fearon (eds) Society and Psychosis. Cambridge: Cambridge University Press, pp. 25–40.

Morgan, C., Knowles, G., and Hutchinson, G. (2019) ‘Migration, Ethnicity and Psychoses: Evidence, Models and Future Directions’, World Psychiatry 18(3): 247–58.

Munafo, M. R., Brown, S. M., and Hariri, A. R. (2008) ‘Serotonin Transporter (5-HTTLPR) Genotype and Amygdala Activation: A Meta-analysis’, Biological Psychiatry 63(9): 852–7.

Murray, R. M. (2017) ‘Mistakes I Have Made in My Research Career’, Schizophrenia Bulletin 43(2): 253–6.

Niewöhner, J. and Lock, M. (2018) ‘Situating Local Biologies: Anthropological Perspectives on Environment/Human Entanglements’, BioSocieties 13(4): 681–97.

Overgaard, M. (2006) ‘Introspection in Science’, Consciousness and Cognition 15(4): 629–33.
Prior, L., Manley, D., and Sabel, C. E. (2019) ‘Biosocial Health Geography: New “Exposomic” Geographies of Health and Place’, Progress in Human Geography 43(3): 531–52.

Ramsden, E. (2012) ‘Rats, Stress and the Built Environment’, History of the Human Sciences 25(5): 123–47.

Rocque, M. and Posick, C. (2017) ‘Paradigm Shift or Normal Science? The Future of (Biosocial) Criminology’, Theoretical Criminology 21(3): 288–303.

Rose, N. (1996) Inventing Our Selves: Psychology, Power, and Personhood. Cambridge: Cambridge University Press.

Rose, N. (2008) ‘Psychology as a Social Science’, Subjectivity 25: 446–62.

Rose, N. (2012) ‘The Human Sciences in a Biological Age’, Theory, Culture & Society 30(1): 3–34.

Rose, N. and Abi-Rached, J. (2013) Neuro: The New Brain Sciences and the Management of the Mind. Princeton, NJ: Princeton University Press.

Selten, J. P. and Cantor-Graae, E. (2004) ‘Schizophrenia and Migration’, in W. F. Gattaz and H. Häfner (eds) Search for the Causes of Schizophrenia. Berlin: Springer, pp. 3–25.

Selten, J. P. and Cantor-Graae, E. (2005) ‘Social Defeat: Risk Factor for Schizophrenia?’, British Journal of Psychiatry 187(2): 101–2.

Selten, J. P., Cantor-Graae, E., and Kahn, R. S. (2007) ‘Migration and Schizophrenia’, Current Opinion in Psychiatry 20(2): 111–15.

Selten, J. P., Van der Ven, E., Rutten, B. P., and Cantor-Graae, E. (2013) ‘The Social Defeat Hypothesis of Schizophrenia: An Update’, Schizophrenia Bulletin 39(6): 1180–6.

Selten, J. P., Van Os, J., and Cantor-Graae, E. (2016) ‘The Social Defeat Hypothesis of Schizophrenia: Issues of Measurement and Reverse Causality’, World Psychiatry 15(3): 294–5.

Sgoifo, A., Koolhaas, J., de Boer, S., Musso, E., Stilli, D., Buwalda, B., and Meerlo, P. (1999) ‘Social Stress, Autonomic Neural Activation, and Cardiac Activity in Rats’, Neuroscience & Biobehavioral Reviews 23(7): 915–23.

Shweder, R. A. and Sullivan, M. A. (1993) ‘Cultural Psychology: Who Needs It?’, Annual Review of Psychology 44(1): 497–523.

Singer, M., Bulled, N., Ostrach, B., and Mendenhall, E. (2017) ‘Syndemics and the Biosocial Conception of Health’, The Lancet 389(10072): 941–50.

Stefanski, V. (2000) ‘Social Stress in Laboratory Rats: Hormonal Responses and Immune Cell Distribution’, Psychoneuroendocrinology 25(4): 389–406.

Stefanski, V. and Engler, H. (1999) ‘Social Stress, Dominance and Blood Cellular Immunity’, Journal of Neuroimmunology 94(1–2): 144–52.

Stenner, P. (2018) Liminality and Experience: A Transdisciplinary Approach to the Psychosocial. New York, NY: Springer.

Tidey, J. W. and Miczek, K. A. (1997) ‘Acquisition of Cocaine Self-Administration After Social Stress: Role of Accumbens Dopamine’, Psychopharmacology 130(3): 203–12.

Van Hazebroek, B. C., Wermink, H., van Domburg, L., de Keijser, J. W., Hoeve, M., and Popma, A. (2019) ‘Biosocial Studies of Antisocial Behavior: A Systematic Review of Interactions Between Peri/Prenatal Complications, Psychophysiological Parameters, and Social Risk Factors’, Aggression and Violent Behavior 47: 169–88.

Vassos, E., Pedersen, C. B., Murray, R. M., Collier, D. A., and Lewis, C. M. (2012) ‘Meta-analysis of the Association of Urbanicity With Schizophrenia’, Schizophrenia Bulletin 38(6): 1118–23.

Weiss, A. P. (1929) A Theoretical Basis of Human Behavior (2nd ed.). Oxford: Adams.
Weiss, A. P. (1930) ‘The Biosocial Standpoint in Psychology’, in C. Murchison (ed.) *International University Series in Psychology. Psychologies of 1930*. Worcester, MA: Clark University Press, pp. 301–6.

Woolgar, S. and Pawluch, D. (1985) ‘Ontological Gerrymandering: The Anatomy of Social Problems Explanations’, *Social Problems* 32(3): 214–27.

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