



# Psychopathy and Facial Expressions of Emotions: Processing, Recognizing and Mimicking

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## **Abstract**

Psychopathy is a personality disorder characterized by several persistent symptoms including problems in interpersonal relations, empathy and emotional functioning. The purpose of this review is examining whether psychopaths emotionally react to and process facial expressions of emotions in the same way as other people and which brain circuits are predominantly involved. The main question is whether psychopaths show abnormalities in recognizing facial expressions of emotions of other individuals.

The results suggest that psychopaths exhibit disproportional deficits in processing, recognizing and mimicking facial expressions of emotions. Psychopaths most likely exhibit amygdala dysfunction and maybe impairment in frontal brain areas. Consequently, the recognition of fear and sadness ('amygdalian emotions') is almost certainly impaired. There also appears to be a delay in cortical maturation in several brain areas in psychopaths. This delay may be associated with impairments in decision-making, morality and empathy. Moreover, facial expression could be of a more primeval character in psychopaths.

Motor mimicry is assumed to be the essence of emotional empathy and to be biologically 'hardwired'. Upper facial mimicry is supposed to predict trait empathy. Therefore, it is thought that psychopaths may have a specific deficit in upper facial mimicry. Finally, there are findings that provide the first evidence for the claim that adolescent pathology is indeed manifest in distinct facial expressions of emotion.

These findings can be useful in clinical practice. More knowledge about the underlying deficits of empathy impairment can be used for developing more suitable and individualized training programs aimed to strengthen the empathic skills of psychopaths. On the other hand, more knowledge about mimicking may be used to determine whether such empathy training would be useful.

Keywords: psychopathy, emotions, mimicking, empathy

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## Introduction

It is widely believed that emotions are evolved and serve an important communicative function (e.g. Ekman, 1992; Hess & Thibault, 2009). Each emotion motivates cognitive processes, physiological responses, and expressive behavior that help the individual respond adaptively to specific problems of survival, such as fleeing from danger, developing and maintaining close social bonds, and avoiding or apologizing for social and moral transgressions (Keltner, Moffitt, & Stouthamer-Loeber, 1995). Emotions also figure prominently in psychological maladjustment; in certain cases, the absence of emotions contributes to psychological dysfunction. On the contrary, abnormal intense emotional responses can also contribute to psychological dysfunction. The link between psychopathology and emotions, in fact, is so pervasive that 45% of the diagnoses listed in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) refer to abnormal emotional responses (*Diagnostic and Statistical Manual of Mental Disorders* (4<sup>th</sup> ed., text rev.; American Psychiatric Association, 2000).

Psychopathy is a personality disorder with several persistent symptoms, such as problems in interpersonal relations and emotional functioning, and several behavioral problems like impulsive and antisocial behaviors and a lack of a sense of responsibility. This complex behavior has been quite thoroughly studied. The results point out that specific brain circuits involved do not function properly in psychopaths; they exhibit disproportional deficits concerning the processing of emotions. The human face is an especially differentiated and reliable medium regarding the expression of emotions, therefore the focus will be on the face instead of the whole body.

The purpose of this review is researching whether psychopaths emotionally react and process emotions in the same way as normal people and what brain circuits are predominantly involved. The main question is whether psychopaths show abnormalities in facial expressions of emotions. The first chapter aims for a complete understanding of emotions; first emotion in general will be discussed, subsequently emotion recognition and the neurobiology of emotions will be treated. The second chapter describes psychopathy in general, psychopathy and emotion processing and the recognition of facial expressions of emotions by psychopaths.

Harrison, Morgan, & Critchley (2010) state that the tendency to mimic others' emotional expressions predicts empathy and may represent a physiological marker of psychopathy. Empathy is related to reduced or different mimicry and psychopaths are known to have a relatively selective empathy deficit. Therefore, mimicking will be described in chapter three. First mimicking in general will be made explicit, subsequently the neurobiology of mimicking will be discussed and next mimicking in psychopaths. Finally, it will be discussed whether psychopaths show deviations in facial expressions of emotions and if the emotional disturbances psychopaths exhibit express themselves via facial expressions of emotions.

## Emotions

Emotions play an important role in our behavior and are of great importance in our daily living. Theories about emotions stretch back at least as far as the stoics of ancient Greece, like Plato and Aristotle. Later theories are not mutually exclusive and many researchers incorporate multiple perspectives in their work.

According to Charles Darwin, emotions are evolved and adaptive (at least at some point in the evolution) and serve an important communicative function (Darwin, 1871, as cited by Hess & Thibault, 2009). Darwin stressed that the expressive behavior that he described was part of an underlying emotional state, so emotional expressions derived their

communicative value from the fact that they were outward manifestations of an inner state. Darwin considered these expressions to be inheritable and evolved; therefore he assumed parallels between human emotions, the emotions of animals and the emotions of our humanoid ancestors. On the basis of his evolution theory, Darwin postulated universals in facial behavior (Hess & Thibault, 2009).

Facial expression did not receive very much attention in the 1950s and 1960s, but Plutchik (1962) and Tomkins (1962) both provided influential evolutionary accounts of facial expressions. Tomkins and McCarter (1964) provided the first evidence that observers in the judgment of facial expression could achieve very high agreement. Tomkins directly influenced both Paul Ekman and Wallace Friesen in their research (Keltner & Ekman, 2000).

Currently, Paul Ekman is an influential researcher with respect to facial expressions of emotions. Ekman established six basic emotions: happiness, sadness, anger, surprise, disgust and fear. Ekman and Friesen (1971) found particular facial behaviors to be universally associated with particular emotions by showing populations, who were not exposed to the media, pictures from Western people expressing specific emotions (Ekman & Friesen, 1971). Furthermore, Ekman (1992) found that each emotion has unique features: behavioral signs, physiology, and antecedent events. Each emotion also has characteristics in common with other emotions: rapid onset, short duration, unbidden occurrence, automatic appraisal, and coherence among responses. These shared and unique characteristics are the product of our evolution, and distinguish emotions from other affective phenomena (Ekman, 1992).

Furthermore, researchers have developed objective measures of facial expression. “Affective facial expressions can be quantitatively analyzed by trained experts coding elementary facial actions, or by automated systems recognizing facial expressions through visual analysis of facial movements. Another method is recording electromyographic (EMG) signals of specific facial muscles. Both visual and EMG methods have their strengths and weaknesses.” (van Boxtel, 2010). EMG responses are objective and unbiased while systems relying on the analysis of observable facial movements are subjective and may be biased. Another weakness associated with systems relying on the analysis of observable facial movements is that weak or moderate affective responses may be accompanied by visually undetectable facial actions (Tassinari & Cacioppo, 1992, as cited by van Boxtel, 2010). Using EMG techniques even the weakest responses, remaining under the visual detection threshold, can be detected. Additionally, EMG signals have a good time resolution so that rapid changes in activity can be reliably measured. Using techniques relying on observable facial movements, small dynamic transitions in activity may be less well observed since they may be masked by the stiffness of overlying cutaneous and subcutaneous tissues. Good dynamic response properties are necessary for accurate measurement of response latencies to affective stimuli or rapid changes in emotional state during social interactions, e.g. emotional mimicry. Taking these strengths and weaknesses together, EMG recording may be considered a sensitive technique for inferring subjective mood states or affective responses. However, this technique has limitations as well. Due to its obtrusiveness and the fact that facial activity is influenced by many other, nonaffective, behavioral factors (as described later) it pertains limitations for many applications under natural life circumstances. “Also, methodological improvements are necessary to enhance its effectiveness as a tool for reliable differentiation between specific emotions.” (van Boxtel, 2010).

The results from the studies of Ekman and Friesen have interesting implications. If the recognition of emotions were indeed universal, then it would be possible that emotions are congenital, like Darwin stated. It also says something about the evolution of the human being. If facial expressions of emotions are considered adaptive mechanisms, then facial expressions of emotions somehow helped to survive the human being at some point in history. If thereby

some messages could be communicated by facial expressions, people could help other people survive by using these expressions. Accordingly, emotions could prepare an organism to act verbally and non-verbally in response to environmental stimuli and challenges. Therefore it is important to interpret facial expressions as accurate as possible to function well in social situations (Kring & Bachorowski, 1999). On the basis of facial expressions people can decide what to do in a specific situation. The question rises if emotions today still play an important role in helping to survive the human race.

It is certain that emotions play a crucial role in interpersonal communication (Ekman, 1999). Emotional signals, either visual or auditory, can be considered as aspects of both an emotional response and social communication (Adolphs, 2002). By facial expressions people often express emotions, either intentional or unintentional. However, the human face not only displays affective responses. The human face also produces a large variety of activities unrelated to emotional processes like speech, mental effort or mental fatigue, task involvement or performance motivation, anticipation of sensory stimuli, preparation of motor responses, orienting responses, and startle reflexes. Consequently, facial expressions are not necessarily expressions of emotions. The influences of these omnipresent and perhaps confounding factors should be carefully evaluated to avoid invalid conclusions regarding a person's affective state (van Boxtel, 2010). The next question is how people recognize emotions shown by facial expressions.

### *Emotion recognition*

There are several theories concerning recognition of facial expressions of emotion. One of the first theories is from Ekman and Friesen, the facial feedback hypothesis (e.g. Buck, 1980). This hypothesis is based on earlier work from William James and Carl Lange, who stated that the direct perception of a particular somatic state (visceral, postural, or facial) was the essence of what it meant to have a particular emotional experience (Davis, Senghas & Ochsner, 2009). The facial feedback hypothesis focuses on facial expressions and the influence of these expressions on emotional experience. This hypothesis states that when people perceive a facial expression, they automatically tense the facial muscles responsible for the expression they perceive. This part of the facial feedback hypothesis in fact states that mimicry occurs. When they become aware of their own muscle tension, or their mimicry, by feedback of muscle contractions to the brain, the perceived facial expression is interpreted. So facial expressions are either necessary or sufficient to produce emotional experience (Davis et al., 2009).

Keillor, Barret, Crucian, Kortenkamp and Heilman have criticized this theory in 2002. They examined the role of facial expressions in determining emotion by studying a patient (F.P.) suffering from a bilateral facial paralysis. Despite her inability to convey emotions through facial expressions, F.P. reported normal emotional experience. When F.P. viewed emotionally evocative slides, her reactions were not dampened relative to the normative sample. F.P. retained her ability to detect, discriminate, and image emotional expressions. These findings are neither consistent with theories stating that feedback from an active face is necessary to experience emotion while processing emotional facial expressions, nor to recognize emotion from such expressions (Keillor et al., 2002).

A second theory about the recognition of emotions is the model by Bruce and Young (Ellis & Young, 1988). This model distinguishes several components: recognition of facial identity, the analysis of facial expressions, and the analysis of facial speech by which people are able to encode faces. By analyzing faces structurally, different sorts of information can be reduced independently, but in a comparable way. Through this independent way of information processing the model is able to explain specific losses of function caused by brain

damage. This model thus considers that it is possible that a person with brain damage is not able to identify familiar faces, while he is able to recognize the emotions expressed on these familiar faces (Ellis & Young, 1988). This model however, does not explain how the brain processes the dissimilar facial expressions. The next questions are how the brain processes emotions and how the brain is involved in recognizing emotions. First the neurobiology of emotions will be discussed.

### *Neurobiology of emotions*

There is much neuropsychological research regarding emotions and the recognition of emotions. This research consists of experiments with healthy subjects and subjects with brain damage. Borod (1993) proposed a theoretical model (the componential approach) for the neuropsychological study of emotional processing. This model describes processing modes, communication channels, emotional dimensions, and discrete emotions. Processing modes include perception, experience and expression. Each of these three aspects can be individually affected by brain damage (Borod, 1993). Communication channels include facial expressions, language, prosody, sign language, and body language. There are several emotional dimensions like pleasant-unpleasant and exciting-not exciting. However, most research regards discrete emotions; the six basic emotions found by Ekman: happiness, sadness, anger, surprise, disgust, and fear (van Strien, 2000).

Most brain structures that participate in recognizing basic emotions involve both perceptual processing and the recognition of the emotional meaning of a stimulus. Perceptual processing means identifying the geometric configuration of facial features in order to discriminate among different stimuli on the basis of their appearance. Recognition of the emotional meaning of a stimulus means knowing that a certain expression for example signals fear. Recognition relies on disparate strategies. For instance, recognition of fear from a facial expression may occur by linking the perceptual properties of the facial stimulus to various knowledge-based processes. These include the knowledge components of the concept of fear, the lexical label 'fear', and the perception of the emotional fear response (or a central representation thereof) that the stimulus triggers in the subject, and knowledge about the motor representations required to produce the expression shown in the stimulus (Adolphs, 2002).

A large number of different structures participate in recognizing the emotion shown in a face: e.g. the occipitotemporal cortices, amygdala, orbitofrontal cortex, basal ganglia, and right parietal cortices. These structures are engaged in multiple processes and at various points in time, making it difficult to assign a single function to a structure (Adolphs, 2002).

Regions of the occipital and posterior temporal visual cortices play a critical role in perceptual processing of socially and emotionally relevant visual stimuli. Single-unit studies in monkeys, intracranial field potential studies in neurosurgical human patients and functional imaging studies, have all provided evidence that cortical areas in the lateral parts of the inferior occipital gyrus, fusiform gyrus, and superior temporal gyrus are disproportionately important in face processing. The earliest activity that discriminates between emotional facial expressions is seen in midline occipital cortex as early as 80 ms to 110 ms after stimulus onset. Monkey single-unit recordings have provided evidence that neurons in the temporal cortex encode information about faces variably with time. Although information sufficient to distinguish faces from other objects is encoded in about 120 ms, responses encoding fine-grained, subordinate information sufficient to distinguish between different emotional expressions only appear after about 170 ms (these latencies would be somewhat longer in humans). These findings suggest the possibility that responses to emotional stimuli in visual cortices are modulated by feedback, perhaps from structures such as the amygdala and

orbitofrontal cortex (Adolphs, 2002). The question is now if dysfunctions of the amygdala and the orbitofrontal cortex play a role in recognition of emotions.

