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PAPER IN HISTORY AND PHILOSOPHY OF SCIENCE

Kant's essentialism and mechanism and their relevance for present-day philosophy of psychiatry

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Abstract

This paper aims to evaluate the relevance of Kant's much discussed essentialism and mechanism for present-day philosophy of psychiatry. Kendler et al. (Psychological Medicine 41(6):1143–1150, 2011) have argued that essentialism is inadequate for conceptualizing psychiatric disorders. In this paper, I develop this argument in detail by highlighting a variety of essentialism that differs from the one rejected by Kendler et al. I show that Kant's essentialism is not directly affected by the argument of Kendler et al. (Psychological Medicine 41(6):1143–1150, 2011), and that Kendler et al.'s (Psychological Medicine 41(6):1143–1150, 2011) argument also does not affect other essentialist positions in psychiatry. Hence, the rejection of essentialism in psychiatry needs more arguments than the one supplied by Kendler et al. Nevertheless, the study of current psychiatry also provides reasons to reject Kant's essentialism and his transcendental project. I argue that Kant's theory of mechanical explanation is more relevant for analyzing present-day philosophy of psychiatry, insofar as (a) modern psychiatric research into the causes of psychiatric disorders fits the mechanist paradigm, (b) Kant's theory of mechanical explanation is importantly similar to modern theories of mechanical explanation applicable to psychiatry, such as those of Bechtel and associates, and (c) Kant's stance that mechanism constitutes a regulative ideal points to useful arguments for the pursuit of mechanical explanations in psychiatry.

Keywords Kant · Essentialism · Mechanism · Psychiatry

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1 Introduction

Psychiatry is a complex science, with clinical psychiatry viewed as a medical specialty that diagnoses and treats mental disorders. Some researchers in psychiatry are concerned with explaining the causes of mental disorders, an enterprise that is often taken to also have value for the classification of mental disorders (see Hyman, 2010, who argues that in disease classification the gold standard is etiology). In this paper, I will look into the relevance of Kant's essentialism and mechanism for research into the nature and causes of mental disorders. I will evaluate the scope of Kendler et al.'s (2011) critique of essentialism, which holds that psychiatric disorders are caused by an essence or single causal agent. I will also argue that the search for mechanical explanations in psychiatry constitutes a fruitful research programme. I follow Kendler et al (2011) as taking the best psychiatric science to imply that mental disorders are features of the brain that can have multiple causes (genetic, psychological, environmental, interactions among symptoms, etc.).

In recent philosophy of psychiatry, there has been debate on whether psychiatric disorders constitute natural kinds (See Hacking, 1991, 2007; Cooper, 2004, 2014). There is a related discussion on whether essentialism can help us conceptualize the nature of psychiatric disorders (Adriaens & De Block, 2013). Kendler et al (2011) argue that essentialism cannot capture the nature of psychiatric disorders. According to these authors, essentialists assume that for psychiatric disorders there exists a single etiological agent that explains all of the defining features of a disorder (ibid.). Kendler et al (2011) reject essentialism on empirical grounds because this assumption is mistaken (see also for earlier discussion Samuels, 2009).

In this paper, I argue that history shows that there are different theories of essentialism, whereas Kendler et al (2011) mistakenly construe essentialism as a uniform view. I reconstruct the essentialism of Immanuel Kant (1724–1804), which is intimately tied to his transcendental project. I show that Kant's essentialism is not directly affected by the argument of Kendler et al (2011). In addition, I show that there are other variants of essentialism in psychiatry that Kendler et al. (2011) argument does not affect. Hence, the rejection of essentialism needs more arguments than the one supplied by Kendler et al. Nevertheless, I show that Kant's essentialism needs to be rejected in light of modern science. More specifically, I argue that an analysis of the plurality of concepts of causality and methods of causal inference in psychiatric research shows that the idea that there are necessary a priori conditions of psychiatric research must be rejected. Hence, core assumptions of Kant's project are unable to do justice to the fact of pluralism in concepts of causality and methods of causal inference. My motivation for discussing essentialism is partly that it highlights facets of Kant's philosophy that are not compatible with assumptions concerning present-day philosophy of science that are upheld by a significant amount of people, such as pluralism. This is an important conclusion when evaluating the relevance of Kant's philosophy of science for present-day science. Kant's conception of mechanical explanation, developed while discussing the life sciences, is more relevant than

his essentialism for understanding current psychiatric research. I argue that (a) modern psychiatric research into the causes of psychopathology fits the mechanist paradigm, (b) that Kant's theory of mechanical explanation is similar to modern theories of mechanical explanation, such as those of Bechtel and associates, and (c) that Kant's stance that mechanism constitutes a regulative ideal points to useful reasons for the pursuit of mechanical explanations in psychiatry.

In the end my judgment on Kant will thus be mixed. It is important to stress that I will not be concerned with providing a merely historical contribution. Although the paper has historical relevance, especially the highlighted similarities between Kant's concept of mechanism and that of present-day mechanists such as Bechtel, I will discuss central and accepted tenets of Kant's thought and evaluate in how far they are relevant for present-day philosophy of psychiatry. I will also not be concerned with discussing the relevance of Kant's views on psychology, philosophy of mind, and anthropology for present-day psychiatry, nor his views on mental disorder (see on these topics Sturm, 2009; Frierson, 2014; Scholten, 2016; Kraus, 2020). Kant's views on the latter topics can be interpreted as being quite different from those of many present-day psychiatric researchers. Sturm (2008, 2009) has argued that Kant rejected (neuro-)physiological explanations in his anthropology, arguing that the investigation into how the organs of the body are related to thoughts is *irrelevant* for his pragmatic anthropology. Many researchers within modern psychiatry do not take (neuro) physiological investigations to be irrelevant. Hence, Kant's views on the science of the human mind may be different from the parts of his philosophy that I identify as being relevant for present-day psychiatric research.

What justifies the comparison between Kant and present-day philosophy of psychiatry? I argue that this comparison provides more insight into discussions of essentialism in philosophy of psychiatry and into the fruitfulness of searching for mechanical explanations in psychiatry. I will discuss Kant's account of essence and show that Kendler et al (2011) define essentialism as the idea that psychiatric disorders have a single cause. However, Kant has a completely different account of essence, according to which a priori features ascribed to objects constitute an object's essence. Kendler et al.'s (2011) argument is thus ineffective against Kant's version of essentialism, and, as I will argue, against other versions of essentialism in psychiatry. Hence, a rejection of essentialism in psychiatry requires more arguments than developed by Kendler et al.

Against present-day Kantians I argue for the limited fruitfulness of Kant's essentialism and his transcendental philosophical framework for understanding present-day pluralist science, which psychiatry exemplifies. In a recent paper, the Kantian inspired philosophers Breitenbach and Choi (2017) argue for a Kantian conception of the unity of science as a regulative ideal. More specifically, they argue for a unified pluralism, where the advantages of pluralist science are tied to a collaborative ideal of the unity of science, in which we seek for example consistency among theories. In this paper I stress with Cartwright (2016) that although we can always, sometimes with good reason, as Breitenbach and Choi claim, adopt an ideal of unity in our investigations, it should also be a task of philosophy of science to describe the thick, varied, and pluriform concepts and methods that pluralist science employs. In this respect, Kant's philosophy is defective, as I will show.

I will finally be concerned with Kant's account of mechanism because, in contrast to his essentialism and his transcendental philosophy, it provides a fruitful systematic framework for understanding present-day psychiatry. My overarching goal is to show through a comparison of Kant and present-day philosophy of psychiatry that Kant's account of mechanism can help provide support for mechanism arguments in psychiatry. Through achieving my goals I show that much psychiatric research fits the mechanist paradigm and, moreover, that there are good reasons to pursue mechanical explanations in psychiatry, even if, as yet, mechanical explanations have been hard to articulate. I articulate a broad conception of mechanism that captures many types of purported explanations in psychiatry.

In Sects. 2, I reconstruct the essentialism and mechanism of Immanuel Kant, relying on existing influential literature. In Sect. 3, I evaluate how Kant's essentialism and mechanism fare in providing an analysis of present-day psychiatric research. Section 3.1 discusses the concept of a multifactorial disease and argues that essentialism, as defined by Kendler et al., must be rejected, but that Kant's essentialism and other versions of essentialism remain unaffected. Section 3.2 discusses different influential conceptions of causation and methods of causal inference in (psychiatric) epidemiology, and argues that Kant's essentialism and his theory of a priori conditions of scientific knowledge cannot make sense of pluralist psychiatric research. Finally, Sect. 3.3 argues that Kant's account of mechanism sheds light on how many psychiatric researchers strive to give mechanical explanations of psychiatric disorders.

2 Kant's accounts of essence and mechanism

In recent years Kant's account of essence has received substantial attention, mainly because of the popularity of the Necessitation Account of laws attributed to Kant, according to which the essential natures of things ground the necessity of laws (see Watkins, 2005, 2019; Kreines, 2009; Stang, 2016; Massimi, 2017; Patton, 2017; Messina, 2017; Breitenbach, 2018; Engelhard, 2018; Cooper, 2023a, 2023b). In this paper, I will restrict myself to making just two points: (a) that Kant's account of real essence, as has been noted by Stang (2016), is related to his transcendental philosophy, insofar as Kant thinks that necessary properties of objects are a consequence of a priori (essential) features we ascribe to objects. This entails that essences are partly mind-dependent and also that, contrary to what some have argued (Kreines, 2009), we know at least part of the real essence of some objects of experience. (b) That Kant's account of mechanical explanation makes mechanism an a priori regulative assumption that constrains the life sciences.

Stang has given a convincing account of essence in Stang (2016, pp. 234–235). He notes that Kant distinguishes between the logical essence of concepts and the real essence of things. The logic essence of a *concept* consists in the partial concepts that are contained in a concept, e.g., the concept "rational" is contained in the concept "man" and thus is part of the logical essence of the latter concept. The real essence of a thing is given by a real definition, and concerns the essence of *objects*. Stang argues that Kant allows for the possibility of knowing (part of) the

real essence of objects of experience. In the *Metaphysical Foundations*, which Stang cites, Kant states:

> A property on which the inner possibility of a thing rests, as a condition, is an essential element thereof, Hence, repulsive force belongs to the essence of matter just as much as attractive force, and neither can be separated from the other in the concept of matter (Kant, 2002, p. 222. AA 4: 511.)

Kant also gives attractive force as an example of belonging to the real essence of matter in the metaphysics lectures (Stang, 2016, p. 251). Stang explains that we can know part of the real essence of matter by arguing that it is in virtue of the fact that we ascribe a priori conditions of experience to objects of experience such as matter that we can know part of their essence, and by arguing that the fact that matter has attractive and repulsive forces is an a priori condition of our being able to experience matter. The fact that matter has attractive and repulsive forces is an a priori condition of our being able to experience matter is thus a reason for taking attractive and repulsive force as belonging to the real essence of matter. Stang's interpretation is consistent with the lectures on logic. There, Kant remarks that we cannot know the complete real essence of *individual objects*, i.e., the complete objective ground of all that belongs to a thing, but that we can know the real essence of the *genera* of objects, for example, the real essence of *body* in general (Kant, 1992, p. 92. AA 24: 118.).

