Affiliative temperament: Concept, neurobiology and implications for antisocial behaviors

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ABSTRACT

Human affiliative behaviors are indispensable for the physical and psychological wellbeing and normal development of individuals. However, how to study the neurobiology of affiliation from temperamental domain is little examined. Based on theoretical arguments about affiliative behaviors in temperamental domain, this review clarifies the concept of affiliative temperament, and then provides a review of the neurobiology of affiliative temperament by the integration of the mirror neuron system (MNS), the dopaminergic reward system, and the neuropeptides. Reviews show that affiliative temperament is firstly a complex and as a part of it, emotional contagion roots in the MNS from the birth. Secondly, the dopaminergic system relating to affiliative temperament can be divided into the regulative system including dorsolateral prefrontal cortex (DLPFC), orbitofrontal cortex (OFC), anterior cingulated cortex (ACC), and the evaluative system including superior temporal sulcus (STS), ventral tegmental area (VTA), nucleus accumbens (NAcc), insula, and amygdala. Thirdly, oxytocin (OT) and vasopressin (VAP) modulate neural circuits in VTA and NAcc. Finally, structural and functional abnormalities in affiliation-related brain circuits contribute to the pathogenesis of psychopathological disorders as well as antisocial behaviors.

Introduction

Humans are inherently social, and one can develop social bonds with other people around himself in order to adapt to the complex social environment (Young, 2008). Although humans share with animals many of the same types of social behavior such as affiliation and aggression, the establishment of hierarchy and territoriality, human affiliative behaviors are essential for the physical and psychological wellbeing and normal development of individuals, and impairments in these behaviors are clearly associated with maladaptive interpersonal patterns and psychiatric disorders (Bora, Yucel & Allen, 2009).

Affiliation research dramatically increased throughout the 1990s, with a broadened emphasis on a variety of attachments including the mother-infant bond, pair bonding, and colony membership (Hammock & Young, 2007). Today, researchers in this area seek to understand the integration of hormonal states and neural circuits in regulating complex behaviors. Along with this idea, this paper will firstly specify the concept of affiliative temperament in theory. The second section will review the neurobiology of affiliative temperament with the integration of the MNS, the dopaminergic system and the neuropeptides. The third section will discuss implications of affiliative temperament for antisocial behaviors. Finally, the future directions will be mentioned.

Affiliative temperament as an individual construct

Affiliation behaviors are very intricate and comprise phenomena at multiple levels of analysis including physiological and psychological systems, whole individuals, short-term interactions between individuals, longer-term relationships, and

groups (Weinstein & Capitanio, 2005). So it is a challenge undertaking to study their neurobiological bases. Recently, Bora et al. (2009) have reviewed the neurobiology of human affiliation behaviors mainly about human brain imaging and neuropeptide researches, which is helpful for us to understand human psychiatric disorders from neurobiology. However, we know that the brain is functional. Behaviors are not different, but the neural mechanism underlying these behaviors may be similar. That is, one neural mechanism may govern many similar behaviors. Therefore, it is unreasonable to study the neural mechanism of each behavior and almost impossible for people to study a myriad of human affiliation behaviors one by one considering researchers' limited time and funds.

One of scientific methods is to introduce some constructs. They can gather up behaviors of the similar neurobiological basis together. Methodologically, people sample typical behaviors from a construct to investigate, and then infer the neurobiology of more other similar behaviors belonging to the same construct, which makes it easier to research the relationship between brain and behaviors. For example, based on animal studies, Depue et al. (2005) have brought forward a neurobiological model about affiliative bonds from a personality point of view. However, the default of personality research is not to explain affiliation early in life, because the newborn's personality is not innate, but gradually shapes as time goes by. In addition, personality is subject to a great deal of experiential influences, and involves abilities, standards and values, self-concepts, and defense mechanism. In contrast, temperament emerges at birth and does not include those compounds. In what

follow we firstly review theoretical arguments about affiliative behaviors in temperamental domain, then give a working definition of affiliative temperament, and finally differentiate affiliativeness with other temperament components such as approach-withdrawal and effortful control (EC) (sometimes called constraint; Nigg, 2006) so that we can study the neurobiology of affiliation from a new perspective.

Theoretical arguments about affiliative temperament

In modern temperament research, social behaviors have been highly concerned all the while, which mainly embody in Buss and Plomin's Behavior-Genetic Theory of Temperament and Rothbart's Developmental Model of Temperament. Framing temperament as a developmental precursor to adult personality, Buss and Plomin (1975) defined temperament as a set of inherited personality traits that appeared early in life, preferably infancy (the first two years of life). They identified four temperamental dimensions: emotionality, activity, sociability and impulsivity, and developed two questionnaire measures, the Colorado Child Temperament Inventory (Rowe & Plomin, 1977) and the EAS Survey for Children (Buss & Plomin, 1984). To be specific, sociability is the tendency to prefer the presence of others to being alone (Buss & Plomin, 1984, p.63). This tendency has its roots in intrinsic rewards which result from social interaction with other persons. Individuals high on sociability are more reinforced by social rewards and more upset by their deprivation. As expressed in social interaction, sociability is developmentally specific. In infancy it plays a role in mother-infant interactions while it can be considered one of the two crucial components of extraversion in adult. Unfortunately, there is little neurobiological research using the two scales.

Comparatively, Rothbart and her colleagues emphasized on attentional and neurobiological mechanisms. Specifically, they defined temperament as constitutional differences in reactivity and self-regulation, in the domains of affect, activity, and attention (Rothbart & Bates, 2006). They developed a series of parent-report questionnaires, including the Infant behavior Questionnaire-Revised (ages 3-12 months) (IBQ-R; Rothbart, 1981), the Early Children Questionnaire (18-30 months) (ECBQ), and the Children's Behavior Questionnaire (CBQ; Rothbart, Ahadi, & Hershey, 1994). In addition, they also developed self-report questionnaires, the Early Adolescence Temperament Questionnaire-Revised (EATQ-R; Muris & Meesters, 2008; Ellis & Rothbart, 2001) and the Adult Temperament Questionnaire (ATQ; Evans & Rothbart, 2007). Affiliation/Cuddliness emerges in IBQ, ECBQ and EATQ-R, which is defined as desire for, pleasure in, warmth and closeness with others. ATQ includes affiliative subscale assessing emotional empathy (affective responses congruent with the feelings of others), empathic guilt (distress in response to negatively affecting other people), and social closeness (feelings of warmth, closeness, interest and involvement with others) (Evans & Rothbart, 2007). Developmental changes in temperament characteristics go along with the maturation of the nervous system, mainly stressing the executive attention systems (Posner, Rothbart & Sheese, 2007). Affiliative temperament emerges like cuddliness, soothability as well as orienting in infancy, but by toddlerhood and early to middle childhood, it is anchored by attentional control and labeled as EC. In adolescence, affiliativeness separates from EC again (Nigg, 2006).

From the above, we can see that affiliative temperament exist a few differences in two theories. These may be attributed to lack of clear boundary of temperamental concept and their different understandings of temperament. Out of question, their researches provide useful references for our following concept of affiliative temperament.

The concept of affiliative temperament

Considering ambiguous boundary of temperamental concept and its complicate expressions, how to define affiliative temperament is still a debating issue. Here we bring forward a transient working definition. We conceptualize affiliative temperament as a relatively stable prosocial tendency to wanting to be close to others, form and sustain close contacts with others (Hill & Werner, 2006; Lahey, 2004; Nigg, 2006).

