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# Bio-psycho-social interaction: an enactive perspective

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**ABSTRACT**

What are the respective roles of physiological, psychological and social processes in the development of psychiatric disorders? The answer is relevant for deciding on interventions, prevention measures, and for our (self)understanding. Reductionist models assume that only physiological processes are in the end causally relevant. The biopsychosocial (BPS) model, by contrast, assumes that psychological and social processes have their own unique characteristics that cannot be captured by physiological processes and which have their own distinct contributions to the development of psychiatric disorders. Although this is an attractive position, the BPS model suffers from a major flaw: it does not tell us how these biopsychosocial processes can causally interact. If these are processes of such different natures, how then can they causally affect each other? An enactive approach can explain biopsychosocial interaction. Enactivism argues that cognition is an embodied and embedded activity and that living necessarily includes some basic form of cognition, or sense-making. Starting from an enactive view on the interrelations between body, mind, and world, and adopting an organizational rather than a linear notion of causality, we can understand the causality involved in the biopsychosocial processes that may contribute to the development of psychiatric disorders.

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## Psychiatry's integration problem

Recently I have been interviewing people who suffer from recurrent major depressive episodes. 'John' was one of them: a kind, clever man, 63 years old, who had just come to accept that he would not be holding a paid job anymore. He wanted to understand how these depressions that had, and still have, so much impact on his life had come about. He felt the strong need to make sense of his depressions – also in order to do as much a possible to prevent them from happening again. Was it down to genetics?, he wondered. Looking back, he concluded that his mother must have had depressive episodes herself, even though she was never diagnosed. Or was it his upbringing in an emotionally unsafe environment, with a largely absent father and an emotionally frail mother? As the eldest son he was expected to be tough, not show any feelings, and help out with his younger brothers and sisters. It was also clear that his depressive episodes typically coincided with feeling overburdened at work, by too much responsibility and too many tasks. Some of his personality traits probably did not help either, he thought, like his perfectionism, his tendency to feel responsible, and to prioritize helping others instead of recognizing his own needs. But then again, where did these personality traits come from? And what did it mean that some medication worked quite well for him?

John's struggles to make sense of his recurrent depressions are not 'just' an individual problem. It is a problem that anyone who tries to make sense of the development of psychiatric disorders encounters – whether driven by one's own experiences, or the experiences of loved ones, or as clinicians or researchers. With so many potentially contributing factors of such different natures – e.g. genes, neuronal specificities, (childhood) trauma's, social and economical disadvantages, existential worries – one of the holy grails in psychiatry is to get clear about how to relate these factors and assess their precise roles. What influences what? What is cause and what is effect? The answers to these questions are not only important for our (self)understanding, but also for determining how to

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best intervene in and possibly even prevent the occurrence of psychiatric disorders.

## Reductionist versus holist models

Questions about causality are questions about ontology: how do different processes relate? There are various explanatory models available for psychiatry, but when it comes to comprehending the relation between the potential contributions of these widely diverging factors there are two main options: reducing all factors to one central one, or granting multiple factors a formative share in the development of psychiatric disorders. *Reductionist models* propose a hierarchy between factors with one type of factors being primary; providing the underlying cause of the problems at hand. As with the rest of medicine, in psychiatry too it is typically physiological processes that are considered primary. In the past decades, the causal roots of psychiatric disorders have specifically been sought in genes and the brain. The neuroreductionist idea that psychiatric disorders are, in one way or another, diseases of the brain, has in particular gained much popularity.

The advantage of reductionist models is that they are coherent and simple. Simple in terms of their main structure, that is: of course the modelling of specific failures in specific brain mechanisms for specific psychiatric disorders remains very complex. But the overall structure of the explanation is nicely straightforward: all symptoms of psychiatric disorders can be traced back to abnormalities in the brain. The biggest downside of reductionist models is that their preference for only one type of factors is unwarranted. So far, despite many research efforts and much funding, no clear genetic or neuronal causes of psychiatric disorders have been found. Few people would contest that the brain is *implicated* in psychiatric disorders, but this does not justify the a priori assumption of its causal primacy. In fact, even when we focus our attention only on genes or the brain, we are still forced to recognize the impact of someone's environment. Both epigenetics and the brain's plasticity attest that our experiences and interactions with the world shape us. Our interactions affect our gene expressions and lead to alterations in our brain's anatomy and functional connectivity. Yet as soon as we have to give up on the idea of either genes or brains as automatically unfolding 'blueprints', we can no longer meaningfully speak of their causal primacy either. Another important drawback is that meaning drops out of the picture when phenomena occurring at a personal level are explained purely in terms of physiology. Finally, adopting a neuroreductionist explanatory model does not have the hoped for effect of reducing the stigma of psychiatric disorders; it may even increase this stigma (Pescosolido et al., 2010).

