Connecting minds and sharing emotions through human mimicry

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Chapter 3

The neurocognitive model of emotional contagion (NMEC)

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The neurocognitive model of emotional contagion (NMEC)

The core assumption of the perception action model of empathy proposed by Preston and de Waal (2002) is that perceiving a target's state automatically activates the corresponding representations of that state in the observer, which in turn activates somatic and autonomic responses. In line with the perception–action mechanism, a number of behavioral studies using EMG, demonstrated that viewing facial expressions triggers similar expressions on the observer's own face (Dimberg et al., 2000, Kret et al., 2013a, Kret et al., 2013b). This observation has been related to the discovery of the Mirror-neurons system (MNS) in the premotor area, F5, of the macaque monkey which discharges not only during action execution but also during action observation (Di Pellegrino et al., 1992). Further fMRI studies in humans have shown that the perception of a specific affective state activates similar neural systems in the observer that are responsible for the generation of that state. This has been observed in the domains of pain (Jackson et al., 2006b), fear (de Gelder et al., 2004, Hadjikhani and de Gelder, 2003), disgust (Wicker et al., 2003), anxiety (Prehn-Kristensen et al., 2009), reward (Mobbs et al., 2009), as well as higher order affects such as envy (Takahashi et al., 2009) and embarrassment (Krach et al., 2011).

In 2011, Anders, Heinzle, Weiskopf, Ethofer, and Haynes used information-based fMRI to investigate the flow of affective information between two interactive brains of romantic partners. In this experiment, both partners were engaged in on-going face-to-face communication whilst inside the scanner. The sender (either male or female) was instructed to pose different emotional expressions to share his or her feelings with his or her partner (the receiver), while the partner was trying to understand the affective experience of the sender. Apart from cerebral blood flow, skin conductance responses (SCR) were also measured to assess the partners' physiological arousal during each interaction period. The neuroimaging results revealed that the level of neural activity within the emotion-specific network predicted the neural activity in the same network in the perceiver's brain, and this neural coupling was associated with synchrony of the autonomic system. Importantly, this was achieved with the same temporal resolution corresponding to the phase of partners' affective interactions. These findings fundamentally showed that during face-to-face interactions, the movements in the partner's face are directly projected to and can be decoded from the observer’s neural activation. This has been dubbed ‘neural resonance’ or ‘shared neural activation’ and has been demonstrated to be a robust and consistent phenomenon in emotion perception studies. This observation has transformed the way we think about neural architecture as it suggests that affective perception and its expression are not separate neurocognitive entities. These accounts provide empirical support for the hypothesis that neural resonance and automatic mimicry are directly involved in emotional contagion and empathy (Hatfield et al., 1994, Preston and de Waal, 2002).

In addition to this view, we propose that while mirror neuron activation and mimicry reflect on the degree to which people internally simulate perceived emotions, it is the perceptual input, not the neural/physiological resonance per se, that drives emotional contagion between species. Hasson et al. (2012) proposed that environmental stimuli, including faces, emit different forms of mechanical, chemical and electromagnetic energy. The sensory receptors convert these elements into electrical impulses that the brain then uses to gather environmental information and to coordinate an appropriate action. In this way, via the transmission of a signal through the environment, the neural processes in one brain can couple to the neural processes in another (Hasson et al., 2004, Stephens et al., 2010). To the best of our knowledge, at the time of writing this review, no neurocognitive model has incorporated different emotional signals into one neurocognitive framework. The present review therefore illustrates how affective information passes from one person's facial display to another's body and brain, and how the transition of perceptual input improves emotional understanding. The NMEC is illustrated in Fig. 3 and Fig. 4, which depicts a situation where a sender is experiencing a sudden feeling of anxiety while being observed by a receiver.