First, affiliative temperament is composed of behavioral tendencies rather than specific instances of behaviors. Thus affiliation disposition is not the same as preferences, bonds and relationships which imply the involvement of a second person (Weinstein & Capitanio, 2005). Sociability refers to a much broader field including affiliative attachment to such things as ethnic group, sports teams, and religious values than affiliativeness. Mating behavior and maternal care are also not the same as affiliation, because these are mainly subject to hormones. For example, once pups of most rodent species are fully weaned, the relationship between mothers and pups ends abruptly (Weinstein & Capitanio, 2005).

Second, affiliative temperament can be only inferred from concrete behaviors. There are many affiliative behaviors to be selected for inference, but here we only select attachment, empathy or dispositional sympathy (Elizabeth, et al., 2009; Evans & Rothbart, 2007), cooperation (Bora et al., 2008), friendship (Güroğlu et al., 2008), love (Ortigue et al., 2007) and interpersonal trust (Zak, 2005). Because as the years pass, each individual has more chances to seek out others, and affiliation tends to be expressed in different ways. From a developmental point of view, we regard these behaviors as different manifestations of affiliative temperament at different stages of development. To be specific, in infancy the quality of parent-infant attachment decides later trust in others. Later on, cooperation plays become more and more important in childhood while friendship among adolescents stands out. During young adulthood, love becomes the principal form of affiliation. Especially, affiliative tendency implicitly or explicitly needs to recognize others' intention or mental states, so we regard empathy as a necessary component in the process of affiliative temperament. Although theory of mind relating to empathy matures after about three years, emotional contagion as a predecessor to empathy appears at birth. The MNS constitutes the biological basis of emotional contagion and makes great contribution to affiliation from the birth, so we consider the MNS as the inborn biological basis of affiliation.

Finally, expressions of affiliative disposition vary across different context and over time, only with modest stability. For instance, there is evidence showing that agreements between mother and father ratings of their child's temperament are consistently higher than agreements between mother and teacher (Goldsmith, 1996), from which we can infer that children's temperament is expressed in somewhat different ways at home versus school. Now we turn our steps to the relationship between affiliation and other temperamental components.

Differences from approach-withdraw and EC

Nigg (2006) made a detailed review about the issue. He considered affiliation as a subcomponent of approach. We admit that approach-withdrawal reflects temperament reactivity and touches upon broader stimulus range more than affiliation. However, as accepted widely, approach (sometimes called positive affect (PA)) is associated with extraversion while withdrawal is associated with negative affect (NA). In ATQ, Evans & Rothbart (2007) put extraversion corresponding with approach and NA to parallel with affiliation, showing that affiliation is a temperamental component different from approach and NA. Here we specify that affiliativeness is in response to socially affiliative stimuli while approach-withdrawal is associated with non-social stimuli in this paper.

From the above, we have known that affiliativeness interweaves EC during the neonatal period, and then separates from EC again in adolescence. What's more important, we note that EC and affiliativeness have different neural bases. EC depends on anterior neural systems relating to executive function, and shows a role for top-down prefrontal modulation of subcortical regions while affiliativeness mainly relies on the mesolimbic system (see the following).

The neurobiology of affiliative temperament

In this section we discuss the neurobiology of affiliative temperament, including the MNS, the dopaminergic system and the neuropeptides (OT and VAP). The MNS comprises the innate neurobiological basis of empathic contagion and imitation which are necessary for affiliativeness, and can explain affiliation behaviors early in life. Overlying with the MNS within DLPFC and STS, the dopaminergic system originating from VTA is dominant for the formation and maintenance of affiliativeness. The neuropeptides interact with the dopaminergic system in mesolimbic circuits (originates in the VTA; innervates the limbic cortices, septo-hippocampal complex, NAcc, and amygdala), and modulate affiliative responses by affecting the hypothalamic-pituitary-adrenal (HPA) axis activity. We separately introduce them in a brief way as follows.

The MNS and emotional contagion

Rizzolatti and his colleagues (1996) discovered a particular class of motor neurons, entitled mirror neurons which constitute a link between what the monkey sees other people doing and what the monkey does himself, in a sector of the ventral premotor cortex of monkeys, called F5 (BA44 and BA6 in human). Further, research has also shown that mirror neurons are related not only to imitation, but also to the recognition of the intentions of others (Rizzolatti & Craighero, 2004). Mirror neurons have also been found in monkey's STS (or the middle temporal gyrus in human) and Area 7b (the rostral inferior parietal lobule in human), which constitute shared circuits together with F5. These shared circuits have already been confirmed by human fMRI (see Iacoboni & Dapretto, 2006 for review). As the biology basis of emotion contagion (Agnew, Bhakoo & Puri, 2007), these circuits can translate the vision and sound of what other people do and feel into the observers' own actions and feelings (Keysers & Gazzola, 2006). And the MNS is fundamentally linked with emotion-related circuitry. Individual differences in the activity of the MNS are correlated with behavioral indices of children's empathic behaviors and interpersonal skill (Pfeifer et al., 2008).

Emotional contagion is defined as the tendency to automatically mimic and synchronize facial expressions, vocalizations, postures and movements with those of another person (Chakrabarti & Baron-Cohen, 2006). Imaging studies have demonstrated involuntary facial mimicry as well as activity in regions of the brain where the existence of mirror neurons has been suggested (Jackson et al., 2005; Nummenmaa et al., 2008). Watching another person being touched also activates a similar neural circuit to actual touch, and for some people with mirror-touch synesthesia, doing this can produce a felt tactile sensation on their own body (Banissy & Ward, 2007). This is consistent with the notion that people empathize with others through a process of simulation.

Emotional contagion is also well-known to develop in the first year, and becomes an important aspect of affiliative temperament. Its mirroring effect allows infants to distinguish emotional and intentional states of significant others, which become important contextual stimuli predictive of reward in the pursuit of their own drive satisfaction, including satisfaction of the affiliative drive. For example, human infants are born with a bias to preferentially track and fixate stimuli with face-like configurations, and show reactions to crying or distress that might be considered empathy, or at least some precursor to empathy such as emotional contagion. When the baby is around the age of 2 months, mother and infant attune to each other's affective expressions; that is, they synchronize their affective behaviors (Feldman, Greenbaum & Yirmiya, 1999). Thus, an inborn tendency to imitate may allow the infant to re-experience emotional states of conspecifics and learn about the association of these states with the delivery of affiliative and other rewards, which helps affiliative temperament gradually outspread under the influence of affiliative stimuli. About 14-18 months, as children more clearly differentiate selves from others, they show more variations and more other-directed empathic and sympathetic responses to others' distress (Vaish et al., 2009).

In summary, the MNS provides the innate equipment for emotional contagion, and help infant synchronize expressions of caretaker. However, if infant wants to satisfy his/her affiliative motivation, get reward experience, and suppress incompatible aversion, the MNS needs to interact with the dopaminergic reward system.

The dopaminergic system

Originating from VTA, the dopaminergic system covers multiple pathways. Here we focus on the mesocortical pathway which can be divided into two parts (see fig. 1). The first part including DLPFC, OFC and ACC exert regulative functions and correspond with EC (Whittle et al., 2006). Because affiliation behaviors can not arise individually without the regulation of EC, we fit this part into the neurobiology of affiliativeness. The second part including STS, VTA, NAcc, insula and amygdala form the evaluative system of affiliativeness. We discuss their functions one by one in the following.

