How do practising clinicians and students apply newly learned causal information about mental disorders?

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Abstract

Rationale, aims and objectives New causal theories explaining the aetiology of psychiatric disorders continuously appear in the literature. How might such new information directly impact clinical practice, to the degree that clinicians are aware of it and accept it? We investigated whether expert clinical psychologists and students use new causal information about psychiatric disorders according to rationalist norms in their diagnostic reasoning. Specifically, philosophical and Bayesian analyses suggest that it is rational to draw stronger inferences about the presence of a disorder when a client’s presenting symptoms are from disparate locations in a causal theory of the disorder than when they are from proximal locations.

Method In a controlled experiment, we presented experienced clinical psychologists and students with recently published causal theories for different disorders; specifically, these theories proposed how the symptoms of each disorder stem from a root cause. Participants viewed hypothetical clients with presenting proximal or diverse symptoms, and indicated either the likelihood that the client has the disorder, or what additional information they would seek out to help inform a diagnostic decision.

Results Clinicians and students alike showed a strong preference for diverse evidence, over proximal evidence, in making diagnostic judgments and in seeking additional information. They did not show this preference in the control condition, in which they gave their own opinions prior to learning the causal information.

Conclusion These findings suggest that experienced clinical psychologists and students are likely to use newly learned causal knowledge in a normative, rational way in diagnostic reasoning.

Introduction

New scientific knowledge about psychiatric conditions appears in the literature steadily and continuously. Of particular interest in the current paper is that causal knowledge about the aetiology of disorders is increasing at a rapid rate. Evidence suggests that this kind of knowledge may appeal strongly to clinicians in that they have a marked tendency to reason causally [1]. For example, clinicians conceptualize disorders as falling along a single continuum of aetiological causes (i.e. from biological to environmental), and they base judgments of treatment efficacy on whether those treatments directly act upon these causes [2]. Similarly, clinicians’ ratings of intervention effectiveness can be systematically predicted from their own reported causal models of clients’ problems [3], and clinicians weight causal symptoms more heavily than their effects in making diagnostic decisions [4].

Although such work has shown that clinicians’ own causal theories of disorders strongly influence their judgment and decision making, it has not addressed the degree to which new, externally presented causal information directly influences clinical decisions. For example, when a practising clinician picks up a research journal and reads about a new theory explaining how a psychiatric disorder comes about, how might he or she apply that knowledge in practice, to the degree that he or she accepts it? The question of how clinicians use such newly acquired information in their diagnostic reasoning processes has important implications for both researchers and practitioners. In many cases, as we discuss next, causal reasoning strategies can be considered strategies that conform to rationalist norms. For example, students and experienced clinicians rate interventions that affect assumed root causes of disorders as more effective than interventions affecting factors with less causal impact [2,5].
In the same vein, the current study investigated whether the causal structure of new information about psychiatric conditions affects clinicians’ and students’ diagnostic reasoning. Current theories of psychiatric conditions typically explain a fairly large range of symptoms with a smaller number of causes (e.g. genetics, brain and/or environment), forming what we will refer to as a hierarchical causal structure. That is, many explanatory theories assume that the manifestation of a given psychiatric condition can be traced back to just one or a few broad categories of root causes [6]. Please see Fig. 1 for an illustrative example. Our study therefore specifically asks how clinicians and students might make use of theories of psychiatric conditions that take the form of hierarchical causal structures.

In previous research, undergraduate students were presented with hierarchical causal structures of various medical conditions (e.g. rheumatic fever) and asked them to judge which of two patients was most likely to have the condition in question: a patient presenting with two symptoms originating from the same causal path (‘causally proximal’ symptoms, henceforth), or a patient presenting with two symptoms originating from different paths (‘causally diverse’ symptoms) [7]. Students most often chose the patient with diverse symptoms. The influence of hierarchical causal information earlier on in the diagnostic reasoning process has also been examined in undergraduate students [8]. Students presented with a patient exhibiting a single symptom were asked whether they would prefer to seek out information about the presence of a causally diverse versus causally proximal symptom in determining whether the patient had the medical condition. The results showed that students preferred to seek out information regarding the presence of causally diverse over causally proximal symptoms [8]. Students did so even though they also reported that patients were more likely to have causally proximal than causally diverse symptoms.