The amygdala plays a significant role in the recognition of emotional signals via at least two pathways: a subcortical route via the superior colliculus and the pulvinar thalamus, and a cortical route via the visual neocortex. Structures in the subcortical route are activated when normal subjects are presented with subliminal facial expressions of fear (Morris, Öhman, & Dolan, 1999), and when subjects with blindsight, due to striate cortex damage, discriminate emotional facial expressions (Morris, de Gelder, Weiskrantz, & Dolan, 2001).

Some studies show that bilateral amygdala damage causes impaired recognition of facial expressions of emotions, principally the recognition of fear (Anderson & Phelps, 2000; Calder, Lawrence & Young, 2001). In another study most subjects with bilateral amygdala damage were impaired on several negative emotions in addition to fear, but no subject was impaired in recognizing positive emotions. The results from this study imply that the amygdala plays an important role in the signaling of threat and danger present in facial expressions (Adolphs et al., 1999). There has been a discussion about the interpretation of these results. Adolphs and his colleagues (1999) state that the amygdala is principally involved in processing negative emotions like fear, sadness and disgust. Whalen (1999) stressed that the amygdala activates cognitive resources to solve ambiguity in the direct environment. At last, emotions of which recognition strongly depends on the amygdala, like fear, disgust and sadness, may be related to behavioral withdrawal (Anderson, Spencer, Fulbright & Phelps, 2000).

Damage to the orbitofrontal cortex, especially on the right side, can result in impaired recognition of facial expressions of emotions and impaired recognition of emotions from the voice. In contrast to the amygdala's activation in response to passive viewing of emotional faces, prefrontal regions may be activated when subjects are engaged in a cognitive task requiring explicit identification of emotions. Disruption of processes within the medial prefrontal cortex with transcranial magnetic stimulation produces longer reaction times in response to angry facial expressions, but not in response to happy facial expressions (Adolphs, 2002). These findings indicate that the medial prefrontal cortex plays a role in the recognition of anger, but not of happiness.

Furthermore, the amygdala and the orbitofrontal cortices may participate in the processing of facial expressions of emotions in at least three distinct ways (Adolphs, 2002). First, they may adjust perceptual representations via feedback. This mechanism might contribute, in particular, to fine-tuning the categorization of a specific facial expression. Second, the amygdala and orbitofrontal cortices may activate associated knowledge, via projections to other regions of the neocortex. This mechanism might contribute especially to retrieval of conceptual knowledge about the particular emotion involved. Third, they may generate an emotional response in the subject, via connections to motor structures, hypothalamus, and brainstem nuclei, where components of an emotional response to the facial expression can be activated. This mechanism might contribute to the generation of knowledge about another person's emotional state via the process of simulation, and would draw on somatosensory related cortices in the right hemisphere for representing the emotional changes in the perceiver. However, it is possible that the simulation of another's emotion could proceed via the generation of an image of the associated body state, even in the absence of actual motor mimicry (Adolphs, 2002).

### **What is Psychopathy?**

As stated before, it is important to interpret facial expressions as accurate as possible to function well in social situations (Kring & Bachorowski, 1999). Some parts of the brain



play a significant role by processing and interpreting facial expressions. When these areas are impaired or damaged, one can hypothesize that the interpreting and processing of emotions are impaired as well. There are neurologic and psychiatric disorders, whereby the recognition of expressions of emotions is known to be impaired. One of these disorders is psychopathy (Berking & Wupperman, 2012). Here it is important to know what psychopathy exactly comes down to. To give a description of psychopathy, it is important to take the classification, the diagnostic criteria and the differences in relation to other disorders in account.

The classification of psychopathy is not identical to diagnoses as conduct disorder or antisocial personality disorder (ASPD); it is rather an expansion of these disorders. The essential feature of ASPD is a pervasive pattern of disregard and violation of the rights of others that begins in childhood or early adolescence and continues into adulthood. This pattern has also been referred to as sociopathy or dissocial personality disorder. Conduct disorder involves a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate social norms or rules are violated. Both conduct disorder and ASPD are DSM-IV diagnoses (DSM-IV-TR, 2000). Robert Hare (1996) criticizes the classification system of the DSM-IV-TR. He argues that the diagnoses are badly itemized and focus too much on the antisocial behavior. Psychopathy, as he states, is not just antisocial behavior but includes social damage as for instance lack of guilt as well. High scores on antisocial behavior, as measured by the Hare Psychopathy Checklist-Revised (PCL-R), are associated with the diagnoses of conduct and antisocial personality disorder. The social damage shown by psychopaths however, is not associated with both of these disorders. In fact, most psychopaths meet the criteria for ASPD, but most individuals with ASPD are not psychopaths. The differences between psychopathy and ASPD are further highlighted by recent laboratory research involving the processing and use of linguistic and emotional information.

Psychopaths differ significantly from nonpsychopaths in their performance of a variety of cognitive and affective tasks. Compared with normal individuals, for example, psychopaths are less able to process or use the deep semantic meanings of language and to appreciate the emotional significance of events or experiences (Hare, 1996). It is worth noting that it is the interpersonal and affective components of psychopathy (as measured by PCL-R) that are most discriminating in these experiments. In sharp contrast, those with a diagnosis of ASPD (in which interpersonal and affective traits play only a small role) differ little from those without ASPD in their processing of linguistic and emotional material. Thereby, a diagnosis of psychopathy, in contrast to ASPD, can be informative with relation to the prospective risk of criminal behavior of the patient (Hare, 1996).

To provide an impression of the dimensions of psychopathy, the two main checklists regarding psychopathy will be shortly discussed here. The Hare Psychopathy Checklist (PCL) precedes the PCL-R (Hare, 1996). Both the PCL and the PCL-R are based on the description for psychopathy by Cleckley, which will be extensively described next. Both the PCL and the PCL-R consist of two parts: a semi-structured interview and a review of the subject's file records and history. The PCL measures two correlated factors. Factor 1 describes a cluster of affective-interpersonal traits central to psychopathy and factor 2 describes traits and behaviors associated with an unstable, unsocialized lifestyle, or social deviance. During the interview, the clinician scores the items (22 items in the PCL and 20 items in the PCL-R) that measure central elements of the psychopathic character. The 20 items or traits assessed by the PCL-R score are: "glib and superficial charm; grandiose (exaggeratedly high); estimation of self need for stimulation; pathological lying; cunning and manipulativeness; lack of remorse or guilt; shallow affect (superficial emotional responsiveness); callousness and lack of empathy; parasitic lifestyle; poor behavioral controls; sexual promiscuity; early behavior problems; lack

of realistic long-term goals; impulsivity; irresponsibility; failure to accept responsibility for own actions; many short-term marital relationships; juvenile delinquency; revocation of conditional release; and criminal versatility.” (Hare, 2003). Like the PCL, the PCL-R provides a total score that is most important for the overall assessment of psychopathy. The total score can be interpreted dimensionally in terms of degree of match to the prototypical psychopath, or it can be used categorically to help identify/diagnose psychopaths. The PCL-R retains the original two factors that reflect the two major facets of psychopathy: the callous, selfish, remorseless use of others (factor 1) and a chronically unstable and antisocial lifestyle (factor 2). The interpretive power of the PCL-R has been enriched, however, through the evolution of four subfactors. Factor 1 and factor 2 are divided into two empirically derived and validated subfactors: Factor 1a, interpersonal; factor 1b, affective; factor 2a, impulsive lifestyle; and factor 2b, antisocial behavior (Hare, 2003).

The first publication of Cleckley’s text, *The Mask of Sanity* (1941), marked the beginning of the modern clinical construct of psychopathy, and his characterization has remained relatively stable to the present day. Cleckley based his description of the psychopath on observations of white, middle-class male patients, residing as inpatients of a mental hospital (Arrigo & Shipley, 2001). Cleckley listed characteristic points emerging from his observations: superficial charm and good ‘intelligence’; absence of delusions and other signs of irrational thinking; absence of ‘nervousness’ or psychoneurotic manifestations; unreliability; untruthfulness and insincerity; lack of remorse or shame; inadequately motivated antisocial behavior; poor judgment and failure to learn by experience; pathologic egocentricity and incapacity for love; general poverty in major affective reactions; specific loss of insight; unresponsiveness in general interpersonal relations; fantastic and uninviting behavior often involving alcohol; suicide rarely carried out; impersonal sex life, trivial and poorly integrated; failure to follow any life plan. In his definition, he does not explicitly refer to aggressive behavior, he even puts forward that a psychopath does not necessarily display aggressive or criminal behavior. Cleckley viewed moral feelings or the human conscience as learned, and the learning process as directed and reinforced by emotional feelings. In addition, Cleckley argued that if normal human emotions were diminished, the development of morality or socialization would be jeopardized (Cleckley, 1976).

Cleckley recognized that many psychopaths never became involved with the criminal justice system. Moreover, he states that many can succeed in business or in other endeavors, particularly in careers offering considerable material success. Cleckley stated that the true difference between the ‘successful’ psychopaths and the psychopaths who continually go to jails or psychiatric hospitals is that they keep up a far better and more consistent outward appearance of being normal. Cleckley observed that the primary psychopathic characteristics of glibness, superficial charm, emotional detachment, and lack of remorse or guilt could be used for both successful criminal and noncriminal careers. Psychopaths pursue what they want without experiencing anxiety attributable to a concern for how their actions might impact others.

Cleckley also described the psychopath’s behavior. He noted that it is impossible for the psychopath to take even the slightest interest in the tragedy or joy or the striving of humanity presented in serious literature or art. He is also indifferent to all these matters in life itself. Beauty and ugliness (except in a very superficial sense), goodness, evil, love, horror, and humor have no actual meaning, no power to move him. Cleckley also said that the psychopath lacks the ability to see that others are moved. He says: “it is as though psychopaths were color-blind, despite their sharp intelligence, to this aspect of human existence. It cannot be explained to them because there is nothing in their orbit of awareness that can bridge the gap with comparison. A psychopath can repeat the words and say glibly

that he understands, and there is no way for him to realize that he does not understand.” (Cleckley, 1976). By saying this, Cleckley in fact states that psychopaths have a deficit in empathy where they are not aware of themselves.

When looking at the characteristics described above, the question rises how a person becomes a psychopath. First, the etiology of psychopathy will be considered. In clarifying the conceptual boundaries of psychopathy, two prominent approaches have emerged. One group of scholars view psychopathy primarily from a personality-based approach. This is exemplified by Cleckley’s classic clinical description of psychopathy as a constellation of deviant personality traits. Other scholars however, conceptualize psychopathy as a behavioral syndrome that should instead be operationalized in terms of a history of chronic antisocial behaviors. Such behavioral, categorical conceptualizations continue to dominate the DSM-IV-TR although the concept psychopathy does not appear in the DSM-IV-TR.

Although genetic behavior studies have attempted to ascertain the relative influence of genetic and environmental etiological factors to the disorder, they have not specifically tapped the core personality features as defined by Cleckley (Blonigen, Carlson, Krueger and Patrick, 2003).

Wootton, Frick, Shelton and Silverthorn (1997) found that ineffective parenting was associated with conduct problems only in children without significant levels of callous (e.g. lack of empathy, manipulateness) and unemotional (e.g. lack of guilt, emotional constrictedness) traits. In contrast, children high on these traits exhibited a significant number of conduct problems, regardless of the quality of parenting they experienced. This implicates that the emotional deficits psychopaths exhibit, form part of the personality.

Assuming that the personality-based conceptualization of psychopathy comprises the etiology of psychopathy best, psychopathy can be considered a personality disorder. According to Cleckley (1976), psychopathy can be seen as a lifelong personality disorder rather than a somehow treatable disorder. Psychopaths do not seem to benefit from treatment or learn from life experiences, they can convince their psychotherapist that treatment has been effective, that it has brought true insight and profound changes making him no longer a danger to society. The daily newspapers, however, report many cases of armed robbery, rape, and murder resulting from such confidently optimistic estimates of therapeutic success. Blair et al. (2004) looked at the passive avoidance learning in individuals with psychopathy compared with healthy individuals. Psychopaths made more passive avoidance errors than the comparisons. In addition, while the level of reward modulated the performance of both groups, only the performance of psychopaths was not modulated by punishment.

Harpur and Hare (1994) examined the assessment of psychopathy as a function of age in 889 male prison inmates. Ratings of psychopathy were made with the PCL, which measures two correlated factors as described before. Scores on factor 1 (affective-interpersonal traits) seemed to be stable across the age-span and mean scores of factor 2 (social deviance) declined with age. The prevalence of antisocial personality disorder, and to a lesser extent of PCL-defined psychopathy, also declined with age. The results suggest that age-related differences in traits related to impulsivity, social deviance, and antisocial behavior are not necessarily paralleled by differences in the egocentric, manipulative, and callous traits fundamental to psychopathy (Harpur & Hare, 1994). Because of the possible chronic course of mainly the affective-interpersonal traits central to psychopathy, it may be interesting to look at the neurobiology of psychopathy.

### *Neurobiology of psychopathy*

It is suggested that amygdala dysfunction is one of the core neural systems implicated in the pathology of psychopathy (Blair, 2003). Two recent neuroimaging studies have

confirmed amygdala dysfunction to be associated with psychopathy. Tiihonen, Hodgins & Vaurio (2000) used volumetric magnetic resonance imaging (MRI) to investigate the relationship between amygdaloid volume and degree of psychopathy in violent offenders as measured by the PCL-R. They found that high levels of psychopathy were indeed associated with reduced amygdaloid volume. Kiehl, Smith and Hare (2001) used functional MRI to examine neural responses in individuals with respectively high and low scores on the PCL-R during an emotional memory task where the participant processed words of neutral and negative valence. They found a reduced amygdala response in the high-scoring group, compared to the low-scoring group during the processing of words of negative valence.

