EuJAP | Vol. 17 | No. 2 | 2021
UDC: 122:159.9]001
https://doi.org/10.31820/ejap.17.2.7

THE BIOPSYCHOSOCIAL MODEL OF HEALTH AND DISEASE: RESPONSES TO THE 4 COMMENTARIES

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Discussion – Received: 01/10/2021 Accepted: 21/10/2021

ABSTRACT

I respond to the 4 commentaries by Awais Aftab & Kristopher Nielsen (A&N), Hane Htut Maung (HHM), Diane O'Leary (DO'L) and Kathryn Tabb (KT) under 3 main headings: "What is the BPSM really?" & Why update it?; "Is our approach foundationally compromised?", and finally, "Antagonists or fellow travellers?".

Keywords: *Biopsychosocial model; causation; George Engel; information*

Preamble

First and foremost, I would like to thank the commentators—Awais Aftab & Kristopher Nielsen (A&N this issue), Hane Htut Maung (HHM this issue), Diane O'Leary (DO'L this issue) and Kathryn Tabb (KT this issue)—for the generous giving of their time to critical commentary of Derek Bolton & Grant Gillett's proposed update of the Engel's (1997) Biopsychosocial Model (B&G). I should say that while the book was co-written, this Reply is written by DB only, so the text varies between plural 'we' for the B&G book, and singular 'I' for the Reply. Our proposed update of the BPSM is in the spirit of trying to get things as straight as we can about the conceptual foundations of health, disease, and healthcare. I thank the commentators for their generous comments about the book and for their critiques on how things could be improved. There are some common and some distinctive themes in the critiques, and I will respond to them under 3 main headings: "*What Is the BPSM Really?*" & *Why Update It?*; "*Is Our Approach Foundationally Compromised?*", and finally, "*Antagonists or Fellow Travellers?*". I have aimed to include supplementary material (additional to what is in B&G) where relevant.

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1. What Was or is the BPSM Really? And Why Update It?

1.1. Was Engel Interested in Causes?

A&N highlight that biopsychosocial causation, while the main problem in B&G, was not Engel's main problem, indeed they suggest that it may not have been one of his problems at all (p. 7). At one level, this is about terminology; "causation" is semantically linked to many other expressions in the health sciences and therapeutics such as "factors" and "influences". So for example, Engel's (1977) list of what the biomedical model fails to take into account includes, quoted by A&N (p. 8-9): "for some conditions such as schizophrenia and diabetes, the effect of conditions of living on onset, presentation and course"—and we take this to refer to causal risks for onset and risk/protective factors (causally) affecting course, putting the issues squarely in the areas of epidemiology and clinical therapeutics. Another connected example, A&N propose that:

> The matters that preoccupy Engel are more to do with psychosocial influences in the form of illness interpretation and presentation, sick role, seeking or rejection of care, the doctor-patient therapeutic relationship, and role of personality factors and family relationships in recovery from illness, etc. (Aftab and Nielsen this issue, 9)

But presumably "*influences*" = something like "*make a difference to*" = "*has a causal role in*".

A&N present a convincing case that one of Engel's main and general concerns was to bring many aspects of the psychological, social dimensions of illness including the doctor-patient relationship within the realm of medical and scientific inquiry. I agree with this, but suggest that this aspiration relies on the working assumption that these dimensions are causally relevant to health outcomes of interest. This is because science, so far as I understand it, is basically concerned with causes, and this is especially so for the applied sciences that aim to make a difference. To put it briefly, healthcare will take an interest in e.g. subjective accounts of illness if it makes a difference to something relevant, e.g. to agreement on whether there is a need to treat, and how; or will take interest in social context of living if it makes (or might make) a difference to e.g. falls at home and emergency admissions; or an interest in the quality of doctor-patient communication if it affects continuing trust, attendance and acceptability of treatment; and so on. As I read Engel, much of what he says on this issue was with the intention of rejecting the dichotomy between medicine as science and medicine as 'art' (Bolton 2020).

However, this project relies on psychosocial/interpersonal factors making a difference to relevant health outcomes. In other words, this strand of Engel's BPSM is the proposal that the causal processes (factors or influences) involved in disease and healthcare are not limited to the biological, but involve the whole person in their social/interpersonal context, and, as such, they are amenable to scientific enquiry.