We find further evidence for Stang's reading of Kant's account of essence in Kant's *Opus postumum* and the reflexionen. As van den Berg (2014, p. 218) makes clear, there Kant argues that the essence of things is form, and that the form of things is a priori prescribed to things by the understanding (the categories) and sensibility (the forms of intuition space and time). This fits Stang's interpretation according to which a priori conditions of experience, which are ascribed to objects a priori, constitute an object's essence. Note that on this reading all objects have essential properties. Note further that being a necessary condition for something, as a referee pointed out to me, as for example repulsive force is a necessary condition for experiencing matter, is not sufficient for something being an essence. However, Kant is an idealist who thinks that necessary a priori conditions of an object are *constitutive* of an object, i.e., they make that object what it is, and as such it makes sense to take a priori conditions of the experience of objects to belong to the object's essence. As Kant also sometimes puts it, our mind makes the phenomenal world by projecting a priori concepts and principles upon that world, this is the core of his idealism, and as such these a priori concepts and principles belong to the object's essence. Kant's concept of essence, as a referee pointed out, resembles that of Aristotle insofar as he takes an essence to make an object what it is and to ground the object's necessary properties. Moreover, insofar as he took a priori concepts and principles, such as the principle of causality, to belong to the essence of phenomenal objects, he adopted an essentialist notion of causality that is shared by everything we call a cause, a view that also fits the view of Aristotle. Insofar as Kant denies that we know the real essence of individual objects, his views are, however, somewhat Lockean.

My account of Kant's essentialism is brief and there are competing accounts of Kant's notion of essence (see e.g., Anderson, 1994; Hannah, 1998; Kroon & Nola,

1987). However, I here focus on well established, and in my view uncontroversial, aspects of Kant's essentialism and a full discussion of all Kant debates on essentialism would take up too much space in an article that is ultimately concerned with present-day psychiatry. I find the interpretation of Stang convincing and uncontroversial because it allows us to unify Kant's idealism with tenets of essentialism, and as such allows us to make sense of most of his remarks on concepts of essence in his published works and logic and metaphysics lectures. The unifying power of Stang's interpretation is thus considerable.

Let us summarize Kant's essentialism. For Kant, the a priori features we ascribe to objects that are conditions of having experience of these objects are part of the real essence of objects. For example, Kant argues that space and time are a priori forms of experience, necessarily ascribed to objects of experience. Hence, having a position within space and time is a necessary and essential property of objects of experience. Similarly, being subject to causal relations is a necessary condition of us having experience of individual objects. Hence, standing in causal relations belongs to the essence of individual objects of experience. Note that Kant's version of essentialism is a version of essentialism as defined by present-day authors: (a) essential properties are understood as properties that an object necessarily has (Robertson Ishii & Atkins, 2023). For Kant, these are the a priori features we ascribe to objects. (b) Kantian essentialism can also be understood as a doctrine that states that objects, i.e., objects of experience, have at least some essential properties.

We may now turn to Kant's account of mechanical explanation, which has been discussed by Van den Berg (2013, 2014, chapter 3. See also McLaughlin, 1990, Ginsborg, 2004, 2015, Breitenbach, 2006, 2009a, Cohen, 2009, Geiger, 2022). According to Van den Berg, Kant takes mechanical explanations, which he construes as explanations of wholes in terms of their parts and the structure of the parts, a conception of mechanical explanation that is similar to that of his predecessor Wolff, as an ideal of explanation in natural science. Reason prescribes this ideal of explanation to nature a priori, adopting a maxim of mechanism. Hence, Kant is committed to the claim that, ideally, we should explain properties of organisms mechanically even if—as Kant also notes—this is not always possible. Van den Berg (2014, chapter 3) also argues that mechanical explanations can be construed as a species of causal explanation, and differ from the latter insofar as mechanical explanations specify a mechanism which explains why certain consequences obtain. Finally, like Wolff, Kant took mechanical explanations to provide a reductive procedure that we can continuously apply: thus we can explain wholes in terms of their parts, and then explain these parts in terms of their parts, and so on (Van den Berg, 2014, p. 121).

Kant argued that in the life sciences we should combine teleology and mechanism (Lenoir, 1989). Kant himself states that in the life sciences we should subordinate mechanism to teleology. The idea behind this claim, as many have noted (Breitenbach, 2009b, van den Berg, 2014), is that teleology provides a heuristic strategy for providing mechanical explanations. As one referee stated: teleology specifies explanatory relevance in the life sciences. Once we have identified sight as the purpose of the human eye, we have a teleological description of the eye that guarantees that it is relevant to subsequently provide mechanical explanations of how the human eye is able to perform the function of sight. Kant's claim that we should

subordinate mechanism to teleology also implies that we assume the complex purposive organization of organisms and organs as given before we subsequently try to provide mechanical explanations of these organisms and organs. For example, when attempting to provide mechanical explanations of the process of pregnancy, life scientists may assume the complex organization of the uterus as given and assume teleologically that the uterus is there for providing nutrition to the embryo. The task of the life scientist remains, however, to provide mechanical explanations of this process. Thus, Kant's teleology is completely geared to providing a mechanist research programme, and it is this aspect of Kant's thought that I will argue is relevant for present-day philosophy of psychiatry. The general idea is that if we assume that mental disorders are natural phenomena, e.g., as diseases of the brain, we are compelled to view mental disorders as complex wholes that can be accounted for mechanically in terms of multiple causes (e.g., environmental, psychological, genetic, etc.).

Kant's maxim according to which we should strive to explain organisms mechanically is a *regulative* maxim that guides scientific investigation (Van den Berg, 2014, chapter 3, pp. 84–86). This means, firstly, that the maxim is a guideline that informs scientists on how to proceed in investigations in the life sciences. This maxim is a priori prescribed to scientists and is necessary insofar as it guides us to provide ideal explanations (i.e., mechanical explanations) of nature. However, secondly, the maxim should not be interpreted ontologically. It is, as Van den Berg (2014, p. 85) explains, a methodological principle that we follow in order to obtain the most perfect scientific knowledge of nature, but it does not imply (as Descartes and Wolff thought) that the entirety of nature or all objects of nature actually are mechanisms, nor does it stipulate that all the properties of objects must be explained in terms of a single and necessary mechanism. Kant's principle of mechanism thus remains neutral on the ontological interpretation of nature and on how mechanisms are instantiated in nature, although Kant does think that nature exhibits causal laws (this is a minimal ontological assumption that underlies his account of explanation).

We can further explain Kant's position by noting that although the a priori and necessary principle of causality, which articulates a general conception of causality valid for all objects, is *constitutive* of (our experience of) all objects of nature, and is thus part of their essence, Kant denies that mechanism, which is a *particular species* of causality in which interactions among parts constitute a whole, is constitutive of (our experience of) objects of nature. Rather, the principle of mechanism is *regulative*, which means, in part, that Kant remains neutral on the ontological question of whether objects of nature actually are constituted by mechanisms.

3 Psychiatry, essence, and mechanism

In this section I consider whether Kant's essentialism and account of mechanical explanation allow us to fruitfully analyze present-day research into the causes of psychiatric disorders. In Sect. 3.1, I consider the concept of a multifactorial disease and show, following Kendler et al (2011), that the multifactorial nature of psychiatric disorders provides an argument against one version of essentialism, but not

against Kant's essentialism and other varieties of essentialism. In Sect. 3.2, I discuss conceptions of causation presupposed in psychiatric research and note that our discussion discredits Kant's essentialism and transcendental philosophy. Finally, in Sect. 3.3, I argue that aspects of Kant's account of mechanical explanation provide us with tools to fruitfully analyze research into psychiatric disorders, insofar as mechanical explanations are conceptualized by present-day mechanists in a manner that is very similar to the conceptualization of Kant and insofar as Kant provides sound methodological reasons for pursuing mechanical explanations in science.

3.1 Multifactorial diseases and the systematic relevance of Kant's concepts of essence and mechanism

Kendler et al (2011) provide a very strong definition of an essentialist conception of (causes of) mental disorder as the view that some single essence is causally responsible for all key features of the disorder (p. 1144). To be precise, this is a form of essentialism about causes: a single essence or cause is responsible for all the features of a disorder. Kendler et al (2011) reject essentialist conceptions of (causes of) mental disorders because such disorders are known to be multifactorial, i.e., they arise from a wide range of causes.

To better understand Kendler et al's (2011) argument we can sketch the different conceptions of disease that it refers to. Kendler et al. note that in the nineteenth and early twentieth century some researchers came to believe that single discrete causes exist for major psychiatric disorders, inspired by the demonstration of a single cause for general paralysis of the insane (2011, p. 1144). These researchers adopted a *monocausal model of disease* (Broadbent, 2009). Broadbent (2009, pp. 302–303) provides a clear account of the monocausal model, and notes that what is distinctive of the model is the belief that every disease has one cause that is *necessary* for the disease (the disease does not occur in the absence of the cause) and that is *sufficient* for the disease (the disease always occurs when the cause is present). The conditions are interpreted as implying that diseases have *one* cause (Broadbent, 2009). One problem for this model is that many diseases do not clearly have necessary or sufficient causes (p. 305). Fuller (2018, p. 8) illustrates the point by discussing the risk factors for cardiovascular disease, including stroke, noting that a risk factor like smoking is not sufficient for the disease (not every smoker has a stroke) nor is it necessary (not everyone who has a stroke smokes). The same applies to psychiatric diseases. For this reason, essentialism concerning psychiatric disorders, which is tied to the monocausal model of disease, must be rejected.

The question now becomes whether Kendler et al's (2011) argument provides a reason to reject Kant's accounts of essence and mechanism. I want to argue that the argument of Kendler et al (2011) does not imply that we should reject Kant's notions of essence and mechanism. Let us start with Kant's account of mechanism. We have seen that Kant thinks we should always strive to provide mechanical explanations of nature, but we have also argued that Kant nowhere argues that natural phenomena must be explained in terms of a single and necessary mechanism. Hence, Kant's account of mechanism leaves room for the possibility that phenomena come about

by multiple mechanisms, and can accommodate the view of Kendler et al (2011) that multiple mechanisms bring about psychiatric disorders.

