

Alma Mater Studiorum – Università di Bologna

DOTTORATO DI RICERCA IN

International Ph.D. Program in Cognitive Neuroscience

Ciclo XXVII

Settore Concorsuale di afferenza: AREA 11/E1

Settore Scientifico disciplinare: M-PSI/02

Deficit in the Emotional Embodiment in Alexithymia

Presentata da: **Dott.ssa Cristina Scarpazza**

Coordianatore Dottorato

Prof.ssa Elisabetta Làdavas

Relatore

Prof.ssa Elisabetta Làdavas

Esame finale anno 2015

*To my parents, Enrico and Fiorella
Since emotions are widely and intensely experienced by children,
I dedicate this thesis to the little kids
From whom these grown-up grew.*

*Thanks for being
Different as colors
And fundamental as breath.*

Table of Content:

Abstract	1
Introduction	3
CHAPTER 1: Emotions and Embodiment	11
1.1) Different theories of Emotions.....	12
1.2) The Embodiment of Emotions.....	14
CHAPTER 2: Alexithymia: emotional dumbness or numbness?.....	17
2.1) Historical hints.....	18
2.2) The construct of alexithymia.....	19
2.3) Subtypes of alexithymia.....	21
2.4) Literature’s review.....	25
2.4.1) Deficit in emotional word processing.....	26
2.4.2) Deficit in the cognitive interpretation of emotions.....	27
2.3.3) Alteration in emotional arousal.....	28
2.3.4) Deficit in emotional perception.....	30
2.3.5) Deficit in emotional embodiment.....	31
2.5) The subject’s selection problem.....	32
2.6) Conclusion.....	37
CHAPTER 3: In touch with Emotions	43
3.1) Introduction.....	43
3.2) <u>Experiment 1</u> : Emotional modulation of touch in Alexithymia	46
3.2.1) Material and Methods.....	46
3.2.2) Results.....	51
3.3) <u>Experiment 2</u> : Neuropsychological profile in Alexithymia.....	58
3.3.1) Material and Methods.....	59
3.3.2) Results.....	61
3.4) Discussion.....	62

CHAPTER 4: Invisible sides of Emotions	68
4.1) Introduction.....	69
4.2) <u>Experiment 3</u> : Somato-motor reactions to emotional facial displays.....	73
4.2.1) Material and Methods.....	73
4.2.2) Results.....	78
3.3) <u>Experiment 4</u> : Visceral reactions to emotional facial displays.....	83
4.3.1) Material and Methods.....	83
4.3.2) Results.....	85
4.4) Discussion.....	87
 CHAPTER 5: Emotions and the body	 95
5.1) Introduction.....	96
5.2) <u>Experiment 5</u> : Dissociation between Emotional Remapping of Fear and Disgust.....	99
5.2.1) Material and Methods.....	99
5.2.2) Results.....	103
5.2.3) Discussion of Experiment 5.....	109
5.3) <u>Experiment 6</u> : The role of interoception in embodiment of disgust.	111
5.3.1) Material and Methods.....	111
5.3.2) Results.....	113
5.3.3) Discussion of Experiment 6.....	116
5.4) Discussion.....	118
 CHAPTER 6: Testing the functional contribution of the amygdale	 125
6.1) Introduction.....	126
6.2) <u>Experiment 7</u> : Disruption of the fear enhancement of tactile perception after Amygdala lesions.....	129
6.2.1) Material and Methods.....	129
6.2.2) Results.....	136
6.3) Discussion.....	141

CHAPTER 7: Are alexithymic impaired in face perception or in emotion perception conveyed by faces?.....	146
7.1) Introduction.....	146
7.2) <u>Experiment 8</u> : n170 emotional modulation in alexithymia.....	150
7.2.1) Material and Methods.....	150
7.2.2) Results.....	156
7.3) Discussion.....	165
CHAPTER 8: General Discussion.....	173
8.1) Alexithymia and happiness.....	176
8.2) Alexithymia and disgust.....	179
8.3) Alexithymia and fear.....	181
8.4) A unifying interpretation: the amygdalar dysfunction.....	185
8.5) Final consideration about the term “Alexithymia”.....	189
8.6) Future dicrections.....	191
References.....	194

Abstract

Alexithymia refers to difficulties in recognizing one's own emotions and others emotions. Theories of emotional embodiment suggest that, in order to understand other peoples' feelings, observers re-experience, or simulate, the relevant component (i.e. somatic, motor, visceral) of emotion's expressed by others in one's self. In this way, the emotions are "embodied". Critically, to date, there are no studies investigating the ability of alexithymic individuals in embodying the emotions conveyed by faces.

In the present dissertation different implicit paradigms and techniques falling within the field of affective neuroscience have been employed in order to test a possible deficit in the embodiment of emotions in alexithymia while subjects were requested to observe faces manifesting different expression: fear, disgust, happiness and neutral. The level of the perceptual encoding of emotional faces and the embodiment of emotions in the somato-sensory and sensory-motor system have been investigated. Moreover, non-communicative motor reaction to emotional stimuli (i.e. visceral reactions) and interoceptive abilities of alexithymic subjects have been explored.

The present dissertation provided convergent evidences in support of a deficit in the processing of fearful expression in subjects with high alexithymic personality traits. Indeed, the pattern of fear induced changes in the perceptual encoding, in the somato-sensory and in the somato-motor system (both the communicative and non communicative one) is widely and consistently altered in alexithymia. This support the hypothesis of a diminished responses to fearful stimuli in alexithymia.

In addition, the overall results on happiness and disgust, although preliminary, provided interesting results. Indeed, the results on happiness revealed a defective perceptual encoding, coupled with a slight difficulty (i.e. delayed responses) at the level of the communicative somato-motor system, and the emotion of disgust has been found to be abnormally embodied at the level of the somato-sensory system.

Introduction

“I know of a planet where there is a red-faced gentleman. He has never smelled a flower. He has never looked a star. He has never loved anybody. He has spent all this time adding up figures. And all day, he keeps on repeating <<I’m busy with serious matters>>, over and over again. And he swells up with pride. But he is not a man, he is a mushroom.”

The Little Prince

Antoine de Saint-Exupéry

Human’s lives are intertwined with those of other people and this makes them intensely social creatures. In this social environment, successful interactions require the ability to precisely understand emotions displayed by other individuals, which is critical to react in an adaptive way. Indeed, emotions imbue our lives. We choose a friend because we like him; we choose work because we enjoy it; we choose where to go on holidays because we consider it exiting, or relaxing. We smell a flower because we like the smell; we look at the sunshine because it makes us happy. Citing the words of the Little Prince in Antoine de Saint-Exupéry’s book, these are the important things of life. If someone never smelled a flower, never saw sunshine, never loved anyone, he/she is not a human being, but a mushroom, i.e. a living being without the possibility of making any kind of choice. Indeed, emotions guide every choice in our life. As a consequence, emotions are not ephemeral like the roses.

There are three reasons why I’ve always been fascinated by emotions.

The emotions in everyday life and relationship.

Humans automatically and effortlessly experience emotions and detect emotions in each other routinely each and every day. I've always been fascinated by this ability. Since childhood I was able to understand if my mother had an headache, if my father had problems at work, if my grandmother was worried about something, just looking at them, at their faces, at their movement. During my professional experience I had the opportunity to work in a psychiatric hospital, where I had to deal with people who were unable to control their own emotions, above all the fear and the rage. Then, I worked in a children's house with adolescent teenagers coming from very different ethnicity, many from Morocco and Egypt, where they were not allowed to overtly express most of the positive (i.e. they cannot express attachment, or happiness) and negative (i.e. they were considered weak if they manifested fear, or sadness) emotions, while some other emotions were considered important for them to be considered real men, for instance rage. The psychiatric disorder, in the first case, or the different ethnicity, in the second case, lead to different expressions of emotions that interfere with a normal life and a normal socialization in those individuals. For instance, "my" children experienced huge difficulties in dealing with other people. I remember Bassim, 15 years old, who always screamed at me, who threw a cup of hot tea at me just because I gave him some rules. He was not used to listening to women's advice, and he expressed the emotions he felt with great rage. This is only one example of how uncontrollable emotions could interfere with socialization and, in this case, with integration. But our daily life is full of situations similar to this one, but less striking.

So, I'm interested in emotions first of all because successful socialization depends entirely on emotions

Emotions' influence on art.

Emotions have inspired every form of art for centuries. For each form of art, it would be possible to produce a million examples.

Concerning MUSIC, just think of the violin concerto in D major, Op. 35, my favorite composition written by *Pyotr Ilych Tchaikovsky* in 1878. The concert was written during the recovery from the depression brought about by the disastrous marriage of the composer and Antonina Miliukova. Roughly in the same period of his life, Pyotr started an homosexual relationship with his pupil, the violinist Iosif Kotek. The Concert is impregnated with emotions: the composer alternated passages in which anguish and uncertainty are clearly perceived, defined by the critics “winding melody and melancholy”, with other passages in which a sense of liberation and enthusiasm are evident, “the absolute amount of liveliness”. Although initially disliked, the violin concerto in D major was then considered an innovative composition, since it was no more a mere academic composition, but “this work is pure emotion”.

Concerning SCULPTURE, I firmly think that the best example of emotional sculpture is provided by my favorite sculptor, August Rodin. His popularity is ascribed to his emotion-laden representations of ordinary men and women, to his ability to find the beauty and pathos in the human being. Some of his most popular works, such as *The Burghers of Calais* (Figure 1, left) and *Fugit Amor* (Figure 1, right) are widely used outside the fine arts as symbols of human emotion and character. Rodin's art stressed the figure's rough physicality and the emotional tension emanating from it. Indeed, the artist believed that an individual's character was revealed by his emotional profile.



Figure 1. Rodin's most beautiful examples of emotional sculpture. In the left, a particular of *The Burghers of Calais* (1884-1889). In the right, is it possible to appreciate *Fugit Amor* (1892-1894), which appeared originally on the Gates of Hell, representing the Dantesque characters Paolo and Francesca.

Concerning PAINTING, between thousands of possible examples (just think to the famous *The Scream* of Edvard Munch, or Vincent Van Gogh's *At Eternity's Gate*, see Figure 2), my heart has always been fascinated by the non obvious emotions hidden in Magritte's paintings. Magritte had the wonderful ability to express through painting the emotions related to one's own identity. The persons painted by Magritte often hide behind objects or disappear within the background. It's frustrating you can't see the person's face and hence its identity. This is actually how in our society every person is often seen in daily life. People judge others on what they see, their facial expression, the clothes they're wearing, but underneath all that there may be a whole unexpected other identity. And this is so horrible that sometimes people are not even able to see themselves (Figure 2).

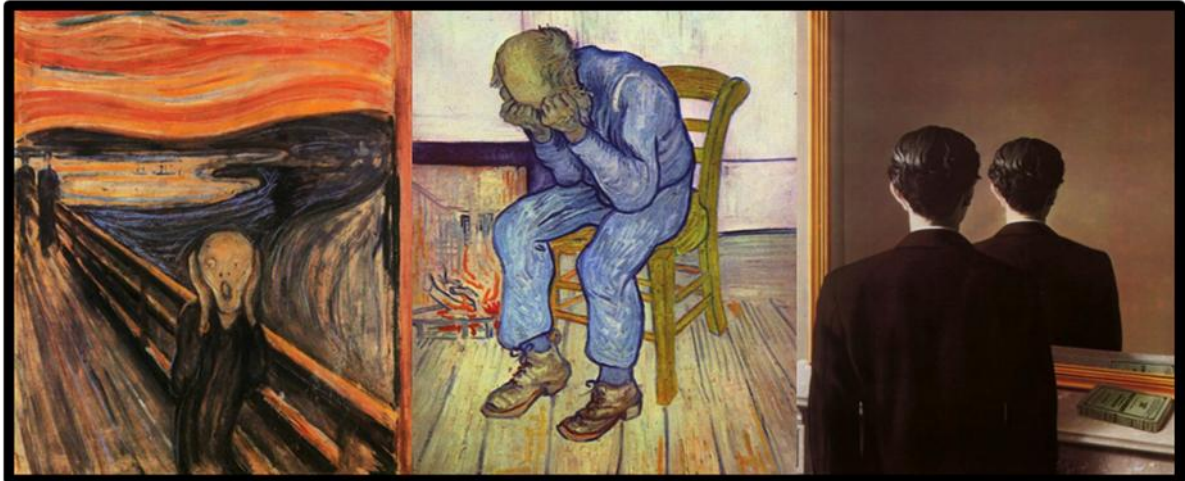


Figure 2. Emotional Painting. From the left to the right side: Edvard Much, “The Scream” (1893); Vincent van Gogh, “At Eternity’s Gate” (1890); Rene Magritte, “Not to be Reproduced” (1937).

Finally, concerning POETRY, I’ve always considered it astounding how the mastery in using words could evoke in the reader the very same emotions felt by the poet. Moreover, the same emotion could be characterized by different faces: just think of the typically human emotion of love, which could be described as romantic love, as in Dante’s sonnet “*Tanto Gentile e tanto onesta pare*”, or, contrarily, as a destructive love, as in Guido Cavalcanti’s sonnet “*Voi che per li occhi mi passaste ‘l core*”.

Emotions might be altered in some diseases, and this may lead to profound changes in personal life.

It is extremely interesting how emotional experience might be, selectively or not, impaired in some diseases. This change in emotional experience might, in turn, affect social, affective life and even artistic life. Just think of a couple of examples (I’ll skip Vincent van Gogh’s well known bipolar disorder since I’m more interested in citing lesser-known examples).

The poet Guillaume Apollinaire used to write his girlfriend passionate letters nearly every day before being wounded in the head, more precisely in the right temporal lobe, during the World War I, in 1916. As a result of this, his personality and emotional behavior changed dramatically and his affective relationships were deeply modified as well. The most significant example being his rapid disinterest in his fiancée. A few weeks after the brain injury, he went back to his literary writing. However, while his lyricism did not decline, his general tone became darker, more defiant and nostalgic, referring to “something lost”. On the contrary, no significant changes in cognitive ability were ever noted by Apollinaire’s contemporaries. The descriptions provided to us by his contemporaries are unanimous in telling that after this injury his personality changed drastically and its affective relationships were profoundly altered (Bogousslavsky, 2005).

Another important example is provided by Caspar David Friedrich, who is recognized as the most important German Romantic painter. He’s always been characterized by melancholic traits, very evident in his painting (Figure 3). However, his creativity had been substantially influenced by several episodes of major depression, during which he preferred techniques requiring only little manual effort, e.g. sepia and pencil drawing (he completely stopped painting in oils). Moreover, his drawings were filled with death symbols, i.e. graveyards, vultures sitting on spades, skeletons (Figure 3) (Dahlenburg and Spitzer, 2005).

Friedrich never tried to gain distance from his own mental and emotional condition. Rather, he resolved them in his art. “A painting is not to be invented, but is to be felt” was one of his maxims (Hinz, 1968).



Figure 3. Examples of Friedrich paintings. On the left side: “Wanderer Looking over a sea of fog” (1809/10) in which his melancholic personality is evident. On the right side: “Skeletons in a dripstone cave” (1826), painted during a depressive episode. The two paintings widely differ both in the color used and in the painting technique adopted.