The other option is to assume that multiple factors may be involved in the development and persistence of psychiatric disorders. The best-known representative of such an encompassing account is the *biopsychosocial (BPS) model* as put forward by Engel (1977, 1980). Engel (1977) criticized the reductionism of what he called the biomedical model and argued that 'inclusion of … psychosocial factors is indispensable' (p. 131) in order to account for such phenomena as patients' experiences of their disease, the effect of living conditions on the development and course of the disease, and the effect of the relationship between physician and patient on the outcome of treatment. The BPS model has been widely adopted in clinical and health educational programs worldwide (Bolton & Gillett, 2019). It does justice to the intuition and evidence that social/environmental and psychological factors matter in the development of psychiatric disorders (Bolton & Gillett, 2019) and it fits with the clinical practice of anamnesis, in which patients' problems are explored in the context of their current situation and previous history.

Despite these advantages, the BPS model has in recent years been strongly criticized. Its main problem is that it lacks a clear account of how the 'bio' the 'psycho' and the 'socio' interact (Ghaemi, 2009; Kendler, 2010; Van Oudenhove & Cuypers, 2014), which in turn may affect its practical applicability (Ghaemi, 2009). Engel (1980) referred to Weiss' and Von Bertalanffy's General Systems Theory to explicate biopsychosocial relations. He argued for a 'hierarchically arranged continuum' (p. 536), spanning from molecules and cells to nervous systems, persons, families, communities, nations and finally, the whole biosphere. These levels are all systems in their own right, while at the same time the lower level systems are the components of the higher level systems. So a cell is a system of its own while it is also part of the nervous system, of the person and so on. Each system is unique, and requires its own unique study methods and types of explanation. By stressing each system's uniqueness, Engel makes a strong case against the reductionist principle to explain 'higher level' phenomena in terms of 'lower level' phenomena. The downside of this is that it makes it harder to envision how these unique systems interact: if they are so different, how can there be causal relations between

them? Unfortunately, Engel (1980) only tells us that across the boundaries of these systems, 'material and information flow' (p. 537). That is not very helpful.[1]

## Bio-psycho-social interaction from an enactive perspective

Any model that wants to avoid reductionism and capture the complexity of multiple processes (potentially) contributing to the development of psychiatric disorders encounters the integration problem. It is a notoriously difficult problem: how should we characterize the causal relations between such different factors as someone's neurotransmitter uptake and release, their tendency to avoid conflicts, and the quality of their friendships? What we are looking for is not only a solution of the mind-body problem, but of what we could call the 'mind-body-world problem': how do body, mind, and world relate?

A recent paradigm, *enactivism*, offers a novel outlook on the relation between body, mind and world.[2] Developed initially for cognitive science, enactivism stresses that cognition can only be understood by taking the whole embodied organism and its environment into account (Varela et al., 1991). It draws on a wide range of sources, notably biology's developmental systems theory, phenomenology, and dynamical systems theory. There are several strands and applications of enactive ideas (Di Paolo & Thompson, 2014), but most relevant for the integration problem is enactivism's so called '*life-mind-continuity thesis*' (Thompson, 2007; Di Paolo, 2009; Froese & Di Paolo, 2009). It states that mind, or *sense-making*, is central to living. Living beings are special compared to non-living matter in that they are self-organizing unities: they maintain themselves through a constant exchange with their environment. In order to stay alive, organisms need to take up nutrients and dispose of their waste. Organisms are thus dependent on continuous interactions with their environments. This means that they need to be able to distinguish what is relevant for their survival in their environment: what is food and what is not, what is dangerous and what is safe. Without such an ability to make some basic sense of their environments, living would not be possible. It is in this way that life and mind are continuous: living requires sense-making.

Now there is obviously more to the human mind than the sense-making of an amoeba or an ant. The basic forms of sense-making (distinguishing, sensing relevant aspects of the environment) are present in all life, but humans are capable of such remarkable behaviours as feeling empathy, making promises, doing maths, and writing poetry. The difference is that humans are capable of *reflexive* or *existential* sense-making: turning sense-making onto itself.[3] We do not only experience things, but we are *aware* of these experiences, and of ourselves, and of how others see us. This capacity for reflexive sense-making opens up a whole new array of abilities, as well as greatly expanding the domain of relevance. For while basic sense-making is about the immediate relevance of the here and now for surviving, existential sense-making implies that it is no longer just survival that counts, but living a good, dignified life. Our self-awareness opens up a moral life and we move from functional values to existential values (de Haan, 2020a, 2020b). Still, our sophisticated sense-making remains just as embodied and embedded. It is in and through our interactions with others, embedded in specific socio-cultural practices, that we develop our existential sense-making in the first place (de Haan, 2020a; Reddy, 2008).

