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## **Sensory and somatosensory underpinnings of emotion processing**

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## Abstract

Alexithymia is characterized by difficulties in recognizing one's own emotions and others' emotions, specially fear. Recognizing emotions is associated with remarkable changes in somatosensory and sensory (particularly visual) processing. For instance theories about emotion processing suggest a strong association between emotion processing and somatic markers. The aim of the present thesis is to assess whether the difficulties in emotion processing shown by alexithimic subjects can affect somatosensory and sensory (especially visual) processing. To this end different somatosensory modalities (e.g. temperature, pain, tactile, touch, etc) and visual stimuli (e.g. face and body expressions) were used to compare the somatosensory and sensory processing in people with high and low scores of alexithymia. These experiments provided evidence that emotional processing deficit seems to be related to the alterations in somatosensory processing (Experiments 1, and 2), in visual processing, early visual encoding (Experiments 3, 4, and 6), and in physiological reactivity, particularly visceral reactivity (Experiment 5), which prevents these individuals to correctly perceive emotions. Together, these studies suggest that the emotional difficulties in alexithymia might be grounded in the specific low-level somatosensory system. Moreover, the lack of emotional modulation at the early stage of visual processing indicates that the rapid modulation of the amygdala over the visual cortices may be reduced, thus suggesting a hyporeactivity of the amygdala in individual with high levels of alexithymia.

# CHAPTER 1: Emotion and the Brain

Emotional processes and emotional states are complex and can be analyzed from various aspects. Therefore, presenting a coherent picture and definition for emotion has been an arguable topic. “Traditionally, feelings have been defined as the elementary subjective experiences, which form the foundation for the more complex processes called emotion” (Izard, 1971). In everyday life we experience a range of different emotions and react, make decisions, and behave based on the experienced emotions. Hence it is important to know how people understand other’s emotions. In this chapter different theories about emotion and the role of underpinning neural networks will be introduced.

## 1.1. Feedback theories

Emotional feelings and experiences, do not rely only on the external physical characteristics of a stimulus. Rather, they may depend on both internal and external factors, such as one’s physiological and psychological states, and social contexts. According to feedback theories of emotion, emotional feelings require afferent input into the brain, and the prerequisite for this afferent input is efferent activity, which could be either muscular or visceral. Efferent activity is essential for producing emotional expressions, and, crucially, the afferent feedback resulting from those expressions is important for perceiving the emotion. The two major feedback theories are the facial and visceral theories.

### **1.1.1. Facial feedback**

Darwin proposed that freely expressing an emotion intensifies the experience of that emotion, while controlling and repression an emotion attenuates the emotional experience (Darwin, 1872). Cross-cultural studies of emotional facial expressions provided support for Darwin's hypothesis that emotional expressions might be innate (Izard and Ekman, 1977; Friesen, 1969). Tomkins (1963) noted that the somatosensory receptors in the face are active during emotional facial expressions, and this activity feeds back into the brain. He proposed it is the perception of one's own emotional facial expressions through this afferent feedback that induces emotional experience. Although facial expressions may exaggerate emotions, as Darwin suggested, this effect may not be driven by sensory feedback from facial muscles. Rather, it may arise entirely from within the central nervous system (CNS) through associative networks by linking the semantic memory to emotions that have been experienced before. For instance associating negative valenced words to negative emotions (Heilman., 1997) .

### **1.1.2. Visceral feedback**

William James (1950) argued that discrete emotional experiences can be identified by particular patterns of bodily changes. Emotional stimuli induce visceral changes, and the perception of these changes may lead the perceiver to experience the emotion. James also proposed that some cerebral forms of emotions (e.g., pleasure) do not need to be perceived through bodily changes.

However, the visceral feedback model of James was questioned by Cannon (1927). He proposed that the dissociation of visceral responses from the brain, for example, after cervical spinal injuries, would not necessarily lead to absence of emotional experience. Furthermore, spinal cord injuries would not interfere with the

vagus nerve's primary role to provide the CNS with feedback from the viscera (Zaidel, 1994). Cannon proposed that the afferent input produced by the viscera is not sufficient for inducing emotional experience. Later, however, it was shown that patients with lower spinal cord injuries demonstrate stronger emotions than those with higher lesions (Hohmann, 1966), providing some evidence for the visceral feedback theory. This theory was also supported by the finding that individuals with stronger emotional responses, particularly to negatively valenced stimuli, performed better on a heartbeat detection task (Hantas et al., 1982).

The brain has feedforward connections to the viscera through the autonomic nervous system (ANS; Zaidel., 1994). The ANS consists of two components: the sympathetic nervous system and the parasympathetic nervous system. Sympathetic nerves originate in the spinal cord, and receive projections from the hypothalamus, the ventral pons and the medulla. The ventral lateral medulla also receives projections from the hypothalamus, and the hypothalamus itself receives projections from limbic and paralimbic areas like the amygdala (Zaidel, 1994). The vagus nerve, which is the most important parasympathetic nerve, originates in the dorsal motor nucleus in the brainstem, and projects to visceral organs such as the heart. The amygdala also projects to the dorsal motor nucleus of the vagus nerve. Indeed, the amygdala is thought to be the most important part of the limbic system for influencing the ANS and the viscera. The insula and the orbitofrontal cortex also, to some extent, provoke autonomic and visceral changes. The amygdala, insula, and orbitofrontal cortex are likely to be the key structures for coding and using the information gained by stimulus appraisal to changes of ANS and visceral system. The vagus nerve is the major nerve that takes visceral afferents back to the brain. Then visceral afferent information end up in medulla and afterwards this nucleus projects to the amygdala and insula. Stimulation



of the vagus nerve induces excitation of the amygdala and the insula, and these structures then project to other neocortical areas such as the temporal, parietal, and frontal lobes (Heilman., 1997).

Another emotion theory, based on physiological arousal and cognitive set, is Schachter and Singer's cognitive-arousal attribution theory of emotions (Schachter and Singer., 1962) For instance they injected epinephrine into participants and showed that inducing visceral and autonomic activations per se could not produce emotional experience. According to this theory, the experience of emotion contains cognitive attributions superimposed on a state of spreading physiological arousal. This theory suggested that visceral feedback along with centrally mediated cognition are necessary for experiencing emotion. On the other hand, clinical studies showed that patients with partial seizures (medial temporal lobe or amygdala seizure) may experience emotions such as fear, as part of their seizure. Schachter and Singer's cognitive-arousal attribution theory cannot explain this finding, since, in these patients, the cognitive set would come only after the emotional experience.

## **1.2. Central theories**

### **1.2.1.Diencephalic:**

Cannon (1927) proposed that afferent signals enter the brain, and are transmitted from the thalamus to the hypothalamus. The hypothalamus activates the endocrine system and the ANS, and these systems lead to changes in the viscera. Whereas James suggested that information from the viscera feeds back directly into the cortex, Cannon asserted that this pathway was routed through the hypothalamus. Recently, it has been shown that the amygdala, together with the thalamic-

hypothalamic circuit, plays a role in many emotional experiences and critically in threatening situations. However, Cannon did not consider the important role of the cortex in translating the emotional stimulus.

### **1.2.2. Limbic system**

Papez (1937) was the first to suggest that a circuit in the limbic system is crucial for emotion processing. This circuit includes the mammillary bodies of the hypothalamus that project via the mammillo thalamic tract to the anterior thalamus, which in turn projects to the cingulate gyrus. The cingulate gyrus itself projects to the hippocampus, and the hippocampus projects back to the mamillary bodies via the fornix. Importantly, the anterior temporal cortex projects to the amygdala, and the amygdala projects to the orbitofrontal cortex (Morris et al., 1998). This is important for how human experience emotion from complex visual stimuli.

### **1.3. Modular theory**

Wundt (1903) proposed that emotional experiences vary along three dimensions: quality, activity, and excitement. These categories have been recently modified to consist of valence (positive/negative, pleasant/unpleasant), arousal (calm/excited), and control (in control/out of control) (Heilman., 1997). For instance, fear is an unpleasant, highly arousing emotion that results in a loss of control.

It had been suggested that positive and negative emotions are mainly related to approach (regulated in the left hemisphere) and avoidance (regulated in the right hemisphere) behaviors (Fox and Davidson, 1984). Yet, it remained unclear how the two

hemispheres were differentially organized. Tucker and Williamson (1984) suggested that the hemispheric valence asymmetry might be related to asymmetrical control of neurotransmitter systems, with the left hemisphere being more cholinergic and dopaminergic, and the right hemisphere being more noradrenergic.

Arousal is another aspect of emotion which refers to the excitatory state of CNS neurons that discharge when activated. In the peripheral nervous system, arousal activates the sympathetic system, with consequences such as increased heart rate. Arousal and attention are closely linked, and the cortical limbic reticular network is thought to mediate both (Heilman, 1981; Mesulam, 1981). Changes in the activity level of the peripheral ANS mirror changes in CNS arousal. For instance, ANS arousal often leads to hand sweating, and this can be measured as an index of peripheral arousal using galvanic skin response (GSR; Heilman et al., 1978). It has been shown that right hemisphere damage leads to reduced GSR and lower heart rate (Yokoyama et al., 1987). The right hemisphere may play a significant role in assessing stimulus significance.

While some emotions do not excite the action system (e.g., sadness) others do (e.g., fear, anger, and joy). The type of action that some emotions cause could either be an action toward the stimulus (approach) or away from the stimulus (avoidance). For instance, fear and disgust may be associated with avoidance, while anger and joy are associated with approach behaviors. Aberrant approach behaviors and avoidance responses have been seen after frontal and parietal lesions, particularly to the right hemisphere (Grafman et al., 1986).

## CHAPTER 2: Emotion and alexithymia

### 2.1. Alexithymia concept

The term alexithymia (from the Greek: *a*=lack, *lexis*=words, *thymos*=emotion) was used by Sifneos (1973) to describe the difficulties associated with a particular psychosomatic disorder, including: difficulty in describing feelings, difficulty in distinguishing the affective component of emotional arousal from the physiological (somatic) component, an impoverished fantasy life, and an externally oriented cognitive style (Sifneos, 1973). During the mid-1970s, the cognitive and affective characteristics described by Nemiah and Sifneos were more precisely defined and integrated into a multidimensional personality construct that is now referred to as the alexithymia construct (Nemiah et al., 1976). An important reason for drawing attention to alexithymia was its association with many psychiatric and psychosomatic disorders, such as depression, anxiety, schizophrenia, eating disorders, and chronic pains (Arturcedo et al., 2001; Honkalampi et al., 2000; Marchesi et al., 2000; Lumley et al., 1996; Taylor et al., 1996;; Schmidt et al., 1993).

Over the past decades, alexithymia has been defined as a personality trait construct that is characterized by three major indicators: difficulties in identifying, differentiating, and verbalizing one's feelings, a paucity of fantasies, and an externally oriented, concrete cognitive style (Taylor et al., 1997). The prevalence of alexithymia in the general population has been reported to be around 10% (Salminen et al., 1999; Taylor et al., 1991). A validated and widely used self-report measure of alexithymia is the 20-item Toronto Alexithymia Scale (TAS-20; Bagby et al., 1994), which assesses three major indicators of alexithymia: Difficulty Identifying Feelings (DIF), Difficulty

Describing Feelings (DDF), and Externally Oriented Thinking (EOT). A score of 61 or higher qualifies one as a high alexithymic on the TAS-20 (Taylor et al., 1997).

## **2.2. Alexithymia dimensions**

As described earlier, alexithymia is thought to be a multifaceted construct that is generally divided into its affective and cognitive dimensions (Vorst and Bermond, 2001). The cognitive dimension refers to emotional processing at a cognitive level, and consists of deficits in identifying, describing, and analyzing feelings and emotions. The affective dimension refers mostly to the subjective component of one's emotional experience, and involves a lack of imagination and fantasizing, as well as decreased emotional arousal from emotional stimuli (Bermond et al., 2008). The cognitive dimension is measured by the DIF and DDF subscales, and the affective dimension is measured by the EOT subscale of the TAS-20.

## **2.3. Alexithymia: a deficit in understanding emotion**

Impairment and difficulty in identifying, recognizing, and describing the emotions of others is another crucial aspect of alexithymia. Because the recognition of others' emotions and affective states is crucial for everyday social communication, this aspect of alexithymia is of particular importance. Alexithymia is associated with impairments in identifying others' facial expressions of emotions such as fear (Parker et al., 2005, Jessimer and Markham., 1997; McDonald and Parkchin, 1990; Lane et al., 1996; Cook et al., 2013; Prkachin et al., 2009; Berthoz et al., 2014). The automatic reactivity of the amygdala, a key structure in emotion processing and facial emotion recognition, has been shown to be negatively related to cognitive dimensions of alexithymia (Kugel et al., 2008). Because of weak signaling from the amygdala to the visual occipito-temporal areas, the superior temporal gyrus, the insula, the parahippocampal gyrus, and the

fusiform gyrus, these areas also show a decreased response to facial expressions in alexithymia (Reker et al., 2010; Karlsson et al., 2008; van der Velde et al., 2013). Alexithymics also demonstrate difficulties in empathizing with others. For instance, they empathize less with someone in pain, and show reduced activity in the anterior insula, a key neural structure underlying empathy (Moriguchi et al., 2007; Haviland et al., 2004; Grynberg et al., 2010; Bird et al., 2010; Patil & Silani, 2014; Silani et al., 2008). Relevant to these difficulties at both the cognitive and affective levels, alexithymics exhibit poor interpersonal and intrapersonal skills in social communication (Lumley et al., 1996; Kauhanen et al., 1993), as well as difficulties with more complex social decisions, such as moral judgments (Patil & Silani, 2014; FeldmanHall et al., 2013).

#### **2.4. Neural underpinnings of alexithymia**

Since the greatest difficulties in alexithymia involve emotional experience and emotion processing, most neuroimaging studies on alexithymia have focused on identifying brain areas associated with emotion processing in alexithymics. Because the right hemisphere is crucial for processing emotional information and regulating emotional behavior (Adolphs et al., 2000), some neurobiological theories of alexithymia propose a right hemisphere deficit or a left hemisphere preference (Bermond, 1997; Bermond et al., 2005).

The anterior cingulate cortex (ACC) and the insula are other brain regions involved in emotion processing. The ACC is thought to be involved in conscious detection of emotional information at an interoceptive level, as well as in directing attention towards emotional signals and pain processing; therefore, ACC dysfunction may be a neural correlate of alexithymia (Lane et al., 1997). Due to the association

between high levels of alexithymia and decreased ACC activity during emotional arousal, the term “blind feel” has been attributed to alexithymia (Lane, 1997).

While some studies found no difference in limbic system activity between high and low alexithymics (Kano et al., 2003; Berthoz et al., 2002; Heinzl & Schafer, 2010), others showed changes in the activity of the amygdala, a key subcortical structure for automatic emotion processing. For instance, some studies have found a negative correlation between scores on alexithymia scales and amygdala activation during an emotion detection task (Kugel et al., 2008; Reker et al., 2010). Other studies have shown lower amygdala activation in response to emotional stimuli, particularly negatively valenced, arousing stimuli, as well as a smaller amygdala volume in people with alexithymia (Bermond et al., 2006; Goelich-Dobre et al., 2015; Ihme et al., 2013; Kano and Fukudo., 2013; Moriguchi and Komaki., 2013; Leweke et al., 2004; Larsen et al., 2003; Wingbermuehle et al., 2012; van der Velde et al., 2015).

Recently, a review study concluded that alexithymics, in response to positively valenced stimuli, have higher activation in the dorsal ACC and the middle cingulate cortex, and lower activation in the precuneus, the cuneus, the right posterior and anterior insula, and the left superior temporal gyrus. In response to negatively valenced stimuli, people with high levels of alexithymia showed greater activation in the dorsal ACC and the right middle temporal gyrus.. Moreover, decreased activation in the left premotor cortex, the bilateral fusiform gyrus, the bilateral amygdala, the supplementary motor area, the left dorsomedial prefrontal cortex (dMPFC), the left middle occipital gyrus, the right putamen, and the left superior parietal gyrus were found with high levels of alexithymia during performing the task (van der Velde et al., 2013).

In conclusion, alexithymia is a personality trait involving crucial alterations in emotional experiences and emotion processing, making it a good population for studying emotion processing. Hence, the experiments in this thesis compared samples of people with high and low scores on an alexithymia scale to further study emotion processing mechanisms. Investigating emotion processing in this population, with a particular focus on sensory and somatosensory underpinnings, shed light on the necessary aspects of emotion analysis at multiple levels, from primary sensory, physiological, and perceptual levels to higher behavioral levels.



## **CHAPTER 3: Emotion and somatosensory processing**

### **3.1. Somatosensory related cortices in emotion processing**

The important role of somatosensory related cortices in right hemisphere for emotion recognition has been found through a large number of lesion studies in this area. For instance Adolphs and colleagues in 2000 showed lesions in right somatosensory related cortices, including primary and secondary somatosensory areas, insula, anterior supramarginal gyrus, and lesions in the left frontal operculum resulted in compromised emotion recognition (Adolphs et al., 2000). One theory to justify this activation in somatosensory areas is that viewing an emotional expression triggers an emotional response in the viewer and this causes the representation of that emotion in somatosensory cortices which helps the perceiver to recognize that emotion (Wild et al., 2001; Damasio., 1994). Interestingly, it has been shown the same neural network needed for understanding one's own emotions and feelings is involved in recognizing others' emotions (Preston and de Waal, 2002; Lamm et al., 2007). For instance it has been shown that using beta blockers for dampening autonomic reaction in response to emotion would lead to deficit in recognition of sad facial expressions (Harmer et al., 2001). The role of insular cortex as a visceral somatosensory cortex has been emphasized specially in recognizing emotions that engage interoceptive systems like disgust (Phillips et al., 1998).

Overall activation in somatosensory cortices appears to be a crucial fundament during emotion recognition. Some of the studies on alexithymia reported altered activation in somatosensory substrate that is thought to have an important role in reporting higher somatoform and psychosomatic disorders in people with high scores

on alexithymia. A tendency to experience normal bodily sensations as intense, noxious, and disturbing as if there is a pathogenic mechanism for that, has been reported in some studies in alexithymia. However other studies failed to find similar results. In this chapter, regarding the important role of somatosensory processing in emotion recognition, I investigated the processing of a number of somatosensory modalities in individuals with high and low alexithymia to examine whether there is a difference between two groups.

### **3.2. Experiment 1: “Lacking warmth”:** Association between alexithymia trait and specific somatosensory signals

Alexithymia is a multifaceted personality construct, expressed with varying intensity in the general population. Self-report measures like the Toronto Alexithymia Scale (TAS) (Bagby et al., 1994), the most widely used and well-validated assessment tool (Bagby et al., 1994; Parker et al., 2003) characterize alexithymia through three main facets: difficulties in identifying feelings (DIF), difficulties in describing feelings (DDF), and externally-oriented thinking or a preoccupation with the details of external events (EOT). Dimensional analysis suggest alexithymia comprises an affective dimension, involving emotionalizing, and fantasizing, and a cognitive dimension, involving identifying, differentiating and describing feelings) (Goerlich-Dobre et al., 2014; Herbert et al., 2011; Parker et al., 2008; Taylor et al., 1991). Importantly, people with high levels of alexithymia exhibit difficulties not only in processing their own emotions, but also in processing the emotions expressed by others (Sifneos, 1973; Jessimer & Markham, 1997; Parker et al., 1993, 2005; Borhani et al., 2016). Thus, alexithymic individuals show altered recognition of emotional stimuli (Grynberg et al., 2012; Ihme et al., 2014) and decreased activation of the amygdala during presentation of emotional stimuli (Jongen et al., 2014; Moriguchi and Komaki, 2013), and particularly negative stimuli (Kugel et al., 2008; Pouga et al., 2010; Reker et al., 2010; for a recent meta analysis: van der Velde et al., 2013).

The relation between alexithymic traits and specific neural systems or pathways remains controversial. In particular, it is unclear whether specific patterns of somatosensory processing accompany these cognitive and affective deficits. The strong association between emotion processing and somatic markers (Damasio et al,

1996) suggests that alexithymia might involve abnormalities of somatosensory or autonomic processing. Disruption in regulation of emotions in alexithymia, particularly negative emotions, is thought to result in chronic sympathetic hyperarousal, high sensitivity to painful stimulation, ) somatosensory amplification (the tendency to experience somatic sensation as intense, noxious, and disturbing - Barsky et al., 1988) and complaints of physical symptoms (Lumley et al., 1996; Kano et al., 2007). Somatosensory brain regions, including the insula and somatosensory cortices, were hypothesized to be overactive during emotional processing in alexithymia, potentially explaining the alexithymic tendency to experience physical symptoms when emotionally aroused (Karlsson et al., 2008). However other studies failed to find associations between somatosensory amplification and alexithymia (Lesser et al., 1979; Kosturek et al., 1998; Gregory et al. 2000). For instance while Nyklicek and Vingerhoets (2000) reported hypersensitivity for pain and touch in alexithymia, De Zwaan et al., 1996 failed to find any association between the degree of alexithymia and thermally and mechanically induced pain threshold, in a group of patients with eating disorders (De Zwaan et al.,1996). Table 1 summarizes a number of key studies providing evidence regarding somatosensory processing in alexithymia.

Table 1

*Studies relating alexithymia to atypical somatosensory processing*

No	Authors	Year	N	Participants	Experimental paradigm	Results
1	Wise et al,	1994	101	Psychiatric patients	Self reports: TAS-20 SSAS	Significant positive correlation between TAS-20 and SSAS scores
2	Nyklicek and Vingerhoets	2000	41	Healthy participants	Painful electrical stimulation	Significant positive correlation between alexithymia score and sensitivity to pain
3	Nako et al,	2002	81	Psychosomatic vs patient control groups	Self reports: TAS-20 SSAS	Significant positive correlation between TAS-20 and SSAS scores
4	Kano et al,	2007	45	Healthy participants	Brain processing of visceral sensation induced by colonic distension	Significant positive correlation between alexithymia and sensitivity to visceral stimulation
5	Millard and Kinsler	1992	195	Patients with chronic nonmalignant pain	Self report: TAS Pain intensity	No relation between alexithymia and sensitivity to pain
6	Cox et al,	1994	55	Patients with somatoform pain disorder	Self report: TAS_20 McGill Pain Questionnaire	No significant pain severity difference between alexithymics and non alexithymics
7	de Zwaan et al,	1996	72	Patients with anorexia nervosa, Patients with bulimia nervosa, Healthy participants	Detection threshold for mechanically and thermally induced pain	No relation between alexithymia and pain threshold
8	Gregory et al,	2000	140	Patients with chronic nonmalignant pain	Self reports: TAS SSAS	No significant association between alexithymia and SSAS score
9	Jackson et al,	2002	116	Healthy participants	Pain tolerance to cold pressor test	No significant correlation between alexithymia and sensitivity to unpleasant stimuli
10	Karlsson et al,	2008	21	Healthy participants	Watching emotional videos	Somatosensory brain regions were more strongly activated during watching emotional videos in high alexithymics than low alexithymics

Indeed, existing accounts make conflicting predictions about the *direction* of any putative association between alexithymia and somatosensation. On the one hand, somatosensory cortical areas (e.g. right primary and secondary somatosensory areas and the insula) are positively activated during emotion recognition in healthy volunteers (Adolphs., 2002), suggesting that high alexithymia might be associated with reduced somatosensory processing. On the other hand, the somatosensory amplification concept predicts increased somatosensory processing (Perez et al., 2015; Barsky et al., 1988; Ihme et al., 2014) in alexithymics, at least for some somatic stimuli. A detailed understanding of relations between alexithymia and somatosensory processing has been hampered by lack of psychophysiologicaly-validated measures of somatosensory processing. We have therefore performed two studies relating alexithymia scores to scores on an established quantitative sensory testing (QST) battery (Rolke et al., 2006). QST refers to a series of psychophysical tests, assessing the neurophysiological function of the major afferent fibre pathways in the skin (Hansson et al., 2007). QST is widely used in neurology, and extensive norms are available ( Rolke et al., 2006a; Rolke et al., 2006b). Importantly, the various QST subtests each focus on a specific somatosensory submodality, so QST has potentially high sensitivity to identify selective deficits in specific neurophysiological mechanisms. We used the following QST subtests: warm threshold, cold threshold, pinprick pain threshold, tactile acuity, and somatosensory detection test were tested. In addition, we included established tests of interoceptive awareness (Critchley et al., 2004), and affective touch ( McGlone et al., 2014), which are not part of classical QST, but involve a degree of psychophysiological specificity, and which have previously been linked to alexithymia. We hypothesised that, if emotion recognition impairment in alexithymia has a somatosensory grounding; this

should be revealed by a relation between TAS scores, and performance on one or more subtests of QST.

### **3.2.1. Methods:**

Participants: 189 volunteers (fifty one males, mean age=23.7, range:18-40) filled out the 20-item Toronto Alexithymia Scale (TAS-20;Taylor et al., 2003). For experiment 1, 40 healthy Individuals with high (six males, mean age=24.65, range: 19-35) and low (six males, mean age=25.7, range:19-40) TAS-20 totals were selected in order to obtain a sample varying widely in levels of the alexithymia trait. The 20 low alexithymia (LA) participants had a TAS mean score of 31.05 (SD=3.18, range: 25-35, corresponding to lower TAS quartile), while the 20 high alexithymia (HA) participants had a TAS mean score of 66.2 (SD=5.69, range: 61-77, corresponding to upper TAS quartile). In addition the alexithymia module of the structured interview for the Diagnostic Criteria for Psychosomatic Research (DCPR) (Mangelli et al., 2006; Porcelli and Rafanelli, 2010; Porcelli and Sonino, 2007) was also used to confirm the presence or absence of alexithymia (LA:1.6, SD=.5, range:1-2;, HA:4.6, SD=1.14, range:3-6). Participants were included in the study if *i*) they had no history of neurological, major medical or psychiatric disorder and *ii*) their scores on the TAS-20 and the DCPR were congruent (congruency was defined as both scores on TAS20 and DCPR had to indicate the same level of alexithymia) . No participant was excluded due to discrepancy between TAS-20 and DCPR score. Two participants were excluded due to previous history of neurological or psychiatric disorder.

The data were obtained from 40 healthy participants in a comprehensive standardized QST protocol consisting of 6 tests: cold and warm thermal perception threshold, pinprick

pain threshold, tactile acuity, affective touch, interceptive awareness, and somatosensory detection test were tested. All participants performed the tests in the same, stated order. The whole experiment took about two hours and a half. Participants provided written informed consent prior to the experiment and were paid £7.50 per hour. The experiment was approved by the UCL Research Ethics Committee, and carried out in accordance with the provisions of the World Medical Association Declaration of Helsinki.

**Thermal detection threshold test:** Contact thermal stimuli were delivered to the back of the left hand using a 13 mm circular diameter Peltier-type thermode (NTE-2A, Physitemp Instruments Inc). Contact warm and cold threshold was estimated by the method of limits (Yarnitsky et al., 1995). The probe temperature was fixed for a random time between 28-30 s at an initial level of 32 °C and then ramped up or down by 2 °C/s. To avoid possible pain and tissue damage, maximum temperature was limited to 50 °C and minimum temperature was limited to 14 °C. Participants were asked to press a button using their right hand as soon as they felt any change in temperature, and then report the direction of the change, whether the stimulus got colder or warmer. Three cold and three warm stimulus ramps were delivered in random order. The three trials were averaged to obtain a warm threshold and a cold threshold measurement. Interstimulus intervals were 20 s.

**Pinprick pain threshold and discrimination task:** Noxious radiant heat stimulation was delivered by an infrared CO<sub>2</sub> laser stimulation device with a wavelength of 10.6 μm (SIFEC, Ferrières, Belgium). The laser pulse (100 ms duration) was transmitted via an optic fiber to reach a spot diameter of 6 mm on the dorsum of participants' left hand. These laser pulses selectively excite A $\delta$ - and C-fibers without co-activating lower-threshold A $\beta$ -fibers. For each participant, we identified the A $\delta$  threshold for 'pinprick pain' using ascending-descending-ascending staircases. The threshold was



identified by finding the lowest skin temperature that elicited both a report of “pinprick sensation”, and a reaction time (RT) < 650 ms (Mouraux et al., 2003; Churyukanov et al.,2012). Starting at 38°C, the temperature was increased in steps of 4°C until RT was less than 650 ms. Then the temperature was decreased in steps of 2°C until the RT became longer than 650 ms. Finally, the temperature was increased steps of 1°C until RT was less than 650 ms again, and the participant reported a pinprick sensation for 3 consecutive repetitions of the same temperature (HA, M = 47.4°C; LA, M=47.94). To estimate nociceptive discrimination, we then set a low stimulus intensity at 2°C above pinprick threshold, and the high stimulus intensity at 8°C above pinprick threshold. Participants were familiarized with the high and low levels of stimulation, and received some discrimination practice trials. Then they performed a forced-choice task in which they received 30 low and 30 high intensity stimuli, randomly interleaved with a random 10-15 s interstimulus interval. After each set of fifteen stimuli there was a 5 min pause to prevent habituation to painful stimuli. Participants were asked to discriminate whether the perceived stimulus was of high or low intensity, responding with a keyboard. Signal detection theory ( Green & Swets, 1966) was used to obtain independent estimates of nociceptive perceptual sensitivity and response bias.

**Tactile acuity test:** The Grating Orientation Test (GOT; van Boven & Johnson, 1994) consists of a series of square wave gratings with graded spatial frequencies, and 50% duty cycle. It was used to measure each participant’s grating orientation discrimination threshold – an established measure of the A $\beta$  light touch pathway. Beginning with the largest ridge width (3 mm) the experimenter applied the grating to the participant’s index fingertip while they were blindfolded. Each grating was presented three times for approximately .5 s, randomly changing the orientation, so the ridges could run either along or across the axis of the index finger. Participants made unspeeded verbal forced-choice judgments regarding the orientation of the gratings, responding “along” or

“across” the finger. If all three trials were perceived correctly, the next lowest ridge width was used. This procedure continued with gratings of decreasing ridge width until the participant made at least one error (i.e., accuracy of 66.6% or less over three trials. The ridge width was then increased again until the participant answered correctly on 100% of three trials. This ridge width was then used as the participant’s threshold. The whole procedure repeated 3 times and results were averaged to obtain a single threshold score.

**Affective touch:** participants sat at a table with their left forearm resting palm-up. Three tactile stroking stimuli at velocities of 0.3, 3, 30 cm/s were delivered over a 10 cm distance. The stimuli were delivered either by experimenter’s index, middle and ring finger or by three joined paintbrushes (Daler Rowney Oval Wash Brush size ½) positioned to form the same shape as

the experimenters’ fingers, and moved by a robot (Phantom premium 1.0). Stimuli were blocked across the type of agent (experimenter or robot). Inside each block, stimulus speed was randomized. E speed repeated twice. The interstimulus interval was 30 s. To keep stimulation duration constant, 1 stroke at 0.3 cm/s, or 10 consecutive strokes at 3 cm/s or 100 strokes at 30 cm/s was applied. The experimenter was trained to apply stroking similarly to the robot. Following each stroke the participants were instructed to rate the pleasantness and softness of stimulus using two separately-presented paper and pencil visual analog scales (VAS), with the endpoints unpleasant to pleasant (– 10 to 10). Prior to the experiment participants were familiarised with one trial for each stimulus with different velocity and delivered by either the experimenter or robot. Previous studies have shown higher pleasantness for 3 cm/s stroking, and linked this velocity-specific pleasant sensation to C-tactile afferents (Bessou et al., 1971; Löken et al., 2009) .

**Interoceptive sensitivity:** The Heartbeat Perception Task was used as a measure of interoceptive sensitivity (ISt). The ECG was measured through nonpolarizable Ag-AgCl electrodes attached to the left ulna styloid process, and right wrist and referenced to the right radial styloid process. Signals were recorded by a BioSemi amplifier system (BioSemi, Amsterdam, The Netherlands). A sampling rate of 1000 Hz was used. R-waves were detected online and were stored on a trigger channel. The heartbeat perception task was performed according to the Mental Tracking Method proposed by Schandry (1981), using four intervals of 25, 45, and 60 seconds. The four perception intervals were separated by standard resting periods (30 seconds). For all trials, participants were asked to silently count their heartbeats by concentrating on their heart activity. During heartbeat counting, participants were not permitted to take their pulse or to attempt any other physical manipulations that could facilitate the detection of heartbeats. Following the stop signal, participants were asked to verbally report the number of counted heartbeats. The participants were not informed about the lengths of the counting phases or about the quality of their performance. ISt was measured as a heartbeat perception score, calculated by taking the mean score across the three heartbeat perception intervals according to the following transformation:  $1/3 \sum (1 - (|\text{recorded heartbeats} - \text{counted heartbeats}|) / \text{recorded heartbeats})$ . The heartbeat perception score varies between 0 and 1. The maximum score of 1 indicates absolute accuracy of heartbeat perception. This heartbeat detection task is widely used to assess interoceptive sensitivity (Dunn et al. 2007; Herbert et al. 2007). It has good test-retest reliability, and correlates highly with other heartbeat detection tasks (Knoll and Hodapp, 1992).

**Somatosensory detection test:** Tactile stimulation with a duration of 10 ms was delivered using a Digitimer DS5 constant current stimulator (Digitimer Ltd., Welwyn

Garden City, UK) connected to a pair of disposable press-stud electrodes (Biosense Medical, Chelmsford, UK) placed on dorsum of the left hand.

Electro-tactile stimulation was used to determine each participant's detection threshold. Starting at 0.5 mA, the current was increased in steps of 0.5 mA until the participant detected the stimulus. The current was then reduced in 0.5 mA steps until the stimulus was no longer detected, and then increased again until the stimulus was again perceived. This last value was taken as the detection threshold. Next, the current was increased rapidly to near-pain threshold, and then the same procedure was used to measure the participant's pain threshold. The low and high levels of stimulation for the main experiment were then set to 45% and 55%, respectively, of the range between the detection and pain thresholds. These levels were chosen based on a pilot study in a separate group of volunteers which indicated that this difference between high and low electro-tactile intensities would approximately match the discrimination performance used in the test of nociceptive discrimination between high and low laser heat-pain stimuli. The mean difference between the high and low intensities was 1.05 mA (range = 0.55-1.15 mA). Participants were familiarized with the high and low levels of stimulation. Then they performed a forced-choice task in which they received 80 (40 high stimulus intensity) randomly delivered stimuli. Inter stimuli interval was randomised between 8 to 10 s. Participants were asked to discriminate whether the perceived stimulus was high or low stimulus intensity, and respond with the keyboard. SDT was used to obtain independent estimates of perceptual sensitivity and response bias.

**Data analysis:**

The participants were required to identify pinprick pain and electrotactile stimuli as 'high' or 'low'. Their reports in the task were recast as attempts to detect the high intensity stimuli, so that the results could be analysed using signal detection analysis

(Macmillan & Creelman, 1991). We considered the number of hits for high intensity level of noxious or electro-tactile stimulus (number of high intensity stimulus trials in which participants responded 'high'), false alarms (number of low intensity stimulus trials in which participants responded 'high'). Hit rates [P('high' response|high intensity stimulus), proportion of hit trials to which subject responded 'high'] and false alarm rates [P('high' response|low intensity stimulus), proportion of trials in which low intensity stimuli were reported as 'high'] were calculated. These were used to obtain the perceptual sensitivity ( $d'$ ) in detecting the high intensity stimulus  $D'$  does not require homogeneous variance and can be calculated even if the hit or false alarm rates are 1 or 0. The tendency to report stimuli as 'high', irrespective of actual intensity, (C, response bias) was also obtained. Sensitivity and response bias were calculated for high intensity noxious and electro-tactile stimuli.

### **3.2.2. Results:**

The full results are shown in table 2. The only somatosensory sub-modality test which significantly differed between low and high alexithymia was thermal detection threshold. Therefore we start with reporting thermal detection threshold results and then we carry on with non significant results.