Emotions guide our inner and social life, inspire every form of art, and, when altered, influence all individual life. **For these reasons, I’ve always been interested in emotions. This is the reason why the topic of my Ph.D. is emotions.**

Due to the extreme importance of emotion comprehension, affective neurosciences have focused on the understanding of how our brain perceives and reacts to emotional stimuli. Since its beginnings, affective neuroscience studies started to investigate the behavioral correlates and the neural basis underlying emotional faces observation. **For this reason, I chose to study emotions using faces as experimental stimuli.**

Moreover, since I’m not only a neurocognitive researcher, but I’m also a psychologist, I’m interested in disorders of affective regulation, above all those subclinical disorders which could interfere with the full understanding of one’s own and others emotions, which could, in turn, interfere with social relationships and with

psychological and physical health. **Thus, I chose to study the subclinical phenomenon called Alexithymia.**

CHAPTER 1

Emotions and embodiment

“Seeds are invisible. They remain dormant in the depth of the earth until one of them suddenly decides to wake up. (...). So it happens that there were some terrible seeds on the little prince’s planet... they were baobab seeds. The soil of the planet was infested with them. But if you intervene too late, you will never get rid of a baobab. It spreads over the entire planet. Its roots bore clear through it. And if the planet is too small and if there are too many baobabs, the planet explodes.”

The Little Prince
Antoine de Saint-Exupéry

In psychology and philosophy, an emotion is a subjective, conscious experience characterized primarily by psychophysiological expressions, biological reactions, and mental states.

In everyday life, emotions are initially like seeds, which are invisible. When the seeds grow, it is sometimes hard to unambiguously distinguish a rose shoot from a baobabshoot. *“If it is merely a sprig of rosebush it can be left to grow”*, explained the Little Prince to Antoine de Saint- Exupéry. Indeed, roses, like the positive emotions, make our planet more beautiful, i.e. our life. On the contrary, *“one must pull out the baobabs as soon as they can be distinguished from the rosebushes”*. Indeed, baobabs, like the negative emotions, might potentially cause our planet to explode. This is what happens daily in the world surrounding us. The individual’s inability to manage their own negative emotions, e.g. fear, rage, sadness, shame, frustration, drives them to commit terrible acts, violence, murders, etc.

Emotions trigger every reaction, every movement. Indeed, etymologically the term emotion derives from the Latin *e-movere*, meaning moving out. This is the reason

why it is important to understand how people understand their own and other's emotions. In the current chapter, different theories of emotions and the concept of emotional embodiment will be introduced. This chapter does not aim to review the existent literature, but only to introduce the theories of emotions and embodiment.

1.1 Different theories of emotions.

In many emotional states the heart races, the hands and face become warm, the palms sweat, and the stomach feels queasy. These sensations are the results of the activation of the autonomic nervous system, either the sympathetic nervous system, which generally activates the body for action, or the parasympathetic nervous system, which generally prepares the body to relax and recuperate.

Several theories have tried to explain the close ties between the subjective feelings of emotions and autonomic activity.

William James and Carl Lange suggested that the emotions we experience are caused by bodily changes. From this perspective, we experience fear because we perceive the activity that dangerous conditions trigger in our body (Fehr and Stern, 1970). In light of this theory, different emotions feel different because they are generated by a different constellation of physiological responses. James-Lange inspired many attempts to link specific emotions to specific bodily responses. These attempts mostly failed because it turns out that there is no distinctive autonomic pattern for each emotion. For instance, fear, surprise and anger, tend to be accompanied by sympathetic activation, while joy and sadness by parasympathetic.

Moreover, as the physiological reactions are rather slow, Walter Cannon and Philip Bard proposed that the brain decides which particular emotion is an appropriate

response to the stimuli (Cannon, 1927). Accordingly to this model, the brain simultaneously decides on the appropriate emotional experience and activates the autonomic nervous system to prepare the body. Thus they posited that our experience of emotion is independent of the simultaneous physiological changes that accompany it.

Following the Cannon-Bard theory, Stanley Schachter emphasized cognitive mechanisms in emotion and proposed the cognitive attribution model (Schachter, 1964). Within this model, emotional labels are attributed to relatively non specific physiological states. According to this model, our emotional experience results from cognitive analysis of the context around us, so that the physiological changes may accentuate emotions, but not specify which emotion we experience. In other words: which emotion we experience depends on cognitive systems that assess the context, i.e. our current social, physical and psychological situation.

In a famous experiment to verify this idea, people were injected with adrenaline and told either that there would be no effect or that their hearts would race (Schachter and Singer, 1962). Participants who were warned of the reaction reported no emotional experience, but some participants who were not forewarned experienced emotions when their bodies responded to the drug. However, which emotion was experienced could be affected by whether a confederate in the room acted angry or happy. The subjects injected with adrenaline were much more likely to report feeling angry when in the presence of an angry confederate, and more likely to feeling happy in the presence of an happy confederate. These findings clearly contradict the James-Lange prediction that emotions of anger and happiness should each be associated with unique profile of autonomic reactions. Schachter and Singer concluded that the misinformed subjects experienced their physiological arousal as whichever emotion seemed appropriate,

dependent on the environment. Thus, our emotional state is the result of an interaction between physiological arousal and cognitive interpretation of that arousal. Critically, this cognitive theory of emotions proposes that our interpretation of the context determines which emotion we'll experience.

Schachter's theory has its critics. For example, the theory asserts that physiological arousal is non-specific, affecting only the intensity of a perceived emotion but not its quality. However, when subjects were asked to adopt facial expressions distinctive for particular emotions, autonomic patterns of the subjects were different for several emotions, such as fear and sadness (Levenson et al. 1990). Indeed, positive emotions elicit a different array of autonomic responses than do negative emotions (Cacioppo et al. 1986), although, within those categories, different emotions elicit approximately the same autonomic profile.

1.2 The Embodiment of Emotions

Emotions are complex events involving a perceptual, visceral (i.e. heart frequency alterations) and somato-motor (i.e. smiling or frowning) experience. The above reported theories of emotions explained how one's own emotions processing happen. However, they lacked in explaining how the other's emotion processing happen.

Recent theories suggest that the processing of emotions expressed by others involves the re-experience, or simulation, of these relevant emotions component in one's self, referred to as "embodiment" (Niedenthal, 2007). Embodied simulation theories state that, since covert emotional states (e.g. happiness or fear) are often associated with overt motor behaviors (e.g. smiling or frowning), or covert emotion

related changes (e.g. increase in heart frequency), observers can automatically understand the covert emotional state of others by embodying their overt motor behavior or covert bodily changes (Gallese and Sinigaglia, 2011; Niedenthal et al. 2010; Bastiaansen et al. 2009; Oberman et al. 2007). Congruence between the recipient's and the sender's covert expression of emotion facilitates, whereas incongruence impairs, comprehension of the other's emotional state. Embodied simulation theories state that affective embodiment is intrusive in the observers' and determines a matching emotion, thus providing a direct way of "emotion understanding" (Gallese and Sinigaglia, 2011; Niedenthal et al. 2010; Bastiaansen et al. 2009; Oberman et al. 2007).

Thus, embodiment theories suggest that people understand other's emotions because they are able to embody their emotion in their selves. As a consequence, the ability to understand one's own emotion is critical for the comprehension of the emotion of others.

One example of emotional embodiment is the topic provided by the imitation of facial expressions (Dimberg, 1982, Bavelas et al. 1986). In Bavelas et al. (1982), for example, a confederate faked an injury and then frowned in pain. When participants observed the frown, they frowned themselves. Furthermore, the magnitude of participants' frowns increased with how clearly they could see the confederate's frown. Emotion imitation appears to be relatively automatic and to even be elicited outside awareness, as when participants react with slight smiles and frowns to subliminal happy and angry expressions (Dimber et al. 2000). Further evidence suggests that embodied consequences of subliminal facial expressions extend beyond these simulative rapid facial reactions. In one study, for example, participants were first subliminally exposed to happy or angry faces and were then asked to try a novel beverage. The results showed

that participants exposed to subliminal happy faces later behaved more in an approach-oriented fashion (by pouring and drinking more beverage) than subjects who were exposed to subliminal angry faces (Winkielman et al. 2005).

According to embodiment views, bodily responses should facilitate cognitive processing of emotion stimuli. Niedenthal et al. (2001) demonstrated that simulative rapid facial reactions play a causal role in the processing of emotional expression. Participants watched one facial expression morph into another and had to detect when the expression changed. Some participants were free to mimic, whereas others were prevented from mimicking by holding a pencil laterally between their lips and teeth. Consistent with the embodiment hypothesis, participants free to mimic the expressions detected the change in emotional expression earlier (more efficiently) for any facial expression than did participants who were prevented from mimicking the expressions.

In conclusion: the embodiment of emotions is considered to be a fundamental step in the process of emotion understanding. Although this phenomenon is now widely studied in normal populations, it is still under-investigated in sub clinical populations experiencing difficulties in emotional processing, such as alexithymia. In the following chapter, the construct of alexithymia will be explained.

CHAPTER 2

Alexithymia: emotional dumbness or numbness?

“If someone loves a flower of which there is only one in the millions and millions of stars, it is enough to make him happy when he looks at them for he can say to himself: <<My flower is somewhere out there...>>. But if the sheep eats the flower, it is for him as if, all of a sudden, all the stars went dark!”

The Little Prince
Antoine de Saint-Exupéry

“All the stars went dark”: this is the extraordinary way the Little Prince uses to describe how he would feel if he lost a friend. He has the terrific ability to use language to communicate his deeper feelings with others. The Little Prince has been able to communicate, using few words, a pervasive sadness, which is clearly perceived and felt by the reader.

Not all people have the mastery to put their feelings into words and for some individuals this task is especially daunting. There are some people who daily experience difficulty in communicating their own feelings. These individuals are called “Alexithymics”. Harvard psychiatrist Peter Sifneos first used the term alexithymia to describe individuals who appeared to have limited ability in emotion recognition, and who easily developed psychosomatic symptoms. The term Alexithymia derives from the Greek and is composed by the privative prefix a-, which means the lack of something, in this particular case the term refers to the absence of words (from the Greek λέξις = lexis) for emotions (from the Greek θυμός = thymos). Thus, the term alexithymia

etymologically means the absence of words for emotions, and denotes those people who are unable to describe and communicate to others their own emotions.

In the present chapter, a general overview of alexithymia construct and of the state of the art on alexithymia research is outlined. This should help in understanding the importance and the innovation of my thesis in the context of alexithymia research.

2.1 Historical hints:

The idea that emotions, in particular excessive and unmodulated levels of emotions, can adversely influence mental and bodily health has been expressed by physicians and philosophers since ancient times. In the Greco-Roman era, for example, Asclepiades attributed mental disorders to emotional disturbances: Galen classified the passions as the sixth of the non-natural causes of disease; Plato associated “erotic madness” with human love; and Cicero describes four main perturbations of passions (sorrow, fear, joy and libido), which could be moderate by reason, but could cause disease of the soul if they became excessive. During the nineteenth century, physicians began to approach in a scientific way the problem of how emotions, personality and health might be linked, by investigating the direct effects of various emotions on different bodily functions (Stainbrook, 1952). Beaumont, for example, demonstrated that heightened fear and anger reduce secretion from the gastric mucosa (Beaumont, 1933). From this pioneering study, scientists have always been fascinated by the way in which abnormal emotions are associated with higher vulnerability to diseases. Indeed, the majority of psychiatric diseases are characterized by the abnormal expression of at least one emotion. For example: anxiety disorders are characterized by a heightened manifestation of fear; obsessive compulsive disorder by a manifestation of both fear and

disgust; mood disorders by a manifestation of sadness (in depression) and euphoria (in bipolar disorder). Thus, the concept that abnormal and heightened emotional reaction might lead to mental (and organic) disorders is now well documented.

In this context, the concept of alexithymia is of particular innovation, since it denotes people with dampened emotional reactivity. For the first time, physicians considered the possibility that not only a heightened but also a weakened emotional experience might increase the vulnerability to illness. The clinical features of alexithymia were originally observed by [Ruesch \(1948\)](#), who described patients suffering from classical psychosomatic diseases or other chronic diseases that lacked imagination and manifested difficulties with the verbal and symbolic expression of emotions. [MacLean \(1949\)](#) also noted that many patients with psychosomatic disorders showed an apparent inability to verbalize feelings. He speculated that, in those patients, distressing emotions find immediate expression through autonomic pathways and are translated into a kind of “organ language” (the psychosomatic symptoms). [Horney \(1952\)](#) and [Kelman \(1952\)](#), subsequently described patients who were difficult to treat psychoanalytically because of a lack of emotional awareness, paucity of inner experiences, concreteness of thinking, and externalized style of living. Such patients were prone to developing psychosomatic symptoms. The term alexithymia was coined in the 1973 by [Sifneos \(Sifneos, 1973\)](#), who used it to describe the symptomatology often observed in psychosomatic patients.

2.2 The alexithymia construct:

During the past decade, the alexithymia construct has undergone theoretical refinement and empirical testing and has evolved into a potential new paradigm for

understanding the influence of emotions and personality on physical illness and health. Although the clinical features of alexithymia were originally described by [Sifneos \(1973\)](#) in patients with psychosomatic disorders, today alexithymia is not regarded as specific to those disorders but rather as a stable personality feature expressed with variable intensity in the general population ([Salminen et al. 1999](#); [Kokkonen et al. 2001](#)). The prevalence of alexithymia is expected in around 10% of the general population ([Taylor et al. 1991](#)). Moreover, there is evidence that alexithymia increases vulnerability not only to psychosomatic disorders, but to a broad range of psychiatric disorders, such as depression, schizophrenia, panic disorders, eating disorders, substance abuse, post-traumatic stress disorders and personality disorders ([Taylor et al. 1996, 1997](#); [Honkalampi et al. 2000](#); [Henry et al. 2010](#); [Todarello et al. 2005](#); [van't Wout et al. 2007](#); [Parker et al. 1993](#); [Marchesi et al. 2005](#); [Loas et al. 2000](#); [Bach et al. 1994](#); [Frewen et al. 2008](#)).

Classically, the construct of alexithymia has been defined to include multiple salient features ([Nemiah et al. 1976](#); [Taylor et al. 1991](#)):

- Difficulty in identifying emotions;
- Difficulty in describing and verbalizing emotions;
- Difficulty in distinguishing between emotions and bodily sensations of emotional arousal;
- Constricted imaginative processes as evidenced by a paucity of fantasies;
- Externally oriented thinking style, consisting in a tendency to focus on external events rather than inner experiences and to describe facts and actions without affective involvement;
- Poor empathizing.

At first glance, some so-called alexithymics appear to contradict this definition of the construct because they manifest, for example, outbursts of weeping or rage. Intensive questioning, however, reveals that they know very little about their own emotions and, in most instances, they are unable to link them with memories, fantasies or specific situations (Taylor, 1984).

It has been hypothesized that limited ability of the alexithymic individual to process emotions cognitively, so that these are experienced as conscious feelings states, leads to focusing on the somatic sensations accompanying emotional arousal. This is called the Somatosensory Amplification Hypothesis in alexithymia (Nyklicek & Vingerhoets, 2000; Nakao et al. 2002; Kano et al. 2007), which posited that alexithymia exhibits hyposensitivity to physical sensations associated with a tendency to rely on or to amplify physical symptoms. This might explain the apparent tendency of individuals who have alexithymic characteristics to develop hypochondriasis and somatization disorders (Demartini et al. 2014), as well as their alleged proneness to regulate tension through compulsive behaviours such as binge eating (Nowakowski et al. 2013), psychoactive substance abuse (Lyvers et al. 2013; Haviland et al. 1994; Taylor et al. 1990), or anorexia nervosa (Beadle et al. 2013; Torres et al. 2015).

2.3 Subtypes of Alexithymia

The term alexithymia does not refer to a single and unitary construct. Indeed, alexithymia could be primary or secondary. Moreover, individuals with a high level of alexithymia could mainly manifest affective or cognitive dimensions of alexithymia, thus leading to different alexithymia subtypes.