The students’ preference for causally diverse evidence in these medical reasoning tasks is in line with a broad preference for feature diversity in category-based induction tasks in classic cognitive science research [9–11]. This preference for diverse evidence has been previously argued to be normative and rational [12,13], and can be justified via Bayesian computation [14–16]. A preference for causally diverse evidence can be similarly justified. The likelihood that a specific root cause is present, given proximal or diverse evidence, is equal to the probability that the evidence occurs given the root cause \( P(e|c_1) \) multiplied by the probability of the root cause independent of any evidence \( P(c_1) \), controlling for the prior probability that the evidence occurs (i.e. the summed probabilities that the evidence occurs with all possible causes – hypothesized root cause and alternative causes). See Equation (1):

\[
P(c_1|e) = \frac{P(e|c_1) P(c_1)}{\sum_{c_{i=2}} P(e|c_i) P(c_i)}
\]

(1)

Possible causes for proximal evidence may include the possible causes for the presence of diverse evidence, and more. Thus, the denominator in the equation gets larger for proximal evidence compared with diverse evidence, leading to a lower probability that, with proximal evidence, the hypothesized root cause is present. With fewer alternative causes, as with diverse evidence, the denominator gets smaller, resulting in a higher probability of the hypothesized root cause. Conversely, the presence of one symptom increases the likelihood of the preceding causal chain in the hierarchy, and thus the presence of the other proximal symptom, whereas it does not similarly increase the likelihood of the other causal chain, and thus the presence of diverse symptoms. Proximal symptoms can thus, rationally, be more expected to occur than diverse symptoms.
Of course, it is completely unknown whether actual clinical practitioners would show the same preference for causal diversity in diagnostic reasoning as did students in prior research regarding medical conditions. In category-induction tasks in cognitive science, domain expertise has been found to lead to other reasoning strategies that can instead lead to an apparent preference for feature proximity [17–19]. Interestingly, experts in these studies directly applied their pre-existing, additional knowledge about the causal relationships between features and were thereby able to circumvent the need to draw inferences from diversity. Thus, relying on feature diversity might be a default strategy for novices. However, in these studies, no new information was presented; both domain experts and novices reasoned using the prior knowledge they had.

In the current work, our driving question was whether experienced clinicians, as well as students, show a causal diversity effect in diagnostic reasoning with new causal information. With increasing scientific knowledge about the aetiology of psychiatric conditions, insights regarding how clinicians use such newly acquired knowledge in their reasoning can ultimately enable us to anticipate and improve clinicians’ diagnostic and treatment decisions by means of prescriptive guidelines, decision aids or education. In this study, we presented experienced clinical psychologists and undergraduate students with causal hierarchical structures for five psychiatric conditions. Each structure was a theory, derived from recent, published clinical literature, explaining how symptoms of a psychiatric condition are thought to originate from a deeper cause. We tested whether clinicians and students would show a preference for causally diverse information in making diagnoses [7] and in seeking information to help inform a diagnosis [8]. Because we anticipated that individual clinicians would probably vary widely in terms of their prior knowledge about specific psychiatric conditions, all participants served as their own controls in a time-delayed, within-subject design.

Method

Participants

Sixty-two people participated in the study; of these, 29 were experienced clinical psychologists and 33 were undergraduate students, all recruited by mail, phone, or e-mail. We randomly assigned the clinicians to carry out either the diagnosis task \( (n = 15, \ 10 \) United States and 5 the Netherlands) or the information-seeking task \( (n = 14, \ 8 \) United States and 6 the Netherlands). Clinicians had a mean of 19.9 years (SD = 8.7) of experience in clinical practice. Fifteen were female, and their mean age was 48.6 years (SD = 10.2). US clinician participants were paid 40 dollars and the Netherlands’ clinician participants received a 10 euro gift certificate. Undergraduate students from various fields of study were recruited to participate at Northeastern University in the United States and Radboud University Nijmegen in the Netherlands. Students were randomly assigned to either the diagnosis task \( (n = 16, \ 8 \) United States and 8 the Netherlands) or the information-seeking task \( (n = 17, \ 9 \) United States and 8 the Netherlands). Their mean age was 20.5 years (SD = 1.8), and 29 were female. Students had no clinical experience and received course credit for their participation.

Materials

We derived summary causal diagrams for five DSM-IV-TR [20] psychiatric disorders from published theories. Each diagram showed how a root cause branched out in two divergent causal paths, each containing two intermediate steps, and ultimately leading to two pairs of symptoms each. See Fig. 1.

The other psychiatric disorders were anorexia nervosa [23,24], attention-deficit/hyperactivity disorder [25], panic disorder [26] and borderline personality disorder [27]. See Appendix for the components of these diagrams. Three additional experienced clinical psychologists who did not participate in the study itself provided feedback on the plausibility of all diagrams, and on how best to equate the proximal versus diverse symptoms in terms of their salience and prevalence. We made adjustments to the diagrams accordingly, while still maintaining as much as possible the integrity of the original theories.

Procedure

All participants received two questionnaires, with a 1-week time lag in between. All clinicians received and returned the questionnaires by mail. All undergraduate participants were tested in person, either individually or in groups. For all participants, the first questionnaire completed was always the control condition questionnaire, to prevent any possibility of carry-over influence of causal information. The second questionnaire was always the causal condition questionnaire, in which we presented causal information. In sum, clinician and student participants were randomly assigned to complete either the diagnosis task or the information-seeking task.