1.2. Was the BPSM Ever a Model?

A&N reiterate the criticisms of Nassir Ghaemi and others to the effect that the BPSM is not a model and is of no clinical or scientific value (p. 10-11). I don't want to insist that it is a model. It is probably no more of a model than the model with which Engel contrasted it, the biomedical model (BMM). Both expressions, and probably any others that summarise complex foundational issues in a word or two (such as also 'biological psychiatry', or 'phenomenology') lend themselves to various kinds of uses ranging between slogan-like and substantially theorised, with being a shorthand for a theory somewhere in between. A theorised version of the BMM would include core concepts and principles of the biomedical sciences, along with basic research and therapeutic paradigms. A theorised version of the BPSM would be the same for the biopsychosocial sciences, and this is what we attempted in B&G. We defined some core ontological and causal features of the three relevant domains and their interactions (contrast the BMM that has only one relevant domain), illustrated by some new paradigmatic biopsychosocial health-related pathways, such as those involving chronic stress and pain perception. We emphasised the theory of causal interactions between the three domains, because they are traditionally so problematic, as well as because causal explanation is central to science and its ontology.

A&N repeat Nassir Ghaemi's charge that the BPSM helped everybody to win, linked to the fact that it had no substantial scientific content (p. 10). I suspect there may be a difference here in the way that the BPSM has played out in the US and the UK. While in the US there may have been a tendency to use the BPSM as a way of being inclusive and open-minded about causes and cures, the more usual perspective in the UK seems to have been that the BPSM is more a matter of empirical data from particular studies, for example in social epidemiology and studies of stress (see e.g. White 2005). Certainly UK colleagues of mine showed some surprise at Nassir Ghaemi's interpretation of the BPSM and one UK reviewer, Julian Leff, did implicate UK/US differences (Leff 2010). This issue is probably linked to the history of "pluralism" on which more below in section 3.3.

1.3. Something's Wrong Somewhere However

Insofar as the BPSM was or has been used as a half-baked attempt at a model that served mainly to reduce uncertainty and make everybody happy, then by all means it doesn't warrant updating, just exposing and moving on. This view, however, does not sit well with the popular proposal that, nevertheless, it serves a valuable educational function, endorsed (though with apparent ambivalence) by A&N (pp. 11-13).

It seemed to us when we embarked on B&G that it was no good at all having these three propositions all being endorsed together:

(1) BPSM is the most popular model (often observed, including by HHM is his opening sentence "The [BPSM] (…) is perhaps the most widely accepted model of health and disease in contemporary medicine.")

(2) However, it is philosophically, scientifically and clinically useless—not a model at all

(3) However, it's useful in education

The combination of these three positions in the literature seemed to demand some work; doing nothing with the conjunct (1) & (2) & (3), as we saw it, was not an option.

If (2) is correct we need to abandon (1) & (3) ASAP; or we accept and retain (1) & (3), and refute or remedy (2)—and it was in this spirit of this second option that we undertook to update the BPSM.

1.4. Engel's Vision and the Value of the BPSM

At the beginning of her paper, KT uses a metaphor of psychiatry being buffeted about by centrifugal and centripetal forces, adapted from Scott Lilienfeld's paper (2014) on the DSM-5, and recognizes the potential value of the BPSM as providing a unifying, 'centripetal' force (pp. 7). KT goes on to discuss centrifugal forces in psychiatry including specialisms, by condition, by profession, by tradition and orientation. Importantly, there is sometimes conflict between specialisms, potentially leading to confusion for end users. The problem gets bigger when splitting occurs, when one side doesn't envisage the other, when there is no perceived whole, whether this be a person, healthcare, or health science. Centripetal forces, by contrast, see a conceptual unity, replacing splitting by something more holistic, and KT sees Engel's (1977) BPSM as, perhaps, the most notable

centripetal project (*loc. cit*.). I agree with that, and would add that its biggest message in this regard is not so much centripetalism within psychiatry (though this is probably an implication), but centripetalism across healthcare as a whole, positing a unity and common involvement of somatic and psychological processes.

Linked to its centripetal force, KT correctly observes that Engel's BPSM project drew on the systems theory in vogue at the time (p. 10). I suggest, however, that this was not just a sign of a temporary fashion, but was more a foretaste, a vision of what was coming: the increasing use of systems theoretic concepts and principles within and across many fields. The systems theory approach is closely linked to the acceleration of inter-disciplinary research and problem-solving programmes over recent decades, providing some general and integrating concepts and principles. In Margaret Boden's typology of interdisciplinarity, the highest levels are 'generalising' and 'integrated', involving a unified single theoretical perspective and integration around shared themes and questions (Boden 1999; see also Strijbos 2010, and Committee on Facilitating Interdisciplinary Research, Committee on Science, Engineering, and Public Policy 2004).

