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Causal modeling of panic disorder theories ☆

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ABSTRACT

We compare a variety of theories of panic disorder using a neutral framework: causal modeling. The framework requires identification of key constructs and specification of their interaction. Biological, cognitive, and behavioral elements of the theory have to be clearly distinguished, as do critical past events and current trigger conditions. The theories compared were drawn from the psycho-dynamic, cognitive, and neurobiological literature. We conclude that there are substantive differences among the cognitive theories and between the biological theories reviewed. However, cognitive and biological theories appear to be compatible in principle. It is not clear whether substantive differences among theories are due to the existence of subtypes of PD or due to the predominance of multifactorial cause. It is argued that current treatment methods imply particular theories, and that particular patterns of success and failure can be understood in relation to theory through the methods we have employed.

Contents

1. Causal modeling as a framework

Increasing work by psychologists in the clinical field has led to an explosion of theories of disorders. Unfortunately, very little work has been done in comparative evaluation of various theories, particularly when they come from different approaches: for example biological versus cognitive. Ideally, such comparisons should be done with techniques which are unbiased with respect to the nature of the

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competing theories. This theoretical neutrality is characteristic of what we call a *framework*, a term that contrasts with *theory* and *model*. A model is, in this way of thinking, representation of a theory within a particular framework.

The causal modeling framework was developed by Morton and Frith (1995) and Morton (2004) in order to represent theories of developmental disorders, making clear distinctions among biological, cognitive, and behavioral components of the theories. The framework has been applied to a variety of disorders, especially autism (Frith, Morton, & Leslie, 1991), dyslexia (Morton & Frith, 1993a,b, 1995, 2002), psychopathy (Blair, 1995), conduct disorder (Krol, Morton & de Bruyn, 2004) and DCD (Sims & Morton, 1998). The framework has also been particularly useful for comparison of different, often competing, causal theories of dyslexia (BPS, 1999; Frith, 1999). Here, the advantage of using the causal modeling framework has been to demonstrate that some theories that were perceived as being totally different from each other were essentially similar, differing only in the relative weighting of the primary causes. In other cases, the weakness of particular theories, for example of autism (Morton, 2004), has been highlighted following their representation in the causal modeling notation. The framework has also been beneficial in the training of education psychologists (Monsen, Graham, Frederickson, & Cameron, 1998; Frederickson & Cline, 2002) who have adapted the framework for understanding pathways of special educational needs.

A causal model of a particular theory, then, is a representation of that theory within the causal modeling framework. The techniques are particularly useful for considering theories of panic disorder since they help to clarify, in each theory, the extent to which biological and environmental factors interact with cognitive processes in determining behavior.

Use of the causal modeling framework involves creation of a directed graph in which the elements of a theory are connected with arrows. Thus, an arrow linking element A to element B will have the force of "A causes B", meaning that, in the particular theory, if A is not present, B would not be found. Note that in different theories, elements may be more or less specific. For example, one might postulate a genetic cause without specifying locus.

The model space is divided up into three components, corresponding to biological, cognitive and behavioral elements. We will also use the concept of *equivalence*. Equivalence emerges when there is both a biological element and a cognitive element to a theory which are effectively identical for the purposes of the theory. Thus, we shall see below that the hippocampus is named as the location where the context of a trauma is stored. This context would serve as a cue for the retrieval of the traumatic experience from memory. The hippocampus has a direct connection with the amygdala, and this connection is entirely in the biological domain. On the other hand, we want to represent the effect of the reoccurrence of the original context in triggering a panic attack. Such input has to be cognitive since the elements of the context have to be recognized, and interpreted, and this process is subject to a variety of cognitive influences. Thus the individual's expectations may play a role in accessing the trigger memory. In general, such cognitive processes are not specified biologically, nor would such a biological specification be relevant to PD. All the PD theory needs is that expectations influence the interpretation of the environment. It is useful in this example, then, to keep the hippocampus as the biological specification and the associative memory as the *equivalent* cognitive specification. In the Causal Model, the hippocampus would be the element linking to other biological elements, and the associative memory would be the link to other cognitive components of the theory.

In previous work, causal models have represented the history of development. In this paper, we will also use a slightly different kind of model which includes current processing. They are to be thought of as additional information to help us understand the theories and data that underlie the causal hypotheses. We also want to make a distinction between antecedent conditions on the one hand and immediate causes on the other. For example, a traumatic event will have a long-term consequence in cognitive and brain structures. This will be part of the causal model. However, there will often be a local cause of a symptomatic breakdown which need not necessarily have permanent consequences. This distinction is the same as that drawn by Roth, Wilhelm, and Pettit (2005) between distal cause and proximal cause. They consider that each of the causal theories of panic attack that they consider "presupposes that there is a *unique* underlying proximal cause, a single underlying event that is both causally necessary and *under normal background conditions* causally sufficient for a PA" (Roth et al., 2005, p. 172, their italics). They then examine falsifiability of each theory in terms of the relation between proximal cause and PA. We will not attempt to identify such a unique element, but rather, will contrast developmental history of the individual contributing to the antecedent conditions with the processing occurring at the time of an attack.

Causal modeling consists of linking elements at the same or at different levels. It can conveniently represent the influence of the environment either at the biological level, as modifying the phenotypic expression of genetic predisposition, or at the cognitive level. As such, it can be an efficient tool to represent the multicausality of disorders and to represent biosocial or bio-psycho-social theories (Kiesler, 1999). This makes it ideal for representation of theories of panic disorder.

2. Panic disorder

The key event in panic disorder is the panic attack. This is defined by Clark (1986) in the following way:

> "A panic attack consists of an intense feeling of apprehension or impending doom which is of sudden onset and which is associated with a wide range of distressing physical sensations. These sensations include breathlessness, palpitations, chest pain, choking, dizziness, tingling in the hands and feet, hot and cold flushes, sweating, faintness, trembling and feelings of unreality" (p. 461).

Normally, an individual is aware of the source of any fearful sensations they have. In panic disorder, the same types of sensation are seemingly unprovoked, unexplained and often occur out of the blue. Furthermore, the characteristic panic could be the principal symptom in terms of temporal precedence; on the other hand, it might be secondary and be seen as a severity marker of a co-morbid illness (Roy-Byrne, Craske & Stein, 2006).

It is believed that, during a panic attack, anxiety can have deleterious physical, social, and psychological consequences that extend beyond any immediate physical discomfort. That is to say, some of the observed symptoms are secondary rather than primary. Let us give an example from the clinical practice of LF. A patient was anxious about swallowing food because he was scared that the food could become stuck in his windpipe and consequently he could choke. The consequence of this was that he ate less than normal, almost only liquid food, and there was an accompanying loss of weight. From the social point of view, because he was scared of being choked, he ate very slowly, and avoided going out to dinner. Because he avoided situations that would expose his problem, he became more isolated, his habits changed and he became depressed. Clearly, treating the depression without addressing the panic disorder would be inappropriate or inadequate.

In addition, some symptoms are not specific to panic disorder, but occur in various combinations in other anxiety disorders, such as post-traumatic stress disorder and social anxiety. For example, phobic avoidance of a certain location could occur in PTSD or social anxiety as well as PD. Again, treatment of the symptom without consideration of the cause would be limited in value.