Metacognition, affect regulation and symptom expression: A transdiagnostic perspective

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ABSTRACT

The editors of this special section commissioned this commentary to bring together some of the conceptual, empirical and measurement issues arising from this series of articles. This commentary explores metacognition in relation to its neurobiology, and diverse syndromes and clinical phenotypes, including schizophrenia, alexithymia, and personality disorders, as well as its relation to assessment and prospects for the further delineation of mechanisms of change in psychological therapy.

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1. Introduction

It seems reasonable to assert that a fundamental aspect of our humanity is our capacity to attune to our own minds, the minds of others and make use of our mind-mindedness to accommodate our own and others’ needs, plans and intentions. Following from this, these capacities enable us to negotiate and resolve intrapersonal and interpersonal conflicts in desires and goals in sophisticated ways that seem so typical for our species. Through understanding our own and others’ minds, we are able to enter into pretend modes of functioning that enable us to take the perspective of others, share their joy and happiness as well as their pain and distress. Thus, we can be moved to help and support those around us, to express care, love, respect, sympathy and compassion. Indeed our utilisation of these capacities to alleviate distress in others moves beyond simple bonds of family and community, towards helping other human beings and other species. Basic information, such as gaze, posture, facial expression, voice tone and volume, provide us with subtle (and sometimes not so subtle) signals of the well-being, intentions, needs, emotions and states of mind of individuals around us. As we respond to and interpret these signals, we adjust our own behaviour and response, often accommodating those around us. This constant daily interplay between multiple states of mind is remarkable in light of the day to day pressures we face getting onto crowded buses, subways, and trains, requiring the inhibition our own irritability and frustration. We might stop to help a mother lift her child’s pram onto the bus; after a long day and despite our tired legs, we might give up our comfortable seat on a subway to an elderly man we’ve never seen before and may never see again. All these things happen on a daily basis without ever a word passing between us. Each day we co-operate with a community of others with whom we share scarce resources (Hrdy, 2009). At the same time we can also deploy our capacities for mental state understanding in the services of other interpersonal and evolutionary priorities (Liotti and Gilbert, 2011). Understanding the mental states of others enables us to anticipate the actions of others including their errors. In this way we can compete, exploit weaknesses, outwit and defeat our opponents. Alternatively, we can anticipate and attend to threats from others and defend ourselves against attacks to our psychological and physical integrity. It is also in our capacities to appreciate and understand the cognitive and emotional states of others where the darkest aspects of our humanity reside, that is, in our utilisation of mental state information to exploit, manipulate and hurt others for our own selfish goals.

Therefore, we understand three key aspects of this analysis. First it acknowledges our capacities for metacognition and mentalization. Second, it acknowledges our utilisation (how and when we use and apply these competences) of these capacities. Finally, these capacities can be utilised in the service of separate and distinctive evolutionary priorities including affiliation, competition and threat. Based on this...
understanding, we can conceptualise symptoms as an expression of processes of adaptation within which metacognition is at the heart of where psychopathology might be best understood in terms of the interplay between metacognitive capacities and competences, the utilisation of these competences, and the overarching social mentalities governing their deployment.

2. Metacognition and complex mental health problems

Dimaggio, Nicolò, Brüne and Lysaker, the guest editors of this special section, have brought together a stunning collection of articles from respected researchers and clinicians around the globe to deliver a body of work concerning the key role of metacognition in adult psychiatric and psychological problems. This is an important and growing domain of research, which has the potential to revolutionise our understandings of the expression of psychopathology and the alienation of emotional and interpersonal distress. A crucial aspect of Dimaggio and colleagues’ volume is the recognition that metacognition is a transdiagnostic process, which has much to tell us about the unfolding of symptoms over time and candidate mechanisms of change in recovery (whether this recovery is assisted or unassisted).

Metacognition (the concept of which overlaps with Mentalization and Theory of Mind as used in this section) has been variously defined as the capacity to conceive of one’s own and others’ mental states as explanations of behaviour (Fonagy and Target, 2006); the cognitive ability to attribute mental states such as thoughts, beliefs and intentions to people, allowing an individual to explain, manipulate and predict behaviour (Sprong et al., 2007); or the ability to think about one’s own inner states, and the inner states of others, allowing for complex self-experience and coping with distress (Semerari et al., 2003). Whilst these various definitions of metacognition appear to reflect a degree of commonality in approach, this masks significant complexity and differences in the underlying disorder-specific models of metacognition. Brüne et al. (2011-this issue) have argued that in the absence of an agreed definition of metacognition there is agreement that a wider definition of metacognition involves the perception and processing of social signals to construct representations that flexibly guide social behaviour, and that more narrowly defined definition of metacognition represents the ability to form and manipulate mental representations of one’s own and others’ mental experiences, which includes beliefs, desires, intentions, feelings and dispositions.