There have been suggestions that other neural systems are dysfunctional in individuals with psychopathy. On the basis of neuropsychological and neuroimaging findings for violent offenders, it has been argued that the frontal cortex could be dysfunctional (Morgan & Lilienfeld, 2000; Soderstrom, Tullberg, Wikkelso, Ekholm, & Forsman, 2000, both as cited by Blair, 2003). However, these studies have been with violent offenders rather than psychopaths. As already mentioned before, there are crucial differences between the general population of violent offenders and psychopaths. Neuropsychological studies with psychopaths, unlike studies with violent individuals, have repeatedly found frontal functioning to be intact (Blair 2003).

The volumetric MRI study conducted by Raine, Lencz, Bihrlé, LaCasse, & Coletti (2000) assessed individuals scoring high on the PCL-R. This study reported reduced prefrontal grey, but not white, matter volume in these individuals. However, they were unable to differentiate between grey matter from different regions of the frontal cortex. It appears that there is no generalized frontal cortical dysfunction in psychopaths, but one region of the frontal cortex that could be impaired; the orbitofrontal cortex (Raine et al., 2000 as cited by Blair, 2003).

De Brito et al. (2009) examined children with callous-unemotional traits, which are thought to be antecedents of psychopathy. They used voxel-based morphometry to compare whole brain grey matter volumes and concentrations of boys with elevated levels of callous-unemotional conduct problems and typically developing boys. Both grey matter volume and concentration were examined controlling for cognitive ability and hyperactivity-inattention disorder symptoms. Boys with callous-unemotional conduct problems, as compared with typically developing boys, presented increased grey matter concentration in the medial orbitofrontal (OFC) and anterior cingulate cortices, as well as increased grey matter volume and concentration in the temporal lobes bilaterally. These findings may indicate a delay in cortical maturation in several brain areas implicated in decision-making, morality and empathy in boys with callous-unemotional conduct problems.

The OFC, especially the medial OFC, receives extensive projections from, and sends extensive projections to, the amygdala. Moreover, the medial OFC is involved in instrumental learning and response reversal, both of which functions are impaired in individuals with psychopathy (Blair, 2003). These findings support the results by de Brito et al. (2009).

Taken these results together, it is suggested that the neural structures implicated in psychopathy include the amygdala and OFC. However, the basic causes remain unclear. The lifestyle of psychopaths may exacerbate any neurobiological impairment. One feature associated with psychopathy is substance misuse. This could be contributing to the apparent impairments discussed above. Chronic amphetamine misuse, for example, has been shown to lead to a disturbance in functions mediated by the OFC (Rogers et al., 1999, as cited by Blair, 2003). As already seen before, there may be a delay in cortical maturation in several brain areas in boys with callous-unemotional conduct problems (Blair, 2003). Jones and colleagues additionally found that the neural substrates of emotional impairment associated with callous-

unemotional antisocial behavior are already present in childhood (Jones, Laurens, Herba, Barker & Viding, 2009). The cause for later dysfunction of the OFC may be ambiguous; the impairment of neural substrates in psychopaths shows up early in life but their lifestyle may exacerbate these impairments.

### *Psychopathy and emotion processing*

Cleckley believed that the diagnostic symptoms of psychopaths are consequences of a deep-seated affective disturbance. He claimed that a psychopath is not able to experience deep positive or negative feelings (Cleckley, 1976). Lykken (1995) on the other hand stated that psychopaths, as defined by Cleckley, display a specific disturbance in fear reactivity. Psychopaths display for instance a disturbance in passive avoidance learning and they learn only by reward, not by punishment. This could indicate that they do not fear punishment as much as healthy persons do (Blair et al., 2004).

Currently, there are two main positions on the nature of the affective characteristics of psychopathy: the 'fear' position and the 'empathy' position. The fear position is concerned with a lack of fear; it considers the aspects of psychopathy related to stimulation seeking and insensitivity to punishment. The empathy position considers the aspects of psychopathy related to reduced sensitivity to emotional signals of others, particularly sadness and fear (Blair, Colledge, Murray, & Mitchell, 2001). James & Blair (2006) assumed that empathic dysfunction is one of the major features of psychopathy. They distinguished two main forms of empathy: cognitive empathy and emotional empathy. Cognitive empathy, or Theory of Mind, refers to simply knowing how other individuals feel and what they might be thinking. Cognitive empathy is sometimes called perspective taking; this kind of empathy can be useful in for example a negotiation or in motivating people. James and Blair (2006) point out that psychopaths show no impairment in cognitive empathy. Emotional empathy on the other hand can be considered the result of the translation of the non-verbal aspects of the emotional expressions of others. It is potentially reliant on both cortical and sub-cortical processing routes of facial expressions. These routes convey the communication to regions of the brain involved in emotional processing (the amygdala, insula and orbital and ventrolateral prefrontal cortex). These regions together allow a dedicated response to the facial expressions of others. Psychopaths are supposed to have a selective emotional empathy deficit (James & Blair, 2006).

Recently, the fear and empathy positions have been integrated within the expanded, neurocognitive Violence Inhibition Mechanism model (VIM) (Blair et al., 2001). The VIM is thought to be an innate mechanism for the control of aggression, typically activated by the sad and fearful expressions of others. The VIM posits that reduced autonomic responses to distress cues may result from deficits within the VIM. At its simplest, the VIM is thought to be activated whenever distress cues, the sad and fearful expressions of others, are displayed. This activation results in increased autonomic activity, attention and activation of the brainstem threat response system, usually resulting in freezing. Hence, activation of the VIM results in the interruption of on-going (aggressive) behavior. According to the model, moral socialization occurs through the pairing of the activation of the mechanism by distress cues with representations of the acts that caused the distress cues. These distress cues are moral transgressions (the exceeding of due bounds or limits) (Blair, 1995 as cited by Blair et al., 2001). A process of classical conditioning results in these representations of moral transgressions becoming triggers for the mechanism. The appropriately developing child initially finds the pain of others' aversive. Through socialization, these children find thoughts of acts that cause pain to others aversive as well. It is thought that a failure in the conditioning process is the fundamental cause of the difficulty of the psychopathic individual to be

socialized. The amygdala is crucial for the formation of emotional classical and instrumental associations. Therefore, the suggestion is that the amygdala is crucial for associating the aversive unconditioned stimulus of another's distress with representations of the act that have caused that distress. It is this process that allows socialization. Deficits within the VIM are therefore supposed to result from a more general amygdala dysfunction. These deficits within the VIM may lead to the development of aggressive behavior particularly seen in psychopaths (Blair et al., 2001).

Recently, there has been a lot of research regarding emotional processing in psychopaths. Hoff, Beneventi, Galta and Wik (2009) compared fMRI-BOLD (functional MRI-blood oxygen level dependent) responses from a psychopathy group to healthy controls. In a block design, subjects were exposed to drawings of facial expressions alternated with scrambled drawings. The face drawings consisted of a circle with two dots as the eyes, eyebrows, and mouth line. The control stimuli were created by scrambling the features in the face circle, so that they did not give an impression of a face or a face like expression. The fMRI observations of psychopaths were markedly different from those of the controls. Facial expressions activated a higher number of brain regions (a total of 24; 18 in the left and six in the right hemisphere) in the psychopaths than controls (a total of eight; three in the left and five in the right hemisphere). Moreover, the activation pattern in the psychopaths included broad activation of older brain regions, which may have relevance for implicit stimuli processing. The regional activation in the psychopaths included the cerebellum, the left insula, the left thalamus, the left putamen, the left cingulate, the right caudate body, the left medial frontal gyrus, and the right substantia nigra, whereas the eight regions activated in the controls were all neocortical. This might indicate that high-level cognitive functions were less active in the psychopath's processing of facial expressions. Thus, facial expression could be more of a primeval character in psychopaths (Hoff et al., 2009). Despite obvious differences, no firm conclusions can be made due to the case-report paradigm used.

Patrick, Bradley and Lang (1993) tested non-psychopaths including college students, and psychopaths. They measured startle-elicited blinks during presentations of affective slides to test emotional responding in psychopaths. The non-psychopaths showed a significant linear relationship between slide valence and startle magnitude, with startle responses being largest during unpleasant slides and smallest during pleasant slides. This effect was absent in psychopaths. Psychopathy on the other hand, had no effect on autonomic or self-report response to the slides. These results suggest once more an abnormality in the processing of emotional stimuli by psychopaths of which they are not aware themselves. The startle reflex is mediated by subcortical defense mechanisms, so this study supports the findings by Hoff and colleagues (2009) that the neural processing of facial expression could be more of a primeval character in psychopaths. A very prominent finding of the study by Patrick et al. (1993) is that psychopaths who only displayed the antisocial behavior characteristics of psychopathy, showed in general a normal relationship between slide valence and startle magnitude. This may imply that psychopaths who differ in the personality traits of psychopathy can be distinguished by their reactions to emotional stimuli.

Deeley et al. (2006) used event-related fMRI for comparing six people scoring high on the PCL-R with nine non-psychopathic controls during an implicit emotion processing task using fearful, happy and neutral faces. The psychopathy group showed significantly less activation than the control group in the fusiform and extrastriate cortices when processing happy and fearful emotions. However, type of emotion affected the response pattern. Both groups of people showed increased fusiform and extrastriate cortex activation when processing happy faces compared with neutral faces, but this increase was significantly smaller in the psychopathy group. Moreover, when processing fearful faces compared with

neutral faces, the control group showed increased activation but the psychopathy groups showed decreased activation in the fusiform gyrus. It may be concluded, at least from these data, that psychopaths have biological differences from controls when processing facial emotional expressions, and that the response pattern differs according to emotion type.

Verona, Patrick, Curtin, Bradley and Lang (2004) investigated physiological reactions to emotional sounds in prisoners selected according to scores on the two original factors of the PCL-R. Psychopaths high on the emotional-interpersonal factor, regardless of scores on the social deviance factor, showed diminished skin conductance responses to both pleasant and unpleasant sounds, suggesting a deficit in the action mobilization of emotional responses. Psychopaths who scored high only on the social deviance factor showed a delay in heart rate differentiation between affective and neutral sounds. These findings may indicate abnormal reactivity to both positive and negative emotional stimuli in psychopaths.

A major result of the fMRI study by Müller et al. (2003) is that when compared with normal subjects, psychopaths show a highly significant increase in BOLD response in the right amygdala (not in the left amygdala) while viewing negative emotional pictures. They also found increased activation through negative contents in the right-sided prefrontal regions. Activation was reduced right-sided in the subgenual anterior cingulate and the medial temporal gyrus, and left-sided in the dorsal anterior cingulate and the parahippocampal gyrus. Increased activation through positive contents was found left-sided in the orbitofrontal regions. Activation was reduced in right medial frontal and medial temporal regions. Unfortunately, the sample size of this study was small (6 psychopathic males and 6 controls), so the results have to be interpreted with caution.

Another study by Marsh et al. (2008) found that callous and unemotional traits are associated with reduced amygdala response to distress-based social cues. They also used fMRI to assess amygdala activation patterns during processing of fearful facial expressions. The amygdala activation was reduced relative to healthy comparison subjects and youth with ADHD while processing fearful expressions, but not to neutral or angry expressions. The results of this study also suggest that symptom severity in the callous-unemotional traits was negatively correlated with the connectivity between amygdala and ventromedial prefrontal cortex.

Adolphs and colleagues (2005 as cited by Han, Alders, Greening, Neufeld & Mitchell, 2011) have shown that the fear-recognition deficit observed in patients with amygdala lesions is associated with a failure to attend to the eye region of faces, and can be reversed by instructing the patient to focus on the eyes. Despite the potential utility of attention as an empathy arousal mechanism, it remains unclear whether this manipulation is associated with recovery of function in neural regions considered critical for empathy, or reflects compensatory patterns of activity that may not have the same implications for supporting prosocial behavior.

The study by Han et al. (2001) begins to address this question. Han et al. (2011) examined the impact of isolating distinct regions of the face (i.e. the eyes versus the remaining facial features) on activity in empathy-related brain regions in a community sample of individuals with high versus low levels of callous traits. One possibility given by Han et al. (2011) is that isolating the eyes acts to enhance empathy in individuals with high callous traits, and so will be associated with enhanced activity in empathy-related brain regions including the amygdala and medial prefrontal cortex. Alternatively, isolating the eyes may result in compensatory engagement of other neural regions implicated in cognitive control or attention, such as dorsal prefrontal and parietal areas (regions not traditionally associated with prosocial behaviors). A third possibility is that rather than representing the level of emotion expressed, the amygdala may act to direct processing resources toward the most salient

elements of a stimulus in order to resolve ambiguity. According to this perspective, amygdala activity should be greatest in healthy individuals when the most ambiguous facial features are present. Thus, on the basis of this view, the prediction can be made that any existing functional amygdala abnormalities associated with high relative to low callous traits should be most apparent when viewing fearful faces with the eyes occluded. Their participants completed an emotion recognition task that varied whether the most or least socially meaningful facial features were visible: the eyes were isolated or occluded. They found that the individuals high in callous traits showed an inverse pattern of activity relative to the low callous trait group, with greater activity in the amygdala and prefrontal cortex for the most relative to the least-informative (eyes isolated or occluded) of fearful and happy faces. Han et al. (2011) suggest that these empathy-related regions may have failed to adapt to emotional ambiguity by seeking out the most socially meaningful stimulus components. They also observed overall (in both conditions) reduced activity in dorsal prefrontal cortex and inferior parietal lobe in the individuals high in callous traits. This was unexpected, because these areas are supposed to be functionally intact in psychopathy. They assume that it is likely that the observed abnormalities resulted from a downstream effect of dysfunction in the amygdala and medial prefrontal cortex. They give the following explanation: "In the low callous trait group, attention-related areas like the inferior parietal cortex may be recruited in response to emotional conflict signaled by frustrated attempts of the amygdala and medial prefrontal cortex to locate disambiguating information. In contrast, the high callous trait group may not have generated these emotional conflict-related signals in response to the least informative conditions and, as a result, showed relatively less recruitment of secondary attention-related regions." (Han et al., 2011). The effects observed in the amygdala do not implicate that the amygdala is specific to represent or embody fearful stimuli in the context of emotion recognition. The results instead support the theory that the amygdala helps to orient attention to cues necessary to disambiguate a stimulus. Other regions outside the amygdala may also play a role in orienting attention to socially meaningful cues, because the medial prefrontal cortex and a frontoparietal attention network were also activated to a greater extent in the low callous trait group when these cues were occluded (Han et al., 2011).

