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A neural model of mechanisms of empathy deficits in narcissism

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From a multidimensional perspective, empathy is a process that includes affective sharing and imagining and understanding the emotions of others. The primary brain structures involved in mediating the components of empathy are the anterior insula (AI), the anterior cingulate cortex (ACC), and specific regions of the medial prefrontal cortex (MPFC). The AI and ACC are the main nodes in the salience network (SN), which selects and coordinates the information flow from the intero- and exteroreceptors. AI might play a role as a crucial hub – a dynamic switch between 2 separate networks of cognitive processing: the central executive network (CEN), which is concerned with effective task execution, and the default mode network (DMN), which is involved with self-reflective processes. Given various classifications, a deficit in empathy may be considered a central dysfunctional trait in narcissism. A recent fMRI study suggests that deficit in empathy is due to a dysfunction in the right AI. Based on the acquired data, we propose a theoretical model of imbalanced SN functioning in narcissism in which the dysfunctional AI hub is responsible for constant DMN activation, which, in turn, centers one's attention on the *self*. This might hinder the ability to affectively share and understand the emotions of others. This review paper on neural mechanisms of empathy deficits in narcissism aims to inspire and direct future research in this area.

Key words: narcissism • empathy • salience network • anterior insula • anterior cingulate cortex

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Empathy and Narcissism

The definition and operationalization of empathy have been subjects of scientific debate over the past decades. In the affective domain, empathy is often understood as the ability to share and co-experience the feelings of others [1–3]. From the cognitive perspective, empathy is the ability to imagine and understand the emotions and motives of others [3–7] and the ability to be consciously aware of their thoughts, intentions, and desires, which is known as mentalizing [8] or having a theory of mind [5,9]. Currently, the multidimensional conceptual framework of empathy combines all of the aforementioned perspectives and describes empathy as a complex process [3,4].

Considering the wide spectrum of often-conflicting definitions in the literature, narcissism is a difficult concept to precisely define. Currently, there exist 2 separate axes of distinction. The first axis divides narcissism into 2 categories with respect to the severity of the symptoms – a healthy type and a clinical (dysfunctional) type. The healthy type can be considered as a functional and sometimes advantageous set of personality traits [10]. The characteristics of this axis involve high self-esteem, deficits in interpersonal relationships, a high likelihood of career success, and low empathy. The clinical type is synonymous with narcissistic personality disorder (NPD). The characteristics of this axis involve a fixed and inflexible pattern of delusions of self-importance and uniqueness, an excessive need for admiration, and a lack of empathy, diagnosed according to the DSM IV [11]. The second axis distinguishes between grandiose and vulnerable types of narcissism [12], often alternatively referred to as overt and covert types, respectively [11]. High levels of extraversion, self-confidence, self-esteem, exhibitionism, and aggression characterize the grandiose type; introversion, low self-esteem, anxiety, and high susceptibility to traumas are associated with vulnerable type [12–14]. The traits that are associated with all narcissism subtypes are selfishness, disregarding others, self-centeredness, and low empathy.

Neural Mechanisms of Empathy – from Affective Sharing to Understanding Others

The most primal and automatic component of empathy involves the bottom-up processing of perceived emotions and reacting to others with a similar affect. This phenomenon is described either as affective resonance [15] or affective sharing [16]. However, there is an on-going debate as to whether this 'shared' feeling is. Are we expressing qualitatively similar sadness while observing someone experiencing such an emotion, or is our expressed emotion simply a state of negative arousal, caused by feeling uncomfortable around someone who is sad? In the latter case, the term 'affective arousal' would be more fitting. On-going research in this area has not yet provided a definitive answer, and the resulting reports seem to contradict one another [17].

The 'perception-action' model [18] explains this phenomenon as activation of similar brain regions in the observer and in the others when the observer watched or imagined the emotional state of others. This hypothesis supports the affective sharing concept of connected mirror neurons [18,19]. Most research on pain empathy indicates co-activation (in both the self and observed pain conditions) of the anterior cingulate cortex (ACC) and anterior insula (AI) [20–30]. Both structures are constituents of the pain matrix. Although such results partially support the perception-action hypothesis, it remains unclear why activation of the primary and secondary somatosensory cortices was never noted in the observed pain condition [20,21].

There are also results supporting the 'affective arousal' hypothesis that emphasizes the differences in activation between the conditions [17,27,31–33]. According to Zaki et al. [31], the AI and the ACC are overlapping parts of 2 distinct circuits: one is involved in experiencing pain, and the other is involved in observing it. A meta-analysis of 32 pain empathy-related studies led Lamm et al. [34] to hypothesize that the differences in activation areas may be due, at least partly, to discrepancies among experimental paradigms. Somatosensory cortices in the pain-observing condition were active only when the presented pain involved deep punctures.