The neurobiology of affiliativeness depends on the integration of the MNS, the dopaminergic system divided into the regulative system (DLPFC, OFC, and ACC) and the evaluative system (VTA, NAcc, insula, and amygdala), and the neuropeptides (OT and VAP) at different levels. First, the MNS interacts with the dopaminergic system within DLPFC at the highest level. DLPFC takes executive control for imitation and emotional contagion, together with the interaction with OFC and ACC. OFC is involved in dynamic reassessment and relearning of affiliative associations, dependent on individual's motivational states. ACC may be to monitor motivational conflict (especially in the aversive context) and select appropriate response with respect to emotional priorities and

goals. Second, STS integrates multiple stimuli's values and evaluate others' intention. The VTA, NAcc, insula and amygdala separately process affiliative stimuli and evaluate their values. Then they provide afferent information for higher-order cortex upwards while they activate the response system downwards. Third, on the lower level, OT interacts with dopaminergic circuits in the NAcc and VTA. Arginine vasopressin also interacts with similar circuits in the NAcc. OT and VAP modulate activity in dopaminergic reward pathways associated with socially affiliative behaviors, directly influence the HPA axis activity, and determine behavioral tendency of affiliative temperament and the strength of subjective experience.

DLPFC: dorsolateral prefrontal cortex; OFC: orbitofrontal cortex; ACC: anterior cingulated cortex; STS: superior temporal sulcus; VTA: ventral tegmental area; NAcc: nucleus accumbens; OT: oxytocin; VAP: vasopressin; HPA: hypothalamic-pituitary-adrenal.

Fig. 1. The hypothesized neurobiological model of affiliative temperament
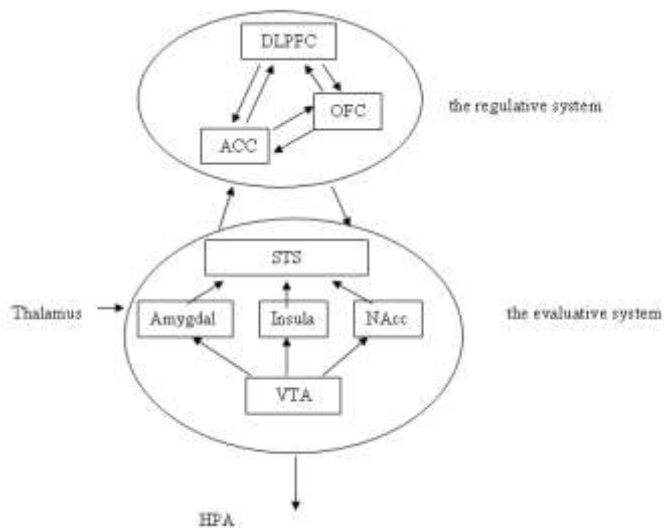
The regulative system
DLPFC

DLPFC comprises a part of the MNS (see the above). And an abundance of neuroimaging researches have showed that DLPFC plays a key role in EC, emotional processing as well as working memory (Geier, Garver & Luna, 2007). Thus it is very necessary to be associated with affiliation behaviors. For example, Güroğlu et al. (2008) found that compared to celebrities, peers evoked a significantly higher level of activation in DLPFC. In another fMRI study, mothers showed the specific pattern of response to their own infant's attachment behavior, with greater activation in DLPFC by viewing their infant in the separation situation than the play situation (Noriuchi et al., 2008). DLPFC was also activated in response to subliminal presentation of the name of a beloved person, but not to the name of friends or passionate interests (Ortigue et al., 2007; Aron et al., 2005).

However, there are some evidences showing that the left and right DLPFC have distinct functions. For example, in contrast to healthy subjects, left DLPFC activity during emotional judgment was not parametrically modulated by negative emotional valence and was inversely modulated by positive emotional valence in major depressive disorder patients (Grimm et al., 2008). On the other hand, Sollberger et al. (2009) found that interpersonal traits high in agency related to left

DLPFC, whereas interpersonal traits high in affiliation related to right DLPFC. These findings seem not to be explained by the valence-lateralization theory which postulates that the left hemisphere is dominant for positive emotions and the right hemisphere is dominant for negative emotions (Wager et al., 2003). In line with this theory, Whittle et al. (2006) proposed that left DLPFC was associated with PA whereas right DLPFC was associated with NA.

As aforementioned, we think that PA is a temperamental component different from affiliativeness. Affiliativeness involves a complex set of processes including sensory detection of conspecifics, recognition of familiar individuals, and motivation to initiate proximity or contact (Hammock & Young, 2007). In addition, incompatible processes such as aversion or aggression must be suppressed. Affiliativeness does not correspond with PA completely because people do not always benefit from affiliation behaviors, but pay social costs when facing motivational dilemmas.

In summary, DLPFC is certainly associated with affiliativeness, but whether the left and right of DLPFC exert different functions in the process of affiliative temperament is not clear and needs more researches in future.

OFC

The OFC is involved in multi-modal sensory integration, representing the affective value of reinforcements, decision-making and expectation. In particular, the human OFC is thought to regulate planning behavior associated with sensitivity to reward and punishment (Kringelbach, 2005). Cloutier et al. (2008) conducted an event-related fMRI experiment during which participants provided explicit attractiveness judgments for faces of the opposite sex. The results revealed that the OFC showed a linear increase in activation with increased judgments of attractiveness.

Activation of the OFC has also been reported in response to viewing diverse affiliation-related stimuli such as one's own child (Noriuchi et al., 2008), an intensely loved person (Ortigue et al., 2007) or friends (Güroğlu et al., 2008). Damage to the caudal OFC in the monkey produces emotional changes (e.g., decreased aggression to humans and stimuli such as a snake and a doll). In humans, irresponsibility, lack of affect, and impulsiveness can follow OFC damage (Rolls & Grabenhorst, 2008).

ACC

Based on attributed functions, the ACC can be divided anatomically into cognitive (dorsal), and emotional (ventral) components. A ventral affective subdivision of ACC (BA25, 32, 33) has connections to amygdala, NAcc, OFC, anterior insula, and autonomic brain stem regions. A dorsal cognitive subdivision (BA24, 32 and cingulate motor area) has anatomical connections with parietal cortex, posterior cingulate, supplementary motor area, and DLPFC (Devisky, Morrell & Vogt, 1995).

The ACC is connected with the prefrontal cortex and parietal cortex as well as the motor system and the frontal eye fields (Posner & DiGirolamo, 1998), making it a central station for processing top-down and bottom-up stimuli and assigning appropriate control to other areas in the brain. The ACC seems to be especially involved when effort is needed to carry out a task such as early learning and problem solving. Many studies attribute functions such as error detection, anticipation of tasks, motivation, and modulation of emotional responses to the ACC (Whittle et al., 2006). In contrast, ventral ACC may be to

monitor and evaluate external stimuli (especially when aversive or painful) and select appropriate responses with respect to ongoing emotional priorities and goals (Davidson et al., 2002).

There is evidence showing that the ACC is related to individual differences in temperament. For example, Whittle et al. (2008) found that higher affiliativeness was associated with larger volume of the bilateral rostral/ventral ACC across males and females. Moreover, the ACC is also involved in affiliation behaviors. For example, the paracingulate cortex (BA32) was involved in building a trust relationship by inferring another person's intentions to predict subsequent behaviors (Krueger et al., 2007). By combining the intranasal, double-blind, administration of OT with fMRI, Baumgartner et al. (2008) found that the placebo group demonstrated increased activation in the dorsal part of the ACC than the experimental group during prefeedback trust game periods.