Disruption in the capacity for or utilisation of metacognition has far-reaching consequences for an individual’s self-experience and functioning in the interpersonal environment (Brüne et al., 2011-this issue). This is crucial since at the heart of many forms of adult psychopathology the impairments of social functioning (as in those diagnosed with schizophrenia; see Lysaker et al., 2011a,b,c-this issue), interpersonal functioning (as in those diagnosed with personality disorders; see Carcione et al., 2011-this issue) and affective functioning (as observed in alexithymia; see Vanheule et al., 2011-this issue) are likely to be strongly linked to metacognition. Simultaneously we can also observe disturbance of affective expression with the diminution of positive and negative affect observed in schizophrenia (Stratta et al., 2011-this issue), cognitive disorganisation in schizophrenia (Lysaker et al., 2011a,b,c-this issue) or the overregulation of affect observed in alexithymia and personality disorders (Nicolò et al., 2011-this issue). Of course, these observations of the expression of psychopathology (reflected in social, interpersonal and affective functioning) in adulthood should not be regarded as separate or compartmentalized by diagnosis. In this sense, diagnosis might be regarded as a marker of symptom expression. Diagnosis may therefore create artificial boundaries between forms of human distress that have common mechanisms and shared developmental pathways (Gumley, 2010). An important implication of this is the exploration of neurobiological mechanisms of metacognition.

3. Neurobiology of metacognition

Brunet-Gouet et al. (2011-this issue) specify three levels of processing relevant to metacognition. At the basic representational level of social information, we can consider the inputs we experience from the external environment (such as others’ gestures, facial expressions, and speech utterances) or from the internal environment (internal cognitive, affective, physiological and kinaesthetic events, as well as our own actions and reactions). At a second level for these inputs to have salience, the events must activate corresponding internal associated representations stored in episodic/procedural or semantic memory. In this model, metacognition represents a higher-level executive mechanism involved in managing multiple aspects of representations that are concurrently activated by inherently complex everyday social interactions. In this context the “mentalizing process thus acts on the set of activated representations (or enables retrieval of additional representations from memory) and allows their sequencing, contextualization, selection, and inhibition of prevalent representational properties in order to reach the most likely inference”. In keeping with this higher order function of mentalization, Raposo et al. (2010) explored patterns of functional magnetic resonance imaging (fMRI) activation associated with different types of mentalizing tasks that were focused on Self, Other and the relationship between self and other (Relational). They found that Relational mentalizing was associated with increased activation of the lateral frontopolar cortex (FPC) relative to mentalizing tasks involving Self only. Tasks involving Other relative to Self demonstrated medial but not lateral FPC activation. These findings suggested functional dissociation of mentalizing skills along the medial-lateral axis of the FPC. Therefore, mentalizing is more than just understanding the self and understanding the other but is also understanding the intersubjectivity of self-other relatedness. Brunet-Gouet et al. (2011-this issue) have called for an integrated model of social cognition based in neuroscience. Their Theory of Shared Representation (based on Decety et al., 2007) proposed that these representational systems each have corresponding underpinning neurobiological mechanisms. For example, a growing number of fMRI studies have shown that the observation of pain in others is mediated by several brain areas that are implicated in processing the affective and motivational aspects of pain. Jackson et al. (2005) found that the anterior medial cingulate cortex (aMCC) and the anterior insula are closely involved in the coding of motivational-affective dimensions of pain. Level of activity within the aMCC was strongly correlated with ratings of imagined pain in oneself and others, suggesting that common neural circuits are involved in representing one’s own and others’ affective pain-related states.

This link between social cognition, neurobiological organisation and brain development is crucial. Metacognition is regarded as an evolved mental capacity designed to enable primates and particularly humans to function in complex social environments requiring the development of affiliative and affective bonds and necessitating competition for resources (Brüne and Brüne-Cohrs, 2006; Fonagy and Target 2006). Evolutionary approaches map metacognition closely to brain development and organisation. Brüne and Brüne-Cohrs (2006) concluded that theory-of-mind tasks appear to map onto a neural network comprising the temporal lobes, the inferior parietal cortex, and the frontal lobes. Saxe and her colleagues (Saxe and Wexler, 2005; Saxe et al., 2006) have found a role for the right temporoparietal junction for performance on theory-of-mind, self-reflective and autobiographical memory tasks, which is consistent with the developmental evidence on the inter-relationships amongst these functions. Fonagy et al. (2007) have suggested that the brain structures underpinning social cognition are also implicated in emotion processing and suggest that a two-component model of metacognition based on implicit (automatic) and explicit (reflective and controlled) processing systems with separate but related underlying neurobiological systems. Metacognition-based approaches to schizophrenia have tended to emphasise the cognitive components of theory of mind, that is, those
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