There is also evidence of AI and ACC activation from the sparse research collected on disgust empathy [35–37], fear empathy [38], anger empathy [39], and sadness empathy [40]. These data support the hypothesis that these 2 structures are essential for bottom-up empathetic processing (Figure 1).

Common bottom-up neural pathways could explain how we can experience the feelings of others as our own [41]. However, emotional contagion or mimicry cannot be considered empathy. Furthermore, cognitive processes must lead to putting those feelings into perspective and distinguishing between ones concerning oneself and ones concerning others. This top-down processing is performed by the prefrontal regions [17], modulating the intensity of the ascending affective path [19]. The other person's perspective to take and then understand others' feelings and intentions enables the observer to act in a context-specific manner; for example, expressing empathetic concerns. Neuroimaging studies show different patterns of activity while imagining and understanding others' affective states, thoughts, and desires. While an individual is imagining, the most active regions include the MPFC [42–46], the temporo-parietal junction (TPJ), the superior temporal sulcus (STS), and the temporal pole (TP) [47]. During understanding, however, the MPFC, VMPFC, and temporo-occipital junction are the regions most involved in processing [17].

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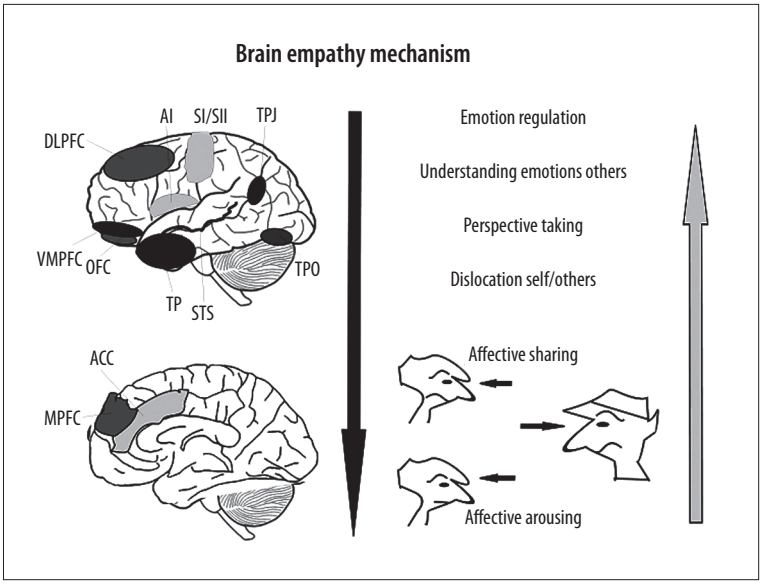

Figure 1. Brain empathy mechanisms. The figure presents brain regions that are active during empathy. Ascending bottom-up processing pathways are highlighted in light-grey colour and descending top-down pathways are highlighted in dark grey and black. MPFC – medial prefrontal cortex, TPJ – temporo-parietal junction, STS – superior temporal sulcus, TP – temporal pole, ACC – anterior cingulate cortex, AI – anterior insula, SI/SII – somatosensory cortex, VMPFC – ventral medial prefrontal cortex, TPO – temporo-occipital junction, DLPFC – dorsolateral prefrontal cortex, OFC – orbitofrontal cortex; bottom-up (light-grey arrow); top-down (black arrow) [after: 16,19; modified].

Another crucial skill involving empathy is control over the expression of experienced emotions, referred to as emotion regulation. This skill enables behavioral control over the intensity and quality of expressed emotions, depending on the social context. Emotion regulation is extremely important from an evolutionary perspective. In our culture, lack of behavioral control in a given context (for example, if one were to burst out laughing after seeing someone fall down the stairs, or to assault a bullying boss at work) is often considered inappropriate and may lead to social ostracism or even loss of social and economic status. Emotion regulation correlates with traits such as self-control, inner discipline, and the expression of sympathy [48]. Structures engaged in emotion regulation are the orbitofrontal cortex (OFC), the MPFC, the DLPFC, and the ACC. Together, these structures constitute a network that sends information to regions involved in processing emotional information, such as the STS or the amygdala [17].