Patrick, Cuthbert and Lang (1994) tested the hypothesis that the response mobilization that normally accompanies imagery of emotional situations is deficient in psychopaths. They found that psychopaths and non-psychopaths did not differ on self-ratings of fearfulness, imagery ability, or imaginary experience. Non-psychopaths however, showed in this study larger physiological reaction during fearful imagery than psychopaths. In naturalistic settings, an affective imagery deficit would be manifested as a failure to review the harmful consequences of one's actions and as an inability to entertain new behavioral strategies. This helps to account for the reckless, impulsive life-styles of psychopaths. It also helps to explain why verbally oriented approaches to treatment, which rely on language-affect connections, are so ineffective with this population.

All these findings from different studies underline that psychopathy is indeed neurobiologically reflected by dysregulation and disturbed functional connectivity of emotion-related brain regions. It may be concluded that psychopaths, as compared with nonpsychopaths, process emotions differently. The next question is whether psychopaths are able to recognize facial expressions of emotions.

#### *Recognition of facial expressions of emotions by psychopaths*

Several studies examined whether psychopaths are able to recognize facial expressions like normal people do. Some results of these experiments are homologous, while others contradict or complement each other. While most studies investigating facial affect



recognition by psychopaths have found some deficit(s), the specific type of deficit varies across studies and samples.

A meta-analysis by Marsh and Blair (2008) shows a consistent, robust link between antisocial behavior and impaired recognition of fearful facial affect. Relative to comparison groups, antisocial populations showed significant impairments in recognizing fearful, sad, and surprised expressions. They were not reliably impaired in recognizing happiness, anger, or disgust expressions. Thereby, deficits for recognizing fear appeared to be significantly greater than for any other expression. It should be noted however, that fear is considered to be the most difficult expression to recognize, whereas sadness is usually considered one of the expressions easiest to recognize. As stated before, fear recognition relies disproportionately on the amygdala (Anderson & Phelps, 2000; Calder et al., 2001). Sampling participants on the basis of their fear recognition ability shows that reduced ability to identify fearful expressions is associated with amygdala hyporesponsivity (Corden, Critchley, Skuse, Dolan, 2006 as cited by Marsh & Blair, 2008). Together, this evidence supports the association between specific fear recognition deficits and amygdala dysfunction.

It should be emphasized that identifying any emotional expression, including fearful expressions, is a complex task that requires visual scanning, perceptual processing, effortful attention, working memory, and semantic processing. Accordingly, such processing relies on a large, distributed network of neural structures. At the most basic level, intact functioning of occipitotemporal visual cortex is required to process the geometric configuration of the features of the face (Allison, Puce, Spencer, & McCarthy, 1999, as cited by Marsh & Blair, 2008). The superior temporal gyrus and fusiform gyrus (part of the inferior temporal cortex) play central roles in processing faces (LaBar, Crupain, Voyvodic, & McCarthy, 2003 as cited by Marsh & Blair). As already said, when processing fearful faces compared with neutral faces, the control group showed increased activation but the psychopathy groups showed decreased activation in the fusiform gyrus (Deeley et al., 2006). Once configural features have been assessed, intact functioning of structures in the temporal lobes is required to link the configural properties of facial expressions with stored knowledge about what those properties represent (Haxby et al., 2002 as cited by Marsh & Blair, 2008). Dysfunction in any of these structures may lead to individuals who show generalized impairments in processing facial emotion. Finally, subcortical routes that involve the thalamus and superior colliculus appear to be involved in processing emotional expressions, particularly fearful expressions (Luo, Holroyd, Jones, Hender, & Blair, 2007 as cited by Marsh & Blair, 2008). Dysfunction in these regions could exist among antisocial populations.

Marsh and Blair (2008) also found deficits in sadness recognition are associated with antisocial behavior. This is theoretically consistent in that sadness, like fear, is a distress cue. Similar neural structures, including the amygdala, are associated with processing fear and sadness expressions (James & Blair, 2006). These findings are also consistent with the VIM model, in that psychopaths have difficulty attributing fear and sad facial affects (Blair et al., 2001).

Blair et al. (2001) found that children with psychopathic tendencies have specific impairments. In their experiment, psychopathic children needed significantly more tries before they could successfully recognize the sad expressions and even when the fearful expressions were at full intensity, they were significantly more likely to misclassify fear as one of the other five basic emotions. They also found that the amygdala-mediated expressions were not significantly more difficult to process than the nonamygdala mediated expressions by the normal comparisons (Blair et al., 2001).

Fairchild, van Goozen, Calder, Stollerey, & Goodyer (2009) also found a link between psychopathy and impairment in the recognition of fear and sadness, but they also found

impairment in surprise recognition. They did not examine children, but male adolescents with conduct disorder and psychopathic traits. The results of this study and the study by Blair et al. (2001) taken together, suppose that psychopathy is a neurocognitive disorder that is apparent across the lifespan, because adult psychopaths show the same impairments as children in this study.

Other studies found additional specific deficits. Hastings, Tangney and Stuewig (2008), for instance found that psychopathy was associated with overall difficulty identifying facial expressions, as well with a specific deficit in identifying happy and sad facial expressions. In addition, psychopathy was associated with difficulty identifying less intense facial displays of emotion. Their last finding was that neither the affective/interpersonal features nor the antisocial lifestyle features of psychopathy were uniquely related to these deficits above and beyond that of psychopathy as a whole. These findings are partially positive of the VIM model in finding significant deficits in attributing sad facial expressions. However, no such deficits were found for angry and shame expressions and the observed deficits in identifying happy expressions are not expected in this model. Results of this study are consistent with previous work by Blair et al. (2004) in finding deficits in identifying less intense displays of emotion.

Blair et al. (1995) also examined the emotion attributions made by psychopaths and non-psychopathic controls. Their study revealed that the emotion attributions of both groups are very similar when the conditions elicited happiness, sadness or embarrassment attributions. The emotion attributions differed significantly however, when the conditions elicited guilt. This finding was predicted by Blair's VIM model. According to this model, guilt and other moral emotions are products of cognitive interpretation of the arousal generated by the activation of the Violence Inhibition Mechanism. Psychopaths, when assuming they have suffered early dysfunction within the VIM, should respond to guilt as controls would. The present findings indicate that psychopaths have a specific difficulty in understanding this emotion. It should be noted however, that there are other, non-affective interpretations of the present findings. For example, psychopaths are more likely to have been involved in violent situations (Hare, 1996). The relative lack of guilt attributions by the psychopaths may reflect their experience of these situations rather than an inability to feel guilt. However, there is evidence to suggest that psychopaths are not emotionally aroused by another's distress (Cleckley, 1976).

Kosson, Suchy, Mayer and Libby (2002) found that psychopathic offenders showed deficits in facial affect recognition only under specific circumstances. Their participants were asked to classify the emotional expression on each of 30 adult male and female Caucasian faces. The six categories were happy, sad, angry, afraid, surprise, and disgust. In particular, they found evidence that deficits were specific to the classification of facial disgust and to conditions designed to minimize the involvement of left-hemisphere mechanisms. These findings fit with the report that, unlike nonpsychopaths, psychopaths failed to show startle potentiation while viewing affective slides depicting mutilation (Patrick et al., 1993). Both the affective modulation of startle responses and the experience of disgust have been linked to right-hemisphere mechanisms (Kosson et al., 2002). Their results do not provide evidence that psychopaths are deficient in classifying fear or sadness, as found by other studies described before. However, they note themselves that their study has important limitations regarding the identification of sadness: "Although we were successful at producing a discriminating task overall (i.e., collapsing across all six emotions), it appears that the current task did not provide a sensitive enough measure for obtaining individual differences in the ability to categorize certain specific emotions." (Kosson et al., 2002). Nevertheless, with respect to fear, the absence of group differences does not appear to reflect limited variance or

low power.

Pham & Philippot (2010) investigated the possibilities of general or specific deficits in psychopaths' decoding of facial expression of emotion. They specifically tested the amygdala dysfunction hypothesis proposed by Blair et al. (2001). According to this hypothesis, psychopaths are specifically impaired in processing the recognition of facial expressions of sadness and fear (amygdalian emotions) but not of the recognition of happiness, anger, and disgust (nonamygdalian emotions). They found that the amygdalian nature of emotions only significantly modulated criminal non-psychopath's accuracy: criminal non-psychopaths were more accurate than criminal psychopaths in decoding nonamygdalian emotions, but no differences emerged regarding amygdalian emotions. The differences in accuracy between the two criminal groups were fully accounted for by their difference in terms of psychopathy. Overall, the healthy controls performed better than the two criminal groups. Interestingly, the healthy controls tended to report more difficulties, especially for weak intensity emotions. In contrast, the criminal participants, and particularly the psychopaths for high intensity displays, reported less difficulties, while their objective performance was lower than that of controls. Obviously, the criminal groups, and especially the psychopaths, did not perceive that they suffer from a deficit in decoding facial expressions of emotions. To the contrary, compared to normal controls, they over-estimated their ability. In sum, according to this study, psychopathy does indeed modulate the ability to decode amygdalian versus nonamygdalian emotions. The pattern of the modulation observed however, is not consistent with the one reported by Blair et al. (2001), but the other way around. It should be noted that this study examined criminal psychopathic adults, while other studies also examined children with psychopathic tendencies. According to this study, there is no evidence of a severe, clinically significant, bias or impairment in the processing of emotional facial expressions in adult criminal psychopaths.

Woodworth and Waschbusch (2007) examined children with callous-unemotional traits. The children were shown 18 pictures that included six photographs of a male face, six photographs of a female face, and six drawings of a cartoon face. Each of the three sets of six faces (i.e. six male faces, six female faces, six cartoon faces) depicted one of the following emotions: anger, disgust, fear, happiness, sadness or surprise. The participants were instructed to identify what emotion the face expressed. Woodworth and Waschbusch (2007) found deficits regarding attributing emotions of sadness in children with callous-unemotional traits. This is consistent with several studies mentioned before. However, they also found that children with high scores on callous-unemotional traits were more accurate than controls in identifying fear expressions, and that children with high conduct problems and low callous-unemotional traits were less accurate in identifying fear expressions. Therefore, these results provided some evidence (although these were trend effects) that callous-unemotional traits actually improve the psychopaths' ability to identify the facial expressions of fear, while youth with a high level of conduct problems (in the absence of a high level of callous-unemotional traits) were less able to identify the facial expression of fear. Overall, this is not consistent with most of the literature described above, with an exception for the study by Kosson et al. (2002). One potential explanation for the finding that higher callous-unemotional traits were associated with more accurate perceptions of fearful expressions is that there is something specific to fear recognition for individuals with more psychopathic, e.g. callous-unemotional, traits that actually make them more successful for observing or recognizing fearful expressions. This would seem to be beneficial of facilitating an ability to prey on, persuade and manipulate others, as Cleckley described in 1976. This recognition does not prevent them from having an inability to being negatively affected by fearful effect in others (e.g. not freezing or not ceasing their aggressive behavior), or having an inability to

comprehend what fear actually constitutes. They may instead recognize fear for their own purposes but processing or understanding it at a more fundamental level (or even perhaps experiencing it themselves) is lacking (Woodworth & Waschbusch, 2007). This explanation is consistent with the VIM model in that psychopathic individuals may not have a deficit in the recognition of a fearful expression per se, but rather a deficit in using fearful expressions as submissive cues to suppress their aggressive behavior. In fact, for psychopaths, fearful cues may actually signal an increased vulnerability and susceptibility to be preyed upon that may actually facilitate their aggressive behavior. They also found that children high in callous-unemotional traits were significantly less accurate in identifying sad facial expressions, just like some studies described above. They give a plausible explanation for this discrepancy: “while recognition of fear may actually prompt an increase in aggressive behavior, the expression of sadness is not as salient for identifying a potential weakness or vulnerability and does not serve as a predatory cue for individuals high in callous-unemotional traits. Therefore, considering their deficit in empathy, sadness may not serve the same function (to potentially facilitate predatory behavior), and is not as readily considered or processed in the same manner as less psychopathic individuals.” (Woodworth & Waschbusch, 2007).

Taken all these results together, one can conclude that psychopaths have certain deficits in recognizing facial expressions of emotions. The recognition of both fear and sadness are most likely to be impaired, however the studies are not mutually conclusive. More specific deficits are also found, e.g. the deficits in the recognition of surprise, happiness and guilt. However, more research is needed before firm conclusions can be drawn.

### **The role of mimicking in recognizing emotional facial expressions**

Darwin stated that emotions are evolved and adaptive and serve an important communicative function. He therefore proposed that facial expressions have a biological basis (Hess & Thibault, 2009). Consistent with this proposition, it has been suggested that they are controlled by particular ‘facial affect programs’ (Tomkins, 1962). It has also been suggested that humans are predisposed to react emotionally to facial stimuli, and in particular to have facial reactions to facial expressions (Dimberg, 1982).