Primary vs Secondary Alexithymia

Alexithymia is considered to be primary when emerging “as a life-long dispositional factor that can lead to psychosomatic illness” (Lesser, 1981). Hence, primary alexithymia is currently thought of as a stable personality trait that becomes molded during childhood and early adult years (Messina et al. 2014). Therefore, primary alexithymia is developmental in nature. It has recently been suggested that genetic polymorphisms of the 5-HT transporter-linked promoter region (i.e. L/L alleles) (Kano et al. 2012) or of the Catechol O-methyltransferase (COMT) gene (i.e. Val/Met alleles) (Ham et al. 2005) may influence the occurrence of alexithymia.

Contrariwise, secondary alexithymia is posited to arise not during development, but as a consequence of events occurring later in life. These may be events with psychological significance and/or medical events (illnesses or diseases) that have a direct or indirect effect on brain functioning. Therefore, secondary alexithymia may have both psychological and /or somatic (organic) mechanisms (Wise et al. 1990; de Vente et al. 2006). For instance, alexithymia secondary to a psychologically significant event may be construed as a defense or protection against highly emotional events. This view is supported by the higher levels of alexithymia found in holocaust survivors (Yehuda et al. 1997) and sexual assault victims (Zeitlin et al. 1993). Similarly, alexithymia may occur *ex novo*, as a consequence of brain injury in regions supporting emotion processing and awareness (Williams and Wood, 2010). In this latter case, alexithymia is considered to be “organic”, and is not associated with a premorbid personality or with cognitive impairment. To date, there are a lot of studies investigating organic alexithymia and the regions involved seem to mainly be the anterior cingulated cortex (Sturm and Levenson, 2011; Paradiso et al. 2008); the frontal lobe (Becerra et al.

2002); the corpus callosum (TenHouten et al. 1986; Ernst et al. 1999); the basal ganglia (Huang et al. 2010) mainly in the right hemisphere (Spalletta et al. 2001). Finally, alexithymia might also emerge as a symptoms of neurological (i.e. multiple sclerosis, Bodini et al. 2008) and psychiatric (i.e. schizophrenia, Henry et al. 2010) disorders.

Thus, whereas primary alexithymia may play a role as a vulnerability factor for mental illness (Kooiman, 1998; Lumley et al. 1996), secondary alexithymia is thought to be a consequence of the illness (de Vente et al. 2006; Messina et al. 2014). This distinction is of fundamental importance for clinical practice. Indeed, if a clinician would like to prevent the insurgence of diseases, mainly somatization and conversion disorders, she/he must be able to recognize primary alexithymia features.

Critically, the present thesis contains 8 experiments, all of them but one conducted on individuals with primary alexithymia. Indeed, aspiring participants have not been enrolled if they had history of neurological or psychiatric diseases. Moreover, they have been asked for the occurrence in their life of major psychological traumatic events that might lead to alexithymia secondary to a psychologically significant event (i.e. sexual assault, road accident, etc). None of them declared to having experienced psychologically traumatic events.

Affective vs Cognitive Alexithymia

As previously reported, alexithymia is a multifaceted construct, characterized by different features. Whereas some researchers regard the cognitive, evaluative aspects of alexithymia as most important (Parker et al. 1993), others suggest that the fundamental deficit in alexithymia is a limited ability to consciously experience emotion (Lane et al. 1996). Bermond (1997) distinguished two main forms of alexithymia: type I and type II. Type I alexithymia is characterized by the absence of emotional experience and,

consequently, by the absence of the cognition accompanying the emotion. Type II alexithymia is characterized by a selective deficit in emotional cognition with scant emotional experience. Type I alexithymia is also called “affective” alexithymia, while type II alexithymia is also called “cognitive” alexithymia (Bagby et al. 2009). The affective dimension refers to the level of subjective emotional experience and mainly consists of the degree to which someone is emotionally aroused by emotion-inducing events. Contrarily, the cognitive dimension refers to the processing of emotions at a cognitive level and mainly consists of the degree of which an individual is able to identify, analyze and describe feelings.

In support of the existence of these two distinct forms of alexithymia, recent studies revealed that the two alexithymia dimensions may indeed be related to different gray matter volumes. It was shown that cognitive alexithymia might be more associated with larger insula volume (Goerlich-Dobre et al. 2013), while affective alexithymia seemed to be related to larger cingulated volume (Goerlich-Dobre et al. 2013). Thus, these different anatomical profiles underlying alexithymia suggested that alexithymia should not be regarded as a unitary construct.

Critically, the present thesis contains 7 experiments on alexithymic individuals. All of them have been included in the experiments based on the Toronto Alexithymia Scale scores (TAS-20, please see the paragraph 2.5 for details). This instrument is considered to mainly measure the cognitive features of alexithymia. Thus, the present participants are considered to belong to the cognitive alexithymia form.

2.4 Literature review:

As previously mentioned, alexithymia, which literally means “absence of words for emotions”, is classically considered to be an appraisal problem, in other words, a problem in the cognitive interpretation of emotional arousal (Taylor et al. 1991; Sifneos, 1973). Thus, the cognitive model of emotion that is most suitable to review alexithymia literature is the one by Schachter (Schachter, 1975, please see Chapter 1 for details). Simplifying, Schachter’s model of emotions could be represented by the **Figure 2.1**.



Figure.2.1. Schachter’s model of emotions. The stimulus perception leads to an autonomic arousal. However, it is the cognitive analysis of the situation that affects what emotions we experience. Thus, for instance, sympathetic activation may increase the intensity of the emotion we experience, but it does not determine which emotion we experience. The cognitive interpretation of autonomic arousal determines which emotion we experience. It is now possible to label the emotion.

In the following paragraphs, I will provide a review of alexithymia literature for each of these points, starting from the end of the diagram (labeling) and proceeding backwards. The reason why I decided to start from the labeling of emotion is that the term Alexithymia seems to suggest that the difficulties experienced by alexithymic individuals refer only to the last part of the process of emotion processing, i.e. the labeling of emotions. Moreover, the alexithymia construct has always been defined as an appraisal problem, due to a deficit in the cognitive processing of emotional arousal (Taylor et al. 1991, 1997; Taylor, 2010). Thus, if we consider the etymology of the term and the definition of the construct, one could suppose that the difficulties experienced by these individuals are mainly due to a deficit in the last boxes of the diagram. However, recent literature reveals that this is not the case, since alexithymic individuals

also manifest a deficit in early reactivity (Pollatos et al. 2008) and in emotional arousal (Neumann et al. 2004). Thus, I would like to proceed to review the literature following a chronological order.

2.4.1 Deficit in emotional word processing

Among human beings, the ability to express emotions requires processing at the linguistic level. Thus, linguistic processing plays a key role in alexithymia. Indeed, alexithymic people display impaired processing of emotional language at multiple levels. These have been the first symptoms of alexithymia noted by Nemiah and Sifneos (1970), giving the name to the construct.

At a basic, perceptual level, alexithymic people display poorer sensitivity to the emotional meanings of language. For instance, relative to low-alexithymia individuals, high-alexithymia individuals show less facilitation from priming emotional contexts on the processing of emotion words (Suslow and Junghanns, 2002). Alexithymic people are further impaired in the perception and processing of speech prosody with emotional content (Goerlich-Dobre et al. 2013).

At communicative level, alexithymic individuals further demonstrate problems in emotional language production and comprehension. In particular, alexithymic individuals display a limited ability to talk about interpersonal relationships (Meganck et al. 2009), describe other's emotional experiences (Bydlowski et al. 2005) and understand the emotions of others (Moriguchi et al. 2006; Swart et al. 2009). In personal narratives, alexithymic individuals tend to use vocabulary of limited complexity and their emotional discourse lacks any vivid descriptions (Meganck et al. 2009).

Alexithymia is also linked to concrete thinking and avoidance of metaphors (Kreitler, 2002).

2.4.2. Deficit in cognitive interpretations of emotions

People with alexithymia may be excessively focused on their own bodies, such that they notice otherwise common and benign sensations and they tend to experience emotions in a physical/bodily way. In either case, alexithymic people may focus on and amplify those bodily sensations associated with emotional arousal, thereby increasing sensation magnitude through a positive, autonomic feedback loop. Amplified sensations may be experienced as symptoms of physical illness, perhaps because of an attribution of sensations to biological rather than psychological or benign causes (Lumley et al. 1996).

Some studies reported that alexithymia was associated with being decoupled or discordant in one's physiological and subjective indices of arousal (Newton and Contrada, 1994; Papciak et al. 1985; Martin and Phil, 1986; Rabavilas, 1987). A pivotal study on this topic is the one conducted by Stone and Nielson (2001). These authors investigated the physiological state (heart rate and skin conductance responses) and the self-reported emotional intensity before and after exposure to an emotionally arousing videotape, in individuals with low and high level of alexithymia. Whereas the emotionally negative stimuli produced increased physiological responses in all subjects, the subjects with high alexithymia did not report increased subjective emotional intensity. Thus, the results revealed a dissociation in alexithymia between the physiological responses to negative stimuli, which are spared according to the authors, and the cognitive experiential component, i.e. the subjective experience of emotions,

which are impaired. According with this decoupling hypothesis (Papciak et al. 1985), alexithymia produces a level of physiological reaction to stressors similar to that seen in non alexithymia. However, alexithymic is characterized by a decreased ability to recognize physiological changes as emotions, and this results in prolonged exposure to stressors (Martin and Phil, 1985).

Moreover, a paper (Grabe et al. 2004) found that high score on difficulty in identifying emotions is particularly effective in predicting the development of somatization symptoms. These results support the hypothesis that the difficulty in identifying feelings in participants with high level of alexithymia is highly predictive of a broad range of somatization symptoms and this is likely due to their inability to understand the link between their emotion related bodily changes and the stimuli that caused them. As a consequence, they are more likely to misinterpret their visceral changes as a bodily disease, thus causing somatization.

2.4.3 Alteration in emotional arousal

There is evidence that alexithymia may be in part mediated by abnormalities in the autonomic state and reactivity, but the data are weak and contradictory (Taylor and Bagby, 2004). Indeed, affected people's physiologic reactions to emotional stimuli have been reported to be nil, increased or inhibited.

Intense arousal might be a causal factor in the development of alexithymia (Rabavilas, 1987). People with alexithymia may have unusually high baseline electrodermal reactivity (Stone and Nielson, 2001). Electrodermal responses to erotic images are higher than normal in people with difficulty in identifying and verbalizing emotions, and frightening images induce unusually strong responses in people with

reduced emotionalizing and fantasizing (Bermond et al. 2010). Alexithymia also predicts greater heart rate and blood pressure to stressors (Luminet et al. 2004; Waldstein et al. 2002). Moreover, significant correlation between alexithymia and the norepinephrine / cortisol ratio has been reported in men (Spitzer et al. 2005). Finally, recent studies found that people with alexithymia, rather than being tonically over-aroused, have a hyper-responsive sympathetic nervous system (Bodganov et al. 2013). Thus, these studies theorized an hyper-arousal in alexithymia, which probably originates in impaired cognitive control of emotions, particularly the negative ones.

However, these findings are contradicted by other studies suggesting either that alexithymia has no effect on stressor reactivity (Friedlander et al. 1997; Fukunishi et al. 1999), or that alexithymia predicts reduced stressor reactivity (Hyer et al. 1990; Linden et al. 1996; Neumann et al. 2004; Roedema & Simons, 1999; Wehmer et al. 1995). For instance, some studies found that people with alexithymia had blunted cardiac (Pollatos et al. 2008) and electrodermal (Wehmer et al. 1995) responses to emotion-evoking pictures. Interestingly, Pollatos et al. (2008) compared the electrodermal activity in response to masked, i.e. unconscious, emotional stimuli between alexithymic and non alexithymic participants and found that alexithymia was associated with smaller skin conductance responses to negative pictures. Moreover, they also manifest smaller skin conductance responses during speech preparation and speech (Pollatos et al. 2011). Thus, these studies theorized an hypo-arousal in alexithymia, which posits that dampened sympathetic nervous system activation and limited affective reactivity is associated with alexithymia during emotional provocation. This hypo-arousal might produce an emotional ambiguous context, which is highly difficult to interpret for people with high level of alexithymia.

Thus, although the literature produced contrasting results, the consistent data is that alexithymia is characterized by an alteration in autonomic responses. What is still matter of debate is the direction of this alteration.

2.4.4. Deficit in emotional perception

Although alexithymia has classically been defined as a difficulty to consciously understand and experience emotions (Taylor et al. 1991), recent studies hypothesized that hampered regulation of emotion in alexithymia might be based on deficits in the perception and further processing of emotional stimuli (Aleman, 2005; Lane et al., 2000). Furthermore, very few studies indicate that early perceptual-related processes are also altered in alexithymia. Schaefer et al. (2007) demonstrated that high alexithymic participants exhibited higher P1 (reflecting early visual processing, Liu et al. 2012) and N1 (reflecting difficulty of stimulus encoding, Nittono et al. 2007) amplitudes compared with low alexithymic when confronted with acoustic stimuli of increasing intensity. Further evidence for perceptual differences in alexithymia stems from research on anhedonia, a personality trait associated with a decrease in the ability to feel pleasure. Anhedonia is conceptualized as linked to alexithymia, an assumption which was partly supported empirically (e.g., Deborde et al., 2006; Loas et al., 1997, 1998). Franken et al. (2006) used a visual oddball paradigm and found that both early, middle and late ERP components of subjects with low levels of hedonic tone were attenuated compared with ERPs of subjects with high levels of hedonic tone. The authors suggested that decreased hedonic tone is associated with reductions in both automatic and effortful cognitive processing of relevant stimuli. In accordance to this finding Rey et al. (2010) reported that anhedonics experience less positive feelings when confronted with positive pictures

differing in luminance which might be associated with the perceptual encoding and emotional processing in anhedonia.

Recently, a new study provided new electrophysiological evidence for early processing deficits in response to emotional stimuli for alexithymic persons as mirrored in significantly reduced P1 amplitudes to emotional pictures (Pollatos and Gramann, 2011), which was driven by the affective features of alexithymia. P1 amplitudes systematically co-varied with P3 amplitudes supporting the assumption that deficits in early emotional processing contribute to later processing deficits. Importantly, the early deficits on a perceptual level precede the observed lack in cognitive and emotional capacities for modulating emotions in alexithymic persons (Vermeulen et al., 2006; Mantani et al., 2005; Moriguchi et al., 2006).

From the analysis of the existent literature, it is now clear that alexithymia should not be considered as emotional dumbness anymore, but needs to be considered as emotional numbness in all the emotional aspects.

2.4.5 Deficit in emotional embodiment

Although emotional embodiment is not included in Schachter's model of emotions, I've already underlined the importance of the embodiment of emotions in the modern emotional theories (please refer to Chapter 1, paragraph 1.2 for details).

To the best of my knowledge, nothing has yet been published on the ability of alexithymic individuals to embody other's emotions. In particular, to date only one study on alexithymia, which used imaging techniques, marginally dealt with this topic (Moriguchi et al. 2009). This study investigated the activity of the mirror neuron system

network in alexithymia by means of functional magnetic image analysis. As widely known, the mirror system is considered crucial for human imitation (Cattaneo and Rizzolatti, 2009; Rizzolatti et al.2009), thus it is involved in the embodiment of the somato-motor features of emotions. The study revealed a greater activation of the mirror neuron system in participants with high level of alexithymia compared with those with low level of alexithymia in response to a classic task, i.e. the passive observation of video depicting goal directed movement. However, without a behavioral performance, it is impossible to understand if this observed hyper-activation reflected a better functioning of this network in alexithymia or, instead, was related to a greater difficulty for alexithymia in understanding the task, such that a greater activation was needed for those subjects to understand what was going on.