Table 2.a  
QST and interoceptive sensitivity results

Tested modality	HA		LA		t-value	df	p-value
	Mean	SD	Mean	SD			
Warm threshold (°C)	36.73	3	34.43	1.46	3.08	38	0.003
Cold threshold (°C)	28.56	5.24	29.96	0.76	-1.18	38	0.24
Warm detection accuracy (%)	80.11	40.80	80.11	40.80	0.00	38	1.00
Cold detection accuracy (%)	85.04	36.51	95.03	22.21	-1.04	38	.30
Pinprick laser heat-pain threshold (°C)	47.55	2.64	48.10	2.22	-0.71	38	0.48
RTs to noxious laser stimulus (ms)	512	80.23	518.3	65.27	-0.27	38	0.8
Sensitivity ( $d'$ )	1.79	0.75	1.82	0.56	0.12	38	.9
Response bias (C)	0.39	0.44	0.27	0.36	0.94	38	0.35
Tactile acuity threshold (mm)	1.66	0.38	1.61	0.36	0.42	38	0.67
Sensory detection task:							
Tactile detection threshold (mA)	1.50	0.56	1.58	0.73	-0.46	38	0.64
Near pain tactile threshold (mA)	1.63	0.63	1.73	0.73	-0.48	38	0.63
Sensitivity ( $d'$ )	1.40	0.49	1.69	0.62	-1.66	38	0.10
Response bias (C)	0.079	0.28	-0.023	0.23	1.23	38	0.22
Interoceptive sensitivity ( )	0.74	0.153	0.73	0.19	0.09	38	0.92

Table 2.b  
Affective touch  
results

Main effects	Mean	SD	F-value	Df	p-value
<b>Pleasantness ratings</b>					
Group:			4.30	38	0.04
HA	1.87	1.21			
LA	.92	1.64			
Velocity:			46.39	76	<.0001
0.3 cm/s	0.056	2.21			
3 cm/s	2.52	1.57			
30 cm/s	1.73	1.53			
<b>Interactions</b>					
Agent x velocity			4.43	76	0.015
Experimenter	0.025	2.34			
0.3 cm/s					
Experimenter 3	2.9	1.67			
cm/s					
Experimenter30	1.57	2.06			
cm/s					
Robot 0.3 cm/s	-0.13	2.36			
Robot 3 cm/s	2.15	1.38			
Robot 30 cm/s	1.88	4.42			
<b>Softness ratings</b>					
Main effects					
Group			5.32	38	0.026
HA	2.37	1.03			
LA	1.5	1.35			
Velocity			13.03	76	<0.0001
0.3 cm/s	1.43	1.84			
3 cm/s	2.65	1.32			
30 cm/s	1.73	1.45			
Interaction					
Agent x velocity			3.87	76	0.025
Experimenter	1.16	2.19			
0.3 cm/s					
Experimenter	2.86	1.14			
3cm/s					

Experimenter	1.55	1.60
30cm/s		
Robot 0.3cm/s	1.7	2.3
Robot 3 cm/s	2.43	1.67
Robot 30 cm/s	1.92	1.61

#### **Thermal detection threshold:**

Since the pathways for processing warm and cold sensations are different, separate t-tests were run for warm and cold threshold estimates.

An independent samples t-test was conducted to compare thermal detection threshold for warm and cold sensations in HA and LA groups. This analysis was done only on correct answered trials. There was a significant difference in warm detection threshold between HA (M= 36.73 °C, SD= 3.00) and LA (M=34.43 °C, SD= 1.46), ( $t(38) = 3.08$ ;  $p = .003$ ). There was no significant difference in cold detection threshold between HA (M=28.56 °C, SD=5.24) and LA (M=29.96 °C, SD=.76), ( $t(38) = -1.18$ ;  $p = .24$ ).

In addition to threshold measures, we also analysed the accuracy of identifying direction of thermal change. There was no significant difference between HA and LA groups in detection of warming (M= 80.11%, SD= 40.80, vs M=80.11%, SD= 40.80), ( $t(38) = 0$ ;  $p = 1.00$ ) or cooling (M= 85.04%, SD= 36.51, vs M=95.03%, SD= 22.21), ( $t(38) = -1.04$ ;  $p = .30$ ).



### **Pinprick pain threshold and discrimination task:**

An independent samples t-test was conducted to compare pinprick threshold in HA and LA groups. There was no significant difference in pinprick threshold in HA (M= 47.55 °C, SD= 2.64) and LA (M=48.10 °C, SD= 2.22), ( $t(38) = -.71$ ;  $p=.48$ ). Similarly results for reaction times in response to noxious stimulus revealed no significant difference between HA (M=512 ms, SD=80.23) and LA (M=518.35 ms, SD=65.27), ( $t(38) = -.27$ ;  $p=.8$ ). Conducting a t-test showed sensitivity ( $d'$ ) did not differ between HA (M=1.79, SD=.75) and LA (M=1.82, SD=.56), ( $t(38) = .12$ ;  $p=.9$ ).

Response bias (C) results revealed no significant difference between HA (M= .39, SD=.44) and LA (M=.27, SD=.36), ( $t(38) = .94$ ;  $p=.35$ ).

### **Tactile acuity test:**

An independent samples t-test was conducted to compare tactile acuity threshold in HA and LA groups. There was no significant difference between HA (M= 1.66 mm, SD= .38) and LA (M=1.61 mm, SD= .36), ( $t(38) = .425$ ;  $p=.674$ ).

### **Affective touch:**

A mixed factors analysis of variance (ANOVA) with the between-subjects factor group (HA, LA) and the within-subjects factors agent that applied the touch stimulus (experimenter, robot), velocity (0.3, 3, 30 cm/s) was run on pleasantness and softness ratings.

**Pleasantness rating:** There was a significant main effect of group ( $F(1, 38) = 4.30$ ;  $p = .044$ ,  $\eta_p^2 = .10$ ), with higher given pleasantness ratings in HA (M = 1.875, SD=1.21) than in LA

( $M=0.925$ ,  $SD=1.643$ ). There was also a main effect of velocity ( $F(2, 76) = 46.39$ ;  $p<.0001$ ,  $\eta_p^2 = 0.549$ ). Participants rate the optimal velocity, 3 cm/s, as more pleasant ( $M=2.52$ ,  $SD=1.57$ ), than higher velocity, 30 cm/s, ( $M=1.73$ ,  $SD=1.539$ ;  $p=0.013$ ) and lower velocity, 0.3 cm/s, ( $M= -.056$ ,  $SD=2.21$ ;  $p=0.0001$ ).

Moreover an Interaction between agent and velocity was found ( $F(2,76)=4.43$ ;  $p=0.015$ ,  $\eta_p^2 = 0.104$ ). Post-hoc showed participants rated the 3 cm/s, and experimenter applied stroke more pleasant ( $M=2.9$ ,  $SD=1.72$ ) than all other conditions (all  $p<0.05$ ).

**Softness rating:** There was a significant main effect of group ( $F(1, 38) = 5.32$ ;  $p = .026$ ,  $\eta_p^2 = 0.12$ ), with higher given softness ratings in HA ( $M = 2.37$ ,  $SD=1.03$ ) than in LA ( $M=1.5$ ,  $SD=1.35$ ).

There was also a main effect of velocity ( $F(2, 76) = 13.03$ ;  $p<.0001$ ,  $\eta_p^2 = 0.25$ ). Participants rate the optimal velocity, 3 cm/s, as the softest ( $M=2.65$ ,  $SD=1.32$ ), than higher velocity, 30 cm/s, ( $M=1.73$ ,  $SD=1.45$ ;  $p=0.001$ ) and lower velocity, 0.3 cm/s, ( $M= 1.43$ ,  $SD=1.84$ ;  $p=0.0001$ ).

Moreover an Interaction between agent and velocity was found ( $F(2,76)=3.87$ ;  $p=0.025$ ,  $\eta_p^2 = .09$ ). Post-hoc showed participants rated the 3 cm/s and experimenter applied stroke more pleasant ( $M=2.9$ ,  $SD=1.72$ ) than all other conditions (all  $p<0.008$ ) except 3cm/s and robot delivered stroke ( $M=2.43$ ,  $SD=1.67$ ;  $p=0.58$ ).

### **Interoceptive sensitivity:**

An independent samples t-test was conducted to compare interoceptive sensitivity in HA and LA groups. There was no significant difference between HA ( $M= 0.741$ ,  $SD= 0.153$ ) and LA ( $M=0.735$ ,  $SD=0.196$ ), ( $t(38) = 0.095$ ;  $p=0.924$ ).

### **Somatosensory detection task:**

An independent samples t-test was conducted to compare electro-tactile detection and near-pain threshold in HA and LA groups. There was no significant difference in tactile detection threshold in HA (M= 1.501 mA, SD= 0.561) and LA (M=1.589 mA, SD=0.731), ( $t(38) = -0.46$ ;  $p=0.647$ ). Results for near-pain tactile threshold revealed no significant difference between HA (M=1.63 mA, SD=0.639) and LA (M=1.735 mA, SD=0.731), ( $t(38) = -.483$ ;  $p=0.631$ ). Sensitivity ( $d'$ ) did not differ between HA (M=1.402, SD=0.496) and LA (M=1.699, SD=0.625), ( $t(38) = -1.66$ ;  $p=0.104$ ).

Response bias results revealed no significant difference between HA (M= 0.079, SD=0.285) and LA (M= -0.023, SD=0.234), ( $t(38) = 1.23$ ;  $p=0.222$ ).

### **3.3. Experiment 2:**

The second experiment focused only on thermal threshold tests. We investigated the relation between thermal detection threshold and alexithymia score in a new group of participants. Further, instead of selectively picking extremes of the alexithymia distribution, we sampled more evenly across the population range of alexithymia scores.

#### **3.3.1. Methods:**

**Participants:** 211 volunteers filled out the TAS-20. Twenty healthy Individuals with low, 20 with medium, and 20 with high TAS-20 total scores ( $n=20$ , bottom tertile score  $36 \leq$ ,  $n=20$ , middle Tertile score  $36 \leq$ ,  $61 \geq$ ,  $n=20$ , top tertile score  $61 \geq$ ) were selected in order to take part in thermal detection threshold. Tertile definitions were used for participant selection, to ensure the broadest range of alexithymia expression, but statistical analysis was based on a model of a continuous relation between thermoception and alexithymia. The same procedure as described above for thermal detection test was used to measure the warm and cold detection thresholds.

### 3.3.2. Results:

An ANOVA with three independent factors (LA, middle, and HA group) was conducted to compare accuracy in detecting warm temperature in LA, middle, and HA groups. There was no significant difference in warm detection accuracy among LA group ( $M= 85.09\text{ }^{\circ}\text{C}$ ,  $SD= 36.39$ ), middle group ( $M=75.16^{\circ}\text{C}$ ,  $SD= 44.13$ ) and HA group ( $M=85.09^{\circ}\text{C}$ ,  $SD=36.39$ ), ( $F(2,57)=1.11$ ;  $p=0.33$ ). There was no significant difference in cold detection accuracy within three groups. ( $F(2, 57) =0.429$ ;  $p=0.65$ ). LA ( $M=95.03\%$ ,  $SD= 22.21$ ), middle group ( $M= 80.11\%$ ,  $SD= 40.80$ ), and HA ( $M= 90.06\%$ ,  $SD= 3.57$ ).

A linear regression was calculated to predict warm detection threshold based on TAS-20 score across all 60 participants (figure 1). A significant regression equation was found ( $F(1,58)=15.14$ ,  $p<.0001$ ), with an  $R^2$  of 0.207. Similarly a regression performed to predict cold detection threshold based on TAS-20 score (figure 1). No significant regression equation was found ( $F(1,58)=0.664$ ,  $p=0.419$ ), with  $R^2$  of 0.011.

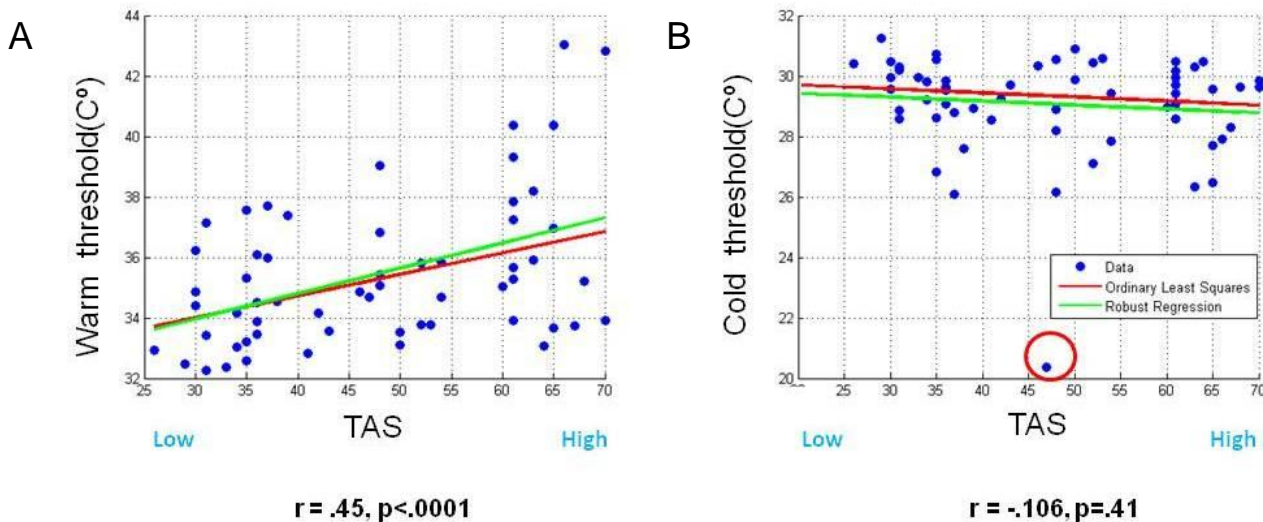


Fig 1.

A linear regression was calculated to predict warm (A) and cold detection threshold (B) based on TAS-20 score.

Further we explored relations between alexithymia subscales (Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF), and Externally Oriented Thinking (EOT) and warm and cold detection thresholds. A linear regression was calculated to predict warm detection threshold based on DIF subscale. An adjusted significance level of 0.0133 was used, since 3 separate tests were performed, but the probabilities are reported uncorrected. A significant regression equation was found ( $F(1,58)=9.92$ ,  $p=0.003$ ), with an  $R^2$  of 0.146. Likewise a significant regression equation was found between DDF subscale and warm threshold ( $F(1,58)=17.35$ ,  $p<.0001$ ), with an  $R^2$  of 0.230. No relation was found between EOT subscale and warm threshold ( $F(1,58)=3.53$ ,  $p=0.065$ ) with an  $R^2$  of 0.057.

Likewise, a linear regression was calculated to predict cold detection threshold based on DIF subscale. No regression was found between DIF subscale and cold threshold ( $F(1,58)=0.226$ ,  $p=0.63$ ) with an  $R^2$  of 0.004. There was no significant relation between either DDF or EOT and cold threshold DDF: ( $F(1,58)=0.756$ ,  $p=0.388$ ) with an  $R^2$  of 0.013; EOT: ( $F(1,58)=0.547$ ,  $p=0.463$ ) with an  $R^2$  of 0.009.

Next, an ANOVA with three independent factors (LA, middle, and HA group) was conducted to compare accuracy in detecting warm temperature in LA, middle, and HA groups. There was no significant difference in warm detection accuracy among LA group ( $M= 85.09\%$ ,  $SD= 36.39$ ), middle group ( $M=75.16\%$ ,  $SD= 44.13$ ) and HA group ( $M=85.09\%$ ,  $SD=36.39$ ), ( $F(2,57)=1.11$ ;  $p=.33$ ). There was no significant difference in cold detection accuracy within three groups. ( $F(2, 57) =0.429$ ;  $p=0.65$ ). LA ( $M=95.03\%$ ,  $SD= 22.21$ ), middle group ( $M= 80.11$ ,  $SD= 40.80$ ), and HA ( $M= 90.06\%$ ,  $SD= 3.57$ ).

A further linear regression was calculated to predict accuracy in detecting warm temperature based on TAS-20 score. No significant relation was found ( $F(1,58)=.012$ ,  $p=0.91$ ), with an  $R^2$  of 0.00. Similarly a regression performed to predict accuracy in

detecting cold temperature based on TAS-20 score and no significant regression equation was found ( $F(1,58)=0.007$ ,  $p=0.93$ ), with an  $R^2$  of 0.00. Also a regression was performed to predict accuracy in detecting warm and cold temperature based on warm and cold detection threshold. No significant regression equation was found, neither for warm ( $F(1,58)=.42$ ,  $p=0.52$ ) with an  $R^2$  of 0.007, nor for cold temperature ( $F(1,58)=2.05$ ,  $p=0.15$ ) with an  $R^2$  of 0.034.

### **3.4. Discussion:**

Alexithymia is defined by difficulties in identifying and describing feelings, and a tendency to focus on external events rather than inner experiences (Taylor et al. 1991). Alexithymia has been characterised as a difficulty in cognitively mapping feeling states onto internal bodily responses (Taylor 2000). Besides, alexithymia has been shown to be associated with atypicalities in sensory processing. While some studies have found hypersensitivity to some somatosensory modalities (e.g. pain, touch, heat, and visceral stimulation) in high alexithymics (Kosturek et al., 1998; Kano et al. 2007; Katz et al. 2009; Nyklicek and Vingerhoets 2000) others did not find similar results (De Zwaan et al., 1996; Millard and Kinsler 1992; Cox et al., 1994 – see table 1 for summary). Therefore, it seems possible that emotion recognition difficulties in alexithymia could be caused by atypical somatosensory processing.

We have investigated this issue using QST as an established gold standard method for assessing multiple somatosensory submodalities. Experiment 1 showed that perception of warm temperature was the only somatosensory sub-modality that differed between HA and LA, with HA showing a higher threshold for detecting warm

temperature that LA. Many QST batteries measure cold and warm thresholds in separate blocks of trials (Rolke et al., 2006a; Rolke et al., 2006b). This practice confounds sensitivity to thermal stimuli with response bias, so that between-group differences in thresholds could reflect differences in bias rather than in perceptual sensitivity. For example, a liberal decision criterion, due to some non-perceptual factor such as trait impulsivity, would lead to low thresholds, and could be mistaken for high thermosensitivity. Importantly, we randomly intermixed cold and warm stimuli, and asked participants to identify the direction of temperature change that they had detected. In this arrangement, a participant with a liberal decision criterion would make less accurate judgments than one with a more conservative criterion. Crucially, we found no difference in accuracy between HA and LA groups in detecting either warm or cold temperature. While this null result cannot rule out any contribution from response bias, it does clarify interpretation of our threshold measures. Specifically, the higher threshold for warmth detection in the HA group, compared to the LA group, appears to be a genuine difference in perceptual sensitivity within the warm thermoreceptive pathway, rather than merely a response bias.

Experiment 2 sought to replicate this result in a new sample, and across the entire distribution of alexithymia trait expression. We modelled thermoception thresholds as a continuous function of alexithymia scores, and found a strong linear relation between warmth perception threshold and level of alexithymia, with higher levels of alexithymia being associated with lower sensitivity for warmth. Sub-analyses of different TAS subscales suggested the correlation was due to the feeling facets of alexithymia, rather than external orientation of thought.

Activation of the insula during emotion recognition is reduced in alexithymics, possibly explaining the cognitive and affective impairment of high alexithymics (Kano et

al., 2003; Reker et al., 2010; see Moriguchi and Komaki., 2013 for a review). Interestingly, the insula also plays a key role in thermoception (for a review see Rolls., 2010). Alexithimics also lack emotional warmth and empathizing with others, for instance while observing painful stimulation (Moriguchi et al., 2003; Bird et al., 2010). Here we showed that in addition to cognitive and affective problems, alexithymia is associated with specific low-level somatosensory deficits, namely low sensitivity to warmth. Alexithymic deficits in cognitive processing of emotion might therefore be grounded in the low-level thermoceptive system: insensitivity to physical warmth could potentially explain the lack of social warmth found in high levels of alexithymia.

Unlike some previous studies, we used a fairly comprehensive battery, and physiologically-selective tests to investigate the links between somatosensory function and alexithymia. The QST approach is inspired by the fact that different qualities of somatosensation are each transmitted by distinct neural pathways, associated with specific receptor types, and afferent fiber types. Thus, the specific association we found between alexithymia and warmth perception can be linked to specific physiological and anatomical substrate. Importantly, we found effects that were defined by physiological substrate, rather than by physical stimulus dimensions, such as temperature. That is, alexithymia was associated with altered perception of non-noxious warmth, while perception of cold and noxious heat were unaffected. The sensation of warmth is transmitted via unmyelinated C-fibers whereas nonpainful cold is conducted by small myelinated A $\delta$  fibers (Schepers and Ringkamp., 2008). This may indicate that while the pathway for perception of cold is intact in HA, the warm-conducting pathways are specifically hypoactivated in persons with high alexithymia. Some neuroimaging studies suggest that the peripheral distinction between warm and cold processing is also maintained in central thermoceptive processing. While both pathways activate the insula



(Rolls et al., 2008; Olausson et al., 2005; Craig et al., 1996; Casey et al., 1996; Davis et al., 1998), while cold stimulation preferentially activated secondary somatosensory cortex (Casey et al., 1996) warm stimulation mostly activates S1, anterior cingulate and the opercular-insular areas (Casey et al., 1996; Iannetti et al., 2003). This indicates that central processing of warmth and cold are at least partially different

Several studies have linked somatosensory processing of thermoception to social-affective processing. In particular, areas such as insular cortex were implicated in processing both physical warmth but also social warmth (Williams and Bargh., 2008; Inagaki and Eisenberger., 2013). On one view, physical warmth has been interpreted as internal, interoceptive signal, associated with both physical and socio-affective contexts (Inagaki and Eisenberger., 2013 ; Williams and Bargh., 2008). Others have urged caution in linking social emotions to somatic sensations. For example, several studies have reported overlap in the fMRI activations during noxious stimulation, and during social exclusion. However, it remains unclear whether the phenomenology of these experiences is truly comparable. Further, the common activation of largely non-specific brain areas could involve a reverse inference fallacy (Iannetti and Mouraux., 2010). Our study provides direct mechanistic evidence for the possible missing link between sensation and social emotion in such arguments, at least in the case of alexithymic traits, by showing a plausible, low-level neural impairment relevant to alexithymia.

In one series of studies, individuals with low levels of warm social interactions reported more taking more frequent hot showers, and appeared to unconsciously substitute physical for social warmth (Bargh and Shalev, 2012). This effect remains controversial, and others have failed to replicate the association (Donnellan et al., 2015). However, our results potentially point to an explanation: individuals in our studies with the poor socio-affective cognition that characterises alexithymia appeared relatively insensitive to

warmth. They might therefore choose more frequent exposures or higher temperature levels to achieve a target perceptual effect.

Warm threshold was the only somatosensory or interoceptive function among the six QST subtests that was significantly linked to alexithymia scores. We cannot draw strong conclusions from subtests with null results. However, the overall pattern across subtests does point to a specific link between alexithymic traits and the warm thermoceptive pathway. Moreover, the association found in experiment 1 with warm, but not cold, thermoception was replicated in experiment 2.

The results of one particular test deserve special comment. Several authors have speculated that the c-tactile mechanoreceptor pathway may play a special role in social emotion. This pathway's unique role in pleasant touch might be relevant to behaviours such as grooming and caressing, providing a link between a specific somatosensory submodality and positive social emotion. We therefore included an affective touch test in our battery, although it is not a classic element of most QST batteries. Quantitative testing of affective touch is problematic, for several reasons. First, the relevant receptors and pathway cannot be activated selectively: any stimulus that activates c-tactile mechanoreceptors will also activate light touch receptors and their associated A $\beta$  fibres. Second, the assessment relies on the observation that subjective pleasantness of stroking varies with movement velocity with a similar tuning profile to individual c-tactile afferents recorded microneurographically (Olausson et al., 2002). However, similar velocity tuning for pleasantness ratings cannot prove that pleasantness judgments derive exclusively from the c-tactile pathway. In our experiment 1, individuals with high alexithymia gave overall higher pleasantness ratings than those with low alexithymia, but no interaction with stroking velocity was found. In the absence of

velocity-specific interaction, differences in overall ratings between the alexithymia groups might not reflect differences in specific sensory channels, but general biases in judgement. Thus, we found no evidence for a specific deficit in affective touch pathways associated with alexithymia. Instead, we unexpectedly found that high alexithymics gave generally higher pleasantness ratings overall, but without any interaction with velocity.

Several limitations of our method and results should be kept in mind. Some QST subtests may have been too insensitive to detect differences between HA and LA. Thus, we may have missed associations between alexithymia and other sub-modalities, beyond warm thermoception. Second, our affective touch test differed from the other QST subtests in two ways. First, it was based on a subjective rating, rather than a conventional psychophysical judgement. Second, it could not show the same level of pathway specificity as the other tests. Other limitations relate to the sample. We could not clinically assess all relevant comorbidities (e.g. anxiety, eating disorder, somatoform disorder, etc) although our screening procedure did exclude severe depression, and history of any psychiatric or neurologic disorders.

Future studies needed to be done to further investigate the function of somatosensory pathways and whether the activation of somatosensory correlates differs during emotion processing in alexithymia. Particularly, neuroimaging studies are needed to provide neural evidence for the lower sensitivity to warmth in high alexithymics.

Overall, current study provides evidence that alexithymia does not characterise only with cognitive and affective deficits but rather it also involves low level somatosensory alterations and the cognitive dimension of this trait might be partially grounded in somatosensory level.

## CHAPTER 4: Processing emotions through faces

In humans, emotional information is displayed and conveyed by the face, the body, and speech prosody. Importantly, the human face is the most important body part for communicating emotional information to others.

Face perception refers to the early processing of the visual features of faces and their spatial configuration, which mostly relies on early sensory cortices (Adolphs, 2002). In contrast, recognition does not solely rely on the visual structure of the stimulus; rather, additional knowledge about the stimulus that has been held in memory is necessary. Based on one of the most important models of face processing, proposed by Haxby, Hoffman, and Gobbini (2000) specific neural regions are assigned to particular functional components of face processing. According to this model, neurons in inferior occipital cortex selectively respond to early feature processing. Gaze, lip movement, and expression processing mainly occurs in the superior temporal sulcus. More specifically, neuroimaging studies have shown that emotional expressions are processed in the amygdala and the insula, speech perception is processed in auditory cortex, and spatial attention is processed in the intraparietal sulcus (Haxby et al., 2000).

Emotional expressions might be processed at a level after early perceptual processing that already requires recognition, but prior to the processing of other facial information like form, gender, and age (Adolphs, 2002). On the other hand, some studies have shown that emotional facial perception at least partially requires information about the configural relations between facial features (Calder et al., 2000).

Observing emotional faces provides a wide range of social and affective information. Perceiving emotion normally leads to changes in somatic systems, including visceral, endocrine,

and autonomic changes, as well as changes in the musculoskeletal system such as mimicry (Adolphs, 2002; Niedenthal, 2007; Niedenthal et al., 2010; Gallese & Sinigaglia, 2011).

#### **4.1. The recognition of emotional faces**

There are different possible mechanisms for recognizing emotions through faces, and these mechanisms are linked to specific neural structures. Below, three different mechanisms that are known to play a role in the recognition of facial expressions are considered. The focus of this chapter is mainly on the first two cognitive mechanisms.

##### **4.1.1. Perceptual mechanisms**

One possibility might be that emotions are recognized through perception of stimulus features. This mechanism suggests that geometric and visual properties of emotional faces might be sufficient for identifying, categorizing, and discriminating facial expressions. Although this mechanism for recognition is poor, it might be sufficient for discriminating or labeling different emotions in a matching task when the labels of facial emotions are provided beforehand. Studies on the categorization of morphed faces in normal participants showed a distinct perceptual difference between facial expressions, even for structurally similar emotional expressions (Calder et al., 2001; Gelder et al., 1997). This evidence reveals the important role of categorical perception in recognition of facial expressions.

Additionally, mechanisms of selective attention focus the processing resources of the visual system by functioning as an information gating mechanism (Parkhurst et al., 2002). This visual attention mechanism plays an important role in the selection of relevant visual information for recognizing facial expressions.

#### **4.1.2. Recognition through simulation**

An observed facial expression may be simulated within the somatosensory and motor systems of the observer (Niedenthal, 2007). This simulation mechanism could trigger the conceptual knowledge necessary for recognizing that emotion. It has been shown that the experience of an emotion is correlated with the expression of that emotion (Rosenberg & Ekman, 1994). Therefore, producing somatovisceral responses and emotional face expressions could affect our emotional experience. Previous studies have shown that covert emotional states are linked with both bodily reactions (e.g., visceral changes and rapid facial muscle movements) and overt motor responses. Recent emotion theories have suggested that emotional face expressions are automatically perceived by embodying the overt motor behavior and covert somatovisceral changes associated with the observed emotion (Bastiaansen et al., 2009; Gallese & Sinigaglia, 2011; Niedenthal et al., 2010; Oberman et al., 2007). The simulation required for facial emotion recognition generally involves somatomotor changes (mimicry) and somatovisceral changes (changes in heart rate).

#### **4.1.3. Recognition through generation of associated knowledge**

Another recognition mechanism focuses on the impact of our past experiences with emotions. This mechanism involves components that are not related to perception of the visual structure of the face. For instance, when we see a happy facial expression, we might recognize the identity of the person making that expression, and recall that she is likely to jump or laugh loudly when she is happy. The association of previous experience and knowledge with the perceived emotion facilitates recognition of that emotion. The neural pattern for implementing the above mechanism requires the binding of information between separate neural representations (Adolphs, 2002).

Recent evidence suggests that individual emotional skills (Meaux et al., 2014), empathic dispositions (Choi et al., 2014), and personality traits might affect the processing of emotional stimuli. Since the rapid perception of negative cues in social environments is highly adaptive, the effect of personality traits on the processing of emotional stimuli, such as emotional face expressions, is an important avenue for research. One relevant trait is alexithymia, a multifaceted personality construct that is characterized by deficits in identifying, differentiating, and describing feelings (Herbert et al., 2011; Parker et al., 2008; Taylor et al., 1991). Importantly, people with high levels of alexithymia exhibit difficulties not only in processing their own emotions, but also in processing the emotions expressed by others (Parker et al., 1993; Sifneos, 1973). Alexithymic individuals show altered recognition of emotional stimuli (Grynberg et al., 2012; Ihme et al., 2014) and decreased activation of the amygdala in response to emotional stimuli (Jongen et al., 2014; Moriguchi & Komaki, 2013), particularly negative stimuli (Kugel et al., 2008; Pougá et al., 2010; Reker et al., 2010; for a recent meta-analysis, see van der Velde et al., 2013). However it is unknown whether and how different mechanisms for recognizing emotion through faces might be similarly affected. This chapter describes three studies (Experiments 3-5) that investigated the perceptual and visceral mechanisms for recognizing facial expressions in people with low and high levels of alexithymia.

Experiment 3 used an explicit, category-specific emotional facial recognition task to investigate whether there is a difference between people with high and low levels of alexithymia in recognizing facial expressions at a late behavioral level.

Then, Experiment 4 investigated the selective attention mechanism as a subcomponent of the perceptual mechanism for recognizing emotional facial expressions. Specifically, eye movement patterns were used as a reliable measure of changes in visual attention between different emotional expressions in groups of participants with high and low levels of alexithymia. Visual attention is directed to particular regions of face to select visual signals for processing

facial emotions. Visual scanpath can be used as an objective psychophysiological marker of visual attention (Horley et al., 2003). The visual scanpath refers to fixations and saccadic movements that trace the direction and duration of eye movements when observing a face (Noton & Stark, 1971b). Therefore, studying eye movement patterns is a way of understanding differences in visual attention between facial expressions of different emotions.

Finally, Experiment 5 studied visceral responses, as a subcomponent of the simulation mechanism, to investigate how alexithymia influences this mechanism of emotion recognition. Heart rate was used as a reliable measure of autonomic nervous system (ANS) activity to study the modulation of somatovisceral responses to different facial expressions, and, more importantly, the effect of alexithymia on this simulation mechanism.



#### **4.2. Experiment 3: Investigating emotional face processing in alexithymia**

Alexithymia has been described as a reduced ability to recognize and communicate emotions, as well as a limited capacity for imagination (Taylor and Bagby, 2004). Moreover, people with high levels of alexithymia exhibit interpersonal impairments and difficulties in decoding others' facial expressions (Prkachin et al., 2009; Berthoz et al., 2008; Grynberg et al., 2010; Guttman & Laporte, 2002; Vanheule et al., 2007). Accurate perception of emotional expressions allows people to adjust to the emotional states and behaviors of others (Critchley et al., 2000; Montagne et al., 2005). The processing of emotional facial expressions is one of the fundamental requisites in interpersonal relations and social communication. For instance, a lack of empathy, which has been shown in alexithymia (Moriguchi et al., 2007; Singer et al., 2009; Bird et al., 2010), could be, to some extent, due to the impairment in recognizing others' facial expressions of emotions.

Some studies have shown impaired facial emotion recognition in alexithymia (Mann et al., 1994; Lane et al., 1996; Lane et al., 2000). Others suggest that there is no difference between high and low alexithymics in emotion identification, or, if there is a difference, that it is only revealed under specific demanding conditions (e.g., time constraints and high speed conditions, masked faces, etc. Mc Donald & Prkachin, 1990; Mayer et al., 1999; Parker et al., 2005). Hence, it is not clear whether this difference in emotion identification reflects a consistent difficulty at a late behavioral level, or a difference only in earlier stages of emotion processing. This study was designed to test categorical perception as a part of a perceptual mechanism for recognizing facial emotions in alexithymia. To this end, a morphing task was used to measure facial emotion recognition in alexithymia. The hypothesis was that a more rigorous

psychophysical paradigm like a dynamic facial morph task could be a tool for demonstrating any subtle emotion recognition difficulties at an explicit and behavioral level. It was expected that participants who scored highly on the TAS-20 would perform worse on the emotion recognition task, particularly with negative (fearful) facial expressions.

#### **4.2.1. Methods**

##### **Participants**

Three-hundred university students completed the 20-item Toronto Alexithymia Scale (TAS-20; Taylor et al., 2003). Individuals with high and low total TAS-20 scores ( $n = 17$ , top quartile score  $> 61$ ;  $n = 17$ , bottom quartile score  $< 36$ ) were selected in order to obtain a sample with as large a variance on alexithymia as possible. The alexithymia module of the structured interview from the Diagnostic Criteria for Psychosomatic Research (DCPR) (Mangelli et al., 2006; Porcelli & Rafanelli, 2010; Porcelli & Sonino, 2007), previously used in alexithymia research (Grandi et al., 2011), was also used in the present study to further confirm the presence or absence of alexithymia. In addition, due to the high association between alexithymia and depression (Allen et al., 2011; Hintikka et al., 2001; Honkalampi et al., 2000), the Beck Depression Inventory (Beck et al., 1961) was administered to exclude participants with high levels of depression. Participants were included in the study if i) they had no history of neurological, major medical, or psychiatric disorders, and ii) their scores on the TAS-20 and the DCPR were congruent. Two participants with a high TAS-20 score and a low DCPR score were excluded. No participant reported a high level of depression on the BDI. All

participants had equivalent educational backgrounds and were students at the University of Bologna. Thirty-four healthy volunteers were selected to take part in the experiment after being screened for alexithymia: 17 high alexithymic (HA) participants (TAS, mean  $\pm$  standard deviation:  $64.94 \pm 4.37$ ; 8 males; mean age  $\pm$  SD:  $22.7 \pm 1.93$  years old) and 17 low alexithymic (LA) participants (TAS, mean  $\pm$  standard deviation:  $32.1 \pm 2.98$ ; 8 males; mean age  $\pm$  SD:  $23.1 \pm 2.36$  years old). The two groups were matched in terms of sex and age. The two groups did not differ in terms of BDI score ( $t(32) = .957$ ;  $p = .345$ ). All participants gave their written informed consent to participate after they were informed about the procedure and the purpose of the study. The study was designed and performed in accordance with the ethical principles of the Declaration of Helsinki, and was approved by the Ethics Committee of the University of Bologna Psychology Department.