This is just what we were aiming at in B&G: a unified theoretical perspective and common themes (constructs and principles), relevant to health and disease, throughout the biological, psychological, and social sciences. We supposed that the BPSM could only be a truly interdisciplinary framework, able to accommodate the many kinds of factors now known to be implicated in health and disease, by having a common set of constructs and principles that operate within and between previously disparate domains. Further, we believed that, as Engel foresaw, the required set of constructs were those in systems theory, such as *function*, *design*, *ends*, *feedback*, *communication/information*, *regulation*, and *control*. Since the 1970s the systems theory approach has developed in many existing and new sciences, applied to functional structures, natural or artificial, from biology to engineering to models of social organisations, criss-crossing previous disparate domains, underpinning interdisciplinarity (see e.g. Strijbos 2010).

In fact, in the relevant recent history of ideas, there is a direct line to be traced from Schrödinger's new and original definition of life, used in B&G to characterize biology, to Engel's (1977) paper, via von Bertalanffy's General System Theory (1968). Schrödinger's work was cited by von Bertalanffy, in turn cited by Engel as a key example of the then new systems approach. Originally proposed for biology, the new systems perspectives were fast extended to cover psychological and social systems,

organised in hierarchies of complexity, from cells to societies. Engel was among those quick to recognise the relevance of these new systems perspectives to health, disease, and healthcare, along with contemporaries such as Alan Sheldon (1970), Ervin Laszlo (1972) and Howard Brody (1973). Engel used the name "biopsychosocial model" in his paper, explicitly announcing it as a new model for medicine, readily interpretable as an extension of biomedicine—and this is the name that caught on, to become now the most widely accepted model. This was a background reason for us wanting to retain the name "BPSM": the belief that its intellectual history was substantial, valid, and visionary.

By all means, along with the name came its accumulated baggage, and several colleagues and pre-publication reviewers advised that we jettison both—the name and its baggage—and propose an explicitly novel theory. However, as is well-known and noted above, the name *BPSM* is still a leading currency. We supposed that this points to the intellectual need to update it and validate the BPSM, rather than abandon it as intellectually vacuous, which is not only hard to square with its being educationally useful, but also, as suggested above, does not recognize its solid foundations.

1.5. What Moves Healthcare Mountains? Metaphysics As Continuous with Science

As noted above, KT discusses centripetal versus centrifugal forces in psychiatry, and sees the BPSM as a centripetal project, but her main concerns in her paper are the centrifugal forces that support the BMM, which she identifies as socio-economic-political (Tabb this issue, sec. 3). Given this reasonable assumption that such forces are important maintaining factors for the BMM, KT then reasonably infers that as such they are unlikely to be affected by a metaphysical argument, which she supposes to B&G to be.

In response to this I would say that the argument in B&G is not metaphysical but is meant to be scientific; actually, more accurately put, the intention is to operate in the dynamic space where metaphysics and scientific theory, and hence also data, merge. In other words, B&G buys into the idea, common in much 20th century philosophy, that philosophy (as metaphysics) is continuous with science, construed broadly as empirical knowledge. I will not spend time on this complicated issue here, but references include Quine's (1951) famous rejection of two dogmas of empiricism, and, in a different way, Lakatos' (1970) highly sophisticated philosophy of science. Importantly, metaphysics so construed is not a permanent set of truths but changes from time to time and place to place.

It undergoes major transformations, shifts in core theory (in Lakatos' 1970 terminology) or paradigm shifts (in Kuhn's 1962 terminology). This is what B&G is about, new (or relatively new) ideas in the life and human sciences that underpin the BPSM, such as Schrödinger's new characterisation of biological organisms in terms of decreasing entropy, the appearance of code in biology, AI, cognitive psychology, embodied cognition, agency, recognition of social recognition and social status *vs.* social disqualification and exclusion as processes that affect health and disease.

As this last example illustrates, interwoven with these deep theory shifts are new technologies and empirical findings, and it is these, I believe, that can move healthcare mountains—over time.

For example, I once heard the opinion that Aaron Beck and colleagues' decision to trial their new CBT for depression against meds, as being truly inspired, because, when the psychotherapy was found to outperform the pharmacotherapy (Rush et. al. 1977), it made the medical community sit up and pay attention. The data scored a reasonably direct hit on the biomedical model that envisaged biological causation only. The rest—the massively increased use of psychological therapies in healthcare systems––is recent history.

Empirical work in epidemiology has also been critical in showing the need for a broader biopsychosocial model. The new social epidemiology has shown that various forms of social exclusion, not only from biological necessities but also exclusion from psychological and psychosocial necessities, such as recognition, security, and civil rights—is bad for your health.