Imaging research has demonstrated that the anterior cingulate cortex (ACC), the medial prefrontal cortex (mPFC) and the temporoparietal junction (TPJ) play roles in perspective taking, self-awareness, and in more cognitive types of empathy (Carr et al., 2003, Decety and Lamm, 2007, Nummenmaa et al., 2008). The subcortical areas associated with, among other things, emotions, and the regions that are known to be part of the MNS such as the inferior frontal gyrus (IFG, Brodmann area 44), are associated with more basic emotional contagion/emotional empathy (Carr et al., 2003;

Shamay-Tsoory, Aharon-Peretz, and Perry, 2009). In the NMEC, we focus on these emotional contagion areas, which develop from early infancy onward.

NMEC: the autonomic mimicry pathway (Fig. 3)

One conceptual challenge mimicry poses is that it is difficult to determine when exactly a response qualifies as mimicry. For example, if one expresses fear following another's outburst of anger, this is an emotional reaction. But what if the result is an expression of anger? Is this still a reaction or should this be defined as mimicry? In our theoretical framework, we would like to conceptualize any type of interaction that results in corresponding autonomic levels between sender and receiver as autonomic mimicry. Accordingly, in the NMEC we argue that autonomic mimicry relies largely on the same neural substrates and pathways that are also involved in emotion responses and ANS activation. Aversive responses, including feelings of fear or anxiety, are modulated by hardwired neural circuits that share common neuroarchitectures among mammals (Le Doux, 2012, Parr and Waller, 2006). These basic evaluative systems are associated with the ANS and motor responses that together aid the adaptive responding of the organism (Decety, 2011). The feeling of fear is related to the activation of the sympathetic nervous system (Armony and Le Doux, 1997). Sympathetic nerves are located near the brainstem and the stress response is initiated by activation of the hypothalamic-pituitary-adrenal (HPA) axis (Herman et al., 2005). When the HPA axis is activated, the adrenal medulla secretes acetylcholine increasing (adrenaline) epinephrine and (noradrenaline) norepinephrine release. This, in turn, activates the cardio- vascular system and, as a result, heart and respiration rates increase and digestion slows down (Herman et al., 2005). Importantly, sympathetic nerves are directly connected to sensory channels on the surface of the body, such as the pupils, the muscles, and the skin (Ekman et al., 1983). For this reason, arousal can lead to autonomic pupil dilation, blushing, skin conductance, as well as involuntary facial/bodily expressions (see Fig. 3, Sender).

