

# A frame of mind from psychiatry

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**Abstract** A distinctive characteristic of psychiatry is that it is a discipline that deals with both the physical and the mental lives of individuals. Largely because of this characteristic, different models are used for different disorders, however, there is still a remnant tendency towards reductionist views in the field. In this paper I argue that the available empirical evidence from psychiatry gives us reasons to question biological reductionism and that, in its place, we should adopt a pluralistic explanatory model that is more suited to the needs of the discipline and to the needs of the patients it is meant to help. This will allow us to retain psychiatry as an autonomous science that can productively co-exist with neuroscience while also giving patients the kind of attention they need. I further argue that this same evidence supports a view of the mind that is anti-reductive and that allows that causation can be both bottom-up and top-down and that such a view is available in emergentism coupled with an interventionist model of causation.

**Keywords** Emergence · Mechanisms · Mind–body problem · Explanatory Pluralism · Reduction · Psychiatry

## Introduction

The question of the nature of the mind and its relation to the brain is a question that has puzzled philosophers for centuries. Though it may seem that a theoretical question like the mind–body problem is not of immediate

importance in mental health professions like psychiatry that have a practical orientation, aspects of this problem are endemic to disciplines that seek to understand the mind and its disorders. Not only the methodological approach one will use will depend on what the constituent parts of the problem are and how they are related, but also, the ultimate status of the mind has repercussions for the status of psychiatry as a science. At the same time, empirical evidence from psychiatry can challenge explicit and tacit views we have about the mind.

In this paper I look into what current empirical evidence from psychiatry can tell us about the mind–brain relation and, given that evidence, what sorts of explanations we should strive for in psychiatry. I conclude that we should aim for explanatory pluralism and, in the end, suggest that the theory of mind in terms of which we should understand the mind is emergentism. Because of the limited space of this paper and the scope of the issues it touches upon, this can only be an outline of a position that unavoidably will leave a lot of issues untouched—the attempt, however, is to point to a view of explanation and of the metaphysics of the mind supported by the data we have from psychiatry.

## The mind–body problem and explanation

The mind–body problem is the problem of explaining the relation between our mental states like beliefs and desires, to the physical, neurological, processes in our brain. The two seem to differ qualitatively; the former being essentially private and introspectively accessible while the latter are objective states of affair that can only be accessed from a third person point of view. The main obstacle to solving this problem is to find a way to explain the distinctive characteristics of our mental states while also, and

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consistently, explaining how it is possible for our mental states to affect our physical states and vice versa. Though many solutions have been proposed, none seem to adequately address both and, as a result, no one solution is accepted by all or even the majority of scientists and philosophers.

In the seventeenth century René Descartes faced with this problem concluded that there are two aspects of reality: the physical realm and the mental realm—two entirely different substances that causally interact in a way that we cannot quite specify through the pineal gland. This position is known as Cartesian, or substance, interactionist dualism. For both theoretical/philosophical reasons, like the problem of how two different kinds of things can causally interact, but also due to advances in our knowledge of brain biology, this is not considered a viable position any more.

The rejection of Cartesian dualism and the advances in science in the early twentieth century led to the beginning of an era of reductionist enthusiasm that promised to expunge any possible supernatural, or spiritual, addition to our world view and to guarantee a scientifically respectable monism, the view that there is only one kind of thing that the world is made of, in this case matter.

Reductionism has been understood in many ways in philosophy and science but, for our purposes, we can say that it is a view about explanation according to which phenomena at one level are explained in terms of phenomena at a lower level. This is mirrored in the hierarchy of the sciences that studies each level—with physics being at the bottom and the so-called “special sciences” at each level above that, starting with chemistry and biology and going all the way up to sociology. The core idea of reductionism, then, is that each special science can be explained by the science at the level below it; sociology can be reduced to psychology, psychology to biology, biology to chemistry and that, in turn, to physics. In psychiatry, the reductionist turn led to biological reductionism, the view that exclusively privileges explanations in terms of biology—the idea being that to explain mental disorders all we need is to explain the biology underlying them and how it gives rise to them.

It wasn't long, however, before reductionism was challenged. In the 1960s Putnam's arguments for multiple realizability cast the first stone and, later, with Fodor's influential argument for the autonomy of the special sciences, reductionism came under serious scrutiny. As a result, anti-reductive views took hold that view the special sciences as autonomous dealing with phenomena at different and irreducible levels.

For a while the debate was in terms of which of these different approaches available—reductionism or anti-reductionism—was to dominate. Lately, however, a new view has been suggested, that of explanatory pluralism. Explanatory pluralism is a view about explanation

according to which in order to understand complex phenomena different kinds of explanations are required addressing different questions at different levels. When it comes to the mind, this means that different approaches and methodologies need to be used if we hope to shed light on mental phenomena.

### Integrative explanatory pluralism

Mitchell (2004) has proposed a model of explanatory pluralism that she calls “integrative pluralism” which aims at the integration of different explanatory models. Unlike other kinds of explanatory pluralism that see the world as dappled and the sciences as disunited (Dupré 1993; Cartwright 1999), in Mitchell's multilevel approach a full explanation will integrate different explanations at different levels—e.g. the neuronal, the computational and the personal level.