Here are some other, emerging candidates of research programmes closer to core biomedicine than the examples above, in cardiology and surgery. In cardiology, studies suggest that about three quarters of patients referred to rapid access cardiology clinics have non-cardiac chest pain or other symptoms, while, or but, commonly there is no management protocol for these patients and they are discharged, often to seek assessment or treatment again later (Tenkorang et al. 2006; Sekhri et al. 2007; Debney and Fox 2011; Chambers et al. 2014; Lenderink and Balkestein 2019). In surgery, there is increasing evidence that for some presentations dominated by pain, surgical procedures do not outperform placebo (Wartolowska et al. 2014; Jonas et al. 2015; Louw et al. 2017). These emerging findings appear in the context of new models of pain and subsequent new treatments. In brief, the perception and severity of pain, while typically localized in a specific part of the body, is now understood to be only partly,

and sometimes not at all, associated with local damage, but also involves higher cortical pathways processing information about the meaning and consequences of the pain for the person's life, potentially modifiable by psychosocial interventions such as psychological therapy and neuroscience education programmes (Quartana et al. 2009; Edwards et al. 2016; Andias et al. 2018). Bearing in mind that pain and associated distress and impairment of functioning are major drivers of service use, these emerging findings are of potential massive interest to healthcare provision and health economics.

To sum up, if the question is posed: what brings about major shifts in practices and great institutions such as healthcare?—then the answer is going to be complicated. Same goes for a closely related question: what kinds of factors are barriers to change? KT notes that major factors maintaining the BMM include social, cultural, economic and professional interests, noting that Engel said as much, and then infers that metaphysical considerations are unlikely to move such things. This inference looks completely right, if 'metaphysics' is understood as an exercise in the academy, in departments of philosophy, divorced from scientific theory and data. But B&G never intended this. We see the move towards a biopsychosocial framework in the health sciences, therapeutics, and epidemiology as being fundamentally a scientific paradigm shift (or series of interconnected paradigm shifts), driven by deep theory changes in combination with new empirical data. It may be that, as indicated previously (sec. 1.2.), interpreting the BPSM as a scientific project—in the broad sense including deep theory, new technologies and empirical findings—as opposed to metaphysics, or ideology, could be an interpretation more common in the UK than in the US.

KT argues for the importance of bioethics in advocating for improvements in healthcare (Tabb this issue, sec. 4) and many of her points I would agree with. I would add, however, that commonly the choice between two courses of action is based not only on the values assigned to the possible outcomes, but also on data-sensitive beliefs about how these outcomes are best likely to be achieved. Especially, whether a biomedical approach is the best way forwards or a biopsychosocial approach, or just psychosocial, will depend partly on what outcomes are desired, but also on empirical evidence about probabilities of how best to achieve them. This applies at every level, from choice of individual treatment, to choice of population level prevention programmes (options include doing nothing), to decisions on research funding priorities.

2. Is Our Approach Foundationally Compromised?

Having outlined above the intended rationale, purpose and method of B&G, the question arises whether and how far it worked out. The commentators present several major challenges to the B&G project.

2.1. Muddle about Dualism?

DO'L proposes that the BPSM always has been contradictory because on the one hand it separates the biological and the psychological, while on the other hand it rejects dualism, fudging this by inadequate definition of dualism, in the original and in B&G (pp. 8-10). She proposes that this contradiction is already in the BMM, and it transfers to the BPSM. She notes the complexity and multiple interpretations of key terms involved in defining dualism, physicalism, and reductionism (pp. 9-10).

We supposed in B&G, staying close to Engel's text, that he charged the BMM with being dualistic and committed to physicalistic reductionism. We interpreted this as meaning, briefly, that BMM is committed to ontological dualism and causal-explanatory reductionism, i.e., to the view that body and mind are ontologically distinct, but that all causing takes place at the physical level, especially that there is no causing of bodily events by mental events. This interpretation involves no contradiction between dualism and physicalist reductionism. There would be a contradiction in affirming both dualism and physicalist ontological reduction, but we don't interpret BMM as being ontologically reductionist, only causal-explanatory reductionist. The contrast is then with the BPSM, which is not explanatory reductionist, but envisages causal interactions within and between all of its three levels or domains. By all means it would be possible then to maintain that the three levels or domains were all ontologically separate, but then good luck with trying to make sense of causal interactions between them. Rather, the coherent shift is to suppose that causal interactions between the three levels of domain is possible because they are in the same ontological space, and hence our proposal that BPSM embraces the current science of embodied and embedded mind, as well as health and disease relevant aspects of the social sciences and the environmental sciences.

2.2. Clinical Utility and the "Psychosomatic" Conditions

DO'L goes on in her commentary to discuss the clinical utility of the BPSM, especially but not only for conditions that expose the unhelpful effects of dualism on healthcare, namely the so-called "psychosomatic" conditions (pp. 15-16). She expresses approval for aligning the BPSM with

evidence-based medicine. In B&G we supposed this to be now the obvious place to look for clinical guidance; substantial evidence from clinical trials and systematic reviews is available to us, unlike to Engel when he formulated the BPSM. On the other hand, DO'L criticizes B&G for placing too much faith in clinical guidance (p. 14). However, we had no intention of suggesting that clinical decision-making can be read off from clinical guidelines alone, the evidence for which is always partial, provisional, and selective (depending on the designs of the trials that have been done), without detailed history-taking and accounting for individual features of the presentation. So far as I know this crucial caveat is integral to EBM, even if there is a risk of it getting lost in practice.