Kant's account of essence, which specifies that a priori features that we ascribe to objects constitute their essence, is tied to his conception of causation, insofar as the concept of cause and causal principles are a priori principles that we ascribe to natural objects. Now Kant's conception of causation is not monocausal in the sense of the monocausal conception of disease. In the second analogy of the first *Critique*, Kant argues for an a priori causal principle according to which "alterations occur in accordance with the law of the connection of cause and effect" (Kant, 1998, p. 304. KrV, B 233. See for classic discussions of causality in Kant, Guyer, 1987, Allison, 2004, Watkins, 2005, De Pierris & Friedman, 2018). This principle implies that every event has a cause, but is silent on the existence of necessary and sufficient causes in the monocausal model of disease. Kant's concept of cause does imply, as Van Cleve (1973, p. 73) has shown, that A causes B if B necessarily follows A according to an absolutely universal rule, which means that whenever an event of kind A occurs, an event of kind B occurs. Hence, Kant's concept of causation implies that a cause of some event is always a sufficient cause of that event (the effect always occurs when the cause is present). However, Kant does not argue that effects must have *necessary causes* and seems to accept the possibility that an effect can have multiple causes. As such, he does not endorse a monocausal conception of causation, which was tied to the doctrine of essentialism by Kendler et al (2011), who rejected essentialism because they rejected the monocausal conception of disease. As such, Kendler et al's (2011) critique of essentialism does not directly affect Kant.

As an anonymous referee has stressed to me, there are also other versions of essentialism in psychiatry which are not affected by Kendler et al.'s (2011) critique of essentialism. Thus, for example, as described by Urfer (2001), the phenomenological psychiatrist Minkoski thought it was the task of phenomenology to grasp the essential features of the patient's experience (Urfer, 2001, p. 281). Psychiatrists must not only describe symptoms, but also their underlying organizing structure (ibid.). Phenomenological psychiatry must describe the essence of disorders, and this is what Minkowski called the *trouble générateur,* the unifying generative essence of a disorder. This is not a single etiological agent, to which Kendler objects. Similarly, as an anonymous referee has stressed, some researchers on cognitive inflexibility in bipolar disorder see cognitive inflexibility as an essential trait or core of the disorder and is in this sense considered to be essential (see O'Donnell et al., 2017 and the discussion in this article). This is an essential trait, not a single cause. Clearly then, there are many versions of essentialism, including that of Kant, and Kendler et al's (2011) argument against essentialism is insufficient to reject all of them. Rejecting essentialism in psychiatry thus requires more arguments than delivered by Kendler et. al. and until these variants are rejected essentialism remains a live option for psychiatry.

Let us now return to Kant. We have seen that Kendler's argument against essentialism does not affect Kant's variety of essentialism. It is probably the case that since Kant thinks that causes are sufficient for their effects, his conception of causation will be rejected by researchers who adopt a multifactorial conception of disease.

After all, these researchers often suggest that diseases do not have sufficient causes. To give a psychiatric example, we might say that cannabis use causes psychosis even if smoking cannabis is not sufficient for developing psychosis (see on cannabis and psychosis, Van Os et al., 2002). I will not develop this train of thought further. Instead, I will in what follows adopt a systematic perspective and argue that a core assumption of Kant's essentialism, namely that sciences are based on a necessary a priori principle of causality, cannot be upheld for modern medicine and psychiatry. An evaluation of Kant's philosophy from the perspective of modern epidemiological and psychiatric research will yield novel insight into concepts of causality and causal inference within psychiatry and a positive argument for pluralism concerning causality in psychiatry. Moreover, in a paper that discusses the relevance of Kant for present philosophy of science, it is necessary to highlight core commitments of Kant's philosophy that are incompatible with the pluralist commitments of many present-day philosophers and scientists.

3.2 Causation and causal inference in (psychiatric) epidemiology: a plea for pluralism

Kant's essentialism is based on the idea that the a priori principles that we presuppose in science define the essential features of objects. If causality is an a priori concept presupposed by all the sciences, standing in causal relations is a necessary and essential feature of objects of experience. The question of whether Kantian essentialism can contribute to our understanding of present-day research in psychiatry then boils down to the question of whether it makes sense to say that some concept of causality is a priori presupposed in psychiatric research.

We might think that the idea that some concept of causation is a priori presupposed in psychiatric research is not outdated. For medicine in general, including psychiatry, is fundamentally concerned with investigating the causes of diseases.

Despite the importance of causation for medicine, Kant's essentialism does not provide us with many tools for fruitfully analyzing medicine and psychiatry. The reason is that Kant's essentialism and philosophy stipulates that sciences presuppose one *necessary* a priori concept of cause. However, a study of research practices in medicine and psychiatry provides an argument for pluralism concerning (a) multiple *non-necessary* concepts of causation, and (b) multiple *non-necessary* methods of causal inference. Insofar as Kant proposed a necessary a priori concept and principle of causality, Kant was not a pluralist concerning the concept of causation, and hence his essentialism is of limited value for understanding present-day medicine and psychiatry. In the following, I will, following Illari and Russo (2014), present some arguments for pluralism concerning causality, and contribute to the literature by extending their analysis to concepts of causation and causal inference in psychiatry and by providing an analysis of pluralist viewpoints articulated in textbooks on epidemiology.

Illari and Russo (2014) give a rationale for adopting pluralism concerning causality. They state that there is no single theory of causality that can meet the diverse *needs* of different sciences. Different scientists have different legitimate questions

concerning causality, different needs, and different methods for inferring causality. For these reasons scientists must adopt theories of causality and methods of causal inference that are best tailored to these different needs. I will argue that pluralism concerning causality is an attractive philosophical position for understanding psychiatric research.

Illari and Russo (2014) distinguish between different types of pluralism (see also Godfrey-Smith, 2009). First, they discuss ontological pluralism concerning causality, which we will not discuss in this paper. Second, Illari & Russo discuss pluralism concerning concepts of causation, noting that some authors think that in science there exists not a single concept of causation but multiple related concepts of causation. I will argue that a study of research practices in epidemiology and psychiatry provides evidence for conceptual pluralism concerning causality. Finally, we can distinguish between pluralism concerning evidence for causality and pluralism concerning methods for causal inference (Illari & Russo, 2014). In the following I argue that the study of (psychiatric) epidemiology supports pluralism concerning methods. My argument moves from an analysis of general medical epidemiology to an argument for pluralism regarding causality in psychiatry. This presupposes that what I say about general medical epidemiology also holds true for psychiatry. To substantiate this assumption, I also at times provide analysis of a textbook on psychiatric epidemiology (Susser et al., 2006).

In his *Epidemiology: an Introduction* (2002), Kenneth Rothman dedicates the second chapter of the textbook to the question "What is causation?" Interestingly, Rothman does not provide a plurality of concepts of causation, but focuses mainly on the so-called sufficient-component cause (SSC) model, which he developed from the 1970 s onwards (see Rothman, 1976 reprinted in 2017). As Illari and Russo (2014, p. 29) show, Rothman's model is similar to Mackie's analysis of causality from the 1960 s (Mackie, 1965), insofar as these approaches analyze causality in terms of *necessary and sufficient components of causes*.

Rothman argues that what we take to be causes are often not sufficient causes themselves but component causes (see Illari & Russo, 2014). For example, Rothman (1976) notes that measles virus is often referred to as the cause of measles, whereas a sufficient cause for getting measles also involves lack of immunity to measles virus and other factors. Rothman defines a cause as an act or event which initiates, alone or together with other causes, an effect. A sufficient cause is a cause which produces the effect, and typically consists of many component causes graphically represented by parts of pies. Thus, for example, a whole pie with parts ACD represents a sufficient cause of a disease with parts (component causes) A, C and D. Take for example lung cancer as a disease. It has many different sufficient causes, all consisting of many component causes. The sufficient causes are represented by pies with parts (component causes) ACD, ADE, and AFG. These component causes (e.g., C, D, G) are typically not sufficient by themselves. However, it may be the case that there is a component cause which functions as a member of every (complex) sufficient cause, which is then called a *necessary cause* (in our example this is component cause A).

Illari and Russo (2014) remark that Rotham takes a sufficient cause to be a complete *causal mechanism*, which is a minimal set of conditions sufficient for the disease to occur. Rothman (2002) remarks that core features of his model are, among

others, the idea of (i) *multicausality or multifactoriality*, i.e., causal mechanisms involve the joint action of multiple component causes, (ii) that diseases typically have genetic and environmental determinants, and (iii) that some component causes are more important than others in bringing about a disease. These more important causes (e.g., smoking as a cause of lung cancer) can be understood on Rothman's model as component causes that figure in many complex sufficient causes. The multifactorial model also significantly influences psychiatric epidemiology. In their textbook on psychiatric epidemiology, Susser et al (2006) note that "we no longer think of *the* cause of a disease, but rather assume that diseases are produced by multiple interacting causes" (Chapter 2, Sect. 3). This type of analysis is taken to inform the psychiatry of the future, and it should inform multilevel analysis of psychiatric disorders such as anorexia nervosa, such as analysis of biological levels, societal levels (the effect of high-income countries), and other relevant levels (Chapter 3)..

In the textbook *Modern Epidemiology* (Lash et al., 2021), Rothman's sufficient-component cause model is no longer represented as the primary conception of causation of epidemiology. Rather, it is presented, along with other models of causality, as one among many useful approaches in reasoning about causal inference. Lash et al., 2021 adopt a pluralist conception of causality, noting that different formal causal models have, in different contexts, strengths and limitations. We will return to the pluralism of Lash et al. below, but first we will describe one of the dominant causal models in epidemiology that differs from Rothman's causal model, namely the potential outcome (counterfactual) model.

Lash et al (2021) note that the potential outcome (counterfactual) model is a *quantitative* model that specifies what happens under alternative patterns of intervention or exposure. It articulates a counterfactual conception of causality insofar as it is assumed that an association is causal when one believes that had the cause or exposure been altered, the effect or outcome would have changed. This description is counterfactual insofar as it refers to what would happen, if contrary to the fact, the exposure had been different from what it was. In their psychiatry textbook, Susser et al (2006) also adopt a counterfactual definition of a cause, noting that an exposure is a cause of an outcome if the outcome would not have occurred had the exposure been different (Chapter 5). Returning to the potential outcome model, Illari and Russo (2014) add that the counterfactual potential outcome is in turn a species of thinking about causality in terms of *difference-making*: we adopt the idea that causes make a difference to whether an effect obtains (see Kment, 2010).

The core idea of potential outcome models is described by Illari and Russo (2014, p. 95–96): ideally, e.g., if one wants to know whether aspirin is a cause of an effective treatment against headache, one compares two versions of oneself at the same time, one taking aspirin and one taking nothing. The causal effect would then be the difference between the effect of not taking aspirin and not taking aspirin. This, however, is impossible, and we need to establish the effectiveness of treatments by comparing groups of individuals. The potential outcome model is based on the following idea, which approximates how we reason about causes in the aspirin case: it provides a formal method for estimating what is called the *average causal effect*, which is the difference between the average response if all individuals were assigned treatment and the average response if all individuals were assigned to the control.

In this way, the potential outcome model provides a formal model to reason about causes.