Thus, is it clear that the emotional embodiment is a topic still highly under-investigated in alexithymia research.

2.5 The subject's selection problem:

The research on alexithymia, although still at its beginning, is characterized by the presence of contrasting results. For instance, alexithymia has been found to be coupled with a poor visceral reactivity to emotional stimuli in some studies, and with increased bodily reactions to emotional stimuli in other studies. These results allow the researcher to advance the hypo-arousal hypothesis (Neumann et al. 2004) and the hyper-arousal hypothesis (Waldstein et al. 2002), respectively.

One possible reason that could account for these contrasting results is the diversity in the participant's selection across studies. Indeed, both the questionnaires/semi-

structural interviews used to identify alexithymic individuals, and the cut-off chosen for each instrument varied enormously across studies.

In the current paragraph the procedure used in the following experiments in order to correctly identify high and low alexithymia participants is widely explained.

First of all, a large number of individuals were screened using the 20-item Toronto Alexithymia Scale (TAS-20; Taylor et al. 2003). The TAS is the main instrument used in alexithymia research and is a self-administered questionnaire that provided a reliable (Taylor et al. 2003; Parker et al. 2003), valid (Taylor et al. 2003; Parker et al. 2003) and common metric to measure alexithymia. There are now evidences that the TAS-20 demonstrates reliability and factorial validity in many different languages and cultures (Taylor et al. 2003), which enables comparisons and a greater generalizability of findings from studies in different countries.

The items are scored on a 5-point scale from strongly disagree to strongly agree. The TAS-20 has a 3-factor structure. Factor 1 assesses the difficulty in identifying feelings (DIF) (example: “When I am upset, I don’t know if I’m sad, frightened or angry”). Factor 2 assesses the difficulty in describing feelings (DDF) (example: “It is difficult for me to find the right words for my feelings”). Factor 3 assesses externally oriented thinking (EOT) (example: “I prefer to analyze problems rather than just describe them”). Although the TAS-20 is consider an instrument that mainly measures the cognitive features of alexithymia compared with other instruments, within the three subscales the first two factors, i.e. DIF and DDF, correspond to the reduced emotional awareness aspect of the alexithymia construct (Nemiah et al. 1976; Nemiah and Sifneos, 1970) and reflected the so called “affective” features of alexithymia. The third factor,

i.e. EOT, corresponding to the *pensée opératoire*, thus reflecting the “cognitive” features of alexithymia (Taylor et al. 2004).

Importantly, in the following experiments, the TAS-20 cut-off for high alexithymia was considered to be 61 (i.e. >60), reflecting the top quartile of the normal distributions, while the cut-off for low alexithymia was considered to be 38 (i.e. <39), reflecting the bottom quartile of the normal distribution. These cut-offs, previously suggested in literature (Taylor et al. 2003) were selected in order to obtain a sample with as large a variance on alexithymia as possible. Notably, these cut-offs were not unanimously used in alexithymia research. Indeed, some researchers used the score of 51 as cutoff for high alexithymia (e.g. Pollatos and Gramann, 2011; Franz et al. 2004) or for low alexithymia (e.g. Borsci et al. 2009), thus reducing the differences of scores between groups, and increasing the likelihood of including low alexithymia participants in the high alexithymia group and vice versa.

Despite its wide use in alexithymia research, TAS-20 has two main limitations.

1. First of all, the TAS-20 uses self-assessment, thus its validity may be attenuated in groups characterized by reduced insight into their psychological functioning. To cope with this first limitation, in alexithymia research the use of a semi-structured interview is often associated to the TAS-20 score in order to confirm the presence of this personality trait.

2. The second main limitation is that TAS-20 investigates only three dimensions of the alexithymia construct, i.e. the difficulty in identifying and describing feelings and the externally oriented cognitive style. To cope with this second limitation, an instrument investigating different factors of alexithymia should be used. Although the Toronto Semi-structured Interview

for Alexithymia (TSIA) has recently been validated in Italian (Caretti et al. 2011), this has not been used for the following experiments since it shares the second limitation with the TAS-20. Instead, the Alexithymia module of the Diagnostic Criteria for Psychosomatic Research (DCPR) (Mangelli et al. 2006) was selected among the existing semi-structured interviews for alexithymia.

The alexithymia module of DCPR is a semi-structured interview which assesses alexithymia through six items concerning those characteristics most frequently encountered in the description of alexithymia in the psychosomatic literature: 1) difficulty in describing feelings; 2) tendency to speak about feelings; 3) impoverished fantasy life; 4) externally oriented thinking; 5) the ability to associate bodily arousal with emotions; 6) sporadic, sudden abnormal reactions to stressful situations. Critically, questions are scored based on both verbal and nonverbal information provided by participants. Moreover, the investigator might ask the participants to provide additional explanation or some examples in order to further explore a participant's answers. Participants were included in the studies if their scores on TAS-20 and DCPR were congruent, i.e. if they manifest high level of alexithymia in both the TAS-20 and in the DCPR.

Moreover, due to the high association between alexithymia and depression (Allen et al. 2011; Honkalampi et al. 2000; Hintikka et al. 2001), the Italian version of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al. 1997), mood disorders subscale, was used in order to exclude participants with clinical relevant depression.

Finally, to further investigate the participants' emotional profile, they were asked to respond to three self-reporting questionnaires: the Emotional Inhibition Scale (EIS; Grandi et al. 2011), the Interpersonal Reactivity Index (IRI; Davis, 1983), and the Body Perception Questionnaire (BPQ; Porges, 1993).

The diagram represented in **Figure 2.2** provides a graphical representation of the participants' inclusion procedure.

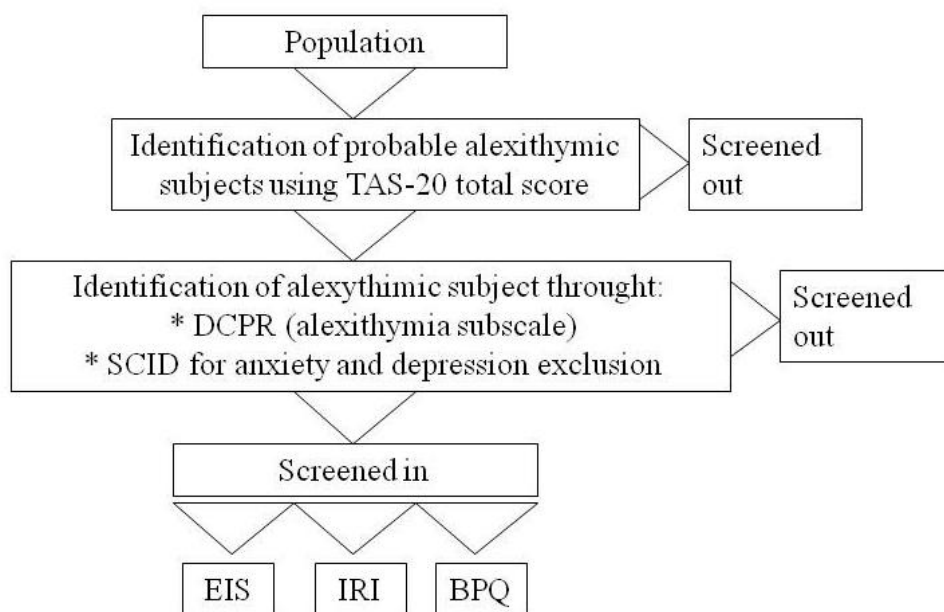


Figure 2.2. Participants' inclusion procedure. TAS-20: 20-items Toronto Alexithymia Scale (Taylor et al. 2003); DCPR: Diagnostic Criteria in Psychosomatic Research (Mangelli et al. 2006); SCID: Structured Clinical Interview for DSM-IV Axis I Disorders (First et al. 1997); EIS: Emotional Inhibition Scale (Grandi et al. 2011); IRI: Interpersonal Reactivity Index (Davis, 1983), BPQ: Body Perception Questionnaire (Porges, 1993).

2.6 Conclusion

To date, the research on alexithymia has been mainly focused on verbal expression and cognitive interpretation of emotions. In the last few years only, studies began to explore the emotional arousal and the perception of the emotional features of the stimuli. However, the ability of alexithymic individuals in the embodiment of emotions in a topic still highly under-investigated. The emotional embodiment is considered a process that involves the re-experience, or simulation, of the relevant component of emotion's expressed by others (i.e. somatic, motor, visceral) in one's self (Niedenthal et al. 2005, 2010; Niedenthal, 2007; Halberstadt et al. 2009). In other words, embodiment theories suggest that the perceiver is able to understand other's emotions by simulating/re-experiencing/"embodying" other's emotion in its own body. Thus, the understanding of the emotions of others is tightly linked to the understanding of one's own emotions.

Since the embodiment of emotion is necessary to the understanding of the emotional state of others, and thus for correct socialization, in the current thesis, the embodiment abilities of alexithymic subjects have been explored. This topic was investigated in 6 experiments comparing the performance of individuals having high and low level of alexithymia in tasks using highly homogeneous stimuli, i.e. emotional faces, to remove a possible confound on the results due to the heterogeneity of the stimuli.

First, the somatic embodiment was explored, by investigating the ability of high and low alexithymia subjects to remap the emotions of others onto their own self. To this end, the Visual Remapping of Touch paradigm has been used (Serino et al. 2008), a multisensory stimulation paradigm in which seeing a face being touched improves

detection of near-threshold tactile stimulation concurrently delivered to one's own face. In a normal population, the VRT effect is modulated by the emotion of fear, so that seeing a fearful faces induces an enhanced VRT effect. In *Experiment 1*, the VRT performance of high and low alexithymia individuals while observing fearful, happy or neutral faces have been compared. The results revealed that, while the participants with low alexithymia showed the fear specific enhancement of the VRT effect, this fear modulation is completely absent in high alexithymic individuals. Thus, alexithymic participants seem to be unable to remap the emotion of fear conveyed by faces. Critically, this impairment seems to be specific for fear. Indeed, in *Experiment 5* using the same paradigm, the results revealed that the VRT effect is enhanced in alexithymic participants while seeing disgusted faces, while this disgust induced modulation is absent in low alexithymic participants. It is worth noting that in *Experiment 5*, the emotion of disgust has been chosen amongst the negative emotion instead of sadness and anger. The reason for this choice istwofold. First of all, disgust shares with fear not only the negative valence, but also the threat related connotation. Despite these similarities, disgust and fear could be considered very different emotions based on their physiological underpinning (see Chapter 5 for further details). Disgust and fear are both specular emotions, i.e. seeing a disgusted/fearful expression will induce an emotion of disgust/fear, while anger is a reactive emotion, i.e. seeing an angry expression will induce an emotion of fear (ref). This is the reason why the emotion of anger has been *a priori* excluded from *Experiment 5*. Moreover, it is well known that the expression of sadness produces less arousal compared with the other negative emotions (for example, see [Balconi et al. 2014](#)), and for this reason, the emotion of sadness has been *a priori* excluded from the experiment. These findings cannot be the results of variables other

than emotion processing abilities. Indeed, in *Experiment 2*, high and low alexithymia individuals were administered a comprehensive neuropsychological battery to explore their cognitive abilities, and the two groups did not differ in any of them.

Secondly, the motor embodiment was explored, by investigating the emotional resonance expressed through the congruent muscle activation in response to affective facial displays. To this aim, in *Experiment 3*, the high and low alexithymia participants' rapid facial reactions (RFR) to emotional faces have been compared. The electromyographic activities in *Corrugator Supercilii* and *Zygomaticus Major* were recorded while participants were performing an emotion-unrelated task (gender classification) on facial displays of various affective states. Low alexithymia participants showed congruent RFRs in response to both fearful and happy. On the contrary, high alexithymia participants showed absent congruent RFRs in response to fearful faces and congruent, but delayed, RFRs in response to happy faces.

Moreover, to better investigate a possible relationship between the alexithymic behavioral patterns that emerged during the embodiment tasks and physiological responses, in *Experiment 4* and *6* physiological measures were analyzed. In particular, in *Experiment 4*, the heart frequency has been recorded, to investigate whether the between groups differences in RFRs might be partially explained by a reduced emotion related heart frequency variability. The results revealed that low alexithymia participants showed a fear related bradychardia, which is absent in the high alexithymic individuals. These results are consistent with the "hypo-arousal theory" of alexithymia. Moreover, given that the emotion of disgust is the emotion that mainly induces visceral/bodily changes, in *Experiment 6*, participants' interoceptive abilities, i.e. the abilities to respond to stimuli originating from inside the body, were investigated in

order to explore the possibility that the dissociation between the embodiment of fear and disgust revealed in *Experiment 5* might be related to the tendency of alexithymic to be mainly focused on their own bodily signals. The findings seem to support this hypothesis.

Experiments 1, 3 and 5 provided convergent evidences in support of the hypothesis of abnormalities in the negative emotional embodiment in alexithymic participants. Indeed, in these individuals, the embodiment of happiness is nearly spared (please see *Experiments 1 and 3*), while the embodiment of negative emotions is shown to be insufficient in the case of fear (please see *Experiments 1, 3, 4 and 5*) and abnormally high in the case of disgust (please see *Experiment 5*), compared with the embodiment patterns showed by non alexithymic participants.

However, the data on the emotion of disgust are only preliminary. On the contrary, the data on the expression of fear are very consistent across experiments. Is it possible to speculate that alexithymic difficulties in the embodiment of fear might be due to reduced amygdalar modulation in response of a fearful expression (Friedman et al, 1986; Sah and Lopez De Armentia, 2003; Höistad and Barbas, 2008). In fact, the amygdala has been reported to play a key role in the early stage of facial expression processing, particularly for fearful expressions (Breiter et al. 1996; Morris et al. 1998; Calder et al. 2001; Zald, 2003; Schultz, 2005).

To investigate whether the amygdala could account not only for the early stage of facial expression processing, but also for the embodiment of emotions, in *Experiment 7* the performance of patients with amygdalar lesions, patients with extra-temporal lesions and healthy participants at the emotional VRT tasks were compared. While control patients and healthy participants showed the typical enhanced VRT for fearful faces

compared to other faces, in patients with lesions to the amygdala the VRT for fearful faces was disrupted, suggesting that the eVRT effect for fear relies on the activity of the amygdala. Hence, the behavioral performance at the eVRT of patients with amygdalar lesions mirrors the one of high alexithymia participants since in both the experimental groups the embodiment/remapping of the expression of fear is absent.

Although the reported findings agreed in supporting the hypothesis that alexithymia is characterized by abnormalities in the embodiment of the emotional features conveyed by faces, is still unknown how alexithymic individuals encode the perceptual features of the emotional stimuli. Preliminary evidences revealed that the perceptual encoding might be impaired as well (Pollatos and Gramann, 2011). However, specific studies on emotional faces are still missing. To fill this gap, a last experiment was conducted in which the amplitude of the n170 wave, particularly evident during the perceptual encoding of faces, has been measured in low and high alexithymic individuals to investigate if the well known emotional modulation of the n170 amplitude (Pizzagalli et al. 2002; Batty and Taylor, 2003; Ashely et al. 2004; Blau et al. 2007) is evident also in alexithymia (*Experiment 8*). Results revealed that the emotion of fear modulated the n170 amplitude in low alexithymia but not high alexithymia participants.

In conclusion: the results of the current thesis provided convergent results supporting the hypothesis of a difficulty of alexithymic participants in embodying the emotion of fear, whose perceptual encoding is also defective. Preliminary but less conclusive data were also provided for the emotion of disgust. Further studies will

explore the relationship between early processing deficit (i.e. for instance the n170) and embodiment.