From an enactive perspective, the 'biological' of the BPS model is thus inevitably bound up with the 'psychological' and the 'social'.[4] Living bodies require interactions with their environments just as much as they require the capacity to make sense of these environments. In other words, physiological processes could not exist without the mind or interactions with the environment.

Applied to psychiatry, an enactive approach sees psychiatric disorders as problems of sense-making; structurally disordered patterns of sense-making (de Haan, 2020a, 2020b). As sense-making is the activity of a bodily person interacting with her material and social environment, this is also the preferred unit of analysis in psychiatry, following this view. From a mathematical perspective, a person-interacting-in-her-world is not only a complex system, but also a dynamical system, as the interactions imply that it changes over time.

So what does this all mean for the question we started out with, about the causality involved? There are two main tendencies that typically lead to confusion about bio-psycho-social interactions. The first is the tendency to think of 'bio', 'psycho', and 'socio' as different domains, and the second is the tendency to assume some form of linear causality between them. As we saw, from an enactive perspective both physiological processes and sense-making are dependent upon each other and on interactions with the (social) world. Our (social) world is in turn shaped by our interactions with it. This means that we cannot

properly understand any of the three factors – body, mind, and world – in isolation from each other. They are instead *different excerpts* of one and the same, complex, dynamical person-in-her-world system. They stand in a *mereological*, part-whole relation. Of course, physiological processes have different characteristics than social or sense-making processes – but this does not mean we have to think of them as separate domains. In fact, when we manage to think differently about causality, we can also see how the idea of separate domains is unnecessary and confusing. When we think of causality, the first thing that comes to mind is a simple, linear picture of cause and effect: one billiard ball or domino hits another. There is a clear cause and a clear effect. Combined with the assumption of 'bio', 'psycho', and 'social' as different domains, we end up with the mind-boggling question of how a psychological domino could possibly 'hit' a physiological one, and vice versa. How could they possibly intervene in each other's territory?

Adopting a complex, dynamical systems approach allows for a different, non-linear, take on causality. Complex systems consist of many interacting components. The back-and-forth of causal influences and typically also (multiple) feedback loops means that a complex system cannot be analysed into a vertical, stratified hierarchy of layers – as in reductionist pyramid models. Complex systems are often pictured as networks. With so many complex processes going on, typically operating at different time-scales, the causality involved is non-linear, rather than linear. An analogy might help. Instead of bumping billiard balls, we can think of the causality involved in baking a cake (de Haan, 2020a, 2020b). By mixing together various ingredients, we make a mix that we can put in the oven to bake – with a hopefully tasty cake as result. As any baker – or any regular watcher of baking shows – knows, the ingredients affect each other. The amount of sugar for instance not only contributes to the sweetness of the cake, but also affects the dough's gluten, thereby affecting the structure of the sponge. It is not only the precise amounts of flour, eggs, baking powder, milk, and butter that influence the cake's eventual taste; it also matters how long you knead the dough, and at which temperature and how long you bake it. Now, the amount of sugar (or any other ingredient) obviously affects the overall taste of the cake. The sugar, however, does not cause the cake's taste in a billiard-ball fashion. Rather it co-determines the cake's taste *by being part of it*. A change in the amount of any of the ingredients is a change of the cake as a whole. There are thresholds: not all changes at a local level will be noticeable at the global level. Adding a few more grains of sugar will not make much of a difference on the cake's taste, but adding several spoons of sugar will. But it is not only the local ingredients that affect the cake's global taste: there are global-to-local influences as well, such as the effects of the baking time on the various ingredients of the dough. Here too, the billiard ball model does not apply: the heat of the oven does not change the cake as a whole, which change then *in turn* influences the ingredients. Instead, influencing the cake as a whole *implies* influencing its parts. Here too, the global intervention can have different effects on different parts of the whole: the cake's icing can be burned before the dough is cooked.