Lash et al (2021) argue that given different needs one can adopt different models of causality. For example, they state that potential outcome models represent the limit of what one can learn about individual causes with no knowledge of *mechanism of action*. By contrast, Rothman's sufficient-component cause model considers causal mechanisms. Thus, if knowledge of mechanisms is an aim, one should use Rothman's model. However, Rothman's model is limited in scope, as is stressed by Illari and Russo (2014), who note that the model is not applicable to all diseases. To give one simple example, Rothman's model is multifactorial, hence it is a poor fit for diseases which are not multifactorial and which inspired the monocausal model of disease that arose in the nineteenth century. Moreover, as Illari and Russo (2014) stress, Rothman's pie charts do not fare well when conceptualizing the so-called induction period, i.e., the period from causal action until disease initiation. On the other hand, potential outcome models require that causes are manipulable, which, as Illari & Russo note, has the limitation that we cannot conceptualize factors such as ethnicity or gender as a cause (see also Vandenbroucke et al., 2016). Based on such considerations, many researchers, including Lash et al (2021), conclude that all causal models or frameworks for thinking about causality have their strengths and weaknesses and that one should adopt a pluralist stance for adopting frameworks.

We have provided an argument for adopting pluralism concerning the concept of causality in (psychiatric) epidemiology. There is also an argument to be made for adopting pluralisms concerning methods of causal inference. Different methods of causal inference in epidemiology have been described by Illari and Russo (2014) and applied to the field of psychiatric epidemiology by Ohlsson and Kendler (2020). Ohlsson and Kendler remark that causal methods in psychiatric epidemiology include randomized clinical trials (RCT's), natural experiments, and statistical models. They argue that each causal method has strengths and limitations. According to Ohlsson and Kendler, RCT's meet three criteria: (i) the response of experimental participants assigned to exposure is compared to the response of non-exposed participants in a control group, (ii) participants are randomly assigned to exposure and control groups, and (iii) the manipulation of the exposure is controlled. Susser et al (2006), in their study of psychiatric epidemiology, note that in such trials the two groups are observed for a period of time to ascertain the proportion in each group who develop the study outcome (chapter 6). Ideally, RCT's are double blind, which means that neither the research team nor the participants know which participants are in which group. The random assignment secures that the groups do not differ because of systematic factors at the start of the trial (ibid.). Susser et al. note that RCT's have led to important findings in psychiatry, such as the etiology for pellegra. Ohlsson and Kendler (2020) note that RCT's have limitations, insofar as individuals participating in RCT's are not necessarily representative of the population exposed to the intervention, which hinders extrapolation of the inferred causal claims in RCT's. In addition, in psychiatry RCT's are often unethical and thus unfeasible. This means that researchers have to adopt other methods of causal inference, such as natural experiments, i.e., observational studies in which variables of interest are not influenced by the researcher, and statistical models. Ohlsson and Kendler (2020)

conclude that all methods have strengths and limitations and that researchers should adopt a plurality of methods to infer causality.

Susser et al (2006) note about natural experiments in psychiatry that they are sometimes founded on historical events that assign individuals to exposed and unexposed groups. One group is then affected by an event or exposed, whereas a second otherwise similar group is not (unexposed). Natural experiments can be based on catastrophic events, such as famines, floods and wars. Finally, Susser et al. discuss cohort studies in psychiatry. In such studies, as in natural experiments, the investigator does not control the assignment of individuals to the exposed and unexposed groups. However, unlike the natural experiments, we cannot assume that selection into exposure was beyond the control of participants (Chapter 6). Although Susser et al. mention RCT's as an ideal for psychiatry, they also note RCT's are often not feasible and are rather pragmatic in their account of the different methods of causal inference. They thus seem to think that for different purposes different methods can be employed and adopt a form of pluralism in psychiatry.

The study of (psychiatric) epidemiology proves that researchers adopt (i) a plurality of concepts of causation and (ii) a plurality of methods of causal inference. If we recognize that in different contexts different causal concepts and methods will be more or less suitable, the study of (psychiatric) epidemiology provides a forceful argument for pluralism concerning causality. It is important to realize, however, that the lure of a single concept of causation or method of causal inference is strong. In a series of articles, Kendler (Kendler & Campbell, 2009; Kendler, 2011), who adopts pluralism concerning methods of causal inference, has argued that interventionism is an approach to causality that is *most appropriate* for psychiatry and which provides "a single, clear empirical framework for the evaluation of all causal claims of relevance to psychiatry" (Kendler & Campbell, 2009, p. 881). These claims illustrate the temptation to adopt a single concept of causality. Kant also thought it necessary to adopt a single concept of causality, and in their recent Kantian paper Breitenbach and Choi (2017) argue for the systematic relevance of Kant's regulative idea of a systematic unity of the sciences, arguing for what they call unified pluralism. According to this idea, we should accept pluralism but also accept as an ideal the idea of a complete and unified science, e.g., in the search for consistency among theories. In a plea for pluralism, Vandenbroucke, Broadbent and Peirce 2016 note that one can formulate a general concept of causality that unifies various theories of causality. What is the danger of this approach, which Kant adopts?

Nancy Cartwright (2016) provides an answer to this question, discussing the search for a unified method that underlies the plurality of scientific methods. Cartwright acknowledges that it might be possible to find a suitable abstract conception of method that homogenizes different scientific methods. This may even be a useful enterprise for some purposes! But she notes that it is essential that we are able to specify and make precise this abstract concept, so that we have a detailed description of the many different scientific methods which guide scientific practice. In this latter sense we stress disunity and plurality. This is one of the core points of the pluralist. I think philosophers like Kant would agree. Kant had, as Michael Friedman has shown (1992a, 1992b, 2001, 2013) specific concepts and principles of causality, articulated in the *Critique of Pure Reason*, that were tailored to the science of his

time (roughly, Newtonian science). In the *Metaphysical Foundations*, Kant *specified* these concepts and principles, to deliver, among others, a priori principles of mechanics that functioned as specific principles of Newtonian science. Hence, Kant, like the pluralist Cartwright, agrees that in philosophy of science *mere* abstract talk is dangerous because it is not specific enough. However, Kant was not a pluralist insofar as he thought his principles of causality were *necessary* and did not offer a model of causality useful for analyzing psychiatry in the twenty-first century. This means his essentialism should be rejected in light of (psychiatric) medical research practice.

The point, to reiterate, is that Kant adopted one necessary concept of causation in contrast to the pluralist, who adopts multiple non-necessary concepts of causation. Kant's necessary general concept of causality also *excludes* certain conceptions of causation, which are relevant in medical and psychiatric contexts. Thus, insofar as Kant held that causes are sufficient for their effects, his conception of causation will probably be rejected by researchers who adopt a multifactorial conception of disease, who suggest that diseases do not have sufficient causes. Similarly, like Hume, Kant adopted the principle "same cause same effect", which, as Illari and Russo remark (2014, p.164) does not suit many medical contexts and does not fit multifactorial models of disease. For these reasons, Kant's account of causality does not fit with pluralism regarding causation.

To conclude this section, we may note that psychiatry itself has had influential practitioners who have argued for the necessity of pluralism. These arguments also provide solid reasons for adopting pluralism in psychiatry. One such practitioner was Karl Jaspers, whose views have been described by Ghaemi (2007). Ghaemi notes that Jaspers adopted a pluralism of methodology partly because "multiple methods never exhaust the uniqueness of individuals" (p. 79). Hence, to do justice to the uniqueness of individuals, we have to view individuals through multiple methods. Jaspers adopted a methodological pluralism according to which "methodological pluralism consists of recognizing the strengths and limits of each method, and applying the ones that are best suited for specific circumstances (diseases, diagnosis, conditions)" (ibid.). Hence, to give an example apt for the current paper, clinicians may adopt different methods for curing and diagnosing disorders than the methods adopted by researchers who search for the causes of disorders, and moreover individual patients bring with them unique problems which are to be tackled by tailor made methods. According to Jaspers, pluralism replaces dogmatism in psychiatry (ibid.). Gheami notes that contemporary authors, such as Leston Havens in his *Psychiatric Movements* and Paul McHugh and Philip Slavney in their *The Perspectives of Psychiatry*, contain a successful and renewed plea for pluralism in psychiatry (p. 80). Thus, there are good reasons for adopting pluralism in psychiatry.

3.3 Kant and mechanisms in psychiatric research

We have seen that the study of psychiatric epidemiology provides reasons for rejecting Kant's essentialism. In this section, I wish to argue that Kant's theory of mechanism provides more fruitful insights for analyzing the role of mechanism and

mechanical explanation in present-day psychiatric research. I will argue for three claims: (a) that modern research into the causes of mental disorders fits the mechanist paradigm (Sect. 3.3.1), (b) that Kant's theory of mechanical explanation anticipates and is similar to modern theories of mechanical explanation applicable to psychiatry (Sect. 3.3.2), and (c) that Kant's stance that mechanism is a regulative ideal points us to reasons to pursue a mechanistic research programme in psychiatry (Sect. 3.3.2).

3.3.1 Mechanism, mechanical explanation, and psychiatry

In this section I describe how philosophical views on mechanism and mechanical explanation provide a suitable framework for analyzing explanation in present-day psychiatric research. I will start by giving definitions of key concepts. A conception of *mechanism* that is widely adopted by new mechanists is the minimal conception of mechanism. According to Glennan et al. (2022, p. 145), the minimal definition of mechanism is "that a mechanism for a phenomenon consists of entities (or parts) whose activities and interactions are organized so as to be responsible for the phenomenon." We will discuss the benefits of this definition shortly. According to Glennan (2017, p. 68), *mechanical explanations* are *how explanations*, providing us with mechanist models that show how organized activities and interactions give rise to a phenomenon to be explained. By doing so, mechanical explanations show how phenomena come about and exhibit (objective) dependencies (Glennan, 2017, p. 237). Finally, in debates on mechanical explanation, there is debate between the *epistemic conception* of explanation, according to which explanations are representations, and the *ontic conception of explanation*, according to which explanations are physical entities (Wright & Van Eck, 2018, pp. 997–999). The assumption underlying the epistemic conception is that any attempt at explanation wishes to account for something, and any account of something necessarily represents something in a certain way (Wright & van Eck, 2018). Thus, as Wright & van Eck note, Hempel and Oppnheim for example took explanations as relations between descriptive and elucidatory sentences (Wright & van Eck, 2018, note 1). Salmon took the epistemic conception to primarily consist in an inferential version, as Wright and van Eck (2018) explain, according to which explanation involves making an inference from the explanans to the explanandum. According to the ontic explanation as described by Craver, by contrast, explanations are not texts but things, and facts instead of representations (Craver, 2007 as quoted in Wright & van Eck, 2018). For example, Illari and Williamson (2011) take mechanisms to explain phenomena by being *causally responsible* for these phenomena (Illari & Williamson, 2011). By arguing in this way, proponents of the ontic conception seek to reconceive the traditional notion of explanation. Glennan (2017) traces this debate to the work of Salmon and notes that among new mechanists we can find defenders of the ontic conception of explanation (Craver, 2013) and the epistemic account (Bechtel & Abrahamsen, 2005). Glennan (2017) argues that the debate between the positions is overstated since good explanations must satisfy both ontic and epistemic constraints and both the ontic and epistemic accounts are complementary (see also Illari, 2013). I will argue that Kant's

account of explanation, influenced by Aristotle, shows that the ontic and epistemic accounts were indeed historically conceived of as complementary.