CHAPTER 3

In touch with emotions

“<<If you tame me, my life will be full of sunshine. (...) And look yonder! Do you see the cornfields? I do not eat bread. Wheat is of no use to me. Those cornfields don't remind me of anything. But you have hair the color of gold. Wheat, which is also golden, will remind me of you. And I shall love the sound of the wind in the wheat.>> (..) Thus the Little Prince tamed the fox. And when the time came for his departure the fox said: <<Oh... I shall cry>> (...) << It has helped me because of the color of the wheat fields>>.”

The Little Prince
Antoine de Saint-Exupéry

Here the wonderful character of the Fox refers to some senses to express emotions. She speaks about the *vision* of the color of the wheat and the *sound* of the wind. Given the similarities between the color of the wheat and the color of the Little Prince's hair, the wheat will remind her of the Little Prince. The Fox uses multiple senses to express strong emotions: she shall cry because of the color of the wheat fields. In the present chapter, an experimental paradigm involving multiple senses, vision and touch, has been used to investigate the emotional embodiment in alexithymia.

3.1 Introduction

Although alexithymia has been classically defined as a difficulty in understanding and describing one's own emotions (McDonald and Prkachin, 1990; Taylor et al. 1991; Mantani et al. 2005; Herbert et al. 2011), some recent studies on alexithymia also demonstrate difficulties in recognizing the emotions of others, both in direct tasks, in which participants are explicitly asked to recognize emotions (e.g. McDonald and

Prkachin, 1990; Vermeulen et al. 2006; Pollatos et al. 2008; Swart et al. 2009; Lee et al. 2011), and indirect tasks, in which emotional information is present in the stimuli but irrelevant for performing the task (Kano et al. 2003; Mueller et al. 2006).

It is worth noting that impaired direct emotion recognition in alexithymia has only been found using difficult tasks (e.g. recognition of micro-expression, matching verbal and nonverbal material, recognition of emotion with temporal constrain or evaluation of emotional scenes), which are sensitive enough to detect subtle differences between groups (Prkachin et al. 2009; Lane et al. 1996; Parker et al. 2005; Pollatos et al. 2008). In contrast, when the task is easy, no differences in direct emotion recognition have been revealed between subjects with high and low alexithymia scores (Grynberg et al. 2012; Kano et al. 2003; Lee et al. 2011; Heinzl et al. 2010; Meriau et al. 2006; Pollatos and Gramann, 2011; Pollatos et al. 2008).

What is still unknown is whether alexithymic subjects have the ability to refer what they see expressed on the faces of others to their own face, i.e. the ability to remap the emotion of others onto the self.

In order to understand other peoples' feelings, observers implicitly match what they see expressed on the faces of others with face-dependent traces associated with personal experience of the same emotion (Carr et al. 2003; Niedenthal, 2007). Thus, the observers re-experience, through their own sensory systems, the observed emotion, simulating how the other individual would feel when displaying that facial expression (Damasio, 1994; Adolphs et al. 2000). Accordingly, previous studies found that our ability to understand the emotions of others relies on the same neuronal network underlying our capacity to understand our own feelings (Preston and de Waal, 2002; Singer et al. 2004, 2009), i.e., somatosensory, insular and anterior cingulate cortices and

the amygdalae (Carr et al. 2003; Wicker et al. 2003; Singer et al. 2004; Jabbi et al. 2007). Hence, in alexithymia (which is defined as a deficit in identifying emotional states in oneself) the difficulty in representing others' emotions could result from a difficulty in representing one's own emotions.

The aim of *Experiment 1* was to investigate the ability of alexithymic subjects to remap the emotional expressions of others onto their own somatosensory systems using an indirect task. To this end, we used a multisensory paradigm called Visual Remapping of Touch (VRT; Serino et al. 2008), in which seeing a face being touched, compared to a face merely approached by fingers, improves detection of near-threshold tactile stimulation concurrently delivered to one's own face. The VRT effect is due to increased activity in the somatosensory cortices (Cardini et al. 2011; Malacuso et al. 2005), which are responsible for processing tactile information (Malacuso et al. 2005). In fact, viewing someone being touched activates brain regions normally recruited during tactile perception (Ebisch et al. 2008; Keysers et al. 2004). This overlap of brain activity for perceiving and viewing touch has been taken as an evidence for a neural mechanism remapping tactile sensation seen on the body of others onto one's own somatosensory system (Cardini et al. 2011; Blakemore et al. 2005; Ebisch et al. 2008).

Critically, in VRT, the performance of subjects is influenced by the emotional content of the stimuli (Cardini et al. 2012): tactile perception is enhanced when viewing touch on a fearful face compared with viewing touch on neutral and happy faces. The enhancement of tactile perception in the emotional VRT when viewing fearful faces has been interpreted as a preliminary activation of the somatosensory system in response to threat (Cardini et al. 2012); the more efficient remapping of fear onto the self could have an adaptive role in alerting the observer by signaling the presence of a potential

threat in the environment and, as a consequence, facilitating preparation of a defensive response (Cardini et al. 2012).

If alexithymic subjects have difficulties in remapping the emotions of others onto the self, particularly the negative ones, we would predict that subjects with high alexithymia levels should not show the expected enhancement of tactile acuity in emotional VRT when they see a fearful face. In contrast, the effect should manifest itself in the subjects with low alexithymia levels.

In a separate experiment, participants were also asked to directly rate the emotional stimuli on the dimensions of arousal and valence to make sure that eVRT task performance was not impaired due to a deficit in explicit emotion recognition. Since this task is very simple and the emotions expressed by the stimuli are easy to recognize, we expected similar ratings of both valence and arousal between the two groups, in accordance with previous studies that used the same rating task (Aftanas et al., 2003; Aftanas and Varlamov, 2007; Karlsson et al. 2008; Kugel et al. 2008; Recker et al. 2010; Heinzl et al. 2010; Pollatos and Gramann, 2011).

3.2. EXPERIMENT 1: Emotional modulation of touch in Alexithymia

3.2.1 Material and Methods

Participants:

Participants have been selected using the procedure described in Chapter 2, paragraph 2.5. Briefly, 280 university students were screened for alexithymia using the 20-item Toronto Alexithymia Scale (TAS-20; Taylor et al. 2003). Individuals with high and low TAS-20 total scores (n=18, top quartile score >60; n=20, bottom quartile score <39) were selected in order to obtain a sample with as large a variance of alexithymia as

possible. The alexithymia module of the structured interview for the Diagnostic Criteria for Psychosomatic Research (DCPR) (Porcelli and Sonino, 2007; Mangelli et al. 2006; Porcelli & Rafanelli, 2010), previously used in alexithymia research (Grandi et al. 2011), was used to further confirm the presence or absence of alexithymia. Moreover, due to the high association between alexithymia and depression (Allen et al. 2011; Honkalampi et al. 2000; Hintikka et al. 2001), the Italian version of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al. 1997), mood disorders subscale, was used in order 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 TAS-20 and DPCR were congruent. One participant with a high TAS-20 score and a low DCPR score was discarded, as well as a participant with a high TAS-20 score who had a previous diagnosis of bipolar disorder. *Table 3.1* gives comparative information about the resulting low alexithymia (LA; n=20) and high alexithymia (HA; n=16) groups. All participants had equivalent educational backgrounds and were students at the University of Bologna.

The intention of this work was also to characterize the emotional and cognitive profiles of HA and LA subjects. To do that, the included participants were asked to respond to three self-report questionnaires: the Emotional Inhibition Scale (EIS; Kellner, 1987; Grandi et al. 2011), the Interpersonal Reactivity Index (IRI; Davis, 1983), and the Body Perception Questionnaire (BPQ; Porges, 1993).

	Whole	LA	HA
n (male/female)	36 (11/25)	20 (5/15)	16 (6/10)
Age, mean (SD) (years)	23.19 (1.83)	23.4 (1.42)	22.93 (2.26)
<i>TAS-20</i>	<i>Minimum-maximum, mean (SD)</i>		
Total	24-80, 47.13 (16.22)	24-39, 33.4 (4.47)	61-80, 64.31 (4.75)
F1	5-27, 13.19 (6.5)	5-11, 7.8 (1.7)	16-27, 19.93 (2.86)
F2	7-32, 16.91 (7.19)	7-15, 11.3 (2.12)	15-32, 23.93 (4.5)
F3	9-26, 17.02 (4.51)	9-21, 14.3 (3.16)	12-26, 30.43 (3.53)
DPCR	0-5, 2.83 (1.41)	0-2, 1.25 (0.63)	3-5, 3.81 (0.54)

Table 3.1. Demographic and alexithymia profile of the whole sample, low and high alexithymia groups. TAS-20: twenty-item Toronto Alexithymia Scale; F1 (factor 1): difficulty in identifying feelings; F2 (factor 2): difficulty in describing feelings; F3 (factor 3): externally oriented thinking. SD: standard deviation. Low Alexithymia (n=20) and High Alexithymia (n=16) groups were obtained excluding the participants with discrepancy between TAS-20 and DPCR scores (cf. Material and Methods). DPCR: Alexithymia Module of Diagnostic Criteria for Psychosomatic Research scores.

Emotional VRT: To test the ability of participants to remap others' emotions onto the neural systems that generate the same emotional expressions, we used an indirect task: the Emotional Visual Remapping of Touch (eVRT, [Cardini et al. 2012](#)).

Stimuli: The eVRT is a multisensory integration paradigm consisting of both tactile and visual stimuli. *Tactile stimuli* were delivered by 2 constant current electrical stimulators (DS7A, Digitimer), via 2 pairs of neurological electrodes (Neuroline, AMBU) placed on the participant's right and left cheeks. For half of the LA group and half of the HA group, the tactile stimulus on the left cheek was set to be more intense than that on the right cheek, and vice-versa for the other half. *Visual stimuli* consisted of video trials presented on a 17" computer screen placed in front of the participant at a distance of about 60 cm. Faces with neutral, fearful or happy expressions were presented in three different blocks. The faces were static black-and-white pictures selected from the Pictures of Facial Affect (PFA) database ([P. Eckman and W.V.](#)

Friesen, Consulting Psychologists Press, Palo Alto, CA, 1976). Overall, 3 different actors were used for each expression.

Procedures: Prior to the experiment, each participant looked at static, neutral faces not included in the main experiment while the intensity of the electrical stimuli was calibrated with a staircase procedure to a threshold detection rate of 100% for the stronger stimulus and 60% for the weaker. As a confirmation of correct calibration of the stimuli, the mean accuracy for unilateral strong and unilateral weak stimuli was $93.4\% \pm 1.1\%$ (mean \pm s.e.m.) and $61.4\% \pm 3.3\%$ respectively (paired sample t-test: $p < 0.001$) ($93.9\% \pm 1.3\%$ and $63\% \pm 5\%$ in HA and $92.9\% \pm 1.7\%$ and $60.1\% \pm 4.3\%$ in LA; independent sample t-test: $p=0.68$ and $p=0.67$ for stronger and weaker stimuli, respectively). This stimulus calibration results in a tendency for participants to fail to report the weaker stimulus during trials with bilateral stimulation. Mean accuracy for bilateral tactile detection was $52\% \pm 2.5\%$ ($51.2\% \pm 3.9\%$ in HA group and $52.5\% \pm 3.3\%$ in LA group, independent sample t-test: $p=0.79$). Errors consisted mostly of reporting the side of the stronger stimulus: the mean probability of reporting the side of the stronger stimulus in the case of errors during bilateral stimulation was $95.4\% \pm 1\%$ ($94.6\% \pm 1.8\%$ in HA group and $96\% \pm 1.1\%$ in LA group, independent sample t-test: $p=0.48$).

The faces were presented as a central, static image in the background of the movie. In the foreground, two fingers were initially positioned on the lower part of the screen, one on the right and one on the left side. During the movie the fingers moved towards the face and then returned back to their initial position. In different trials, the finger-motion followed one of two trajectories: in the Touch condition, the fingers touched the cheeks of the shown face, in the same position where tactile stimulation on

the participants' cheeks was administered. In the No-Touch condition, the fingers stopped about 5 cm alongside the face (see *Figure 3.1*). In different trials, either the finger on the right, on the left or both fingers moved. Visual and tactile stimuli were synchronized so that when the fingers reached the peak of their trajectory a tactile input was delivered to the participant's face. Each movie lasted a total of 2700 ms, and tactile stimulation was delivered at 750 ms from movie onset.

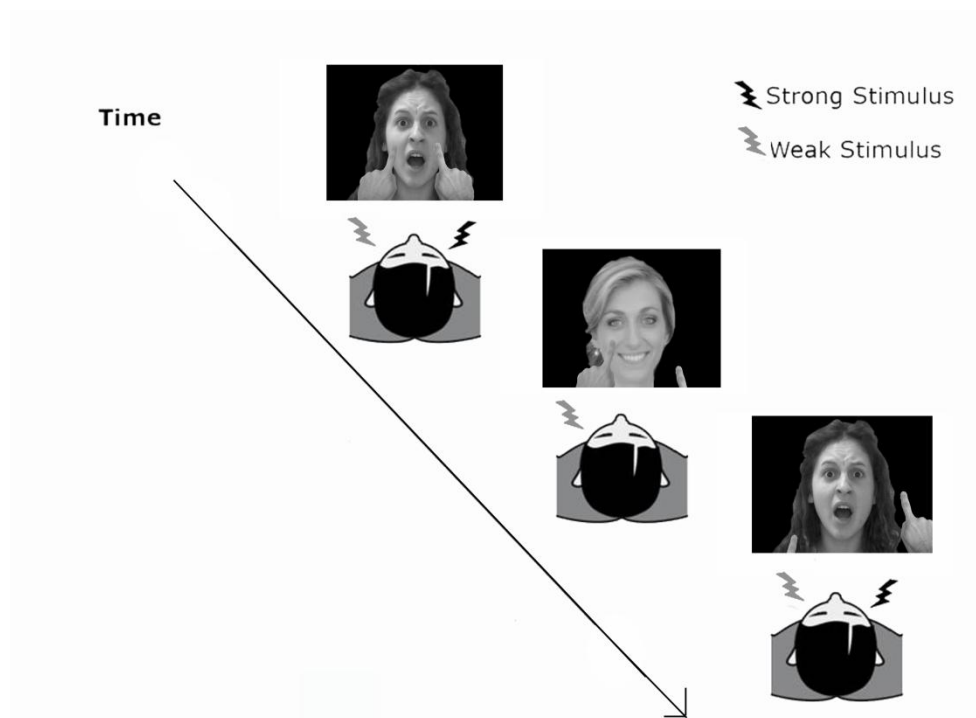


Figure 3.1. Experimental paradigm. All participants performed 6 randomized blocks of the tactile confrontation task, lasting ~ 5 min each. Three different actors for each emotion (fear, happy, neutral) were presented in the movie. One emotion (fear, happiness or neutral) was presented in each block. In different trials fingers moved towards the image and then backwards to their starting position. Fingers either touched the cheeks of the shown face or stopped about 5 cm alongside the face. In different trials, either the finger on the right, on the left or both fingers moved. As soon as the fingers reached the image, a tactile input (either the weak, the strong or both stimuli) was delivered on the participant's cheeks.

A PC running C.I.R.O. software (www.cnc.unibo.psice.unibo/ciro) was used to control the presentation of the stimuli and record the responses. Each experiment consisted of 6 counterbalanced experimental blocks of the tactile confrontation tasks,

two blocks for each facial emotion presented in the video trials. Stimuli comprised a combination of the two types of tactile stimulation (unilateral left or right and bilateral), the two types of visual stimulation (unilateral left or right and bilateral) and the two finger movement trajectories (Touch and No-Touch). Each block consisted of 48 trials, presented in a random order. A pause of 3 minutes, during which tactile thresholds were recalibrated, was interposed between blocks.