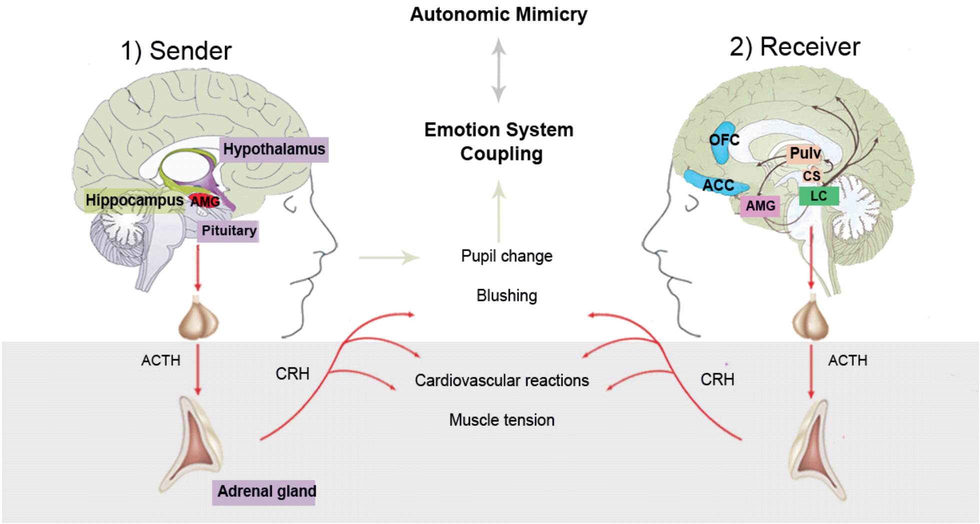

Fig. 3. Neurological Mechanisms of Autonomic Mimicry. Sender: (1) Sender's stress response is initiated by hypothalamus-pituitary-adrenal axis activation. (2) Adrenal gland secretes ACTH increasing the level of CRH in the bloodstream. (3) The neuroendocronological reactions are accompanied by cardiovascular changes, muscle tension, pupil dilation, blushing, and sweating. Receiver: (4) The affective information is implicitly registered by receivers' senses and passes through (5) the CS-Pulv pathway to the AMG. (6) The AMG and LC activate the HPA. (7) AMG and LC project to higher cortical networks such as OFC, ACC and VMPFC influencing social decisions. (8) Sender and receiver emotionally converge on physiological (gray) and cognitive (white) levels.

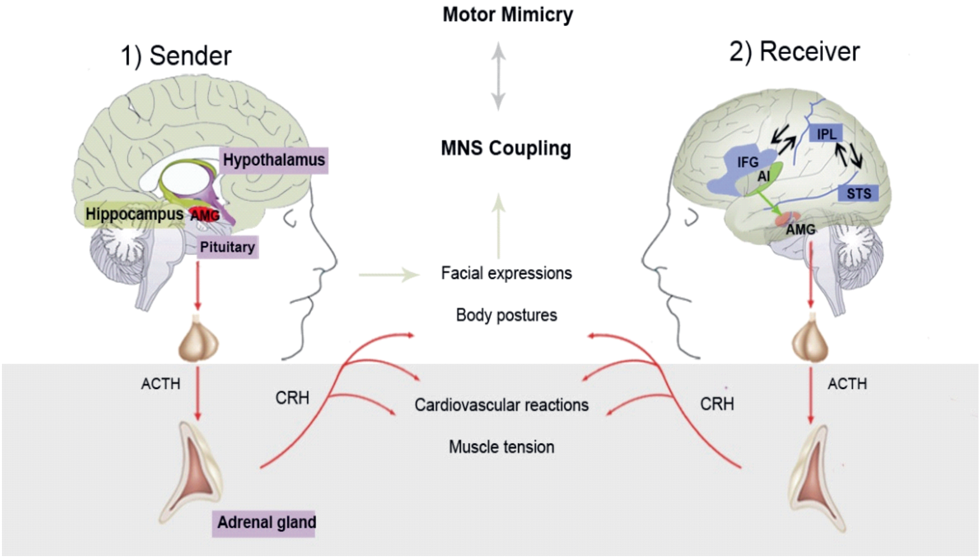

Fig. 4. Motor mimicry (1) Observation of bodily movements activates the STS which is involved in early visual description of actions. (2) The STS projects to the IPL with mirror neurons tracking precise kinaesthetic movements and (3) passes this information to the IFG coding for 'the goal of the action'. (4) The goal directed motor plans are sent from the IFG via the IPL back to the STS. (5) The MNS coupling initiates motor mimicry. The anterior insula AI (green) connects MNS with AMG and provides a possible neurological crossroad between these two independent, yet mutually interacting systems.

In nature, organisms survive and thrive by detecting unconditioned arousal signals. In the case of both olfactory and visual unconditioned signals (such as a partner's pupil size), the signals are generally processed implicitly, passing through the superior colliculus (CS)- pulvinar (Pulv) pathway to the amygdala (AMG; Tamietto and de Gelder, 2010). The amygdala is a brain region located in the deep layers of the limbic cortex and is mainly associated with detecting biologically relevant cues including emotions expressed by peoples' faces and bodies (Adolphs, 2001, Atkinson and Adolphs, 2005). This area is also used to direct the appropriate action following threat detection (Armony and Le Doux, 1997) and is an important regulator of stress-related glucocorticoids in response to physical or psychological stressors (Dedovic et al., 2009). When a receiver perceives a signal of a partner's increased arousal, the amygdala activates the locus coeruleus (LC) part of the noradrenergic system (Tamietto and de Gelder, 2010). The locus coeruleus has connections to the ventromedial hypothalamus, which in turn, outputs to motor control areas to promote adaptive behavioral responses to the event (Phillips and Le Doux, 1992).