One of the virtues of the minimal definition of mechanism, as described by Glennan et al (2022, p. 146), is its generality. This definition of mechanisms captures mechanisms within (molecular) biology and neuroscience, but also in physics and the social sciences. The fact that the definition is widely applicable and captures social mechanisms is important, for sciences such as psychiatry, and medicine in general (Kelly et al., 2014), must be able to model social and cultural causes of mental disorders. The fact that psychiatry must be able to make sense of different so-called *levels of explanation* (Kendler, 2005, 2008, 2014), e.g., different causes or mechanisms of mental disorder spanning biological, social, psychological and other factors, means that mechanisms must be able to accommodate different levels of explanation. That mechanisms can indeed accommodate different levels of explanation, as Kendler argues following Bechtel, is one of the attractive features of mechanisms.

Bechtel (2006) explains why mechanisms can involve different *levels of organization and explanation*. He defines mechanisms as wholes consisting of parts and operations (p. 30). For example, the heart consists of parts such as the atria and ventricles, whereas operations include the contraction and relaxation of the atria and ventricles (ibid.). Bechtel notes that the components of a mechanism (parts and operations) occupy a lower level than the mechanism itself, which is a structure with a function (p. 40). For Bechtel, a mechanistic explanation "decomposes a mechanism into its parts and operations" (p. 41). We can continue this decomposition and research lower levels of organization, investigating for example the parts, composition, and operations of the atria and ventricles. In this way, mechanisms involve different levels of organization. Bechtel stresses that mechanists also investigate how mechanisms function within the context of things inhabiting a higher level (p. 42). Thus, we might investigate how an organism functions within its environment, or how individual humans function within urban cities, and so forth. According to Bechtel, interactions with (things in) the environment influence the behavior of the mechanism, and it can be the case that "the organization in the higher-level mechanism results in the imposition of constraints on the behavior of the first mechanism", which is important to understand the functioning of the first mechanism (p. 42). That mechanisms involve multiple levels of organization is crucial for researchers who investigate the causes of psychiatric disorders.

While explaining mental disorders we must be attentive to different types of causes. Kendler (2014) illustrates the different kinds of causes of psychiatric disorders. He reviewed articles discussing the causes of psychiatric disorders in four 2013 issues of 12 major psychiatry journals. On the basis of this review he identified three categories of explanation. These categories include: (i) a biological category (including causes belonging to the molecular, genetic, and neurochemical level), (ii) a psychological category (including causes such as neuropsychological traits, personality traits, and so forth), and (iii) an environmental level (including discussion of individuals, the family, and the community, society or culture). This overview shows that explanations of the causes of psychiatric disorders must take into account multiple causes and levels of organization. According to Kendler,

mechanistic explanations are ideally suited to this cause: "while the integration of biological, psychological, and social elements into causal processes was a tortured one using law-based models of science, it flows easily from a multilevel mechanistic approach." (Kendler, 2008, p. 696). That mechanistic explanations can incorporate and model social causes is also stressed by Kelly et al. (2014), who argue that so-called *mixed mechanisms* allow for the integration of biological with social and behavioral causes in an etiological mechanism of disease (see also Clarke & Russo, 2017).

Mixed mechanisms are already used in psychiatry, which proves that the idea of a mechanism is a useful category for analyzing psychiatric practice. To give one example, Van Os et al. (2010) argue that environmental factors play an important role in the etiology of schizophrenia.[1] The authors highlight that psychotic outcomes are associated with growing up in urban areas, cannabis use, and other environmental factors. To comprehend how genetic factors and environmental factors cooperate in bringing about schizophrenia, Van Os et al. (2010, p. 206) provide a model. According to this model (i) environmental exposure influences the phenotype (psychotic syndrome) and the phenotype influences environmental exposure, (ii) genes control environmental exposure and the environment can occasion epigenetic mutations, and (iii) genes influence the phenotype. In this model, we have (a) multiple levels of explanation (most importantly genes and environmental factors, such as cannabis use or living in urban areas) and multiple complex interactions between causal factors (most importantly between genes and environmental factors). However, the model of Van Os et. al. provides a *mechanistic* perspective on the etiology of schizophrenia insofar as it details *interactions* between various *entities* (e.g., between genes responsible for genetic liability and cannabis use) that operate at different levels of organization. As such, this example illustrates Kendler's contention that mechanisms can integrate biological, social and other causes operating at different levels.

Interpreting the model of Van Os et al (2010) as a mechanistic model shows that the notion of mechanism is very broad. Explanations of psychotic syndromes that incorporate environmental factors as causes can count as mechanisms. In a similar way, as an anonymous referee asked, societal and social events that cause post traumatic disorder can function in mechanistic explanations of post traumatic disorder, for mechanisms can easily incorporate environmental causes. In a forthcoming article, Russo and van Eck (forthcoming) argue that explanations of mental disorders on the basis of network models are also mechanistic. Network models (as explained by Borsboom, 2017) explain mental disorders in terms of networks of causally connected symptoms. For example, as explained by Borsboom, 2017, we can take delusions to generate paranoia, which in turn leads to social isolation, which again strengthens the delusion. Here, as Russo and van Eck explain, multiple component parts or causes or activities interact by being organized in specific ways and constitute mental disorders. Thus, network models can be seen as mechanistic

[1] The term and concept of schizophrenia are contentious. See Guloksuz & Van Os (2018). Since I report on past research, I will use the term and concept, which are still used.

explanations, which shows that the notion of mechanism is very broad indeed. Thus, it is false to only view explanations of mental disorders in terms of the brain or in terms of genes as mechanistic.

The research we have discussed concerning the environmental causes of schizophrenia shows the complexity of our explanations of mental disorders. We have multiple causes of psychiatric disorders, and these causes interact (e.g., genes and environmental factors). Such complexity provides a challenge for researchers who wish to give mechanical explanations of the causes of mental disorder. Bechtel and Richardson (2010) describe what they call *decomposition* and *localization* as heuristic strategies for providing mechanical explanations of phenomena. *Decomposition* assumes "that one activity of a whole system is the product of a set of subordinated functions performed in the system" (p. 23). *Localization* is "the identification of the different activities proposed in a task decomposition with the behavior or capacities of specific components.'(p. 24).

Bechtel and Richardson note (p. 25) that decomposition and localization function well for *aggregative systems*, which following Wimsatt they ascribe the following properties: (1) "intersubstitubility of parts, (2) qualitative similarity with a change in the number of parts, (3) stability under reaggregation of parts, (4) minimal interactions among parts". However, very few biological and mental systems are aggregative systems, and this poses challenges for mechanistic explanations in these fields. Kendler (2008) provides various psychiatric examples that show that psychiatric disorders are not aggregative in the sense of Bechtel and Richardson. We can focus on the example from Van Os et. al., which illustrates that environmental risk factors and genetic risk factors interact in the etiology of schizophrenia, thus showing that the assumption (4) for aggregative systems, i.e., "minimal interactions among parts", is not satisfied in the case of schizophrenia.

Although dynamic systems pose challenges for mechanical explanations, Bechtel (2006) argues that we should never give up the mechanist research programme. He argues, first of all, that mechanists acknowledge that mechanisms are not only explained in terms of their parts, but that the *organization* of the parts matters for the functioning of the system. Thus, for example, the heart would not function as it does without the organization of all its parts. In addition, Bechtel argues that a key feature of biological systems is the "incorporation of feedback and other kinds of control systems that allow the behavior of some components of the mechanism to be regulated by other components of the mechanism" (2006, p. 33). Incorporating such kinds of feedback and control systems, which also characterize psychiatric phenomena (Kendler, 2008), thus provide the mechanism with a fruitful research programme for studying and explaining dynamic systems.

In his 2008 *Mental Mechanisms*, Bechtel illustrates the mechanistic research programme by referring to the work of Bichat (1771–1802). He notes that Bichat thought that living tissues cannot be explained mechanically because they behave (a) indeterministically, and (b) the living tissues withstand and oppose physical forces, two features of living systems that illustrate that they maintain themselves as living systems longer than non-living systems (p. 207). Bechtel (2008) notes that subsequent mechanists nevertheless found mechanical explanations of the phenomena that Bichat took to be mechanically inexplicable. Claude Bernard (1813–1878), focusing

as Bechtel recommends on the *internal organization* of living things, argued that internal parts of organisms reside in an internal environments that is different from the external environment of the organism as a whole, and that the behavior of parts of organism in response to the internal environment is deterministic and in accordance with physical laws (p. 209). Later mechanist researchers invented the concept of homeostasis and conceived of negative and positive feedback as organizational principles that allow us to understand complex biological and mental systems. In this way, the mechanistic research programme was established as a fruitful research programme. Kendler (2008) agrees and argues that we can fruitfully search for mechanical explanations of psychiatric phenomena, akin to the successful search of biologists for mechanical explanations in the field of biology.

To conclude: mechanisms are useful explanatory devices in psychiatry because (i) psychiatrists often invoke mechanisms to explain phenomena (Kendler, 2008; Van Os et al., 2010), and (ii) mechanisms, insofar as they can represent multiple levels of organization and multiple types of causes, can accommodate multicausal and multilevel explanations in psychiatry (Kendler, 2005, 2008, 2014). These arguments provide reasons to take psychiatric research to fit the mechanist paradigm. In the next section, we will see that if we adopt a Kantian perspective we can identify additional plausible reasons to adopt a mechanistic research programme in psychiatry.

3.3.2 Kant, mechanism, and psychiatry

In this section, I will describe how Kant's account of explanation in general and his account of mechanical explanation in particular provides insight into the debate between the ontic and epistemic conception of explanation, anticipate some of Bechtel's ideas concerning mechanical explanations of complex dynamic systems, and points to fruitful reasons for pursuing a mechanistic research programme in psychiatry.

In the previous section, we have described the *epistemic conception* of explanation and the *ontic conception* of explanation. Kant's account of explanation, which derives from Aristotle (Van den Berg, 2014, Chapter 2 and 3), shows that historically these two types of explanations were seen as complementary. For Aristotle and Kant, explanations are deductive arguments, typically construed as syllogisms (Van den berg, 2014, chapter 2). As such, Aristotle and Kant subscribed to the epistemic conception of explanation. However, true and proper explanations must represent the causal structure of the world. This is what mechanistic explanations do according to Kant, which are a subset of causal explanations (Van den Berg, 2014, chapter 3). In what follows, I will focus on the general conception of explanation shared by Aristotle and Kant.