Direct Task: The direct emotion recognition task consisted of arousal and valence ratings of emotional facial expressions. The very same Ekman faces used in the eVRT were presented to participants: three different actors expressing fear, happiness and a neutral expression. Participants were requested to rate arousal and valence using a nine point Likert scale, with 1 meaning “not very arousing” or “very negative emotion” and 9 meaning “highly arousing” or “very positive emotion.”

3.2.2 Results

Self Reported questionnaire. Table 3.2 summarizes the differences between the two groups in these questionnaires. Regarding *emotion management*, revealed by EIS, HA groups had higher scores on the EIS verbal inhibition and disguise of feelings subscales, but not on the timidity and self-control subscales. These data are in line with previous literature (Grandi et al. 2011) and with the definition of the construct of alexithymia as an “absence of words for feelings” (Sifneos, 1973; Nemiah et al. 1976; Taylor et al. 1991). Interestingly, given the different scores between the HA and LA groups in two out of the four EIS subscales, our results pointed out that alexithymia and emotional inhibition are only partially overlapping constructs.

	Low Alexithymia	High Alexithymia	t	p
EIS total score	25.25 (5.72)	38.43 (6.62)	-6.409	<0.001*
EIS verbal inhibition	4.5 (2.11)	9.43 (2.85)	-5.967	<0.001*
EIS timidity	6.3 (2.22)	7.93 (2.97)	-1.889	0.067
EIS disguise of feelings	5 (2.33)	11.21 (2.49)	-7.81	<0.001*
EIS self-control	9.45 (2.06)	9.75 (2.74)	-0.374	0.710
IRI total score	71.7 (11.93)	67.12 (11.87)	1.145	0.260
IRI empathy concern	20.65 (4.9)	18.68 (3.3)	1.370	0.180
IRI fantasy	18.65 (4.35)	18.5 (5.36)	0.093	0.927
IRI perspective taking	20.95 (4.57)	15.37 (4.82)	3.547	0.001*
IRI personal distress	11.45 (4.12)	14.56 (3.48)	-2.408	0.022
IRI cognitive scale	39.6 (7.3)	33.87 (8.18)	2.215	0.034
IRI affective scale	32.1 (5.98)	33.25 (5.48)	-0.595	0.556
BPQ total score	217 (42.65)	244.8 (39.64)	-1.976	0.056
BPQ awareness	120 (29.58)	130.2 (23.22)	-1.120	0.271
BPQ stress	25.8 (6.5)	31.25 (7.8)	-2.287	0.029
BPQ reactivity	44.15 (9.7)	50 (12.54)	-1.579	0.124
BPQ stress style	27.45 (4.12)	33.43 (4.41)	-4.198	<0.001*

Table 3.2: Psychological Results. Values denotes mean (standard deviation). t = independent sample t-test; p = p-value at t test. EIS: Emotional Inhibition Scale; IRI: Interpersonal Reactivity Index; BPQ: Body Perception Questionnaire. * denotes scales still significant after correction for multiple comparisons.

Regarding the *empathy profile*, revealed by the IRI, the HA subjects seemed less able to take the perspectives of other people and showed more discomfort when facing the negative feelings of others, indicating that HA subjects have difficulties in managing emotions in social relationships. Lastly, regarding *awareness of peripheral nervous system reaction in response to emotions*, revealed by BPQ, HA participants showed more intense physical reactions to stress compared to LA participants and greater awareness of their bodily changes due to stressful events. These results are in line with the frequent observation that alexithymia is highly prevalent in patients suffering from psychosomatic diseases (Sifneos et al. 1973; Taylor et al. 1991). Taken together, these results suggest that subjects with HA are less able to express their own feelings and deal with the feelings of others, and they tend to misinterpret their bodily reactions.

These features decrease the likelihood of having a false positive in the HA group and a false negative in the LA group.

eVRT task: In order to investigate the effect of the emotional content of the viewed face on the VRT effect in alexithymia, HA and LA subjects' accuracy in responding to bilateral tactile stimuli was compared when both fingers did or did not touch the faces. We conducted an analysis of variance (ANOVA) on bilateral stimulation detection accuracy using Group (two levels: Low and High alexithymia) as a between-subjects variable, and Emotion (three levels: Fear, Happy and Neutral) and Finger Trajectory (two levels: No Touch and Touch) as within-subjects variables. Post-hoc comparisons (Duncan's test) were performed, when necessary, to compare single effects. The partial eta squared (η_p^2) was reported as an estimate of effect size (Cohen, 1969). The analysis revealed a significant main effect of Finger Trajectory ($F[1,34]=71.2$; $p<0.001$, $\eta_p^2 = 0.67$). Moreover, both the critical double and the triple interactions were significant: Emotion x Finger Trajectory ($F[2,68]=11.86$; $p<0.001$, $\eta_p^2 = 0.25$) and Group x Emotion x Finger Trajectory ($F[2,68]=8.07$; $p<0.001$, $\eta_p^2 = 0.19$). The significant triple interaction led us to analyze LA and HA subjects separately. Consequently, an ANOVA on bilateral stimulation detection accuracy using Emotion and Finger Trajectory as within-subjects variables was performed on each group.

In the LA group, the analysis revealed a significant main effect of Finger Trajectory ($F[1,19]=42.28$; $p<0.001$, $\eta_p^2 = 0.68$). Moreover, the critical double interaction Emotion x Finger Trajectory was also significant ($F[2,38]=19.88$; $p<0.001$, $\eta_p^2 = 0.50$). Post-hoc tests showed that the Touch condition enhanced the accuracy of bilateral stimulation perception in comparison to the No-Touch condition for neutral

faces (mean accuracy no touch: 56.8%±4.3%, mean accuracy touch: 69.7%±4.3%, $p<0.001$) and for fearful faces (54.8%±2.9% vs 73.9%±3.4%, $p<0.001$). However, this effect failed to reach statistical significance when participants saw happy faces (58.5%±4.7% vs 61.7%±4.2%, $p=0.08$). Moreover, whereas in the No-Touch condition there was no difference in the accuracy of bilateral stimuli detection between the three emotions (fear vs happy: 54.8%±2.9% vs 58.5%±4.7%, $p=0.10$; fear vs neutral: 54.8%±2.9% vs 56.8%±4.3%, $p=0.27$; happy vs neutral: 58.5%±4.7% vs 56.8%±4.3%, $p=0.34$), in the Touch condition the accuracy of perception of bilateral stimuli was higher for fearful faces (73.9%±3.4%) than for happy (61.7%±4.2%, $p<0.001$) or neutral faces (69.7%±4.3%, $p=0.025$), and was higher for neutral faces (69.7%±4.3%) than for happy faces (61.7%±4.2%, $p<0.001$). The lack of significant differences found in the No-Touch condition is important to rule out the possibility that the results are due to an increased attention to fearful faces.

In the HA group, the analysis revealed a main effect of Finger Trajectory ($F[1,15]=32,18$; $p<0.001$, $\eta_p^2 = 0.68$). Critically, in contrast to the LA group, the double interaction Emotion x Finger Trajectory was not significant ($F[2,30]=0.26$; $p=0.76$, $\eta_p^2 = 0.01$). Subjects were more accurate when seeing faces being touched (64.2%±3.7%, 64.3%±3.8%, 67.5%±3.3% of bilateral stimuli detection for fear, happy and neutral faces, respectively) compared with seeing faces being just approached by fingers (53.8%±3.4%, 55.5%±2.4%, 58.5%±3.2%). However, the Touch condition enhanced the accuracy of bilateral stimulation perception in comparison to the No-Touch condition to the same degree for all the emotions (fear: 53.8%±3.4% vs 64.2%±3.7%; happy: 55.5%±2.4% vs 64.3%±3.8%; neutral: 58.5%±3.2% vs 67.5%±3.3%). Results are depicted in **Figure 3.2**.

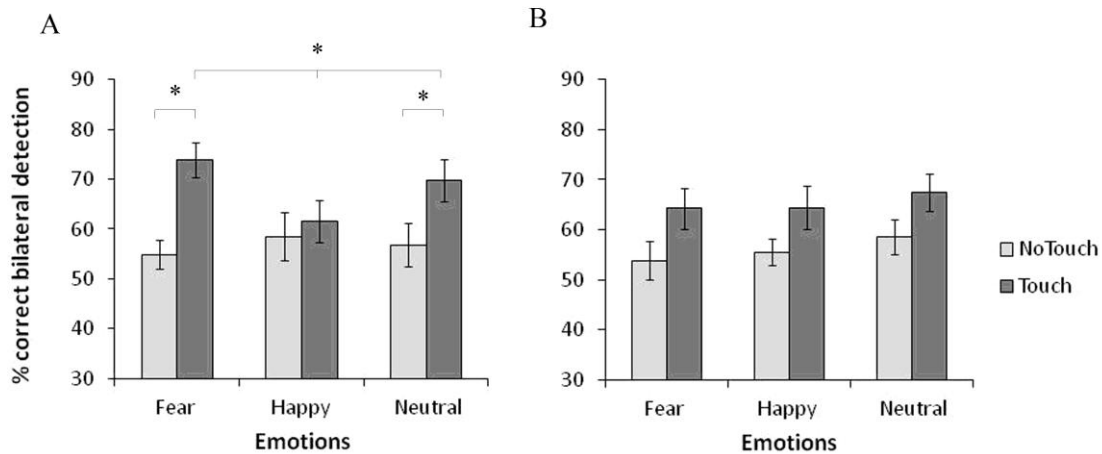


Figure 3.2. Results for the eVRT in LA (A) and HA (B) participants. Accuracy in detecting bilateral tactile stimulation while viewing video trials showing either a fearful face, a happy face or a neutral face that could be touched or just approached by two human fingers. Error bars show standard errors of the mean across LA and HA participants.

To further explore the difference between the LA and HA groups on the influence of emotional expression on bilateral stimuli detection, an emotional VRT index was calculated, consisting of the difference between accuracy of bilateral stimuli detection in the No-Touch and Touch conditions (Touch – NoTouch). LA and HA participants' emotional VRT index for each emotion was compared. We performed an analysis of variance (ANOVA) on VRT indices using Group (two levels: LA and HA) as a between-subjects variable, and Emotion (three levels: Fear, Happy and Neutral) as a within-subjects variable. The analysis revealed a significant Group x Emotion interaction ($F[2,68]=8.07, p<0.001, \eta_p^2 = 0.19$). The post-hoc test showed that in LA the VRT index was bigger (i.e. bigger difference between no touch and touch) when participants saw fearful faces ($19\% \pm 2.4\%$) than happy ($3.2\% \pm 1.6\%$, $p<0.001$) or neutral faces ($12.9\% \pm 2.7\%$, $p=0.018$) and was also bigger for neutral faces than for happy faces ($p<0.001$). On the contrary, in HA the VRT index was the same for all three emotions

(fear: 10.4%±1.6%; happy: 8.8%±2.9%; neutral: 8.9%±1.7%, all the comparisons $p>0.54$).

Explicit Task: To investigate the explicit emotion recognition profile in alexithymia, LA and HA subjects' explicit arousal and valence ratings of the emotional facial expressions were compared. Mean arousal and valence ratings for each group of subjects were calculated separately for the three emotion conditions (fear, happy and neutral) and submitted to mixed factors ANOVAs with Group (two levels: LA and HA) as a between-subjects variable and Emotion (three levels: Fear, Happy and Neutral) as a within-subjects variable.

With respect to arousal ratings, a significant main effect of Emotion ($F[2,68]=850.9$; $p<0.001$, $\eta_p^2 = 0.96$) was found. Positive and negative faces were rated as more arousing than neutral ones ($p<0.001$ at post-hoc test). Neither the between-subjects factor Group ($F[1,34]=0.36$; $p=0.55$, $\eta_p^2 = 0.01$) nor the double interaction ($F[2,68]=0.71$; $p=0.49$, $\eta_p^2 = 0.01$) was significant. With respect to valence ratings, the statistical analysis revealed a highly significant effect of Emotion ($F[2,68]=1892$; $p<0.001$, $\eta_p^2 = 0.98$). Post-hoc tests revealed that valence ratings for happy faces (mean 7.68±0.1) were significantly higher than neutral (mean: 4.5±0.09, $p<0.001$) and fearful faces (mean: 1.92±0.09, $p<0.001$). In addition, valence ratings for neutral faces were significantly higher than fearful faces ($p<0.001$). Neither the between-subjects factor Group ($F[1,34]=0.24$; $p=0.62$, $\eta_p^2 = 0.005$) nor the interaction Group x Emotion ($F[2,68]=1.96$; $p=0.14$, $\eta_p^2 = 0.005$) was significant.

Correlations: To further investigate the association between alexithymia and behavioral results, we first computed bivariate correlation between scores on each TAS-20 subscales (DIF, DDF, EOT) with the emotional three VRT indexes (fear, happy and neutral). We performed an outlier analysis and identified one subject in the LA group as an outlier. A strong negative correlation was found only between the DIF subscale of TAS-20 and the emotional VRT index for fear (Spearman $R = -0.47$; $p = 0.003$, see *Figure 3.3*). On the contrary, all the others correlations were not significant (eVRT index for fear and DDF: $r = -0.32$, $p = 0.06$; eVRT index for fear and EOT: $r = -0.18$, $p = 0.28$; eVRT index for happy and DIF: $r = 0.28$, $p = 0.11$; eVRT index for happy and DDF: $r = 0.25$, $p = 0.15$; eVRT index for happy and EOT: $r = 0.15$, $p = 0.37$; eVRT index for neutral and DIF: $r = -0.17$, $p = 0.30$; eVRT index for neutral and DDF: $r = -0.11$, $p = 0.51$ and eVRT index for neutral and EOT: $r = -0.11$, $p = 0.48$). Note that the significant correlation between DIF and VRT for fear ($p = 0.003$) persists after multiple comparison correction (corrected alpha level was 0.005).

However, since the two groups widely differed in subscales score, the correlations using these subscales are likely to suffer from non-independence. As such, the effect size we reported might be inflated. We also used z transformations (Meng et al. 1992) to further confirm that VRT performance viewing fearful expression was more strongly associated with the “affective” dimensions (DIF and DDF subscales of TAS-20), relative to the “cognitive” dimension (EOT subscale of TAS-20), of alexithymia. To this end, a total of four comparisons were conducted comparing the strength of correlations between TAS subscales and VRT indices. Results from these follow-up analyses revealed that DIF was more strongly associated with VRT index for fear as compared to the association of DIF with VRT index for happy ($z = 5.27$, $p < 0.05$), DIF

with VRT index for neutral ($z=2.26$, $p<0.05$), and EOT with VRT index for fear ($z=1.98$, $p<0.05$). There were no significant differences between associations of DIF with VRT index for fear and DDF with VRT index for fear ($z=1.17$, $p>0.05$). In sum, these analyses further confirm that VRT indices for fear were more strongly associated with the “affective” dimensions, relative to the “cognitive” dimension, of alexithymia.

