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Editorial

The neuroscience of empathy – from past to present and future

Research on the neural basis of empathy has undergone a significant transformation over the past fifty years. From early studies focusing on basic neuroanatomy, this field has developed into many branches that explore the complex etiology and mechanisms underlying empathy, and its implications for mental health and social behavior. As early as the 1970s, and 80s, researchers began to theorize about the potential neural basis of empathy (Heilman et al., 1975; Brothers, 1989). Early empathy research, throughout the 1990s, focused primarily on brain lesion studies and implicated cerebral and frontal lobe lesions in empathy deficits (e.g. Grattan et al., 1994).

The solid foundation of lesion studies laid the groundwork for neuroimaging studies examining the neural underpinning of empathy in healthy populations. With the technological advances of the 21st century, studies began using functional magnetic resonance imaging (fMRI) to research empathy, frequently using empathy for pain as their experimental paradigm. Two seminal studies (Decety and Jackson, 2004; Singer et al., 2004) simultaneously posited that a specific set of regions of the 'pain matrix' (specifically the anterior cingulate cortex [ACC] and anterior insula [AI]) are activated both by experiencing pain and by watching others experience pain. A surge of studies of empathy ensued, replicating these findings and showing how empathy related activations are modulated depending on the context (Hein and Singer, 2008, for review; Lamm et al., 2011, for meta-analysis).

Based on an increased understanding of the mechanisms underlying empathy, research concerning different subtypes of empathy has emerged. This evolution in defining empathy has led to more nuanced research, as subsequent studies often investigated these subtypes. One model divides empathy into emotional empathy – sharing the emotions of others – and cognitive empathy – understanding the thoughts and motivations of others (Shamay-Tsoory et al., 2009). Another model divides empathy into personal distress (also called empathetic or vicarious distress) – which is self-oriented and focused on alleviating one's own pain – and empathetic concern (also called compassion) – which entails feeling sympathy for another person (Eisenberg and Eggum, 2009; Singer and Klimecki, 2014).

Given these advances in understanding empathy, further research has explored the societal issues of empathy. One line of research focuses on empathy biases and group membership. Many fMRI and EEG studies have shown reduced levels of empathy directed towards people of different racial or social groups (Han, 2018, for review). Another line of research focused on the role of empathy in psychopathology, as variety of psychiatric and neurological conditions - including autism, schizophrenia, bipolar disorder, borderline personality disorder and psychopathy - are associated with deficits (or surpluses) in different subtypes of empathy (Gonzalez-Liencres et al., 2013). Other research has focused on the neurochemical basis of empathy, implicating opioids (Rutgen et al., 2017, in this issue) and neuropeptides, mainly oxytocin, in empathy (e.g. Hurlemann et al., 2010). This line of research has clinical implications for a range of disorders, including autism and schizophrenia.

In recent years criticisms of the methodologies commonly used to study the neural basis of social behavior are beginning to emerge (e.g. Schilbach et al., 2013), motivating methodological advances towards increasing the external validity of the findings in social neuroscience. Owing to the increasing importance of understanding social behavior in natural settings the field of empathy recently took a step towards understanding more real-life empathic interactions with either interactive avatars (Jackson et al., 2015) or real face-to-face interactions (Goldstein et al., 2018). Indeed, given that empathy by its nature requires active participation in social exchange with social agents, measuring a response of an isolated passive observer may fail to capture the genuine mechanisms of empathy. Based on the more widespread use of brain stimulation methods as well as of psychopharmacological manipulations, we are also beginning to gain more causal-mechanistic insights into the neural underpinnings of empathy, and how they are related to (pro)social behavior (Lamm et al., 2017, for review).

In light of this history and the more recent developments, the present special issue of *Neuropsychologia*, "The neuroscience of empathy – from past to present and future," has compiled a broad range of contributions advancing our understanding of the intricacies of empathy, which we would like to summarize and highlight as follows.

1. Exploiting individual differences to better understand the neuroplasticity of empathy

The papers by Engen et al. (2017) and Patil et al. (2017) address questions related to how variations in brain structure may underpin individual differences in empathy, compassion and prosocial behavior. Using a costly helping task implemented in a virtual reality scenario, Patil & colleagues show that prosocial helping is associated with higher trait empathic concern and an enlarged anterior insula, which as outlined above has been consistently associated with empathy in fMRI studies. Engen et al. directly complements these findings, by showing that long-term practitioners of a Buddhist meditation practice cultivating "loving kindness" (an attitude akin to compassion) show higher cortical thickness in the anterior insula as well. However, there is an interesting discrepancy in between the two studies, with Patil et al. reporting differences in right and Engen et al. in left AI. This raises the question whether trait- vs. training-related neuroplasticity targets different aspects of AI, a brain region generally linked to emotional

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awareness and interoception (Craig and Craig, 2009).