We can use this analogy to understand the 'bio-psycho-social' causality that is at stake in psychiatry. Within the complex system of a person-in-her-world, we can similarly distinguish between local-to-global and global-to-local effects. A small change in the serotonin levels in the brain (i.e. a local physiological process) will not be noticeable at the global level of the person's mood, but a bigger change will. Conversely, an insightful therapy session can calm down the patient and this intervention at the global level implies several local physiological changes, for example in neurotransmitter levels and muscle tension. Serotonin levels and moods are not part of different domains, one physiological and the other experiential: they are rather both part of the same person-in-her-world, different excerpts of this same system, at different levels of globality, operating at different timescales.

By distinguishing between more local and more global processes within the one person-world system we can do justice to the fundamental differences between physiological processes and experiential and social processes – something reductionist models cannot offer. Yet we can also make clear how these processes relate: as they are part of the same system they cannot be opposed and therefor there cannot be any *linear* causality between them either. The 'bio', the 'psycho', and the 'social' do affect each other, but through what we might call mereological or *organizational* causality. By distinguishing between local-to-global and global-to-local causality, we clarify how different interventions have different effects and involve different causal trajectories. Even though at the global level the results may be similar, it is important to recognize that this result is achieved through different causal trajectories. For example, both psychotropic drugs and psychotherapy can

reduce someone's anxiety levels, but they obviously do so in different ways.

Coming back to John, the BPS model would tell him that his recurrent depressions were probably due to a combination of biological (genetic), psychological (personality traits) and social factors (upbringing, current stressors). It could accordingly list the concrete correlations that have been found in these respective areas of depression research. Based on more concrete details of John's situation, an estimation could be made of the likely relevance of these general findings. An enactive perspective, on the contrary, would start from John in relation to his world as forming one system, already offering an outlook on the interrelation of the physiological, psychological, and environmental processes involved. Compared to such an enactive approach, the BPS model appears to artificially break elements down into domains with no clear view on their relation.[5]

From an enactive perspective, a person's interactions with her (social)environment are crucial. We 'lay down a path in walking' as enactivists (Varela et al., 1991) like to say: like a path emerges from repeatedly walking the same walk, patterns in our behaviour emerge from repeatedly interacting with our environment in a certain way. Psychiatric disorders refer to structurally disordered patterns of sense-making: when sense-making is no longer flexibly attuned to the situation, but instead biased in a certain direction. One for instance feels anxious all the time, regardless of whether the situation is indeed threatening. Such 'stuck' sense-making typically occurs when sense-making that was once appropriate given one's situation, is repeated even though the present situation no longer calls for that response. Sense-making patterns reflect the history of one's interactions.

John's *upbringing*, and more generally the environment he grew up in, have shaped his inclinations in his way of reacting to the world. Later on, his work as a nurse also fitted his self-effacing tendency to be directed at caring for others. His *personality traits* at least partly reflect this deeply engrained pattern of behaviour. Anything we do, and especially what we do repeatedly, changes our *brains*. Here too, paths are laid down by walking: functional connectivity between brain regions strengthens due to repeated behaviours. This also explains the efficacy of *medication* and other brain-targeted interventions. Their efficacy does not imply that the brain is at the root of everything, as neuroreductionists would say, but rather reflects that the brain is what we could call a 'mediating organ' (Fuchs, 2018). In terms of network models, the brain is a highly connected node, so intervening in it can potentially have wide-ranging consequences. The precise role of his *genes* cannot be seen in isolation from his interactions with his (social) environment either, as these co-determine their expression. In fact, histories of interactions stretch over generations, modifying gene structures as well. This intertwinement of physiology, psychology and (social) environment also clarifies that changing such a complex person-world system can be achieved in many ways, targeting local (e.g. neural) processes or global (e.g. behavioural, social) processes.

## What do we gain by adopting an enactive perspective?

Just like the BPS model, an enactive approach argues for a holistic conception of psychiatric disorders. Unlike the BPS model, however, enactivism offers a coherent account of how physiological, psychological, and social processes relate; an account that explains their unique characters as well as their mutual interactions. Enactivism thus provides a sound theoretical foundation for a holistic psychiatry.

Even though the discussion of holism versus reductionism is pretty abstract, which model one (implicitly) assumes will have important practical implications. Our assumptions inform empirical research: the kinds of questions we ask, the kinds of places we look for answers, and the kinds of methodologies that we prefer. They also inform treatment decisions (which interventions seem most worthwhile?), and communication with patients and professionals. A sound holistic model helps us resist the temptation to a priori single out one type of process as 'the' defining matter of psychiatric disorders and to unquestioningly assume that there are such things like 'underlying' causes or mechanisms of psychiatric disorders. A sound holistic model does justice to psychiatry's complexity in a manageable way and offers us (self)understanding. And, importantly, it supports the holistic practice of cooperation in interdisciplinary teams of social workers, psychologists, psychiatrists, nurses, and other professionals, and as such supports optimal care. Enactive psychiatry is such a view.