Aristotle and Aristotelians, including Kant (Van den Berg, 2014, chapter 3. See for a similar interpretation also Watkins, forthcoming), distinguished between (a) syllogisms in which we reason from the cause to the effect and (b) syllogisms in which we reason from the effects to the cause. For example, Aristotle took the following syllogism in natural science to reason from cause to effect: (a) The planets are near, (b) What is near does not twinkle, (c) The planets do not twinkle. By contrast, he took the following syllogism to reason from effect to cause: (a) The

planets do not twinkle, (b) What does not twinkle is near, (c) The planets are near (both Aristotelian examples are taken from Beaney & Raysmith, 2024). Syllogisms in which we reason from the cause to the effect represent the causal structure of the world and are properly explanatory. These syllogisms show *why* something is the case and were traditionally called a *demonstratio propter quid* (de jong & Betti, 2010). In contrast, syllogisms in which we reason from effect to cause, called a *demonstratio quia*, merely showed *that* something was the case. These syllogisms did not represent the causal structure of the world and were not the most proper explanations. We probably now have good reasons to reject the idea that only (deductive) arguments are explanatory. But the Aristotelian and Kantian conception of explanation shows that the epistemic and ontic conceptions of explanation were always viewed as complementary. This historical conception of explanation does justice to Bechtel and Abrahamsen's view that offering explanations is an epistemic activity (2005, p. 425) as well as to the insight of Craver that "idealizations count as conveying explanatory information in virtue of the fact that they represent certain kinds of ontic structures" (2013, p. 29). Hence, history supports contemporary philosophers of science who wish to stress similarities between the epistemic and ontic conception of explanation.

We have seen that the minimal definition of mechanism was suitable for psychiatry since it can be applied to a wide range of (social, psychological, biological, etc.) causes and mechanisms. Kant, of course, did not invent the minimal definition of mechanism. However, as Van den Berg (2013, 2014, chapter 3) has shown, the conception of mechanical explanation adopted by Wolff and Kant must be situated in a historical context in which in disciplines in logic and metaphysics it was common to speak about explanations in terms of the parts and the structure of the parts. Thus, in Wolffian logic, *definitions* were construed as explanations of wholes (species) in terms of their parts (partial concepts contained in the species) (see also de Jong, 1995). Take for example the definition "man is a rational animal". Here, the species concept ("man") was conceptualized in the eighteenth century as a complex or whole, that was composed of a *differentia* ("rational") and a *genus* ("animal"), which were both conceived of as *parts* of the species concept (the whole) (note that in definitions, just as in mechanical explanations, even the order or *organization* of concepts matters. To give Anderson's, 2004 example: the concepts "long pieces of clothing with stripes' and 'pieces of clothing with long stripes" can differ qua extension).

To conclude: mechanical explanations were seen as a variety of explanations of wholes in terms of their parts (see also Geiger, 2022). Moreover, as van den Berg (2014, p. chapter 3) stresses, Kant lived in the eighteenth century where the concept of mechanism also applied to Newtonian explanations invoking attractive forces, so the concept of mechanism that Kant adopted was broader than the traditional Cartesian one. Hence, although Kant did not invent a minimal definition of mechanism, his philosophy and historical context shows that Kant and his contemporaries adopted general accounts of explanations of wholes in terms of their parts and mechanisms that were applicable to a wide variety of explanations and phenomena.

Kant recognized that in organisms there is an apparent influence of the whole on the parts and that the parts of organisms mutually interact and are dependent upon

one another. Hence, he recognized, in Bechtel's terms, that organisms are not aggregative systems. He also claimed that some features of organisms are mechanically inexplicable (Breitenbach, 2006; Geiger, 2022; Ginsborg, 2004; McLaughlin, 1990). However, as Van den Berg (2014, chapter 5) has stressed (see also Breitenbach, 2017), Kant still insisted that we should explain as much as possible of organisms mechanically, because mechanical explanations are ideal scientific explanations that provide insight into the workings of nature. Hence, it is not the case, as Zammito (2006) seems to suggest, that Kant denied that we can explain anything of organisms. Instead, Kant proposed a positive research programme for organisms (see Van den Berg, 2014, chapter 5. Ginsborg, 2006, Geiger, 2022).

According to Kant's research programme, we must, as we have seen, in investigating complex systems such as organisms, assume as a principle the *complex organization* of these systems, and then investigate mechanically how the component parts and operations of these systems bring about certain functions. Thus, for example, we assume the complex organization of the heart as given, and then investigate how its parts and operations of the parts secure how the heart pumps around blood. The idea is that by making this assumption, we can understand and explain the role of parts within a complex organization of parts. This programme resembles that of Bechtel to a great extent, insofar as Bechtel also argued that in investigating complex dynamic systems we must assume the *complex organization* of a system, which constrains the operation of the parts, and allow for positive and negative feedback loops in these systems. Kendler articulated this mechanical research programme for psychiatry. As van den Berg (2014, chapter 5) shows, Kant was aware of both the claims of vitalists who showed that organic phenomena such as self-maintenance and self-reproduction (phenomena indicating homeostasis) are difficult to explain mechanically, while he also argued that we must always try to explain organic phenomena mechanically as far as possible. Thus, for example, in Kant's time there were attempts to provide mechanical and chemical accounts of phenomena such as nutrition, even if many life scientists took the way in which nutrition cyclically and continuously contributes to self-maintenance to be difficult to explain in mechanical terms (ibid.). Kant combined these perspectives and formulated a mechanical research programme that could be employed in emerging sciences such as biology (whether life scientists followed this programme is much debated among historians of science, and I will not consider this debate here. See Zammito, 2018 and Cooper, 2023a, 2023b). Hence, although this is not recognized by Kant scholars, Kant formulated a fruitful mechanistic research programme for the life sciences that resembles the mechanist research programme of new mechanists.

Bechtel argued for a mechanistic research programme that can be extended to many sciences. Kendler (2008) extends the mechanistic research programme to psychiatry. I have suggested that Kant also viewed the mechanistic research programme as a research programme that guides natural sciences. Evidence for this reading is that Kant assigns the status of a *regulative principle* to the principle of mechanism. For Kant, as Zuckert (2017) has explained (see also Spagnesi, forthcoming), regulative principles *guide scientific investigation* towards specific aims, such as systematic science. Moreover, regulative principles and the associated regulative ideas function as *demands of reason* or *standards of knowledge*, in terms of

which we investigate nature. Finally, regulative ideas function as impulses to extend our knowledge and to prevent what Zuckert calls empirical complacency. Hence, Kant characterizes regulative principles as we would now characterize research programmes, and insofar as he sees the principle of mechanism as a regulative principle that guides empirical research, he provides a reflection on guidelines that guide the development of empirical science. Kant's reflections fit the mechanist research programme described by Bechtel, who sketches how vitalist challenges such as those of Bichat have been overcome by persistent mechanists such as Bernard. Kant did not himself extend this research programme to psychology or psychiatry, but his account of mechanisms does provide some insight into how different sciences can adopt this research programme.

But why should psychiatry adopt the mechanist research programme? Since psychiatry has often not been able to provide explanations, mechanical or otherwise, of most of the causes of most psychiatric disorders, what gives us confidence that we should pursue a mechanist research programme in psychiatry? It is with respect to this question that I think Kant's views on mechanisms are interesting for current mechanists in psychiatry. Note that psychiatry in the twenty-first century is somewhat similar to the life sciences in the eighteenth century: both have had very little success in providing mechanical explanations. Yet, in spite of this limited success, Kant insisted that we should nevertheless strive for mechanical explanations in the life sciences. Can we similarly say that we should strive for mechanist explanations in psychiatry?

As we have seen, Kant treats the maxim according to which we must search for mechanical explanations of nature as a regulative maxim. This implies that the justification for adopting this maxim is not ontological. It is not the case for Kant, as Descartes thought, that we know that nature and natural objects are mechanisms and that we must therefore explain nature mechanically. For all we know, God created the world as composed of immaterial souls or monads. I think that an ontological justification for pursuing mechanical explanations is also not easily available for modern psychiatric researchers. Why would psychiatric researchers argue that the mind is a mechanism if mechanical explanations of the (dysfunctions of) the mind have been so elusive? Although we lack ontological reasons, Kant argues that we nevertheless have good methodological reasons for giving mechanical explanations of natural objects. Such methodological reasons consist, for example, in the fact that mechanical explanations provide *unified* explanations of nature or that they provide *scientific understanding* (see for Kant's views on virtues guiding science, including unification and understanding, Van den Berg, 2020, 2021; Falkenburg, 2000). In the following, we will see that psychiatric researchers indeed have good methodological reasons for providing mechanical explanations.

We have already seen one influential argument, by Kendler, for giving mechanistic explanations in psychiatry, namely that mechanisms, insofar as they can represent multiple levels of organization and multiple types of causes, can accommodate multicausal and multilevel explanations in psychiatry (Kendler, 2005, 2008, 2014). Russo and Williamson (2007) provide another important methodological reason for why one ought to strive for knowledge of mechanisms in medicine (and we may add) psychiatry. The reason is that they argue that mechanisms constitute evidence

for establishing causal claims in medicine. The argument is that mechanisms explain dependencies between cause and effect and that they provide evidence of the stability of the causal relationship, allowing us to generalize causal claims. Thus, for example, the causal link between smoking and lung cancer was established, one might argue, after at least a plausible physiological mechanism of smoking was established. It is never the case, according to Russo and Williamson, that we accept a causal link in complete abstraction from mechanist considerations. If Russo and Williamson are right, they provide an important methodological reason for providing mechanisms of phenomena. Note that this argument also applies to psychiatry. If Van Os et al. (2010) are correct in claiming that genes and environmental factors mechanistically interact to cause psychosis, we have explained dependencies between cause and effect and have a reason to accept a causal link.

Another methodological reason why one might strive for providing mechanical explanations in psychiatry is that these explanations are suited to providing *scientific understanding*. Kant thought causal mechanical explanations provide understanding by showing how a phenomenon comes about, and were in this respect to be preferred to teleological accounts, which did not yield such understanding (Van den Berg, 2014, chapter 4). The idea that mechanisms yield understanding is a methodological reason for providing mechanical explanations in psychiatry. Understanding is here understood as de Regt (2017) defines it, namely as an ability of scientists to use a relevant theory which involves skills and judgments (p. 36). Understanding thus depends on the skills of scientists who use a theory. de Regt notes that causal-mechanical explanations were viewed by its proponents to yield understanding (p. 48). Thus, for example, Wesley Salmon held that causal mechanisms constitute the key to our understanding of the world (p. 59). The idea is partly that mechanical explanations gives you insight into how a phenomenon comes about, i.e., how the parts generate a more complex phenomenon, and also allows you to manipulate the causes that yield a phenomenon. This allows scientists to use a particular account of phenomena and thus, in de Regt's terms, to understand such a phenomena. Applied to psychiatry, if we know, as Van Os et al. (2010) claim, that genes and environmental factors such as growing up in urban areas and cannabis use, interact to cause psychosis, we know how the parts interact to produce a complex phenotype such as the psychotic syndrome and can also manipulate these causes to prevent the psychotic syndrome from happening. In this way, the mechanisms constituting schizophrenia allow us to understand schizophrenia.