Apart from the LC in the brain-stem, the AMG also projects to temporal and frontal regions including the orbitofrontal cortex (OFC) and the anterior cingulate cortex (ACC). The amygdala and the OFC share reciprocal connections with the superior temporal sulcus (STS) that underlies rapid and prioritized processing of affective signals (Decety, 2011). These areas are involved in emotional control and higher forms of empathy such as perspective-taking (Adolphs, 2001, Mutschler et al., 2013). These higher-order regions fully develop relatively late in development (Gogtay et al., 2004). In early development, subcortical circuits including the amygdala, hypothalamus, hippocampus, and OFC, are essential components of affective arousal. The NMEC (Fig. 3) shows that autonomic responses of the sender directly modulate neural activity in the emotion system of the receiver. In line with the AIM, we argue that this form of emotional contagion is fast, automatic, shared by most vertebrates, and does not require extensive training. For example, human infants possess an innate mechanism which automatically converts the sensory signals related to senders' autonomic states to their own corresponding autonomic states. The mimicry of autonomic responses (such as pupil size change, facial redness, cardiovascular responses and hormonal level) detected by the receiver results in emotion system coupling between the infant and its caregiver. Yet, how are the autonomic states of a sender mapped onto the receiver?

Kilner et al's. (2007) predictive coding framework of the mirror neuron system provides a promising account of its potential mechanisms. These predictive computations are not necessarily tied to one specific neural system but rather to a network of regions that also include the emotion system. Similar to PAM (Preston and de Waal, 2002), in the predictive coding framework, perception and action are tightly coupled (Barrett and Simmons, 2015). Through sensorimotor feedback, an organism's body receives essential information from its environment. For example, the receptors in the skin inform us about the angle of the surface we walk on, the temperature of the air and the taste of food. These highly specialized innate mechanisms transmit environmental stimuli to impulses in our brain, making us move and act adaptively in our environment. Similarly, the autonomic signals of an expresser, such as his or her pupil size, changes in facial redness, cardiovascular responses, and hormonal levels are implicitly (unconditionally) detected by the receiver. As information arrives via receptors of the body (visual, olfactory, auditory, tactile receptors, among others), predictions are sent through the cortex. Limbic cortices, with their simple laminar structure, issue predictions within every sensory system with a well-developed laminar structure (Chanes and Barrett, 2016). These predictions induce the discharge of neurons in regions anticipating the trajectory of an emotional reaction, while receiving actual sensory input from the environment. Hence, predictions function as hypotheses about the world that can be tested against sensory signals that arrive in the brain (Barrett and Simmons, 2015). A mismatch between sensory input and prediction is registered as a prediction error. The brain tries to minimize 'prediction error' by reducing such a mismatch. One way to do this is via mimicry. By generating a response to mimic the observed sensory input, the prediction error is sent back along cortical connections to update predictions about the situation. A newborn's brain has strongly developed limbic structures but underdeveloped neocortex. Because human behavior often fails to follow an anticipated pattern of action, and because infants lack prior experience, the model of the world is yet to be established. During this process of establishment, autonomic mimicry can be beneficial to reduce prediction errors and to establish emotion system coupling between the infant and its caregiver. For a detailed description of computations, see Kilner et al. (2007) and Chanes and Barrett (2016).