Integrative explanatory pluralism is not reductive *per se* yet it is compatible with reduction. Though it neither identifies phenomena of one level with phenomena in another nor is it committed to the idea that *explanations* of “higher” level phenomena (in this case certain mental phenomena) will be exclusively in terms of phenomena at a “lower” level (in this case brain mechanisms), since for Mitchell's explanatory pluralism a full explanation involves explanations at different levels that are interconnected, a full explanation will also involve a downward—reductive—explanation. In this sense, explanatory pluralism is compatible with reduction understood as reductive explanation. Indeed, there is no contradiction between the claim that to understand mental phenomena different kinds of explanations from different sciences are needed and the claim that mental phenomena can be explained in terms of underlying mechanisms. After all, one form of reductionism is reductive explanation and finding physical mechanisms can be a way of giving a reductive explanation. That is compatible with there being other explanations at different levels that are also explanatory. Therefore higher level, special science explanations remain intact—given, in the case of the mental, that it remains to a large extent irreducible to the physical—and are an integral part of a full explanation in terms of mechanisms. So explanatory pluralism and explanatory reduction are compatible.<sup>1</sup> This is worth noting because, in itself, there is nothing wrong with reduction. Indeed, where available it should be welcome because by describing the physical mechanism that brings about a higher-level phenomenon we may gain explanatory insight into how the phenomenon works and

<sup>1</sup> For an argument that mechanistic explanation is a form of reduction see Bickle (2003) and Wimsatt (2000). For the opposite view see Craver (2007).

why (or why not) a theory of the higher-level phenomena is true.

### The medical model of mental disorder

Today a strongly endorsed approach in psychiatry is the so-called medical or disease model of mental disorder (Harland et al. 2008). The medical model is a view of mental disorder according to which a mental disorder is a medical disease (Murphy 2006; Andreasen 1984, 2001; Kandel 1998). This is often understood to be a reductionist view, and though this is true in some cases, it is neither necessarily the case, nor predominantly the case. In itself, the view that a mental disorder is a medical disease does not entail anything further about whether the causes should be identified at the level of genes, molecules, or at the cognitive level. In the same way that to say that a heart condition is a medical disease does not say anything about what caused such a condition—e.g. whether it was caused by purely organic causes or, say, stress. Indeed, though there are reductionist views that privilege one level of explanation over all others that would fall under the medical model, such as Kandel's (1998) proposed framework for psychiatry that aims for a reduction of mental disorders to molecular biology, the general consensus in psychiatry is that the etiology of mental disorders is multifactorial and involves causes at different levels.

The medical model is to a large extent a mechanism-based framework in which explaining a disorder is associated with identifying the mechanism that describes how it works. The search for mechanisms goes back to the scientific revolution of the seventeenth century and the general idea behind mechanisms is that, like a watch whose mechanism can be understood in terms of its parts and their operations, so the function—or dysfunction—of a system can be understood mechanistically in terms of the system's constituent parts and the way they interact. Recently, the mechanistic view of explanation has been put forward as an alternative to the covering-law model of explanation that does not seem to cover the reality of many sciences or the practice of the health sciences. Though the notion of a mechanism has been understood in different ways, a common working definition of it is “entities and activities organized such that they are productive of regular changes from start or set-up to finish or termination conditions” (Machamer et al. 2000, 3). In this sense, then, we have an explanation of a phenomenon when we can describe in full detail the behaviour of a mechanism in terms of its parts and their interaction. This has been primarily understood in reductive, biological, terms the *relata* being mechanisms at higher levels and their component parts in lower levels—even though it is not micro-reductionist since it does not

exclusively privilege one fundamental biological level, e.g. molecular biology, over another (Craver 2007).

However, we can use a more liberal notion of mechanism that need not be reductive that can accommodate explanatory pluralism. Essentially, a mechanistic explanation is a causal explanation that shows how parts of a mechanism come together in order for the mechanism in its entirety to perform a function. As such, a mechanistic explanation can include psychological causes too. For example, an explanation in terms of trauma can be an explanation in terms of a mechanism. So a view like the medical model that is committed to the idea that mental disorders involve dysfunctional mechanisms is not limited as to the scope of possible explanations, and therefore need not be an exclusively reductive view but is compatible with explanatory pluralism.

### Why integrative explanatory pluralism?

Though the search for mechanisms has undoubtedly proven to be beneficial in many sciences including the medical sciences, mechanisms may not be sufficient for a full explanation of complex phenomena because, though they capture how a system works, they leave out why it works the way it does. Not only this, but mechanisms seem to leave out what seem to be characteristic features of complex systems; e.g. weather systems and the flocking patterns of birds can be described as mechanisms appealing to the component parts and their organisation but it is not clear that mechanisms can describe the self-organisational aspects of such complex systems. Mitchell's integrative pluralism aims to address these shortcomings by addressing not only the “how?” questions, but by also incorporating historical explanations like evolutionary or developmental explanations that address the “why?” questions (Mekios in press).

In a field like psychiatry that spans the mental and the physical aspects of humans and in which the personal history of a patient is important, such a multifactorial approach seems particularly suited. According to explanatory pluralism it is the nature of mental disorders to have certain (at least proximal) organic causes but identifying them is only part of the explanation of what the disorder is. Explanatory pluralism thus requires that in order to achieve a full understanding of mental disorders different explanations addressing different aspects of the problem need to be taken into account.