### **Stimuli and task**

Facial emotion recognition was assessed using a dynamic facial morph task. The task consisted of 30 randomized trials, each showing a dynamic facial expression changing from neutral into one of three basic facial expressions: fear, happiness, and disgust. Photographs of 4 men and 4 women were chosen from a validated facial emotion database (Pictures of Facial Affect) assembled by Ekman and Friesen (1976). Stimulus size was 17 x 24.5 cm, and the pictures were trimmed to fit an oval with a black background to remove hair and non-facial contours. Participants sat in a relaxed position on a comfortable chair in front of a 17" PC monitor (refresh rate: 60 Hz) at a distance of approximately 57 cm. The pictures of facial expressions were morphed, using FantaMorph software (Abrosoft, 2005), from 0% (neutral) to 100% of a particular emotion in 1% steps. Each step lasted 1 sec, resulting in a video with a total duration of 100 sec (Figure 1). As soon as participants recognized the emotion in the video, they

pressed the button corresponding to that emotion. They used the index and middle fingers of the right hand and the index finger of the left hand on three keyboard buttons (D, J, and K). The emotions assigned to the buttons were counterbalanced across participants. Reaction times, percentages of correct responses, and inverse efficiency scores were analyzed.

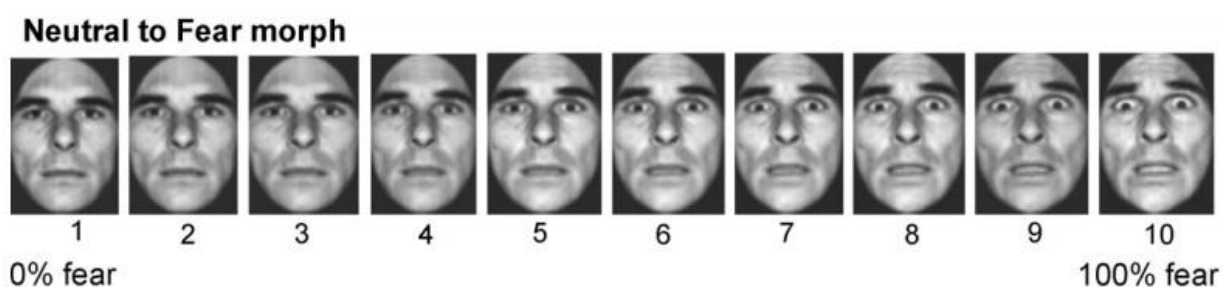


Figure1. Graphical representation of a dynamic facial morph trial from 0% fear (neutral) to 100% fear.

#### 4.2.2. Results

A mixed factors analysis of variance (ANOVA) with Group (LA and HA) as a between-participants factor and Emotion type (fear, happiness, disgust) as a within-participants factors was performed on reaction times, accuracy, and inverse efficiency scores (IES = reaction time/accuracy). The groups did not differ in their reaction times ( $F(1, 32) = .530$ ;  $p = .471$ ;  $\eta_p^2 = .016$ ). A significant main effect of Emotion type was found ( $F(2, 64) = 2.084$ ;  $p < .0001$ ;  $\eta_p^2 = .656$ ). Post-hoc comparisons (Newman keuls) revealed faster reaction times to happy expressions (24712 ms) than to the other emotions (fear = 32791 ms; disgust = 36705 ms; all  $p$ -values  $< .0001$ ). Also, the slowest reaction times were in response to disgust (36705 ms; all  $p$ -values  $< .001$ ). Similarly, the ANOVA on accuracy scores showed no significant difference between groups

( $F(1,32) = .583$ ;  $p = .450$ ;  $\eta_p^2 = .0178$ ). A significant main effect of Emotion type was revealed ( $F(1,32) = 10.382$ ;  $p < .001$ ;  $\eta_p^2 = 2.449$ ). Post-hoc tests showed that participants were more accurate in responding to happy expressions (95.88%,  $SD=7.43$ ) compared to the other emotions (fear = 91.12%,  $SD=12.5$ ; disgust = 87.059%,  $SD=15.67$ ; all  $p$ -values  $< .02$ ). They were less accurate in responding to disgust (87.059%) compared to the other emotions (all  $p$ -values  $< .04$ ). No interactions with the factor Group were significant (all  $p$ -values  $\geq .93$ ). Finally, the ANOVA on inverse efficiency scores revealed no difference between groups ( $F(1,32) = .095$ ;  $p = .759$ ;  $\eta_p^2 = .003$ ). A significant main effect of Emotion type was revealed ( $F(2, 64) = 67.38$ ;  $p < .0001$ ;  $\eta_p^2 = .678$ ), showing significantly lower scores (reflecting better performance) for happy expressions (256.6 ms; all  $p$ -values  $\leq .0001$ ), compared to fear (358.78 ms) and disgust (421.57 ms). In addition, the IES for disgust was significantly higher (reflecting worse performance) than for the remaining emotion types (all  $p$ -values  $\leq .00015$ ).

#### **4.2.3. Discussion**

Several investigators have found people with alexithymic characteristics to be less accurate at recognizing emotional expressions (Jessimer & Markham, 1997; Lane et al., 1996; Lane et al., 2000; Parker et al., 2005; Parker et al., 1993). However, other studies report contradictory results (Parker et al., 2005; Mc Donald & Prkachin, 1990; Mayer et al., 1999). Here, we used a dynamic facial morph task to study whether impairment in facial emotion recognition could be seen in an explicit task, and at a late stage of emotional face processing.

The results showed no differences between high and low alexithymics in recognizing facial expressions of happiness, fear, and disgust--neither in terms of

accuracy, nor reaction times. Considering the characteristics of our task (e.g., the temporal duration, and the gradual emergence of the emotional expression in the morph), these results indicate that there was no difference between high and low alexithymics in recognizing emotional faces at a late behavioral level. This suggests that the categorical perception of facial expressions at a behavioral level does not differ between high and low alexithymia.

Additionally, we found that, for both high and low alexithymics, the happy facial emotion was the easiest type of facial expression to recognize, while disgust was the hardest. However, it is of great importance to note that electrophysiological studies on lower perceptual, structural levels of face processing in alexithymia have shown that the early stage of visual facial expression processing for happiness, fear, and disgust is altered. For instance, Scarpazza et al. (2015) showed that the emotional modulation of the N170 ERP component that indexes structural processing of human faces is impaired in high alexithymia. In low alexithymics, the N170 is modulated by facial expressions of fear and disgust, but this modulation is absent in high alexithymics. In accordance with the results of Experiment 3, they did not find any differences in facial emotion processing at a behavioral level (Scarpazza et al., 2015). This suggests that the emotion processing difficulties in alexithymia may only appear under exceptionally demanding circumstances, or, alternatively, at early stages of emotional face processing.

Further studies are needed to better understand the perceptual mechanism of emotional face recognition, specifically for categorical perception of facial expressions. These studies could use a dynamic morphing task with a shorter duration, and different percentages of emotional expressions in the morphs (e.g., up to 40%, up to 60%, up to 80% of emotional expression) with respect to the one used in the present study (e.g. up

to 100% of emotion). This might allow the detection of subtle differences in explicit facial emotion recognition between high and low alexithymics.

#### **4.3. Experiment 4a: Eye movement patterns during emotional face processing**

The social information exhibited by facial expressions is crucial for interpreting others' mental states, goals, and emotions, a process which affects interpersonal interactions and social behaviors. The complex visual information displayed by faces must be integrated in order to correctly classify affective expressions. Searching and scanning the target visual stimulus is the primary stage of visual processing, and is done with eye movements. Eye movements refer to voluntary or involuntary movements of the eyes, which help to acquire, fixate, and track visual stimuli (O'Regan et al., 1983).

It has been shown that normal individuals obtain visual information mostly from the eye and mouth regions of faces (Henderson, Williams, & Falk, 2005; Yarbus, 1967). The recognition of facial expressions from various types of face stimuli, including only the eye region, only the mouth region, the eye and mouth regions together, a full face except for a missing nose, and a full face, were studied to investigate analytic and holistic modes of facial emotion processing (Kestenbaum, 1992). Facial expressions of fear, anger, and surprise were better recognized from the eye region than from the mouth region. In contrast, happy facial expressions were better recognized from the mouth region. Also, Adolphs et al. (2005) showed that normal participants obtained visual information mostly, and consistently, from the eye region when discriminating fearful, angry, and sad facial expressions. Moreover, they found that eliminating the eye region from faces caused a significant reduction in recognition accuracy for these facial expressions (Adolphs et al., 2005).

Importantly, studies on visual scan path in clinical populations (e.g., autism and schizophrenia) have not revealed any preferences for a specific facial area (Dalton et



al., 2005; Hernandez et al., 2009; Loughland, Williams, & Gordon, 2002; Streit et al., 1997). Additionally, brain lesions can lead to impaired patterns of eye movements and, therefore, deficits in emotional face recognition. For instance, patients with amygdala lesions are impaired at decoding fearful faces, and, interestingly, these patients are not able to use visual information from the eye region. This was shown by a study that showed decreased spontaneous fixations on the eye region during passive observation of facial expressions (Adolphs et al., 2005).

People with alexithymia show alterations in emotion processing. They exhibit difficulties not only in identifying and discriminating their own emotions, but also in understanding others' emotions (Kugel et al., 2008; Prkachin et al., 2009). In the present study, we investigated an early stage of visual processing at which eye movements are used to acquire information about the face. Using both an explicit (emotion discrimination) and implicit (gender discrimination) tasks, we tested whether people with high and low levels of alexithymia show different patterns of eye movements for exploring faces. Numerous studies have investigated the ability to recognize emotions from faces in alexithymia, using various methods such as discrimination, matching, and free labeling. However, to our knowledge, no study had yet explored whether the difficulty in emotion recognition with alexithymia results from an alteration in the perceptual mechanism for emotional face recognition. Specifically, a visual attention mechanism, as indexed by visual scanning behavior, might be altered.

Visual scanning of facial expressions can be evaluated using eye tracking technology, by measuring the fixation percentage within each region of the face during a facial emotion recognition task. This technique discloses the source of visual information in the observed object (e.g., eyes or mouth), allowing us to explore the

strategies used by high and low alexithymics exposed to a certain stimulus (e.g., an image of a facial expression).

The current study investigated, for the first time, the eye movement patterns of high and low alexithymics during facial expression categorization (fear, disgust, happiness, or neutral). Two parameters--the percentage of fixations prior to the response, and the first saccade--were used to investigate visual attention during emotional face processing.

We hypothesized there would be a significant difference between high and low alexithymic groups at the stage of the first saccade. Moreover, we expected that the percentages of fixations in each face region would be modulated by the emotional expression of the face in low alexithymics, but not in high alexithymics. Specifically, based on previous findings that high alexithymics have greater difficulties in recognizing negative emotions (Kugel et al., 2008; Scarpazza et al., 2015; Pouga et al., 2010; Reker et al., 2010; for a recent meta-analysis, see van der Velde et al., 2013), we expected a difference between high and low alexithymia groups in eye movement patterns while viewing fearful facial expressions.

#### **4.3.1. Methods**

##### **Participants**

Three-hundred university students completed the 20-item Toronto Alexithymia Scale (TAS-20; Taylor et al., 2003). Individuals with high and low total TAS-20 scores ( $n = 20$ , top quartile score  $> 61$ ;  $n = 20$ , bottom quartile score  $< 36$ ) were selected in order to obtain a sample with as large a variance on alexithymia as possible. The alexithymia module of the structured interview from the Diagnostic Criteria for Psychosomatic Research (DCPR) (Mangelli et al., 2006; Porcelli & Rafanelli, 2010; Porcelli & Sonino,

2007), previously used in alexithymia research (Grandi et al., 2011), was also used in the present study to further confirm the presence or absence of alexithymia. In addition, due to the high association between alexithymia and depression (Allen et al., 2011; Hintikka et al., 2001; Honkalampi et al., 2000), the Beck Depression Inventory (Beck et al., 1961) was administered to exclude participants with high levels of depression. Participants were included in the study if i) they had no history of neurological, major medical, or psychiatric disorders, and ii) their scores on the TAS-20 and the DCPR were congruent. Two participants with a high TAS-20 score and a low DCPR score were excluded. No participant reported a high level of depression on the BDI. All participants had equivalent educational backgrounds and were students at the University of Bologna. Forty healthy volunteers were selected to take part in the experiment after being screened for alexithymia: 20 HA participants (TAS, mean  $\pm$  standard deviation:  $64.60 \pm 4.08$ ; 8 males; mean age  $\pm$  SD:  $23.5 \pm 2.41$  years old) and 20 LA participants (TAS, mean  $\pm$  standard deviation:  $31.3 \pm 3.21$ ; 8 males; mean age  $\pm$  SD:  $23.8 \pm 2.7$  years old). The two groups were matched in terms of sex and age. The two groups did not differ in terms of BDI score ( $t(38) = .572$ ;  $p = .570$ ). All participants gave their written informed consent to participate after they were informed about the procedure and the purpose of the study. The study was designed and performed in accordance with the ethical principles of the Declaration of Helsinki, and was approved by the Ethics Committee of the University of Bologna Psychology Department.

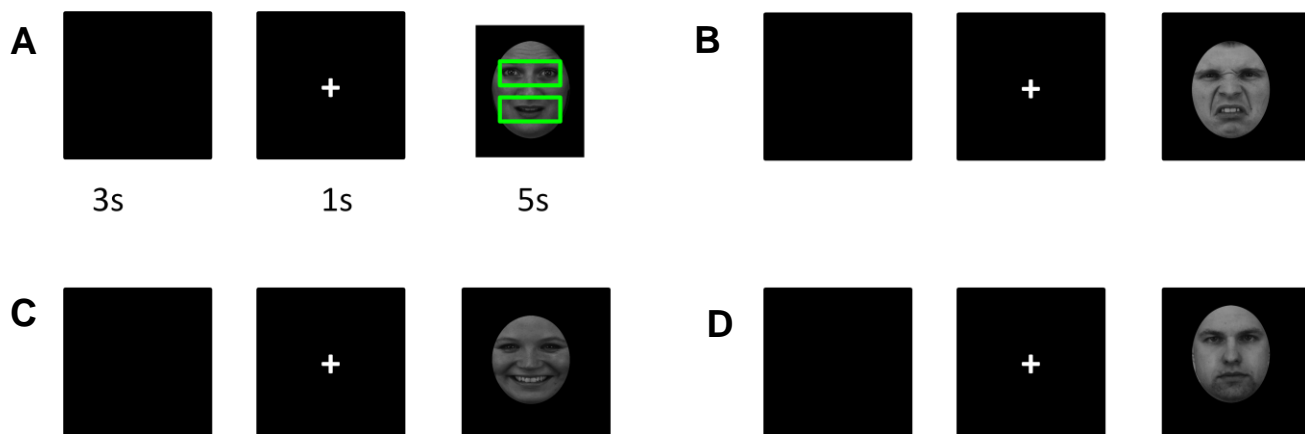
### **Stimuli and procedure**

The stimuli used in Experiments 4 and 5 were chosen from the same database (Karolinska Directed Emotional Faces (KDEF) database; Lundqvist et al., 1998). A pilot study was carried out to select a set of stimuli in each emotional category that would be similarly difficult to recognize. In the pilot study, 268 stimuli were taken from the KDEF

and rated by pilot participants on valence, arousal, and the amount of perceived fear, happiness, sadness, disgust, surprise, and anger in each stimulus on a Likert scale from 1-9 for arousal and perceived emotion, and from -2 to 2 for valence. Ultimately, participants were asked to label each of the emotional facial expressions using one of the following labels: fear, happiness, disgust, and neutral. Finally, 10 stimuli from each category of emotion were selected for Experiments 4. (A separate set of stimuli was selected for Experiment 5.). There were no significant differences in arousal ratings between the different emotional categories (fear, happiness, disgust;  $F(2,27) = .765$ ;  $p = .475$ ). Similarly an ANOVA was performed on ratings for perceived emotion on the same three types of emotion and no significant difference was revealed ( $F(2,27) = .81$ ;  $p = .50$ ).

The stimuli used in Experiment 4 consisted of 40 black and white photographs showing facial expressions (4 emotions x 2 genders x 5 characters). Each image depicted one of the basic emotions (fear, happiness, disgust, or neutral) from the validated KDEF database (Lundqvist et al., 1998; Goeleven et al., 2008). Stimuli subtended a horizontal visual angle of  $14^\circ$  and a vertical visual angle of  $20^\circ$ . The pictures were trimmed to fit an oval with a black background to remove hair and non-facial contours. Participants were comfortably seated in a silent, dimly lit room in front of the screen, at a viewing distance of 60 cm from the eye-tracker and 75 cm from the screen. The eye-tracker was positioned under the screen, and was centered relative to both the screen and the participant. Eye movements were recorded using a Pan/Tilt optic eye-tracker (Eye-Track ASL-6000) which registers real-time gaze at 50 Hz. Data acquired during the facial emotion recognition task were analyzed offline using EyeNal Analysis Software (ASL). Fixation percentages were measured in two specific areas of interest (AOI): the eye region, corresponding to the 12.6 x 3.3 cm rectangle at the top,

centered around the eyes, and the mouth region, corresponding to the 12.6 x 3.3 cm rectangle at the bottom, centered around the mouth. Eye movements and behavioral responses were collected throughout the experiment and stored for offline analysis. Participants placed their chin on a chin rest and were asked to remain as still as possible to avoid confounding effects on eye-movements. The whole experiment was conducted in a dark room to facilitate eye-movement recording. The experimental session began with calibration of the eye-tracker device, during which the participant fixated nine specific points on the computer screen. Then the participant performed a discrimination task with four different types of facial expressions: fear, disgust, happiness, and neutral. The task consisted of 40 randomized trials. Each trial began with 3 s of a black screen, followed by 1 s of a fixation cross. After the fixation cross disappeared, the stimulus appeared for 5 s (Fig. 1).



**Figure 1.**

Graphical representation of the trial structure in the behavioral task. Task started with 3s of black page continued by 1s fixation cross and then 5s of stimulus presentation. (A-D) represents an example of fear, disgust, happy, and neutral facial expression stimuli. The fixation percentage was measured for two specific areas of interest, eye and mouth region corresponding to the 12.6 x 3.3 cm rectangular (A).

Participants were told to answer as soon as they identified the emotional facial expression, but the duration of the stimulus was independent of the response. Four buttons on the keyboard (1, 2, 8, and 9) corresponded to fear, disgust, happiness, and neutral, and participants responded with the middle and index fingers of their right and left hands. They were told to keep their gaze on the monitor, and not to look at the buttons. Prior to the main experiment, a practice session was performed to make sure participants memorized the response buttons. The response buttons were counterbalanced across participants.

The first saccade landing position (i.e., the location of the second fixation), and fixation percentages in the eye and mouth regions during the presentation of the stimulus (5 seconds) were analyzed with a mixed factors ANOVA with AOI (eye region, mouth region) and emotion (fear, disgust, happiness, and neutral) as within-subjects variables and group (HA, LA) as a between-subjects variable. To compensate for violations of sphericity, Greenhouse–Geisser corrections were applied whenever appropriate (Greenhouse & Geisser, 1959) and corrected p-values (but uncorrected degrees of freedom) are reported. Post-hoc comparisons were performed using the Newman–Keuls test.

#### **4.3.2. Results**

##### **Fixation percentages during the presentation of the stimulus**

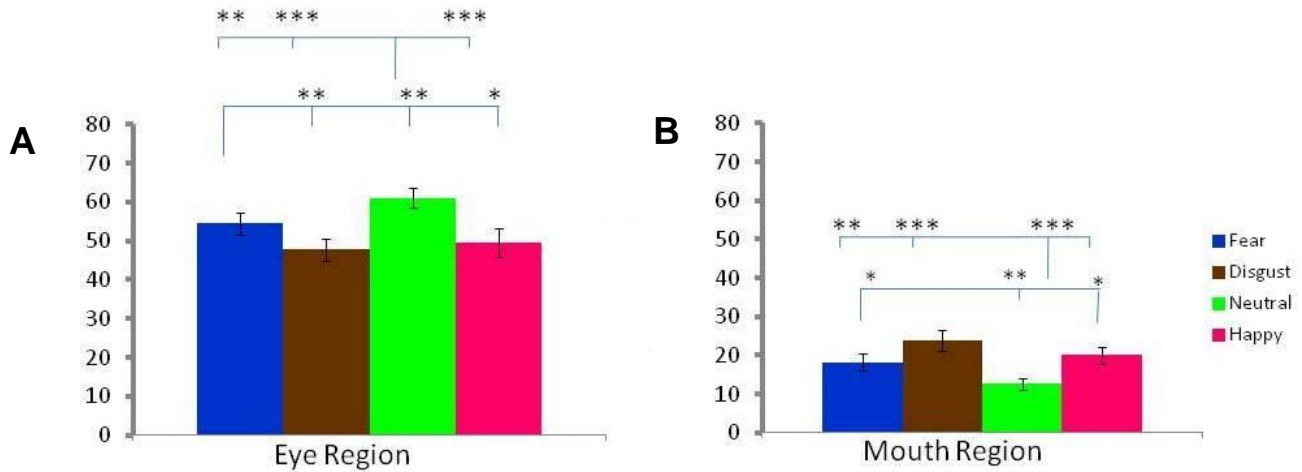
The ANOVA showed no significant main effect of group ( $F(1,38) = .226$ ;  $p = .637$ ;  $\eta_p^2 = .006$ ), demonstrating that fixation percentages were comparable between HA and LA participants. A main effect of AOI was found ( $F(1,38) = 276.73$ ;  $p < .0001$ ;  $\eta_p^2 = .879$ ), revealing more fixations in the eye region ( $M = 59.12\%$ ,  $SD = 12.20\%$ ) than in the

mouth region ( $M = 15.22\%$ ,  $SD = 8.50\%$ ). More importantly, the AOI x emotion x group interaction was significant ( $F(3, 114) = 3.66$ ;  $p = .018$ ;  $\eta_p^2 = .088$ ). This interaction was further explored with separate two-way ANOVAs for each group (HA and LA) with AOI (eye region, mouth region) and emotion (fear, disgust, happiness, and neutral) as within-subjects factors, to investigate possible differences between the two groups.

The results of the ANOVA in the LA group showed a significant main effect of AOI ( $F(1,19) = 102.09$ ;  $p < .0001$ ;  $\eta_p^2 = .843$ ), revealing more fixations in the eye region ( $M = 54.91\%$ ,  $SD = 11.233\%$ ) than in the mouth region ( $M = 18.63\%$ ,  $SD = 7.71\%$ ). Notably, there was a significant interaction between AOI and emotion ( $F(3,57) = 27.059$ ;  $p < .0001$ ;  $\eta_p^2 = .587$ ). Post-hoc tests showed a greater percentage of fixations in the eye region for neutral faces ( $M = 62.98\%$ ,  $SD = 10.76\%$ ) than for any of the other faces (all  $p$ -values  $< .001$ ; fear:  $M = 55.7\%$ ,  $SD = 11.08\%$ ; disgust:  $M = 49.43\%$ ,  $SD = 12.167\%$ ; happiness =  $51.55\%$ ,  $SD = 15.32\%$ ). Moreover, the percentage of fixations was greater for fear than for disgust or happiness (all  $p$ -values  $< .05$ ). The percentage of fixations was smaller for disgust than for fear and neutral (all  $p$ -values  $< .01$ ), but it was not significantly different from the percentage of fixations for happiness ( $p = .3$ ).

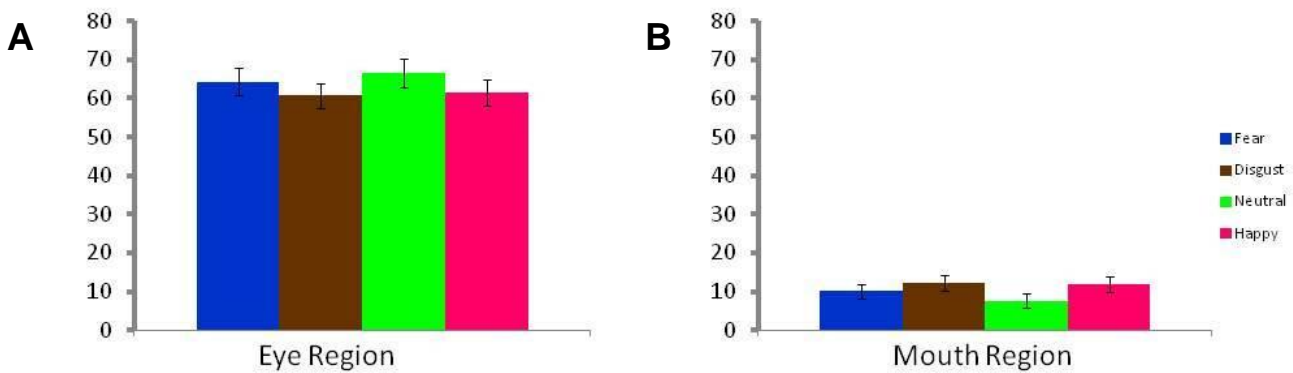
Post-hoc tests showed a greater percentage of fixations in the mouth region for disgust ( $M = 23.86\%$ ,  $SD = 10.41\%$ ) compared to the other emotions (fear:  $M = 18.59\%$ ,  $SD = 8.21\%$ ; neutral:  $M = 12.30\%$ ,  $SD = 6.48\%$ , happy:  $M = 19.75\%$ ,  $SD = 8.83\%$ ; all  $p$ -values  $\leq .050$ ). The percentage of fixations for neutral faces was significantly smaller than for all other emotions (all  $p$  values  $\leq .003$ ). The fixation percentage for fear was not significantly different from the fixation percentage for happiness ( $p = .57$ ) (Figure. 2).

On the other hand, the ANOVA in the HA group showed a significant main effect of AOI ( $F(1,19) = 177.38$ ;  $p < .0001$ ;  $\eta_p^2 = .903$ ), revealing more fixations in the eye



**Fig 2.** Mean fixation percentage measured in eye region (A) and in mouth region (B) when fear, disgust, neutral, and happy were presented (A) for low alexithymia group.

region ( $M = 63.33\%$ ,  $SD = 11.94\%$ ) than in mouth region ( $M=11.81$ ,  $SD=8.04$ ). A significant interaction between AOI and emotion was also revealed ( $F(3,57) = 4.23$ ;  $p = .009$ ;  $\eta_p^2 = .18$ ). Crucially, post-hoc tests revealed that this interaction is mainly driven by a greater percentage of fixations in the eye region than in mouth region. There were no significant differences between emotions in the eye region (all  $p$ -values  $\geq .06$ ) or in the mouth region (all  $p$ -values  $\geq .17$ ), showing that eye movement patterns did not differ between emotional expressions (Figure. 3).





**Fig 3.**

Mean fixation percentage measured in eye region (A) and in mouth region (B) when fear, disgust, neutral, and happy were presented (A) for high alexithymia group.

**First saccade**

Furthermore, we analyzed the first saccade (the location of the second fixation), to explore whether differences between the HA and LA groups are present from the beginning of the trial.

The ANOVA showed a significant main effect of AOI ( $F(1,38) = 32.20$ ;  $p < .0001$ ;  $\eta_p^2 = .458$ ), revealing a greater percentage of first saccades to the eye region ( $M = 60\%$ ,  $SD = 29\%$ ) than to the mouth region ( $M = 19\%$ ,  $SD = 22\%$ ). More importantly, the AOI x emotion x group interaction was significant ( $F(3, 114) = 3.036$ ;  $p = .04$ ;  $\eta_p^2 = .073$ ). This interaction was further explored with separate two-way ANOVAs for each group (HA and LA) with AOI (eye region, mouth region) and emotion (fear, disgust, happiness, and neutral) as within-subjects factors, to investigate possible differences between the two groups.

The ANOVA in the LA group showed a significant main effect of AOI ( $F(1,19) = 4.63$ ;  $p = .044$ ;  $\eta_p^2 = .19$ ); revealing more first saccades to the eye region ( $M = 51\%$ ,  $SD = 27\%$ ) than to the mouth region ( $M = 27\%$ ,  $SD = 25\%$ ). Notably, a significant interaction between AOI and emotion was revealed ( $F(3,57) = 9.22$ ;  $p < .0001$ ;  $\eta_p^2 = .32$ ). Post-hoc tests showed a greater percentage of first saccades to the eye region for neutral faces ( $M=65$ ,  $SD=27$ ) compared to all the emotional faces (fear:  $M = 49\%$ ,  $SD = 31\%$ ; disgust:  $M = 48\%$ ,  $SD = 32\%$ ; happiness =  $43\%$ ,  $SD = 38\%$ ; all  $p$ -values  $\leq .037$ ). No other differences were significant (all  $p$ -values  $> .5$ ).

Post-hoc tests comparing differences between emotional expressions in the mouth region showed a smaller percentage of first saccades to the mouth for neutral faces ( $M = 11\%$ ,  $SD = 15\%$ ) compared to all other emotions (fear:  $M = 26\%$ ,  $SD = 24\%$ ; disgust:  $M = 36\%$ ,  $SD = 30\%$ ; happiness:  $M = 36\%$ ,  $SD = 29\%$ ; all  $p$ -values  $\leq .04$ ). No other differences were significant (all  $p$ -values  $> .15$ ).

In contrast, the ANOVA in the HA group only showed a main effect of AOI ( $F(1,19) = 39.90$ ;  $p < .0001$ ;  $\eta_p^2 = .677$ ), revealing a greater percentage of first saccades to the eye region ( $M = 68\%$ ,  $SD = 28\%$ ) than to the mouth region ( $M = 11\%$ ,  $SD = 13\%$ ). No other main effects or interactions were significant (all  $p$ -values  $> .06$ ).

## **Behavioral results**

Reaction times and accuracy were analyzed in ANOVAs with group (HA, LA) as a between-subjects factor, and emotion (fear, disgust, happiness, neutral) as a within-subjects factor. The ANOVA on reaction times showed no significant differences between the two groups ( $F(1,38) = .40$ ;  $p = .52$ ;  $\eta_p^2 = .01$ ). There was only a significant main effect of emotion ( $F(3,114) = 44.49$ ;  $p < .0001$ ;  $\eta_p^2 = .54$ ). Post-hoc tests showed that participants were faster at responding to neutral facial expressions ( $M = 1253$  ms,  $SD = 419.97$ ) than to disgust ( $M = 1594$  ms,  $SD = 459.16$ ;  $p < .001$ ) and fear ( $M = 1826$  ms,  $SD = 538.54$ ;  $p \leq .0001$ ). There was no significant difference between reaction times to neutral and happy faces ( $M = 1305$  ms,  $SD = 412.76$ ). Moreover, reaction times to fear were longer than reaction times to all other emotions (all  $p$ -values  $\leq .0001$ ). The differences between other emotions were not significant ( $p = .3$ ). No interactions with the factor group were significant (all  $p$ -values  $> .25$ ). Similarly, the ANOVA on accuracy scores showed no significant differences between groups ( $F(1,38)$

= 2.37;  $p = .13$ ;  $\eta_p^2 = .06$ ). There was only a main effect of emotion ( $F(3,114) = 37.18$ ;  $p < .0001$ ;  $\eta_p^2 = .5$ ). Post-hoc analysis revealed participants were more accurate at identifying neutral faces ( $M = 99\%$ ,  $SD = 4\%$ ) compared to all other emotions (fear:  $M = 77\%$ ,  $SD = 13\%$ ; disgust:  $M = 93\%$ ,  $SD = 10\%$ ; happiness:  $M = 91\%$ ,  $SD = 10\%$ ; all  $p$ -values  $\leq .01$ ). Also, they were less accurate at identifying fear, compared to all other emotions (all  $p$ -values  $\leq .0001$ ). No other differences were found between emotions (all  $p$ -values  $> .4$ ). The group  $\times$  emotion interaction was not significant ( $p = .24$ ).

### 4.3.3. Discussion

Alexithymia is associated with difficulties in emotion recognition, mainly for negative emotions. It is not clear whether this difficulty is grounded in early stages of visual processing, when visual information should be gathered from relevant places on the face. This difficulty may result from differences in stimulus processing in alexithymia. Considering the critical role that the human face plays as a perceptual category, we studied how different facial emotions modulate eye movements, and whether different patterns of eye movements are needed to recognize emotions. Using eye tracking technology, we compared patterns of eye movements during an emotional face recognition task consisting of fearful, disgusted, happy, and neutral facial expressions.

Our results revealed that, in both the high and low alexithymia groups, the eye region was more explored than other regions of face, both for emotional faces (fear, disgust, happiness) and for neutral faces. This is in accordance with previous studies (Adolphs et al., 2005; Domes et al., 2007; Haxby et al., 2002; Heinrichs et al., 2003; Kirsch et al., 2005). Secondly, our results showed that neutral expressions received a

higher percentage of eye fixations than other emotional expressions. This might be driven by greater ambiguity in neutral faces that leads to more exploration (Todorov et al., 2008). In low alexithymics, after neutral faces, the fearful expression received the highest percentage of fixations in the eye region. This is in line with previous findings, and supports the idea that the eyes are an important source of salient information in fearful faces (Adolphs et al., 2005). Moreover, in line with previous studies disgusted expressions received a higher percentage of mouth fixations than all other emotions (Calder et al., 2000). Therefore, low alexithymics performed similarly to normal people in exploring emotional faces. Instead, in high alexithymia, no differences was seen in eye movement patterns between facial emotions, neither in the eye region, nor in the mouth region.

One possible explanation for this lack of any modulation by emotional expression in the high alexithymic group might be amygdala hypoactivity (Goerlich-Dobre et al., 2015; Suslow et al., 2016; van der Velde et al., 2013). Amygdala hypoactivity in high alexithymia might diminish the function of this key structure in directing the visual system to “seek out, fixate, pay attention to, and make use of” visual information (Adolphs et al., 2005). The reason that eye movement patterns in the eye region do not differ between neutral and other facial expressions in high alexithymia might be a lack of activity in the amygdala and, hence, a lack of differentiation in seeking visual information from faces showing different emotions. Moreover the absence of any difference between fearful facial expressions and other emotions (disgust, happy) in the percentage of fixations in the eye region might, again, be due to amygdala hypoactivity in response to salient, fear-related information in alexithymia. Additionally, the lack of any difference between disgust and other emotions in eye

movement patterns within the mouth region might be driven by altered insula activity in alexithymia (Kano et al., 2010; Wingbermühle et al., 2012; Scarpazza et al., 2015).

The difference in eye movement patterns between the high and low alexithymia groups was evident in both the first saccades and the fixation percentages. This suggests that a difference in coding visual emotional information exists even from the early stages of visual search. Additionally, it suggests that there was early holistic perception of emotional faces when participants were asked to recognize the facial emotion. This was revealed by first saccade results that showed a significant difference between neutral and emotional facial expressions in low alexithymia group.

Overall, this study showed that both the eye and mouth regions are crucial for obtaining visual information and using it to recognize emotional expressions. Additionally, exploration of and attention to these two regions differed by facial expression in the low alexithymia group, while no differences between emotions were seen neither in either region in high alexithymics. However, it is important to note that, despite the differences we found at an early stage of visual search for perceptual processing, no difference was found at the behavioral level in facial emotion recognition between high and low alexithymics. This suggests that subtle differences in emotion processing could possibly lead to considerable difficulties for alexithymics in more complex and ambiguous social interactions.

#### **4.4. Experiment 4b: Gender discrimination**

Experiment 4a revealed using an emotion categorization task, a difference between high and low alexithymics in eye movement patterns was revealed. Experiment 4b studied whether high and low alexithymics differ in terms of eye movement patterns in a control task that did not require facial emotion discrimination. In this task, the emotional aspects of facial expressions were task-irrelevant, and participants were asked to judge the gender of the faces using an otherwise identical procedure and the same stimuli. In this task recognition of emotion was less relevant because participants had to categorize the gender. Therefore, no differences in eye movement patterns between the two groups were expected.