The fact that arousing stimuli and others' reactions toward arousing stimuli induce arousal in the observer has obvious evolutionary benefits. However, it is important to note that we cannot just assume that perceived autonomic states of the sender must only elicit corresponding autonomic states because of the adaptive value. Just because default responses are in place, it does not mean that they determine human actions. Instead of the brain being a 'stimulus–response' organ stimulated by a specific type of emotion (e.g. fear, happiness or anger), the brain functions as a generative system which constructs others' emotions as affective information accumulates over time. While visual information (i.e. pupil size, facial redness) gives a description of the visible affective components, it does not provide a full explanation sufficient for understanding the other's emotional state in all its complexity. A variety of autonomic input is essential to estimate the prior probability (from past experiences) to create the posterior probabilities that serve as a prediction about the action. Predictions guide our actions and perception by continually constructing possible representations relative to the present context. In other words, the organism has the opportunity to choose from the repertoire of actions based on past experience, yet does not require extensive training as innate “default processes” are already in place. This gives organisms greater flexibility and avoids the single input–output relationship criticized in mirror neuron theories (Hickok, 2009, Kilner et al., 2007).

Kleckner et al. (2017) argued that ascending sensory inputs from the organs, such as autonomic visceral and vascular function, neuroendocrine fluctuations are similarly anticipated by the brain to anticipate bodily needs before they even arise. In support of NMEC, researchers began to identify analogous introspective mechanisms for representing sensations from within the body (Kleckner et al., 2017). With the use of tract-tracing experiments in macaque monkeys, followed by fMRI studies in humans, researchers were able to map the intrinsic allostatic/interoceptive system supported by subcortical, hippocampal, brainstem, and cerebellar connectivity. In a follow-up fMRI experiment, subjects viewed arousing photos. The results showed that individuals with stronger functional connectivity within the allostatic/interoceptive system also reported greater arousal while viewing images and also demonstrated a greater sympathetic nervous system activity while viewing arousing images. This evidence suggests that these networks transfer emotional information across individuals and that connectivity of this network is essential for vicarious experiences (concordance between objective and subjective measures of bodily arousal). We propose that measures of autonomic mimicry, along with these system hubs, may provide an implicit index of interoceptive ability related to autonomic fluctuations.

In the first part of NMEC (Fig. 3), we have described how the intra- individual coupling between partners’ amygdala and HPA axis underpins autonomic mimicry. Yet, the synchrony of autonomic signals (heart rate, skin conductance, pupil diameter and hormonal expression) is not sufficient for emotional contagion to occur. This is because emotions have at least two fundamental dimensions: arousal (intensity) and valence (Russell, 1978). In a recent review, Wood et al. (2016) argued that in order to recognize facial expressions, humans must integrate several perceptual and contextual inputs at once. This is because modalities of perceptual input (auditory, visual, tactile and olfactory) are often incomplete, and the brain needs to generate predictions by integrating information from other modalities (Driver and Noesselt, 2008). For example, increased facial redness and sweating may be interpreted as either positive or negative, depending on additional visual input (e.g., facial expression). In this way, visual input from one sensory modality can affect the perception of another modality (Wood et al., 2016). While autonomic mimicry communicates intensity (the arousal level) of observed emotion, the motor movement of facial expression and gestures provides visual input that helps observers to label the increase in physiological arousal with the appropriate emotional valence. In other words, by pairing physiological synchrony with motor synchrony, emotional meaning can be transferred from one individual to another.

NMEC: the motor mimicry pathway (Fig. 4)

In addition to synchrony of autonomic arousal, a mechanism that plays a fundamental role in emotional contagion is the mirror neuron system (Gallese, 2005, Iacoboni, 2009, Likowski et al., 2012, Nummenmaa et al., 2008). The second part of the NMEC (see Fig. 4) depicts neurological pathways of the MNS through which motor signals can be registered. In humans, the MNS system is a neural network connecting several brain areas including the inferior parietal lobe (IPL), the inferior frontal gyrus (IFG) and the superior temporal sulcus (STS; Dinstein et al., 2007, Iacoboni, 2009). The regions of the MNS are assumed to contain 'mirror' neurons similar to those studied in analogous regions in macaque monkeys (Rizzolatti et al., 1996). In the macaque monkey, the mirror neurons in the ventral premotor area (F5) responded both when the monkey executed a specific movement and when the monkey observed another individual performing that same movement (Di Pellegrino et al., 1992, Rizzolatti et al., 1996). However, the MNS system is also activated when goal-directed hand movements are performed (Di et al., 1992). In addition to hand movements, the MNS is activated when people observe others enacting object or non-object related actions made with the mouth, hand or foot (Buccino et al., 2001, Grafton et al., 1996), or even when individuals only imagine that someone is performing a motor action (Grafton et al., 1996).