In psychiatry most mental illnesses are defined as syndromes in terms of symptoms, progress, outcomes etc. rather than some physical pathology or cause. One reason for this is that for most disorders we do not have reductive explanations and no such explanations are foreseeable any

time soon. There seem to be different causes at play for the onset of mental disorders and there is such variation between individuals that exhibit symptoms of mental disorders and such variation within the same individual at different times, that a reductive explanation might well be unattainable. Of course, this does not mean that reductive explanations are impossible, but it does mean that, so far, they are inductively improbable.

With rare exceptions like Alzheimer's for which biological markers have been found and Huntington's disease for which a fully penetrant and dominant gene has been identified, increasing evidence points to the fact that though genes and neurophysiology are relevant to the etiology of mental disorders, there is no direct causal link from one to the other. According to the widely accepted "stress-vulnerability hypothesis", what genes do is contribute in terms of dispositions by increasing the probability that someone may develop a mental disorder (Schaffner 1994). For example, a genetic predisposition for depression may express itself only if triggered by a major stressful life event (Kendler et al. 2003). If we take the case of schizophrenia, the disorder for which the medical model is most strongly endorsed (Harland et al. 2008), there is no evidence for one common underlying biological correlate of psychosis, either genetic or neurophysiological (Kidd 1997; Flashman and Green 2004). Rather, psychosocial/environmental factors like living in an urban area, living with families with high expressed emotion, drug use during adolescence or belonging to a minority group are contributing causes of a large number of schizophrenia onset incidents and their post-onset course (Kavanagh 1992; Sundquist et al. 2004; Broome et al. 2005; Van Os et al. 2008; van Os et al. 2010; Pharoah et al. 2010). Such findings support that schizophrenia cannot be fully understood just by looking at a person's brain or his genetic makeup but, rather, the interplay between genes/physiology and psychosocial/environmental factors needs to be taken into consideration when trying to understand the causes, and the nature, of schizophrenia. So since both genetic and environmental factors are causally relevant, to give primary importance to a genetic explanation seems as unwarranted as giving primary importance to environmental explanations because the environment triggers the gene expression.

Of course, on its own the fact that environmental factors contribute to the onset of mental disorder is not enough to preclude an exclusively reductive explanation. As Mitchell (2008) has argued about genetic explanations, if the non-genetic components merely act as background conditions, genes can still be offered as the cause, in the same way that though oxygen is necessary for a match to light, it is still my striking of the match that causes it to light. However, in the cases of mental disorders the presence of environmental

factors affects gene expression in a stronger sense: the very causal properties of the genes involved depend on environmental factors, so a reductive description solely in terms of genes would not be a complete causal explanation since the behaviour of the parts is itself a function of the behavior of the whole. When a system's properties depend not only on its composition but also on the context sensitive organization of the properties of its parts, then reductive explanations will fail (Mitchell 2008, pp. 127–129). This is important not only on a theoretical level but also on a clinical one. If we know that an individual has a strong genetic predisposition towards a disease we can preventively address this or manage its symptoms by intervening on environmental factors that (can) act as triggering agents.

Beyond the lack of available or foreseeable reduction, the importance of the causal history of a disorder also speaks for the necessity of pluralistic explanations in psychiatry. For instance, one can be depressed because of financial insecurity in a collapsing economy, because of a loss and so on, or one can be depressed as a result of the purely physical causes of a disease, say dementia. In all such cases the neurobiological correlates of the depression are probably the same, yet it could be a perfectly normal response of a healthy individual to exhibit depressive behaviour for a certain period of time as a response to, say, the death of a loved one or a painful divorce. In such a case looking merely at one level would not give us a full and correct view.

Yet another reason to adopt a pluralistic view of mental disorders is that it is not clear how reductive views can explain how interventions at higher (psychological) levels, i.e. non-pharmacological therapies, can be effective, as evidence shows that they are. Indicatively, though schizophrenia has medication as an intervention it usually also requires non-pharmacological intervention like family therapy or CBT. Studies show that patients that undergo family therapy in addition to taking their medication maintain improvement and have a lower relapse rate than patients who are only administered medication. Also, CBT when used with antipsychotic medication has a proven role in the management of positive symptoms of schizophrenia (Mueser et al. 1997; Dickerson 2000; Turkington et al. 2004; Pharoah et al. 2010). Similar conclusions can be reached by looking at PTSD. This is a disorder at the other end of the spectrum from schizophrenia in the sense that the latter is usually taken to be the paradigmatic example of a disorder in which genetic/physiological factors are very important, while for PTSD environmental factors are pivotal. PTSD is an anxiety disorder caused by severely traumatic events like being in a severe accident, being sexual abused or having been to war. However, not everyone that undergoes a severely traumatic experience develops PTSD; apart from the severity of the stressor,

whether someone will develop PTSD also depends on the psychological support he will get after the trauma is inflicted and the personal and emotional resources that the person has for coping with his experiences (Andreasen 2001, p. 303ff.). Such, and other, anxiety disorders are treated predominantly with behavioural and CBT interventions and evidence clearly shows that individual and group trauma focused CBT, eye movement desensitization and reprocessing, and stress management are effective in the treatment of PTSD (Bisson and Andrew 2007).

Also, though the causes of PTSD are environmental they physiologically affect the brain. Patients with severe PTSD have shrunken hippocampi due to persistent high levels of adrenalin and cortisol as a result of exposure to trauma (Andreasen 2001; Winter and Irlle 2004; Jatzko et al. 2006). Similarly, studies show that experiences at the personal level, including psychotherapy, can induce physiological changes in a person's brain like medication does. Findings such as these strengthen the support for top-down causation (Le Van Quyen et al. 1997; Schmid-Schonbein 1998; Mayberg et al. 2002; Fuchs 2004).