In all causal condition questionnaires, participants were shown the causal diagram information, each divided across four pages (four chains per disorder) to eliminate spatial distance cues (following [7]). Each chain was labelled with the name of the disorder and a reference to the publications used for the construction. The four chains for one disorder were presented in pseudo-randomized order on separate sheets, such that the same causal chain was never seen twice consecutively. After having read the four chains for the first disorder, participants were asked to write a summary paragraph of the information to demonstrate their understanding; for subsequent disorders they were asked to mentally summarize the information.

Next, participants assigned to complete the diagnosis task were asked to imagine two clients, one presenting with two symptoms resulting from the same causal path (proximal symptoms), and the other presenting with two symptoms resulting from different causal paths (diverse symptoms). The two clients always had one symptom in common. For autism, participants were presented with the statements: ‘Client N.W. has impairment in social interactions and problems with communication’ and ‘Client C.F. has a need for structure and rules and problems with communication.’ As Fig. 1 illustrates, Client N.W. has causally proximal symptoms, and Client C. F. has causally diverse symptoms. To ensure that participants would not simply discount the presented theories when they did not agree with their content, we asked them to determine which of two clients would be most likely, according to the theory they had read, to have the disorder. Continuing our example, we asked:
The means ran in the same direction for all five disorders. There was a significant preference for diversity reached significance for both clinicians and students, such that our participants across the board showed a relatively direct, positive impact on clinical practice. Additional work will be needed to test whether clinicians would accept new causal information spontaneously in practice, when causal structure is not primed as it was in our study. As discussed earlier, however, a number of studies have shown that clinicians’ judgments are reliably and systematically predicted from their causal beliefs [2–4,30]. As our work currently stands, we conclude that it suggests clinicians and even students are likely to apply theoretical work on psychiatric conditions to diagnostic reasoning in a way that is normative and rational. To the extent that clinicians are aware of and accept theoretical models of psychiatric conditions (and to the degree that those theoretical models are in fact accurate), these models may be expected to have a relatively direct, positive impact on clinical practice.

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References


## Appendix: Components of the psychiatric disorder stimuli

<table>
<thead>
<tr>
<th>Components</th>
<th>Anorexia nervosa</th>
<th>ADHD</th>
<th>Panic disorder</th>
<th>Borderline personality disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Root cause</td>
<td>Too severe dietary restrictions due to an extreme need for self-control</td>
<td>Inhibitory dysfunction due to alterations in prefrontal control circuits</td>
<td>Specific trigger activates malfunctioning amygdala</td>
<td>Interaction of genetic predisposition and environmental stressors</td>
</tr>
<tr>
<td>Intermediate causes 1</td>
<td>Breaking of (too) strict dietary rules; perceived failure</td>
<td>Cognitive dysregulation; poor planning and working memory functioning</td>
<td>Amygdala sends overly strong signal to lateral hypothalamus; overactive sympathetic nervous symptom</td>
<td>Low serotonin activity; lack of inhibitory influence over limbic system</td>
</tr>
<tr>
<td>Terminal effects 1</td>
<td>Negative self-evaluation; abuse of laxatives</td>
<td>Failure to complete tasks; careless in daily activities</td>
<td>Chills; Sweating</td>
<td>Outbursts of anger; Impulsivity</td>
</tr>
<tr>
<td>Intermediate causes 2</td>
<td>Severely low caloric intake; state of starvation</td>
<td>Behavioural dysregulation; impulsiveness</td>
<td>Amygdala misdirects signal to the locus caeruleus; enhanced excitability</td>
<td>Increased amygdala activation; Neurochemically based difficulty regulating emotions</td>
</tr>
<tr>
<td>Terminal effects 2</td>
<td>Preoccupied with food and eating; social withdrawal</td>
<td>Risky behaviour; interrupts others frequently</td>
<td>Pounding heart; chest pain</td>
<td>Fluctuating emotions; stress-related transient paranoia</td>
</tr>
<tr>
<td>Patient symptoms Presenting</td>
<td>Abuse of laxatives</td>
<td>Careless in daily activities</td>
<td>Sweating</td>
<td>Stress-related transient paranoia</td>
</tr>
<tr>
<td>Diverse Proximal</td>
<td>Social withdrawal</td>
<td>Interrupts others frequently</td>
<td>Chest pain</td>
<td>Outbursts of anger</td>
</tr>
<tr>
<td></td>
<td>Negative self-evaluation</td>
<td>Failure to complete tasks</td>
<td>Chills</td>
<td>Fluctuating emotions</td>
</tr>
</tbody>
</table>

Note: A 5th psychological disorder used in this study, autism, is depicted in Fig. 1. ADHD, attention-deficit/hyperactivity disorder.