Movements in a partner's face are registered in the superior temporal sulcus (STS), a multisensory area which activates when observing biological motion (Iacoboni, 2009). From the STS, motor information is transferred to the inferior parietal lobe (IPL; BA 39,40) and then to the inferior frontal gyrus (IFG; Brodmann's Area 45/44/6); (Carr et al., 2003). The IFG region is an important region in social cognition, being associated with coding the 'goal of the action' (Gazzola et al., 2006). These goal-directed motor plans are then sent back to the IPL and the STS (Carr et al., 2003). Cattaneo et al. (2010) provided convincing neurobehavioral evidence for mirror neurons contribution to cognition by means of transcranial magnetic stimulation (TMS). In this experiment, blindfolded participants repeated an object-directed action (push or pull). When participants categorized others' actions, this resulted in a visual after-effect, as a result of motor-to-visual adaptation (of mirror neurons). TMS over the ventral premotor cortex suppressed the after-effect. These data are consistent with the existence of premotor mirror neurons associated with action meaning in humans.

In the past decades, the MNS has attracted scientific attention as it has been suggested that, in addition to motor imitation, the MNS also supports social functions (Gallese and Goldman, 1998, Keysers and Gazzola, 2010, Rizzolatti et al., 2009). Specifically, since the same neural networks are involved in motor production and observation, it has been theorized that the MNS may play a critical role in empathy as it allows for feedback from facial and bodily actions that simulation reflects on the emotions of others.

Anterior insula connecting the MNS and the emotion systems

A key neural structure believed to connect the mirror neuron system with the emotion system is the anterior insula (Carr et al., 2003). The anterior insula has been implicated in playing a role in the perception and experience of pain (Jackson et al., 2006a, Mutschler et al., 2013). Apart from vicarious physical pain, the AI is also associated with feelings of embarrassment or social pain (Krach et al., 2011). The anterior insula is structurally and functionally coupled to limbic structures including the amygdala

(Augustine, 1996). For this reason, the AI has been proposed to be the neural structure connecting the mirror neuron system with the emotion systems in empathy. Carr et al. (2003) showed in an fMRI study that activation of the AI correlated with activity in the premotor cortex, IFG, and AMG – all areas associated with emotional contagion, as demonstrated in numerous empathy reviews (Decety, 2010, Gazzola et al., 2006, Iacoboni, 2009). Interestingly, in several fMRI experiments (Jackson et al., 2006a, Jackson et al., 2006b), subjects were presented with people in a painful situation and instructed to imagine perceiving the pain from first and second person perspectives. The first perspective of pain led to increased pain ratings as well as increased activation in the somatosensory cortex, the ACC, and the insula. Taking the perspective of others increased activation in the precuneus and the right TPJ, areas involved in theory of mind and mentalizing. These results indicate that perceptions of pain processed in the insula, as well as in the ACC, represent self-centered experiences, while the TPJ and the precuneus play a role in self-other discrimination, which are crucial aspects of human empathy.