##### **4.4.1. Methods**

Using the same stimuli and procedure as Experiment 4a, participants were asked to discriminate the gender of the faces rather than their emotional expressions. Response buttons on a keyboard (1 and 9) corresponded to male and female, and were counterbalanced between participants. The order of emotional face discrimination and gender discrimination tasks was also counterbalanced between participants. Half of the participants performed the emotional face discrimination task first, and the other half did the gender discrimination task first. Prior to the experiment, participants practiced the task for a short time.

##### **4.4.2. Results**

**Fixation percentage:** the ANOVA showed a significant main effect of group ( $F(1,38)=7.79$ ;  $p\leq.008$ ;  $\eta^2=.17$ ) revealing more fixation percentage in LA group

(M=38.21, SD=3.22) rather than in HA group (M=33.90, SD=6.1). Moreover a main effect of AOI was revealed ( $F(1,38)=252$ ;  $p<.0001$ ;  $\eta^2=.87$ ) showing that there were more fixations in eye region (M=58.17, SD=10.79) than in mouth region (M=13.94, SD=7.83). There was a significant interaction between AOI and emotion ( $F(3,114)=11.4$ ,  $p<.0001$ ;  $\eta^2=.23$ ). Post-hoc analysis showed in eye region the smallest fixation percentage was during presentation of disgust (M=54.54, SD=13.33) compared with other emotions: fear(M= 58.58, SD=12.41), happy(M= 58.21, SD=14), neutral(M= 61.33, SD=14.11) all  $ps\leq.017$ . In mouth region the smallest fixation percentage was during presentation of neutral (M=10.95, SD=7.32) compared with disgust (M=16, SD=9.37) and happy (M=15.10, SD=8.81). There was no significant difference between fear (M=13.72, SD=8.8) and neutral ( $p=.06$ ). The difference among all other emotions were not significant (all  $P\geq.06$ ). Crucially no interactions with the factor group were significant (all  $ps > .17$ ).

#### **4.4.3. Discussion:**

Experiment 4b showed there is no difference between high and low alexithymics regarding to eye movement patterns when they had to perform gender discrimination task. This might suggest that the difference in the eye movement pattern that was found in high alexithymia is not due to basic visuoperceptual inabilities to process information conveyed through the face, rather it is an alteration in emotion processing. This might be because of amygdale's hypoactivity to direct the visual attention to relevant regions on face and make use of emotional information exhibited through these regions.

#### **General discussion for experiments 4a, and 4b:**

Overall, experiments 4a and 4b investigated the visual scanning in high and low alexithymia in different facial recognition tasks (emotion recognition, and gender

recognition). Results in facial emotion recognition task suggested a different pattern of exploring visual information through important regions on face (eyes and mouth) in high alexithymics compared to low alexithymics. Importantly this difference was found even in the first saccade stage. On the other hand our results from gender discrimination task showed high and low alexithymic are not different in general visuperceptual abilities since no difference was found between them when they had to judge the gender instead of emotion. This might be due to the fact that in the used gender task, emotional information was less relevant, instead when the emotional content was relevant to task, the difference between two groups was found.

In conclusion, both high and low alexithymics process the information from faces, via allocating the gaze and attention towards eye region. However in low alexithymic group different emotions modulate the proportion of fixations in eye and mouth region, while this modulation was absent in high alexithymics. In low alexithymia group, visual scanning pattern is modulated by different facial emotions and this modulation is influenced by personality traits, more specifically alexithymia trait.

Although there was no significant difference in processing emotional faces at behavioral level between high and low alexithymics, a difference in where the eye fixations land and what visual information is processed during observing facial expressions was seen. This difference might be due to the alterations in amygdala's role to seek out and allocate the visual attention to relevant regions on face.



#### **4.5. Experiment 5: Visceral reactivity during emotional face perception**

Emotion, like many other cognitive functions of the brain, has an intuitive definition. It has been defined as any relatively brief conscious experience characterized by intense mental activity and a high degree of pleasure or displeasure (Cabanac, 2002; Schancter, 2011). One aspect of emotion that is generally agreed upon is its association with physiological responses, such as somatic, reflexive, and visceral reactions. These physiological responses play a crucial role in emotion recognition. Recent theories have suggested that an important requisite for recognizing others' emotions is re-experiencing, or simulating, the same emotional states in one's own sensory and motor systems (Hess & Fischer, 2013; McIntosh et al., 2006). Embodied simulation theories state that somato-motor reactions to others' emotional expressions intrude upon the observer's affective state to produce a matching emotion, thus providing a direct form of emotion understanding (Niedenthal, 2007; Niedenthal et al., 2010; Oberman et al., 2007; Gallese & Sinigaglia, 2011). Some physiological reactions related to emotion include changes in heart rate, sweating palms, muscle tension, blood pressure changes, and other arousal responses driven by emotional stimuli (Bradley & Lang, 2000). For instance, previous studies have shown that the corrugator muscles of the face (which draw the brows together in expressions of displeasure) are more active when viewing unpleasant pictures than when viewing pleasant pictures. In contrast, the activity of the zygomatic majoris (a muscle involved in smiling) increases while observing pleasant pictures (Bradley et al, 2001). Another physiological response to emotional stimuli is the startle reflex (a blink response that occurs when there is a sudden noise). This reflex is larger when viewing negatively valenced stimuli compared to positively valenced stimuli (Lang, 1995; see Bradley et al., 1999 for an overview). Interestingly, skin conductance is a physiological response

that increases in response to highly arousing stimuli, both pleasant and unpleasant. Another important early autonomic reaction is changes in heart rate. Heart rate is a psychophysiological measure related to autonomic nervous system activity that is commonly used in emotion research. Heart rate is generally thought to distinguish between pleasant and unpleasant emotions. In healthy populations, heart rate decelerates when observing unpleasant stimuli, compared to pleasantly rated stimuli (Bradley et al., 1996; Bradley et al., 2000).

Experiment 4 revealed the cognitive and perceptual aspects of the emotion recognition deficit in alexithymia. In Experiment 5, we assessed whether this emotion recognition impairment has a physiological (visceral) aspect, as well. We measured heart rate changes during passive viewing of emotional facial expressions in HA and LA participants. Previous studies have shown contradictory findings. For instance, some studies did not find any difference between HA and LA participants in terms of cardiovascular reactivity (Friedlander et al., 1997; Roedema & Simons., 1999; Waldstein et al., 2002), while, in other studies, alexithymics showed decrements in heart rate in response to emotion inducing tasks (Linden et al., 1996; Newton & Contrada, 1994; Wehmer et al., 1995). These findings are suggestive of the hypoarousal model of alexithymia, which states that attenuated sympathetic nervous system responses account for the dampened emotional reactivity (Neumann et al., 2004; Papciak et al., 1985). In contrast, the hyperarousal model states that alexithymia is related to higher tonic levels of sympathetic activity and/or exaggerated sympathetic reactivity (and possibly parasympathetic withdrawal) in response to emotional stressors (Neumann et al., 2004).

It is necessary to note that none of the aforementioned studies used emotional faces to assess heart rate changes. In Experiment 5, we used basic facial expressions,

including fear, happiness, disgust, and neutral expressions, to study how the autonomic system reacts to others' emotional expressions in HA and LA participants. Based on the hypoarousal model in alexithymia that suggests attenuated visceral responses due to perceived emotions (Neumann et al., 2004), we expected to find reduced autonomic reactivity in response to emotional facial expressions in the HA group, compared to the LA group.

#### **4.5.1. Methods**

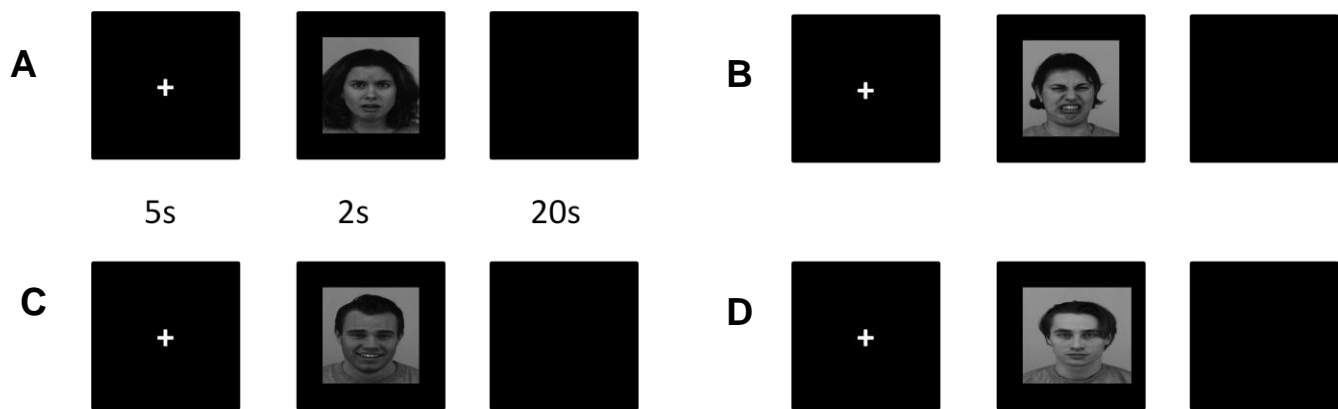
##### **Participants**

The same individuals who took part in Experiment 4 also participated in Experiment 5. These participants performed Experiments 4 and 5 on the same day. The order of experiments was counterbalanced between participants (half of them performed Experiment 4 first, and the other half performed Experiment 5 first). In total, 40 participants (20 HA, 20 LA) took part in this experiment. (For demographic information, please see Experiment 4, Methods).

##### **Stimuli and task**

Stimuli were selected from the Karolinska Directed Emotional Faces (KDEF) database (Lundqvist et al., 1998), and consisted of 40 black-and-white photographs (4 emotions, 2 genders, 5 actors) showing facial expressions. Each image depicted a basic emotion (i.e., fear, happiness, disgust, or neutral) from the validated KDEF database (Lundqvist et al., 1998; Goeleven et al., 2008). Stimuli subtended a horizontal visual angle of 16° and a vertical visual angle of 23°. Participants were seated comfortably in a silent room in front of a screen, at a viewing distance of 57cm from the screen. They were asked to remain as still as possible during the experiment, and to pay attention to the task. The task was passive viewing of facial expressions. It consisted of

two blocks, each containing 40 trials. Each trial started with 5 s of a fixation cross, followed by 2 s of stimulus presentation, and then 20 s of a black screen (Fig. 1). This inter-trial interval let the heart rate return to baseline prior to the next trial, preventing any carryover effects from the previous trial. The two blocks were identical, except for the order of trials, which was randomized in each block. The experiment was programmed and run in PRESENTATION software (Neurobehavioral Systems, Inc., San Francisco, CA).



**Fig. 1.**

Graphical representation of the trial structure in the behavioral task. Task started with 5s of fixation cross, continued by 2s of stimulus presentation and then 20s of black page. Fearful (A), disgust (B), happy (C), and neutral facial expressions (D) were presented in separate trials.

### **Electrocardiographic (EKG) recording and processing**

EKG measurements were taken using non-polarizable Ag-AgCl electrodes attached to the left and right wrists, and referenced to the left mid-clavicle. Signals were recorded by a computer-based data acquisition system (Biopac MP150) and its corresponding software, AcqKnowledge (BIOPAC Systems Inc., Santa Barbara, CA). The signal was amplified x1000 and digitized at 100 Hz. Data were analyzed using custom routines in MATLAB 7.0.4 (The Mathworks, Natic, MA). The series of consecutive heart beats starting 6 beats before stimulus presentation and ending 6 beats after stimulus presentation was considered for analysis. QRS complexes were discriminated from the EKG recordings by triggering the R-wave peaks. The time interval between consecutive QRS complexes was then determined (R-R interval), and its inverse value was calculated. This value is an index of instantaneous heart rate (HR), and was multiplied by 60 in order to have an HR signal expressed in beats/minute. At this point, the data were simplified by calculating, for each trial, the mean values of HR before the onset of the visual stimulus and after the onset of the visual stimulus. These mean HR values were analyzed with a mixed factors analysis of variance (ANOVA) with time (before and after stimulus presentation), emotion (fear, disgust, happiness, and neutral) as within-subjects variables, and group (HA, LA) as a between-subjects variable. To compensate for violations of sphericity, Greenhouse–Geisser corrections were applied whenever appropriate (Greenhouse & Geisser, 1959). Corrected p-values (but uncorrected degrees of freedom) are reported. Post-hoc comparisons were performed using the Newman–Keuls test.

### **Control task**

Since participants must be as still as possible while heart rate is being measured, as any verbal or non-verbal responses could affect the heart rate, we had to use a passive viewing task. To ensure that participants paid attention to the stimuli, we

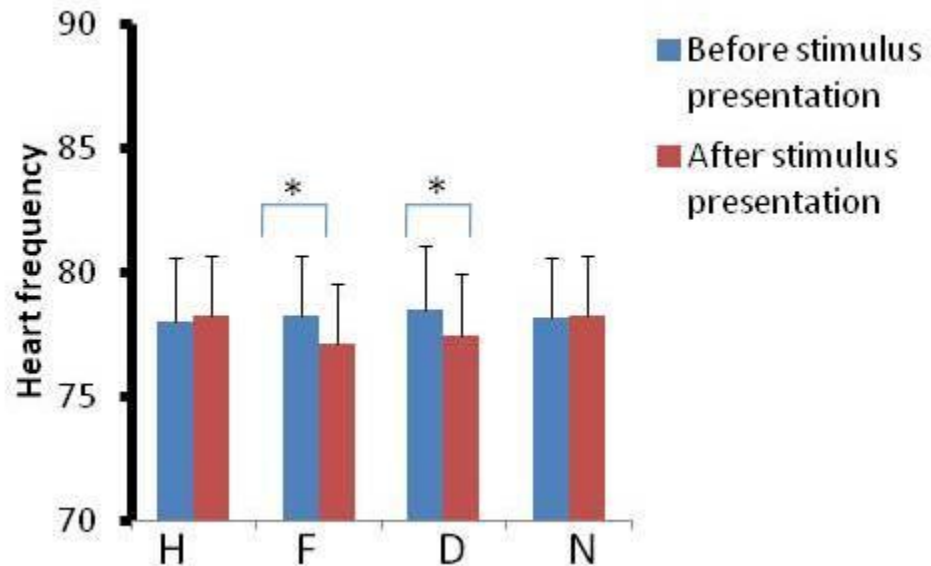
carried out a control task in which 24 stimuli from the KDEF database were shown to participants. Half of the stimuli had been used in the passive viewing task, and the other half had not been used. Stimuli subtended a horizontal visual angle of 16° and a vertical visual angle of 23°. Participants were asked to judge whether they had seen the same stimulus (same actor and same emotion) in the passive viewing task with a yes-or-no verbal response.

## **4.5.2. Results**

### **Heart rate changes**

The ANOVA showed no main effect of group ( $F(1,38) = 1.053$ ;  $p = .31$ ;  $\eta_p^2 = .027$ ). A main effect of time was revealed ( $F(1,38) = 4.56$ ;  $p = .04$ ;  $\eta_p^2 = .107$ ), showing that heart rate was higher before stimulus presentation ( $M = 76.45$ ,  $SD = 1.49$ ) than after stimulus presentation ( $M = 76.21$ ,  $SD = 10.35$ ). More importantly, the group x time x emotion interaction was significant ( $F(3,114) = 4.52$ ,  $p = .011$ ;  $\eta_p^2 = .106$ ). This interaction was further explored with separate two-way ANOVAs for the HA and LA groups, with time (before, after) and emotion (happiness, fear, disgust, and neutral) as within-subjects factors. The ANOVA in the LA group showed a main effect of time ( $F(1,19) = 9.46$ ,  $p = .006$ ;  $\eta_p^2 = .33$ ), revealing that heart rate was higher before stimulus presentation ( $M = 78.25$ ,  $SD = 10.99$ ) than after stimulus presentation ( $M = 77.79$ ,  $SD = 10.91$ ). Interestingly, the time x emotion interaction was significant ( $F(3,57) = 4.98$ ;  $p = .014$ ;  $\eta_p^2 = .207$ ). Post-hoc comparisons showed that heart rate did not differ between emotion conditions before stimulus presentation (happiness:  $M = 78.43$ ,  $SD = 11.41$ ; fear:  $M = 78.25$ ,  $SD = 10.92$ ; disgust:  $M = 78.54$ ,  $SD = 10.79$ ; neutral:  $M = 78.18$ ,  $SD =$

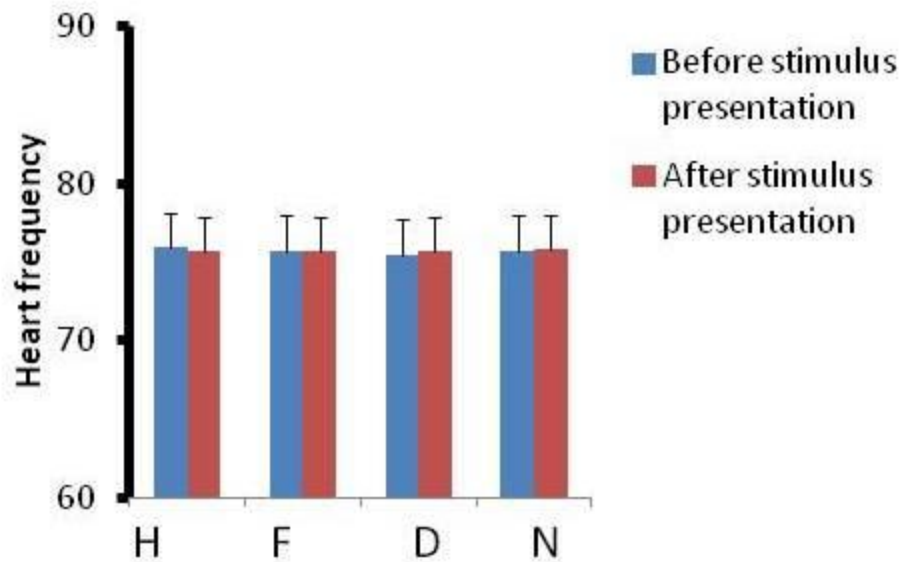
11.01; all  $p$ -values  $> .64$ ), but heart rate after stimulus presentation was lower in response to fear ( $M = 77.12$ ,  $SD = 11.22$ ) than to happiness ( $M = 78.25$ ,  $SD = 11.25$ )



**Fig 2.**

Heart frequency results in low alexithymia group. Error bars show standard error of the mean.

or neutral expressions ( $M = 78.31$ ,  $SD = 10.70$ ; all  $ps \leq .013$ ), but was not significantly different from disgust ( $M = 77.47$ ,  $SD = 10.84$ ;  $p = .29$ ) (Fig. 2). Moreover, fear and disgust were the only emotions for which heart rate was significantly modulated by stimulus presentation (fear:  $p = .013$ ; disgust:  $p = .03$ ). That is, LA participants showed bradycardia (a decrease in heart rate) after observing fearful and disgusted facial expressions. In contrast, the ANOVA in the HA group revealed no significant main effects or interaction (all  $p$ -values  $\geq .74$ ) (Fig. 3).



**Fig 3.**

Heart frequency results in high alexithymia group. Error bars show standard error of the mean.

### **Control task**

A binomial test of significance was run on each participant's percentage of correct responses to test whether they were above chance level. The binomial tests showed that all the participants performed above chance ( $p < .04$ ). Additionally, a t-test was used to compare the number of correct responses between the high and low alexithymic groups. No significant difference was found between the two groups. ( $t(38) = .90, p = .37; HA: M = 22.3; LA: M = 22.95$ ).



### **4.5.3. Discussion**

In this study, we investigated visceral responses (changes in heart rate) to emotional facial expressions. Participants with low scores on an alexithymia scale showed bradycardia (heart rate deceleration) in response to fearful and disgusted facial expressions, but this modulation was absent in the HA group. The finding of fear-related bradycardia in the low alexithymia group is in line with previous studies in normal populations that also found fear-related bradycardia (Hermans et al., 2013; Lang & Davis, 2006). This deceleration in heart rate is considered a physiological component of a complex freezing reaction (Azavedo et al., 2005; Hagenaaars et al., 2012; Roelofs et al., 2010), i.e., a pause in locomotion characterized by a predominantly parasympathetic autonomic nervous system response (Hermans et al., 2013). Interestingly, the current study adds to previous findings by showing a deceleration in heart rate in response to disgusted faces, as well as fearful faces.

Crucially, our findings showed that heart rate was comparable at the pre-stimulus baseline in both HA and LA groups. The lack of an autonomic response to negative emotions in alexithymia is in line with previous studies showing that alexithymia is associated with dampened physiological reactivity to such stimuli (Bermond et al., 2010; Neumann et al., 2004; Wehmer et al. 1995; Linden et al. 1996; Roedema & Simons, 1999). Several studies found that higher alexithymia scores predicted lower skin conductance and diminished heart rate deceleration responses to emotional stimuli (Linden et al., 1996; Wehmer et al., 1995; Roedema & Simons, 1999). All these findings, as well as the results from the present study, are consistent with the hypoarousal (hyporeactivity) model of alexithymia that proposes dampened

physiological reactivity--particularly visceral reactions--to emotional contents (Linden et al., 1996; Nemiah et al., 1997; Newton & Contrada, 1994; Wehmer et al., 1995; Neumann et al. 2004). The finding of hypoarousal, or hyporeactivity, in response to negative emotions (fear and disgust) indicates attenuated sympathetic activation for these emotional expressions. Together with the findings of Experiment 4, which showed differing patterns of visual exploration for emotional expressions in high and low alexithymics, the findings of Experiment 5 support the model of amygdala hypoactivation in response to emotional facial expressions, especially negative valencely expressions such as fear (Kugel et al., 2008; Reker et al., 2010; van der Velde et al., 2013). The amygdala is a key subcortical structure for signaling negative emotions and salient information (Adolphs, 2013; LeDoux, 2014), and it is highly activated in response to human faces (LeDoux 2012; Morris et al., 1996; Breiter et al., 1996; Whalen et al., 1998). Importantly, the central amygdaloid nucleus, located in the dorsal amygdala, is probably involved in the autonomic correlates of emotional arousal (Gentile et al., 1986; Hitchcock & Davis, 1986; Kapp et al., 1979). Classical conditioning studies have shown that lesions in areas that receive projections from the central amygdaloid nucleus lead to disrupted autonomic responses to fear (LeDoux et al., 1988). Moreover, patients with amygdala lesions demonstrate less fear conditioning and bradycardia (Amorapanth et al., 2000, LeBar et al., 1995). Our results also extend prior research findings by showing that the hypoarousal model of alexithymia is supported not only by responses to inner emotions (i.e., one's own emotional states) but also by responses to externally oriented emotions (e.g., emotions conveyed by others' faces). These findings provide evidence that alexithymia is characterized by difficulties in identifying and recognizing the emotions of others, in addition to one's own emotions.

Experiment 5 showed that people with high levels of alexithymia have attenuated visceral reactivity in response to negative facial expressions, and Experiment 4 showed that they have different patterns of eye movements for exploring facial expressions, compared to people with low levels of alexithymia. Overall, it is likely that the impaired emotion recognition associated with alexithymia results from autonomic and perceptual alterations.

### **General Discussion for experiment 3, 4, and 5:**

Experiments 3, 4, and 5 investigated the impact of alexithymia on perceptual, attentional, and simulation mechanisms of facial emotion recognition. The results showed no differences between high and low alexithymics at a perceptual level. Investigating the attentional mechanism, by measuring eye movement patterns, revealed that patterns of visual scanning were modulated by facial expressions in low alexithymia. For instance, in the emotion categorization task, low alexithymic participants fixated the eye region more when viewing fearful expressions than when viewing the other facial expressions (except neutral).. In contrast, this modulation was absent in the high alexithymia group. This might be due to amygdala hypoactivation, specifically in response to negative emotions, which might affect the direction of attention toward relevant visual information.

Similarly, participants with high and low levels of alexithymia showed differences in the visceral component of emotional responses. The low alexithymia group showed bradycardia in response to negative emotional expressions (fear and disgust). This modulation was absent in high alexithymics, perhaps because of altered functioning in the amygdala, which is involved in the autonomic correlates of emotional arousal (Kapp et al., 1979; Gentile et al., 1986).

Overall, these findings indicate that emotion processing is altered in people with high levels of alexithymia, even when they show no impairments in emotion recognition at a behavioral level.

## CHAPTER 5: Recognizing emotion through body postures

**The role of body language in understanding emotion:** nonverbal language plays an essential role in social interactions and communication. Movement of the body as a whole object or its individual parts makes a meaningful contribution to nonverbal language. As Darwin proposed, emotions are adaptive because they evoke an action which is beneficial for organism's survival. Human body postures along with human faces are the most important sources for obtaining information about others' feelings and emotions. In contrast to faces, body postures exhibit not only the type of emotion one is experiencing, but also it shows the undertaken actions in response to that specific experienced emotion.

Neuroimaging studies as well as single-cell recordings on visual recognition of bodies showed existence of a preference for either face or body images in the superior temporal sulcus (STS) (Perrett et al., 1922; Rizzolatti et al., 1996). More specifically, a region near the middle occipital gyrus, extrastriate body area (EBA), found to respond selectively to bodies but very little to faces (Peelen and Downing., 2005; Hadjikhani and de Gelder., 2003). Emotional body language has been reported to be percept through a rapid automatic, non-conscious route in subcortical areas which is called primary network (LeDox., 2000). This route involves the superior colliculus, pulvinar, striatum (putamen and caudate) and amygdala. It has been shown superior colliculus is an important structure involved in defensive reflexes for instance freezing and withdrawal (Schiller et al., 1971). Beside the primary system, there is also a second system which is involved in perception of emotional body language that connects awareness of bodily states to decision making (Damasio et al., 2000). This system consists of frontoparietal motor system, and connectivity between amygdala and prefrontal and ventromedial

prefrontal cortex. This system plays an important role in perception of emotional body language in detail and analyzes the subsequent behavioral responses.

In this chapter I tried to investigate how body postures conveying emotional and non emotional movement related information influence the visual processing. To this end firstly in experiment 6, the modulations of early structural and late attentional stages of visual processing in normal population were studied. Then regarding the findings in emotional face perception experiments (3, 4, and 5) that revealed impairment in alexithymia, I was interested to study the perception of bodily emotions in alexithymia and see whether there is also a deficit in perception of emotional information conveyed through body postures. Therefore in experiment 7 the early visual coding of body postures and the late attentional stage of visual processing of human bodies were studied.

### **5.1. Experiment 6:** Modulation of visual processing by emotional and movement-related body postures

Human body postures comprise a biologically salient category of stimuli, whose efficient perception is crucial for social interaction. Although in natural environments human bodies and faces are usually integrated into a unified percept, the neural networks underlying the processing of these two categories of stimuli, though closely related, seem to be distinct. In particular, neuroimaging evidence has demonstrated selective responses to human bodies in two focal brain regions: the extrastriate body area (EBA), located in the lateral occipitotemporal cortex (Downing et al., 2001), and the fusiform body area (FBA), in the posterior fusiform gyrus (Peelen and Downing, 2005; Taylor et al., 2007). Interestingly, both EBA and FBA responses generalize to schematic depictions of bodies, suggesting that body representation in these two areas is independent of low-level image features (Downing et al., 2001; Peelen et al., 2006). As is the case with faces (e.g. Adolphs, 2002), the perceptual processing of bodies seems to represent a specialized mechanism, in which perception is configural (i.e. based on relations among the features of the stimulus), rather than based on the analysis of single body features. This is suggested, for example, by the inversion effect, a phenomenon in which bodies presented upside-down are more difficult to recognize than inverted objects (Reed et al., 2003). At the electrophysiological level, event-related potentials (ERPs) in response to bodies show a prominent negative deflection at occipitotemporal electrodes peaking in a range between 150 and 230ms after stimulus presentation (Stekelenburg and de Gelder, 2004; Meeren et al., 2005; Van Heijnsbergen et al., 2007; Minnebusch et al., 2010).

More specifically, Thierry et al. (2006) found a negative component peaking at 190ms post-stimulus onset (N190), reflecting the structural visual encoding of bodies, which was distinct in terms of latency, amplitude and spatial distribution compared with the typical negative component elicited by the visual encoding of faces (i.e. the N170; Rossion and Jacques, 2008). The neural generators responsible for the negative deflection in response to bodies are thought to be located in a restricted area of the lateral occipitotemporal cortex, corresponding to EBA, as suggested by source localization analysis (Thierry et al., 2006), magnetoencephalographic recordings (Meeren et al., 2013) and electroencephalogram (EEG)-fMRI correlation studies (Taylor et al., 2010). Studies on the perceptual processing of faces have shown that the component reflecting visual encoding (N170) is modulated by the emotional expressions of faces processed both explicitly (Batty and Taylor, 2003; Stekelenburg and de Gelder, 2004) and implicitly (Pegna et al., 2008, 2011; Cecere et al., 2014), suggesting that relevant emotional signals are able to influence the early stages of structural face encoding. In addition, non-emotional face movements, such as gaze and mouth movements, seem to be encoded at an early stage of visual processing and to modulate the N170 amplitude (Puce et al., 2000; Puce and Perrett, 2003; Rossi et al., 2014).

At a later stage of visual processing (typically around 300ms after stimulus onset), salient emotional faces are known to modulate the amplitude of the early posterior negativity (EPN), which reflects stimulus-driven attentional capture, in which relevant stimuli are selected for further processing (Sato et al., 2001; Schupp et al., 2004a; Fruhholz et al., 2011; Calvo and Beltran, 2014).

Although faces represent a primary source of information about others' states (Adolphs, 2002), human bodies can also be a powerful tool for inferring the internal



states of others (de Gelder et al., 2010). Indeed, body postures convey information about others' actions and emotions, both of which are useful for interpreting goals, intentions and mental states. Neuroimaging studies have shown that motion and emotion-related information conveyed by bodies activates a broad network of brain regions (Allison et al., 2000; de Gelder, 2006; Peelen and Downing, 2007). On the one hand, the observation of human motion increases activation in occipitotemporal areas close to and partly overlapping with EBA (Kourtzi and Kanwisher, 2000; Senior et al., 2000; Peelen and Downing, 2005), the superior temporal sulcus (STS), the parietal cortex (Bonda et al., 1996) and the premotor and motor cortices (Gre`zes et al., 2003; Borgomaneri et al., 2014a), which might take part in perceiving and reacting to body postures (Rizzolatti and Craighero, 2004; Urgesi et al., 2014).

On the other hand, emotional body postures, compared with neutral body postures, enhance activation not only at the approximate location of EBA, the fusiform gyrus and STS but also in the amygdala (de Gelder et al., 2004; Van de Riet et al., 2009) and other cortical (e.g. orbitofrontal cortex, insula) and subcortical structures (e.g. superior colliculus, pulvinar) known to be involved in emotional processing (Hadjikhani and de Gelder, 2003; Peelen et al., 2007; Gre `zes et al., 2007; Pichon et al., 2008). Although the pattern of neural activation for bodies conveying motion and emotion-related information suggests a similarity between perceptual mechanisms for faces and bodies, it is still unclear whether, like the information conveyed by faces, the information conveyed by body postures is already encoded at the early stage of structural representation and is therefore able to guide visual selective attention to favor the recognition of potentially relevant stimuli. Thus, this study was designed to investigate, using the high temporal resolution of ERPs, whether the structural encoding of bodies, reflected in the N190 component and visual selective attention, measured by the

subsequent EPN component, are influenced by motion and emotion-related information represented in body postures.

To this end, an EEG was recorded from healthy participants performing a visual task in which they were shown pictures of bodies. These bodies had static postures (without implied motion or emotional content), implied motion postures without emotional content or implied motion postures expressing emotion (fear or happiness). In addition, stimuli were peripherally presented to the left or the right of a central fixation point to investigate whether the two hemispheres differentially contribute to the processing of body postures. In keeping with previous evidence that the right hemisphere plays a prominent role in responding to bodies (Chan et al., 2004; Taylor et al., 2010) and processing emotional information (Gainotti et al., 1993; Ladavas et al., 1993; Adolphs et al., 2000; Borod, 2000), a more detailed perceptual analysis of the different body postures was expected in the right hemisphere, compared with the left. More specifically, a low-level discrimination of motion-related information, reflected by an enhancement of the N190 component in response to postures with implied motion (either neutral or emotional) compared with static postures, was expected in both hemispheres.

In contrast, discrimination of emotional content, reflected by an enhanced N190 in response to fearful compared with happy bodies, was only expected in the right hemisphere. Finally, at a later stage of visual processing, the salience of fearful body postures was expected to increase visual selective attention, resulting in an enhanced EPN component. Unlike the emotion-related modulation of the N190, we expected the EPN enhancement for salient fearful postures to occur in both hemispheres, since attention-related emotional modulations are known to occur in a widespread bilateral network of brain regions, including extrastriate occipital cortex, superior and inferior

parietal areas and medial prefrontal regions (for a review, Pourtois and Vuilleumier, 2006).

### **5.1.1. Methods**

**Participants:** Twenty-two right-handed healthy volunteers (two males; mean age: 21.6 years; range: 20–26 years) took part in the experiment. They all had normal or corrected-to-normal vision. Since alexithymia is a relatively stable personality trait (Nemiah et al., 1976; Taylor et al., 1991),

which is known to affect emotion recognition and processing (Jessimer and Markham, 1997; Parker et al., 2005), all volunteers underwent a screening for alexithymia, using the 20-item Toronto Alexithymia Scale (TAS-20; Taylor et al., 2003). Only volunteers with scores in the normal range (TAS score: >39 and <61) were selected to participate. Participants were informed about the procedure and the purpose of the study and gave written informed consent. The study was designed and performed in accordance with the ethical principles of the Declaration of Helsinki and was approved by the Ethics Committee of the Psychology Department at the University of Bologna.

**Experimental task:** The experimental session was run in a sound-attenuated and dimly lit room. Participants sat in a relaxed position on a comfortable chair in front of a 17" PC monitor (refresh rate 60Hz) at a distance of 57cm. Prior to the experiment, a short practice session was administered to familiarize the participants with the task. The stimuli were presented on a PC running Presentation software (Version 0.60; www.neurobs.com) and consisted of 64 static color pictures of human bodies (two males and two females; 10°x 16°) with the faces blanked out. The images were selected from a validated database (Borgomaneri et al., 2012, 2014a). Half of the stimuli were the original pictures and the other half were mirror-reflected copies. Stimuli represented

bodies in different postures, in which implied motion was absent (static body posture) or motion was implied with different emotional expressions and body movement.

In particular, the body images included 16 static body postures (static body stimuli; S) in which neither motion nor emotion was implied, 16 neutral body postures in which motion was implied (neutral body stimuli; N), 16 fearful body postures in which motion was implied (fearful body stimuli; F) and 16 happy body postures in which motion was implied (happy body stimuli; H; Figure 1). Two independent psychophysical studies (Borgomaneri et al. 2012, 2014a) provided evidence that N, F and H are subjectively rated as conveying the same amount of implied motion information and as conveying more body motion information than S stimuli. Moreover, H and F were rated as more arousing than N and S. Critically, although H and F were rated as conveying positive and negative emotional valence, respectively, these two classes of stimuli received comparable arousal ratings. The stimuli were displayed against a white background, 118 to the left [left visual field (LVF) presentation] or the right [right visual field (RVF) presentation] of the central fixation point (28). Each trial started with a central fixation period (100ms), followed by the stimulus (500ms). Participants were asked to keep their gaze fixed on the central fixation and decide whether the presented stimulus was emotional (fearful or happy) or non-emotional (static or neutral) by pressing one of two vertically arranged buttons on the keyboard. The task was selected to balance the number of stimuli assigned to each response while maximizing the number of correct responses to minimize the rate of rejected epochs. Behavioral responses were recorded during an interval of 2400ms. Half of the subjects pressed the upper button with the middle finger to emotional stimuli and the lower button with the index finger to non-emotional stimuli, while the remaining half performed the task with the opposite button arrangement. Eye movements were monitored throughout the task

with electrooculogram (EOG; see below). Participants performed 12 blocks in an experimental session of 45min. In half of the blocks, the stimuli were presented in the LVF, while in the remaining half, they were presented in the RVF. Blocks with LVF and RVF presentation were interleaved, and the sequence of the blocks was counterbalanced between participants. In each block, 67 trials were randomly presented (16 trials x 4 body stimuli: static, motion neutral, motion fearful, motion happy=64 trials+3 practice trials). Each participant completed a total of 768 trials (384 trials in the LVF and 384 in the RVF).