It is, therefore, an established fact in psychiatry that for mental disorders non-pharmacological therapy works, even if sometimes only in conjunction with medication, and there is evidence that psychotherapy affects the physiology of the brain of patients. And even though we don't know the mechanism through which psychological experiences have consequences at the physical level, the effectiveness of non-pharmacological therapies is, at least *prima facie*, evidence that causation in mental illness is not only multifactorial but also top-down.

So it seems that empirical evidence is in accord with the view that we need to approach mental disorders from different perspectives at different levels if we hope to achieve a comprehensive understanding of them. For instance, to use Murphy's (2006, p. 262–263) example of eating disorders, we may appeal to social factors to explain why there is a higher rate of eating disorders in some parts of the world, but that will not suffice to explain why some individuals exposed to these factors suffer from it while others do not. For this, a biological explanation may be what is needed.

Yet, though lip service is paid to the attempt to bring together the different levels, such integration is to a large extent currently missing from the actual practice of psychiatry where the predominant tendency is to have different explanatory models for different disorders (Harland et al. 2008)—something that may in part be due to the practical training of psychiatrists. So what is needed is to try and incorporate pluralism more in medical practice.

Some studies suggest that this lack of integration is the result of a reductionist bias that is exhibited by the fact that in many cases medication is the first choice in dealing with

a disorder and is often seen as the only efficient way to deal with certain symptoms (Olfson et al. 2002; Olfson and Marcus 2010; Deacon 2013). Though, undoubtedly, this is sometimes correct—in the sense that medication is often the default choice in dealing with mental disorders and sometimes the tendency to (over)medicate *is* the result of a reductionist bias—it is not always the case. The tendency to medicate and the commitment to reductionism need not go hand in hand.

In the health sciences we often need to be pragmatic, and prescribing medication and searching for pharmacological therapies that contain symptoms is less complicated, less expensive and less time-consuming than addressing and researching other relevant factors that can lead to long-term solutions. Also, addressing sociocultural factors or have patients undergo psychotherapy will not have the immediate effects that a drug will have and will cost more. This pragmatic aspect of a clinician's work highlights the fact that though we may use one way to intervene in a mental disorder, this does not necessarily imply something about the causes of mental disorders. For instance, a doctor may administer drugs for a specific disorder not because she is committed to its causes being purely physical but because that is what works. So the tendency towards use of drugs may to some extent also be the result of factors other than a bias towards reductionism—for instance sometimes therapies show weak results and have undesirable side effects and, sometimes, patients themselves refuse interventions other than pharmacological ones<sup>2</sup> (which, again, may be a result of pragmatic considerations or theoretical commitments).

Having said that, however, given that we don't know precisely how drugs work, why they help some patients but not others, and that we don't know the causes of mental illness, it is true that focusing on medication can lead us astray from the search for a pluralistic explanation of a disorder—for instance, by resulting in allocating most funding to biomedical research.

Of course, a thorough-going reductionist will retort at this point that environmental factors act through mechanisms that are themselves mechanistically explainable,<sup>3</sup> so there is no need to bring in explanations other than physical ones. But the point being presently made is that though one could arguably make the case for mechanisms all the way up (and down) we have no reductive explanations—in the sense of being able to describe a purely *physical* mechanism—of how psychosocial factors affect brain biology, as we have evidence that they do. How is it, for instance, that the semantic properties of what is being said in

<sup>2</sup> I'd like to thank an anonymous reviewer for bringing this point to my attention.

<sup>3</sup> I owe this point to an anonymous reviewer.

psychotherapy sessions affect the brain structure of patients? Regardless of the insistence on the side of the reductionist that it must be a purely physical mechanism that explains this, we have no idea how that can be the case—as Jerry Fodor (1984, 232) put it 30 years ago, “the semantic (and/or the intentional) [proves] permanently recalcitrant to integration to the natural order”. This is not to say that no theoretical progress has been made, but it is to say that we do not have much to show when it comes to explaining just how different levels connect. In fact, even when it comes to describing mechanism in all their details, in most cases of cognitive mechanisms we still have gaps in our understanding.

Still, a reductionist might argue, though we have not yet managed to reduce mental disorders, reductionism as a methodological strategy has been very fruitful in other fields so it is plausible to assume that it will be fruitful in psychiatry also. But the fact remains that we have no biological markers for the vast majority of disorders and we have evidence that they are the result of a variety of cooperating factors—and this is of vast importance in psychiatry where what is at play is the well-being of patients who might be getting less than the best we can currently do to help them based on reductionist hopes.

However, though a thorough-going reductionism does not seem to be enough to tackle the complexity of mental disorders it is undoubtedly true that the reductionist methodology has offered valuable explanations in terms of their neurobiological underpinnings—our understanding of which, albeit limited, has already changed our view of mental disorders and has helped us find drugs to address some of the symptoms that such disorders exhibit. Adopting this sort of pluralism, then, that combines aspects of both the reductionist and the anti-reductionist perspective is not to exclude any methodology or divide research, but, instead, is a recognition of the fact that psychiatry has different concerns and aims that need to all be taken into consideration—e.g. the patient as a biological organism who, though, is also part of a larger ecosystem and in a constant commercium with his sociocultural and physical environment, our ultimate aim to understand a mental disorder in its entirety, our need to tend to the needs of patients, our desire to find cures and methods of prevention and so on.