However, clinical practice and the clinical studies and trials that guide it are only as good as the nosology, and as noted above, DO'L focuses particularly on the important clinical categories linked to unhelpful dualism. While there been many nosological problems and debates within physical and psychological medicine, probably none have been as conceptually problematic as those about conditions that do not fit into either of those two kinds but fall somewhere in-between. These are the called-by-many-names 'psychosomatic' conditions, themselves comprising many kinds, and, as DO'L points out, accounting for a high proportion of health conditions (p. 14). People with these conditions, associated with varying levels of distress and impairment of functioning, can be transferred between general hospitals and neurological, psychiatric or psychological clinics, too often falling between them. One aspect of this unfortunate state of affairs is the dualism that has permeated healthcare, separating the biomedical study and treatment of conditions below the neck, roughly, with neurology, psychiatry and psychology between them sharing, more or less harmoniously, the brain and mind. At the same time, the mental well-being aspects of physical health conditions have less visibility, and the same for the somatic aspects of psychiatric conditions. The continuing and probably increasing popularity of the BPSM belongs with a move towards more holistic healthcare. An important aspect of this are the new models of pain, distress and associated impairment, implicating central, not only peripheral, involvement—noted previously in section 1.5 as potentially contributing to changing healthcare practice.

2.3. Is Biological Information Still Problematic?

HHM and A&N both emphasise that the presumed normative, semantic characterization of biological information is a problematic foundation for B&G's proposed update of the BPSM. There is a substantial philosophical literature which finds such a construct problematic in biology as opposed to psychology. As A&N (p. 18) remark, we are unlikely to settle this

problem here and now, but I will summarise some aspects of the rationale why B&G proceeded in this way, and address some of the criticisms they make.

Firstly, in B&G we purposely made *regulation* and *regulatory mechanism* the primary characterization of what we suggest is a new kind of science in biology; rather than fronting the more familiar 'information-processing'. This was partly to work around the familiar philosophical objections to biological information-talk, but it was also in the belief that biology has actually moved on since the original information-processing revolution that started in the 1950s/1960s following discovery of the genetic code, and is now more involved with regulation and regulatory mechanisms throughout biological systems. These processes and mechanisms are visible: physical-chemical processes stop/start, increase/decrease; caused by observable events that lend themselves to descriptions such as 'switches' and 'gates' that e.g. increase or decrease concentration of catalysts. *Information flow* by contrast is a more abstract construct—you can't see it—and the next step of supposing that what is 'flowing' has semantic, normative content, seems to turn this abstraction into a philosophical error (horror)—at least it does when certain philosophical assumptions about content are being made, on which more below. However, as this new biological science has developed, the concept of information is not, or does not have to be seen as, doing the conceptual heavy lifting; rather it appears rolled up in a whole family of interconnected constructs, along with coding, signalling, feedback, function, and so on. This is evident in, for example, the relatively new and rapidly expanding subfields of molecular biology, cell signalling and genetic regulatory networks. As part of these developments, the construct of information is itself changing, shifting towards *programming* and *instructions*, for e.g. building complex molecules, or for the operation of regulatory mechanisms. In these theory-shifts, it is less easy to identify information-talk as having semantic content. I mean, while it is easy to assume that information is supposed to have content 'that p', where 'p' is a proposition with a truth-value expressible in language, there is no corresponding easy assumption of true/false propositional content when 'information' has the sense of *instruction*. Instructions are not true/false, though they can be e.g. normal/abnormal, or they can lead to the wrong result, in the circumstances, and they can be issued by the wrong agent. Here the reference is to the pervasive normativity in current biological models, evident in constructs such as *dysregulation*, *error*, *mutation*, *correction*, *deception/mimicry*, etc., but which is not best interpreted in terms of true/false semantic content. As to the grounds of this biological normativity, they are fundamentally to do with staying alive or dying, at the individual and/or species level.

Let me return to the point that biological semantic information or normativity is problematic only if certain philosophical assumptions about content/normativity are being made. HHM makes the criticism (p. 12), that while concepts of informational content and normativity are valid in the psychosocial domains, they are problematic in the biological domain at the sub-personal level. But apart from being familiar in folk usage, what is the metaphysics or science behind this claim? This is probably the same question as: what is the metaphysically acceptable *literal* meaning of 'informational content' and 'normativity', such that application of these terms to biological, sub-personal processes is not *literal*, but only *metaphorical*? (A&N pp. 17-18; HHM pp. 13, 15). I suggest two, completely different justifications.