If facial expressions are indeed generated by biologically given affect programs, one would expect that these programs operate automatically by eliciting facial muscle reactions spontaneously, quickly, and independently of conscious cognitive processes (Ekman, 1992). It has been found that when people are exposed to pictures of emotional facial expressions, they spontaneously and rapidly react with distinct facial EMG reactions in muscles relevant for positive and negative emotional displays (Dimberg, 1982). Pictures of happy faces usually evoke a spontaneous increase in zygomatic muscle activity whereas angry faces usually evoke a spontaneous increase in corrugator supercilii (CS) muscle activity, typically after only 500 ms of exposure. The zygomatic muscle (ZM) is the basis of a smile, whereas the corrugator muscle knits the eyebrows during a frown. It has been reported that these muscles more generally distinguish between positive and negative emotional reactions (Dimberg & Thunberg, 1998, as cited by Dimberg, Thunberg, & Elmehed, 2000).

Another characteristic of an automatic reaction, besides being spontaneous and rapid, is that it can occur without attention or conscious awareness. Zajonc (1980, as cited by Dimberg et al., 2000) proposed that the initial response to affective stimuli could be generated without conscious cognitive processes. Dimberg et al. (2000) found that the initial facial reactions are controlled by rapidly operating affect programs that can be triggered independently of conscious cognitive processes. It is thus possible to unconsciously evoke a physiological response that is more than an attention-arousal response (e.g. an aversively conditioned skin conductance response to angry faces). The distinct positive and negative

facial emotional response patterns can be spontaneously evoked without awareness of the positive and negative eliciting stimuli. These results support the proposition that important aspects of emotional face-to-face communication can occur on an unconscious level (Dimberg et al., 2000). However, from these results it is not clear to what degree the different facial reactions originate in unconscious mimicking behavior, or to what degree the facial reactions are initially readouts of underlying emotional states. In fact, one could argue that the response to a happy picture could be well confounded by mimicry and a reciprocating response, both resulting in increased zygomatic activity and thus a smile. Furthermore, the corrugator response to angry targets could be anger, which could be confounded by mimicry and a reciprocating response, but the corrugator response could also be a fear response.

Mimicry is also called the chameleon effect, and it refers to the unconscious imitation of postures, facial expressions, mannerisms, and other verbal and nonverbal behavior (Guéguen, 2011). As seen above, behavioral mimicry is an automatic social behavior; it refers to changing ones' behavior unintentionally in order to match that of the other person in a social interaction. It has been suggested that mimicry communicates affiliation, liking of, and rapport with the mimicked interaction partner (Vrijesen, Lange, Becker, & Rinck, 2010). This relation between mimicry and rapport is bi-directional; being mimicked creates an affiliation with the interaction partner, and individuals are more inclined to mimic a person they like better. It has been found that people react more positively to a mimicking virtual man than to a virtual man that did not mimic (Vrijesen et al., 2010).

Stel et al. (2010) pointed out that third-party observers make judgments about individuals' competence on the basis of their decisions whether and whom to mimic. As also stated by Vrijesen et al. (2010), mimicry is not necessary beneficial to the mimicker, people who mimicked an unfriendly or disliked model were rated as less competent than people who did not mimic. Moreover, Stel et al. (2010) found that when people are asked to mimic a disliked person, mimicry did not increase liking for that person, whereas liking increased when people 'intentionally mimicked' a liked target. Intentionally mimicking however, is not really mimicking but mere acting because it is neither automatic nor spontaneous. The effects of a priori liking and mimicry on liking were mediated by the individual's affiliation with the target. According to these results, a positive reputation depends not only on the ability to mimic, but also on the ability to discriminate whom not to mimic.

### *Neurobiology of mimicking*

Unlike most species, humans are able to learn from imitation, and this faculty is at the basis of human culture. A neurophysiological mechanism, the mirror-neuron mechanism, appears to play a fundamental role in both imitation and action understanding (Rizzolatti & Craighero, 2004). Mirror neurons are a particular class of visuomotor neurons, which are originally discovered in area F5 of the monkey premotor cortex, that discharge when monkeys perform a particular action and when it observes another individual (monkey or human) performing a similar action. In order to be triggered by visual stimuli, mirror neurons require an interaction between a biological effector (hand or mouth) and an object. The sights of an object alone, of an agent mimicking an action, or of an individual making intransitive (nonobject-directed) gestures are all ineffective. An important functional aspect of mirror neurons is the relation between their visual and motor properties. Virtually all mirror neurons show congruence between the visual actions they respond to and the motor responses they code (Rizzolatti & Craighero, 2004).

Two main hypotheses have been advanced on what might be the functional role of mirror neurons. The first is that mirror-neuron activity mediates imitation; the second is that mirror neurons are at the basis of action understanding (Rizzolatti et al. 2001, as cited by

Rizzolatti & Craighero, 2004). Rizzolatti and Craighero (2004) consider these two hypotheses most likely correct. However, they note that two points should be specified. First, although they are fully convinced that the mirror neuron mechanism is a mechanism of great evolutionary importance through which primates understand actions performed by their conspecifics, there is no evidence to claim that this is the only mechanism through which actions done by others may be understood. Second, the mirror-neuron system is the system at the basis of imitation in humans. Imitation is however no very primitive cognitive function. There is a vast agreement among etiologists that imitation, the capacity to learn to perform an action from seeing it being performed, is only present in humans and probably in apes. Therefore, the primary function of mirror neurons cannot be action imitation. On the other hand, the proposed mechanism in how mirror neurons mediate understanding of actions performed by others is rather simple. Each time an individual sees an action performed by another individual, neurons that represent that action are activated in the observer's premotor cortex. This automatically induced motor representation of the observed action corresponds to that which is spontaneously generated during active action and whose outcome is known to the acting individual. The mirror system thus transforms visual information into knowledge (Rizzolatti et al., 2001, as cited by Rizzolatti & Craighero, 2004).

Direct evidence for the existence of mirror neurons in humans is lacking, there is however a rich amount of data proving, indirectly, that a mirror-neuron system does exist in humans. Evidence of this comes from neurophysiological and brain-imaging experiments. Neurophysiological experiments demonstrate that when individuals observe an action performed by another individual their motor cortex becomes active, in the absence of any overt motor activity. Cochin et al. (1998) observed several times that the desynchronization of an EEG rhythm recorded from central derivations (the so-called mu rhythm) not only occurs during active movements of studied subjects, but also when the subjects observed actions performed by others. Their study showed that the desynchronization during action observation includes rhythms originating from the cortex inside the central sulcus (Cochin et al., 1998, as cited by Rizzolatti & Craighero, 2004). Furthermore, transcranial magnetic stimulation (TMS) studies indicate that a mirror-neuron system (a motor resonance system) exists in humans and that it possesses important properties not observed in monkeys. First, intransitive meaningless movements produce mirror-neuron system activation in humans, whereas they do not activate mirror neurons in monkeys. Second, the temporal characteristics of cortical excitability during action observation, suggest that human mirror-neuron systems code also for the movements forming an action and not only for the action itself as monkey mirror-neuron systems do. These two differences in properties of the mirror-neuron system should play an important role in determining the humans' capacity to imitate others' action (Rizzolatti & Craighero, 2004).

A couple of studies show that the observation of actions performed by others activates a complex network formed by occipital, temporal, and parietal visual areas, and two cortical regions whose function is fundamentally or predominantly motor (e.g. Decety, Chaminade, Grezes, & Meltzoff, 2002 as cited by Rizzolatti and Craighero, 2004). These two last regions are the rostral part of the inferior parietal lobe and the lower part of the precentral gyrus plus the posterior part of the inferior frontal gyrus (IFG). These regions form the core of the human mirror-neuron system (Rizzolatti & Craighero, 2004).

Achaibou, Pourtois, Schwartz, & Vuilleumier (2008) combined simultaneous EEG and EMG recordings to assess the pattern of neural activation associated with involuntary mimicry of emotional facial expressions. By tracking the time-course of both EEG and EMG responses to dynamic emotional faces, they were able to identify processing stages that were differentially activated as a function of mimicry intensity on a trial-by-trial basis, i.e. when

covert facial imitation was higher as compared to when covert facial imitation was lower. Their study indicates that facial mimicry can be repeatedly elicited over many successive trials, supporting a strong degree of automaticity for this phenomenon, and establishing an opportunity for repeated measurements of the concomitant neural activity. Secondly, their EMG data revealed temporal dynamics for CS and ZM responses. The CS activity was enhanced in response to angry faces as soon as 200 ms after stimulus onset, while ZM activity was increased in response to happy faces after 500 ms only. This contrasts with the findings of Dimberg and Thunberg, 1998, who found similar latencies for both expressions, but this is likely due to the fact that Achaibou et al. (2008) used a different set of face stimuli with dynamic expressions whereas Dimberg and Thunberg used static pictures, and because their paradigm did not evoke an early emotion-independent increase in CS activity, which was present across all conditions in Dimberg and Thunberg's study. A more rapid onset of the angry CS response, relative to the happy ZM response, might reflect faster processing of negative, threat-related signals or some intrinsic advantage for the innervations of the upper facial musculature. They did not find any asymmetry in muscular activity for the left and right side of the face for both emotion expressions. The most novel aspect of their study is the link between brain activity and intensity of facial mimicry. It turned out that this link was stronger for intense mimicry of happy faces (with greater ZM activity) than for intense imitation of angry faces (greater CS activity). However, they found no reliable correlation between the intensity of facial mimicry in EMG and empathy level (Achaibou et al., 2008). The early increase (200 ms) in corrugator response in Dimberg's studies may not be reliable. In addition, the greater CS activity could be due to a sudden transition from a dark or unlit computer screen to a bright projected picture and is therefore probably a little startle response in the corrugator.

Harrison et al. (2010) state that the anatomical connectivity between amygdala, cingulate motor cortex (M3, M4) and the facial nucleus demonstrate a potential neuroanatomical substrate for mimicry. They found just like Achaibou et al. (2008) a relationship between intensity of facial mimicry and trait emotional empathy. Thereby, they found a specific relationship between upper (CS) but not lower (ZM) facial mimicry and trait emotional empathy. The corrugator but not the zygomatic mimicry thus predicts trait empathy, which is consistent with greater anatomical connectivity between the amygdala and M3 coding upper facial muscle representations. Their results also show that norepinephric modulation, in contrast to its effects on emotion perception, does not modulate emotional mimicry or its relationship with emotional empathy.

Harrison et al. (2010) also state that individuals with disorders of social communication such as Asperger's syndrome and conduct disorder show a selective impairment in mimicry responses in the upper face; the CS. Interestingly, this upper facial mimicry predicts trait empathy. Harrison and his colleagues therefore suppose that the tendency to mimic others' emotional expressions predicts empathy and may represent a physiological maker of psychopathy.

### *Psychopathy and mimicking*

Lipps (1905, as cited by de Wied, van Boxtel, Zaalberg, Goudema & Matthys, 2006) suggested that attention was the possible role of motor mimicry in the automatic transmission of emotions. Lipps proposed that people tend to mimic the facial, vocal and postural expressions of emotions displayed by an interaction partner, and that such mimicry may evoke matching emotions in the observer. Today, many researchers suppose that motor mimicry is the very essence of emotional empathy and that it is biologically 'hardwired' (de Wied et al., 2006).

Hoffman (2000, as cited by de Wied et al., 2006) expands Lipps ideas by suggesting a developmental model of empathy, which says that mimicry is an early component in the process of empathy. He states that the tendency to automatically mirror emotional expressions manifests itself already in the first days of life by reflexive crying in response to other babies' crying. Mimicry thus contributes to the development of empathy in the early preverbal period, and continues to develop and operate in childhood. When the cognitive system develops, higher-order cognitive processes come to play a more important role, but the mature empathic responses are generated by more sophisticated cognitive processes and the primitive (automatic) mechanisms already present in young children.

Sonnby-Borgström (2002) investigated the link between emotional empathy and mimicking. She examined differences between individuals high and low in emotional empathy. The parameters compared were facial mimicry reactions, as represented by EMG activity when subjects were exposed to pictures of angry or happy faces, and the degree of correspondence between subjects' facial EMG reactions and their self-reported feelings. The comparisons were made at different stimulus exposure times in order to elicit reactions at different levels of information processing. High-empathy individuals showed mimicking reactions at short exposure times and reported feelings that were reflected in their muscular reactions. This result supports the hypothesis that automatic mimicry is an early, automatic element involved in emotional empathy. In contrast, the low-empathy group reacted with increased zygomaticus activity (smiling) when exposed to angry faces and showed a higher level of zygomaticus activity when they reported negative feelings. The high- and low-empathy groups were not characterized by contrasts in self-reported feelings (a controlled, conscious process) when they were exposed to angry and happy faces or by differences in muscle reactions at the controlled level. Accordingly, individual differences in emotional empathy appear to reflect differences in spontaneous somatic reactions based on primary memory systems rather than differences in controlled reactions to the emotional situation based on secondary memory systems. This provides support for the idea that the process involved in emotional empathy is related to automatic, spontaneous reactions rather than a product of controlled cognitive interpretation of the emotional situation (Sonnby-Borgström, 2002).

Sonnby-Borgström, Jönsson, and Svensson (2003) also examined mimicking in individuals high and low in emotional empathy. They distinguished between spontaneous facial reactions and facial expressions associated with more controlled or modulated emotions at different information processing levels, first at a preattentive level and then repeatedly at more consciously controlled levels: their subjects were exposed to pictures at three different exposure times (17, 56, and 2350 ms). They found that in contrast to high empathic individuals' significant mimicking reaction, low-empathy individuals showed no mimicking reaction, neither at the automatic level (56 ms) nor at the controlled level (2350 ms). On the contrary, low-empathy individuals showed a tendency to increased zygomaticus activation, when exposed to angry faces at the controlled level. This last finding is consistent with the results of the study by Sonnby-Borgström (2002).

Skin conductance responses reflect sympathetic arousal, which is generally interpreted as an orienting response to salient, or novel stimuli and can indicate emotional arousal, independent of stimulus valence (Westbury & Neumann, 2008). Westbury and Neumann (2008) found that skin conductance responses (SCR) are greater in moderately empathic individuals than in highly empathic ones. This is interesting because one would expect those higher in the emotional trait empathy to be more affectively reactive to, and oriented towards, empathy-eliciting scenes. They also found that individuals low in empathy showed less corrugator muscle EMG activity than moderate and high empathic individuals as a reaction to



the stimuli presented. The stimuli presented were short film clips depicting humans, primates, quadruped mammals and birds in victimized circumstances.