Cingulate pathology may be associated with emotional and social behavioral disturbances. Bilateral cingulate lesions made in three macaques were associated with decreases in social interactions, time spent in proximity with other individuals, and vocalizations but an increase in manipulation of an inanimate object (Hadland et al., 2003). In human, impairment of ACC was associated with autism spectrum disorders only in social tasks (Martino et al., 2009).

The evaluative system
STS

The STS belongs to the MNS and is a key component of the social brain. It receives multiple inputs from third-order sensory association areas and other polymodal areas (parietal, prefrontal cortex, limbic, and paralimbic regions), suggesting that multimodal STS areas are involved in the highest level of cortical integration of both sensory and limbic information (Boddaert et al., 2004).

Recent researches confirmed the role of the STS in affiliation. For example, Ida Gobbini et al. (2004) measured neural activity using fMRI when subjects viewed faces of personally familiar individuals (i.e. friends and family), faces of famous individuals, and faces of strangers. Result showed that personally familiar faces evoked a stronger response than did famous familiar faces and unfamiliar faces in STS. Neuroimaging studies in normal subjects and single-cell recordings in monkeys have emphasized the role of STS in the MNS (Rajmohan & Mohandas, 2007), empathy (Hein & Singer, 2008), and theory of mind (Otsuka et al., 2009). The STS also plays an important role in affiliation behaviors such as attachment (Noriuchi et al., 2008) and friendship (Güroğlu et al., 2008).

The STS involves processing of dynamic facial attributes which convey information relevant for social interactions such as an individual's emotional state or direction of eye-gaze. The role of the STS in facial attractiveness judgments may be consistent with the "intention- detection" of the observer, the process of assessing the attractiveness of a conspecific as the social evaluation of another's intentions (Iaria et al., 2008).

VTA

The VTA is populated with dopaminergic neurons. The two primary efferent fiber projections of the VTA are the mesocortical and mesolimbic pathways. Almost all areas receiving projections from the VTA project back to it. However, the lateral VTA largely projects to the NAcc core, but not vice versa (Pierce & Kumaresan, 2006). Animal research demonstrates that the positive incentive motivation and experience of reward that underlies a behavioral system of approach is dependent on the functional properties of the VTA (see Depue & Morrone-Strupinsky, 2005 for review).

In human, Vrtička et al. (2008) examined how the three classic prototypes of attachment style (secure, avoidant, and anxious) modulated brain responses to facial expressions conveying either positive or negative feedback about task performance (either supportive or hostile) in a social game context. Activation of VTA was enhanced to positive feedback signaled by a smiling face, but this was reduced in participants with avoidant attachment, indicating relative impassiveness to social reward. In another human study, Conditional trust selectively activated the VTA (Krueger et al., 2007). Experimental studies have also shown that conditioned fear, anxiety and other stressors elicited an activation of VTA-derived dopaminergic pathways to the amygdala and adjacent bed nucleus of the stria terminals, and to the NAcc (see Millan, 2003 for review).

NAcc

The NAcc processes afferent input from many of the cognitive and limbic areas of the brain, including the prefrontal cortex, hippocampus, amygdala, and thalamus. The output neurons of the NAcc send axon projections to the ventral analog of the globus pallidus, which, in turn, projects to the dorsomedial nucleus of the dorsal thalamus. Other efferents from the NAcc include connections with the substantia nigra and pontine reticular formation (Schwienbacher et al., 2004). The release of dopamine within the NAcc is thought to be rewarding and thus could mediate a positive association with attachment (Noriuchi et al., 2008), affiliation trait (Depue & Morrone-Strupinsky, 2005), partner preference formation (Aragona & Wang, 2007), and friendship (Güroğlu et al., 2008).

Insula

The insular cortex is a multimodal sensory region with visceral, gustatory, somatosensory, visual, and auditory afferents and reciprocal connections to amygdala, hypothalamus, cingulated gyrus, and OFC. In addition to its role in interoceptive representation and autonomic control, the insula has also been implicated in the acquisition of inhibitory avoidance behavior (Small, Zald & Jones-Gotman et al., 1999).

The insula not only has a central role in our own disgust experience but also in our empathic response to seeing another person pull a disgusted facial expression (Jabbi, Swart & Keysers, 2007). Several researches showed that insula activation was associated with feeling states during the experience, imitation, or imagination of pain. For example, Singer et al. (2004) found that the insula activated when subjects received pain or when they observed their loved ones experiencing pain. The insula also activated regardless of whether participants perceived or assessed painful stimuli in others (Jackson et al, 2005), and of whether the participants imagined themselves or another person in painful situations (Jackson et al., 2006).

Amygdala

The amygdala is a heterogeneous structure that, in primates, consists of at least 13 anatomically and functionally distinct subnuclei (Amaral et al, 1992; LeDoux, 2000). Besides the complex internal structure, the amygdala has extensive external anantomical connections (Amaral et al, 1992), which allow the amygdala to integrate sensory input from all modalities and affect autonomic and motor output systems. Amygdala has efferent projections to wide regions of the brain, including hypothalamus, basal forebrain, entorhinal cortex, ventral

striatum, cingulated gyrus, orbitofrontal cortex, and brain stem autonomic centers (Amaral et al, 1992). The amygdala seems to be a site of primary appraisal: the automatic evaluation of events in relation to goal and concerns. In particular it seems to be responsible for assigning emotional significance to events that signal dangers and threats, and possibly to emotionally significant events of other kinds (Oatley et al., 2006).

As reviewed above, incompatible processes such as aversion or aggression must be suppressed in the process of affiliation. So it is not surprising that the amygdala involves in affiliative behaviors. For example, a research about rhesus monkeys showed that amygdala lesions yielded several personality changes that precluded positive social interactions (increased exploration and excitability, decreased affiliation and popularity) and altered responses to threatening social signals (Machado & Bachevalier, 2006). In a human fMRI, Güroğlu et al. (2008) assessed neural activity in a social interaction simulation task implementing the factors "type of relationship" (peers vs. familiar celebrities) and "emotional valence" (positive (liked), negative (disliked), and neutral (neither liked nor disliked)). Results revealed that among others, three regions of particular interest as selectively more strongly activated when subjects interacted with their friends than with other peers and celebrities: the amygdala and hippocampus, the NAcc, and the ventro-medial prefrontal cortex.

Neuropeptides

The neuropeptide system gains abundant researches in different animals, which build many animal models to help understand human behaviors. Here we only focus on the two peptide hormones of the posterior pituitary gland, OT and VAP. Their functions can be divided into two parts. The one part interacts with the limbic system by the mesolimbic pathway, and influences the formation and maintenance of affiliativeness. The other influences the HPA axis activity to modulate affiliative behavioral responses (see Carter, 2003 for review). Here we mainly discuss the first functions of OT and VAP.

OT

OT is synthesized in magnocellular neurons in the paraventricular and supraoptic nuclei of the hypothalamus (PVN and SON, respectively), which project to the posterior pituitary, and release the peptide into the peripheral circulation. OT is also produced within the parvocellular neurons of the PVN, which project to limbic sites such as the hippocampus, amygdala, striatum, hypothalamus, NAcc, and to mid- and hindbrain nuclei such as the locus coeruleus and nucleus of the tractus solitarius, as well as the spinal cord (Sofroniew, 1983). OT released within the brain itself is thought to regulate behaviors by acting as a neurotransmitter/neuromodulator.