2. Reward and empathy?

Individual differences also play a key role in Luo and Zhang (2017), whose work points to an aspect that has somewhat been overlooked in research on empathy for pain: what if the target person enjoys rather than suffers from the pain experience? By investigating a sample regularly engaging in sexual practices involving deliberate acts of giving and receiving pain, they indicate that this results in reduced evaluations of the pain of others, and associated event-related potentials (ERPs). Schadenfreude is another example where negative experiences of a target triggers positive affective responses in an observer. However, which factors that decide whether we respond to potentially embarrassing failures of others with positive or with negative affect remains an open question. Paulus et al. (2017) thus investigated the neural networks underpinning schadenfreude or fremdscham (i.e., gloating vs. vicarious embarrassment). They show that modulation of activation and connectivity between the anterior insular cortex and nucleus accumbens seems to determine to what extent someone experiences schadenfreude or fremdscham. This opens up interesting questions on how the reward circuitry including the meso-cortico-limbic dopamine system may shape empathic vs. counter-empathetic responses, especially in social interactions characterized by a competitive or non-affiliative relationship (Dvash et al., 2010; Yamada et al., 2011). Mimicry, i.e. the spontaneous and largely automatic motor matching of other people's actions or expressions, could play an important role in this respect. This aspect is addressed by Hsu et al. in this issue (Hsu et al., 2017). When participants in their study were allowed to spontaneously mimic another person's positive emotions, they showed increased activation in reward-related areas, and this also correlated with individual differences in trait empathy. Investigating such vicarious reward (Morelli et al., 2015, for meta-analysis) and showing that being more empathic can have beneficial effects may also shift the focus away from the “dark side” of empathy - and the prevalent view that we somewhat might need to protect ourselves from the distress of others in order to not get overwhelmed by their plight (Gleichgerrcht and Decety, 2013).

3. Empathy and interaction

In real life, empathy does not happen in a vacuum, and is not a one-way street that only flows from an active observed person to a passive observer. Rather, it serves to facilitate social interaction, as well as to affiliate and form social bonds. Since the dynamics of social interaction are difficult to control and investigate in the lab, Social Neuroscience has for a long time ignored this aspect. However, with the advent of “hyperscanning” techniques and other data collection approaches allowing to capture how empathic interaction unfold over time and what lasting effects this may have, the field has started to fill this research gap (Babiloni and Astolfi, 2014). Two papers in the special issue took on this challenge and examined real-life empathic interactions. In a laboratory study with particularly high ecological validity, Peled-Avron et al. (2017) investigated the effects of consoling touch, a distress-relieving strategy aimed at reducing the suffering of a target. When delivering such touch to a romantic partner in pain, empathy-related neural responses in the consoler were significantly increased. Abraham et al. (2017) exploit a particularly rich dataset that allowed them to investigate how empathy-related brain responses in parents predict their children's stress reactivity in infancy, preschool and at age six. Their findings are exciting as they show that parental responses related to embodied simulation are linked to stress reactivity of their children, while those related to mentalizing are rather associated with emotion regulation skills. This connects to the discourse on “affective” vs. “cognitive” empathy touched upon above, and suggests that this distinction of empathic components is not only of theoretical relevance, but has direct implications for parent-child interaction and education.

4. From neural correlates to causal mechanisms

A great deal of social neuroscience research has been devoted to identify to localize the brain areas associated with in empathy and associated social emotions and behaviors. However, such a “neural correlates” approach necessarily only constitutes a first step towards a mechanistic understanding. Recent years have thus seen an increase in research approaches allowing causal inferences. Several papers in the special issue followed this research approach. Coll et al. (2017) used transcranial direct current stimulation (tDCS) to investigate the role of the right temporo-parietal junction (rTPJ) in empathy for pain. They report that inhibitory stimulation of this brain area results in less intense evaluations of the pain of others, and is associated with matching changes in event-related potentials. This confirms ample evidence linking this brain to self-other distinction (Lamm et al., 2016, for review), a crucial component of any kind of empathic experience. Their findings are extended by those of Paracampo et al. (2018), who used another brain stimulation method – repetitive transcranial magnetic stimulation (rTMS) and demonstrate that the findings of Coll et al. (2017) do not only apply to pain, but also to empathic evaluations of positive emotions, such as laughing. Moreover, their finding that only highly empathic participants showed such inhibitory effects indicate that individual differences in trait empathy might need to be taken into account more systematically in future research. Rutgen et al. (2017) and Qiao-Tasserit et al. (2017) aimed for causal inferences by exploiting a special twist that has recently been brought back into the experimental spotlight. This is to experimentally manipulate affect in their participants to test whether this also changes empathy, allowing a more specific exploration of the role emotions felt by the self play in sharing and understanding the emotions felt by others. Rütgen et al.’s (2015a, 2015b) work extends earlier findings of their group that placebo analgesia also reduces pain empathy (see also Mischkowski et al., 2016), and that this is associated with the endogeneous opioid system (Rütgen et al., 2015a, 2015b). Using ERPs, they show that administration of an opioid antagonist blocks the effects of placebo analgesia on pain empathy not only on the behavioral level, but that also be modulation of a neural signature of pain processing, the pain-related P2. Quiao-Tasserit et al.’s (2017) work points towards an additional factor that needs to be taken into account by future research, which is the valence of the experimentally induced emotion. Their findings indicate that depending on whether positive or negative transient emotional states are induced, participants perceive the pain of others in different ways: while inducing unpleasant emotion reduced neural and autonomic responses linked to pain empathy, as in Rütgen et al.’s work, inducing positive emotion resulted in a more similar response of pain experienced in the self with pain empathy. Interestingly, these effects were partially counteracted by individual differences in trait empathy, thus again pointing towards the importance of considering individual differences in future research. Somewhat relatedly in terms of the experimental approach, the work by Luo et al. (2017) manipulated physical coldness versus warmth to investigate how this affects empathy and racial bias in pain empathy. Following up on social psychological work focusing on felt similarity and simulation mechanisms for empathy (O'Brien and Ellsworth, 2012), they demonstrate that holding a cold pack in one's hand (and thus possibly being in an unpleasant physical and affective state) increases neural responses to facial expressions of pain in same-race as compared to other-race members. Future studies will need to show whether this can be explained by cognitive effects, such as better recognition of emotional expressions matching the bodily state of the observer, and whether this may be the mechanism for increases in affect sharing.