Finally, Gillies (2018) provides two more methodological reasons for providing mechanical explanations in medicine and psychiatry. First, mechanical explanations yield unification. If we explain some phenomena P in terms of a mechanism M, we can subsequently explain M in terms of a lower level mechanism M, and so on, thus unifying multiple phenomena (p. 189). As we have seen, Kant, and later Bechtel, similarly thought we can continue the process of decomposition in mechanical explanations for as long as we like, explaining mechanisms in terms of their parts, these parts in terms of subparts, and so forth, thus obtaining unified explanations. This line of reasoning also applies to psychiatry. Take again the mechanistic explanation of schizophrenia from Van Os et al. (2010). According to this model, as we have seen, (i) environmental exposure influences the phenotype (psychotic

syndrome) and the phenotype influences environmental exposure, (ii) genes control environmental exposure and the environment can occasion epigenetic mutations, and (iii) genes influence the phenotype. On this model, the environment figures as an explanandum of the psychotic syndrome, which can in turn be decomposed in various environmental factors, such as growing up in urban areas, cannabis use, and so forth, and we can, for example, analyze cannabis use again to discover which of the active ingredients of cannabis triggers a psychosis. In this way, we obtain unified explanations in psychiatry. Second, Gillies explains that searching for mechanisms in science aids us in finding cures in medicine (p. 185). Suppose we believe we know the causal claim A causes disease B. We can then find a cure for B by for example blocking A. However, now suppose we have a complex mechanism linking A to B, which consists of a sequence of causes. We can then tackle any of this set of causes to prevent disease B from occurring. As an anonymous referee has pointed out, for clinicians finding a cure is often the most important for learning about etiology in medicine and psychiatry. Note that this argument again also applies to psychiatry. If we know cannabis causes psychotic episodes in teenagers, we can invest in measures that prevent cannabis use in teenagers, such as education on the dangers of cannabis use.

To conclude: we can find multiple methodological or instrumental reasons for providing mechanical explanations in psychiatry without pointing to an ontological justification, which is in line with Kant's reflections on mechanisms. Kant not only anticipated many arguments and ideas of the new mechanists, he also provided fruitful reasons for adopting a mechanist programme which can be adopted by present day psychiatric researchers. His relevant insights for present-day philosophy of psychiatry are that he argued for the necessity of a broad mechanistic research programme in a domain where there was a paucity of mechanistic explanations. Nevertheless, Kant argued, we should pursue mechanistic explanations since they are proper causal explanations that explain why something is the case and that yield scientific understanding and scientific unification. Modern day philosophers have provided more methodological or instrumental reasons for pursuing mechanistic explanations that can be used when they need to justify the reason for pursuing mechanistic explanations. Such reasons are important when in psychiatry obviously ontological justifications will not suffice.

4 Conclusion

This paper has described Kant's essentialism and mechanism and evaluated their relevance in light of present-day psychiatric research. Kendler et al (2011) reject essentialism because essentialist mistakenly assume that mental disorders have a single cause. Kant's theory of essentialism is not disqualified by the argument of Kendler et al (2011) against essentialism, since Kant adopts a different variety of essentialism than described by Kendler et al. In addition, there are other varieties of essentialism in psychiatry that are also not affected by the argument of Kendler et al (2011). Hence, rejecting essentialism requires more and different arguments than provided by Kendler et al., and until such arguments have been articulated

essentialism remains a live option in psychiatry. I have argued that modern psychiatric research supports arguments for adopting pluralism with respect to the concept of causality and methods of causal inference, which is incompatible with Kant's essentialism and transcendental philosophy more generally. More specifically, Kant assumes the existence of one necessary concept of causality underlying science, a concept which excludes other causal concepts, whereas pluralists allow for the adoption of multiple non-necessary concepts of causality. Finally, I have argued that psychiatric research into the causes of psychiatric disorders fits the mechanist paradigm, that Kant anticipated many arguments concerning mechanism adopted by new mechanists that are applicable to psychiatry, and that Kant articulated a mechanistic research programme for complex systems that is similar to the research programme articulated by Bechtel and associates. Kant's methodological reasons for pursuing a mechanistic research programme can fruitfully be adopted by contemporary mechanists. Such methodological and Kantian arguments provide non-ontological reasons for psychiatrists to proclaim that "it is in the nature of science to want to move from findings of causality to a clarification of the mechanisms involved" (Kendler, 2011, p. 76), even if such mechanisms have yet to be found in psychiatry.

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References

Adriaens, P. R., & De Block, A. (2013). Why we Essentialize Mental Disorders. *Journal of Medicine and Philosophy, 38*(2), 107–127.

Allison, H. E. (2004). *Kant's Transcendental Idealism*. Yale University Press.

Anderson, E. (1994). Kant, natural kind terms, and scientific essentialism. *History of Philosophy Quarterly, 11*(4), 355–373.

Anderson, R. L. (2004). It adds up after all: kant's philosophy of arithmetic in light of the traditional logic. *Philosophy and Phenomenological Research, 69*(3), 501–540.
Beaney, M., & Raysmith, T. (2024). "Analysis", *The Stanford Encyclopedia of Philosophy* (Fall 2024 Edition), Edward N. Zalta & Uri Nodelman (eds.), URL = https://plato.stanford.edu/archives/fall2024/entries/analysis/
Bechtel, W. (2006). *Discovering Cell Mechanisms: The Creation of Modern Cell Biology*. Cambridge University Press.
Bechtel, W. (2008). *Mental Mechanisms: Philosophical Perspectives on Cognitive Neuroscience*. Routledge.
Bechtel, W., & Abrahamsen, A. (2005). Explanation: A mechanist alternative. *Studies in History and Philosophy of Biological and Biomedical Sciences, 36*(2), 421–441.
Bechtel, W., & Richardson, R. C. (2010). *Discovering Complexity: Decomposition and Localization as Strategies in Scientific Research*. MIT press.
Borsboom, D. (2017). A network theory of mental disorders. *World Psychiatry, 16*, 5–13.
Breitenbach, A. (2006). Mechanical explanation of nature and its limits in Kant's critique of judgment. *Studies in History and Philosophy of Biological and Biomedical Sciences, 37*(4), 694–711.
Breitenbach, A. (2009). *Die Analogie von Vernunft und Natur: Eine Umweltphilosophie nach Kant*. Walter de Gruyter.
Breitenbach, A. (2009b). Teleology in biology: A Kantian approach. *Kant Yearbook, 1*, 31–56.
Breitenbach, A. (2017). Laws in Biology and the Unity of Nature. In M. Massimi & A. Breitenbach (Eds.), *Kant and the Laws of Nature* (pp. 237–255). Cambridge University Press.
Breitenbach, A. (2018). Laws and Ideal Unity. In W. Ott & L. Patton (Eds.), *Laws of Nature* (pp. 108–122). Oxford University Press.
Breitenbach, A., & Choi, Y. (2017). Pluralism and the unity of science. *The Monist, 100*, 391–405.
Broadbent, A. (2009). Causation and models of disease in epidemiology. *Studies in History and Philosophy of Biological and Biomedical Sciences, 40*, 302–311.
Cartwright, N. (2016). Loose talk kills: What's worrying about unity of method. *Philosophy of Science, 83*(5), 768–778.
Clarke, B., & Russo, F. (2017). Mechanisms and Biomedicine. In S. Glenna & P. Illari (Eds.), *The Routledge Handbook of Mechanisms and Mechanical Philosophy* (pp. 319–331). Routledge.
Cleve, J. V. (1973). Four recent interpretations of Kant's second analogy. *Kant-Studien, 64*(1), 71.
Cohen, A. (2009). *Kant and the Human Sciences: Biology, Anthropology and History*. Palgrave MacMillan.
Cooper, R. (2014). *Psychiatry and Philosophy of Science*. Routledge.
Cooper, A. (2023a). Hypotheses in Kant's philosophy of science. *Studies in History and Philosophy of Science, 99*, 97–105.
Cooper, A. (2023). *Kant and the Transformation of Natural History*. Oxford University Press.
Cooper, R. (2004). Why hacking is wrong about human kinds. *British Journal for the Philosophy of Science*, *55*(1). https://doi.org/10.1093/bjps/55.1.73
Craver, C. F. (2007). *Explaining the Brain*. Oxford University Press.
Craver, C. F. (2013). The Ontic Account of Scientific explanation. In M. I. Kaiser, O. R. Scholz, D. Plenge, & A. Hütteman (Eds.), *Explanation in the Special Sciences: The Case of Biology and History* (pp. 27–52). Springer.
De Jong, W. R. (1995). Kant's analytic judgments and the traditional theory of concepts. *Journal of the History of Philosophy, 33*, 613–641.
De Regt, H. (2017). *Understanding Scientific Understanding*. Oxford University Press.
De Jong, W. R., & Betti, A. (2010). The classical model of science: A millennia-old model of scientific rationality. *Synthese, 174*, 185–203.
De Pierris, G., & Friedman, M. (2018). Kant and Hume on causality. *The Stanford Encyclopedia of Philosophy* (Winter 2018 Edition), Edward N. Zalta (ed.), URL = https://plato.stanford.edu/archives/win2018/entries/kant-hume-causality/
Engelhard, K. (2018). The problem of grounding natural modality in Kant's account of empirical laws of nature. *Studies in History and Philosophy of Science Part A, 71*, 24–34.
Falkenburg, B. (2000). *Kants Kosmologie: Die Wissenschaftliche Revolution der Naturphilosophie im 18. Jahrhundert.* Frankfurt am Main: Vittorio Klostermann.
Friedman, M. (1992). *Kant and the Exact Sciences*. Harvard University Press.
Friedman, M. (1992). Causal Laws and the Foundations of Natural Science. In P. Guyer (Ed.), *The Cambridge Companion to Kant* (pp. 161–199). Cambridge University Press.

Friedman, M. (2001). Matter and Motion in the Metaphysical Foundations and the First Critique: The Empirical Concept of Matter and the Categories. In E. Watkins (Ed.), *Kant and the Sciences* (pp. 53–96). Oxford University Press.

Friedman, M. (2013). *Kant's Construction of Nature: A Reading of the Metaphysical Foundations of Natural Science*. Cambridge University Press.

Frierson, P. (2014). *Kant's Empirical Psychology*. Cambridge University Press.