One is the Cartesian or quasi-Cartesian, that would have semantic content, or intentionality and other related concepts, essentially tied to *mind* and *consciousness*. But this, I suggest, as suggested by the name of the original author, is just yesterday's science/metaphysics; the current science/metaphysics is different.

The other justification for the rejection of biological-semantic/normative talk is very different, but actually points distantly to the relevant deep shifts in science and metaphysics. It is the neo-Wittgensteinian argument, made for example by Hacker (1987), that such semantic/normativity concepts really belong to our activities using language, to language-games, i.e. briefly, to our sending/receiving signs enabling activities such as, to use an example near the start of the *Philosophical Investigations*, fetching and carrying stones for building (Wittgenstein 1953, paras. 2, 7). However, the argument in B&G is that signalling, communication, instructions, obtaining and transporting materials for building structures, is already happening in our biology—this, we contend, is the new biological science. I realise the magnitude of the alleged theory-shift here, which is basically from some idea of meaning (and cognates) as true/false representation of reality (hopefully, in Descartes), something so mysterious that only the conscious mind could do it, to the idea of meaning as communication, command and action. But this is the shift involved in the use of semantic/normative concepts in the biological as well as the psychosocial domains.

It was proposed above that the grounds of this biological normativity are fundamentally to do with staying alive or dying, at the individual and/or species level. Putting the matter thus, however, could be interpreted as grounding biological normativity in our interests and concerns, as opposed to being in independent nature. But as against that, and of course, the emergence of life on Earth and its evolution over deep time much pre-dated

us and our concerns and scientific heuristics. The difference between life and death is in nature itself, independent of us, albeit in only part of nature––the biological part.

However, Schrödinger's theory of the biological goes deeper, seeing life as dependent on building and maintaining counter-entropic dynamic structures and functions—until such time as they break down and die. It is an essential of the part of the argument in B&G, aiming to track this deep theory in current biology/biophysics, that the regularities involved in such as genetic replication, genetic regulatory mechanisms, and cell signalling, can break down. This possibility of breakdown in regularities is an essential and distinctive feature of the new biology. The biological regularities are not immutable laws of nature, like the energy exchange and conservation laws of physics and chemistry, but could be otherwise, and can fail. This refers for example to Crick's consideration of the possibility that the genetic code is a 'frozen accident', that the original allocation of codons to amino acids was "entirely a matter of 'chance'" (Crick 1968, 369-370). The accidental, non-fixed-law-like nature of the code is what allows break-down and error, as in genetic mutation, the condition of evolution, and of death.

HHM proposes (pp. 13-14) inter-linked counter-arguments to those set out in B&G, summarised above, that would distinguish biology from physics (and chemistry) in a way that permits normativity. HHM proposes that Newton's *F=ma* can lead to distinct predictions for experimental setups that are mathematically difficult to resolve. This may be true, but what is needed for to counter the argument in B&G is that *F=ma* can actually break down—and it can't. Or, it is treated in such a way that it is not allowed to break down, as in Lakatos' definitive account of scientific methodology (Lakatos 1970). Biological system-specific, information-based 'laws' always contain *ceteris paribus* clauses, as typically for the causal laws of the 'special sciences', unlike physics which has no such clauses, as argued by Fodor (1987). A statement of the sort that such-and-such genetic sequence codes for a particular protein—unpacked in terms of it producing such a protein under normal cellular operating conditions—fails to apply, breaks down, under abnormal conditions. No *ceteris paribus* clause appealing to normative conditions qualify *F=ma*.

A connected line of thought responds to HHM's connected argument (pp. 14) that teleological language can be used to describe e.g. bodies tending to thermodynamic equilibrium. But the response here is the same as applied in the massive theory-shift from Aristotelian physics to the modern mechanics of Galileo and Newton, namely, that the new non-teleological mechanics did all the work needed to explain objects falling to the ground,

and teleological language added nothing of explanatory value. In biology by contrast, the teleological language, the language of regulatory mechanisms and associated constructs, does a variety of explanatory work that is not done by physical descriptors: especially it picks out invariances among physical realisations involved with functions, tending towards ends; it identifies error and can be used to diagnose breakdown, possible repair, etc.

A specific theme in the literature endorsed by A&N (pp. 15-16) is that Shannon information is enough for biology and is not semantic. In reply to this line of thought, I would reframe but basically repeat the arguments as above: Shannon communication involves a transmitter, a signal and a receiver; information transfer reduces uncertainty in the receiver and is prone to more or less 'error'. These inter-systemic, normative concepts are quite unlike those in the energy-related laws of physics, and are applicable to artificial designed functional systems and evolved biological systems alike.