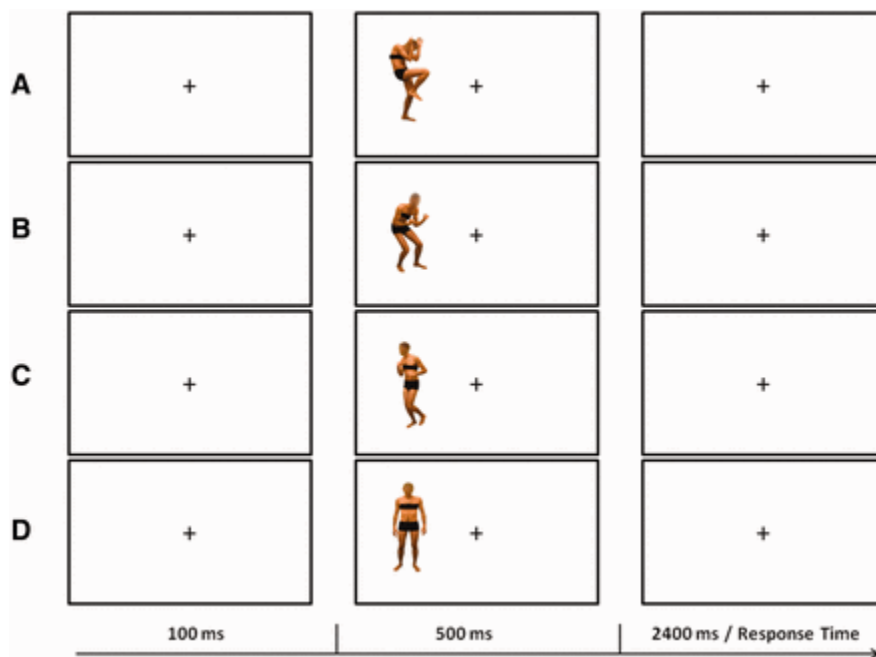


Fig. 1  
Graphical representation of the trial structure in the behavioral task. The figure depicts example trials with stimuli showing fearful (A), happy (B), neutral (C) and static body postures (D).

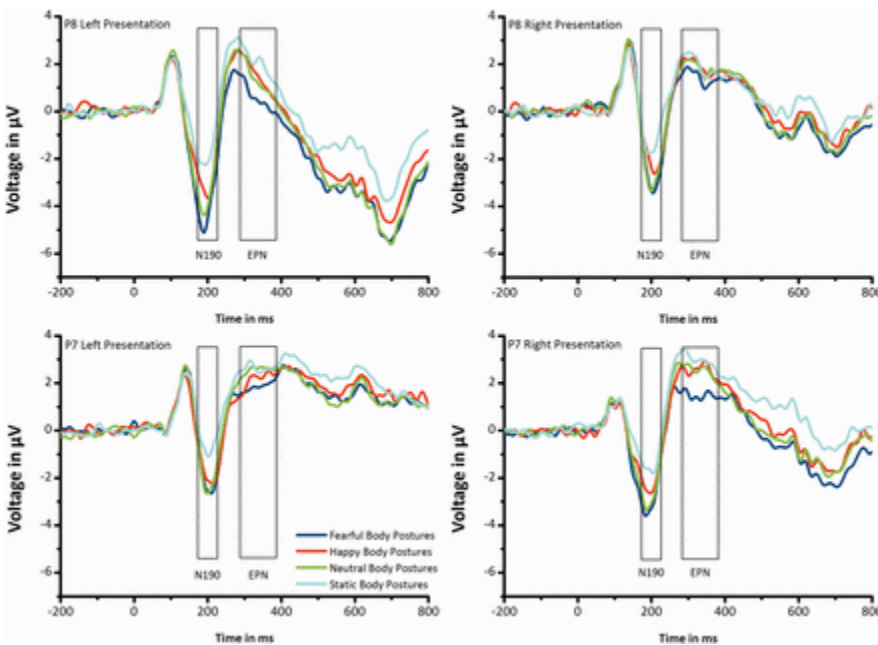
**EEG recording:** EEG was recorded with Ag/AgCl electrodes (Fast'n Easy-Electrodes, Easycap, Herrsching, Germany) from 27 electrode sites (Fp1, F3, F7, FC1, C3, T7, CP1, P3, P7, O1, PO7, Fz, FCz, Cz, CPz, Pz, Fp2, F4, F8, FC2, C4, T8, CP2, P4, P8, O2, PO8) and the right mastoid. The left mastoid was used as reference

electrode. The ground electrode was placed on the right cheek. Impedances were kept below 5k. All electrodes were off-line re-referenced to the average of all electrodes. Vertical and horizontal EOG was recorded from above and below the left eye and from the outer canthi of both eyes. EEG and EOG were recorded with a band-pass of 0.01–100Hz and amplified by a BrainAmp DC amplifier (Brain Products, Gilching, Germany). The amplified signals were digitized at a sampling rate of 500Hz and offline filtered with a 40-Hz low-pass filter.

**ERP data analysis:** ERP data were analyzed using custom routines in MATLAB 7.0.4 (The Mathworks, Natic, MA) and EEGLAB 5.03 (Delorme and Makeig, 2004; <http://www.sccn.ucsd.edu/eeglab>). Segments of 200ms before and 800ms after stimulus onset were extracted from the continuous EEG. The baseline window ran from -100ms to 0ms relative to stimulus onset. Epochs with incorrect responses were rejected (5.8% per body stimulus type). In addition, epochs contaminated with large

artifacts were identified using two methods from the EEGLAB toolbox (Delorme et al., 2007): (i) an epoch was excluded whenever the voltage on an EOG channel exceeded 100mV to remove epochs with large EOG peaks and (ii) an epoch was excluded whenever the joint probability of a trial exceeded five standard deviations to remove epochs with improbable data (mean excluded epochs: 9.6%). Remaining blinks and EOG horizontal artifacts were corrected using a multiple adaptive regression method (Automatic Artifact Removal Toolbox Version 1.3; [http://www.germangh.com/eeglab\\_plugin\\_aar/index.html](http://www.germangh.com/eeglab_plugin_aar/index.html); Gratton et al., 1983), based on the Least Mean Squares algorithm. Finally, epochs were discarded from the analysis when saccadic movements (>30mV on horizontal EOG channels) were registered in a time window of 500ms following stimulus onset (1.73%). The remaining epochs (mean: 83 epochs per body stimulus type) were averaged separately for each participant and

each body stimulus type. The N190 amplitude was quantified as the mean amplitude in a time window of 160–230ms post-stimulus presentation (Figure 2). Scalp topographies for the N190 component were calculated as mean amplitude in a time window of 160–230ms post-stimulus presentation (Figure 3g and h). In addition, the EPN was calculated as the mean amplitude in a time window of 290–390ms post-stimulus presentation (Figure 2). Both the N190 and the EPN mean amplitudes were analyzed with a three-way analysis of variance (ANOVA) with electrode (P8, P7), visual field (LVF, RVF) and body stimulus (static: S; neutral: N; fearful: F; happy: H) as within-subjects variables. To compensate for violations of sphericity, Greenhouse–Geisser corrections were applied whenever appropriate (Greenhouse and Geisser, 1959) and corrected P values (but uncorrected degrees of freedom) are reported. Post-hoc comparisons were performed using the Newman–Keuls test.



**Fig. 2.** Grand-average ERPs elicited by fearful, happy, neutral and static body postures. ERP waveforms at the representative electrodes P8 (A,B) and P7 (C,D) when stimuli were presented in the LVF (A,C) and in the RVF (B,D).

### 5.1.2. Results

#### ERP results:

**N190:** The mean N190 amplitude averaged for all body stimuli (fearful, happy, neutral and static body postures) reached a maximum negative deflection in a time window of 160–230ms on electrodes P7 and P8, as shown in the scalp topographies (Figure 3g and h). Electrodes P7 and P8 were therefore chosen as electrodes of interest in the N190 analyses, in line with previous studies (Stekelenburg and de Gelder, 2004; Thierry et al., 2006). Grand average waveforms for the electrodes P7 and P8 are shown in Figure 2. The ANOVA showed a significant main effect of Body stimulus ( $F(3, 63)=29.14$ ;  $P<0.0001$ ;  $\eta^2=0.58$ ), a significant Electrode x Visual field interaction ( $F(1, 21)=11.84$ ;  $p=0.002$ ;  $\eta^2=0.36$ ) and, more importantly, a significant Electrode x Visual field x Body stimulus interaction ( $F(3, 63)=7.43$ ;  $p=0.0003$ ;  $\eta^2=0.26$ ). This interaction was further explored with two-way ANOVAs with Visual field (LVF, RVF) and Body stimulus (static, neutral, fearful, happy) as within subjects factors for the two electrodes (P8 and P7) separately, to investigate possible differences between the two hemispheres. The results of the ANOVA on the N190 amplitude over electrode P8, located in the right hemisphere, revealed a significant main effect of Visual field ( $F(1, 21)=6.74$ ;  $p=0.016$ ;  $\eta^2=0.24$ ), with larger amplitudes for stimuli presented in the contralateral LVF (-2.83mV), compared with the ipsilateral RVF (-1.65mV;  $p=0.016$ ). Moreover, the main effect of Body stimulus was significant ( $F(3, 63)=18.42$ ;  $P<0.0001$ ;  $\eta^2=0.47$ ). Post-hoc analyses showed a significantly smaller N190 amplitude in response to static postures (-1.27mV) compared with all the motion postures (all  $P_s<0.001$ ; H: -2.10mV; N: -2.65mV; F: -2.93mV). In addition, a significant difference was found between the emotional postures, with a significantly larger N190 amplitude for fearful postures (-2.93mV;  $p=0.003$ ) compared with happy postures (-



2.10mV). More importantly, the Visual field x Body stimulus interaction was significant ( $F(3, 63)=6.99$ ;  $p=0.0007$ ;  $\eta p^2=0.25$ ). Post-hoc analyses revealed that, in both the LVF and the RVF, static postures (S-LVF: -1.60mV; S-RVF: -0.93mV) elicited a significantly smaller N190 compared with all the motion postures (LVF: all  $P_s < 0.0001$ ; H-LVF: -2.66mV; N-LVF: -3.24mV; F-LVF: -3.80mV; RVF: all  $P_s < 0.0006$ ; H-RVF: -1.54mV; N-RVF: -2.06mV; F-RVF: -2.07mV). Also, in both the LVF and the RVF, the N190 amplitude was significantly larger for fearful postures than for happy postures (F-LVF vs H-LVF:  $p=0.0001$ ; F-RVF vs H-RVF:  $P=0.01$ ). In addition, in the LVF, the N190 amplitude was significantly larger for fearful postures (F-LVF: -3.80mV) than for neutral postures (N-LVF: -3.24mV;  $P \leq 0.001$ ; Figure 3a, b, e and f).

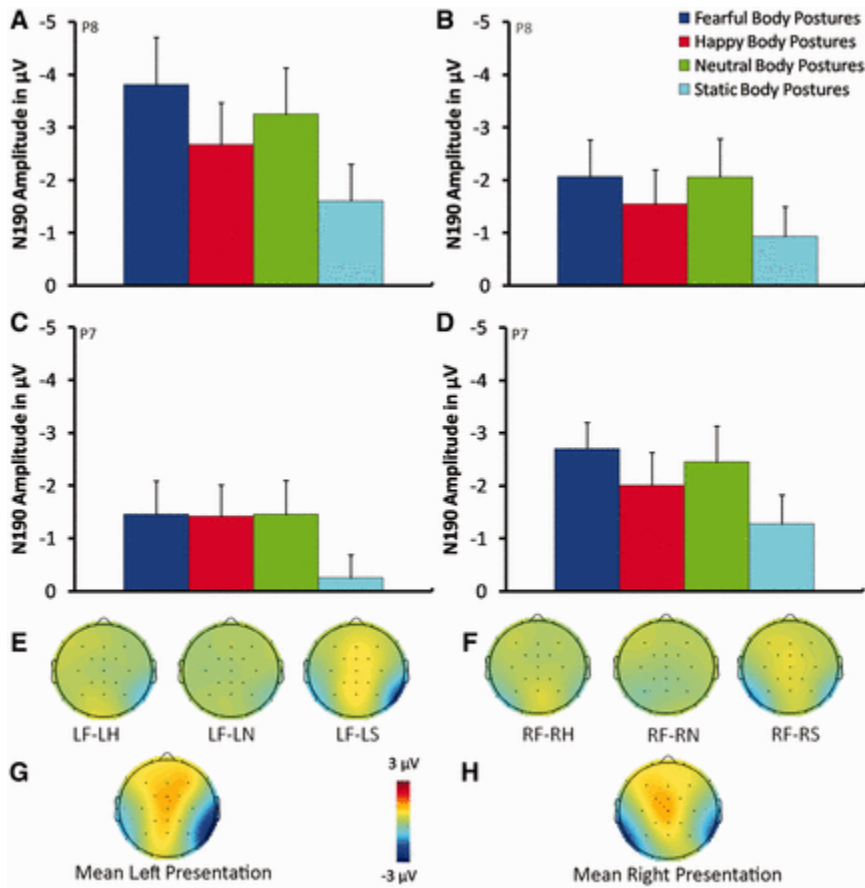


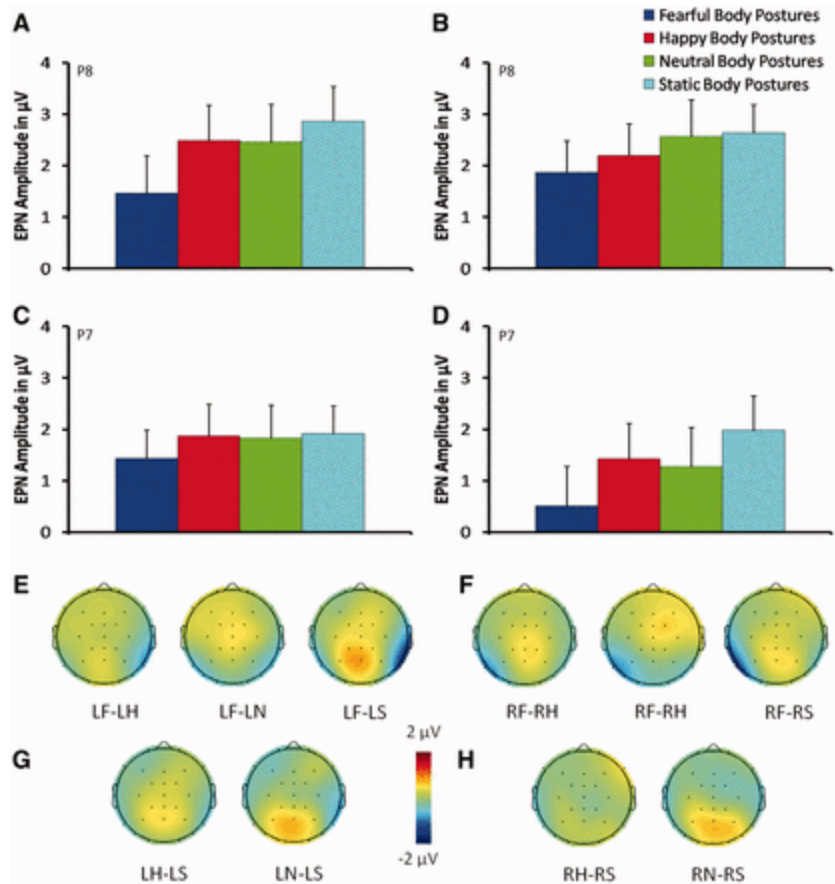
Fig. 3

Mean N190 amplitude elicited by fearful, happy, neutral and static body postures from electrode P8 in the right hemisphere (A, B) and electrode P7 in the left hemisphere (C, D) when stimuli were presented in the LVF (A, C) and in the RVF (B, D). Scalp topographies of the difference in mean N190 amplitude between fearful and other body stimuli (happy, neutral and static) when stimuli were presented in the LVF (E) and in the RVF (F) in a time window of 160–230 ms. (G) and (H) represent scalp topographies of the mean N190 amplitude averaged for all body stimuli (fearful, happy, neutral and static body postures) in a time window of 160–230 ms when stimuli were presented in the LVF and RVF, respectively. Error bars represent standard error of the mean (SEM). LF, left fearful body posture; LH, left happy body posture; LN, left neutral body posture; LS, left static body posture; RF, right fearful body posture; RH, right happy body posture; RN, right neutral body posture; RS, right static body posture.

The ANOVA for electrode P7, located in the left hemisphere, revealed a significant main effect of Visual field ( $F(1, 21)=11.44$ ;  $p=0.002$ ;  $\eta^2=0.35$ ), with larger N190 amplitudes for stimuli presented in the contralateral RVF (-2.11mV) compared with the ipsilateral LVF (-1.14mV;  $P=0.002$ ). In addition, the main effect of Body stimulus was significant ( $F(3, 63)=14.26$ ;  $P<0.0001$ ;  $\eta^2=0.4$ ). Post hoc comparisons revealed a significantly smaller N190 amplitude in response to static postures (-0.76mV), compared with all the motion postures (all  $P_s \leq 0.0001$ ; H: -1.71mV; N: -1.95mV; F: -2.08mV; Figure 3c and d). However, in contrast to the results from electrode P8, the N190 amplitude recorded from electrode P7 did not significantly differ between fearful and happy body postures ( $P=0.24$ ). No other comparisons were significant (all  $P_s > 0.57$ ).

**EPN:** The subsequent EPN amplitudes were measured at the same electrode locations as the N190, in a time window of 290–390ms post-stimulus onset (Figure 2). The ANOVA showed a significant main effect of Body stimulus ( $F(3, 63)=14.87$ ;  $P<0.0001$ ;  $\eta^2=0.41$ ) and, more interestingly, a significant Electrode x Visual field x Body stimulus interaction ( $F(3, 63)=9.07$ ;  $P=0.0002$ ;  $\eta^2=0.3$ ). This interaction was further explored with two-way ANOVAs with Visual field (LVF, RVF) and Body stimulus (static, neutral, fearful, happy) as within-subject factors for the two electrodes (P8 and

P7) separately, to investigate possible differences between the two hemispheres. The ANOVA for electrode P8, in the right hemisphere, revealed a significant main effect of Body stimulus ( $F(3, 63)=7.71$ ;  $P<0.0002$ ;  $\eta^2=0.27$ ), showing a significant more negative amplitude in response to fearful postures (0.97mV), compared with the remaining postures (all  $P_s<0.006$ ; H: 1.64mV; N: 1.55mV; S: 1.95mV). The interaction Visual field x Body stimulus was also significant ( $F(3, 63)=5.63$ ;  $P=0.003$ ;  $\eta^2=0.21$ ). Post-hoc analyses revealed that, both in the LVF and the RVF, fearful body postures (F-LVF: 0.51mV; F-RVF: 1.43mV) elicited the most negative amplitude compared with the remaining postures (LVF: all  $P_s<0.0001$ ; H-LVF: 1.42mV; N-LVF: 1.27mV; S-LVF: 1.98mV; RVF: all  $P_s\leq 0.03$ ; H-RVF: 1.86mV; NRVF: 1.83mV; S-RVF: 1.92mV; see Figure 4a, b, e and f). In addition, in the LVF, happy (1.42mV) and neutral (1.27mV) body postures showed a significantly more negative amplitude than compared with static body postures (1.98mV; all  $P_s<0.02$ ; Figure 4a, b, g and h). The ANOVA for electrode P7, in the left hemisphere, revealed a significant main effect of Body stimulus ( $F(3, 63)=7.47$ ;  $P=0.0002$ ;  $\eta^2=0.26$ ). Post-hoc comparisons revealed that negative amplitude was significantly greater in response to fearful postures (1.66mV), compared with the remaining postures (all  $P_s<0.006$ ; H: 2.34mV; N: 2.51mV; S: 2.75mV; Figure 4c–f). No other comparisons were significant (all  $P_s>0.48$ ).



**Fig. 4**

Mean EPN amplitude elicited by fearful, happy, neutral and static body postures from electrode P8 in the right hemisphere (A, B) and electrode P7 in the left hemisphere (C, D) when stimuli were presented in the LVF (A, C) and in the RVF (B, D). Scalp topographies of the difference in mean EPN amplitude between fear and other body stimuli (happy, neutral and static) when stimuli were presented in the LVF (E) and in the RVF (F) in a time window of 290–390 ms. Scalp topographies of the difference in mean EPN amplitude between static and other body stimuli (happy, neutral) when stimuli were presented in the LVF (G) and in the RVF (H) in a time window of 290–390 ms. Error bars represent standard error of the mean (SEM). LF, left fearful body posture; LH, left happy body posture; LN, left neutral body posture; LS, left static body posture; RF, right fearful body posture; RH, right happy body posture; RN, right neutral body posture; RS, right static body posture.

**Behavioral results:** Reaction times (RTs), accuracy scores and inverse efficiency scores (IES=reaction times/accuracy) were analyzed with separate ANOVAs with Visual field (LVF, RVF) and Body stimulus (static, neutral, fearful, happy) as within-subjects variables. The analysis on RTs revealed a significant main effect of Body

stimulus ( $F(3, 63)=31.81$ ;  $P<0.0001$ ;  $\eta^2=0.6$ ), showing faster RTs for static body postures (654ms) compared with fearful (756ms), happy (764ms) and neutral postures (797ms; all  $P_s<0.0001$ ). In addition, RTs for neutral body postures were significantly slower than for fearful ( $P=0.02$ ) and happy body postures ( $P=0.03$ ). The ANOVA performed on the accuracy scores revealed a significant main effect of Body stimulus ( $F(3, 63)=8.42$ ;  $P<0.0001$ ;  $\eta^2=0.29$ ), showing that participants were slightly more accurate in responding to static body postures (98%), compared with fearful (93%;  $P=0.004$ ), happy (93%;  $P=0.007$ ) and neutral postures (90%;  $P=0.0001$ ). Finally, the ANOVA on inverse efficiency scores revealed a significant main effect of Body stimulus ( $F(3, 63)=21.62$ ;  $P<0.0001$ ;  $\eta^2=0.5$ ), showing significantly lower scores (reflecting better performance) for static body postures (661ms; all  $P_s\leq 0.0001$ ), compared with fearful (811ms), happy (822ms) and neutral postures (891ms). In addition, IES for the neutral body postures was significantly higher than for the remaining postures (all  $P_s<0.02$ ).

### 5.1.3. Discussion

Seeing images of bodies elicits a robust negative deflection peaking at 190ms post-stimulus onset (N190) reflecting the early structural encoding of these stimuli (Thierry et al., 2006) and a subsequent relative negativity (EPN) indexing attentional engagement to salient stimuli (Schupp et al., 2006; Olofsson et al., 2008). This study revealed that information concerning both the presence of motion and the emotions expressed by different body postures are able to modulate the early stage of the visual encoding of bodies and the attentional engagement process as reflected by changes in the amplitudes of N190 and EPN, respectively. In particular, laterally presented pictures of bodies in different postures strongly modulated the N190 component. Interestingly, this component showed differential sensitivity to the observed body postures in the two cerebral hemispheres. On the one hand, the right hemisphere showed a modulation of the N190 both for the motion content (i.e. all the postures implying motion elicited larger N190 amplitudes compared with static, no-motion body postures) and for the emotional content (i.e. fearful postures elicited the largest N190 amplitude).

On the other hand, the left hemisphere showed a modulation of the N190 only for the motion content, with no modulation for the emotional content. These findings suggest partially distinct roles of the two cerebral hemispheres in the visual encoding of emotional and motion information from bodies. In addition, at a later stage of perceptual representation reflecting selective attention to salient stimuli, an enlarged EPN was observed for fearful stimuli in both hemispheres, reflecting an enhanced processing of motivationally relevant stimuli (Schupp et al., 2006; Olofsson et al., 2008). Electrophysiological studies suggest that, akin to the N170 for faces, the N190

component represents the process of extracting abstract and relevant properties of the human body form for categorization (Thierry et al., 2006) and is considered the earliest component indexing structural features of human bodies (Taylor et al., 2010).

Our study expands these ideas by demonstrating that the stage of structural encoding reflected by the N190 entails not only the categorization of the visual stimulus as a body but also an analysis of motion-related and emotional features of the body posture. In other words, the visual encoding stage involves not only a perceptual representation of the form, configuration and spatial relations between the different body parts (Taylor et al., 2007, 2010), but it also reflects a discrimination between body postures conveying information about the presence of actions and emotions. It has been argued that EBA (i.e. the putative neural generator of the N190; Thierry et al., 2006; Taylor et al., 2010) has a pivotal role in creating a cognitively unelaborated but perceptually detailed visual representation of the human body (Peelen and Downing, 2007; Downing and Peelen, 2011), which is forwarded to higher cortical areas for further analysis.

On the other hand, EBA is thought to be modulated by top-down signals from multiple neural systems, including those involved in processing emotion and action information (Downing and Peelen, 2011). Thus, the finding that the N190 is sensitive to information about motion and emotions conveyed by human body postures suggests that emotion- and action-related signals are rapidly extracted from visual stimuli and can exert a fast top-down modulation of the neural processing reflecting structural encoding of bodies in occipitotemporal areas, i.e. the N190. The smaller N190 amplitudes for static bodies than for bodies with implied motion suggest that both hemispheres operate a perceptual distinction between bodies with static postures and bodies performing actions. Because of the highly adaptive value of motion perception,

observers typically extract motion-related information from static images where motion is implied (Freyd, 1983; Verfaillie and Daems, 2002). Occipitotemporal visual areas have been suggested to encode dynamic visual information from static displays of “moving” objects (e.g. human area MT, Kourtzi and Kanwisher, 2000; STS, when implied motion information is extracted from pictures of biological entities, Puce and Perrett, 2003; Perrett et al. 2009) and to respond to static images of human body postures implying an action (Peigneux et al., 2000; Kourtzi et al., 2008). Thus, the static snapshots of moving bodies used here were not only a necessary methodological substitute for real motion that was required to reliably record ERPs but also a sufficient substitute for understanding how the human visual system represents human body movements. Notably, action observation is also known to activate a wide frontoparietal network of sensorimotor regions involved in action planning and execution. Indeed, observing images of humans during ongoing motor acts is known to enhance the excitability of the motor system (Urgesi et al., 2010; Borgomaneri et al., 2012; Avenanti et al., 2013a,b), where the perceived action is dynamically simulated (Gallese et al., 2004; Nishitani et al., 2004; Keysers and Gazzola, 2009; Gallese and Sinigaglia, 2011). Such motor simulation appears to emerge very early in time (<100ms after stimulus onset in some cases, e.g. van Schie et al., 2008; Lepage et al., 2010; Ubaldi et al., 2013; Rizzolatti et al., 2014) and is thought to facilitate visual perception through feedback connections from motor to visual areas (Wilson and Knoblich, 2005; Kilner et al., 2007; Schippers and Keysers, 2011; Avenanti et al., 2013a; Tidoni et al., 2013). Thus, the observed enhancement of structural encoding for postures implying motion and action compared with static postures seems to indicate increased perceptual representation of the bodies, possibly triggered by fast action simulation processes in interconnected frontoparietal areas.



On the other hand, a finer perceptual distinction, discriminating not only the presence of action but also the emotional content of that action, is evident only in the right hemisphere, where the N190 was differentially modulated by fearful and happy body postures, with fearful postures eliciting the largest N190 amplitude. This emotional modulation of structural encoding might reflect an adaptive mechanism, in which the perceptual representation of body stimuli signaling potential threats is enhanced by top-down modulations. In line with this, neuroimaging studies have shown that fearful bodies increase the BOLD signal in the temporo-occipital areas from which the N190 originates and in nearby visual areas (Hadjikhani and de Gelder, 2003; Peelen et al., 2007; Gre`zes et al., 2007; Pichon et al., 2008; Van de Riet et al., 2009). Importantly, fearful bodies are known to enhance activation in the amygdala (Hadjikhani and de Gelder, 2003; de Gelder et al., 2004; Van de Riet et al., 2009), the key subcortical structure for signaling fear and potential threat (Adolphs, 2013; LeDoux, 2014). Notably, the magnitude of amygdala activation predicts activity in EBA and FBA during perception of emotional bodies (Peelen et al., 2007). Therefore, the enhanced N190 over the right occipitotemporal electrodes might reflect a rapid and distant functional influence of the amygdala on interconnected visual cortices, useful for processing threat signals efficiently and implementing fast motor reactions (Vuilleumier et al., 2004; Borgomaneri et al., 2014b). Similarly, somatosensory and motor regions, crucial to the processing of threat-related expressions (Adolphs et al., 2000; Pourtois et al., 2004; Banissy et al., 2010; Borgomaneri et al., 2014a), might also participate in this top-down influence. Indeed, somato-motor regions are connected to occipitotemporal areas via the parietal cortex (Keysers et al., 2010; Rizzolatti et al., 2014) and exert a critical influence on visual recognition of emotional expressions quite early in time (i.e. 100–170ms after stimulus onset; Pitcher et al., 2008; Borgomaneri et al., 2014a), which may be compatible with the observed N190 modulation. Although previous

electrophysiological findings showed a modulation of fearful body expressions at the stage of the P1 component (i.e. before structural encoding of the stimulus has taken place; Mereen et al., 2005; Van Heijnsbergen et al., 2007), the potentials peaking in the range of the N1 seem to offer more reliable measures of both face and body-selective perceptual mechanisms. Indeed, earlier potentials such as the P1 could be modulated to a greater degree by low-level features of the stimuli, as they are highly sensitive to physical properties of visual stimuli (Halgren et al., 2000; Rossion and Jacques, 2008).

The observed emotional modulation of the N190 exclusively over the right hemisphere is in keeping with the idea of a possible right hemisphere advantage in processing emotions (Gainotti et al., 1993; Ladavas et al., 1993; Adolphs et al., 2000; Borod, 2000). Alternatively, the more detailed modulation of structural encoding processes observed in the right hemisphere could be due to a higher sensitivity to human bodies, as suggested by preferential activation in response to body stimuli in the right EBA (Downing et al., 2001; Chan et al., 2004; Saxe et al., 2006) and in a broad network of right cortical areas (Caspers et al., 2010). In keeping with the idea of a right hemisphere advantage in processing emotional body postures, recent transcranial magnetic stimulation studies have shown that motor excitability over the right (but not the left) hemisphere is sensitive to the emotional content of the observed body posture at a latency compatible with the initial part of the N190 component (Borgomaneri et al., 2014a). This suggests a strict functional coupling between visual and motor representations during the processing of emotional body postures, which might favor perception of and adaptive motor responses to threatening stimuli.

Interestingly, at a later stage of visual processing (i.e. 300ms poststimulus onset), the EPN component was enhanced for fearful stimuli in both hemispheres. The EPN is a relative negativity for emotional stimuli (Schupp et al., 2006). This emotional

modulation reflects attentional capture driven by salient emotional stimuli and might reflect the degree of attention needed to recognize relevant signals (Olofsson et al., 2008). Previous studies have shown increases in the amplitude of the EPN in response to both emotional scenes (Schupp et al., 2003, 2004b; Thom et al., 2014) and emotional faces (Sato et al., 2001; Schupp et al., 2004a; Frühholz et al., 2011; Calvo and Beltran, 2014). Similar to the findings of present study, the EPN is also enhanced during observation of hand gestures, with a greater effect for negatively valenced gestures (Flaisch et al., 2009, 2011). This suggests that viewing isolated body parts with emotional relevance also modulates this component. The present results add to the previous studies by showing strong EPN sensitivity to whole body expressions of fear, supporting the idea that fearful body postures represent a highly salient category of stimuli, able to engage selective visual attention to favor explicit recognition of potentially threatening signals (de Gelder et al., 2004, 2010; Kret et al., 2011; Borgomaneri et al., 2014a).

Notably, our data suggest that attentional processes are enhanced by fearful postures in both hemispheres, indicating that, at later stages of visual processing, both the right and the left hemispheres concur to engage attentional resources to aid recognition of salient emotional stimuli. However, it is interesting to note that the right hemisphere also maintains a higher capacity to discriminate between the different body postures at this later stage, as suggested by an increased negativity for happy and neutral body postures compared with static body postures. Interestingly, the emotional modulations observed both at the early stage of structural encoding and at the later attentional engagement stage might be a by-product of the interaction between movement and emotion-related information conveyed by emotional body postures.

Indeed, bodies express emotions through movements, therefore providing concurrent motion-related information.

Further studies are needed to disentangle the contributions of emotion and movement-related information by investigating ERP modulations in response to emotional body postures with a minimal amount of motion content (e.g. sad body postures).

Overall, these results suggest that information pertaining to motion and emotion in human bodies is already differentially processed at the early stage of visual structural encoding (N190), in which a detailed representation of the form and configuration of the body is created. At this early stage, the right hemisphere seems prominent in processing the emotional content of body postures, as shown by the effects of laterally presented body postures on structural encoding.

At a later stage of visual processing (EPN), the relevant and salient information represented by fearful body postures recruits visual attention networks in both hemispheres, which might facilitate recognition of potentially dangerous stimuli. Finally, the modulations observed in the visual processing of different body postures, both at the visual encoding and attentional engagement stages, are reminiscent of modulations seen in visual face processing (Batty and Taylor, 2003; Stekelenburg and de Gelder, 2004; Schupp et al., 2004a; Frühholz et al., 2011; Calvo and Beltran, 2014), suggesting that face and body processing might involve distinct but similar perceptual mechanisms. This highly efficient and specialized structural encoding, and the subsequent attentional engagement for salient stimuli, may represent an adaptive mechanism for social communication that facilitates inferences about the goals, intentions and emotions of others.

## **5.2. Experiment 7:** The effect of alexithymia on early visual processing of emotional body postures

The ability to perceive and categorize emotional stimuli is highly relevant in social environments. Indeed, rapid processing of potentially threatening stimuli is crucial for minimizing the negative consequences associated with unpleasant cues. In support of this view, recent evidence has shown that unpleasant stimuli are detected more quickly than both pleasant and neutral stimuli (Fox et al., 2000; Hansen and Hansen, 1988; Öhman et al., 2001).

In addition, negative stimuli are associated with enhanced activation in perceptual occipito-temporal areas (Taylor et al., 2000) and in subcortical structures, such as the amygdala, that are pivotal for emotional processing (Breiter et al., 1996; FusarPoli et al., 2009; Lane et al., 1998; Oya et al., 2002). These findings suggest that more processing resources are devoted to the visual processing of unpleasant stimuli than to pleasant or neutral stimuli (Carretié et al., 2009; Vuilleumier, 2002). Electrophysiological studies have also shown that fearful faces enhance early event-related potential (ERP) components such as the P1, reflecting exogenous spatial orienting of attention toward fearful stimuli (Pourtois et al., 2005, 2004). In addition, both explicit (Batty and Taylor, 2003; Stekelenburg and de Gelder, 2004) and implicit (Cecere et al., 2014; Pegna et al., 2011, 2008) processing of fearful faces can modulate early stages of perceptual encoding of facial features and configurations, as indexed by the occipito-temporal N170 component (Batty and Taylor, 2003; Bentin et al., 1996).

Moreover, at a later stage of perceptual representation (around 300 ms after stimulus onset), faces expressing negative emotions increase stimulus-driven

attentional capture, as suggested by a pronounced early posterior negativity (EPN; Bayer and Schacht, 2014; Calvo and Beltrán, 2014; Frühholz et al., 2011; Rellecke et al., 2012; Schupp et al., 2004; Valdés-Conroy et al., 2014).