## Notes

1. In their recent defence of the BPS model, Bolton and Gillett (2019) also acknowledge that its value should lie in explaining biopsychosocial causal interactions and that Engel's work does not sufficiently address this issue.

They argue that 'information-based regulatory control mechanisms' (p. 45) can explain biopsychosocial causation. Although their account shares several main ideas with an enactive account (i.e. the shift from physics to biology, acknowledging the causal power of form or structure, and recognizing the normativity inherent in the process of living), there is also a huge difference. By relying on the metaphor of information exchange, they counteract all the non-reductionist explanatory force that comes from accepting the causal powers of structure (i.e. emergence). Instead of relying on the organisational causality of structure, they assume that this causal power must be explained as the exchange of information between higher and lower level processes: again separating these processes (or 'agents', or 'systems') rather than regarding them in their part-whole relationship. Moreover, by defining information 'like a switch, turning processes on and off, hence being representable typically by 0's and 1's' (p. 48), the door is left open to reductionist explanations again, which is exactly what happens when the authors reduce life to genes (p. 46) and equate psychological processes with central nervous system pathways (p. 81, 99): invoking the very reductionist physicalism that they aimed to abolish. For an excellent, in-depth analysis of and critique on the use of the notion of 'information' in genetics and developmental theory, see Oyama's (1985/2000) '*The ontogeny of information*'.

2. A related, yet fundamentally different approach, is extended mind theory as developed by Clark and Chalmers (1998). They argue that the mind can extend into the world and that it is arbitrary to limit the mind to what is happening 'inside the skull'. If a part of the world functions similarly as the cognitive processes 'going on in the head' it is just as much part of the mind. Extending the inner is, however, very different from overthrowing the very inner mind-outer world division – as enactivism proposes. The extended mind theory has recently also been applied to psychiatry (Davies, 2016; Cooper, 2017; Roberts et al., 2019). Like the enactive approach, they reject the idea that psychiatric disorders could be explained by solely referring to what is happening inside an individual and instead stress the fundamental role of patients' interactions with their material and social environment. They fundamentally differ when it comes to explaining how physiological, experiential, and environmental processes relate. Because extended mind theory assumes only a functionalist relation between these processes (instead of a fundamental dependency), it remains stuck in the dualist difficulties of explaining their interactions. Enactivism, by contrast, assumes the physiological and experiential processes to be fundamentally dependent on each other as well as on their interactions with the environment. It thus advocates one person-world system, with organisational rather than linear causality between its more local and more global processes (de Haan, 2020a, 2020b).
3. It is an empirical question whether there are other animals that are capable of this reflexive sense-making too.
4. From an enactive perspective, the terms of the biopsychosocial model are a bit unfortunate. As biology is the study of life and living beings, for enactivists this means that sense-making is necessarily part of biology, so there is no opposition between biology and psychology. Moreover, 'psychological' is such a broad container notion – including such different phenomena and categories as unconscious processes, executive functions, and personality traits – that it is unhelpful when one tries to provide a precise account of causality. Following an enactive take, it would instead be more accurate to speak of physiological and *experiential* processes, or *sense-making* when we are primarily concerned with cognitive capacities. Finally, although the world we interact with is certainly social, it is also cultural and material, so the term 'social' is not quite apt either.
5. Depending on the details of John and his particular situation a *personalized network model* could be constructed with the nodes representing the factors that are of particular (positive or negative) relevance to John (de Haan, 2020a). It is important to note that such a personalized network model differs from *symptom network models* as advocated by Borsboom and colleagues (2018). Symptom network models only include DSM symptoms and models their interrelations. However, as Nielsen and Ward (2020) point out, they *model* symptoms, but they do not *explain* them. That is because, as mathematical models, they offer a helpful template, but no ontological account of what should and should not be part of the model and how the elements are related (de Haan, 2020a). By contrast, the personalized network models that follow from an enactive approach are what we could call 'constitutional' networks. As a consequence, they can include all kinds of relevant factors: not just symptoms, but also, importantly the 'contextual factors' that symptom network models have to leave out of the picture. For an alternative elaboration of an embodied, embedded, enactive method using network models, see: (Nielsen & Ward, 2020).

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