3. Antagonists or Fellow Travellers?

As befits what we argued is a large-scale theory-shift, the BPSM has many fellow-travellers, in Engel's original, and in any update now including B&G. Some among the former are mentioned in B&G, while some of the latter are cited in the commentaries as alternatives, considered below.

3.1. The Interventionist Theory of Causation a Quick Fix?

HHM argues (pp. 19-20) that the complicated and contentious causal/regulatory explanatory model proposed in B&G is not necessary to accommodate biopsychosocial causation because this can be done simply by using the interventionist theory of causation. He notes that we endorse this theory in B&G. However, I suggest, the interventionist theory is not enough by itself.

When conducting an experiment, of some degree of stringency, or by observing a natural experiment, we measure certain variables and estimate the proportion of the variance in the outcome variable that can be explained by (or at least, is associated with) different factors, using regression. It is true that we can put any measured variables that we like into the regression as independent factors, and call them 'biological', 'psychological' or 'social'. Finding that the latter two account for significant variance in health outcomes is of course a major way in which epidemiological and

clinical trials have established the evidence base for biopsychosocial models of particular health outcomes of interest.

The experimental method, however, is well known to be theory-free. So far, we have no idea of causal mechanisms, and also so far no theory of the constructs the variables stand for. In the present case, using the experimental method only, we so far have no idea how to theorise the *biological*, *psychological* or *social*—so far we just have variable names that we are saying are of these sorts. This is particularly important in this area, because of the centuries old presumptions of materialism and the consequent problematic status of psychological and social causes. In the context of this historical prejudice, apparent observations of psychosocial as well as biological causes are wide open to the reductionist pressure that would regard them as noncausal epiphenomena, which obscure the real material causes, e.g. in the brain or genes. Either way, whether we are happy with the untheorized observations, or whether we assume everything is really biological, we have no need to theorise or investigate the causal mechanisms by which e.g. psychological therapy or social exclusion affect health.

In short, the experimental method on its own, philosophically expressed as the interventionist theory of causation, delivers only sparse theory-free empirical findings. No science is satisfied with this; it requires theory, and B&G aims to articulate it for the BPSM. As discussed in B&G, the most worked out theory of how social and psychological factors impact health invokes chronic social-psychological-biological stress, and the explanatory concepts are of the sort that we try to explicate, in terms of environmental and social resources, agency, dysregulation of metabolic processes, etc. See also below section 3.3 on pluralistic approaches that include interactions between kinds of factor.

3.2. Causal Selection

HHM argues that

> the challenge when developing a defensible version of the [BPSM] (…) is not so much providing an adequate account of biopsychosocial causation, but providing an adequate account of causal selection. (Maung this issue, 21)

He notes (*loc. cit.*) that "almost every event that is caused is the outcome of multiple causal factors (...). Nonetheless, we only consider some of these causal factors to be relevant in an explanation". The issue is how we select which factors are causally relevant. HHM goes on to critically discuss

several accounts of causal selection in the literature, and in so doing covers a wide variety of considerations that may come into play in selection, ranging from empirical determination, to distinguishing between explanatorily relevant factors and background conditions, with the addition that this distinction is dependent on contexts, values, and interests, including ethical and political considerations, especially in healthcare (see Maung this issue, 21-23).

In response to this critique, I would say that while B&G does not address the question of causal selection by that name in this way, with reference to the same literature, we do come at more or less the same issues from a different angle, and arrive at quite similar conclusions. In B&G we emphasise that empirical determination is necessary to define what causes affect an outcome, and for empirical study to occur at all, a problem of interest has to have been identified, this being, in health research, a health outcome of interest—i.e. typically, a condition of range of conditions, and within that, onset, course +/- treatment, and quality of life. Once a range of causes implicated in a particular health-relevant outcome of interest has been identified, then, given that healthcare is an applied science aiming to make a difference, at the individual or population level, the challenge is to identify a causal factor that is both of *large enough effect* and is *modifiable*. Many considerations apply in all these stages: in the first step, selection of a health outcome '*of interest*', then also in decisions about what is a large-enough, modifiable target for intervention (prevention or treatment). Considerations include e.g. individual/population burden of illness; healthcare costs; acceptability of interventions, available technology, level of resources, cost-benefit analyses, political priorities—all these of different sorts. While HHM and B&G take different approaches to this question of identifying relevant causes, I don't see that they are wide apart in direction or conclusions.