OT is crucial for the development of attachment behavior in animal models (Brown et al., 2009). In human, OT has also been shown to be associated with adult attachment (Tops et al., 2007; Gordon et al., 2008), maternal-foetal (Levine et al., 2007), and maternal–infant attachments (Feldman et al., 2007).

OT is also associated with trust. For example, using the sequential anonymous "trust game" with monetary payoffs, Zak et al. (2005) found that OT levels were higher in subjects who received a monetary transfer that reflected an intention of trust, compared to an unintentional monetary transfer of the same amount. In addition, higher OT levels were associated with trustworthy behavior (Kosfeld et al, 2005), specifically and powerfully affected generosity (Zak et al., 2007), and increased ratings of trustworthiness and attractiveness of male and female targets in raters of both sexes relative to control ratings. This suggests that higher levels of OT may enhance affiliative tendency towards unfamiliar others (Theodoridou et al., 2009).

Recent studies show that OT also influences social cognitive processes. OT enhances encoding (Guastella et al., 2008b), conceptual recognition of positive social stimuli over social threat stimuli (Unkelbach et al., 2008), and the gaze time to the eye region when viewing faces (Guastella et al., 2008a). It also improves the ability to infer the mental state of others from social cues of the eye region (Domes et al., 2007), and increases the processing of positive emotions while it decreases the processing of negative emotions (Simplicio et al., 2008). But OT may not influence the detection of positive and threatening social stimuli at early perceptual levels of processing (Guastella et al., 2009).

Moreover, OT modulates the expression of evaluative conditioning for socially relevant faces, which is an effect that may explain its prosocial effects (Petrovic et al., 2008). With increased OT levels, Rimmele et al. (2009) found that previously presented faces were more correctly assessed as "known", whereas the ability of recollecting faces was unchanged. This pattern speaks for an immediate and selective effect of the peptide strengthening neuronal systems of social memory.

From the above, we can see that OT is involved in multiple behaviors relating to affiliativeness, and influences social cognitive process. OT appears to be a facilitator for the formation and maintenance of affiliative temperament.

VAP

Like OT, VAP is synthesized in magnocellular PVN and SON neurons and released from the posterior pituitary into the peripheral circulation. VAP is also synthesized within parvocellular neurons in the PVN and supra-chiasmatic nucleus as well as in extra-hypothalamic neurons in the bed nucleus of the stria terminalis and medial amygdala (DeVries & Panzica, 2006). These extra-hypothalamic sources of VAP are androgen dependent and are the likely source of sexually dimorphic projections within the brain. VAP regulates several male-typical social behaviors, including scent marking, courtship, aggression, and paternal care in vertebrates (Lim &Young, 2006).

Few human studies on arginine vasopressin (AVP) suggest that behavioral effects are similar to those found in animal research. Thompson et al. (2004) conducted an experiment using a placebo controlled, double-blind design. The results showed that AVP did not affect attention toward, or autonomic arousal in response to emotionally neutral, happy or angry facial expressions, but it selectively enhanced the corrugator's EMG responses. Thus AVP might influence aggression in human males by biasing individuals to respond to emotionally ambiguous social stimuli as if they were threatening or aggressive. Further, another study indicated that in men, AVP stimulated agonistic facial motor patterns in response to the faces of unfamiliar men and decreased perceptions of the friendliness of those faces (Thompson et al., 2006). In contrast, in women, AVP stimulated affiliative facial motor patterns in response to the faces of unfamiliar women, and increased perceptions of the friendliness of those faces. AVP also affected autonomic responsiveness to threatening faces, and increased anxiety, which may underlie both communication patterns by promoting different social strategies in stressful contexts in men and women.

Thus effects of AVP appear to be sex-specific, but this is just tentative. We can not make a clear conclusion because there

is a lack of enough human literature at present. Especially, it is also necessary to make more researches to clarify how OT interacts with AVP to affect affiliative temperament.

Implications for antisocial behaviors

Antisocial behaviors are characterized by apparent lack of remorse or guilt, inability to make or keep friends, tendency to violate the boundaries and "rights" of others, aggression, callousness and lack of empathy (Blair, 2006). Antisocial behaviors are risk factors of many crimes including substance abusing, stealing, and violence. Persistent antisocial behavior may be a manifestation of the antisocial personality disorder (Berger, 2003). However, the neurobiology of antisocial behaviors is still unclear.

This paper proposes that the neurobiology of affiliative temperament may provide a window into the etiology of a disorder or mechanism behind a developmental phenomenon. Individual differences in multiple aspects of the MNS, the dopaminergic system and the neuropeptides lay a biological foundation of affiliative temperament. Impairment in trait affiliation is a core feature of psychopathology as well as antisocial behaviors. In the following, the role of the neurobiology of affiliative temperament in antisocial behaviors will be explored from the above facets.

First, antisocial behaviors are germane to impairment in the MNS (Decety & Moriguchi, 2007), which contributes to a reduced ability to feel other people's emotional state, although they have intact cognitive empathy capacity (Blair, 2006). Moreover, there are evidences showing that the neural circuit underlying moral feeling and decision making is impaired in antisocial populations, and that dysfunction in the OFC may be associated with poor inhibitory control, emotional decision-making and reward/punishment processing in antisocial individuals (Raine & Yang, 2006).

Second, antisocial behaviors are also associated with structural and functional abnormalities in brain circuit related to affiliative temperament. For example, Researches using MRI show that those antisocial personality disorders have 11% reduction in prefrontal gray matter (Raine et al., 2000). This means that individuals of antisocial behaviors are less able to maintain a plan and inhibit irrelevant information due to the dysfunction of higher cortical regulation or execution ability (Pham et al., 2003). Moran (2006) suggested that abnormalities in the amygdala-orbitofrontal system may be the neurological basis of persistent, impulsive antisocial and violent behaviors. Hypervigilance to emotionally laden affiliative stimuli is further confirmed by studies showing enhanced amygdala reactivity to negative scenes (Herpertz et al., 2001), negative facial expressions (Minzenberg et al., 2007), and particularly towards anger (Domes et al., 2008).

As reviewed by Depue & Morrone-Strupinsky (2005), the animal and human evidences demonstrate that the VTA dopamine-NAcc pathway is a primary neural circuit for incentive reward. Variations in how these neural pathways develop may contribute to the risk for antisocial drug use during adolescence. Mild antisocial traits also appeared to aggregate with heightened dopamine activity (Thatcher & Clark, 2008), which means the abnormality of brain reward circuit in affiliative temperament among individuals of antisocial behaviors. Thus, the disturbance among the central regulative system including PFC and ACC, the negative emotion system mainly referring to amygdala, and the reward system (VTA, NAcc in part) seems to result in different antisocial behaviors in the cortical level.

Finally, antisocial behaviors are also linked with alterations in the OT/AVP system. OT reduces amygdala activity in humans (Kirsch et al., 2005), and thus deficits in OT might contribute to the hostility, fear, and mistrust that may provide the preconditions for the emergence of aggression. Inversely, AVP activates amygdale, which increases aggression. Given the high prevalence of severe childhood trauma and neglect in the borderline personality disorder (Lieb et al., 2004), it has been speculated that early stress interferes with the developing neuropeptide system and alters receptor binding of OT and AVP (Heinrichs & Domes, 2008).