Besides facial expressions, human body postures represent a powerful tool for inferring the internal states of others (de Gelder et al., 2010). Indeed, body postures convey information about others' actions and emotions, both of which are useful for interpreting goals, intentions and mental states. Compared to faces, body postures offer the possibility to capture these signals from longer distances. Similar to the face-related N170, the observation of bodies elicits an early occipito-temporal negative deflection peaking at 190 ms after stimulus presentation, which has been termed the N190. It is thought to be generated in a restricted area of the occipito-temporal cortex, corresponding to the extrastriate body area (EBA; Meeren et al., 2013; Taylor et al., 2010; Thierry et al., 2006). This electrophysiological signature reflects the extraction of abstract properties of the human body form for categorization (Thierry et al., 2006), and represents the earliest component indexing structural features of human bodies (Taylor et al., 2010). Interestingly, both motion- and emotion-related information conveyed by body postures can modulate the N190 (Borhani et al., 2015). Indeed, in a recent electrophysiological study (Borhani et al., 2015), bodies with static or implied motion postures, and with or without emotional content (fearful, happy or neutral), were presented peripherally to the left or the right of a central fixation point. The N190 component, recorded from the right hemisphere, was modulated both by the presence of implied motion (i.e., larger N190 amplitude in response to body postures conveying implied motion compared to static postures) and by emotional content (i.e., larger N190 amplitude in response to fearful body postures). These modulations suggest that this visual processing stage encodes not only a perceptual representation of the visual

stimulus as a body, but also a more detailed analysis of motion and emotion information conveyed by body postures. Notably, the study by (Borhani et al. 2015) did not show any modulation of the early P1 component. This is in keeping with other electrophysiological findings (Stekelenburg and de Gelder, 2004) that emotional stimuli modulate ERP components in the same time window as the N190 (i.e., the Vertex Positive Potential, which is considered the fronto-central counterpart of the N190), but not earlier components. Moreover, evidence for modulations of the P1 in response to emotional bodies is inconsistent (Meeren et al., 2005; van Heijnsbergen et al., 2007), possibly because the P1 is highly sensitive to the physical properties of the stimulus (Halgren et al., 2000; Rossion and Jacques, 2008).

At a later stage of perceptual representation (i.e., 300 ms post-stimulus onset), viewing fearful body postures elicits a pronounced early posterior negativity (EPN; Borhani et al., 2015). The EPN is a ERP difference in the processing of emotionally relevant stimuli and neutral stimuli, and occurs 200- 300 ms after stimulus presentation (Schupp et al., 2006). This differential ERP appears as a negative deflection over temporo-parietal areas, and reflects exogenous attentional capture driven by salient emotional stimuli and the degree of attention needed to recognize relevant signals (Olofsson et al., 2008), such as body postures expressing fear.

These results suggest the existence of a specialized perceptual mechanism tuned to the emotion and action-related information conveyed by human body postures. Recent evidence suggests that individual emotional skills (Meaux et al., 2014), empathic dispositions (Choi et al., 2014) and personality traits, such as antisocial behavioral tendencies (Pfabigan et al., 2012), might affect visual processing of emotional stimuli. Because the rapid perception of negative cues in social environments

is highly adaptive, the influence of personality traits on visual processing of emotional stimuli, such as emotional body expressions, is an important avenue for research.

One relevant trait is alexithymia, a multifaceted personality construct that is expressed with varying intensity in the general population, and characterized by a deficit in identifying, differentiating and describing feelings (Herbert et al., 2011; Parker et al., 2008; Taylor et al., 1991). Importantly, people with high levels of alexithymia exhibit difficulties not only in processing their own emotions, but also in processing the emotions expressed by others (Parker et al., 1993; Sifneos, 1973). Alexithymic individuals show altered recognition of emotional stimuli (Grynberg et al., 2012; Ihme et al., 2014) and decreased activation of the amygdala during presentation of emotional stimuli (Jongen et al., 2014; Moriguchi and Komaki, 2013), specifically negative stimuli (Kugel et al., 2008; Pouga et al., 2010; Reker et al., 2010; for a recent meta-analysis: van der Velde et al., 2013). However, it is unknown whether early visual processing of the emotional information conveyed by body postures might be similarly affected. Thus, this study was designed to investigate, using the high temporal resolution of ERPs, whether participants with low levels of alexithymia (LA) and high levels of alexithymia (HA) show similar electrophysiological modulations in response to body postures conveying information about others' actions and emotions.

We studied both the early stage of structural body encoding, indexed by the N190 component, and the later stage of visual selective attention, reflected by the subsequent EPN component. In line with evidence suggesting impaired processing of emotional stimuli in alexithymia (Grynberg et al., 2012; Ihme et al., 2014), we expected that only LA participants would exhibit detailed visual encoding of the emotional content of body postures, with the greatest N190 amplitude in response to fearful body postures. Indeed, we expected that HA participants would not show any emotional



modulation at the early stage of structural encoding, and, in particular, no fear-related enhancement of the N190 component.

In addition, we explored whether alexithymia might also influence a later stage of perceptual representation, reflecting selective attention to salient stimuli (EPN).

### 5.2.1. Methods

**Participants:** Three-hundred university students completed the 20-item Toronto Alexithymia Scale (TAS-20; Taylor, Bagby, & Parker, 2003). Individuals with high and low TAS-20 total scores ( $n = 18$ , top quartile score  $>61$ ;  $n = 16$ , bottom quartile score  $<36$ ) were selected in order to obtain a sample with as large a variance on alexithymia as possible. The alexithymia module of the structured interview for the Diagnostic Criteria for Psychosomatic Research (DCPR) (Mangelli, Semprini, Sirri, Fava, & Sonino, 2006; Porcelli & Rafanelli, 2010; Porcelli & Sonino, 2007), previously used in alexithymia research (Grandi, Sirri, Wise, Tossani, & Fava, 2011), was also used in the present study to further confirm the presence or absence of alexithymia. In addition, due to the high association between alexithymia and depression (Allen, Qian, Tsao, Hayes, & Zeltzer, 2011; Hintikka, Honkalampi, Lehtonen, & Viinamäki, 2001; Honkalampi, Hintikka, Tanskanen, Lehtonen, & Viinamäki, 2000), the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) was administered to exclude participants with high levels of depression. Participants were included in the study if (i) they had no history of neurological, major medical or psychiatric disorder and (ii) their scores on the TAS-20 and the DCPR were congruent. Two participants with a high TAS-20 score and a low DCPR score were discarded; no participants reported high levels of depression on the BDI. All participants had equivalent educational backgrounds and were students at the University of Bologna. Thirty-two right-handed healthy volunteers were selected to take part in the experiment after screening for

alexithymia: 16 HA participants (TAS, mean  $\pm$  standard deviation:  $63.62 \pm 2.68$ ; 6 males; mean age 20.68; range 18–25 years old) and 16 LA participants (TAS, mean  $\pm$  standard deviation  $31.56 \pm 2.75$ ; 6 males; mean age 21.18; range 19–26 years old). The two groups were matched in terms of sex and age. The two groups did not differ in terms of BDI score ( $t(30) = -1.41$ ;  $p = .16$ ). All participants gave their written informed consent to participate after having been informed about the procedure and the purpose of the study. The study was designed and performed in accordance with the ethical principles of the Declaration of Helsinki, and was approved by the Ethics Committee of the University of Bologna Psychology Department.

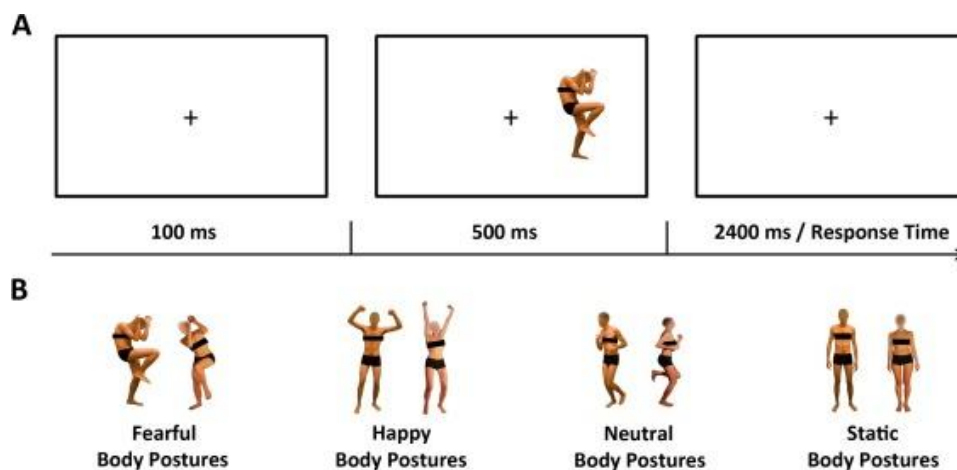


Fig. 1. (A) Graphical representation of the trial structure in the behavioral task. The figure depicts an example trial with fearful body posture stimuli. (B) Example stimuli showing fearful, happy, neutral and static body postures.

### Experimental task

The experimental session was run in a sound-attenuated and dimly lit room. Participants sat in a relaxed position on a comfortable chair in front of a 17" PC monitor (refresh rate: 60 Hz) at a

distance of approximately 57 cm. Prior to the experiment, a short practice session was administered to familiarize participants with the task.

The stimuli were presented on a PC running Presentation software (Version 0.60; [www.neurobs.com](http://www.neurobs.com)), and consisted of 64 color pictures of human bodies (2 males and 2 females;  $10^\circ \times 16^\circ$ ) in which faces were blanked out. The images were selected from a validated database (Borgomaneri et al., 2015, Borgomaneri et al., 2012). Half of the stimuli were the original pictures, and the other half were mirror-reflected copies. The stimuli represented bodies in different postures, in which motion was either absent (static body posture) or implied. The implied motion postures depicted either neutral or emotionally expressive body movements. In particular, the body images included 16 static body postures (static body stimuli), in which neither motion nor emotion was implied, 16 neutral body postures in which motion was implied (neutral body stimuli), 16 fearful body postures in which motion was implied (fearful body stimuli) and 16 happy body postures in which motion was implied (happy body stimuli; Fig. 1). Two psychophysical studies using the same body images (Borgomaneri, Gazzola, & Avenanti, 2015; Borgomaneri, Vitale, Gazzola, Avenanti, 2015) provided evidence that the neutral, fearful and happy body stimuli are subjectively rated as conveying the same amount of implied motion, and as conveying more body motion than the static body stimuli. Moreover, the fearful and happy body stimuli were rated as more arousing than the neutral and static body stimuli. Critically, while fearful and happy body stimuli were respectively rated as negative and positive, in terms of valence, these two classes of stimuli received comparable arousal ratings.

The stimuli were displayed against a white background,  $11^\circ$  to the left (left visual field presentation; LVF) or the right (RVF) of the central fixation point ( $2^\circ$ ). Each trial started with a central fixation period (100 ms), followed by the stimulus (500 ms). Participants were asked to keep their gaze fixed on the central fixation point and decide whether the presented stimulus was emotional (fearful or happy body stimuli) or non-emotional (static or neutral body stimuli) by

pressing one of two vertically-arranged buttons on the keyboard. Behavioral responses were recorded during an interval of 2400 ms. Half the participants pressed the upper button with their middle finger to report emotional stimuli and the lower button with their index finger to report non-emotional stimuli, while the remaining half performed the task with the opposite button arrangement. Eye movements were monitored throughout the task with electrooculogram (EOG; see below).

Participants performed twelve blocks in an experimental session of approximately 45 min. In half of the blocks the stimuli were presented in the LVF, while in the remaining half they were presented in the RVF. Blocks with LVF and RVF presentation were interleaved, and block sequence was counterbalanced between participants. Each block contained 67 trials presented in a random order (16 trials × 4 body stimuli: static, neutral, fearful, happy = 64 trials + 3 practice trials). Each participant completed a total of 804 trials. The analysis was run on a total of 768 trials, i.e., excluding practice trials.

### **EEG recording**

EEG was recorded with Ag/AgCl electrodes (Fast'n Easy-Electrodes, Easycap, Herrsching, Germany) from 27 electrode sites (Fp1, F3, F7, FC1, C3, T7, CP1, P3, P7, O1, PO7, Fz, FCz, Cz, CPz, Pz, Fp2, F4, F8, FC2, C4, T8, CP2, P4, P8, O2, PO8) and the right mastoid. The left mastoid was used as reference electrode. The ground electrode was placed on the right cheek. Impedances were kept below 5 kΩ. All electrodes were off-line re-referenced to the average of all electrodes. Vertical and horizontal EOG were recorded from above and below the left eye and from the outer canthi of both eyes. EEG and EOG were recorded with a band-pass of 0.01–100 Hz and amplified by a BrainAmp DC amplifier (Brain Products, Gilching, Germany). The amplified signals were digitized at a sampling rate of 500 Hz, and off-line filtered with a 40 Hz low-pass filter.

## ERP data analysis

ERP data were analyzed using custom routines in MatLab 7.0.4 (The Mathworks, Natic, MA, USA), as well as EEGLAB 5.03 (Delorme & Makeig, 2004), an open source toolbox for EEG data analysis (EEGLAB toolbox for single-trial EEG data analysis, Swartz Center for Computational Neurosciences, La Jolla, CA; <http://www.sccn.ucsd.edu/eeglab>). Segments of 200 ms before and 800 ms after each stimulus onset were extracted from the continuous EEG. The baseline window ran from -100 ms to 0 ms relative to stimulus onset. Epochs with incorrect responses were rejected (LA: 5% and HA: 7%, across body stimuli and visual fields of presentation). In addition, epochs contaminated with large artifacts were identified using two methods from the EEGLAB toolbox (Delorme, Sejnowski, & Makeig, 2007): (1) An epoch was excluded whenever the voltage on an EEG channel exceeded 100  $\mu$ V to remove epochs with large EEG peaks; (2) An epoch was excluded whenever the joint probability of a trial exceeded five standard deviations to remove epochs with improbable data (mean excluded epochs HA: 12.71%; mean excluded epochs LA: 11.53%). Remaining blinks and EOG horizontal artifacts were corrected using a multiple adaptive regression method (Automatic Artifact Removal Toolbox Version 1.3; [http://www.germangh.com/eeglab\\_plugin\\_aar/index.html](http://www.germangh.com/eeglab_plugin_aar/index.html)), based on the Least Mean Squares algorithm (Gratton, Coles, & Donchin, 1983). Finally, epochs were discarded from the analysis when saccadic movements ( $>30 \mu$ V on horizontal EOG channels) were registered in a time window of 500 ms following stimulus onset (HA: 0.98%; LA: 2.44%). The remaining epochs (HA mean number of epochs per condition: 76; LA mean number of epochs per condition: 79) were averaged separately for each participant and each Body stimulus type. The N190 amplitude was quantified as the mean amplitude in a time window of 170–220 ms post stimulus presentation (Fig. 2). Scalp topographies for the N190 component were calculated as mean amplitude in a time window of 170–220 ms post stimulus presentation (Fig. 3A and B).

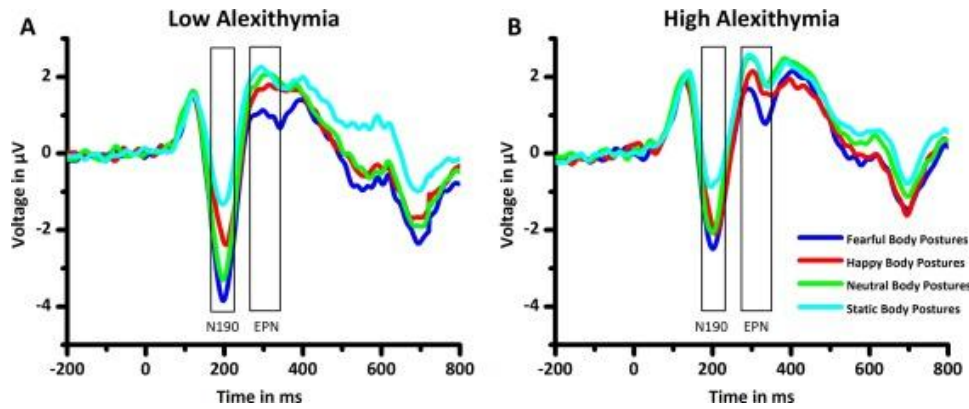


Fig. 2. Grand-average ERPs averaged across electrodes P7 and P8 and across visual fields (LVF and RVF), elicited by fearful, happy, neutral and static body postures in LA participants (A) and HA participants (B).

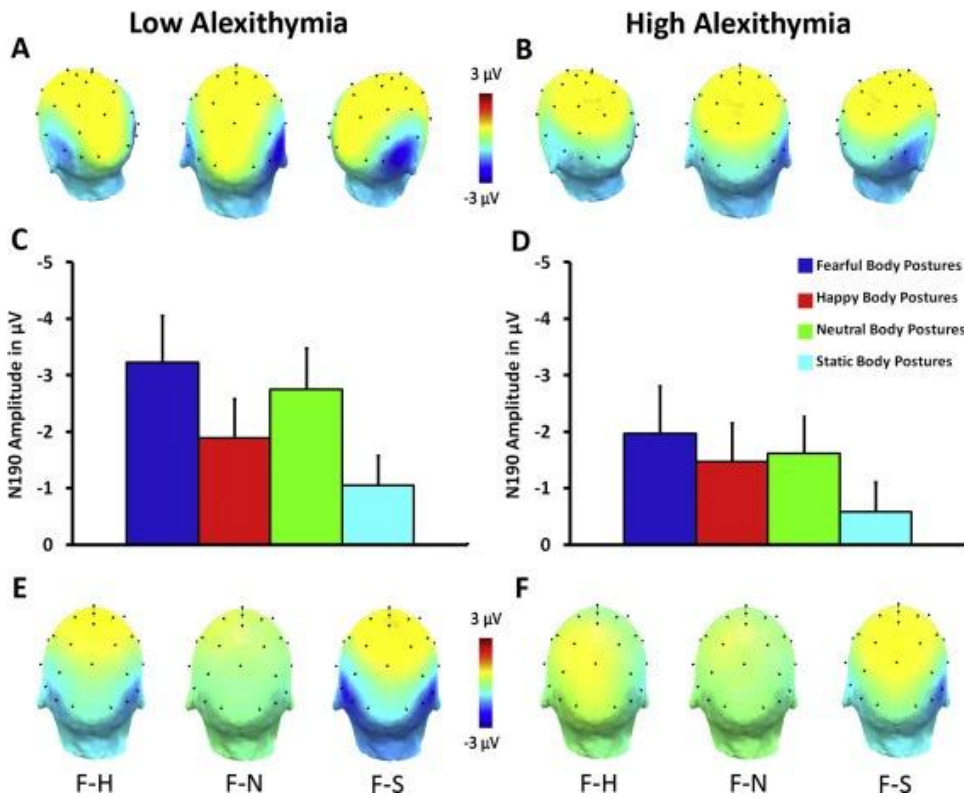


Fig. 3. N190 component. (A and B) 3D Scalp topographies (left back view, central back view, right back view) of the mean N190 amplitude averaged across body stimuli (fearful, happy, neutral and static body postures) and visual fields (LVF and RVF) in a time window of 170–220 ms in LA participants (A) and HA participants (B). (C and D) Mean N190 amplitude elicited by fearful, happy, neutral and static body postures in LA participants (C) and HA participants (D). Error bars represent standard error of the mean

(S.E.M.). (E and F) 3D Scalp topographies of the difference in mean N190 amplitude between fearful and other body stimuli (F–H: fearful–happy; F–N: fearful–neutral; F–S: fearful–static) in a time window of 170–220 ms in LA participants (E) and in HA participants (F).

As shown in the scalp topographies (Fig. 3A and B), the mean N190 amplitude averaged across body stimuli (fearful, happy, neutral and static body postures) and visual fields (LVF and RVF) reached a maximum negative deflection on electrodes P7 and P8. Therefore, electrodes P7 and P8 were chosen as a region of interest (ROI) in the N190 analyses, in line with previous studies (Stekelenburg and de Gelder, 2004 and Thierry et al., 2006). Grand average waveforms for LA and HA participants are shown in Fig. 2.

In addition, the EPN was calculated as the mean amplitude in a time window of 290–350 ms post-stimulus presentation (Fig. 2) at the same electrode locations as the N190 (i.e., electrodes P7 and P8). Both the N190 and the EPN mean amplitudes were analyzed with a mixed-model ANOVA with Group (HA and LA participants) as a between-participants factor and Electrode (P8 and P7), Visual field (LVF and RVF) and Body stimulus (fearful, happy, neutral and static) as within-participants factors. To compensate for violations of sphericity, Greenhouse–Geisser corrections were applied whenever appropriate (Greenhouse & Geisser, 1959), and corrected *p* values (but uncorrected degrees of freedom) are reported. Post-hoc comparisons were carried out using the Newman–Keuls test.

## 5.2.2. Results

### ERP results

**N190:** The ANOVA with Group (LA and HA participants) as a between-participants factor and Electrode (P8 and P7), Visual field (LVF and RVF), and Body stimulus (fearful, happy, neutral and static) as within-participants factors showed no

significant main effect of Group ( $F(1,30) = 0.86$ ;  $p = .36$ ), demonstrating that the N190 amplitude was comparable between LA and HA participants. A significant main effect of Electrode ( $F(1,30) = 5.74$ ;  $p = .02$ ) was found, revealing a larger N190 amplitude over electrode P8 in the right hemisphere ( $-2.29\mu\text{v}$ ) than over electrode P7 in the left hemisphere ( $-1.35\mu\text{v}$ ). Moreover, the ANOVA showed a main effect of Body stimulus ( $F(3,90) = 46.14$ ;  $p < .0001$ ). Post hoc analyses revealed that static body postures elicited a significantly smaller N190 amplitude than implied motion postures ( $-0.82\mu\text{v}$ ; all  $p$ s  $< .0002$ ). Notably, fearful body postures elicited the largest N190 amplitude compared to all the other body postures ( $-2.60\mu\text{v}$ ; all  $p$ s  $< .02$ ). In addition, neutral body postures ( $-2.18\mu\text{v}$ ) elicited a significantly larger N190 amplitude than happy postures ( $-1.68\mu\text{v}$ ;  $p = .002$ ). More importantly, a significant interaction between Body stimulus and Group was found ( $F(3,90) = 3.70$ ;  $p < .02$ ). Post-hoc analyses showed that, in the LA group, static body postures elicited a significantly smaller N190 amplitude ( $-1.05\mu\text{v}$ ) than neutral body postures ( $-2.75\mu\text{v}$ ;  $p = .0001$ ), happy body postures ( $-1.89\mu\text{v}$ ;  $p = .002$ ) and fearful body postures ( $-3.22\mu\text{v}$ ;  $p = .0001$ ). In addition, neutral body postures elicited a larger N190 than happy body postures ( $p = .001$ ). Importantly, a significantly larger N190 amplitude was found in response to fearful body postures ( $-3.22\mu\text{v}$ ) compared to both happy ( $p = .0001$ ) and neutral body postures ( $p = .04$ ; Figure 3C and 3E). Conversely, in the HA group, the N190 amplitude was only modulated by the presence of implied motion. Indeed, static body postures ( $-0.58\mu\text{v}$ ) elicited a significantly smaller N190 amplitude than implied motion postures (fearful =  $1.97\mu\text{v}$ ; happy =  $1.47\mu\text{v}$ ; neutral =  $1.62\mu\text{v}$ ; all  $p$ s  $< .0006$ ).

In contrast, there were no significant differences in N190 amplitude between positive and negative emotional body postures (fear vs happy:  $p = .13$ ) or between emotional and neutral body postures (fear vs neutral:  $p = .27$ ; happy vs neutral:  $p = .51$ ).



(Figure 3D and 3F). Interestingly, no other factors interacted with the factor Group (all  $p$ s > .24).

**EPN:** The ANOVA with Group (LA and HA participants) as a between-participants factor and Electrode (P8 and P7), Visual field (LVF and RVF) and Body stimulus (fearful, happy, neutral and static) as within-participants factors showed no main effect of Group, ( $F(1,30) = 0.06$ ;  $p = .81$ ), suggesting similar EPN amplitudes between LA and HA participants (Figure 4). There was a significant main effect of Body stimulus ( $F(3,90) = 13.09$ ;  $p < .0001$ ). Post-hoc comparisons showed a more negative amplitude in response to fearful body postures ( $1.07\mu\text{v}$ ), compared to all other postures (happy =  $1.76\mu\text{v}$ ; neutral =  $2.07\mu\text{v}$ ; static =  $2.08\mu\text{v}$ ; all  $p$ s < .0005). Notably, at variance with the results of the N190, no significant interaction between Body stimulus and Group was found ( $F(3,90) = 0.08$ ;  $p = .97$ ) (Figure 4). No other interactions with the factor Group were significant (Visual Field x Group:  $p = .06$ ; all the remaining interactions:  $p > .27$ ).

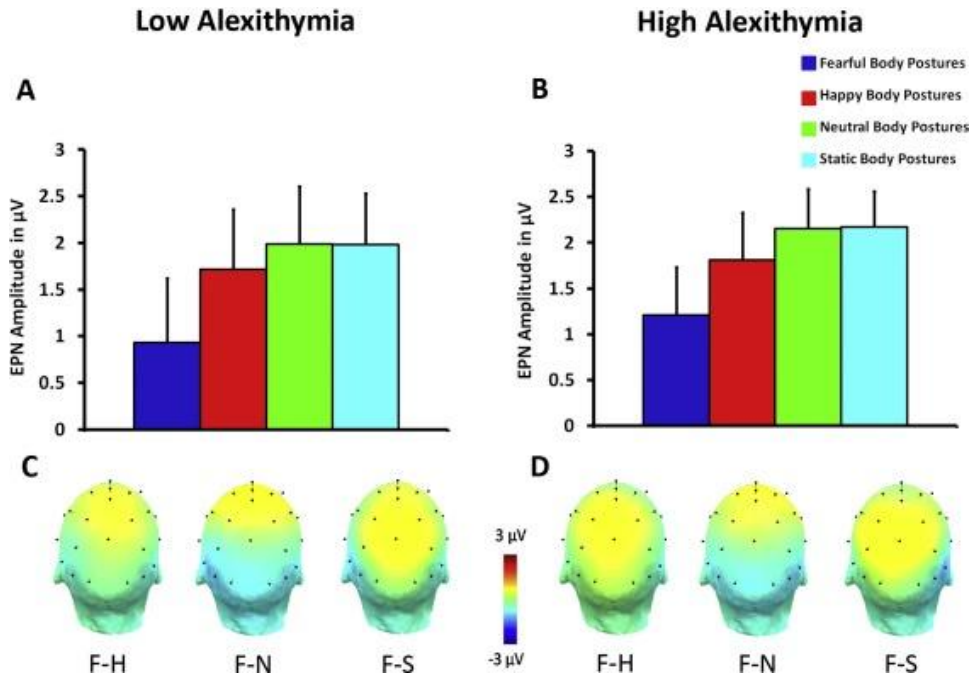


Fig. 4.

EPN component. (A and B) Mean EPN amplitude elicited by fearful, happy, neutral and static body postures in LA participants (A) and HA participants (B). Error bars represent standard error of the mean (S.E.M.). (C and D) 3D Scalp topographies of the difference in mean EPN amplitude between fearful and other body stimuli (F–H: fearful–happy; F–N: fearful–neutral; F–S: fearful–static) in a time window of 290–350 ms in LA participants (C) and in HA participants (D).

### Behavioral results:

Reaction times and accuracy scores were analyzed by means of two separate ANOVAs with Group (LA and HA participants) as a between-participants factor, and Visual field (LVF and RVF) and Body stimulus (fearful, happy, neutral and static) as within-participants factors. The ANOVA on reaction times showed no significant difference between the two groups ( $F(1,30) = 0.06$ ;  $p = .81$ ). Only a significant main effect of Body stimulus ( $F(3,90) = 73.18$ ;  $p < .0001$ ) was found. Post-hoc comparisons showed faster reaction times to static body postures (634ms) than to the other body

postures (fearful = 753 ms; happy = 748 ms; neutral = 769 ms; all  $ps < .0002$ ). No other comparisons were significant (all  $ps > .11$ ). Similarly, the ANOVA on accuracy scores showed no significant difference between groups ( $F(1,30) = 2.47$ ;  $p = .13$ ). A significant main effect of Body stimulus 13 ( $F(3,90) = 12.02$ ;  $p < .0001$ ) was revealed. Post-hoc analyses showed that participants were more accurate in responding to static body postures (99%), compared to all the other postures (fearful = 92%; happy = 94%; neutral = 92%; all  $ps < .0003$ ). No interactions with the factor Group were significant (all  $ps > .18$ ).

### **5.2.3. Discussion**

Human body postures represent a highly relevant vehicle through which both actions and emotions are expressed. Here we tested the hypothesis that individuals with difficulties in emotional processing (i.e., HA participants) may show an altered emotion-related modulation of the visual processing of body postures. Consistent with results from participants in the normal range of alexithymia (Borhani et al., 2015), individuals with low alexithymia scores (LA participants) showed N190 modulation both for implied body motion (i.e., body postures implying motion elicited greater N190 amplitudes than static postures) and for the emotional body expressions (i.e., fearful body postures elicited the largest N190 amplitude).

These findings reveal fine perceptual discrimination of both the presence of motion and the emotional content conveyed by body postures at an early stage of visual processing. In contrast, HA participants did not show emotion-related modulations of the N190 component. Indeed, the N190 amplitude was significantly enhanced by implied motion, but no emotion-related modulation was observed. Most importantly, the

fearful body postures failed to elicit the largest N190 amplitude. These results suggest that, at variance with LA participants and with participants in the normal range of alexithymia (Borhani et al., 2015), HA participants did not show an early detailed perceptual analysis of the emotional content of body postures. In contrast, early discrimination of motion-related information was observed in both LA and HA groups.

At the later stage of visual processing represented by the EPN component, no significant differences were found between HA and LA participants. A more pronounced EPN component was observed in response to fearful body postures in both groups, suggesting a similar attentional capture driven by salient cues in both HA and LA participants. The lack of any difference in the N190 amplitude elicited by emotional (fearful and happy) and nonemotional (neutral) body postures in HA participants suggests the presence of an altered early perceptual process in alexithymia. This result is in line with previous studies showing impaired early processing of emotional stimuli in alexithymic individuals. Indeed, HA participants showed a reduction in the amplitude of the early visual P1 component in response to emotional pictures (Pollatos and Gramann, 2011). In addition, altered modulations of the P1 and the N1 component were found in emotional oddball tasks (Campanella et al., 2012; Delle-Vigne et al., 2014). Notably, in the present study, HA participants also failed to show the enhanced visual encoding of fearful body postures that was evident in LA participants and individuals in the normal range of alexithymia (Borhani et al., 2015). In keeping, individuals with high alexithymia show altered early responses to other types of negative stimuli, such as early auditory-related potentials in response to potentially dangerous acoustic stimuli (Schäfer et al., 2007), reduced electrodermal responses to negative pictures (Pollatos et al., 2008) and impaired internal somatic simulation of fearful faces (Scarpazza et al., 2014). Similarly, HA participants exhibit hypo-responsiveness to negative emotional

stimuli in several cerebral areas, including the fusiform gyrus (Deng et al., 2013; Eichmann et al., 2008; Karlsson et al., 2008), the insula, the superior temporal gyrus and the middle occipital and parahippocampal gyrus (Reker et al., 2010) and in brain areas typically involved in emotional processing, such as the amygdala (Jongen et al., 2014; Kugel et al., 2008; Pouga et al., 2010; see van der Velde et al., 2013 for a recent meta-analysis). The reported hypo-responsiveness of the amygdala to negative stimuli in HA individuals might be responsible for the lack of N190 modulation by fearful body postures. Fearful bodies have been shown to elicit increased activation in the temporo-occipital areas from which the N190 originates (Grèzes et al., 2007; Hadjikhani and de Gelder, 2003; Peelen et al., 2007; Pichon et al., 2008; van de Riet et al., 2009).

In addition, fearful bodies enhance activation in the amygdala (de Gelder et al., 2004; Hadjikhani and de Gelder, 2003; van de Riet et al., 2009), the key subcortical structure for signaling fear and potential threat (Adolphs, 2013; LeDoux, 2014). Therefore, the typical enhancement of the N190 in response to fearful body postures, observed in LA individuals and in those with alexithymia scores in the normal range, might reflect a fast, distant functional influence of the amygdala on interconnected visual cortices (Borgomaneri et al., 2014; Vuilleumier et al., 2004; Wendt et al., 2011). Such a mechanism could be altered in individuals with high alexithymia, in which the reduced amygdala responses to negative stimuli (Jongen et al., 2014; Kugel et al., 2008; Pouga et al., 2010; see van der Velde et al., 2013 for a recent meta-analysis) might weaken feedback projections to the visual areas, preventing fear-related modulation of early perceptual processing.

Interestingly, the altered visual encoding of body postures observed in HA participants was specifically related to their emotional content, while the encoding of motion-related information was unaltered. Indeed, individuals with alexithymia showed

the typical enhancement of structural encoding for implied motion postures compared to static postures, suggesting an intact perceptual representation of bodies performing actions, possibly mediated by a wide fronto-parietal network of sensorimotor regions involved in action observation, planning and execution (Gallese and Sinigaglia, 2011; Gallese et al., 2004; Keysers and Gazzola, 2009; Nishitani et al., 2004).

At a later stage of visual processing, the same fear-related enhancement of the EPN component was found in both HA and LA participants, in line with previous results from participants in the normal range of alexithymia (Borhani et al., 2015). The observed enhancement of the EPN with fearful body postures is consistent with previous evidence showing larger EPN components in response to negatively valenced faces (Bayer and Schacht, 2014; Calvo and Beltrán, 2014; Frühholz et al., 2011; Rellecke et al., 2012; Schupp et al., 2004; Valdés-Conroy et al., 2014).

Similar to the findings of the present study, an emotional modulation of the EPN has also been observed in response to negatively valenced hand gestures (Flaisch et al., 2011, 2009).

These results suggest that, notwithstanding the altered early visual encoding of emotional body expressions, later visual processing of emotional body postures is intact in HA individuals. EPN has been interpreted as a marker of visual selective attention, which might reflect exogenous attentional capture driven by salient and motivationally relevant emotional stimuli (Olofsson et al., 2008; Schupp et al., 2006). The allocation of attentional resources at this later stage relies on a widespread network of brain regions, including extrastriate visual areas, superior and inferior parietal cortices and medial prefrontal regions (for a review, see Pourtois and Vuilleumier, 2006). Therefore, this later stage of visual processing seems at least partially independent of back projections

from the amygdala (Wendt et al., 2011). The involvement of a wide network of high order cortical areas, functionally intact in individuals with high alexithymia, seems to account for the preserved electrophysiological marker of selective attention towards salient fearful body postures in HA participants.

Interestingly, no differences between LA and HA participants were found at the behavioral level, neither in accuracy scores, nor in reaction times. This intact behavioral performance of individuals with high alexithymia is in keeping with the notion that HA individuals show emotional recognition impairments only when exposure times and response windows are short (Grynberg et al., 2012; Ihme et al., 2014).

Overall, the results of the present study suggest that alexithymia specifically affects the early visual encoding of emotional stimuli, without affecting the early visual encoding of motion information. Moreover, alexithymia does not affect fear-related modulation at a later stage of visual processing, which reflects attentional capture of salient stimuli.

## CHAPTER 6: General discussion

Individuals with high levels of alexithymia are known to have difficulties in understanding their own and others' emotions. Crucially, to date there are not so many studies investigating whether difficulties in emotion processing (e.g. alexithymia) may affect somatosensory and sensory (particularly visual) processing. In the present dissertation emotion related modulation of somatosensory and sensory systems was investigated in high and low alexithymic individuals. Human faces and body postures were used as representative stimuli for exhibiting emotion. Different paradigms and techniques falling within the field of cognitive neuroscience have been employed in order to test a possible difficulty in emotion modulation of somatosensory and sensory processing in high alexithymia. As an index of emotional modulation, the effect of emotional stimuli on physiological (e.g. autonomous system), perceptual (e.g. eye movements pattern), and electrophysiological (early structural and late attentional visual coding) levels were studied.

