Abstract: Lindquist et al. present a strong case for a constructionist account of emotion. First, we 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., Bartholomew 2004; Poldrack 2006). Likewise, as 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 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 or 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 (e.g., Roelofs et al. 2008a; 2008b).

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 object will respond with various bodily sensations (e.g., profuse sweating, heart palpitations) to that object, 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 2008; 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 dysfunction causes all symptoms of MD). As such, each symptom is an autonomous causal entity and it is unlikely that such entities share the exact 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 of 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 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.

Emotions as mind organs

doi:10.1017/S0140525X11001695

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Abstract: In matters of the mind, the opposition between what is mind-made or inside and natural or outside the mind is bound to misfire. Lindquist et al. build their analysis on a strong contrast between naturalism, which they reject, and psychologism, which they endorse. We challenge this opposition and indicate how adopting psychologism to combat a naturalistic view of emotional mind/brain areas is self-defeating. We briefly develop the alternative view of emotions as mental organs.

Lindquist et al. challenge the view that the most familiar emotion words and the linguistically expressed emotion experiences are ultimately the natural kinds found in the brain/mind, referred to as "naturalism" for short. Naturalism traditionally refers to the view that some of the entities the mind reasons with and decides about exist outside of and independent from these

mental operations. Let us label this physicalist naturalism. However, when naturalism is used to refer to putative entities in the mind/brain, the situation is much more complex. Mentalistic naturalism as opposed to physicalist naturalism, seems to postulate that there are entities in the mind that are not mind-made. As Lindquist et al. present it, for naturalism basic emotions are such mind-independent entities. Emotions, in the naturalistic view the authors challenge, stand for mind-independent or for biological categories, which are essentially present in the mind-brain. Psychologism, by contrast, does not build on anything given other than its own operations, which are the same whatever the subject matter.

Interestingly, the description of the alternative view Lindquist et al. endorse, psychological constructivism, consists of entirely content-general mental operations that operate over inputs that are not necessarily emotional. So, other aspects aside, the contrast the authors set up is between emotional determinism and emotional indeterminism of the mind's building blocks.

The inherent contradictions of an area-focused meta-analysis. Lindquist et al. are rightfully critical of the approach that has been prominent in the majority of brain imaging studies aspiring to localize the neurofunctional basis of each single emotion in a dedicated brain area. For example, the amygdala was the fear area, the insula was the disgust area, and so forth. Meta-analyses inherit the weak points of still less-than-perfect brain imaging techniques and cannot but endorse and amplify them. fMRI studies vary widely in scanner properties, in settings, in designs, and in tasks, including the involvement of attention, awareness, and contrast stimuli or conditions. The meta-analysis exploits the very procedures under attack by using positive activation levels of isolated brain areas themselves obtained in a wide variety of studies. The meta-analytic conclusion that some areas play or do not play their anticipated role, does not invalidate their role, and this role may or may not show up in fMRI analysis. For example, the amygdala was repeatedly shown to play a role in processing of emotional stimuli, and brain imaging studies of autism are consistent with this. However, patients with Urbach-Wiethe syndrome have a major deficit of the basolateral amygdala, yet show no signs of autistic behavior. There are many more examples illustrating that there is no rigid link between a brain area and a functional deficit. But the suggestion of attributing functions to a network rather that to a single area is likely to beg the question. Another approach to emotions is needed. It must be possible to avoid naive naturalism and extreme psychologism.

Emotions are mind organs. Emotions are mind/body adaptations, evolved in natural and social contexts (in a partly species-specific way). As emotions serve different goals, they have evolved next to each other and inhabit brain/body resources in different ways to fit their goals (Panksepp 1998). Yet in contrast to many approaches, different emotions are interdependent and interrelated. We do not believe that emotions must await neuroanatomical dissection to prove that they operate as cooperating distinct entities, even if functional distinctions can be made and appear in clinical symptoms. We know that this is unrealistic with current functional neuroimaging techniques. For example, different emotions produce different facial expressions in a predictable way, although we can reasonably assume that we are unable at the moment to distinguish between motor activity associated with angry versus fearful expressions. In the very same way, the visceral activation and the associated feeling will be different between disgust and anger, but it is unlikely that these emotional experiences can be disentangled spatially by their cortical somatosensory responses. In our view, emotions entail a distributed neural system, and focusing on its components, whether from a locationist or from a psychological constructionist perspective, is equally and inherently reductionist. First, psychological constructivism reduces emotions to a sum of parts, ignoring that a particular neural component exerts its function in relation to and sometimes driven by the other components of the individual emotion system (e.g., Benuzzi et al. 2009; Liang et al. 2009). This emotion-specific connectivity pattern is an essential and mandatory characteristic of emotions. Second, by attributing a specific psychological operation to a gross anatomical component, the degrees of freedom of the functional contribution of this component to a particular emotional state are reduced. For example, the amygdala may signal motivational salience in some instances but may critically contribute to the fear response in others. Patients with epilepsy caused by sclerosis of the amygdala, for example, may experience intense fear during their seizures, in the absence of any relevant object (Van Paesschen et al. 2001). Third, emotion-specific activation at the cellular level in monkey studies (Kuraoka & Nakamura 2007) somewhat contradicts the postulation of generic regional operations that is made by psychological constructivism.

Just as organs have different functions in the body, emotions serve different functions in the mind. The traditional terminology of basic emotions as states is indeed inappropriate to catch these functions. They encompass not just a network of brain areas, as these activation peaks are the tip of the iceberg. Beneath the neurofunctional facts revealed by brain imaging studies of neurotypical subjects, are structural facts, inhibitory and excitatory modulations in dynamic networks, endocrinological signatures, behavioral engrams laid down by phylogenetic and ontogenetic experience, and so on. Most importantly in this context, the function of an organ, in this case the minds' emotions, needs to be understood in relation to the others and of the whole. Just as the body cannot be reduced to a collection of independent organs, the emotions operate in concert, and whether in health or in sickness, they need to be considered together. Balanced or unbalanced, the interaction between the emotion organs makes and breaks the self. One may still call this "psychologism," but then any view on emotions is "psychologism."

A rigorous approach for testing the constructionist hypotheses of brain function

doi:10.1017/S0140525X1100149X

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Abstract: Although the target article provides strong evidence against the locationist view, evidence for the constructionist view is inconclusive, because co-activation of brain regions does not necessarily imply connectivity between them. We propose a rigorous approach wherein connectivity between co-activated regions is first modeled using exploratory Granger causality, and then confirmed using dynamic causal modeling or Bayesian modeling.

Lindquist et al. seek to distinguish between locationist and constructionist models of emotion by performing a meta-analysis of brain activations during various types of emotional stimuli. Methodologically speaking, the discovery of activated brain areas using the general linear model is primarily geared towards the