Summary and future studies

Affiliative temperament is an individual construct in psychology, and involves a complicate process, from the secondary stimuli processing through intention recognition to the formation of behavioral tendency. There are evidences showing that the neurobiology of affiliativeness depends on the integration of the MNS, the dopaminergic system and the neuropeptides at different levels. First, the MNS interacts with the dopaminergic system within DLPFC at the highest level. DLPFC takes executive control for imitation and emotional contagion, together with the interaction with OFC and ACC. OFC is involved in dynamic reassessment and relearning of affiliative associations, dependent on individual's motivational states. ACC may be to monitor motivational conflict (especially in the aversive context) and select appropriate response with respect to emotional priorities and goals. Second, STS integrates multiple stimuli's values and evaluate others' intention. The VTA, NAcc, insula and amygdala separately process affiliative stimuli and evaluate their values. Then they provide afferent information for higher-order cortex upwards while they activate the response system downwards. Third, on the lower level, OT interacts with dopaminergic circuits in the NAcc and the VTA. AVP also interacts with similar circuits in the NAcc. Thus OT and VAP modulate activity in dopaminergic reward pathways associated with socially affiliative behaviours, directly influence the HPA axis activity, and determine behavioral tendency of affiliative temperament and the strength of subjective experience. Finally, Dysfunction of brain circuits relating to affiliative temperament is associated with psychopathology as well as antisocial behaviors.

In future, we can focus on the followings to make further researches. (1) Using ATQ and EAS to make neuroimaging studies of fMRI, EEG and MEG will provide convergent information about the neurobiology of affiliative temperament. (2) Researches should also be strengthened in psychopathology in order to further clarify the role of affiliative temperament in different psychopathological disorders and testify the neurobiological validity of ATQ and EAS. (3) Longitudinal intervention studies about antisocial behaviors with OT and AVP will be helpful to understanding the developmental mechanism of affiliative temperament in different contexts.

Reference

Agnew, Z.K., Bhakoo, K.K., & Puri, B.K. (2007). The human mirror system: A motor resonance theory of mind-reading. *Brain Research Reviews, 54*, 286-293.

Amaral, D.G., Price, J.L., Pikanen, A., & Carmichael, S.T. (1992). Anatomical organization of the primate amygdaloid complex. In J. P. Aggleton(Ed.), T*he Amygdala:*

Neurobiological Aspects of Emotion, memory and Mental Dysfunction (pp.1-66). New York: Wiley-Liss.

Aragona, B.J., & Wang, Z. (2007). Opposing regulation of pair bond formation by cAMP signaling within the nucleus accumbens shell. *The Journal of Neuroscience, 27*, 13352–13356.

Aron, A., Fisher, H., & Mashek, D.J., et al. (2005). Reward, motivation, and emotion systems associated with early-stage intense romantic love. *Journal of Neurophysiology, 94*, 327–337.

Banissy, M.J., & Ward, J. (2007). Mirror-touch synesthesia is linked with empathy. *Nature Neuroscience, 10*, 815–816.

Baumgartner, T., et al. (2008). Oxytocin Shapes the Neural Circuitry of Trust. *Neuron, 58*, 639–650.

Berger, K.S. (2003). *The Developing Person Through Childhood and Adolescence, 6th edition (3rd publishing)*. Worth Publishers.

Blair, R.J. (2006). The emergence of psychopathy: implications for the neuropsychological approach to developmental disorders. *Cognition, 101*, 414–442.

Boddaert, N., et al. (2004). Superior temporal sulcus anatomical abnormalities in childhood autism: a voxel-based morphometry MRI study. *NeuroImage, 23,* 364-369.

Bora, E., Yucel, M., & Allen, N.B. (2009). Neurobiology of human affiliative behaviour: implications for psychiatric disorders. *Current Opinion Psychiatry, 22*, 320–325.

Brown, S.L., et al. (2009). Social closeness increases salivary progesterone in humans. *Hormones and Behavior*. doi:10.1016/j.yhbeh.2009.03.022.

Buss, A.H., & Plomin, R. (1975). *A temperament theory of personality development.* New York: Wiley.

Buss, A.H., & Plomin, R. (1984). *Temperament: Early developing personality traits*. Hillsdale, NJ: Erlbaum.

Carter, C.S. (2003). Developmental consequences of oxytocin. *Physiology & Behavior*, *79 (3)*, 383-397.

Chakrabarti, B., & Baron-Cohen, S. (2006). Empathizing: neurocognitive developmental mechanisms and individual differences. *Progress in Brain Research, 156,* 403–417.

Cloutier, J., Heatherton, T.F., & Whalen, P.J., et al. (2008). Are attractive people rewarding? Sex differences in the neural substrates of facial attractiveness. *Journal of Cognitive Neuroscience, 20*, 941–951.

Davidson, R.J., Pizzagalli, D., & Nitschke, J.B., et al. (2002). Depression: Perspectives from affective neuroscience. *Annual Review of Psychology, 53,* 545–574.

Decety, J., & Moriguchi, Y. (2007). The empathic brain and its dysfunction in psychiatric populations: implications for intervention across different clinical conditions. *BioPsychoSocial Medicine, 1*, 22-65.

Depue, R.A., & Morrone-Strupinsky, J.V. (2005). A neurobehavioral model of affiliative bonding: Implications for conceptualizing a human trait of affiliation. *Behavioral and brain, 28***,** 313–395.

Devisky, O., Morrell, J., & Vogt, B.A. (1995). Contributions of anterior cingulated cortex to behavior. *Brain, 118*, 279-306.

DeVries, G.J., & Panzica, G.C. (2006). Sexual differentiation of central vasopressin and vasotocin systems in vertebrates: different mechanisms, similar endpoints. *Neuroscience, 138 (3),* 947–955.

Ditzen, B., Neumann, I.D., & Bodenmann, G., et al. (2007). Effects of different kinds of couple interaction on cortisol and heart rate responses to stress in women. *Psychoneuroendocrinology, 32,* 565–574.

Domes, G., Czieschnek, D., & Weidler, F., et al. (2008). Recognition of facial affect in borderline personality disorder. *Journal Personality Disorders, 22*, 135–147.

Domes, G., Heinrichs, M., & Michel, A., et al. (2007). Oxytocin improves mindreading in humans. *Biological Psychiatry, 61*, 731–733.

Elizabeth, A., et al. (2009). Neurobiology of empathy and callousness: implications for the development of antisocial behavior. *Behavioral Sciences and the Law, 27*, 137–171.

Ellis, L.K., & Rothbart, M.K. (2001, April). *Revision of the Early Adolescent Temperament Questionnaire.* Poster presented at the biennial meeting of the Society for Research in Child Development, Minneapolis, MN.

Evans, D.E., & Rothbart, M.K. (2007). Developing a model for adult temperament. *Journal of Research in Personality, 41*, 868–888.

Feldman, R., Greenbaum, C.W., & Yirmiya, N. (1999). Mother-infant affect synchrony as an antecedent of the emergence of self-control. *Developmental Psychology, 35*, 223–231.

Feldman, R., Weller, A., & Zagoory-Sharon, O., et al. (2007). Evidence for a neurobiological foundation of human affiliation. Plasma oxytocin levels across pregnancy and the postpartum period predict mother-infant bonding. *Psychological Science, 18*, 965–970.

Geier, C.F., Garver, K.E., & Luna, B. (2007). Circuitry underlying temporally extended spatial working memory. *NeuroImage, 35,* 904-915.