### Is a physical pathology necessary?

A caveat is needed here. According to some reductionist versions of the medical model, mental disorders are the result of pathologies of the brain. Though it is true that medicine looks for differences in brain function that correlate with clinical mental symptoms, this does not mean

that these differences—or, better, deviances—are the cause of mental disorders.

Though we might find differences at the neurological level between healthy individuals and people with mental disorders, these differences need not necessarily count as disorders at that level. Nor do they necessarily imply the presence of a disorder at all; if London taxi drivers have different size hippocampi than the rest of us as has been suggested, that doesn't mean they are sick. The idea of something being disordered at a certain level implies that there also can be a kind of order at that level. But just because there is a physical difference that does not imply a physical disorder because there is no particular physical order (i.e. an order describable by physics plus a physiological description of the ways the physical parts are organised) which this difference counts as disturbing.

So in order to explain a disorder of rational behaviour (disorders that affect the central, rational, capacities of the mind) it is not enough to identify a physiological difference in the brains of people afflicted with it compared to healthy individuals. Those will be present, of course, since there cannot be a mental difference without a difference in the brain, but we also need an independent explanation of why it is a disorder rather than just a difference or abnormality. Of course, underlying causes, if such there be, are important in order to get a complete understanding of mental disorders. But if some mental disorders are functional disorders of rational behaviour in which a system fails to perform a certain function, then insisting on the presence of a physical pathology is unwarranted, since a person afflicted with a mental disorder may be simply working on faulty input acquired through learning, rather than have something physically wrong with his brain.

It should be noted also that even if neurological or genetic reductions were available, that is, if strong correlations were found at different levels—e.g. if high levels of a neurotransmitter or a gene or a gene cluster were found to be strongly correlated with a symptom of schizophrenia—there would still remain the explanatory gap of why an increase in these chemicals causes these specific symptoms rather than different ones or why these symptoms with this particular content rather than with another. Much like there is gap when it comes to explaining correlations of cognitive deficits and the symptoms that follow from them. Of course, this does not mean that because there is an explanatory gap there is no causal relation between such variables. If interventions on one level consistently produced the same effect on the other, we should accept a causal relation despite the explanatory gap and despite our a priori intuitions on the matter (Campbell 2008; Kendler and Campbell 2009).

So, given the currently available evidence there is no guarantee that there are exclusively physical mechanisms that bring about mental disorders and, even if we could find

some sort of a physical or cognitive mechanism it is not clear that we could connect it in an explanatorily useful way to mental disorders at the cognitive level—as we currently cannot do. In this sense, reductive views that have the inbuilt assumption that physical mechanisms are there to be found and that they will be explanatorily sufficient for understanding mental disorders put the cart before the horse.

### An interventionist view of causation

The fact is that though we strive for an integration of levels and for a complete understanding of the mechanisms involved, when it comes to health issues we must often make do without complete knowledge of the mechanisms involved and we need to go instead with the ways we can intervene to help. So a natural way to understand causes in the present context is as difference makers that we can intervene on. This is central because, as Cartwright (1979) has argued, in the sciences causes are important for instrumental reasons; we want to achieve a certain result—in the health sciences either to prevent or to cure—so our aim is to identify the variables that we can effectively intervene on in order to affect an undesirable outcome and hopefully change it to make a difference in a patient's experience.

Since when it comes to the health sciences causation is associated with effective interventions, with making a difference, what is needed is a view that makes sense of such causation. As Kendler and Campbell (2009) have already suggested, such a view is available in the interventionist theory that sees causation as a relation between variables without posing a priori constraints on what these variables can be or to what level they belong to (Pearl 2000; Woodward 2000, 2003; Craver 2007). According to the interventionist theory, variable X causes variable Y if an ideal intervention on X would affect the value of Y (or the probability distribution over values of Y)—where an ideal intervention is understood as an intervention in which the manipulation of X changes the value of Y and the changes in value of Y occur *only* as a result of the causal connection to X, that is, through a causal route that goes through X. Unlike other views about causation, interventionism starts with the way the notion of cause is used in scientific practice (Pearl 2000) where it is associated with effective interventions, with making a difference. So explanation is understood in terms of a counterfactual dependence that would tell us how the behavior of a system would change if a certain variable were intervened upon.

Because the interventionist theory does not require explanations to be in terms of laws of nature or physical mechanisms, it denies the assumption that all real causal

work must happen at the biological level. So it has the advantage over reductive views that it is more generally applicable since it can accommodate the possibility of top-down causation. Therefore, it is compatible with the currently available evidence in psychiatry because it allows many different variables to have a causal role in mental disorders and it can take the evidence from the effectiveness of psychotherapy at face value allowing for both physical causation and psychological causation.

According to the interventionist view, explanation is to be understood in terms of what-if-things-were-different questions. This view can be combined with the mechanisms view by seeing where one can intervene in a mechanism to change its behaviour (Craver 2007, 93ff). So while looking for mechanistic explanations of mental disorders and trying to fill in the largely incomplete picture of these disorders that we now have, we can pragmatically address mental health issues by understanding causal claims within an interventionist framework and address the immediate needs of patients through the variables that we can intervene on. In fact, this is very much the idea behind what most practicing clinicians actually do.

