The glue of (ab)normal mental life: Networks of interacting thoughts, feelings and behaviors
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Citation for published version (APA):
Cramer, A. O. J. (2013). The glue of (ab)normal mental life: Networks of interacting thoughts, feelings and behaviors

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Appendix D

A constructionist account of emotional disorders

Abstract

This commentary was written in response to a target article by Lindquist, Wager, Kober, Bliss-Moreau and Feldman-Barrett (2012, published in the same issue of Behavioral and Brain Sciences) in which the brain basis of emotion was reviewed. Lindquist et al. present a strong case for a constructionist account of emotion. In this commentary, we first elaborate on the ramifications that a constructionist account of emotions might have for psychiatric disorders with emotional disturbances as core elements. Second, we reflect on similarities between Lindquist et al.’s model and recent attempts at formulating psychiatric disorders as networks of causally related symptoms.

Fear is not localized in the amygdala, nor does sadness exclusively arise in the anterior cingulate cortex. Unfortunately for Gall (Gall & Spurzheim, 1835), and more recent proponents, who hypothesized that single brain areas (later referred to as “particular circuits”; see Kandel & Squire, 1992) correspond to single functions (e.g., arithmetic skills), feelings (e.g., pride) or attitudes (e.g., religiosity); locationist perspectives on such functions, feelings, and attitudes and their hypothesized unique “signature” in the brain increasingly turn out to be wrong (e.g., Bartholomew2004, Poldrack2006).

Lindquist et al. convincingly argue, emotions are not recognized by the brain as separate entities and, as such, do not each have their own seat and unique activation signature in the brain. Instead, Lindquist et al. present a strong case for a constructionist perspective in which emotions are comprised of multiple, more basic processes, which are each associated with their own location and activation signature in the brain. The combined outcomes of these processes result in the individual experience of a particular emotion.

If Lindquist et al.’s constructionist perspective is an accurate representation of the relation between emotions and the brain, what ramifications might this have for those psychiatric disorders that have emotional disturbances as core elements? Among other processes, Lindquist et al. distinguish between core affect (i.e., mental representation of bodily representations) and conceptualization (i.e., sensations from the body or external


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world that are made meaningful). Major depression (MD) is a psychiatric disorder with “sadness” as one of the core elements, and it is well known that, in many cases, an episode of MD is preceded by stressful life events such as marital of health problems (e.g., Kendler et al., 1999). Although such life events are potentially quite aversive in nature, most people do not develop an episode of MD after experiencing them: So why are some people so severely affected by a stressful life event whereas most others are not? One explanation could be that in people who develop an episode of MD after a stressful life event the conceptualization process is dysfunctional; most people would respond with some sadness after a quarrel with a spouse (i.e., “normal” core affect), but in people with MD, this event is overly negatively conceptualized (“See, even my husband does not love me”). Such a hypothesis is consistent with clinical observations that patients with MD often engage in excessive rumination about past events (Roelofs, Huibers, Peeters, & Arntz, 2008; Roelofs, Huibers, Peeters, Arntz, & van Os, 2008).

On the other hand, in disorders with “fear” as a core element, the core affect process might have gone awry. Patients with a specific phobia are extremely fearful of certain objects (e.g., hypodermic needles), situations (e.g., flying an airplane), or animals (e.g., spiders) that do not elicit the same response in most other people. When confronted with, for example, a spider, patients with a phobia for that animal will respond with various bodily sensations (e.g., profuse sweating, heart palpitations) to that animal, whereas people without the phobia will not experience such bodily sensations; in terms of the Lindquist et al. perspective, specific phobia patients react with excessive core affect to phobic objects compared to non-phobic patients.

Distinguishing emotional disorders in terms of Lindquist et al.’s proposed processes might implicate a shift in clinical neuroscience from searching for the dysfunctional brain area causing a particular disorder to searching which brain areas do not optimally work together in perceiving and interpreting external stimuli (e.g., will we find that the conceptualization network is overly active in patients with MD?). This implication of Lindquist et al.’s work, that psychiatric disorders are not likely to be explained in terms of one dysfunctional brain area, bears a striking resemblance to recent attempts at formulating psychiatric disorders as networks of causally related symptoms (Borsboom, 2008b; Cramer et al., 2010; Kendler et al., 2011). In the network approach, psychiatric disorders are hypothesized to stem from direct interactions between symptoms (e.g., feeling tired → sleeping a lot → concentration problems) instead of from one underlying biological dysfunction (e.g., serotonin depletion causes all symptoms of MD). As such, each symptom is an autonomous causal entity and it is unlikely that such entities share the same etiological mechanisms. For example, symptoms such as insomnia and fatigue are likely governed by homeostatic processes, whereas symptoms such as guilty feelings and depressed mood are more likely regulated by cognitive processes (e.g., rumination). This hypothesis also lies at the heart of a theory in which psychiatric disorders are mechanistic property clusters (MPCs): mutually reinforcing networks of causal mechanisms at multiple levels of explanation (e.g., symptoms, brain). Each of these conceptualizations suggests that there are no hard delineations between disorders, as the processes that carry forward disturbances in a network are unlikely to be confined to a single set of symptoms (i.e., have a transdiagnostic character).

Thus, Lindquist et al.’s constructionist account is suggestive mutually reinforcing networks at the brain level that, when working optimally, result in the subjective experience of an appropriate particular emotion (e.g., fear when confronted with an angry grizzly bear). However, if one or more of those networks do not optimally work together, the result can be an inappropriate emotion (e.g., excessive fear when confronted with a spider). Subsequently, the network approach (i.e., mutually reinforcing networks at the symptom level) explains why, for example, a dysfunctional core affect process does not
result in a specific phobia (i.e., the entire syndrome) but results in excessive fear of a particular object or situation: other symptoms of a specific phobia, for example avoiding the feared object or situation, are a result of the excessive fear (i.e., one symptom causing the other). One way to investigate this hypothesis is by gathering intensive time series data with which one can accurately monitor the development of symptoms (and interactions among them) over time. This approach can be combined with frequent fMRI scans in order to link, for example, excessive activation of the conceptualization network, to the subsequent development of MD symptoms.