Empathy Deficits in Narcissism

According to DSM-IV, lack of empathy is one of the key symptoms of NPD. This association has been demonstrated in many experiments and clinical observations [49–51]. Research shows deficits predominantly in the bottom-up processing domain among narcissists, but the cognitive components of empathy seem to be impaired as well [15,33,51]. The use of new tools such as the Multifaceted Empathy Test [52] and the Movie for the Assessment of Social Cognition [53] in studies of NPD patients showed impairments only in the affective arousal/sharing component of empathy. However, a different study [54] that used a classical test of recognizing and understanding facial expressions of fear, anger, disgust, joy, and sadness revealed additional deficits in emotion recognition (mostly fear and anger) that are governed by cognitive top-down processes. This tendency among narcissists to perform worse in the recognition task holds true irrespective of the exposure time.

DSM-IV describes a lack of empathy among people with NPD, as a result of a volitional unwillingness to identify with others' feelings and needs [11]. However, some of the aforementioned studies may suggest that the inability to understand emotional expressions might also be a major cause of empathy impairments [33,51]. The correlation between deficits in facial expression recognition and empathy is well documented [55–57].

A neuroimaging experiment in a sample of non-clinical narcissists was performed by Fan et al. (2010). The group was divided into high narcissism (HN) and low narcissism (LN) subgroups. The task involved empathizing with presented pictures of emotional faces. The results showed lower deactivation of the right AI (rAI) and higher activation of the posterior cingulate cortex (PCC), DLPFC, and premotor areas during the control condition (non-emotional faces) among HN subjects. These results could mean that top-down inhibition might be insufficient to properly modulate the affective arousal due to an overactive rAI, which responds even when non-empathetic stimuli are present. The HN group also scored higher on the Interpersonal Reactivity Index questionnaire; specifically, on the Personal Distress subscale, which measures self-oriented feelings of personal anxiety and unease in stressful interpersonal settings. This result provides further evidence that narcissists might have trouble separating the self from others. Taking into account the prominent role of the rAI in the representation of self [58,59] and high scores on the self scales in the Narcissism Inventory, we might speculate that the rAI plays a crucial role in empathy deficits among narcissists.

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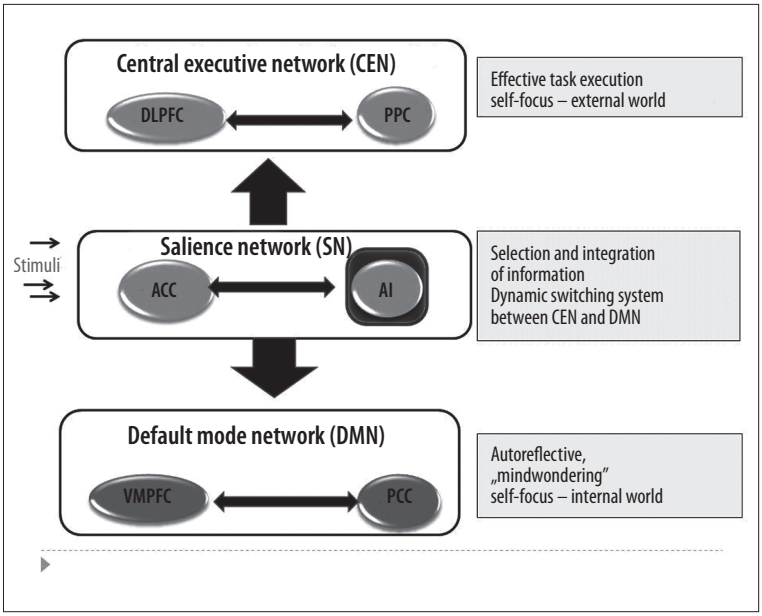

Figure 2. Brain network models of cognitive and emotional processing. Black bolded arrows show the SN dynamic relative to CEN and DMN. DLPFC – dorsolateral prefrontal cortex, PPC – posterior parietal cortex, ACC – anterior cingulate cortex, AI – anterior insula, VMPFC – ventral medial prefrontal cortex, PCC – posterior cingulate cortex [after: 82; modified].

The Role of the Anterior Insula

The AI is connected with the prefrontal (OFC, DLPFC), temporolimbic (TP, parahippocampal area, amygdala, ACC), and subcortical (basal ganglia, thalamus, brain stem) regions [60–62]. Most of those connections are bidirectional [58]. According to Craig's model, the information flow from the interoreceptors and exteroreceptors of the body is processed and mapped in the posterior part of the insula and then represented in the anterior part [58,63–65]. The AI integrates emotionally significant internal and external information, which then becomes the subject of further selection and control in the ACC. This process might be the foundation of conscious awareness of emotionally significant states [63–65]. Many studies have shown that the rAI is active both during experiencing and anticipating emotions [66–69].