### A theory of mind?

We have talked of reductionism as the alternative to dualism, however, though the two views are related it is not quite right to treat them as alternative views of the mind. Dualism is an ontological view about the mind which means that it is a view about what kind of thing (if it is a thing at all) the mind is. Reductionism, in contrast, is a view about explanation, though it does have ontological ramifications. The ontological view associated with reductionism is physicalism, the view that everything is or depends for its existence on the physical (where the physical is understood as the subject matter of physics). This view takes reductionism all the way down, as a reductionist consistently ought to: if everything is explainable in terms of its constitutive parts that will eventually lead us to physics. A central tenet of physicalism is that all real causation takes place at the physical level and so apparent cases of top-down causation reflect our incomplete knowledge and are nothing but epiphenomena. In other words, whatever causal effects higher-level mental properties appear to have are, in fact, causal effects of underlying physical properties. So for reductionists everything is in principle explainable in terms of its physical parts and their interaction alone, and top-down causation is not real.

However, in this paper two main reasons have been put forward to question the physicalist's privileging of the physical level of reality. The first reason is that for at least

some mental properties, there are no available reductions nor any guarantee that they are forthcoming or even possible. The second reason is that causes at different levels seem to be involved in the onset of mental disorders and we can manipulate variables at different levels to bring about changes in the mental states of patients. These two reasons allow us to question the physicalist assumption of a privileged level of causation.

As we have seen, empirical evidence from psychiatry suggests that we need to adopt an explanatorily pluralistic methodology when it comes to mental disorders. So it would seem that we need a view of the mind that allows for explanatory pluralism and top-down causation and that is consistent with empirical evidence. I propose that emergentism is such a view.

The basic tenets of the kind of emergentism I have in mind are, firstly, that at different levels of organization and complexity matter exhibits different properties that are novel relative to the lower levels of organization from which they emerged. Second, that novel emergent properties are something ‘over and above’ the physical properties from which they emerge, that is, they are irreducible to the matter they emerged from. And third, that these higher-level properties are causally efficacious, including towards lower levels.

Emergentism about the mind starts with the unquestionable premise that mental function is dependent on brain function and that any mental change will mirror a change in the brain. But in contrast to reductionist views, according to emergentism mental states cannot be explained in terms of physical facts of the brain alone—that is, mental states are irreducible to physical states, they have no complete low-level mechanistic explanation. Also, since according to emergentism when a system reaches a certain level of organizational complexity new properties emerge that can be causally efficacious in ways that cannot be reduced to the causal efficacy of its constituent parts, emergentism accepts the existence of top-down causation and thus is compatible with the effectiveness of psychotherapy.

Thus understood we can locate emergentism between the extremes of reductionism and substance dualism. Emergentism is neither dualist in the Cartesian sense which posits two different kinds of things, nor anti-biological since mental phenomena depend on biological phenomena. So though emergentism involves different kinds of properties and so it is a property dualist view, it is a monist materialist view since it sees the world as made up of fundamentally one kind of thing; matter. When this matter reaches a certain level of complexity, however, irreducible properties emerge, i.e. mental properties. This does not mean that something is added to the body from the outside like in Cartesian dualism. Rather, in the words of an early

emergentist, novel properties “blossom out” of the pre-existing matter.

It is true that some philosophers and scientists believe that positing irreducible properties is at odds with science and that there *must* be a purely physical explanation of them (e.g. see McGinn 1989, 353). But despite the insistence that mental properties must be physical somehow, the fact remains that science has not yet managed to reduce such properties and the nature of the phenomenal and intentional properties of mental states makes the possibility of such reduction questionable. In fact, it can be argued that a property dualist view is a more naturalist view than reductive views at least in the (loose) sense that it is in line with scientific evidence that brains are plastic and are shaped by experience, including psychotherapy, and with the fact that there are no currently available reductions of mental states nor of the mechanisms through which mental to physical (and vice versa) causal interactions taken place.<sup>4</sup> Of course, this does not establish that mental properties are not physical, but it gives us strong reasons to believe that they are not. And, anyway, even if such reductions turn out to be possible in the end, physicalists at the present time do not have the right to assume that they will.

So emergentism offers a view of the world in which reality is structured in aggregates of different orders (with each order being made up of the kinds in lower orders) with higher orders exhibiting different organizational complexity and new principles of organisation. This is compatible with the principle of the ubiquity of physics, according to which physical principles apply universally and constrain the motions of all physical systems (Anderson 1972; Cartwright 1999; Hendry 2010: 217ff). This is because the principle of ubiquity does not entail that physical principles determine every physical effect—nor does it follow from it that that we can build the whole universe just by knowing the laws physics and the properties of elementary particles (Anderson 1972: 393).

At the same time emergentism provides a plausible potential way of bridging the explanatory gap: if it turns out that all we can get is a nomological connection of physical states to disordered mental states, though we will not have the explanation reductionists require and the explanatory gap will persist, it will not be a problem anymore. Since, according to emergentism, it is possible that among the actual world’s fundamental facts, there are facts concerning the correlation of brain states to conscious states.

So if we see the mind through the emergentist perspective, in order to understand mental phenomena, including mental disorders, we need to take into account

<sup>4</sup> For an extended argument for this position see Vintiadis (2013).



different levels and kinds of explanation and this has important implications for the autonomy of psychiatry. For if the personal level is irreducible to the biological level, then psychiatry is an autonomous discipline that can coexist with neuroscience and related disciplines and is not just a placeholder for more mature versions of the latter. What is more, it is indispensable for a full understanding of the disorders of the mind.