Goldsmith, H.H. (1996). Studying temperament via construction of the Toddler Behavior Assessment Questionnaire. *Child Development, 67*, 218-235.

Gordon, I., Zagoory-Sharon, O., & Schneidermann, I., et al. (2008). Oxytocin and cortisol in romantically unattached young adults: associations with bonding and psychological distress. *Psychophysiology, 45*, 349–352.

Grimm, S., Beck, J., & Schuepbach, D., et al. (2008). Imbalance between Left and Right Dorsolateral Prefrontal Cortex in Major Depression Is Linked to Negative Emotional Judgment: An fMRI Study in Severe Major Depressive Disorder. *Biological Psychiatry, 63*, 369–376.

Guastella, A.J., Carson, D.S., & Dadds, M.R., et al. (2009). Does oxytocin influence the early detection of angry and happy faces? *Psychoneuroendocrinology, 34*, 220-225.

Guastella, A.J., Mitchell, P.B., & Dadds, M.R. (2008a). Oxytocin increases gaze to the eyeregion of human faces. *Biological Psychiatry, 63 (1)*, 3-5.

Guastella, A.J., Mitchell, P.B., & Mathews, F. (2008b). Oxytocin enhances the encoding of positive social memories in humans. *Biological Psychiatry, 64*, 256-258.

Güroğlu, B, Haselager, G.J.T., & van Lieshout, C.F.M., et al. (2008). Why are friends special? Implementing a social interaction simulation task to probe the neural correlates of friendship. *NeuroImage, 39*, 903–910.

Hadland, K.A., et al. (2003). The effect of cingulate lesions on social behaviour and emotion. *Neuropsychologia, 41*, 919–931.

Hammock, E.A.D., & Young, L.J. (2007). Neuroendocrinology, Neurochemistry, and Molecular Neurobiology of Affiliative Behavior. In Lajtha A. & Blaustein, J.D. (Eds.), *Handbook of Neurochemistry and Molecular Neurobiology Behavioral Neurochemistry, Neuroendocrinology and Molecular Neurobiology* (pp.247-285).

http://www.springerlink.com/content/r5476tx83647x073/ doi: 10.1007/978-0-387-30405-2.

Hein, G., & Singer, T. (2008). I feel how you feel but not always: the empathic brain and its modulation. *Current Opinion in Neurobiology, 18*, 153–158.

Heinrichs, M., & Domes, G. (2008). Neuropeptides and social behaviour: effects of oxytocin and vasopressin in humans. *Progress in Brain Research, 170*, 337-350. DOI: 10.1016/S00796123(08)00428-7.

Hill, G.L., & Werner, N. (2006). Affiliative motivation, school attachment, and aggression in school. *Psychology in the Schools, 43(2)*, 231-246.

Iacoboni, M., & Dapretto, M. (2006). The mirror neuron system and the consequences of its dysfunction. *Nature Reviews Neuroscience, 7*, 942-951.

Iaria, G., et al. (2008). The contribution of the fusiform gyrus and superior temporal sulcus in processing facial attractiveness: Neuropsychological and neuroimaging evidence. *Neuroscience, 155(2)*, 409-422.

Ida Gobbini, M., et al. (2004). Social and emotional attachment in the neural representation of faces. *NeuroImage, 22(4)*, 1628-1635.

Jabbi, M., Swart, M., & Keysers, C. (2007). Empathy for positive and negative emotions in the gustatory cortex. *NeuroImage, 34*, 1744–1753.

Jackson, P.L., Brunet, E., & Meltzoff, A.N., et al. (2006). Empathy examined through the neural mechanisms involved in imagining how I feel versus how you feel pain. *Neuropsychologia, 44(5)*, 752–761.

Jackson, P.L., Meltzoff, A.N., & Decety, J. (2005). How do we perceive the pain of others? A window into the neural processes involved in empathy. *NeuroImage, 24*, 771–779.

Keysers, C., & Gazzola, V. (2006). Towards a unifying neural theory of social cognition. *Progress in Brain Research, 156*, 379-401.

Kirsch, P., Esslinger, C., & Chen, Q. et al. (2005). Oxytocin modulates neural circuitry for social cognition and fear in humans. *Journal of Neuroscience, 25,* 11489–11493.

Kosfeld, M., Heinrichs, M., & Zak, P.J., et al. (2005). Oxytocin increases trust in humans. *Nature, 435*, 673-676.

Kringelbach, M. L. (2005). The orbitofrontal cortex: linking reward to hedonic experience. *Nature Reviews Neuroscience, 6*, 691-702.

Krueger, F., McCabe, K., & Moll, J., et al. (2007). Neural correlates of trust. *PNAS, 104(50)*, 20084–20089.

Lahey, B.B. (2004). Commentary: role of temperament in developmental models of psychopathology. *Journal of Clinical Child and Adolescent Psychology, 33(1)*, 88-93.

LeDoux, J.E. (2000). Emotion Circuits in the Brain. *Annual Review of Neuroscience, 23*, 155-184.

Levine, A., Zagoory-Sharon, O., & Feldman, R., et al. (2007). Oxytocin during pregnancy and early postpartum: individual patterns and maternal-fetal attachment. *Peptide, 28,* 1162–1169.

Lieb, K., Zanarini, M.C., & Schmahl, C., et al. (2004). Borderline personality disorder. *Lancet, 364*, 453–461.

Lim, M.M., & Young, L.J. (2006). Neuropeptidergic regulation of affiliative behavior and social bonding in animals. *Hormones and Behavior, 50,* 506–517.

Machado, C.J., & Bachevalier, J.(2006). The Impact of Selective Amygdala, Orbital Frontal Cortex, or Hippocampal Formation Lesions on Established Social Relationships in Rhesus Monkeys (*Macaca mulatta*). *Behavioral Neuroscience, 120(4),* 761-786.

Martino, A.D., et al. (2009). Functional Brain Correlates of Social and Nonsocial Processes in Autism Spectrum Disorders: An Activation Likelihood Estimation Meta-Analysis. *Biological Psychiatry, 65(1)*, 63-74.

Millan, M.J. (2003). The neurobiology and control of anxious states. *Progress in Neurobiology, 70(2)*, 83-244.

Minzenberg, M.J., Fan, J., & New, A.S. et al. (2007). Fronto-limbic dysfunction in response to facial emotion in borderline personality disorder: an event-related fMRI study. *Psychiatry Reserach, 155*, 231–243.

Moran, M. (2006). Brain changes may explain long-term antisocial behavior. *Psychiatric News September 1, 41(17)*, 24.

Muris, P., & Meesters, C. (2008) Reactive and Regulative Temperament in Youths: Psychometric Evaluation of the Early Adolescent Temperament Questionnaire-Revised. *Journal of Psychopathological Behavior Assessment* (www.springer.com). doi 10.1007/s10862-008-9089-x.

Nigg, J.T. (2006). Temperament and developmental psychopathology. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 47(3-4)*, 395-422.

Noriuchi, M., Kikuchi, Y., & Senoo, A. (2008).The functional neuroanatomy of maternal love: mother's response to infant's attachment behaviours. *Biological Psychiatry, 63*, 415–423.

Nummenmaa, L., Hirvonen, J., & Parkkola, R., et al. (2008). Is emotional contagion special? An fMRI study on neural systems for affective and cognitive empathy. *Neuroimage, 43,* 571–580.

Oatley, K., Keltner, D., & Jenkins, J. M. (2006). *Understanding of emotions* (2nd) (pp.139-164). Blackwell Publishing.