The aforementioned reviewed literature suggests that empathy is, in part, based on shared brain-to-brain coupling of affective states. While previous reviews have clarified that neural pathways are involved in the detection of subtle emotional signals in a partner's face and body (Hasson et al., 2012, Kret, 2015, Tamietto and de Gelder, 2010) and that other reviews have addressed the neural underpinnings of motor imitation (Ferrari et al., 2005, Iacoboni, 2009, Rizzolatti et al., 2001), we here propose a new model that incorporates these neurological accounts into one interactive emotional contagion model. In addition, NMEC accounts for how the sender's nonverbal facial characteristics (movements/autonomic responses) lead to brain-to-brain coupling and mimicry between the partner's emotion system (limbic system and HPA axis) and the mirror neuron system (IFG, IPL, STS). Both motor mimicry and the autonomic mimicry indicate a high level of neural coupling between these areas, where autonomic mimicry contributes to the intensity of communicative signals and motor mimicry frames the expression with the appropriate emotional valence. Consequently, by combining autonomic and motor signals, people can extract affective meaning from a partner's face. Disrupted emotion processing has been related to a range of mental disorders and can possibly explain the high comorbidity between mental disorders. Kret and Ploeger (2015) reported evidence for disrupted emotion processing in anxiety disorders, mood disorders, schizophrenia, autism spectrum disorder, borderline personality disorder, and eating disorders. Multiple measures of automatic autonomic mimicry, along with motor mimicry early after birth, could be used as developmental markers of social deficits. If true, pediatrics could intervene early to substantially reduce the adverse symptoms of these disorders.

Discussion

The current review provides an overreaching overview of studies spanning developmental psychology, social sociology, evolutionary biology and neuroscience, supporting the notion that automatic mimicry is essential for the development of empathy. The literature indicates that people are generally not consciously aware of subtle changes in an interaction partner's face and do not voluntarily react to these changes (Dimberg et al., 2000, Tamietto and de Gelder, 2010, Wood et al., 2016). Yet, infants, as well as adults, automatically mimic facial expressions as well as autonomic signals from their partner's face or body and by doing so enhance their understanding of the other's feelings, emotions, intentions, and actions. Automatic mimicry has here been proposed as a potential mechanism that allows humans to recognize and empathize with other's emotions (Buck, 1980). In theory, automatic mimicry of the perceived affective signals, in turn, simulates further neural systems involved in the corresponding emotional state, which helps observers to implicitly infer the expresser's internal state (Wood et al., 2016). This is an evolutionarily adaptive skill, allowing organisms to survive and thrive by detecting unconditioned signals of emotionality or arousal. Nevertheless, causal evidence for this hypothesis remains controversial. The above reviewed literature provides a unique and novel insight into the possible function and underlying mechanisms of mimicry.

Building upon Preston and de Waal’s (2002) perception-action model, we showed that strong synchronization exists between two people. We argued that automatic mimicry provides a physical-cognitive link during an organism's development and is a precursor of healthy social development. To support this argument, we demonstrated that emotional contagion can occur at different levels of processing. We reviewed well established, as well as recent, studies introducing several physiological mechanisms of automatic mimicry through which affective information can be shared. The presently reviewed literature implies that people mimic both autonomic and motor expressions (Dimberg et al., 2000, Niedenthal et al., 2001, Tia et al., 2011, Gregory and Webster, 1996, Webb, 1969, Helt et al., 2010, Goldin-Meadow and Alibali, 2013, Estow et al., 2007). Special attention has been given to autonomic mimicry, which is an underexplored area of current emotional contagion research. The "autonomic mimicry" involves synchrony in heart rate (Feldman, 2011), breathing rhythms (Creaven et al., 2014; van Puyvelde et al., 2015), pupil diameter (Fawcett et al., 2016, Kret et al., 2015, Kret and de Dreu, 2017) and hormonal levels (Laurent et al., 2012, Saxbe et al., 2014). Psychophysiological research indicates that strong autonomic mimicry exists between mothers and neonates and that this physiological association translates to psycho-emotional interactions between the pair. While autonomic mimicry is generally a positive marker promoting attachment, if the mother is in distress, physiological synchrony can actually have negative consequences on the child's social development. This supports the argument that physiological alignments allow for the direct transfer of affective information from one individual to another and thus facilitates implicit emotional communication.