## Conclusion

Psychiatry is important because millions of people are afflicted by mental disorders around the world and so understanding them can help alleviate their suffering. With this in mind, looking at currently available evidence from psychiatry I have argued that in order to understand mental disorders we need to use different approaches in different contexts and that, rather than putting all our eggs in one basket, we need to leave behind reductionist presuppositions and hopes and adopt a broader explanatory strategy. Such a strategy could be an explanatory pluralism in which the different sciences do not work on different aspects of problems in isolation but that cooperate to the extent possible to gain a full understanding of mental disorders.

Understanding mental disorders in a multilevel way involving the interplay of organic, environmental and sociocultural factors leads to a reconception of mental disorders in which it is acknowledged that they are much more complex than was originally thought. It also gives support to a view of the mind that sees it as intimately connected to its environment. This suggests that the mental health sciences stand to gain if they maintain a dialogue not only with the brain sciences but also with disciplines that have the psychosocial environment as their object of study. Such a dialogue can also have repercussions for clinical practice by expanding the scope of possible variables we can intervene on to address mental health issues—for instance, we may be able to intervene at the social level on different variables that can act as triggering agents for psychiatric conditions. In addition, such a dialogue can inform public policy in matters of mental health on a range of issues, starting from training and allocation of resources to questions of access to mental health services and workplace policies.

While retaining a materialist view, we must also resist reductionist views that want to make reality the privilege of only fundamental entities. This suggests a view of the mind, emergentism, according to which the mind depends for its existence on the brain yet that also does justice to the idea that the mind does not develop from the brain in isolation but is the result of complex interactions of the brain-body environment as well as its interaction with the surrounding psychosocial environment. Emergentism is consistent with

the known laws of physics and their universal application, it does not posit anything that smacks of the supernatural and also does not deny the unique status of conscious experience. This allows us to retain psychiatry as an autonomous science that can productively co-exist with neuroscience while also bringing to the forefront the need for an extended interdisciplinary dialogue in our effort to understand the nature of the mind and the various aspects of human cognition.

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## References

- Anderson, P.W. 1972. More is different. *Science* 177: 393–396.
- Andreasen, N.C. 2001. *Brave new brain: Conquering mental illness in the era of the genome*. Oxford: Oxford University Press.
- Andreasen, N.C. 1984. *The broken brain: The biological revolution in psychiatry*. New York: Harper and Row.
- Bickle, John. 2003. *Philosophy and neuroscience: A ruthlessly reductive account*. Dordrecht: Kluwer Academic Publishers.
- Bisson, J., and M. Andrew. 2007. Psychological treatment of post-traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews* 2007, Issue 3. Art. No.: CD003388. doi:10.1002/14651858.CD003388.pub3.
- Broome, M.R., J.B. Woolley, P. Tabraham, L.C. Johns, E. Bramon, G.K. Murray, C. Pariante, P.K. McGuire, and R.M. Murray. 2005. What causes the onset of psychosis? *Schizophrenia Research* 79: 23–34.
- Campbell, John. 2008. Comment: Psychological causation without physical causation. In *Philosophical issues in psychiatry*, eds. K. S. Kendler, M. D. Parnas, 184–196. Baltimore, MA: The John Hopkins University Press.
- Cartwright, N. 1979. Causal laws and effective strategies. *Nous* 13: 419–438.
- Cartwright, N. 1999. *The dappled world*. Cambridge: Cambridge University Press.
- Craver, Carl F. 2007. *Explaining the brain*. USA: Oxford University Press.
- Deacon, Brett J. 2013. The biomedical model of mental disorder: A critical analysis of its validity, utility, and effects on psychotherapy research. *Clinical Psychology Review* 33: 856–861.
- Dickerson, F.B. 2000. Cognitive behavioural psychotherapy for schizophrenia: A review of recent empirical studies. *Schizophrenia Research* 16: 71–90.
- Dupré, John. 1993. *The disorder of things: Metaphysical foundations of the disunity of science*. Cambridge: Harvard University Press.
- Flashman, L.A., and M.F. Green. 2004. Review of cognition and brain structure in schizophrenia: Profiles, longitudinal course, and effects of treatment. *Psychiatric Clinics of North America* 27(1): 1–18.
- Fodor, J.A. 1984. Semantics Wisconsin style. *Synthese* 59: 231–250.
- Fuchs, T. 2004. Neurobiology and psychotherapy: An emerging dialogue. *Current Opinions in Psychiatry* 17: 479–485.
- Harland, R., E. Antonova, G.S. Owen, M. Broome, S. Jandau, Q. Deeley, and R. Murray. 2008. A study of psychiatrists' concepts of mental illness. *Psychological Medicine* 39: 967–976.