Fuller, J. (2018). Universal etiology, multifactorial diseases and the constitutive model of disease classification. *Studies in History and Philosophy of Biological and Biomedical Sciences, 67*, 8–15.

Geiger, I. (2022). *Kant and the Claims of the Empirical World: A Transcendental Reading of the Critique of the Power of Judgment*. Cambridge University Press.

Ghaemi, S. N. (2007). Existence and pluralism: The rediscovery of Karl Jaspers. *Psychopathology, 40*, 75–82.

Gillies, D. (2018). *Causality, Probability, and Medicine*. Routledge.

Ginsborg, H. (2004). Two kinds of mechanical Inexplicability in Kant and Aristotle. *Journal of the History of Philosophy, 42*(1), 33–65.

Ginsborg, H. (2006). Kant's Biological Teleology. In G. Bird (Ed.), *A Companion to Kant* (pp. 455–469). Wiley-Blackwell.

Ginsborg, H. (2015). *The Normativity of Nature: Essays on Kant's critique of Judgement*. Oxford University Press.

Glennan, S. (2017). *The New Mechanical Philosophy*. Oxford University Press.

Glennan, S., Illari, P., & Weber, W. (2022). Six theses on mechanisms and mechanistic science. *Journal for General Philosophy of Science, 53*, 143–161.

Godfrey-Smith, P. (2009). Causal Pluralism. In H. Beebee, C. Hitchcock, & P. Menzies (Eds.), *The Oxford Handbook of Causation* (pp. 326–337). Oxford University Press.

Guloksuz, S., & van Os, J. (2018). The slow death of the concept of schizophrenia and the painful birth of the psychosis spectrum. *Psychological Medicine, 48*(2), 229–244.

Guyer, P. (1987). *Kant and the Claims of Knowledge*. Cambridge University Press.

Hacking, I. (1991). A tradition of natural kinds. *Philosophical Studies, 61*, 109–126.

Hacking, I. (2007). Kinds of people: Moving targets. *Proceedings of the British Academy, 151*, 285–318.

Hanna, R. (1998). A Kantian critique of scientific essentialism. *Philosophy and Phenomenological Research, 58*(3), 497–528.

Hyman, S. E. (2010). The diagnosis of mental disorders. The problem of reification. *Annual Review of Clinical Psychology, 6*, 155–179.

Illari, P. (2013). Mechanistic explanation: Integrating the ontic and epistemic. *Erkenntnis, 78*(Suppl 2), 237–255.

Illari, P., & Russo, F. (2014). *Causality: Philosophical Theory Meets Scientific Practice*. Oxford University Press.

Illari, P., & Williamson, J. (2011). Mechanisms are Real and Local. In P. Illari, F. Russo, & J. Williamson (Eds.), *Causality in the Sciences* (pp. 818–844). Oxford University Press.

Kant, I. (1992). *Lectures on Logic*. Translated and edited by J. Michael Young. Cambridge University Press.

Kant, I. (1998). *The Critique of Pure Reason*. Translated and edited by P. Guyer and Allen W. Wood. Cambridge University Press.

Kant, I. (2002). *Theoretical Philosophy After 1781*. Edited by H. Allison and P. Heath. Translated by G. Hatfield, M. Friedman, H. Allison and P. Heath. Cambridge University Press.

Kelly, M. P., Kelly, R. S., & Russo, F. (2014). The integration of social, behavioral, and biological mechanisms in models of pathogenesis. *Perspectives in Biology and Medicine, 57*(3), 308–328.

Kendler, K. S. (2005). Toward a philosophical structure for psychiatry. *American Journal of Psychiatry, 162*(3), 433–440.

Kendler, K. S. (2008). Explanatory models for psychiatric illness. *American Journal of Psychiatry, 165*(6), 695–702.

Kendler, K. S. (2011). Causal Thinking in Psychiatry. In P. E. Shrout, K. M. Keyes, & K. Ornstein (Eds.), *Causality and Psychopathology* (pp. 66–78). Oxford University Press.

Kendler, K. S. (2014). The structure of psychiatric science. *American Journal of Psychiatry, 171*(9), 931–938.

Kendler, K. S., & Campbell, J. (2009). Interventionist causal models in psychiatry: Repositioning the mind-body problem. *Psychological Medicine, 39*(6), 881–887.

Kendler, K. S., Zachar, P., & Craver, C. (2011). What kinds of things are psychiatric disorders? *Psychological Medicine, 41*(6), 1143–1150.
Kment, B. (2010). Causation: Determination and difference-making. *Noûs, 44*(1), 80–111.
Kraus, K. (2020). *Kant on Self-Knowledge and Self-Formation: The Nature of Inner Experience*. Cambridge University Press.
Kreines, J. (2009). Kant on the laws of nature: Laws, necessitation, and the limitation of our knowledge. *European Journal of Philosophy, 17*(4), 527–558.
Kroon, F., & Nola, R. (1987). Kant, Kripke and Gold. *Kant-Studien, 78*, 442–458.
Lash, T. L., VanderWeele, T., Rothman, K., & Haneuse, S. (2021). *Modern Epidemiology*. Wolters Kluwer.
Lenoir, T. (1989). *The Strategy of Life*. University of Chicago Press.
Mackie, J. L. (1965). Causes and conditions. *American Philosophical Quarterly, 2*(4), 245–264.
Massimi, M. (2017). Grounds, Modality, and Nomic Necessity in the Critical Kant. In A. Breitenbach & M. Massimi (Eds.), *Kant and the Laws of Nature* (pp. 150–170). Cambridge University Press.
McLaughlin, P. (1990). *Kant's Critique of Teleology in Biological Explanation: Antinomy and Teleology*. Edwin Mellen Press.
Messina, J. (2017). Kant's Necessitation Account of Laws and the Nature of Natures. In M. Massimi & A. Breitenbach (Eds.), *Kant and the Laws of Nature* (pp. 131–149). Cambridge University Press.
O'Donnell, L. A., Deldin, P. J., Pester, B., McInnis, M. G., Langenecker, S. A., & Ryan, K. A. (2017). Cognitive flexibility: A trait of bipolar disorder that worsens with length of illness. *Journal of Clinical and Experimental Neuropsychology, 39*(10), 979–987.
Ohlsson, H., & Kendler, K. S. (2020). Applying causal inference methods in psychiatric epidemiology: A review. *JAMA Psychiatry, 77*(6), 637–644.
Patton, L. (2017). Kantian essentialism in the metaphysical foundations. *The Monist, 100*(3), 342–356.
Robertson Ishii, T. & Atkins, P. (2023) Essential vs. accidental properties. *The Stanford Encyclopedia of Philosophy* (Spring 2023 Edition), Edward N. Zalta & Uri Nodelman (eds.), URL = https://plato.stanford.edu/archives/spr2023/entries/essential-accidental/
Rothman, K. J. (1976). [reprinted in 2017]. Causes. *American Journal of Epidemiology, 104*(6), 587–592.
Rothman, K. J. (2002). *Epidemiology: An Introduction*. Oxford University Press.
Russo, F., & Van Eck, D. (forthcoming). Charting the Explanatory Potential of Network Models in Psychopathology. In Kendler, K.S., Parnas, J., and Zachar, P. (eds), *Philosophical Issues in Psychiatry VI: Causal Concepts in Psychopathology: Multidisciplinary Perspectives.* Oxford University Press.
Russo, F., & Williamson, J. (2007). Interpreting causality in the health sciences. *International Studies in the Philosophy of Science, 21*(2), 157–170.
Samuels, R. (2009). Delusions as a Natural Kind. In M. Broome & L. Bortolotti (Eds.), *Psychiatry as Cognitive Neuroscience: Philosophical Perspectives* (pp. 49–79). Oxford University Press.
Scholten, M. (2016). Schizophrenia and moral responsibility: A Kantian essay. *Philosophia, 44*, 205–225.
Spagnesi, L. (forthcoming 2023). The Systematic Unity of Reason and Empirical Truth in Kant's Critique of Pure Reason. *Kant-Studien*.
Stang, N. F. (2016). *Kant's Modal Metaphysics*. Oxford University Press.
Sturm, T. (2008). Why did Kant reject physiological explanations in his anthropology? *Studies in History and Philosophy of Science Part A, 39*(4), 495–505.
Sturm, T. (2009). *Kant und die Wissenschaften vom Menschen*. Mentis.
Susser, E., Schwartz, S., Morabia, A., & Bromet, E. J. (2006). *Psychiatric Epidemiology: Searching for the Causes of Mental Disorders*. Oxford University Press.
Urfer, A. (2001). Phenomenology and psychopathology of schizophrenia: The views of Eugene Minkowski. *Philosophy, Psychiatry & Psychology, 8*, 279–289.
Van den Berg, H. (2013). Wolff and Kant on scientific demonstration and mechanical explanation. *Archiv Für Geschichte der Philosophie, 95*(2), 178–205.
Van den Berg, H. (2014). *Kant on Proper Science. Biology in the Critical Philosophy and the Opus postumum*. Springer.
Van den Berg, H. (2020). Theoretical virtues in eighteenth-century debates on animal cognition. *History and Philosophy of the Life Sciences, 42*(3), 37.
Van den Berg, H. (2021). Kant's ideal of systematicity in historical context. *Kantian Review, 26*(2), 261–286.
Van Os, J., Bak, M., Hanssen, M., Bijl, R. V., De Graaf, R., & Verdoux, H. (2002). Cannabis use and psychosis: A longitudinal population-based study. *American Journal of Epidemiology, 156*(4), 319–327.

Van Os, J., Kenis, G., & Rutten, B. P. (2010). The environment and Schizophrenia. *Nature, 468*(7321), 203–212.

Vandenbroucke, J. P., Broadbent, A., & Pearce, N. (2016). Causality and Causal inference in epidemiology: The need for a pluralistic approach. *International Journal of Epidemiology, 45*(6), 1776–1786.

Watkins, E. (2005). *Kant and the Metaphysics of Causality*. Cambridge University Press.

Watkins, E. (2019). *Kant on Laws*. Cambridge University Press.

Watkins, E. (forthcoming). The Systematicity of Natural Science: Logical and Real. In *Kant and the Systematicity of the Sciences*, eds. G. Gava, T. Sturm, and A. Vesper. Routledge.

Wright, C., & Van Eck, D. (2018). Ontic explanation is either ontic or explanatory, but not both. *Ergo, 5*, 997–1029.

Zammito, J. (2006). Teleology then and now: The question of Kant's relevance for contemporary controversies over function in biology. *Studies in History and Philosophy of Biological and Biomedical Sciences, 37*(4), 748–770.

Zammito, J. (2018). *The Gestation of German Biology: Philosophy and Physiology from Stahl to Schelling*. University of Chicago Press.

Zuckert, R. (2017). Empirical Scientific Investigation and the Ideas of Reason. In M. Massimi & A. Breitenbach (Eds.), *Kant and the Laws of Nature* (pp. 89–107). Cambridge University Press.

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