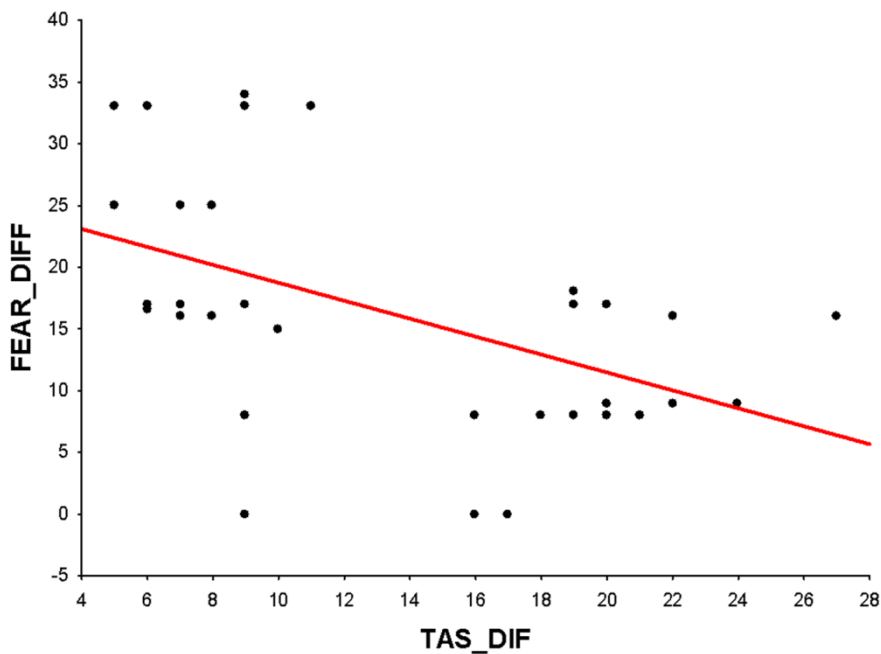


Figure 3.3. Negative correlation (Spearman $R=-0.47$; $p=0.003$; after removing one outlier from the analysis) between DIF subscale of TAS and the difference between no touch and touch in the accuracy of bilateral stimuli detection while subjects saw fearful faces in the eVRT task.

3.3. EXPERIMENT 2: Neuropsychological profile in Alexithymia

In order to test whether the above reported results could be affected by variables other than emotion related features, LA and HA participants underwent a comprehensive neuropsychological examination, investigating participants’ cognitive

profiles. Neuropsychological tests were selected mainly to assess working memory and frontal functions.

This step is of particular relevance since, citing the weird geographer in the Antoine de Saint-Exupery book, “<<*An explorer who told lies would bring into disrepute the geographer’s books. As would an explorer who drank too much, because drunkards see double. As a result, the geographer would note two mountains where, in fact, there was only one.*>>”.

3.3.1 Material and Methods

Participants: the participants are the same as *Experiment 1*. They were asked to undergo the neuropsychological examination after procedures described in *Experiment 1* were completed. None of them refused to participate.

Neuropsychological assessment: A comprehensive neuropsychological examination was performed in order to examine the cognitive profile of participants and to investigate possible differences in cognitive profile between high and low alexithymia subjects. Since the participants were students at the faculty of psychology and they studied neuropsychology, tests that are rarely covered during university courses were selected. This is the reason why, for instance, the TIB (Test di Intelligenza Breve, [Colombo et al. 2002](#), an Italian version of NAART, [Johnstone et al. 1996](#)) was chosen instead of the Raven’s matrices ([Raven, 1947, 1981](#)) to assess general intelligence, and the Delis Kaplan Frontal Function System (D-KEFS) sorting test ([Latzam and Markon, 2010](#); [Homack et al. 2005](#)) was chosen instead of the well known Wisconsin Card Sorting Test ([Heaton et al. 2008](#)) to assess frontal functions.

The neuropsychology battery was composed of the following tests:

- TIB for assessing general intelligence (Colombo et al. 2002): Subjects were asked to read 34 words with irregular accentuation, which could be read only if the reader knew the words. A high correlation between TIB total score and WAIS total IQ has previously been found. The number of errors was recorded.

- Digit span (Spinnler and Tognoni, 1987): This is a measure of short-term memory in which a person listens to someone saying a series of single-digit numbers and must repeat them back in the same order they were given.

- Backward span (Spinnler and Tognoni, 1987): This is a measure of working memory in which a person listens to someone saying a series of single-digit numbers and is asked to recall the items in reverse order.

- 2-back: This is a subtask of the TAP (Zoccolotti et al. 1994), used to measure working memory abilities. In this task, a randomly ordered sequence of 100 digits appeared in the middle of a computer screen at the rate of one stimulus every 3 seconds. Subjects were requested to press a button whenever the presented digit matched the stimulus 2 positions back in the sequence. Twenty target stimuli were given. Percentages of correct answers, omissions, and row number of false positives were recorded.

- PASAT word: This test is a variant of the Paced Auditory Serial Addition Test (Gronwall, 1977) which strongly relies on working memory and information processing speed abilities. Subjects were required to listen to a list of words and produce a word that starts with the third letter of the word they heard 2 positions previously in the sequence. The number of errors was recorded.

- D-Kefs sorting test (Latzam and Markon, 2010; Homack et al. 2005): This test has been used to assess high level functions such as pianification, cognitive flexibility

and capacity for abstraction. The test is formed by 16 different sorting concepts across two different sets of cards. Subjects were requested to categorize the six cards of each set into two abstract categories, based on lexical or perceptual information. A high correlation between performance on the D-Kefs sorting test and the WCST has been found. For each category, scores were based on correctness in categorizing the cards and describing both categories.

- *Spatial Incompatibility*: This is a subtask of the TAP (Zoccolotti et al. 1994) that measures the ability to inhibit irrelevant information. In this task, an arrow appeared on the left or the right part of the screen. Subjects were required to press a right or a left button in accordance with the direction indicated by the arrow, inhibiting the position of the arrow on the screen. Accuracy and the reaction times were recorded.

Statistical analysis: To investigate possible differences in cognitive profile between high and low alexithymia subjects, we compared the performance of the two groups on each neuropsychological test using two independent sample t-tests.

3.3.2 Results:

The analyses revealed no differences between the two groups (all $p > 0.11$). Results are summarized in **Table 3.3**. These results partially disagree with previous findings. Indeed, Zhu et al. (2006) compared HA and LA participants' scores on the Wisconsin Card Sorting Test and found a lower number of correct responses in HA participants. Moreover, two studies provided evidence for a relationship between alexithymia and executive dysfunction using a verbal fluency test (Henry et al. 2006; Lamberty and Holt, 1995). However, these latter two studies were conducted on non-healthy

populations (the first one on brain injured patients and the second one on war veterans), so their results may not generalize to alexithymia in healthy populations. Furthermore, while the studies cited above used only one index of executive functions, in the present work several indices were used, making the results most reliable.

		LA	HA	t	p
TIB	n. errors	2.05 (1.69)	3.12 (2.24)	-1.577	0.125
Digit Span		6.25 (0.96)	5.87 (0.61)	1.34	0.188
Backward Span		4.65 (0.74)	4.56 (0.89)	0.31	0.750
Pasat Word	n. errors	9.82 (7.19)	6.93 (4.55)	1.33	0.192
D-Kefs sorting test		43.57 (5.53)	42.4 (6.04)	0.592	0.558
2-back accuracy	% correct answer	83.63 (12.61)	86.33 (11.35)	-0.647	0.522
2-back omissions	% omissions	16.21 (12.36)	13.6 (11.24)	0.636	0.529
2-back false positive		5.52 (5.84)	4.53 (3.31)	0.587	0.562
Spatial Incomp. Accuracy	% correct answer	97.2 (3.62)	94.86 (4.79)	1.624	0.110
Spatial Incomp. RT	ms	0.49 (0.08)	0.5 (0.09)	-0.440	0.663

Table 3.2: Neuropsychological evaluation results. Values denotes mean (standard deviation); t = independent sample t-test; p = p-value at t test. TIB = Test di Intelligenza Breve. D-Kefs = Delis- Kaplan Executive Function System. RT = Reaction Time.

3.4. Discussion

Difficulty in recognizing others' emotions in alexithymia has previously been demonstrated using both direct (McDonald and Prkachin, 1990; Vermeulen et al. 2006; Pollatos et al. 2008; Swart et al. 2009; Lee et al. 2011) and indirect (Kano et al. 2003; Mueller et al. 2006) tasks. The present study was designed to test the ability of alexithymic subjects to remap observed emotions onto the neural systems involved in generating the same emotional expressions. This has been investigated indirectly by means of the emotional Visual Remapping of Touch (eVRT) paradigm. In keeping with

previous results, the present findings confirm that viewing a face being touched enhances tactile perception on one's own face compared with viewing the same face being just approached by fingers (Serino et al. 2008). Moreover, the present results also confirm that fearful facial expressions modulate this effect (Cardini et al. 2012) in low alexithymia (LA) participants. The new finding is that the emotional expression of the observed face does not modulate the VRT effect in participants with high levels of alexithymia (HA). In contrast, LA and HA subjects did not differ in terms of arousal or valence ratings of the same facial expressions, suggesting that the absence of the VRT effect for fear in HA is due to their inability to remap fear (i.e. an emotion embodiment deficit) and not to their inability to explicitly evaluate emotion (Niedenthal, 2007).

Since no difference between HA and LA in cognitive functioning emerged, we could exclude the possibility that the results of our investigation were driven by this variable rather than alexithymia *per se*. Indeed, results of *Experiment 2* ruled out this possibility. Moreover, the emotional and empathy profiles of LA and HA participants described by means of the self reported questionnaires (i.e. EIS; IRI and BPQ) make unlikely the possibility of having a false positive in the HA group and a false negative in the LA group.

The results of the LA subjects replicate those of previous studies using the same paradigm in a normal population (Serino et al. 2008; Cardini et al. 2012). Viewing a fearful face being touched enhances tactile perception on one's own face compared to viewing the same face not being touched or expressing another emotion (Serino et al. 2008; Cardini et al. 2012). An influential model of emotion processing (Adolphs, 2002a,b) suggests that recognizing others' emotions also depends on activating a representation of that emotion within one's own somatosensory system, which simulates

how the other individual would feel when displaying a certain facial expression (Damasio, 1994; Adolphs et al 2000). The authors interpreted the enhancement of tactile perception when viewing a fearful face as a preliminary activation of the somatosensory system in response to threat. The present data showed that LA participants were able to readily remap fear-specific information onto their own somatosensory system.

The new finding of the present study is that the VRT effect in HA subjects is not influenced by the emotional content of the observed faces. HA subjects exhibited a standard VRT effect, i.e. they were more accurate when seeing faces being touched compared to seeing faces just being approached by fingers. However, the Touch condition enhanced perception of bilateral tactile stimuli in comparison to the no Touch condition to the same degree in all the emotion conditions, at variance with LA subjects. This effect could not be due to HA participants paying less attention to fearful faces compared to LA participants because in the No-Touch condition there was no difference in the accuracy of bilateral stimuli detection between the two groups, nor could it result from lower perceived arousal of fearful stimuli in HA participants because the arousal ratings of HA subjects did not differ from those of the LA subjects. The fact that HA participants exhibited a normal VRT effect suggests that the mechanism of remapping observed touch onto the somatosensory system is not impaired in HA subjects. The main result of the present investigation is the absence of the enhancement of the VRT effect when HA subjects saw fearful faces being touched. Since the general remapping mechanism seems to be unchanged in HA, the absence of an eVRT effect could not be explained as a deficit in the remapping mechanism, but must instead be explained as difficulty in remapping emotional information conveyed by others' faces onto the self. The robustness of this result is emphasized by the negative correlation between the

eVRT index for fear and the difficulty in identifying feelings (as revealed by the TAS-20 subscale score): the bigger the difficulty in identifying feelings experienced by the participant, the smaller the eVRT effect when seeing fearful faces. This negative correlation further corroborates the hypothesis that the lack of the eVRT effect in HA subjects is due to affective dimension of alexithymia and not to possible confounding variables that could influence emotion recognition, for example socioeconomic status (Alvarez and Fuentes, 1994).

The eVRT effect is supposed to be evoked by means of a preliminary activation of somatosensory cortices when viewing fearful faces, resulting in facilitated processing of tactile information delivered to the participant's face (Cardini et al. 2012). Therefore, the lack of the eVRT effect in HA participants could be due to a lack of this preparatory effect in the somatosensory cortices. Since this effect is specific for fear, we could speculate that it is due to reduced amygdalar modulation of somatosensory cortices (Friedman et al, 1986; Sah and Lopez De Armentia, 2003; Höistad and Barbas, 2008). In fact, the amygdala has been reported to play a key role in the early stage of facial expression processing, particularly for fearful expressions (Breiter et al. 1996; Morris et al. 1998; Calder et al. 2001; Zald, 2003; Schultz, 2005), and its activation appears to be automatic (Pasley et al. 2004; Schultz, 2005). Further, previous findings in alexithymia research have shown reduced amygdala reactivity in HA subjects (Kugel et al. 2008; Reker et al. 2010), and an activation based meta-analysis revealed that during the processing of negative emotional stimuli, alexithymia was associated with a diminished response of the amygdala (van der Velde et al. 2013). However, cognitive neuroscience research has only just started to address the neural mechanisms underlying alexithymia, and future research will be necessary to further explore the link between alexithymia

and amygdalar responsivity. The hypothesis that the amygdala may have a pivotal role in the fear related eVRT enhancement is specifically addressed in *Chapter 6, Experiment 7*.

The results of the present experiment suggest that alexithymic subjects' difficulty in recognizing others' emotions could be due to their inability to modulate their own somatosensory system activity according to the seen emotional expression. This would prevent them from generating the internal somatic representation of the other's emotion that is required to recognize it.

Interestingly, in the present experiment there was an unexpected eVRT effect when HA but not LA participants saw happy faces being touched compared to being just approached by fingers. This effect was not found in a previous eVRT experiment (Cardini et al. 2012). Emotion recognition strongly relies on the spontaneous tendency to synchronize our facial expressions with those of another person during face-to-face interaction (Dimberg and Thunberg, 1998; Dimberg et al. 2000; Larsen et al. 2003). In the present experiment, the electrodes that delivered the tactile stimuli in the eVRT task were placed on the cheeks, near the *Zygomaticus Major*, which is involved in producing a happy expression. We could speculate that the contraction of this muscle during spontaneous motor mimicry of happiness introduced noise into the somatosensory signal, leading to difficulty in detecting the electro-tactile stimuli. The absence of a VRT effect in LA subjects (see also Cardini et al. 2012) and the presence of the same effect in HA subjects when they see happy faces could be explained by assuming the absence of motor mimicry in HA participants. The hypothesis that LA and HA participants might differ in the emotional mimicry pattern is specifically explored in Chapter 4, Experiment 3.

To summarize, the present study provides clear evidence that alexithymia is associated with difficulties in remapping seen emotions, particularly fear, onto the observer's own somatosensory system. This impairment could be due to an inability of HA subjects to modulate their own somatosensory system activity according to the seen emotional expression, i.e. to re-experience the observed emotion through their own sensory systems.

CHAPTER 4

Invisible sides of emotions

*“Now here is my secret. It is very simple.
It is only with one’s heart that one can see clearly.
What is essential is invisible to the eye”*

The Little Prince
Antoine de Saint-Exupéry

Emotions are not only what is overtly conveyed by facial expressions (i.e. a smile), but they are also the changes that covertly happen within the bodies (i.e. cardiovascular emotional related changes). Indeed, theories of emotions posit that covert emotional states (e.g. happiness or fear) are often associated both with overt motor behavior (e.g. smiling or frowning) and covert bodily reactions (e.g. rapid facial reactions and visceral changes). Recent theories suggested that observers can automatically understand the covert emotional state of others by embodying their overt motor behavior and covert emotionally related changes (Gallese and Sinigaglia, 2011; Niedenthal et al. 2010; Bastiaansen et al. 2009; Oberman et al. 2007). In the current chapter, the invisible side of emotions will be explored. Indeed, the two experiments presented are focused on emotional related phenomenon that are invisible to the eyes, i.e. covert motor behaviors (*Experiment 3*) and cardiovascular changes (*Experiment 4*) accompanying the perception of emotion conveyed by affective facial displays.