Summarizing the newest discoveries in social neuroscience, we explained that mimicry is likely to be a result of overlapping neural networks. We proposed a new "Neurocognitive Model of Emotional Contagion". At its core, NMEC illustrates how complex processes, such as empathy, might emerge from automatic mimicry of conspecifics. We argued that while shared neural activation and automatic mimicry reflect the degree to which people internally simulate perceived emotional states, it is the emotional signals – not the mimicry – that drive the common patterns of neural representations that underlie empathy. We proposed that emotions are communicated via various communicative channels (Fig. 1, Fig. 2) and the degree of mimicry and brain-to-brain coupling (Fig. 3, Fig. 4) corresponds to the level the receiver is able to "tune in" to one or more of these communicative channels. Therefore, measurements of several types of mimicries at once would provide a more holistic physiological profile of the level to which one individual understands/processes another individual's social signal. Especially useful might be measures of autonomic signals that are not likely to be influenced by learning, social interactions, or conscious control (Kret, 2015). In line with this hypothesis, there is an increasing interest in how mimicry may underlie social deficits in social disorders (Duffy and Chartrand, 2015). Still, to what extent mimicry is necessary for healthy social development remains inconclusive. Considering the potential role of automatic mimicry in social pathologies, we propose that future research should measure the mimicry of emotional signals on different levels of expression.

Future directions

The topics of motor mimicry and especially autonomic mimicry are very new and as of yet, still underexplored. A valuable step in future studies could be to conduct longitudinal studies whereby automatic mimicry is continuously measured throughout a child's development (Feldman et al., 2014). While most previous studies measured automatic mimicry during virtual interactions, a study of mimicry during real-life interactions is highly recommended to provide real-life implications. Importantly, multiple measurements of emotional signals (e.g., facial muscles, eye gaze, pupil-size, blushing and body postures) are needed to measure several processes underpinning emotional contagion during social interactions. Apart from behavioral experiments, future studies may combine neurological techniques (EEG, fMRI) with physiological measures and try to block mimicry in order to shed light on how different forms of mimicries are represented in the brain. Neuroimaging analyses should be hypothesis driven and make use of functional connectivity analysis and dynamic neural network modeling in order to make sense of social cognition as an interactive system.

An alternative way to tackle the correspondence problem of mimicry is to study mimicry in robots. The possibility that empathy can be generated by sensorimotor processes in robots has already excited the cognitive science community. This is because artificial systems provide the benefit of a blank state in which neuroscientific theories of brain functions can be tested (de Kleijn et al., 2015). With robots, researchers can carefully manipulate parameters in a controlled way. There is evidence showing that if robots mimic another person's affective actions, this automatically activates a motor representation and affective experience in the human that is coherent with the robot's affective expression (Hofree et al., 2015, Li and

Chignell, 2011, Mayer et al., 2010). Similarly, as in human-to-human interactions, research shows that during human-robot interactions, humans perceive robots that mimic as more positive and empathic (Fuente et al., 2015, Hofree et al., 2015). Hypothetically, if automatic mimicry would allow for affective representations to be manifested in a robot, it would confirm the theory that mimicry is sufficient for emotional contagion to emerge (Asada, 2015). By developing robots that can detect subtle social signals and exhibit mimicry, we could then test whether robots start to develop social behavior similar to humans. In the future, brain-inspired algorithms and computational models of neural networks (e.g., simulations of neural microcircuits, connectionist networks) could provide well controlled mimicry parameters for social cognitive models (Asada et al., 2001, Watanabe et al., 2007). The application of the NMEC in robots in future studies may provide evidence to refute or support the hypothesis that automatic mimicry is necessary for empathy development.

Conclusion

In conclusion, the current review argues that automatic mimicry is essential for the development of healthy social cognition. The current review provided an interdisciplinary approach to the study of human cognitive developmental. Through the integration of information from social neuroscience and evolutionary biology, this review provided new insights into the development of human cognitive functions.