Low socially anxious individuals regard a mimicking virtual man as more positive than a non-mimicking one, but high socially anxious individuals did not (Vrijnsen, Lange, Dotsch, Wigboldus, & Rinck, in press as cited by Vrijnsen et al., 2010). These results suggest that high socially anxious individuals display a problem with an important social behavior, namely the processing of unintentional mimicry. Vrijnsen et al. (2010) examined this hypothesis and found that high socially anxious people differ from normal controls in displaying a standard, appropriate behavioral response to their interaction partner. They not only lack appreciation of the interaction partner's mimicry, they also show less unintentional mimicry themselves during a one-on-one interaction. They process the interaction partner's behavior, but are not able to respond appropriately. Moreover, Guégen (2011) found that mimicking enhances self-consciousness and reduces social anxiety.

Oppositional defiant disorder (ODD) and conduct disorder (CD) are referred to as disruptive behavior disorders (DBD) in the DSM-IV-TR (DSM-IV-TR 2000). ODD can be a precursor of CD and includes hostility, disobedience, and opposition to authority figures. Although psychopathy is not perfectly identical to such diagnoses but rather an expansion, there are many similarities and therefore DBD children will also be discussed in this paragraph.

De Wied et al. (2006) explored whether DBD boys are less facially responsive to facial expressions of emotions than normal controls. They studied EMG activity in the zygomaticus major and corrugator supercilii muscle regions and heart rate activity during exposure to dynamic happy and angry expressions. They first note that it is important that DBD boys score significantly lower on a self-report measure of emotional empathy than normal controls. This supports the assumption and clinical observations that they are weak empathizers. They also found that angry and happy facial stimuli spontaneously elicit different EMG response patterns. Like the study by Dimberg (1982), they found that angry faces evoke a stronger increase in corrugator activity than happy faces, while happy faces evoke a stronger increase in zygomaticus activity than angry faces. In addition, the corrugator response to angry faces was less distinct for DBD boys than for controls, while no differences between the two groups in the zygomaticus response to happy faces appeared. These results show that DBD boys are less facially responsive than normal controls towards angry faces, i.e. they show less mimicking behavior. De Wied and her colleagues only used angry and happy faces, so it is not certain whether these findings generalize to other negative emotions relevant to empathy and prosocial responding, like fear and sadness. Interestingly, habituation to the angry faces shown is clearly visible in the EMG responses of normal controls, but not the responses in DBD boys. Given the limitations of this study, all these findings must be interpreted with caution.

De Wied, van Boxtel, Posthumus, Goudena and Matthys (2009) examined aspects of emotional empathy across different physiological response systems in clinic-referred DBD boys and normal controls. They monitored facial EMG and heart rate responses during exposure to short film clips involving children experiencing either positive (happiness) or negative (sadness or anger) emotions. By examining anger as well as sadness, they in fact elaborate the study by de Wied et al. (2006). A second goal was to study basal autonomic function in DBD boys versus normal controls, because numerous studies show basal autonomic disturbances in children with externalizing disorders, mainly a low resting heart rate. A low resting heart rate is thought to be driven by sympathetic under activation and is one of the best-replicated biological markers of aggressive antisocial behavior (Lorber, 2004, as cited by de Wied et al., 2009). Their results show that the anger and sadness clips

significantly increased corrugator muscle activity in the normal controls. In DBD boys, the corrugator response was also significantly increased, but was significantly smaller to both sorts of negative clips. Furthermore, the viewing of the positive and negative clips evoked cardiac deceleration in the normal controls while DBD boys showed significantly less cardiac deceleration during sadness. According to these results, DBD boys are less emotionally responsive to others' distress than normal boys, like de Wied et al. also found in 2006. Their results also demonstrate that DBD boys with a high resting heart rate (reflecting high anxiety levels) and low vagal tone (reflecting poor emotional control) show selective impairments in empathy with negative but not with positive emotions.

The two studies mentioned above only examined DBD boys as a single group, but a more recent study by de Wied, van Boxel, Matthys and Meeus (2012) compared male adolescents with disruptive behavior disorders, high and low on callous-unemotional traits. Consistent with the previous studies, they also monitored facial EMG and heart rate responses during empathy-inducing film clips showing sadness, anger or happiness. An expansion in relation to the other studies by de Wied et al. (2006; 2009) is that this study examined autonomic activity together with verbal and facial EMG responses to different target emotions. Their results show that individuals high in callous-unemotional traits report less empathy, show less facial responsiveness, and less heart rate change from baseline during sadness than controls. Furthermore, they present a difference in autonomic (not verbal nor facial) reactions to sadness compared with the low callous-unemotional group. DBD adolescents with high callous-unemotional traits showed significantly less heart rate change from baseline during sadness than those low in callous-unemotional traits. As described before, the VIM model suggests that activation of the VIM results in autonomic arousal and the interruption of on-going (aggressive) behavior. The results are consistent with the VIM model by showing subnormal levels of heart rate reactivity during exposure to sadness in adolescents with high callous-unemotional traits. As mentioned before, the cortical maturation in several brain areas by psychopaths may be delayed, which may be implicated in responses to affective stimuli and the experience of empathy (de Brito et al., 2009). Hence, subnormal heart rate reactivity to sadness by DBD adolescents high in callous-unemotional traits may possibly be related to abnormalities (delayed maturation) in the prefrontal brain regions including the anterior cingulate cortex. Moreover, the DBD adolescents with low callous-unemotional traits showed a more diffuse pattern of empathy dysfunction: they reported less empathic happiness and showed deficits in facial responsiveness to both sadness and happiness. This last finding contradicts earlier work from the Wied et al. (2006; 2009), suggesting that DBD boys are only impaired in empathy with negative (and not positive) emotions. This could be due to a couple of differences between these studies. De Wied et al. (2006; 2009) examined boys instead of adolescents, and this last group was possibly more heterogeneous because an older group is more likely to include both persons with adolescence-limited antisocial behavior and persons with early-onset pathways. Individuals with adolescence-limited antisocial behavior are only antisocial during adolescence while individuals with early-onset pathways are antisocial earlier and their antisociality is present longer in life. Secondly and thirdly, the differences could be due to differences in stimulus material or developmental processes. For example to increasing testosterone levels in older men or changes in frontal lobe maturation, which are both likely to affect empathic behavior (de Wied et al., 2012). Their results regarding baseline autonomic functioning indicate that the DBD individuals high in callous-unemotional traits were characterized by an abnormally low cardiac vagal tone, which is consistent with the studies by de Wied et al. (2006; 2009). All together, the results support the notion that callous-unemotional traits designate a distinct subgroup of DBD individuals (de Wied et al., 2012).

It should be noted that deficits in empathizing are not only a characteristic of psychopathy, but also of autism-spectrum disorders. Results of EMG recordings of the corrugator supercillii muscle revealed reduced automatic mimicry of angry facial expressions in high autistic individuals, but only in females (Hermans, van Wingen, Bos, Putman and van Honk, 2009). Mimicry responses in the zygomaticus major appeared to be less strong in autistic individuals. These findings indicate that the degree to which individuals exhibit spontaneous mimicry may also vary as a function of both gender and autistic traits

Although the results of these studies are not perfect identical to each other, one can assume or conclude that psychopaths exhibit a specific deficit in mimicking facial emotions of others. The last question is whether psychopaths show deviations in facial expressions of emotions and if the emotional disturbances psychopaths exhibit, express themselves via facial expressions of emotions.

#### *Facial expressions of emotions in psychopaths*

As we have seen above, psychopaths process emotions differently, they show deficits in recognizing facial expressions of emotions and they exhibit deficits in mimicking. Unfortunately, there is not much research regarding the facial expressions of emotions of psychopaths outside the content of mimicking.

Keltner et al. (1995) tested hypotheses concerning the relations between externalizing problems and anger, internalizing problems and fear and sadness, and between the absence of externalizing problems and the occurrence of social-moral emotions (embarrassment). They found that adolescents with externalizing problems (both pure and mixed) showed a higher ratio of anger expression than non-disordered adolescents. Pure externalizing adolescents also showed anger expressions of greater duration and intensity. Furthermore, they found that pure internalizing adolescents did show more fear than pure externalizing adolescents. Moreover, non-disordered adolescents showed more embarrassment than the adolescents in the three pathological groups (internalizing, externalizing and mixed), especially compared to adolescents with externalizing problems. These findings provide the first evidence for the claim that adolescent pathology is indeed manifest in distinct facial expressions of emotion. These findings also propose relations between antisocial behavior and deficits in the ability to inhibit or control impulses. First, adolescents with externalizing problems showed a higher ratio of anger than non-disordered adolescents, and they were the least likely to inhibit this antisocial emotion while interacting with the adult examiner. One norm of emotional expression is the inhibition and control of anger in front of authority figures. Externalizing adolescents were not inclined to follow this norm. Second, adolescents who showed more fear were, according to their teachers, less inclined to engage in delinquent and aggressive behavior. Fear could be part of an inhibitor system that enables people to respond to punishment, internalize moral standards, and control antisocial impulses. Finally, externalizing adolescents showed the least embarrassment, an emotion that also reflects inhibitory processes and whose origins are alleged to be in the inhibition of prohibited pleasure. Accordingly, anticipated embarrassment should inhibit antisocial behavior. They suppose that inhibited behavior is part and parcel of the characteristic facial expression of embarrassment, which includes the gaze aversion (which is thought to part social interaction) and the inhibited smile. In this way, the relative absence of embarrassment observed in externalizing adolescents may mark their disinclination to inhibit their emotions and actions according to social morals and conventions. In summary, increased anger and decreased fear and embarrassment may reflect a more general deficit in inhibiting impulses that characterizes individuals prone to antisocial behavior (Keltner et al., 1995).

## Discussion

The purpose of this review is researching whether psychopaths emotionally react in the same way as other people, whether they process their emotions in the same way as normal people and which brain circuits are predominantly involved. The main question is whether psychopaths show abnormalities in the perception of facial expressions of emotions of other individuals.

First the recognition of emotions is discussed. There are several theories proposed and some of them have been proved wrong. The facial feedback hypothesis by Ekman and Friesen states that when we perceive a facial expression, we automatically tense the facial muscles responsible for the expression we perceive. When we become aware of our own muscle tension, we interpret the perceived facial expression. According to this theory, facial expressions are either necessary or sufficient to produce emotional experience (Davis et al., 2009). Keillor et al. proved them wrong by studying a patient suffering from a bilateral facial paralysis who reported normal emotional experience. However, this last study was a case study by an impaired individual so no hard conclusions can be drawn before it is replicated. The second theory about recognition of emotions by Bruce and Young does not explain how dissimilar facial expressions are processed, so that remains unclear.

A large number of different structures participate in recognizing the emotion shown in a face: the occipitotemporal cortices, amygdala, orbitofrontal cortex, basal ganglia, and right parietal cortices, among others. These structures are engaged in multiple processes at various points in time, making it difficult to assign one single function to a structure (Adolphs, 2002). The amygdala seems to be principally involved in processing negative emotions whereas damage to the OFC can result in impaired recognition of facial expression of emotions and recognition of impaired emotions from the voice. Moreover, the prefrontal cortex may play a role in the recognition of anger (Adolphs, 2002).

A couple of studies indicate that the amygdala is indeed dysfunctional in psychopaths (Patrick, 1994, as cited by Blair 2003; Tiihonen et al., 2000; Kiehl et al., 2001). These studies used small sample sizes, which raises the possibility that the observed effects may be sample specific. Thereby, in the study by Kiehl et al. (2001) it cannot be ruled out that history of substance abuse may have contributed to the findings. However, taken these studies together, one can conclude that there is an amygdala dysfunction in psychopaths. The orbitofrontal cortex may also be dysfunctional in psychopaths as indicated by several studies (De Brito et al., 2009; Raine et al., 2000). On the contrary, Blair (2003) states in his review that neuropsychological studies with psychopaths, unlike studies with violent individuals, have repeatedly found frontal functioning to be intact. Clearly, more research is needed with 'pure' psychopaths to study the role of the frontal cortex. The results of de Brito et al. (2009) must be interpreted with caution; they only used twin boys, so their results may not be generalized to girls. However, this could be due to the fact that there are more men than women with psychopathy. This could be due to differences in plasma levels of testosterone. Furthermore, their group was not defined by an expert rated questionnaire or structured clinical psychiatric interview, so there is no detailed information about the features of their participants. Raine et al. (2000) examined only 21 men with a diagnosis of antisocial personality disorder, which is not identical to psychopathy. It is possible that these limitations account for the differences found between these three studies.

There appears to be a delay in cortical maturation in several brain areas in psychopaths (De Brito et al., 2009; Blair, 2003), which may be associated with impairments in decision-making, morality and empathy exhibited by psychopaths. The Violence Inhibition Mechanism proposed by Blair is also thought to be dysfunctional in psychopaths, as supported by several studies (e.g. Hastings et al., 2008; Blair et al., 1995; Woodworth & Waschbusch, 2007; de

Wied et al., 2012). Overall, it can be concluded that psychopaths have biological differences as compared to controls when processing facial emotions, and the pattern of response differs according to emotion type.