The AI and the ACC are believed to be the central nodes in the salience network (SN) [70,71]. The SN responds to behaviorally salient events by integrating them and giving them an emotional context. The rAI shares reciprocal connections with the amygdala, hippocampus, ACC, OFC, TP, and olfactory cortex [71,72]. These regions collectively activate in response to a wide variety of stimuli, such as pain [73], pain empathy [20], and metabolic stress or hunger, as well as activating in response to pleasant stimuli such as touch [63], hearing one's favorite music [74], or seeing familiar faces [75]. The rAI is also active in response to social rejection, high anxiety, or low self-esteem [76], all of which are common characteristics of vulnerable narcissism. Other SN nodes consist of subcortical structures that regulate emotions, homeostasis, and reward signals [77].

A causal analysis of the fMRI signals with Granger's test indicates that AI might be a central hub – an on/off switch between 2 networks: the central executive network (CEN) and the default mode network (DMN). The CEN includes the DLPFC and posterior parietal cortex and is correlated with effective performance (both speed and accuracy) in a given cognitive task. Its efficacy is dependent on the number and strength of reciprocal connections within the network. DMN involves VMPFC and PCC [78] and is active during auto-analysis, 'mind-wandering', and detachment from the outside world [81–83]. This network might be a neural instantiation of the self [84,85]. However, in some cases, DMN shows increases in activity during processing information about the mental states of others [84] and understanding social interactions between others [86–88]. The SN might act as a dynamic system of attention shifting between the outside world and external events controlled by the CEN, and self-focus and internal processes managed by the DMN [78,82,83] (Figure 2).

Research results connect rAI size [89] and its connectivity with neighboring structures [83] with the degree of expressed anxiety. Carlson and Mujica-Parodi [67] noted a strong correlation between rAI and amygdala co-activation during anxious anticipation, suggesting that estimating the degree of a pain hazard is one of the functions of rAI. Ibanez et al. [91] formulated a hypothesis about the insula being the crucial structure in the human threat detection system. Designing their experiment, they assumed that other faces are processed in a different way than one's own face and that they might be perceived as a potential threat. Event-related potential (ERP) results have shown that reaction times in response to pictures of pain when primed with other faces were significantly faster than when primed by one's own face, suggesting that those 2 processes might be managed by slightly different networks [91].

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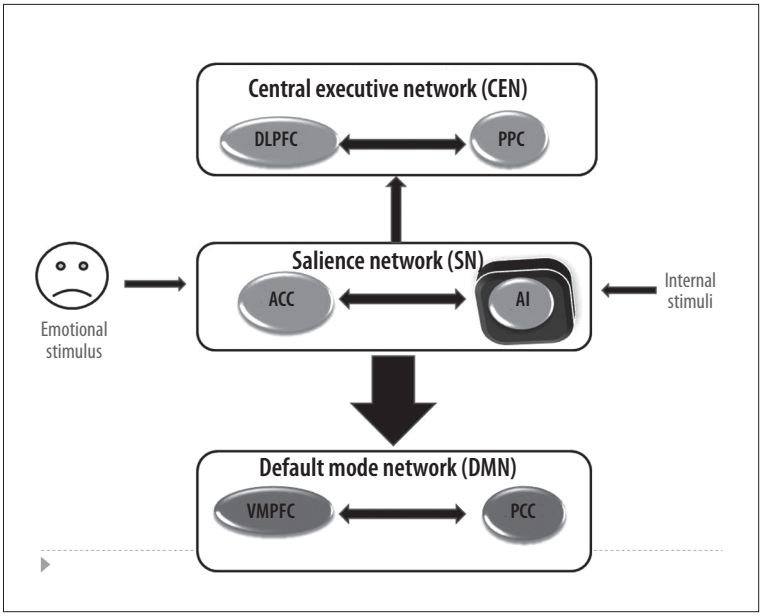

Figure 3. Model of SN dysfunction in NP. DLPFC – dorsolateral prefrontal cortex, PPC – posterior parietal cortex, ACC – anterior cingulate cortex, AI – anterior insula, VMPFC – ventral medial prefrontal cortex, PCC – posterior cingulate cortex [after: 82; modified].