In experiments 1 and 2, regarding the critical role of somatosensory cortices activation in emotion processing (Adolphs., 2002; Damasio., 1996) and existence of somatosensory amplification theory in alexithymia (Wise and Mann., 1994; Lumley et al., 1996; Kano et al., 2007; Barsky et al., 1988), the somatosensory processing in high and low alexithymia was studied. Using a well-established QST paradigm, we compared a number of important somatosensory sub-modalities to find whether alterations in emotion processing seen in high levels of alexithymia could be grounded in somatosensory level. Results of experiments 1 and 2 showed that individuals with high scores on alexithymia are less sensitive to warm thermal



stimulation. One candidate area that is proposed for altered emotion processing in alexithymia is Insula (Kano et al., 2003; Reker et al., 2010; see Moriguchi and Komaki., 2013 for a review) that is involved in thermoception as well (Rolls., 2010). Our results might indicate that alexithymia is not only associated with affective and cognitive alterations but also is associated with low level somatosensory insensitivity, namely insensitivity to warm temperature. However another possible explanation for this could be an alteration at peripheral nervous system. The pathways for processing warm are different from those for cold. While warm is conducted through C-fibers, the cold temperature is transmitted via A-delta fibers (Schepers and Ringkamp., 2008). This association in conductance of warm and cold temperature has been shown also in central nervous system. It has been reported that while warm mostly activates S1, anterior cingulate and the opercular-insular areas (Casey et al., 1996; Iannetti et al., 2003), cold activates the secondary somatosensory cortex (Casey et al., 1996). Overall, although thermoception was the only somatosensory sub-modality that was found to be differently processed between high and low alexithymics, somatosensory processing seems to be varied in high levels of alexithymia. However, the sensitivity of the tests that were used in experiment 1 for all other somatosensory sub-modalities (e.g. pain, SDT, tactile, ...) might have been not enough to pick up the difference between two groups. Therefore, drawing a strong conclusion about somatosensory alteration role in emotion processing impairments in alexithymia requires further research.

In experiments 3, 4, and 5 emotion related modulation of visual processing of human faces as multi-dimensional stimuli that conveys emotion, was studied. In experiment 3, a category-specific emotional facial recognition task was used to investigate whether there is a difference between people with high and low level of alexithymia in recognizing facial expressions at a behavioral level. Experiments 4 and 5 investigated the effect of alexithymia on

early perceptual level of emotional face processing. Eye movement patterns as a measure of perceptual mechanism were used to compare high and low alexithymia during recognition of facial expressions. In addition, a visceral (cardiovascular) response, i.e. the instantaneous variations of heart rate (HR), was investigated, in response to fearful, disgusted, happy and neutral faces.

The behavioral results showed no difference between high and low alexithymics, although the preliminary cognitive prerequisite for emotional face perception differed between the two groups. Eye movement during the recognition of facial expressions showed a different pattern of fixation across different emotions. Results in low alexithymia group showed that concerning eye region, neutral expressions received a higher percentage of eye fixations than emotional expressions. After neutral faces, the fearful expression received the highest percentage of fixations in the eye region. Eye region is an important source of salient information in fearful face (Adolphs et al., 2005) and the work presented here is also in line with previous findings. Moreover, as in previous studies (Calder et al., 2000), disgusted expressions received a higher percentage of mouth fixations than all other emotions. This pattern was absent in high alexithymics. Decreased ability in recognition of emotional faces in alexithymia has been shown to be associated with diminished activity in amygdala (Suslow et al., 2016; Goerlich-Dobre et al., 2015; van der Velde et al., 2013). Also it has been indicated that amygdala is responsible in allocating attention to relevant piece of information and to properly use that information (Adolphs et al., 2005; Pessoa and Adolphs., 2010). Amygdala hypoactivity in high alexithymia might diminish the function of this key structure in directing the visual system to fixate, pay attention to, and make use of visual information (Adolphs et al., 2005). Consequently the absence of eye movements

pattern across emotions in alexithymia might be due to inability of amygdale in using properly that information.

Findings of experiment 5 revealed that, during visual processing of facial expressions, normally autonomic system responds differently to various emotions. For instance in low alexithimycs, bradycardia was found in response to negative facial expressions (fear and disgust). Again this physiological modulation in response to facial expressions was absent in high alexithymics The results of experiment 5 are consistent with the hypoarousal model proposed in alexithymia that indicates a dampened physiological reactivity, particularly reduced visceral reactions to emotional stimuli ( Linden et al., 1996, Nemiah et al., 1997; Newton and Contrada, 1994 and Wehmer et al., 1995; Neumann et al. 2004). The notion that this hypoarousal and hyporeactivity was found for negative emotions (fear and disgust) indicates attenuated sympathetic activation for these emotions. Together with results of experiment 4, these findings support the amygdale hypoactivation hypothesis in response to emotional information conveyed by faces, especially those with negative valence, like fear (Kugel et al., 2008; Reker et al., 2010; van der Velde et al., 2013).

In experiments 6 and 7, the visual processing of emotional information was studied at the electrophysiological level. In these experiments we addressed the question whether emotions conveyed by body postures can also be differently processed in alexithymia. In experiment 6, the early structural processing of fearful, happy, neutral, and static body postures was studied in normal participants. Results showed a greatest modulation of N190, ERP component that indexes structural processing of body postures, in response to fearful body postures. This might reflect a rapid functional influence of the amygdala on interconnected visual cortices, useful for processing threat signals efficiently and implementing fast motor reactions (Vuilleumier

et al., 2004). Similarly, somatosensory and motor regions, crucial to the processing of threat related expressions might also participate in this top-down influence. (Adolphs et al., 2000; Pourtois et al., 2004; Banissy et al., 2010)

Moreover, at the later stage of visual processing the EPN component was enhanced for fearful stimuli. This emotional modulation reflects attentional capture driven by salient emotional stimuli, and might reflect the degree of attention needed to recognize relevant signals (Oloffson et al., 2008). Indeed, the EPN modulation in response to fear indicates that fear is a relevant stimulus that is selected for further processing (Sato et al., 2001; Schupp et al., 2004a; Fruhholz et al., 2011; Calvo and Beltran, 2014).

Then in experiment 7 it was found that the early structural coding stage in high alexithymics was not modulated in response to salient fear signals and this is in line with amygdala malfunction theory in alexithymia. Hypoactivation of amygdala fails to elicit increased activation in the temporo-occipital areas from which the N190 originates (Grèzes et al., 2007; Hadjikhani and de Gelder, 2003; Peelen et al., 2007; Pichon et al., 2008; van de Riet et al., 2009). Interestingly, the later attentional processing of fearful information remained intact in high alexithymics. This might indicate that the later attentional stage of visual processing of body postures is not entirely driven from amygdala back projections (Wendt et al., 2011).

Overall, the findings in individuals with high alexithymia indicate that difficulties in emotion-related domains might ground in low-level perceptual and physiological stages. In addition, the findings in high alexitymics showed dissociation between recognizing emotions at behavioral level, which was intact and the early perceptual and physiological modulations due to emotion, which was altered. This was revealed by lack

of: physiological changes, by the absence of a different eye movement pattern in response to facial emotions, and by the lack of early visual encoding of bodily emotions. The dissociation between emotion recognition level and early perceptual levels is important because it might suggest that a wider network of cortical areas, are functionally intact in high alexithymics and they contribute to recognition of emotional stimuli.

In general, experiments in this dissertation showed that difficulties in emotional processing (e.g. alexithymia) affect sensory and somatosensory processing. The results of these experiments provided convergent evidence that the pattern of influences induced by emotions in sensory and somatosensory systems is different in high and low alexithymia. Consistent results, mainly in relation to fear, were found across different experiments. High alexithymic individuals showed difficulties in processing the signals of fear exhibited by faces and body postures either at perceptual level or at physiological level. This supports the hypothesis of a diminished response to fearful stimuli in high alexithymia compared to low alexithymia. This might be explained by the amygdala hypoactivation which has been revealed in high alexithymia by previous studies, especially in response to negative emotional stimuli (Heinzel et al., 2010; Kano et al., 2003; Kugel et al., 2008; van der Velde et al., 2013). Amygdala has been proposed as a candidate area that underlies emotional processing problems in alexithymia because of its role in detecting the emotional relevance of the stimuli (Kano and Fukudo., 2013; Moriguchi and Komaki., 2013). Notably, amygdala is a key subcortical structure that plays an important role in signaling fear as a relevant stimulus for survival. Therefore decreased activation of this structure could affect the processing of fear.

Finally, it is important to note that studying alexithymia as a personality construct with difficulties in emotion processing and investigating the possible mechanisms responsible for these difficulties is relevant for several reasons. For instance alexithymia has been reported to coexist in so many psychiatric disorders like depression, anxiety, eating disorders, and psychosomatic disorders. Crucially, alexithymia has been shown to influence the onset and progression of psychosomatic disorders (Moriguchi and Komaki., 2013). Moreover, individuals with high alexithymia deal with serious problems in everyday social relations and communication skills that affect their social and personal life. Due to all above reasons, alexithymia was studied in the present dissertation to shed light on the possible underpinning mechanisms of this personality construct.

## References

- Adolphs, R. (2002). Neural systems for recognizing emotion. *Current opinion in neurobiology*, 12(2), 169-177.
- Adolphs, R. (2002). Recognizing emotion from facial expressions: psychological and neurological mechanisms. *Behavioral and Cognitive Neuroscience Reviews*, 1, 21–62.
- Adolphs, R. (2013). The biology of fear. *Current Biology*, 23(2), R79-R93.
- Adolphs, R., Damasio, H., Tranel, D., Cooper, G., Damasio, A.R. (2000). A role for somatosensory cortices in the visual recognition of emotion as revealed by three-dimensional lesion mapping. *The Journal of Neuroscience*, 20, 2683–90
- 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(7021), 68-72.
- Allen, L. B., Lu, Q., Tsao, J., Hayes, L. P., & Zeltzer, L. (2011). Depression partially mediates the relationship between alexithymia and somatization in a sample of healthy children. *Journal of health psychology*, 1359105311402407.
- Allison, T., Puce, A., McCarthy, G. (2000). Social perception from visual cues: role of the STS region. *Trends in Cognitive Sciences*, 4, 267–78
- Amorapanth, P., LeDoux, J. E., & Nader, K. (2000). Different lateral amygdala outputs mediate reactions and actions elicited by a fear-arousing stimulus. *Nature neuroscience*, 3(1), 74-79.
- Arturcedo, Kokoszka, A., Popiel, A., & NARKIEWICZ-JODKO, W. (2001). Alexithymia in schizophrenia: an exploratory study. *Psychological reports*, 89(1), 95-98.
- Avenanti, A., Annella, L., Candidi, M., Urgesi, C., & Aglioti, S. M. (2013a). Compensatory plasticity in the action observation network: virtual lesions of STS enhance anticipatory simulation of seen actions. *Cerebral Cortex*, 23(3), 570-580.

Avenanti, A., Candidi, M., & Urgesi, C. (2013b). Vicarious motor activation during action perception: beyond correlational evidence. *Frontiers in human neuroscience*, 7, 185.

Azevedo, T. M., Volchan, E., Imbiriba, L. A., Rodrigues, E. C., Oliveira, J. M., Oliveira, L. F., & Vargas, C. D. (2005). A freezing-like posture to pictures of mutilation. *Psychophysiology*, 42(3), 255-260.

Bagby, R. M., Parker, J. D., & Taylor, G. J. (1994). The twenty-item Toronto Alexithymia Scale—I. Item selection and cross-validation of the factor structure. *Journal of psychosomatic research*, 38(1), 23-32.

Banissy, M. J., Sauter, D. A., Ward, J., Warren, J. E., Walsh, V., & Scott, S. K. (2010). Suppressing sensorimotor activity modulates the discrimination of auditory emotions but not speaker identity. *The Journal of Neuroscience*, 30(41), 13552-13557.

Bargh, J. A., & Shalev, I. (2012). The substitutability of physical and social warmth in daily life. *Emotion*, 12(1), 154.

Barsky, A. J., Goodson, J. D., Lane, R. S., & Cleary, P. D. (1988). The amplification of somatic symptoms. *Psychosomatic Medicine*, 50(5), 510-519.

Bastiaansen, J. A., Thioux, M., & Keysers, C. (2009). Evidence for mirror systems in emotions. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 364(1528), 2391-2404.

Batty, M., & Taylor, M. J. (2003). Early processing of the six basic facial emotional expressions. *Cognitive Brain Research*, 17(3), 613-620.

Bayer, M., Schacht, A. (2014). Event-related brain responses to emotional words, pictures, and faces: a cross-domain comparison. *Front. Psychol.* 5, 1–10.



Beck, A. T., Ward, C., & Mendelson, M. (1961). Beck depression inventory (BDI). *Arch Gen Psychiatry*, 4(6), 561-571.

Bentin, S., Allison, T., Puce, A., Perez, E., & McCarthy, G. (1996). Electrophysiological studies of face perception in humans. *Journal of cognitive neuroscience*, 8(6), 551-565.

Bermond, B., Bierman, D. J., Cladder, M. A., Moormann, P. P., & Vorst, H. C. (2010). The cognitive and affective alexithymia dimensions in the regulation of sympathetic responses. *International Journal of Psychophysiology*, 75(3), 227-233.

Bermond, B., Bleys, J. W., & Stoffels, E. J. (2005). Left hemispheric preference and alexithymia: A neuropsychological investigation. *Cognition & Emotion*, 19(1), 151-160.

Bermond, B., Righart, R., Ridderinkhof, K. R., & Moormann, P. P. (2008). Alexithymia and the brain potential P300. *Netherlands Journal of Psychology*, 64(2), 65-77.

Bermond, B., Vorst, H. C., & Moormann, P. P. (2006). Cognitive neuropsychology of alexithymia: implications for personality typology. *Cognitive neuropsychiatry*, 11(3), 332-360.

Berthoz, S., Artiges, E., Van de Moortele, P. F., Poline, J. B., Rouquette, S., Consoli, S. M., & Martinot, J. L. (2002). Effect of impaired recognition and expression of emotions on frontocingulate cortices: an fMRI study of men with alexithymia. *American Journal of Psychiatry*.

Bessou, P., Burgess, P. R., Perl, E. R., & Taylor, C. B. (1971). Dynamic properties of mechanoreceptors with unmyelinated (C) fibers. *Journal of neurophysiology*.

Bird, G., Silani, G., Brindley, R., White, S., Frith, U., & Singer, T. (2010). Empathic brain responses in insula are modulated by levels of alexithymia but not autism. *Brain*, awq060.

Bonda, E., Petrides, M., Ostry, D., & Evans, A. (1996). Specific involvement of human parietal systems and the amygdala in the perception of biological motion. *The Journal of Neuroscience*, 16(11), 3737-3744.

Borgomaneri, S., Gazzola, V., & Avenanti, A. (2012). Motor mapping of implied actions during perception of emotional body language. *Brain stimulation*, 5(2), 70-76.

Borgomaneri, S., Gazzola, V., & Avenanti, A. (2013). Temporal dynamics of motor cortex excitability during perception of natural emotional scenes. *Social cognitive and affective neuroscience*, doi:10.1093/scan/nst139.

Borgomaneri, S., Gazzola, V., & Avenanti, A. (2014). Transcranial magnetic stimulation reveals two functionally distinct stages of motor cortex involvement during perception of emotional body language. *Brain Structure and Function*, doi:10.1007/s00429-014-0825-6.

Borgomaneri, S., Vitale, F., Gazzola, V., Avenanti, A., 2015. Seeing fearful body language rapidly freezes the observer's motor cortex. *Cortex* 65, 232–245.

Borhani, K., Borgomaneri, S., Làdavas, E., & Bertini, C. (2016). The effect of alexithymia on early visual processing of emotional body postures. *Biological psychology*.

Borhani, K., Làdavas, E., Maier, M. E., Avenanti, A., & Bertini, C. (2015). Emotional and movement-related body postures modulate visual processing. *Social cognitive and affective neuroscience*, 10(8), 1092-1101.

Borod, J. C. (Ed.). (2000). *The neuropsychology of emotion* (pp. 80-103). New York: Oxford University Press.

Bradley, M. M., & Lang, P. J. (1999). *Affective norms for English words (ANEW): Instruction manual and affective ratings* (pp. 1-45). Technical report C-1, the center for research in psychophysiology, University of Florida.

Bradley, M. M., & Lang, P. J. (2000). Measuring emotion: Behavior, feeling, and physiology. *Cognitive neuroscience of emotion*, 25, 49-59.

Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and motivation I: defensive and appetitive reactions in picture processing. *Emotion, 1*(3), 276.

Breiter, H. C., Etcoff, N. L., Whalen, P. J., Kennedy, W. A., Rauch, S. L., Buckner, R. L., ... & Rosen, B. R. (1996). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron, 17*(5), 875-887. Cabanac, M. (2002). What is emotion?. *Behavioural processes, 60*(2), 69-83.

Calder, A. J., Lawrence, A. D., & Young, A. W. (2001). Neuropsychology of fear and loathing. *Nature Reviews Neuroscience, 2*(5), 352-363.

Calder, A. J., Young, A. W., Keane, J., & Dean, M. (2000). Configural information in facial expression perception. *Journal of Experimental Psychology: Human perception and performance, 26*(2), 527.

Calvo, M. G., & Beltrán, D. (2014). Brain lateralization of holistic versus analytic processing of emotional facial expressions. *Neuroimage, 92*, 237-247.

Cannon, W. B. (1927). The James-Lange theory of emotions: A critical examination and an alternative theory. *The American journal of psychology, 39*(1/4), 106-124.

Carretié, L., Albert, J., López-Martín, S., Tapia, M., (2009). Negative brain: an integrative review on the neural processes activated by unpleasant stimuli. *Int. J. Psychophysiol. 71*, 57–63.

Casey, K. L., Minoshima, S., Morrow, T. J., & Koeppe, R. A. (1996). Comparison of human cerebral activation pattern during cutaneous warmth, heat pain, and deep cold pain. *Journal of Neurophysiology, 76*(1), 571-581.

Caspers, S., Zilles, K., Laird, A. R., & Eickhoff, S. B. (2010). ALE meta-analysis of action observation and imitation in the human brain. *Neuroimage, 50*(3), 1148-1167.

Cecere, R., Bertini, C., Maier, M. E., & Làdavas, E. (2014). Unseen Fearful Faces Influence Face Encoding: Evidence from ERPs in Hemianopic Patients. *Journal of Cognitive Neuroscience*. doi:10.1162/jocn\_a\_00671.

Chan, A. W., Peelen, M. V., & Downing, P. E. (2004). The effect of viewpoint on body representation in the extrastriate body area. *Neuroreport*, 15(15), 2407-2410.

Choi, D., Nishimura, T., Motoi, M., Egashira, Y., Matsumoto, R., & Watanuki, S. (2014). Effect of empathy trait on attention to various facial expressions: evidence from N170 and late positive potential (LPP). *Journal of physiological anthropology*, 33(1), 1.

Churyukanov, M., Plaghki, L., Legrain, V., & Mouraux, A. (2012). Thermal detection thresholds of Delta-and C-fibre afferents activated by brief CO2 laser pulses applied onto the human hairy skin. *PloS one*, 7(4), e35817.

Cook, R., Brewer, R., Shah, P., & Bird, G. (2013). Alexithymia, not autism, predicts poor recognition of emotional facial expressions. *Psychological Science*, 0956797612463582.

Craig, A. D., Reiman, E. M., Evans, A., & Bushnell, M. C. (1996). Functional imaging of an illusion of pain.

Critchley, H. D., Elliott, R., Mathias, C. J., & Dolan, R. J. (2000). Neural activity relating to generation and representation of galvanic skin conductance responses: a functional magnetic resonance imaging study. *The Journal of Neuroscience*, 20(8), 3033-3040.

Critchley, H. D., Wiens, S., Rotshtein, P., Öhman, A., & Dolan, R. J. (2004). Neural systems supporting interoceptive awareness. *Nature neuroscience*, 7(2), 189-195.

Dalton, K. M., Nacewicz, B. M., Johnstone, T., Schaefer, H. S., Gernsbacher, M. A., Goldsmith, H. H., ... & Davidson, R. J. (2005). Gaze fixation and the neural circuitry of face processing in autism. *Nature neuroscience*, 8(4), 519-526.

Damasio, A. R., Everitt, B. J., & Bishop, D. (1996). The somatic marker hypothesis and the possible functions of the prefrontal cortex [and discussion]. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 351(1346), 1413-1420.

Darwin, C. (1972). *The expression of emotion in man and animal*. Murray, London.

Davis, K. D., Kwan, C. L., Crawley, A. P., & Mikulis, D. J. (1998). Functional MRI study of thalamic and cortical activations evoked by cutaneous heat, cold, and tactile stimuli. *Journal of Neurophysiology*, 80(3), 1533-1546.

Davis, M., & Whalen, P. J. (2001). The amygdala: vigilance and emotion. *Molecular psychiatry*, 6(1), 13-34.

De Gelder, B. (2006). Towards the neurobiology of emotional body language. *Nature Reviews Neuroscience*, 7(3), 242-249.

De Gelder, B., Snyder, J., Greve, D., Gerard, G., & Hadjikhani, N. (2004). Fear fosters flight: a mechanism for fear contagion when perceiving emotion expressed by a whole body. *Proceedings of the National Academy of Sciences of the United States of America*, 101(47), 16701-16706.

De Gelder, B., Teunisse, J. P., & Benson, P. J. (1997). Categorical perception of facial expressions: Categories and their internal structure. *Cognition & Emotion*, 11(1), 1-23.

De Gelder, B., Van den Stock, J., Meeren, H. K., Sinke, C., Kret, M. E., & Tamietto, M. (2010). Standing up for the body. Recent progress in uncovering the networks involved in the perception of bodies and bodily expressions. *Neuroscience & Biobehavioral Reviews*, 34(4), 513-527.

De Zwaan, M., Biener, D., Bach, M., Wiesnagrotzki, S., & Stacher, G. (1996). Pain sensitivity, alexithymia, and depression in patients with eating disorders: are they related?. *Journal of psychosomatic research*, 41(1), 65-70.

Delorme, A., & Makeig, S. (2004). EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of neuroscience methods*, 134(1), 9-21.

Delorme, A., Sejnowski, T., & Makeig, S. (2007). Enhanced detection of artifacts in EEG data using higherorder statistics and independent component analysis. *Neuroimage*, 34(4), 1443-1449.

Deng, Y., Ma, X., & Tang, Q. (2013). Brain response during visual emotional processing: an fMRI study of alexithymia. *Psychiatry Research: Neuroimaging*, 213(3), 225-229. Domes, G., Heinrichs, M.,

Gläscher, J., Büchel, C., Braus, D. F., & Herpertz, S. C. (2007). Oxytocin attenuates amygdala responses to emotional faces regardless of valence. *Biological psychiatry*, 62(10), 1187-1190.

Donnellan, M. B., Lucas, R. E., & Cesario, J. (2015). On the association between loneliness and bathing habits: Nine replications of Bargh and Shalev (2012) Study 1. *Emotion*, 15(1), 109.

Downing, P. E., & Peelen, M. V. (2011). The role of occipitotemporal body-selective regions in person perception. *Cognitive neuroscience*, 2(3-4), 186-203.

Downing, P. E., Jiang, Y., Shuman, M., & Kanwisher, N. (2001). A cortical area selective for visual processing of the human body. *Science*, 293(5539), 2470-2473.

Dunn, B. D., Dalgleish, T., Ogilvie, A. D., & Lawrence, A. D. (2007). Heartbeat perception in depression. *Behaviour research and therapy*, 45(8), 1921-1930.

Eichmann, M., Kugel, H., Suslow, T., 2008. Difficulty identifying feelings and automatic activation in the fusiform gyrus in response to facial emotion. *Percept. Mot. Skills* 107, 915–22.

Ekman, P. (1984). Expression and the nature of emotion. *Approaches to emotion*, 3, 19-344.

Ekman, P., Sorenson, E. R., & Friesen, W. V. (1969). Pan-cultural elements in facial displays of emotion. *Science*, 164(3875), 86-88.

FeldmanHall, O., Dalgleish, T., & Mobbs, D. (2013). Alexithymia decreases altruism in real social decisions. *Cortex*, 49(3), 899-904.

Flaisch, T., Häcker, F., Renner, B., & Schupp, H. T. (2011). Emotion and the processing of symbolic gestures: an event-related brain potential study. *Social cognitive and affective neuroscience*, 6(1), 109-118.

Flaisch, T., Schupp, H. T., Renner, B., & Junghöfer, M. (2009). Neural systems of visual attention responding to emotional gestures. *Neuroimage*, 45(4), 1339-1346.

Fox, E., Lester, V., Russo, R., Bowles, R.J., Pichler, A., Dutton, K., (2000). Facial expressions of emotion: are angry faces detected more efficiently? *Cogn. Emot.* 14, 61–92.

Fox, N. A., & Davidson, R. J. (1984). Hemispheric substrates of affect: A developmental model. *The psychobiology of affective development*, 353-381.

Freyd, J. J. (1983). The mental representation of movement when static stimuli are viewed. *Perception & Psychophysics*, 33(6), 575-581.

Friedlander, L., Lumley, M. A., Farchione, T., & Doyal, G. (1997). Testing the alexithymia hypothesis: physiological and subjective responses during relaxation and stress. *The Journal of nervous and mental disease*, 185(4), 233-239.

Frühholz, S., Jellinghaus, A., & Herrmann, M. (2011). Time course of implicit processing and explicit processing of emotional faces and emotional words. *Biological psychology*, 87(2), 265-274.

Fusar-Poli, P., Placentino, A., Carletti, F., Landi, P., Allen, P., Surguladze, S., Benedetti, F., Abbamonte, M., Gasparotti, R., Barale, F., Perez, J., McGuire, P., Politi, P., (2009). Functional atlas of emotional faces processing: a voxel-based meta-analysis of 105 functional magnetic resonance imaging studies. *J. Psychiatry Neurosci.* 34, 418–32.

Gainotti, G., Caltagirone, C., & Zoccolotti, P. (1993). Left/right and cortical/subcortical dichotomies in the neuropsychological study of human emotions. *Cognition & Emotion*, 7(1), 71-93.

Gallese, V., & Sinigaglia, C. (2011). What is so special about embodied simulation?. *Trends in cognitive sciences*, 15(11), 512-519.

Gallese, V., Keysers, C., & Rizzolatti, G. (2004). A unifying view of the basis of social cognition. *Trends in cognitive sciences*, 8(9), 396-403.

Gentile, C. G., Jarrell, T. W., Teich, A., McCabe, P. M., & Schneiderman, N. (1986). The role of amygdaloid central nucleus in the retention of differential Pavlovian conditioning of bradycardia in rabbits. *Behavioural brain research*, 20(3), 263-273.

Goeleven, E., De Raedt, R., Leyman, L., & Verschuere, B. (2008). The Karolinska directed emotional faces: a validation study. *Cognition and Emotion*, 22(6), 1094-1118.

Goerlich-Dobre, K. S., Bruce, L., Martens, S., Aleman, A., & Hooker, C. I. (2014). Distinct associations of insula and cingulate volume with the cognitive and affective dimensions of alexithymia. *Neuropsychologia*, 53, 284-292.

Goerlich-Dobre, K. S., Votinov, M., Habel, U., Pripfl, J., & Lamm, C. (2015). Neuroanatomical profiles of alexithymia dimensions and subtypes. *Human brain mapping*, 36(10), 3805-3818.

Goerlich-Dobre, K. S., Witteman, J., Schiller, N. O., van Heuven, V. J., Aleman, A., & Martens, S. (2013). Blunted feelings: Alexithymia is associated with a diminished neural response to speech prosody. *Social cognitive and affective neuroscience*, nst075.



Grafman, J., Vance, S. C., Weingartner, H., Salazar, A. M., & Amin, D. (1986). The effects of lateralized frontal lesions on mood regulation. *Brain*, *109*(6), 1127-1148.

Grandi, S., Sirri, L., Wise, T. N., Tossani, E., & Fava, G. A. (2011). Kellner's Emotional Inhibition Scale: a clinimetric approach to alexithymia research. *Psychotherapy and psychosomatics*, *80*(6), 335-344.

Gratton, G., Coles, M. G., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and clinical neurophysiology*, *55*(4), 468-484.

Green, D. M., & Swets, J. A. Signal detection theory and psychophysics. 1966. *New York*.

Greenhouse, S. W., & Geisser, S. (1959). On methods in the analysis of profile data. *Psychometrika*, *24*(2), 95-112.

Gregory, R. J., Manning, J., & Berry, S. L. (2000). Pain location and psychological characteristics of patients with chronic pain. *Psychosomatics*, *41*(3), 216-220.

Grèzes, J., Pichon, S., & De Gelder, B. (2007). Perceiving fear in dynamic body expressions. *Neuroimage*, *35*(2), 959-967.

Grèzes, J., Tucker, M., Armony, J., Ellis, R., & Passingham, R. E. (2003). Objects automatically potentiate action: an fMRI study of implicit processing. *European Journal of Neuroscience*, *17*(12), 2735-2740.

Grynberg, D., Chang, B., Corneille, O., Maurage, P., Vermeulen, N., Berthoz, S., & Luminet, O. (2012). Alexithymia and the processing of emotional facial expressions (EFEs): systematic review, unanswered questions and further perspectives. *PloS one*, *7*(8), e42429.

Grynberg, D., Luminet, O., Corneille, O., Grèzes, J., & Berthoz, S. (2010). Alexithymia in the interpersonal domain: A general deficit of empathy?. *Personality and individual differences, 49*(8), 845-850.

Guttman, H., & Laporte, L. (2002). Alexithymia, empathy, and psychological symptoms in a family context. *Comprehensive psychiatry, 43*(6), 448-455.

Hadjikhani, N., & de Gelder, B. (2003). Seeing fearful body expressions activates the fusiform cortex and amygdala. *Current Biology, 13*(24), 2201-2205.

Hagenaars, M. A., Stins, J. F., & Roelofs, K. (2012). Aversive life events enhance human freezing responses. *Journal of Experimental Psychology: General, 141*(1), 98.

Halgren, E., Raji, T., Marinkovic, K., Jousmäki, V., & Hari, R. (2000). Cognitive response profile of the human fusiform face area as determined by MEG. *Cerebral cortex, 10*(1), 69-81.

Hansen, C.H., Hansen, R.D., (1988). Finding the face in the crowd: an anger superiority effect. *J. Pers. Soc. Psychol. 54*, 917–24.

Hansson, P., Backonja, M., & Bouhassira, D. (2007). Usefulness and limitations of quantitative sensory testing: clinical and research application in neuropathic pain states. *Pain, 129*(3), 256-259.

Hantas, M. N., Katkin, E. S., & Reed, S. D. (1984). Cerebral lateralization and heartbeat discrimination. *Psychophysiology, 21*(3), 274-278.

Haviland, M. G., Sonne, J. L., & Kowert, P. A. (2004). Alexithymia and psychopathy: Comparison and application of California Q-set prototypes. *Journal of personality assessment, 82*(3), 306-316.

Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2000). The distributed human neural system for face perception. *Trends in cognitive sciences, 4*(6), 223-233.

Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2002). Human neural systems for face recognition and social communication. *Biological psychiatry*, 51(1), 59-67.

Heilman, K. M. (2000). Emotional experience: A neurological model. *Cognitive neuroscience of emotion*, 328-344.

Heilman, K. M., Schwartz, H. D., & Watson, R. T. (1978). Hypoarousal in patients with the neglect syndrome and emotional indifference. *Neurology*, 28(3), 229-229.

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological psychiatry*, 54(12), 1389-1398.

Heinzel, A., Schäfer, R., Müller, H. W., Schieffer, A., Inghag, A., Northoff, G., ... & Hautzel, H. (2010). Differential modulation of valence and arousal in high-alexithymic and low-alexithymic individuals. *Neuroreport*, 21(15), 998-1002.

Henderson, J. M., Williams, C. C., & Falk, R. J. (2005). Eye movements are functional during face learning. *Memory & cognition*, 33(1), 98-106.

Herbert, B. M., Herbert, C., & Pollatos, O. (2011). On the relationship between interoceptive awareness and alexithymia: is interoceptive awareness related to emotional awareness?. *Journal of Personality*, 79(5), 1149-1175.

Herbert, B. M., Pollatos, O., & Schandry, R. (2007). Interoceptive sensitivity and emotion processing: an EEG study. *International Journal of Psychophysiology*, 65(3), 214-227.

Hermans, E. J., Henckens, M. J., Roelofs, K., & Fernández, G. (2013). Fear bradycardia and activation of the human periaqueductal grey. *Neuroimage*, 66, 278-287.

Hernandez, N., Metzger, A., Magné, R., Bonnet-Brilhault, F., Roux, S., Barthelemy, C., & Martineau, J. (2009). Exploration of core features of a human face by healthy and autistic adults analyzed by visual scanning. *Neuropsychologia*, 47(4), 1004-1012.

Hess, U., & Fischer, A. (2013). Emotional mimicry as social regulation. *Personality and Social Psychology Review, 17*(2), 142-157.

Hintikka, J., Honkalampi, K., Lehtonen, J., & Viinamäki, H. (2001). Are alexithymia and depression distinct or overlapping constructs?: a study in a general population. *Comprehensive Psychiatry, 42*(3), 234-239.

Hitchcock, J., & Davis, M. (1986). Lesions of the amygdala, but not of the cerebellum or red nucleus, block conditioned fear as measured with the potentiated startle paradigm. *Behavioral neuroscience, 100*(1), 11.

Hohmann, G. W. (1966). Some effects of spinal cord lesions on experienced emotional feelings. *Psychophysiology, 3*(2), 143-156.

Honkalampi, K., Hintikka, J., Tanskanen, A., Lehtonen, J., & Viinamäki, H. (2000). Depression is strongly associated with alexithymia in the general population. *Journal of psychosomatic research, 48*(1), 99-104.

Horley, K., Williams, L. M., Gonsalvez, C., & Gordon, E. (2003). Social phobics do not see eye to eye:: A visual scanpath study of emotional expression processing. *Journal of anxiety disorders, 17*(1), 33-44.

Iannetti, G. D., & Mouraux, A. (2010). From the neuromatrix to the pain matrix (and back). *Experimental brain research, 205*(1), 1-12.

Iannetti, G. D., Truini, A., Romaniello, A., Galeotti, F., Rizzo, C., Manfredi, M., & Cruccu, G. (2003). Evidence of a specific spinal pathway for the sense of warmth in humans. *Journal of neurophysiology, 89*(1), 562-570.

Ihme, K., Dannlowski, U., Lichev, V., Stuhrmann, A., Grotegerd, D., Rosenberg, N., ... & Suslow, T. (2013). Alexithymia is related to differences in gray matter volume: a voxel-based morphometry study. *Brain research, 1491*, 60-67.

Ihme, K., Sacher, J., Lichev, V., Rosenberg, N., Kugel, H., Rufer, M., ... & Villringer, A. (2014). Alexithymia and the labeling of facial emotions: response slowing and increased motor and somatosensory processing. *BMC neuroscience*, 15(1), 1.

Ihme, K., Sacher, J., Lichev, V., Rosenberg, N., Kugel, H., Rufer, M., ... & Villringer, A. (2014). Alexithymia and the labeling of facial emotions: response slowing and increased motor and somatosensory processing. *BMC neuroscience*, 15(1), 40.

Ihme, K., Sacher, J., Lichev, V., Rosenberg, N., Kugel, H., Rufer, M., Grabe, H.J., Pampel, A., Lepsien, J., Kersting, A., Villringer, A., Lane, R., Suslow, T., (2014). Alexithymic features and the labeling of brief emotional facial expressions – an fMRI study. *Neuropsychologia* 64, 289– 299.

Inagaki, T. K., & Eisenberger, N. I. (2013). Shared neural mechanisms underlying social warmth and physical warmth. *Psychological science*, 0956797613492773.

Izard, C. E. (1992). Basic emotions, relations among emotions, and emotion-cognition relations.

James, W. (1950). *The Principles of Psychology*. 2 vols. New York: Henry Holt and Co. Reprint.

Jessimer, M., & Markham, R. (1997). Alexithymia: a right hemisphere dysfunction specific to recognition of certain facial expressions?. *Brain and cognition*, 34(2), 246-258.