There exists some discord regarding the results concerning the recognition of facial expressions of emotions by psychopaths. There are studies supporting a link between antisocial behavior and the recognition of fear (Marsh & Blair, 2008; Blair et al., 2001; Fairchild et al., 2009; Hastings et al., 2008; Kosson et al., 2002) but some do not (Kosson et al., 2002; Woodworth & Waschbusch, 2007). Woodworth and Waschbusch (2007) even found that children high on callous-unemotional traits were more accurate than controls in identifying fearful expressions, but these were only trend effects. Some studies suppose that psychopaths have a deficit attributing sadness (Marsh & Blair 2008; Blair et al., 2001; Fairchild et al., 2009; Hastings et al., 2008) while one does not (Blair et al., 2005). There are more deficits found, e.g. deficits in the recognition of surprise (Marsh & Blair, 2008; Fairchild et al., 2009), happiness (Hastings et al., 2008), and guilt (Blair et al., 1995). A limitation in the study by Hastings et al. (2008) is that identifying fearful versus surprised facial expressions significantly confused participants. Participants overwhelmingly misidentified fearful facial expressions with surprised facial expressions. It is not clear what deficits may or may not have been found when 'surprise' had not been included as a response option. However, it is not clear if this is a limitation of the study by Hastings et al. (2008). Namely, this misidentification of fearful facial expressions with surprised facial expressions follows earlier work showing that fear is certainly considered to be the most difficult expression to recognize (although sadness is usually considered one of the easier expressions to recognize) (Ekman & Friesen, 1976 as cited by Blair et al., 2001). Furthermore, while Blair et al. (2001) found that psychopaths show impairments in recognizing amygdala-mediated emotions compared with nonamygdala-mediated emotions, Pham & Philippot (2010) could not replicate these results. The study by Pham & Philippot (2010) is however not overall conclusive; it has certain limitations. First, reaction time is not controlled, so neither impulsivity nor speed-accuracy can be measured. Second, unlike Kosson et al. (2002) they did not assess the possibility of hemispheric asymmetry. Finally, Kosson et al. (2002) found that psychopathic offenders showed deficits in facial affect recognition only under specific circumstances. In particular, they found evidence that deficits in recognition were greatest in conditions designed to minimize the involvement of left-hemisphere mechanisms. Taken all these results together, one can conclude that psychopaths have specific deficits in recognizing facial expressions of emotions. The other results are not mutually conclusive, so future research may be needed.

The last chapter regarded mimicking. Mimicking refers to the unconscious imitation of postures, facial expressions, mannerisms, and other verbal and nonverbal behavior. Mimicking also enhances self-consciousness and reduces social anxiety (Guéguen, 2011). Mimicking involves a complex network formed by occipital, temporal, and parietal visual areas, and two cortical regions whose function is fundamentally or predominantly motor (Decety et al., 2002; Rizzolatti & Craighero, 2004). Individuals with social communication disorder may show a selective impairment in mimicry responses in the upper face, the CS. Interestingly, this upper facial mimicry predicts trait empathy and psychopaths are known to have empathy impairments (Harrison et al., 2010). Motor mimicry is therefore supposed to be the essence of emotional empathy and biologically 'hardwired' (de Wied et al., 2006).

Low empathic individuals are supposed to show a higher level of ZM activity, when exposed to angry faces (Sonnby-Borgström, 2002; Sonnby-Borgström et al., 2003) and when they report negative feelings (Sonnby-Borgström, 2002). A possible explanation of the low-empathy individuals' reversed 'smiling reactions' is that these reactions through facial

feedback may serve defensive goals in inhibiting negative feelings (Sonnby-Borgström, 2002). It has also been found that individuals low in empathy overall showed less CS muscle EMG activity than moderate and high empathic individuals and that skin conductance responses are greater in moderately empathic individuals than in highly empathic ones (Westbury & Neumann, 2008). It cannot be ruled out however that those high in empathy did not look as long as those low in empathy to the film presented to avoid distress resulting in the expected and observed CS activity, but not in the expected skin conductance reactivity. A possible methodological limitation of the study by Sonnby-Borgström (2002) is the fact that the neutral stimulus was not used to obtain measurements of an emotional base-level condition. A definite methodological limitation is that the sampling rate (100 Hz) selected was too low to meet the recommendation of a sampling rate twice that of the most rapid EMG frequencies of interest. This means that the signal was sub-sampled and more reliable measurement data would probably have been obtained if the recommended sampling rate had been employed (Fridlund & Cacioppo, 1986 as cited by Sonnby-Borgström, 2002). Another limitation of both studies lies in that the participants were all students at Lund University. Consequently, the results cannot be generalized to other populations than students. The strength of these three studies can be seen in that they examined men and women, in contrast to many other studies shown.

It has also been found that DBD boys are less facially responsive than normal individuals towards angry faces, i.e. they show less mimicking behavior. It can be concluded that DBD boys are less emotionally responsive to others' distress than normal boys (de Wied et al., 2006; 2009). When one assumes that facial imitation is a factor in the process of emotional empathy, de Wied et al. (2006) propose that deficits in facial reactivity may play a role in DBD boys' impaired empathic responding. The results from de Wied et al. (2012) show that individuals high in callous-unemotional traits report less empathy, show less facial responsiveness, and less heart rate change from baseline during sadness than controls.

These results have to be interpreted with caution because of several limitations. The results from the study by de Wied et al. (2009) were obtained within a film-viewing context where individuals are passive observers and cannot be generalized to situations where individuals are active participants. Furthermore, they cannot exclude the possibility that activity in both CS and ZM muscles partly reflects nonemotional aspects of information processing like voluntary attention to external stimuli. The study by de Wied et al. (2012) used a relatively small sample size (N=44) so the power is limited. Second, they only used male participants, so their results cannot be generalized to female adolescents with DBD. Third, the majority of the DBD boys showed comorbid ADHD, so before firm conclusions can be drawn further research is needed to examine the independent effects of DBD and ADHD upon empathic behavior. Strengths of this study can be seen in the inclusion of a well-defined sample of DBD boys, with high and low callous-unemotional respondents. Another strength is the inclusion of positive and negative target emotions. Their findings encourage further research on empathy problems associated with DBD in relation to a broader range of target emotions, especially for fear and anxiety.

At last, facial expressions of emotions in psychopathy outside the content of mimicking were examined. Although there is not much known about this topic, one study has been found and provides the first evidence for the claim that adolescent pathology is indeed manifest in distinct facial expressions of emotion. Their findings also propose relations between antisocial behavior and deficits in the ability to inhibit or control impulses (Keltner et al., 1995). However, one must exercise caution in generalizing these results. A teacher, who may have been biased, observed their participants to divide them into groups. Moreover, the nature of the experimental situation, the IQ test, may have shaped the pattern of results

observed. Thereby, this study only showed that externalizing adolescents did not show much embarrassment in a school-related context, which may reflect the unimportance of school to these adolescents. In other contexts however, externalizing adolescents may show more embarrassment than during an IQ test. There is a need for further research before firm conclusions can be drawn.

The main question of this review is whether psychopaths show abnormalities in perceiving and producing facial expressions of emotions. Taken the results of the last chapter together, one can conclude that psychopaths indeed show abnormalities in their facial expressions of emotion. If these results will be replicated in the future, the findings may have clinical significance.

### *Clinical implications*

Overall, these findings demonstrate deficits in the automatic perception of the expression of empathy (mainly sadness and fear) across different groups of psychopaths. Boys and adolescents with DBD, with or without callous-unemotional traits have all been discussed. The results suggest that the mechanisms underlying empathy problems may be different for DBD subgroups with high versus low callous-unemotional traits. In clinical practice it is important to discriminate conduct-disordered individuals with callous-unemotional traits from those without such traits because they may require different approaches during intervention. All these studies concerning empathy may advance our current understanding about the mechanisms underlying empathy problems in psychopaths. If laboratory markers of empathy can be developed that distinguish between subtypes of psychopaths, for example between subtypes of DBD individuals, such instruments could be useful as part of an assessment battery (de Wied et al., 2012). More knowledge about the processes underlying deficits in the development of empathy in psychopaths is likely to inform treatment options. Because empathy training is often part of prevention and intervention programs for antisocial youth, knowledge about the nature of empathy problems associated with psychopathy may have important implications for developing more individualized or suitable training programs aimed to strengthen their empathic skills (de Wied et al., 2012).

Moreover, when assumed that motor mimicry is the essence of emotional empathy, biologically 'hardwired' and when upper facial mimicry predicts trait empathy, knowledge about mimicking can be used in clinical practice as well. For instance, this knowledge may be used to determine whether the empathy training described above has been useful and whether the psychopaths treated show more mimicking and consequently more empathy. Clearly, more research about mimicry is needed before it can be useful in clinical practice.

## References

- Achaibou, A., Pourtois, G., Schwartz, S., & Vuillemer, P. (2008). Simultaneous recording of EEG and facial muscle reactions during spontaneous mimicry. *Neuropsychologia*, *46*, 1104-1113.
- Adolphs, R. (2002). Neural systems for recognizing emotion. *Current opinion in Neurobiology*, *12*, 169-177.
- Adolphs, R., Gosselin, F., Buchanan, T. W., Tranel, D., Schyns, P., & Damasio, A. R. (2005). A mechanism for impaired fear recognition after amygdala damage. *Nature*, *433*, 68–72.
- Adolphs, R., Tranel, D., Hamann, S., Young, A., Calder, A., Anderson, A., Phelps, E., Lee, G. P., & Damasio, A. R. (1999). Recognition of facial emotion in nine subjects with bilateral amygdala damage. *Neuropsychology*, *37*, 1111-1117.
- Allison, T., Puce, A., Spencer, D. D., & McCarthy, G. (1999). Electrophysiological studies of human face perception. I: Potentials generated in occipitotemporal cortex by face and non-face stimuli. *Cerebral Cortex*, *9*, 415–430.
- American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders* (4<sup>th</sup> edn., text revision). Washington, DC: American Psychiatric Association.
- Anderson, A. K., & Phelps, E. A. (2000). Expression without recognition: contributions of the human amygdala to emotional communication. *Psychological Science*, *11*, 106-111.
- Anderson, A. K., Spencer, D. D., Fulbright, R. K., & Phelps, E. A. (2000). Contribution of the anteromedial temporal lobes to the evaluation of facial emotion. *Neuropsychology*, *14*, 526-536.
- Arrigo, B. A., & Shipley, S. (2001). The confusion over psychopathy: historical considerations. *International Journal of Offender Therapy and Comparative Criminology*, *45*, 325-344.
- Berking, M., & Wupperman, P. (in press). Emotion regulation and mental health: recent findings, current challenges, and future directions. *Current Opinion in Psychiatry*.
- Blair, R. J. R. (1995). A cognitive developmental approach to morality: Investigating the psychopath. *Cognition*, *57*, 1–29.
- Blair, R. J. R. (2001). Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. *Journal of Neurology, Neurosurgery and Psychiatry*, *71*, 727-731.
- Blair, R. J. R. (2003). Neurobiological basis of psychopathy. *British journal of psychiatry*, *182*, 5-7.
- Blair, R. J. R., Colledge, E., Murray, L. & Mitchell, D. G. V. (2001). A selective impairment in the processing of sad and fearful expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology*, *29*, 491-498.
- Blair, R. J. R., Mitchell, D. G. V., Leonard, A., Budhani, S., Peschardt, K. S., & Newman, C. (2004). Passive avoidance learning in individuals with psychopathy: modulation by reward but not by punishment. *Personality and Individual Differences*, *37*, 1179–1192.
- Blair, R. J. R., Mitchell, D. G. V., Peschardt, K. S., Colledge, E., Leonard, R. A., Shine, J. H., Murray, L. K., & Perret, D. I. (2004). Reduced sensitivity to others' fearful expressions in psychopathic individuals. *Personality and Individual Differences*, *37*, 1111-1122.
- Blair, R. J. R., Sellars, C., Strickland, I., Clark, F., Williams, A. O., Smith, M., & Jones, L.



- (1995). Emotion attributions in the psychopath. *Personality and Individual Differences*, 4, 431-437.
- Blonigen, D. M., Carlson, S. R., Krueger, F., & Patrick, C. J. (2003). A twin study of self-reported psychopathic personality traits. *Personality and Individual Differences*, 35, 179-197.
- Borod, J. C. (1993). Emotion and the brain – anatomy and theory: An introduction to the Special Section. *Neuropsychology*, 7, 427-432.
- Buck, R. (1980). Nonverbal behavior and the theory of emotion: The facial feedback hypothesis. *Journal of Personality and Social Psychology*, 38, 811–824.
- Calder, A. J., Lawrence, A. D., & Young, A. W. (2001). Neuropsychology of fear and loathing. *Nature Reviews Neuroscience*, 2, 352-363.
- Cleckley, H. (1976). *The mask of sanity* (5<sup>th</sup> ed.). St.Louis: Mosby.
- Cochin, S., Barthelemy, C., Lejeune, B., Roux, S., & Martineau, J. (1998). Perception of motion and qEEG activity in human adults. *Electroencephalography and Clinical Neurophysiology*, 107, 287–295.
- Corden, B., Critchley, H. D., Skuse, D., & Dolan, R. J. (2006). Fear recognition ability predicts differences in social cognitive and neural functioning in men. *Journal of Cognitive Neuroscience*, 18, 889–97.
- Davis, J. I., Senghas, A., & Ochsner, K. N. (2009). How does facial feedback modulate emotional experience? *Journal of Research in Personality*, 43, 822-829.
- Darwin, C. (1871). *The descent of man, and selection in relation to sex*. London: John Murray
- De Brito, S. A., Mechelli, A., Wilke, M., Laurens, K. R., Jones, A. P., Barker, G. J., Hodgins, S., Viding, E. (2009). Size matters: increased grey matter in boys with conduct problems and callous–unemotional traits. *Brain* 132, 843–852.
- Decety, J., Chaminade, T., Grezes, J., & Meltzoff, A. N. (2002). A PET exploration of the neural mechanisms involved in reciprocal imitation. *Neuroimage*, 15, 265–272.
- Deeley, Q., Daly, E., Surguladze, S., Tunstall, N., Mezey, G., Beer, D., Ambikapathy, A., Robertson, D., Giampietro, V., Brammer, M. J., Clarke, A., Dowsett, J., Fahy, T., Phillips, M. L., & Murphy, D. G. (2006). Facial emotion processing in criminal psychopathy: Preliminary functional magnetic resonance imaging study. *British Journal of Psychiatry*, 189, 533-539.
- De Wied, M., van Boxtel, A., Matthys, W., & Meeus, W. (2012). Verbal, facial and autonomic responses to empathy-eliciting film clips by disruptive male adolescents with high versus low callous-unemotional traits. *Journal of Abnormal Child Psychology*, 40, 211-223.
- De Wied, M., van Boxtel, A., Posthumus, J. A., Goudena, P. P., & Matthys, W. (2009). Facial EMG and heart rate responses to emotion-inducing film clips in boys with disruptive behavior disorders. *Psychophysiology*, 46, 996-1004.
- De Wied, M., van Boxtel, A., Zaalberg, R., Goudena, P. P., & Matthys, W. (2006). Facial EMG responses to dynamic emotional facial expressions in boys with disruptive behavior disorders. *Journal of Psychiatric Research*, 40, 112-121.
- Dimberg, U. (1982). Facial reactions to facial expressions. *Psychophysiology*, 19, 643-647.
- Dimberg, U., & Thunberg, M. (1998). Rapid facial reactions to different emotionally relevant stimuli. *Scandinavian Journal of Psychology*, 39, 39-45.
- Dimberg, U., Thunberg, M., & Elmehed, K. (2000). Unconscious facial reactions to emotional facial expressions. *American Psychological Society*, 11, 86-89.
- Ekman, P. (1992). An argument for basic emotions. *Cognition and Emotion*, 6, 169-200.
- Ekman, P. (1999). Basic emotions. In: Dalglish, T., & Power, M. (Eds.), *Handbook of*