3.3. Pluralism

HHM and A&N both consider the relation of the BPSM to various types of explanatory pluralism. HHM accepts that the BPSM accommodates or is compatible with explanatory pluralism (pp. 23-24), and I think that's right. A&N by contrast view explanatory pluralism as alternative to the BPSM (p. 11). On the other hand, A&N acknowledge (pp. 11, 13) that B&G's proposal that the content of the BPSM is in the specifics, is not that different to an explanatory pluralism that is guided by data on the specifics. They make the point (p. 11-12) that databased models of specific conditions, such as diabetes or depression, cannot be derived from a general statement of the BPSM, and that is of course correct and exactly part of the argument in B&G.

A&N go on to say (p. 12) that "establishing the psychological and the social as ontologically and causally real", as proposed in B&G, "doesn't help us with the question of how to best integrate the etiological factors in the form of a coherent explanation and how this should inform multidimensional approaches to treatment". My response here is that the intention in B&G is to map out, at least some of, the key constructs and principles that can be used to construct integrated models of risks for onset, maintenance, and treatment of specific conditions.

B&G considers two main models of integration: chronic stress and pain, which between them are major drivers of ill health and service use. As noted in the previous section, we highlight that current models of chronic stress are essentially biopsychosocial, involving the psychological aspect of down-regulation of agency (raising risk of dysregulation of agency, helplessness or inability to cope), interacting with the social aspect of excessive salient task demands in relation to low access to resources, linked to 'low social status', poverty, racism and other kinds of social exclusion, and the biological responses to chronic psychosocial stress that involve dysregulation of metabolic processes, compromising the immune system, creating risk for many kinds of ill health. The intention in B&G was to sketch out the constructs and principles employed in such models of complex biopsychosocial/environmental interactions. Another example considered in B&G in some detail was that of pain, discussed above in section 2.2., highlighting that current models implicate central neuropsychological processing including appraisals of agency/impairment as well as peripheral damage, or even in the absence of detectable sufficient peripheral damage. Again, the aim was to explicate the constructs and principles of these new models that integrate biopsychosocial/environmental factors.

Overall, the intention was to go beyond any general statements to the effect that "it's all very complicated involving lots of things and requiring lots of different approaches", whether such a general statement is labelled as "the BPSM" or as "pluralism". The science has gone way beyond this and there is no need for such general statements in the clinic, or in education, at least not in courses where the learning outcomes include understanding the science or the ability to read scientific papers. We can use the general statements, but hopefully followed by advice that there are ongoing research programmes on the details.

3.4. Enactivism

A&N compare and contrast the proposal in B&G with the 3/4E models of embodied cognition, sometimes called 'enactivist' theories. They note that

we endorse the 4E approach, as does HHM (p. 11), and they note many similarities between B&G and enactivism (A&N, pp. 14-15). For me, the list of similarities is long and substantial enough to regard B&G's version of the BPSM and enactivism as fellow travellers. A&N go onto contrast them, however, in favour of enactivism, citing its advantages over B&G in two respects (p. 19):

(1) Enactivism does without the problematic concept of biological normative/semantic information

(2) Enactivism explicitly bridges the natural-normative gap, by affirming that "all life shares an embodied concern (i.e. a self-perpetuating structure) for the continuation of self" (p. 19)

On the second point (2), the intention in B&G is to affirm something like what A&N propose. Specifically, and as reiterated above in section 2.3., it proposes that the biological in nature has a normativity, grounded in the difference between life and death, adding the connected point that the regularities on which life depends are contingent and mutable, unlike laws of non-biological nature, and are liable to breakdown, eventually in dying and death, the end of the struggle to withstand increasing entropy.

This raises the question of the relation between (1) and (2). Granting that enactivism envisages normativity in all life (2), why should it want to resist accepting normativity in biological information (1)? If all life exhibits normativity—grounded in the difference between life and death—what would be the problem in accepting that this normativity, so grounded, applies to biological information? It is not clear, in other words, that the first supposed advantage of enactivism sits well together with the first.

The broader point here is that models of embodied cognition such as 4E do not necessarily reject the concept of information-processing, though they of course interpret it in the terms of the model, i.e. as tied closely to requirements for action, linked to needs and concerns. What is rejected is the old idea of information-processing as being processing of 'mental representations' (Newen et al. 2018) , i.e. as I understand it, representations of a ready-made, independent world, that has so far nothing to do with the embodied, active cognitive agent. There are many strands involved in models of embodied cognition (Newen et al. 2018), and only some take the radical and problematic step of eschewing the concept of information altogether (Carney 2020).

So far as concerns the BPSM, we supposed in B&G that accounting for the biopsychological (two of the three domains in the model) requires the model of embodied cognition, which also makes explicit its essential environmental involvement. Since the BPSM also requires linkages between the psychological and social, it is also necessary to emphasise that cognition, with action and agency, is constituted by interactions not only with the non-social environment, but also by interpersonal and other social relations.

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