Jongen, S., Axmacher, N., Kremers, N. A., Hoffmann, H., Limbrecht-Ecklundt, K., Traue, H. C., & Kessler, H. (2014). An investigation of facial emotion recognition impairments in alexithymia and its neural correlates. *Behavioural brain research*, 271, 129-139.

Kano, M., & Fukudo, S. (2013). The alexithymic brain: the neural pathways linking alexithymia to physical disorders. *BioPsychoSocial medicine*, 7(1), 1.

Kano, M., Fukudo, S., Gyoba, J., Kamachi, M., Tagawa, M., Mochizuki, H., ... & Yanai, K. (2003). Specific brain processing of facial expressions in people with alexithymia: an H215O-PET study. *Brain*, 126(6), 1474-1484.

Kano, M., Hamaguchi, T., Itoh, M., Yanai, K., & Fukudo, S. (2007). Correlation between alexithymia and hypersensitivity to visceral stimulation in human. *Pain*, 132(3), 252-263.

Karlsson, H., Näätänen, P., & Stenman, H. (2008). Cortical activation in alexithymia as a response to emotional stimuli. *The British Journal of Psychiatry*, 192(1), 32-38.

Katz, J., Martin, A. L., Pagé, M. G., & Calleri, V. (2009). Alexithymia and fear of pain independently predict heat pain intensity ratings among undergraduate university students. *Pain Research & Management: The Journal of the Canadian Pain Society*, 14(4), 299.

Kauhanen, J., Kaplan, G. A., Julkunen, J., Wilson, T. W., & Salonen, J. T. (1993). Social factors in alexithymia. *Comprehensive Psychiatry*, 34(5), 330-335.

Kestenbaum, R. (1992). Feeling happy versus feeling good: The processing of discrete and global categories of emotional expressions by children and adults. *Developmental Psychology*, 28(6), 1132.

Keysers, C., & Gazzola, V. (2009). Expanding the mirror: vicarious activity for actions, emotions, and sensations. *Current opinion in neurobiology*, 19(6), 666-671.

Keysers, C., Kaas, J. H., & Gazzola, V. (2010). Somatosensation in social perception. *Nature Reviews Neuroscience*, 11(6), 417-428.

Kilner, J. M., Friston, K. J., & Frith, C. D. (2007). Predictive coding: an account of the mirror neuron system. *Cognitive processing*, 8(3), 159-166.

Kirsch, P., Esslinger, C., Chen, Q., Mier, D., Lis, S., Siddhanti, S., ... & Meyer-Lindenberg, A. (2005). Oxytocin modulates neural circuitry for social cognition and fear in humans. *The Journal of neuroscience*, 25(49), 11489-11493.

Knoll, J. F., & Hodapp, V. (1992). A comparison between two methods for assessing heartbeat perception. *Psychophysiology*, 29(2), 218-222.

Kosturek, A., Gregory, R. J., Sousou, A. J., & Trief, P. (1998). Alexithymia and somatic amplification in chronic pain. *Psychosomatics*, 39(5), 399-404.

Kourtzi, Z., & Kanwisher, N. (2000). Activation in human MT/MST by static images with implied motion. *Journal of cognitive neuroscience*, 12(1), 48-55.

Kourtzi, Z., Krekelberg, B., & Van Wezel, R. J. (2008). Linking form and motion in the primate brain. *Trends in cognitive sciences*, 12(6), 230-236.

Kret, M. E., Pichon, S., Grèzes, J., & de Gelder, B. (2011). Similarities and differences in perceiving threat from dynamic faces and bodies. An fMRI study. *Neuroimage*, 54(2), 1755-1762.

Kugel, H., Eichmann, M., Dannlowski, U., Ohrmann, P., Bauer, J., Arolt, V., ... & Suslow, T. (2008). Alexithymic features and automatic amygdala reactivity to facial emotion. *Neuroscience letters*, 435(1), 40-44.

Làdavas, E., Cimatti, D., Pesce, M. D., & Tuozi, G. (1993). Emotional evaluation with and without conscious stimulus identification: Evidence from a split-brain patient. *Cognition & Emotion*, 7(1), 95-114.

Lane, R. D., Ahern, G. L., Schwartz, G. E., & Kaszniak, A. W. (1997). Is alexithymia the emotional equivalent of blindsight?. *Biological psychiatry*, 42(9), 834-844.

Lane, R. D., Lee, S., Reidel, R., Weldon, V., Kaszniak, A., & Schwartz, G. E. (1996). Impaired verbal and nonverbal emotion recognition in alexithymia. *Psychosomatic medicine*, 58(3), 203-210.

Lane, R. D., Sechrest, L., Riedel, R., Shapiro, D. E., & Kaszniak, A. W. (2000). Pervasive emotion recognition deficit common to alexithymia and the repressive coping style. *Psychosomatic medicine*, 62(4), 492-501.

Lane, R.D., Reiman, E.M., Axelrod, B., Yun, L.S., Holmes, A., Schwartz, G.E., 1998. Neural correlates of levels of emotional awareness. Evidence of an interaction between emotion and attention in the anterior cingulate cortex. *J. Cogn. Neurosci.* 10, 525–35.

Lang, P. J. (1995). The emotion probe: studies of motivation and attention. *American psychologist*, 50(5), 372.

Lang, P. J., & Davis, M. (2006). Emotion, motivation, and the brain: reflex foundations in animal and human research. *Progress in brain research*, 156, 3-29.

Larsen, J. K., Brand, N., Bermond, B., & Hijman, R. (2003). Cognitive and emotional characteristics of alexithymia: a review of neurobiological studies. *Journal of psychosomatic research*, 54(6), 533-541.

LeBar, I. C. S., LeDoux, JE, Spencer, DD, & Phelps, EA (1995). Impaired fear conditioning following unilateral temporal lobectomy in humans. *The Journal of Neuroscience*, 15, 6846-6855.

LeDoux, J. (1998). *The emotional brain: The mysterious underpinnings of emotional life*. Simon and Schuster.

LeDoux, J. (2012). Rethinking the emotional brain. *Neuron*, 73(4), 653-676.

LeDoux, J. E. (2014). Coming to terms with fear. *Proceedings of the National Academy of Sciences*, 111(8), 2871-2878.



Lepage, J. F., Tremblay, S., & Théoret, H. (2010). Early non-specific modulation of corticospinal excitability during action observation. *European Journal of Neuroscience*, 31(5), 931-937.

Lesser, R. P., Koehle, R., & Lueders, H. (1979). Effect of stimulus intensity on short latency somatosensory evoked potentials. *Electroencephalography and clinical neurophysiology*, 47(3), 377-382.

Leweke, F., Stark, R., Milch, W., Kurth, R., Schienle, A., Kirsch, P., ... & Vaitl, D. (2004). [Patterns of neuronal activity related to emotional stimulation in alexithymia]. *Psychotherapie, Psychosomatik, medizinische Psychologie*, 54(12), 437-444.

Linden, W., Stossel, C., & Maurice, J. (1996). Psychosocial interventions for patients with coronary artery disease: a meta-analysis. *Archives of Internal Medicine*, 156(7), 745-752.

Löken, L. S., Wessberg, J., McGlone, F., & Olausson, H. (2009). Coding of pleasant touch by unmyelinated afferents in humans. *Nature neuroscience*, 12(5), 547-548.

Loughland, C. M., Williams, L. M., & Gordon, E. (2002). Visual scanpaths to positive and negative facial emotions in an outpatient schizophrenia sample. *Schizophrenia research*, 55(1), 159-170.

Lumley, M. A., Stettner, L., & Wehmer, F. (1996). How are alexithymia and physical illness linked? A review and critique of pathways. *Journal of psychosomatic research*, 41(6), 505-518.

Lundqvist, D., Flykt, A., & Öhman, A. (1998). The Karolinska directed emotional faces (KDEF). *CD ROM from Department of Clinical Neuroscience, Psychology section, Karolinska Institutet*, 91-630.

Macmillan, N. A., & Creelman, C. D. (1991). *Detection theory: A user's guide* Cambridge University Press. *New York*.

Mangelli, L., Semprini, F., Sirri, L., Fava, G. A., & Sonino, N. (2006). Use of the Diagnostic Criteria for Psychosomatic Research (DCPR) in a community sample. *Psychosomatics*, 47(2), 143-146.

Mann, L. S., Wise, T. N., Trinidad, A., & Kohanski, R. (1994). Alexithymia, affect recognition, and the five-factor model of personality in normal subjects. *Psychological Reports*, 74(2), 563-567.

Marchesi, C., Brusamonti, E., & Maggini, C. (2000). Are alexithymia, depression, and anxiety distinct constructs in affective disorders?. *Journal of psychosomatic research*, 49(1), 43-49.

McDonald, P. W., & Prkachin, K. M. (1990). The expression and perception of facial emotion in alexithymia: a pilot study. *Psychosomatic Medicine*, 52(2), 199-210.

McGlone, F., Wessberg, J., & Olausson, H. (2014). Discriminative and affective touch: sensing and feeling. *Neuron*, 82(4), 737-755.

McIntosh, D. N., Reichmann-Decker, A., Winkielman, P., & Wilbarger, J. L. (2006). When the social mirror breaks: deficits in automatic, but not voluntary, mimicry of emotional facial expressions in autism. *Developmental science*, 9(3), 295-302.

Meaux, E., Hernandez, N., Carteau-Martin, I., Martineau, J., Barthélémy, C., Bonnet-Brilhault, F., & Batty, M. (2014). Event-related potential and eye tracking evidence of the developmental dynamics of face processing. *European Journal of Neuroscience*, 39(8), 1349-1362.

Meeren, H. K., de Gelder, B., Ahlfors, S. P., Hämäläinen, M. S., & Hadjikhani, N. (2013). Different cortical dynamics in face and body perception: an MEG study. *PLoS one*, 8(9), e71408.

Meeren, H. K., van Heijnsbergen, C. C., & de Gelder, B. (2005). Rapid perceptual integration of facial expression and emotional body language. *Proceedings of the National Academy of Sciences of the United States of America*, 102(45), 16518-16523.

Mesulam, M. (1981). A cortical network for directed attention and unilateral neglect. *Annals of neurology*, 10(4), 309-325.

Minnebusch, D. A., Keune, P. M., Suchan, B., & Daum, I. (2010). Gradual inversion affects the processing of human body shapes. *Neuroimage*, 49(3), 2746-2755.

Montagne, B., van Honk, J., Kessels, R. P., Frigerio, E., Burt, M., van Zandvoort, M. J., ... & de Haan, E. H. (2005). Reduced efficiency in recognising fear in subjects scoring high on psychopathic personality characteristics. *Personality and Individual Differences*, 38(1), 5-11.

Moriguchi, Y., & Komaki, G. (2013). Neuroimaging studies of alexithymia: physical, affective, and social perspectives. *BioPsychoSocial medicine*, 7(8), 1-12.

Moriguchi, Y., Decety, J., Ohnishi, T., Maeda, M., Mori, T., Nemoto, K., ... & Komaki, G. (2007). Empathy and judging other's pain: an fMRI study of alexithymia. *Cerebral Cortex*, 17(9), 2223-2234.

Morris, J. S., Friston, K. J., Büchel, C., Frith, C. D., Young, A. W., Calder, A. J., & Dolan, R. J. (1998). A neuromodulatory role for the human amygdala in processing emotional facial expressions. *Brain*, 121(1), 47-57.

Morris, J.S., Ohman, A., and Dolan, R.J. (1999). A subcortical pathway to the right amygdala mediating "unseen" fear. *Proceedings of the National Academy of Sciences of United States of America*. 96(4):1680-5.

Mouraux, A., Guerit, J. M., & Plaghki, L. (2003). Non-phase locked electroencephalogram (EEG) responses to CO<sub>2</sub> laser skin stimulations may reflect central interactions between A $\delta$ -and C-fibre afferent volleys. *Clinical neurophysiology*, 114(4), 710-722.

Nemiah, J. C., Freyberger, H., & Sinfeos, P. E. (1976). Alexithymia: A view of the psychosomatic process. In O. W. Hill (Ed.), *Modern trends in psychosomatic medicine* (Vol. 3, pp. 430–439). London, UK: Butterworths.

Nemiah, J.C., Sifneos, P.E. and Apfel-Savitz, R. (1997). A comparison of oxygen consumption of normal and alexithymic subjects in response to affect-provoking thoughts. *Psychotherapy and Psychosomatics*, 28, 245-253.

Neumann, S. A., Sollers, J. J., Thayer, J. F., & Waldstein, S. R. (2004). Alexithymia predicts attenuated autonomic reactivity, but prolonged recovery to anger recall in young women. *International Journal of Psychophysiology*, 53(3), 183-195.

Newton, T. L., & Contrada, R. J. (1994). Alexithymia and repression: contrasting emotion-focused coping styles. *Psychosomatic medicine*, 56(5), 457-462.

Niedenthal, P. M. (2007). Embodying emotion. *science*, 316(5827), 1002-1005.

Niedenthal, P. M., Mermillod, M., Maringer, M., & Hess, U. (2010). The Simulation of Smiles (SIMS) model: Embodied simulation and the meaning of facial expression. *Behavioral and brain sciences*, 33(06), 417-433.

Nishitani, N., Avikainen, S., Hari, R., 2004. Abnormal imitation-related cortical activation sequences in Asperger's syndrome. *Ann. Neurol.* 55, 558–62.

Noton, D., & Stark, L. (1971). Scanpaths in saccadic eye movements while viewing and recognizing patterns. *Vision research*, 11(9), 929-938.

Nyklíček, I., & Vingerhoets, A. J. (2000). Alexithymia is associated with low tolerance to experimental painful stimulation. *Pain*, 85(3), 471-475.

Oberman, L. M., Winkielman, P., & Ramachandran, V. S. (2007). Face to face: Blocking facial mimicry can selectively impair recognition of emotional expressions. *Social neuroscience*, 2(3-4), 167-178.

Öhman, A., Lundqvist, D., Esteves, F., (2001). The face in the crowd revisited : a threat advantage with schematic stimuli. *J. Pers. Soc. Psychol.* 80, 381–396.

Olausson, H., Charron, J., Marchand, S., Villemure, C., Strigo, I. A., & Bushnell, M. C. (2005). Feelings of warmth correlate with neural activity in right anterior insular cortex. *Neuroscience letters*, *389*(1), 1-5.

Olausson, H., Lamarre, Y., Backlund, H., Morin, C., Wallin, B. G., Starck, G., ... & Bushnell, M. C. (2002). Unmyelinated tactile afferents signal touch and project to insular cortex. *Nature neuroscience*, *5*(9), 900-904.

Olofsson, J. K., Nordin, S., Sequeira, H., & Polich, J. (2008). Affective picture processing: an integrative review of ERP findings. *Biological psychology*, *77*(3), 247-265.

O'Regan, J. K., & Le, A. (1983). Integrating visual information from successive fixations: Does trans-saccadic fusion exist?. *Vision research*, *23*(8), 765-768.

Oya, H., Kawasaki, H., Iii, M.A.H., Adolphs, R. (2002). Electrophysiological Responses in the Human Amygdala Discriminate Emotion Categories of Complex Visual Stimuli. *J. Neurosci.* *22*, 9502–9512.

Papciak, A. S., Feuerstein, M., & Spiegel, J. A. (1985). Stress reactivity in alexithymia: decoupling of physiological and cognitive responses. *Journal of Human stress*, *11*(3), 135-142.

Papez, J. W. (1937). A proposed mechanism of emotion. *Archives of neurology and psychiatry*, *38*(4), 725.

Parker, J. D., Keefer, K. V., Taylor, G. J., & Bagby, R. M. (2008). Latent structure of the alexithymia construct: a taxometric investigation. *Psychological assessment*, *20*(4), 385.

Parker, J. D., Michael Bagby, R., Taylor, G. J., Endler, N. S., & Schmitz, P. (1993). Factorial validity of the 20-item Toronto Alexithymia Scale. *European Journal of Personality*, *7*(4), 221-232.

Parker, J. D., Taylor, G. J., & Bagby, R. M. (1993). Alexithymia and the recognition of facial expressions of emotion. *Psychotherapy and psychosomatics*, 59(3-4), 197-202.

Parker, J.D.A., Keefer, K. V, Taylor, G.J., Bagby, R.M., 2008. Latent structure of the alexithymia construct: a taxometric investigation. *Psychol. Assess.* 20, 385–396.

Parker, P. D., Prkachin, K. M., & Prkachin, G. C. (2005). Processing of facial expressions of negative emotion in alexithymia: the influence of temporal constraint. *Journal of personality*, 73(4), 1087-1107.

Parkhurst, D., Law, K., & Niebur, E. (2002). Modeling the role of salience in the allocation of overt visual attention. *Vision research*, 42(1), 107-123.

Patil, I., & Silani, G. (2014). Reduced empathic concern leads to utilitarian moral judgments in trait alexithymia. *Frontiers in psychology*, 5.

Peelen, M. V., & Downing, P. E. (2005). Selectivity for the human body in the fusiform gyrus. *Journal of neurophysiology*, 93(1), 603-608.

Peelen, M. V., & Downing, P. E. (2007). The neural basis of visual body perception. *Nature Reviews Neuroscience*, 8(8), 636-648.

Peelen, M. V., Atkinson, A. P., Andersson, F., & Vuilleumier, P. (2007). Emotional modulation of bodyselective visual areas. *Social Cognitive and Affective Neuroscience*, 2(4), 274-283.

Peelen, M. V., Wiggett, A. J., & Downing, P. E. (2006). Patterns of fMRI activity dissociate overlapping functional brain areas that respond to biological motion. *Neuron*, 49(6), 815-822.

Pegna, A. J., Darque, A., Berrut, C., & Khateb, A. (2011). Early ERP modulation for task-irrelevant subliminal faces. *Frontiers in Psychology*, 2, 88.

Pegna, A. J., Landis, T., & Khateb, A. (2008). Electrophysiological evidence for early non-conscious processing of fearful facial expressions. *International Journal of Psychophysiology*, 70(2), 127-136.

Pegna, A.J., Darque, A., Berrut, C., Khateb, A. (2011). Early ERP modulation for task-irrelevant subliminal faces. *Front. Psychol.* 2, 1–10.

Peigneux, P., Salmon, E., Van Der Linden, M., Garraux, G., Aerts, J., Delfiore, G., ... & Franck, G. (2000). The role of lateral occipitotemporal junction and area MT/V5 in the visual analysis of upperlimb postures. *Neuroimage*, 11(6), 644-655.

Perez, D. L., Barsky, A. J., Vago, D. R., Baslet, G., & Silbersweig, D. A. (2015). A neural circuit framework for somatosensory amplification in somatoform disorders. *The Journal of neuropsychiatry and clinical neurosciences*.

Perrett, D.I., Xiao, D., Barraclough, N.E., Keysers, C., & Oram, M.W. (2009). Seeing the future: Natural image sequences produce "anticipatory" neuronal activity and bias perceptual report. *Quarterly journal of experimental psychology*, 62(11):2081-2104.

Pichon, S., de Gelder, B., & Grezes, J. (2008). Emotional modulation of visual and motor areas by dynamic body expressions of anger. *Social neuroscience*, 3(3-4), 199-212.

Pitcher, D., Garrido, L., Walsh, V., & Duchaine, B. C. (2008). Transcranial magnetic stimulation disrupts the perception and embodiment of facial expressions. *The Journal of Neuroscience*, 28(36), 8929-8933.

Pollatos, O., Gramann, K. (2011). Electrophysiological evidence of early processing deficits in alexithymia. *Biol. Psychol.* 87, 113–121.

Pollatos, O., Schubö, A., Herbert, B.M., Matthias, E., Schandry, R. (2008). Deficits in early emotional reactivity in alexithymia. *Psychophysiology* 45, 839–846.

Porcelli, P., & Rafanelli, C. (2010). Criteria for psychosomatic research (DCPR) in the medical setting. *Current psychiatry reports*, 12(3), 246-254.

Porcelli, P., & Sonino, N. (2007). Psychological factors affecting medical conditions. *Advances in psychosomatic medicine*, 200.

Pouga, L., Berthoz, S., de Gelder, B., & Grezes, J. (2010). Individual differences in socioaffective skills influence the neural bases of fear processing: the case of alexithymia. *Human brain mapping*, 31(10), 1469-1481.

Pourtois, G., & Vuilleumier, P. (2006). Dynamics of emotional effects on spatial attention in the human visual cortex. *Progress in brain research*, 156, 67-91.

Prkachin, G. C., Casey, C., & Prkachin, K. M. (2009). Alexithymia and perception of facial expressions of emotion. *Personality and Individual Differences*, 46(4), 412-417.

Puce, A., & Perrett, D. (2003). Electrophysiology and brain imaging of biological motion. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, 358(1431), 435-445.

Puce, A., Smith, A., & Allison, T. (2000). ERPs evoked by viewing facial movements. *Cognitive Neuropsychology*, 17(1-3), 221-239.

Reed, C. L., Stone, V. E., Bozova, S., & Tanaka, J. (2003). The body-inversion effect. *Psychological science*, 14(4), 302-308.

Reker, M., Ohrmann, P., Rauch, A. V., Kugel, H., Bauer, J., Dannlowski, U., ... & Suslow, T. (2010). Individual differences in alexithymia and brain response to masked emotion faces. *Cortex*, 46(5), 658-667.

Rellecke, J., Sommer, W., Schacht, A., 2012. Does processing of emotional facial expressions depend on intention? Time-resolved evidence from event-related brain potentials. *Biol. Psychol.* 90, 23–32.



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

Rizzolatti, G., Cattaneo, L., Fabbri-Destro, M., & Rozzi, S. (2014). Cortical Mechanisms Underlying the Organization of Goal-Directed Actions and Mirror Neuron-Based Action Understanding. *Physiological reviews*, 94(2), 655-706.

Roedema, T. M., & Simons, R. F. (1999). Emotion-processing deficit in alexithymia. *Psychophysiology*, 36(3), 379-387.

Roelofs, K., Hagens, M. A., & Stins, J. (2010). Facing Freeze Social Threat Induces Bodily Freeze in Humans. *Psychological Science*.

Rolke, R., Baron, R., Maier, C., Tölle, T. R., Treede, R. D., Beyer, A., ... & Braune, S. (2006a). Quantitative sensory testing in the German Research Network on Neuropathic Pain (DFNS): standardized protocol and reference values. *Pain*, 123(3), 231-243.

Rolke, R., Magerl, W., Campbell, K. A., Schalber, C., Caspari, S., Birklein, F., & Treede, R. D. (2006b). Quantitative sensory testing: a comprehensive protocol for clinical trials. *European Journal of Pain*, 10(1), 77-77.

Rolls, E. T. (2010). The affective and cognitive processing of touch, oral texture, and temperature in the brain. *Neuroscience & Biobehavioral Reviews*, 34(2), 237-245.

Rolls, E. T., Grabenhorst, F., & Parris, B. A. (2008). Warm pleasant feelings in the brain. *Neuroimage*, 41(4), 1504-1513.

Rosenberg, E. L., & Ekman, P. (1994). Coherence between expressive and experiential systems in emotion. *Cognition & Emotion*, 8(3), 201-229.

Rossi, A., Parada, F. J., Kolchinsky, A., & Puce, A. (2014). Neural correlates of apparent motion perception of impoverished facial stimuli: A comparison of ERP and ERSP activity. *Neuroimage*, 98, 44259.

Rossion, B., & Jacques, C. (2008). Does physical interstimulus variance account for early electrophysiological face sensitive responses in the human brain? Ten lessons on the N170. *Neuroimage*, 39(4), 1959-1979.

Salminen, J. K., Saarijärvi, S., Äärelä, E., Toikka, T., & Kauhanen, J. (1999). Prevalence of alexithymia and its association with sociodemographic variables in the general population of Finland. *Journal of psychosomatic research*, 46(1), 75-82.

Sato, W., Kochiyama, T., Yoshikawa, S., & Matsumura, M. (2001). Emotional expression boosts early visual processing of the face: ERP recording and its decomposition by independent component analysis. *Neuroreport*, 12(4), 709-714.

Saxe, R., Jamal, N., & Powell, L. (2006). My body or yours? The effect of visual perspective on cortical body representations. *Cerebral Cortex*, 16(2), 178-182.

Scarpazza, C. (2015). Deficit in the Emotional Embodiment in Alexithymia.

Scarpazza, C., Di Pellegrino, G., & Làdavas, E. (2014). Emotional modulation of touch in alexithymia. *Emotion*, 14(3), 602.

Schachter, S., & Singer, J. (1962). Cognitive, social, and physiological determinants of emotional state. *Psychological review*, 69(5), 379.

Schäfer, R., Schneider, C., Tress, W., Franz, M. (2007). Cortical augmenting in alexithymic subjects after unpleasant acoustic stimulation. *J. Psychosom. Res.* 63, 357–364.

Schandry, R. (1981). Heart beat perception and emotional experience. *Psychophysiology*, 18(4), 483-488.

Schepers, R. J., & Ringkamp, M. (2010). Thermoreceptors and thermosensitive afferents. *Neuroscience & Biobehavioral Reviews*, 34(2), 177-184.

Schippers, M. B., & Keysers, C. (2011). Mapping the flow of information within the putative mirror neuron system during gesture observation. *Neuroimage*, 57(1), 37-44.

Schmidt, U., Jiwany, A., & Treasure, J. (1993). A controlled study of alexithymia in eating disorders. *Comprehensive psychiatry*, 34(1), 54-58.

Schupp, H. T., Flaisch, T., Stockburger, J., & Junghöfer, M. (2006). Emotion and attention: event-related brain potential studies. *Progress in brain research*, 156, 31-51.

Schupp, H. T., Junghöfer, M., Weike, A. I., & Hamm, A. O. (2003). Attention and emotion: an ERP analysis of facilitated emotional stimulus processing. *Neuroreport*, 14(8), 1107-1110

Schupp, H. T., Junghöfer, M., Weike, A. I., & Hamm, A. O. (2004b). The selective processing of briefly presented affective pictures: An ERP analysis. *Psychophysiology*, 41(3), 441-449.

Schupp, H. T., Öhman, A., Junghöfer, M., Weike, A. I., Stockburger, J., & Hamm, A. O. (2004a). The facilitated processing of threatening faces: an ERP analysis. *Emotion*, 4(2), 189.

Senior, C., Barnes, J., Giampietro, V., Simmons, A., Bullmore, E. T., Brammer, M., & David, A. S. (2000). The functional neuroanatomy of implicit-motion perception or 'representational momentum'. *Current Biology*, 10(1), 16-22.

Sifneos, P. E. (1973). The prevalence of 'alexithymic' characteristics in psychosomatic patients. *Psychotherapy and psychosomatics*, 22(2-6), 255-262.

Silani, G., Bird, G., Brindley, R., Singer, T., Frith, C., & Frith, U. (2008). Levels of emotional awareness and autism: an fMRI study. *Social neuroscience*, 3(2), 97-112.

Singer, T., Critchley, H. D., & Preuschoff, K. (2009). A common role of insula in feelings, empathy and uncertainty. *Trends in cognitive sciences*, 13(8), 334-340.

Stekelenburg, J. J., & de Gelder, B. (2004). The neural correlates of perceiving human bodies: an ERP study on the body-inversion effect. *Neuroreport*, 15(5), 777-780.

Streit, M., Wölwer, W., & Gaebel, W. (1997). Facial-affect recognition and visual scanning behaviour in the course of schizophrenia. *Schizophrenia research*, 24(3), 311-317.

Taylor, G. J. (2000). Recent developments in alexithymia theory and research. *The Canadian Journal of Psychiatry/La Revue canadienne de psychiatrie*.

Taylor, G. J., & Bagby, R. M. (2004). New trends in alexithymia research. *Psychotherapy and psychosomatics*, 73(2), 68-77.

Taylor, G. J., Bagby, R. M., & Parker, J. D. (1991). The alexithymia construct: a potential paradigm for psychosomatic medicine. *Psychosomatics*, 32(2), 153-164.

Taylor, G. J., Bagby, R. M., & Parker, J. D. (2003). The 20-Item Toronto Alexithymia Scale: IV. Reliability and factorial validity in different languages and cultures. *Journal of psychosomatic research*, 55(3), 277-283.

Taylor, G. J., Bagby, R. M., & Parker, J. D. (2003). The 20-Item Toronto Alexithymia Scale: IV. Reliability and factorial validity in different languages and cultures. *Journal of psychosomatic research*, 55(3), 277-283.

Taylor, G. J., Parker, J. D., Bagby, R. M., & Bourke, M. P. (1996). Relationships between alexithymia and psychological characteristics associated with eating disorders. *Journal of Psychosomatic research*, 41(6), 561-568.

Taylor, G.J., Bagby, R.M., Parker, J.D., 1991. The alexithymia construct. A potential paradigm for psychosomatic medicine. *Psychosomatics* 32, 153–64.

Taylor, J. C., Wiggett, A. J., & Downing, P. E. (2007). Functional MRI analysis of body and body part representations in the extrastriate and fusiform body areas. *Journal of neurophysiology*, 98(3), 1626-1633.

Taylor, J. C., Wiggett, A. J., & Downing, P. E. (2010). fMRI–Adaptation Studies of Viewpoint Tuning in the Extrastriate and Fusiform Body Areas. *Journal of Neurophysiology*, 103(3), 1467-1477.

Taylor, S.F., Liberzon, I., Koeppe, R.A. (2000). The effect of graded aversive stimuli on limbic and visual activation. *Neuropsychologia* 38, 1415–25.

Thierry, G., Pegna, A. J., Dodds, C., Roberts, M., Basan, S., & Downing, P. (2006). An event-related potential component sensitive to images of the human body. *Neuroimage*, 32(2), 871-879.

Thom, N., Knight, J., Dishman, R., Sabatinelli, D., Johnson, D. C., & Clementz, B. (2014). Emotional scenes elicit more pronounced self-reported emotional experience and greater EPN and LPP modulation when compared to emotional faces. *Cognitive, Affective, & Behavioral Neuroscience*, 14(2):84960.

Tidoni, E., Borgomaneri, S., Di Pellegrino, G., & Avenanti, A. (2013). Action simulation plays a critical role in deceptive action recognition. *The Journal of Neuroscience*, 33(2), 611-623

Todorov, A., & Engell, A. D. (2008). The role of the amygdala in implicit evaluation of emotionally neutral faces. *Social cognitive and affective neuroscience*, nsn033.

Tomkins, S. (1963). *Affect/imagery/consciousness*. Vol. 2: The negative affects.

Tomkins, S. S. (1962). *Affect, imagery, consciousness: Vol. I. The positive affects*.

Tucker, D. M., & Williamson, P. A. (1984). Asymmetric neural control systems in human self-regulation. *Psychological review*, 91(2), 185.

Ubaldi, S., Barchiesi, G., & Cattaneo, L. (2013). Bottom-Up and Top-Down Visuomotor Responses to Action Observation. *Cerebral Cortex*, doi:10.1093/cercor/bht295.

Urgesi, C., Candidi, M., & Avenanti, A. (2014). Neuroanatomical substrates of action perception and understanding: an anatomic likelihood estimation meta-analysis of lesion-symptom mapping studies in brain injured patients. *Frontiers in Human Neuroscience*, 8, 344. doi: 10.3389/fnhum.2014.00344.

Urgesi, C., Maieron, M., Avenanti, A., Tidoni, E., Fabbro, F., & Aglioti, S. M. (2010). Simulating the future of actions in the human corticospinal system. *Cerebral Cortex*, 20(11), 2511-2521.

Valdés-Conroy, B., Aguado, L., Fernández-Cahill, M., Romero-Ferreiro, V., Diéguez-Risco, T., 2014. Following the time course of face gender and expression processing: A task-dependent ERP study. *Int. J. Psychophysiol.* 92, 59–66.

Van Boven, R. W., & Johnson, K. O. (1994). The limit of tactile spatial resolution in humans Grating orientation discrimination at the lip, tongue, and finger. *Neurology*, 44(12), 2361-2361.

Van de Riet, W. A., Grèzes, J., & de Gelder, B. (2009). Specific and common brain regions involved in the perception of faces and bodies and the representation of their emotional expressions. *Social Neuroscience*, 4(2), 101-120.

van der Velde, J., Gromann, P. M., Swart, M., Wiersma, D., de Haan, L., Bruggeman, R., ... & Aleman, A. (2015). Alexithymia influences brain activation during emotion perception but not regulation. *Social cognitive and affective neuroscience*, 10(2), 285-293.

van der Velde, J., Servaas, M. N., Goerlich, K. S., Bruggeman, R., Horton, P., Costafreda, S. G., & Aleman, A. (2013). Neural correlates of alexithymia: A meta-analysis of emotion processing studies. *Neuroscience & Biobehavioral Reviews*, 37(8), 1774-1785.

Van der Velde, J., Servaas, M.N., Goerlich, K.S., Bruggeman, R., Horton, P., Costafreda, S.G., Aleman, A., 2013. Neural correlates of alexithymia: A meta-analysis of emotion processing studies. *Neurosci. Biobehav. Rev.* 37, 1774–1785.

Van Heijnsbergen, C. C. R. J., Meeren, H. K. M., Grezes, J., & de Gelder, B. (2007). Rapid detection of fear in body expressions, an ERP study. *Brain research*, 1186, 233-241.

van Schie, H. T., Koelewijn, T., Jensen, O., Oostenveld, R., Maris, E., & Bekkering, H. (2008). Evidence for fast, low-level motor resonance to action observation: an MEG study. *Social Neuroscience*, 3(34), 213-228.

Vanheule, S., Desmet, M., Meganck, R., & Bogaerts, S. (2007). Alexithymia and interpersonal problems. *Journal of clinical psychology*, 63(1), 109-117.

Verfaillie, K., & Daems, A. (2002). Representing and anticipating human actions in vision. *Visual Cognition*, 9(1-2), 217-232.

Vorst, H. C., & Bermond, B. (2001). Validity and reliability of the Bermond–Vorst alexithymia questionnaire. *Personality and individual differences*, 30(3), 413-434.

Vuilleumier, P. (2002). Facial expression and selective attention. *Curr. Opin. Psychiatry* 15, 291– 300.

Vuilleumier, P., Richardson, M. P., Armony, J. L., Driver, J., & Dolan, R. J. (2004). Distant influences of amygdala lesion on visual cortical activation during emotional face processing. *Nature neuroscience*, 7(11), 1271-1278.

Waldstein, S. R., Kauhanen, J., Neumann, S. A., & Katzel, L. I. (2002). Alexithymia and cardiovascular risk in older adults: psychosocial, psychophysiological, and biomedical correlates. *Psychology and Health*, 17(5), 597-610.

Wehmer, F., Brejnak, C., Lumley, M., & Stettner, L. (1995). Alexithymia and physiological reactivity to emotion-provoking visual scenes. *The Journal of nervous and mental disease*, 183(6), 351-357.

Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike, M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *The Journal of neuroscience*, 18(1), 411-418.

Williams, L. E., & Bargh, J. A. (2008). Experiencing physical warmth promotes interpersonal warmth. *Science*, 322(5901), 606-607.

Wilson, M., & Knoblich, G. (2005). The case for motor involvement in perceiving conspecifics. *Psychological bulletin*, 131(3), 460.

Wingbermhühle, E., Theunissen, H., Verhoeven, W., Kessels, R. P., & Egger, J. I. (2012). The neurocognition of alexithymia: evidence from neuropsychological and neuroimaging studies. *Acta Neuropsychiatrica*, 24(2), 67-80.

Wundt W (1903) Characteristics of physiological psychology, vol 3, 5th edn. W Engelmann, Leipzig

Yarbus, A. L. (1967). *Eye movements during perception of complex objects*(pp. 171-211). Springer US.

Yarnitsky, D., Sprecher, E., Zaslansky, R., & Hemli, J. A. (1995). Heat pain thresholds: normative data and repeatability. *Pain*, 60(3), 329-332.

Yokoyama, K., Jennings, R., Ackles, P., Hood, P., & Boller, F. (1987). Lack of heart rate changes during an attention-demanding task after right hemisphere lesions. *Neurology*, 37(4), 624-624.

Zaidel, D. W. (Ed.). (2013). *Neuropsychology*. Academic Press.