- cognition and emotions*. Sussex: John Wiley & Sons.
- Ekman, P., & Friesen, W. V. (1971). Constants across cultures in the face and emotion. *Journal of Personality and Social Psychology*, *17*, 2, 124-129.
- Ekman, P., & Friesen, W. V. (1976). *Pictures of facial affect*. Palo Alto: Consulting Psychologists Press.
- Ekman, P., & Friesen, W. V. (1982). Felt, false, and miserable smiles. *Journal of Nonverbal Behavior*, *6*, 238-252.
- Ellis, A. W., & Young, A. W. (1988). *Human Cognitive Neuropsychology: A textbook with readings*. Hove: Psychology Press.
- Fairchild, G., van Goozen, S. H. M., Calder, A. J., Stollery, S. J., & Goodyer, I. M. (2009). Deficits in facial expression recognition in male adolescents with early-onset or adolescence-onset conduct disorder. *Journal of Child Psychology and Psychiatry*, *50*, 627-636.
- Fridlund, A. J. & Cacioppo, J. T. (1986). Guidelines for human electromyographic research. *Psychophysiology*, *23*, 567-589.
- Guégen, N. (2011). The mimicker is a mirror of myself: impact of mimicking on self-consciousness and social anxiety. *Social behavior and personality*, *39*, 725-728.
- Han, T., Alders, G. A., Greenings, S. G., Neufeld, R. W. J., & Mitchell, D. G. V. (in press). Do fearful eyes activate empathy-related brain regions in individuals with callous traits? *Social Cognitive and Affective Neuroscience*.
- Hare, R. D. (1996). Psychopathy and antisocial personality disorder: a case of diagnostic confusion. *Psychiatric Times*, *13*, 39-40.
- Hare, R. D. (2003). *Manual for the revised psychopathy checklist (2nd ed.)*. Toronto: Multi-Health Systems.
- Harpur, T. J., & Hare, R. D. (1994). Assessment of psychopathy as a function of age. *Journal of Abnormal Psychology*, *103*, 604-609.
- Harrison, N. A., Morgan, R., & Critchley, H. D. (2010). From facial mimicry to emotional empathy: A role for norepinephrine? *Social Neuroscience*, *5*, 393-400.
- Hastings, M. E., Tangney, J. P., & Stuewig, J. (2008). Psychopathy and identification of facial expressions of emotion. *Personality and Individual Differences*, *44*, 1474-1483.
- Haxby, J. V., Hoffman, E. A., Gobbini, M. I. (2002). Human neural systems for face recognition and social communication. *Biological Psychiatry*, *51*, 59-67.
- Hermans, E. J., van Wingen, G, Bos, P. A., Putman, P., & van Honk, J. (2009). Reduced spontaneous facial mimicry in women with autistic traits. *Biological Psychology*, *80*, 348-353.
- Hess, U., Thibault, P. (2009). Darwin and Emotion Expression. *American Psychologist*, *64*, 120-128.
- Hoff, H., Beneventi, H., Galta, K., & Wik, G. (2009). Evidence of deviant emotional processing in psychopathy: an fMRI case study. *International Journal of Neuroscience*, *119*, 857-878.
- Hoffman, M. L. (2000). *Empathy and moral development*. Cambridge: Cambridge University Press.
- James, R., & R. J., Blair (2007). Empathic dysfunction in psychopathic individuals. In T. F. D. Farrow, & P. W. R. Woodruff (Eds.), *Empathy in Mental Illness*, (pp. 3-16). Cambridge: Cambridge University Press.
- Jones, A. P., Laurens, K. R., Herba, C. M., Barker, G. J., & Viding, E. (2009). Amygdala hypoactivity to fearful faces in boys with conduct problems and callous-unemotional traits. *American Journal of Psychiatry*, *166*, 95-102.

- Keillor, J. M., Barrett, A. M., Crucian, G. P., Kortenkamp, S., & Heilman, K. M. (2002). Emotional experience and perception in the absence of facial feedback. *Journal of the International Neuropsychological Society*, *8*, 130-135.
- Keltner, D., & Ekman, P. (2000). *Handbook of emotions 2<sup>nd</sup> edition*. New York: Guilford Publications, Inc.
- Keltner, D., Moffitt, T. E., & Stouthamer-Loeber, M. (1995). Facial expressions of emotion and psychopathology in adolescent boys. *Journal of Abnormal Psychology*, *104*, 646-652.
- Kiehl, K. A., Smith, A. M., Hare, R. D., Mendrek, A., Forster, B. B., Brink, I., & Liddle, P. F. (2001). Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. *Biological Psychiatry*, *50*, 677-684.
- Kosson, D. S., Suchy, Y., Mayer, A. R., & Libby, J. (2002). Facial affect recognition in criminal psychopaths. *Emotion*, *4*, 398-411.
- Kring, A. M., & Bachorowski, J. (1999). Emotions and psychopathology. *Cognition and Emotion*, *13*, 575-599.
- LaBar, K. S., Crupain, M. J., Voyvodic, J. T., & McCarthy, G. (2003). Dynamic perception of facial affect and identity in the human brain. *Cerebral Cortex* *13*, 1023–1033.
- Lipps, T. (1905) Das Wissen von fremden Ichen. In Lipps T. (Ed.), *Psychologische Untersuchungen* (pp. 697-722). Leipzig: Wilhelm Engelmann.
- Lorber, M. F. (2004). Psychophysiology of aggression, psychopathy, and conduct problems: A meta-analysis. *Psychological Bulletin*, *130*, 531– 552.
- Luo, Q., Holroyd, T., Jones, M., Hendler, T., & Blair J. (2007). Neural dynamics for facial threat processing as revealed by gamma band synchronization using MEG. *Neuroimage*, *34*, 839–847.
- Lykken, D. T. (1995). *The antisocial personalities*. Hillsdale, N. J.: Lawrence Erlbaum Associates.
- Marsh, A. A., & Blair, R. J. R. (2008). Deficits in facial affect recognition among antisocial populations: a meta-analysis. *Neuroscience & Biobehavioral Reviews*, *32*, 454-465.
- Marsh, A. A., Finger, E. C., Mitchell, D. G. V., Reid, M. E., Kosson, D. S., Towbin, K. E., Leibenlust, E., Pine, D. S., & Blair, R. J. R. (2008). Reduced amygdala response to fearful expressions in children and adolescents with callous-unemotional traits and disruptive behavior disorders. *The American Journal of Psychiatry*, *165*, 712-720.
- Morris, J. S., de Gelder, B. D., Weiskrantz, L., & Dolan, R. J. (2001). Differential extrageniculostriate and amygdala responses to presentation of emotional faces in a cortically blind field. *Brain*, *124*, 1241-1252.
- Morris, J. S., Öhman, A., & Dolan, R. J. (1999). A subcortical pathway to the right amygdala mediating ‘unseen’ fear. *Proceedings of the National Academy of Sciences of the United States of America*, *96*, 1680-1685.
- Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review*, *20*, 113 -136.
- Müller, J. L., Sommer, M., Wagner, V., Lange, K., Taschler, H., Röder, C. H., Schuierer, G., Klein, H. E., & Hajak, G. (2003). Abnormalities in emotion processing within cortical and subcortical regions in criminal psychopaths: evidence from a functional magnetic resonance imaging study using pictures with emotional content. *Biological Psychiatry*, *54*, 152-162.
- Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath: Startle reflex modulation. *Journal of Abnormal Psychology*, *102*, 82-92.

- Patrick, C. J., Cuthbert, B. N., & Lang, P. J. (1994). Emotion in the criminal psychopath: fear image processing. *Journal of Abnormal Psychology, 103*, 523-534.
- Pham, T. H., & Phillippot, P. (2010). Decoding of facial expression of emotion in criminal psychopaths. *Journal of Personality Disorders, 24*, 445-459.
- Plutchik, R. (1962). *The emotions: Facts, theories and a new model*. New York: Random House.
- Raine, A., Lencz, T., Bihrlé, S., LaCasse, L., & Coletti, P. (2000). Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial personality disorder. *Archives of General Psychiatry, 57*, 119-127.
- Rizzolatti, G., & Craighero, L. (2004). The mirror-neuron system. *Annual Review of Neuroscience, 27*, 169-192.
- Rizzolatti, G., Fogassi, L., & Gallese, V. (2001). Neurophysiological mechanisms underlying the understanding and imitation of action. *Nature Reviews Neuroscience, 2*, 661-670.
- Rogers, R. D., Everitt, B. J., Baldacchino, A. J., Swanson, R., Wynne, K., Baker, N. B., Hunter, J., Carthy, T., Booker, E., London, M., Deakin, J. F., Sahakian, B. J., & Robbins, T. W. (1999). Dissociable deficits in the decision-making cognition of chronic amphetamine abusers, opiate abusers, patients with focal damage to prefrontal cortex, and tryptophan-depleted normal volunteers: evidence for monoaminergic mechanisms. *Neuropsychopharmacology, 20*, 322-339.
- Soderstrom, H., Tullberg, M., Wikkelso, C., Ekholm, S., & Forsman, A. (2000). Reduced regional cerebral blood flow in non-psychotic violent offenders. *Psychiatry Research: Neuroimaging, 98*, 29-41.
- Sonnby-Borgström, M. (2002). Automatic mimicry reactions as related to differences in emotional empathy. *Scandinavian Journal of Psychology, 43*, 433-443.
- Sonnby-Borgström, M., Jönsson, P., & Svensson, O. (2003). Emotional empathy as related to mimicry reactions at different levels of information processing. *Journal of Nonverbal Behavior, 27*, 3-23.
- Stel, M., Blascovich, J., McCall, C., Mastop, J., van Baaren, R. B., & Vonk, R. (2010). Mimicking disliked others: effects of a priori liking on the mimicry-liking link. *European Journal of Social Psychology, 40*, 867-880.
- Tassinari, L. G., and Cacioppo, J. T. Unobservable facial actions and emotion. *Psychological Science 3* (1992), 28-33.
- Tiihonen, J., Hodgins, S., & Vaurio, O. (2000). Amygdaloid volume loss in psychopathy. *Society for Neuroscience Abstracts*, 2017.
- Tomkins, S. S. (1962). *Affect, imagery, consciousness: The positive affects*. New York: Springer.
- Tomkins, S. S., & McCarter, R. (1964). What and where are the primary affects? Some evidence for a theory. *Perceptual and Motor Skills, 18*, 119-158.
- Van Boxtel, A. (2010). Facial EMG as a tool for inferring affective states. In A. J. Spink, F. Grieco, O. E. Krips, L. W. S. Loijens, L. P. J. J. Noldus, & P. H. Zimmerman (Eds.), *Proceedings of Measuring Behavior 2010* (pp. 104-108). Wageningen: Noldus Information Technology
- Van Strien, J. (2000). De neuropsychologie van emoties. *Neuropraxis, 5*, 135-143.
- Verona, E., Curtin, J. J., Patrick, C. J., Bradley, M. M., & Lang, P. J. (2004). Psychopathy and physiological response to emotionally evoked sounds. *Journal of Abnormal Psychology, 113*, 99-108.
- Vrijzen, J. N., Lange, W.-G., Becker, E. S., & Rinck, M. (2010). Socially anxious individuals lack unintentional mimicry. *Behavior Research and Therapy, 48*, 561-564.

- Vrijzen, J. N., Lange, W.-G., Dotsch, R., Wigboldus, D. H. J., & Rinck, M. (in press). Do socially anxious like to be mimicked? A virtual reality study. *Cognition and Emotion*.
- Westbury, H. R., & Neumann, D. L. (2008). Empathy-related responses to moving film stimuli depicting human and non-human animal targets in negative circumstances. *Biological Psychology*, 78, 66-74.
- Woodworth, M. & Waschbusch, D. (2007). Emotional processing in children with conduct problems and callous/unemotional traits. *Child: Care, Health and Development*, 34, 234-244.
- Wootton, J. M., Frick, P. J., Shelton, K. K., & Silverthorn, P. (1997). Ineffective parenting and childhood conduct problems: The moderating role of callous-unemotional traits. *Journal of Consulting and Clinical Psychology*, 65, 292-300.
- Zajonc, R. B. (1980). Feeling and thinking: preferences need to inferences. *American Psychologist*, 35, 151-175.