The Dysfunctional Model of SN in Narcissistic Persons (NPs)

Looking at the function of the insula, which is involved in switching between brain networks, together with evidence from a neuroimaging study in NPs of lower deactivation of rAI, we assume that some deficits in empathy at the neurobiological level in NPs may be associated with a dysfunctional SN, resulting in a disorder of affective sharing and understanding the emotions of others. Models of AI dysfunction in SN were proposed in autism [82] and schizophrenia disorder [83]. Both of these disorders are characterized by some deficits in empathizing. We propose that dysfunction of switching in the SN between the DMN and the CEN will lead to the hyperactivity of only 1 of them. In the case of NPs, it is apparently getting attention or thinking of oneself. This effect may arise from the impaired switching of the SN, leading to hyperactivity of the faster DMN. The DMN consists of regions that are typically more active during "mind wandering" or self-referential processing [92]. Interestingly, some areas within the DMN are also activated when participants infer the mental states of other people [85,88,93,94]. It has been suggested that the DMN comprises at least 2 different anatomical and functional subsystems [95], which might be involved in representing both self and others [85]. However, it is likely that in NPs, DMN might have only 1 main function, which involves constant self-monitoring. In NPs, internal stimuli – thoughts concerted around the self – may activate the rAI. NPs tend to think of themselves or to get themselves a neutral stimulus more often than others. If a focus on the self leads to the activation of the same brain regions that are activated during the observation of others' pain, the proper processing of both stimuli simultaneously might be difficult due to the limited efficiency of the rAI. Such thoughts might explain some disorders with distorted processing of affective stimuli derived from the external world, causing deformation of salience judgment and decreased empathy. The model of SN dysfunction in NP is presented in Figure 3.

Considering that the insula is the crucial structure in the human threat detection system, we assume that in NPs, a dysfunction in the rAI might lead to the abnormal evaluation of many emotional stimuli derived from external world as threatening stimuli. This effect might lead to sensitization and difficulty in suppressing the reaction of the threat detection system. Extended activation of the alarm system might travel to the prefrontal cortex and disturb the proper recognition of a threat level. Moreover, this excessive concentration around danger disfavors helping behaviour, especially in regard to someone who presents some source of threat. This explanation is in accordance with studies by Mikulincer et al. [96], who observed a negative correlation between insecurity and helpfulness and supportiveness.

Abnormal activation of the rAI in NPs might hinder grasping and understanding the perspectives of others. The experience of extremely sensitive NPs at any given moment mirrors the experience that they observe in others. This generates a sensitivity to threats that may be connected with dysfunction of the SN and might provide a basis for vulnerable, narcissism-sensitive, highly reactive anxiety that is highly analogous to social phobia. Social phobia is characterized as a mainly negative state of mind [97] and a hyperactive autonomic nervous system. In addition, hyperactive responses to negative stimuli might lead to increased anxiety [98].

Another manner in which the extended activation of the rAI might produce a lessened or slowed self-inhibition is via the bottom-up

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approach, which leads to secondary disorganization of the switching system in the SN. If so, extended (or chronic) activation of the rAI might lead to an extended period of affective stimulation of self-focus in NPs. In this way, NPs appear to have a high level of sensitivity for personal distress, resembling that of functional psychopaths [51]. This is in agreement with the grandiose type of NPs and many clinical observations. Kernberg [99] has suggested that narcissism might be a central part of psychopathy. High levels of impulsiveness and aggressiveness and low levels of guilt and empathy characterize the psychopathic state [11,100]. Many similarities are observed on the physiological level between these 2 disorders, such as similar responses to stress and negative arousal, measured by galvanic skin response (GSR) [101]. Currently, the grandiose type of narcissism is better studied [102].

Conclusions

Despite various definitions of narcissism, the following traits are shared: selfishness, disregarding others, self-centeredness, and low empathy [12]. Some crucial studies of NPs have shown that these deficits of empathy concern the levels of affective sharing or arousal [15,33,51], understanding emotions [54], and emotion regulation [51]. Differences in neuronal activity in the rAI (lower deactivation), DLPFC, and PCC (higher activation) of NP were observed in the non-empathic control condition compared to control samples [33]. The rAI is also an important brain structure in experiencing and anticipating emotions [66–69] and is involved in self-representation [58,59]. Moreover, the rAI and the ACC are typically associated with empathizing with others [103].

Analysis of social networks indicates that these structures are central hubs of the SNs [70,71]. The rAI plays a critical and causal role in switching between the CEN and the DMN [78,83].

Based on these studies, we propose a theoretical model of imbalanced SN function, which may explain the neuronal deficits of empathy in NPs. We assume that there is faulty switching between the CEN and the DMN, leading to increased DMN activation, which, in turn, centers one's attention on the *self*. At the same time, high activation in the SN, caused by a large stimulus while empathizing with others, could lead to decreased affective sharing and increased personal distress. High amounts of stimulus within the internal world in narcissists may impair rAI function even more. As a consequence, the processing of external stimuli may be contorted, and from the psychological point of view, NPs will be observed as having issues grasping the perspectives of others. This is a proposed explanation for some deficits of empathy in NPs. This is a review paper and it aims to inspire and direct future research into the neural mechanisms of empathy deficits in narcissism.

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