- Hendry, R.F. 2010. Emergence vs. Reduction in Chemistry. In *Emergence in mind*, eds. C. Macdonald, G. Macdonald. New York: Oxford University Press.
- Jatzko, A., S. Rothenhöfer, A. Schmitt, C. Gaser, T. Demirakca, W. Weber-Fahr, M. Wessa, V. Magnotta, and D.F. Braus. 2006. Hippocampal volume in chronic posttraumatic stress disorder (PTSD): MRI study using two different evaluation methods. *Journal of Affective Disorders* 94: 121–126.
- Kandel, Eric R. 1998. A new intellectual framework for psychiatry. *American Journal of Psychiatry* 155: 457–469.
- Kavanagh, D.J. 1992. Recent developments in expressed emotion and schizophrenia. *British Journal of Psychiatry* 160: 601–620.
- Kendler, K.S., and J. Campbell. 2009. Interventionist causal models in psychiatry: repositioning the mind–body problem. *Psychological Medicine* 39: 881–887.
- Kendler, K.S., John M. Hettema, Frank Butera, Charles O. Gardner, and Carol A. Prescott. 2003. Life event dimensions of loss, humiliation, entrapment, and danger in the prediction of onsets of major depression and generalized anxiety. *Archives of General Psychiatry* 60: 789–796.
- Kidd, Kenneth K. 1997. Can we find genes for schizophrenia? *American Journal of Medical Genetics* 74: 104–111.
- Le Van Quyen, M.C., C. Adam, J.P. Lachaux, J. Martinerie, M. Baulac, B. Renault, and F.J. Varela. 1997. Temporal patterns in human epileptic activity are modulated by perceptual discriminations. *NeuroReport* 8: 1703–1710.
- Machamer, P.K., L. Darden, and C. Craver. 2000. Thinking about mechanisms. *Philosophy of Science* 67: 1–25.
- Mayberg, H.S., J.A. Silva, S.K. Brannan, J.L. Tekell, R.K. Mehurin, S. McGinnis, and P.A. Jerabek. 2002. The functional neuroanatomy of the placebo effect. *American Journal of Psychiatry* 159(5): 728–737.
- McGinn, Colin. 1989. Can we solve the mind–body problem? *Mind* 98: 349–366.
- Mekios, Constantinos. in press. Explanation in systems biology: is it all about mechanisms? In *Explanation in biology: An enquiry into the diversity of explanatory patterns in the life sciences*, eds. Pierre-Alain Braillard kai Christophe Malaterre.
- Mitchell, Sandra D. 2004. Why integrative pluralism? *Emergence, Complexity and Organization* 6(1): 81–91.
- Mitchell, Sandra D. 2008. Comment: Taming causal complexity. In *Philosophical issues in psychiatry*, eds. K. S. Kendler, Josef Parnas. Baltimore, MD: Johns Hopkins University Press.
- Mueser, Kim T., Robert E. Drake, and Gary R. Bond. 1997. Recent advances in psychiatric rehabilitation for patients with severe mental illness. *Harvard Review of Psychiatry* 5: 123–137.
- Murphy, Dominic. 2006. *Psychiatry in the scientific image*. Cambridge, MA: MIT Press.
- Olfson, M., and S.C. Marcus. 2010. National trends in outpatient psychotherapy. *American Journal of Psychiatry* 167: 1456–1463.
- Olfson, M., S.C. Marcus, M.M. Weissman, and P. Jensen. 2002. National trends in the use of psychotropic medications by children. *Journal of the American Academy of Child and Adolescent Psychiatry* 41(5): 514–521.
- Pharoah, F., J.J. Mari, J. Rathbone, and W. Wong. 2010. Family intervention for schizophrenia. *Cochrane Database of Systematic Reviews* 12. Art. No.: CD000088. doi:10.1002/14651858.CD000088.pub3.
- Pearl, J. 2000. *Causality*. Cambridge: Cambridge University Press.
- Schaffner, K.F. 1994. Psychiatry and molecular biology: reductionist approaches to schizophrenia. In *Philosophical perspectives on psychiatric diagnostic classification*, eds. J. Sadler, O. Wiggins, M. Schwartz. Baltimore, MD: Johns Hopkins University Press.
- Schmid-Schonbein, C. 1998. Improvement of seizure control by psychological methods in patients with intractable epilepsies. *Seizure* 7: 261–270.
- Sundquist, K., G. Frank, and J. Sundquist. 2004. Urbanisation and incidence of psychosis and depression: Follow-up study of 4.4 million women and men in Sweden. *British Journal of Psychiatry* 184: 293–298.
- Turkington, D., R. Dudley, D.M. Warman, and A.T. Beck. 2004. Cognitive-behavioral therapy for schizophrenia: A review. *Journal of Psychiatric Practice* 10: 5–16.
- van Os, J., J. Mitchel, B.P. Rutten, and R. Poulton. 2008. Gene–environment interactions in schizophrenia: Review of epidemiological findings and future directions. *Schizophrenia Bulletin* 34: 1066–1082.
- van Os, J., G. Kenis, and B.P.F. Rutten. 2010. The environment and schizophrenia. *Nature* 468: 203–212.
- Vintiadis, Elly. 2013. Why a naturalist should be an emergentist about the mind. *Sats* 14(1): 38–62.
- Wimsatt, William C. 2000. Emergence as non-aggregativity and the biases of reductionisms. *Foundations of Science* 5: 269–297.
- Hermann, Winter, and Irlé Eva. 2004. Hippocampal volume in adult burn patients with and without posttraumatic stress disorder. *American Journal of Psychiatry* 161: 2194–2200.
- Woodward, J. 2000. Explanation and invariance in the special sciences. *British Journal for the Philosophy of Science* 52: 197–254.
- Woodward, J. 2003. *Making things happen*. New York: Oxford University Press.