Ortigue, S., Bianchi-Demicheli, F., & Hamilton, A.F.C., et al. (2007). The neural basis of love as a subliminal prime: an event related functional magnetic resonance imaging study. *Journal Cognitive Neuroscience, 19*, 1218–1230.

Otsuka, Y., et al. (2009). Individual differences in the theory of mind and superior temporal sulcus. *Neuroscience Letters, 463(2)*, 150-153.

Petrovic, P., Kalisch, R., & Singer, T., et al. (2008). Oxytocin attenuates affective evaluations of conditioned faces and amygdala activity. *The Journal of Neuroscience, 28(26)*, 6607-6615.

Pfeifer, J.H., Iacoboni, M., & Mazziotta, J.C., et al. (2008). Mirroring others' emotions relates to empathy and interpersonal competence in children. *Neuroimage, 39(4)*, 2076–2085.

Pham, T.H., Vanderstukken, O., & Philippot, P.et al. (2003). Selective attention and executive functions deficits among criminal psychopaths. *Aggressive Behavior, 29(5)*, 393 – 405.

Pierce, R.C., & Kumaresan, V. (2006). The mesolimbic dopamine system: The final common pathway for the reinforcing effect of drugs of abuse? *Neuroscience and Biobehavioral Reviews, 30*, 215-38.

Posner, M.I., & DiGirolamo, G.J. (1998). Executive attention: Conflict, target detection, and cognitive control. In R. Parasuraman (Ed.), *The Attentive Brain* (pp. 401–423). Cambridge, MA: MIT Press.

Posner, M.I., Rothbart, M.K., & Sheese, B.E. (2007). Attention genes. *Developmental Science, 10,* 24–29.

Raine, A., & Yang, Y. (2006). Neural foundations to moral reasoning and antisocial behavior. *Social Cognitive and Affective Neuroscience, 1(3)*, 203-213.

Raine, A., Lenez, T., & Bihrle, S.et al. (2000). Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial. *Archives of General Psychiatry, 57*, 119-127.

Rajmohan, V., & Mohandas, E. (2007). Mirror neuron system. *Indian Journal of Psychiatry, 49*, 66-69.

Rimmele, U., Hediger, K., & Heinrichs, M., et al. (2009). Oxytocin makes a face in memory familiar. *The Journal of Neuroscience, 29(1)*, 38-42.

Rizzolatti, G., & Craighero, L. (2004). The mirror-neuron system. *Annual Review of Neuroscience, 27*, 169-192.

Rizzolatti, G., Fadiga, L., & Gallese, V., et al. (1996). Premotor cortex and the recognition of motor actions. *Cognitive Brain Research, 3 (2)*, 131-141.

Rolls, E.T., & Grabenhorst, F. (2008). The orbitofrontal cortex and beyond: From affect to decision-making. *Progress in Neurobiology, 86*, 216–244.

Rothbart, M.K. (1981). Measurement of temperament in infancy. *Child Development, 52*, 569-578.

Rothbart, M.K., & Bates, J.E. (2006). Temperament, In W. Damon (Series Ed.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol3. Social, emotional, and personality development* (6th ed., pp.99-166). New York: Wiley.

Rothbart, M.K., Ahadi, S.A., & Hershey, K.L. (1994). Temperament and social behavior in childhood. *Merrill-Palmer Quarterly, 40*, 21–39.

Rowe, D.C., & Plomin, R. (1977). Temperament in early childhood. *Journal of Personality Assessment, 41,* 150–156.

Schwienbacher, I., et al. (2004). Temporary inactivation of the nucleus accumbens disrupts acquisition and expression of fear-potentiated startle in rats. *Brain Research*, *1027,* 87-93.

Simplicio, M.D., Massey-Chase, R., & Cowen, P.J., et al. (2008). Oxytocin enhances processing of positive versus negative emotional information in healthy male volunteers. *Journal of psychopharmacology, 23(3)*, 241-248.

Singer, T., Seymour, B., & O'Doherty, J., et al. (2004). Empathy for pain involves the affective but not sensory components of pain. *Science, 303(5661)*, 1157–1162.

Small, D.A., Zald, D.H., & Jones-Gotman, M., et al. (1999). Human cortical gustatory areas: a review of functional neuroimaging data. *Neuroreport, 10*, 7-14.

Sofroniew, M.V. (1983). Morphology of vasopressin and oxytocin neurones and their central and vascular projections. *Progress in Brain Research, 60*, 101–114.

Sollberger, M., et al. (2009). Neural basis of interpersonal traits in neurodegenerative diseases. *Neuropsychologia.* doi:10.1016/j.neuropsychologia.2009.06.006.

Theodoridou, A., Rowe, A.C., & Penton-Voak, I.S., et al. (2009). Oxytocin and social perception: Oxytocin increases perceived facial trustworthiness and attractiveness. *Hormones and Behavior*. doi:10.1016/j.yhbeh.2009.03.019.

Thompson, R., Gupta, S., & Miller, K., et al. (2004). The effects of vasopressin on human facial responses related to social communication. *Psychoneuroendocrinology, 29*, 35–48.

Thompson, R.R., George, K., & Walton, J.C., et al. (2006). Sex-specific influences of vasopressin on human social communication. *Proceedings of the National Academy of Science of the United States of America, 103,* 7889–7894.

Tops, M., Van Peer, J.M., & Korf, J., et al. (2007). Anxiety, cortisol and attachment predict plasma oxytocin. *Psychophysiology, 44,* 444–449.

Unkelbach, C., Guastella, A.J., & Forgas, J.P. (2008). Oxytocin Selectively Facilitates Recognition of Positive Sex and Relationship Words. *Psychological Science, 19(11)*, 1092-1094.

Vaish, A., Carpenter, M., & Tomasello, M. (2009). Sympathy through affective perspective taking and its relation to prosocial behavior in toddlers. *Developmental Psychology, 45(2)*, 534–543.

Vrtička, P., Andersson, F., & Grandjean, D., et al. (2008). Individual Attachment Style Modulates Human Amygdala and Striatum Activation during Social Appraisal. *PLOS One, 3*, e2868.

Wager, T.D., Phan, K. L., & Liberzon, I., et al. (2003). Valence, gender, and lateralization of functional brain anatomy in emotion: a meta-analysis of findings from neuroimaging. *NeuroImage, 19*, 513–531.

Weinstein, T.A.R., & Capitanio, J.P. (2005). A nonhuman primate perspective on affiliation. *Behavioral and Brain Sciences, 28*, 366-367.

Whittle, S., Allen, N.B., & Lubman, D.I., et al. (2006). The neurobiological basis of temperament: Towards a better understanding of psychopathology. *Neuroscience and Biobehavioral Reviews, 30*, 511–525.

Whittle, S., Yucel, M., & Fornito, A., et al. (2008). Neuroanatomical correlates of temperament in early adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 47*, 682–693.

Young, S.N. (2008).The neurobiology of human social behaviour: an important but neglected topic. *Journal of Psychiatry Neuroscience, 33(5)*, 391–392.

Zak, P.J., Kurzban, R., & Matzner, W.T. (2005). Oxytocin is associated with human trustworthiness. *Hormones and Behavior, 48*, 522-527.

Zak, P.J., Stanton, A.S., & Ahmad, S. (2007). Oxytocin increases generosity in humans. *PLoS ONE, 2(11)*: e1128. doi:10.1371/journal.pone.0001128.