Experiment 1 suggests that alexithymic participants are unable to remap the somatic aspect of fear on their own somatosensory system. In other words, they seem

unable to re-experience the observed emotion through their own sensory systems, i.e. they exhibit a deficit in the embodiment of fear. The theories of embodiment suggest that this process also involves the re-experience, or simulation, of the seen affective states in one's self. As a consequence, one may wonder if the results of Experiment 1 could be limited to a deficit in the embodiment of the somatic aspect of emotions, or are they extensible to other aspects of emotional embodiment, i.e. the motor aspects. This hypothesis has been explored in *Experiment 3*, in which the motor aspect of embodiment has been explored. Moreover, in *Experiment 4*, in which the visceral emotional reactions have been explored in order to investigate whether the between groups differences in motor embodiment might be partially explained by a reduced emotion related heart frequency variability.

Since alexithymic individuals, who are known to exhibit emotional difficulties, have been shown to manifest an alteration in the embodiment mechanisms, it is possible to state that the emotional sides that are invisible to the eyes are essential for the emotional understanding.

4.1 Introduction

Emotional states are complex events pervasive to the functioning of the mind/brain at almost any level. From a physiological point of view, the experience of affective states recruits specific visceromotor and somatomotor patterns, to the point that some authors identify a specific neural entity referred to as the “emotional motor system” (Niedenthal, 2007, Niedenthal et al. 2010; Gallese and Sinigaglia, 2011). The self-experience of emotions is intimately entangled with the production of emotional

movements, to the point that the causal link between experience of emotions and the related motor phenomena is known to be bi-directional. For example, the visceral motor phenomenon of tachycardia is both the product and the cause of the perception of anxiety; the somatic motor phenomenon of a smile is both the product and the cause of a perceived state of happiness (Niedenthal et al. 2010; Frijda, 2009).

Affective movements, and more specifically somatic affective movements, support inter-individual communicative functions (Falkenberg et al. 2008; Wild et al. 2001; Hess and Fischer, 2013). For example displays of fear, pain or disgust communicate to conspecifics valuable information about environmental threats. Smiles have a fundamental role in consolidating parental bonds, a supporting cornerstone of species survival. Interestingly, recent theories suggest that such communicative functions, i.e. the process of ascribing to others' emotional states, involves the re-experience, or simulation, of the same affective states in one's self (Cacioppo et al. 1986; Larsen et al. 2003; McIntosh, 1996; McIntosh et al. 2006; Moody et al. 2007; Hess and Bourgeois, 2010; Feroni and Semin, 2011; Hess and Fischer, 2013). Embodied simulation theories state that affective movements are intrusive in the observers' affective states and determine a matching emotion, thus providing a direct way of "emotion understanding" (Gallese and Sinigaglia, 2011; McIntosh, 1996; Niedenthal et al. 2010; Bastiaansen et al. 2009; Oberman et al. 2007; Niedenthal, 2007).

Evidence in favor of such an embodied mechanism is given by phenomena of implicit affective mimicry, the best known of which is the one involving the production of emotional facial displays in response to others' emotional facial displays (Dimberg and Thunberg, 1998; Cacioppo et al. 1986). Adult humans exposed to affective displays of conspecifics produce stereotypical facial movements that are specific to the facial

expression that is observed. These movements are referred to as rapid facial reactions (RFRs) (for a review see [Cattaneo and Pavesi, 2014](#)). They may follow a mimetic pattern (as in smiling in response to a smile) or a reactive pattern (as in producing a fearful facial posture in response to an angry face) to the emotional facial expression of the person being observed ([Dimberg et al. 2000, 2002](#); [Dimberg and Petterson, 2000](#); [McIntosh, 2006](#)). Subjects are unaware of RFRs, which are not modified by superimposed voluntary movements. Another characteristic of RFRs is their sub-second onset latency, which has been documented in the 300-700 ms range ([Dimberg et al. 2000](#)).

It is hypothesized that RFRs initiate or modulate affective states in the observer and therefore are potentially a fundamental link in the chain of events that mediate inter-individual affective communication ([McIntosh et al. 1994, 2006](#); [McIntosh, 2006](#)). RFRs would thus play a role in emotional embodiment ([Hatfield et al. 1992](#); [Lundquist and Dimberg, 1995](#); [McIntosh et al., 1994](#); [Vaughan and Lanzetta, 1980](#)), and in the perception and recognition of facial expressions of emotion ([Niedenthal et al. 2001](#)).

RFRs may be impaired in some major neuropsychiatric disorders with severe affective symptoms, such as schizophrenia ([Falkenberg et al. 2008](#)) and unipolar depressive disorder ([Wexler et al. 1994](#)) and some authors have identified the lack of embodied affective communication as a core element in the genesis of such disorders.

However, is it not yet known whether RFRs are impaired in milder (i.e. subclinical) forms emotional disorders such as Alexithymia, which is characterized by a lack of normal ability in reading non-verbal cues during social interactions and by problems in identifying non-verbal facial emotion ([Fitzgerald and Bellgrove, 2006](#)). The research on the affective motor systems of alexithymics is at its beginnings and in

particular no authors have yet investigated the characteristics of RFRs in individuals with such personality traits. The topic is of utmost relevance in the context of an embodied theory of affective states and of affective communications given the causal role attributed by the embodied theory to automatic affective movements in the process of emotional communication. *Experiment 3* was specifically designed to test this issue. The degree of which participants with low and high alexithymia level produce RFRs in *Corrugator supercilii* and *Zygomaticus Major* (Larsen et al. 2003) muscles congruent with the facial expressions displayed in observed faces has been investigated. The *a priori* prediction was that subjects with high alexithymia levels should show attenuated RFRs of the expected enhanced muscles activation in response to the observed emotions, particularly the negative one, while the effect should manifest itself in the subjects with low alexithymia levels.

Besides the somatic emotional responses to affective facial displays (RFRs), in *Experiment 4* a non-communicative emotional movement, namely a visceral (cardiovascular) response, i.e. the variations of heart rate (HR), was investigated. This further investigation was carried out with the aim of circumscribing a possible emotional motor deficit of HA subjects to the social/communicative domain or to a more general domain of self-experience of emotion. Visceral reactions have been previously investigated in alexithymia, leading to contradictory results. On one hand, some studies reported greater heart rate in response to emotional stimuli in alexithymia (i.e. Waldstein et al. 2002; Lumley et al. 1996; Fukunishi et al. 1999; Friedlander et al. 1997; Nemiah et al. 1997), theorizing an hyper-arousal in alexithymia. On the other hand, these findings are contradicted by studies suggesting that alexithymia predicts reduced autonomic reactivity (i.e. Hyer et al. 1990; Linden et al. 1996; Neumann et al.

2004), theorizing an hypo-arousal during emotional stimulation in alexithymia. Critically, these studies do not investigate alexithymic visceral reactions in response to facial displays (i.e. to others' emotion) since the authors measure the heart frequency at rest or after inducing an emotional state in the participants (i.e. one's own emotion). Thus, whether HA subjects manifest visceral reactions in response to other's expressions is still an open question. To this aim, the changes in autonomic activity (as documented by changes in instantaneous heart rate - HR) induced by the observation of affective facial displays in HA and LA subjects were investigated with the *a priori* prediction to find a reduced emotional related autonomic response in HA compared to LA subject.

4.2 EXPERIMENT 3: Somato-motor reactions to emotional facial displays

4.2.1 Materials and Methods

Participants: Participants inclusion criteria were reported in detail elsewhere (please see Chapter 2). In brief, participants were included in the study if i) they had no history of neurological, major medical or psychiatric disorder and ii) they did not show subclinical or clinical depression at the Italian version of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; [First et al. 1997](#)); iii) their scores at two different instrument to assess alexithymia were congruent. The two instrument used to assess alexithymia were the 20-item Toronto Alexithymia Scale (TAS-20; [Taylor et al. 2003](#); cut-off for high alexithymia > 61; cut-off for low alexithymia < 39) and the alexithymia module of the structured interview for the Diagnostic Criteria for Psychosomatic Research (DCPR) ([Mangelli et al. 2006](#)). The TAS-20 is formed by three subscales, namely: Difficulty Identifying Feelings (DIF); Difficulty Describing

Feelings (DDF); Externally Oriented Thinking (EOT). The DIF and DDF subscale are thought to reflect the affective dimension of alexithymia, while the EOT subscale the cognitive dimension (Taylor et al. 2003). The initial number of included subjects was 12 in LA and 11 in HA groups.

Mood questionnaire: Since the emotional state of the subjects during the experiment has been proven to influence the RFRs responses (Moody et al. 2007, Niedenthal et al. 2001), prior to the experiment the participants were asked to fulfill the Positive and Negative Affect Schedule (PANAS, Crawford and Henry, 2004). The PANAS consists of 10 positive affect items (reflecting the extent to which a person feels enthusiastic, active and alert) and 10 negative affect items (reflecting distress, anger, fear and guilt). Participants rated items on a five point scale to what extent they experienced certain mood states (1 = "very slightly or not at all," and 5 = "extremely"). Data on emotions "enthusiastic" and "afraid" were of particular interest for the present study, since these emotions are also the one expressed by observed faces. Moreover, to estimate the level of participants' dedication to the experiment, "interested" and "attentive" affects were analyzed as well. One participant in the HA group and two participants in the LA group were excluded from the study due to the low score (1= "not at all") to the "attentive" and "interested" and "enthusiastic" affects, which would have negatively influenced the results.

Visual stimuli: The subjects, tested individually, were seated with a 19-inch screen in front of them onto which the stimuli were shown. All stimuli were presented on a black background and were resized to a visual angle of 8.23° height and 9.83° width at a viewing distance of 50 cm. Black-and-white stimuli consisted of 15 objects, which were used as catch trials, and 30 static pictures selected from the Pictures of Facial Affect

(PFA) database (P. Eckman and W.V. Friesen, Consulting Psychologists Press, Palo Alto, CA, 1976). The Eckman faces depicted 10 different actors (5 male and 5 female), each of them showing three facial expressions: fear, happy and neutral. The experiment was programmed with the E-Prime software to control picture presentation and to flag the EMG recordings with event onsets.

EMG recording: Facial EMG activity was measured over the *corrugator supercilii* and *zygomatici* muscles (See Cattaneo and Pavesi, 2014 for an overview on the anatomy of facial muscles and see Fridlund and Cacioppo, 1986; Larsen et al. 2003 for surface EMG recordings of facial muscles), with surface Ag/AgCl electrodes (6 mm diameter) in a bipolar montage. The ground electrode was placed on the mastoids. The left side of the face was recorded, consistently with studies reporting more extensive left-hemiface movement during emotional expression (Rinn, 1984; Dimberg and Petterson, 2000). Muscle activity was amplified x1000 with the Biopac MP150 System and sampled at 1000 Hz. A notch filter at 50 Hz was also applied. To obscure the fact that their facial muscular activity was the main data of interest, the participants were told that several muscles were measured throughout the body. To this end, fake electrodes were placed on the left leg, arm and shoulder. When interviewed after the experiment, none of the subjects reported that they were aware of the actual measurement being made. EMG was collected continuously for 2 blocks for each subject. Each block contained 45 trials (10 faces for each emotion, i.e. happy, fear and neutral, and 15 objects)

Trial structure and task: On each trial, a white fixation cross was presented in the center of the screen. After 5 sec, a randomly selected stimulus (i.e. a face or an object) replaced the fixation cross and remained on the screen for 2 s, followed by a 20 s inter-

trial interval second long (Dimberg and Thunberg, 1998; Dimberg et al. 2000; Dimberg et al. 2002). Participants were never told that the experiment's aim was to assess emotional processing and instead were required to carry out an irrelevant task on gender categorization of the face or object as male or female (please note that the Italian language does not support a neutral form, therefore each object is either masculine or feminine). Responses were given manually by pressing a button. The 45 different stimuli were all presented in each block in a random order, therefore each single stimulus was presented twice (once in each block) in the whole experiment.

EMG data processing: Two participant in the LA and one in the HA group were discarded from the data processing and analysis given the massive presence of movement artifacts in the recording. Even though the EMG was acquired continuously, the time window of interest was limited from -500 ms to +1250 ms around the onset of the visual stimuli. Six steps of pre-processing were then carried out prior to statistical analysis. 1) The digitized EMG signals were filtered off-line with a high pass filter of 30 Hz. 2) All recordings were rectified. 3) Trials with excessive EMG activity in the baseline (pre-stimulus) period were defined as those trials with baseline EMG greater than 2 standard deviations over the mean value of baseline EMG from all 90 trials. Outlier trials were then removed from further analysis (overall less than 5%). 4) Baseline correction was performed trial-by-trial by subtracting from each data point the mean pre stimulus activity (-500 to 0 ms). 5) Data were down-sampled to a 5 Hz sampling rate by dividing the recordings into bins 250 ms wide and averaging all data points within that bin to one single value. In this way each trial was reduced to 6 time-points corresponding to the 1-250 ms, the 251-500 ms, the 501-750 ms, the 751-1000 ms and the 1001-1250 ms bins. 6) The EMG associated with emotional facial displays

(happiness and fear) were normalized individually by subtracting from them the mean value of the EMG activity associated with vision of the neutral faces, which served as baseline condition. The data associated with the vision of the objects were not analyzed.

Explicit Rating: After the facial EMG experiment, participants were asked to explicitly rate the arousal and valence of the emotional stimuli. The very same Eckman faces and objects used in the facial EMG were presented to participants. Participants were requested to rate arousal and valence using a nine point Likert scale, with 1 meaning “not very arousing” or “very negative emotion” and 9 meaning “highly arousing” or “very positive emotion.”

Behavioral Statistical analysis: For the analysis of behavioral data, a mixed factor ANOVA on reaction times (RT) with Group (two levels: LA and HA) as a between-subjects variable and Stimulus (four levels: Fear, Happy and Neutral and Object) as a within-subjects variable was conducted. Consequently, an ANOVA on reaction times using Stimulus as within-subjects variable was performed on each group.

EMG statistical analysis. A mixed factor ANOVA with Group (two levels: LA and HA) as a between-subjects variable and Muscle (two level: *corrugator* and *zygomaticus*), Emotion (two levels: Fear and Happy) and Time (five levels: 0-250 ms, 250-500 ms, 500-750 ms, 750-1000 ms, 1000-1250 ms) as a within-subjects variables was conducted.

Analysis of explicit emotion recognition. LA and HA subjects' explicit arousal and valence ratings of the emotional facial expressions were compared. Mean arousal and valence ratings for each group of subjects were calculated separately for the four stimulus conditions (fear, happy, neutral and objects) and submitted to mixed factors

ANOVAs with Group (two levels: LA and HA) as a between-subjects variable and Stimulus (four levels: Fear, Happy, Neutral and Objects) as a within-subjects variable.

Newman-Keuls post-hoc test was used when necessary. For each analysis, the partial eta squared (η^2_p) was reported as an estimate of effect size, such that 0.05 is considered a small effect, 0.1 a medium effect, and 0.2 a large effect (Cohen, 1969).

4.2.2 Results

Table 4.1 gives comparative information about the low alexithymia (LA; n=10) and high alexithymia (HA; n=10) groups.

	Low Alexithymia	High Alexithymia
n (male/female)	10 (2/8)	10 (2/8)
Age, mean (SD) (years)	24.9 (2.28)	24.7 (2.35)
<i>TAS-20</i>		
Total score	33.1 (4.86)	62.9 (1.66)
DIF	10.7 (2.79)	25 (3.05)
DDF	8.6 (2.17)	19.1 (2.55)
EOT	13.8 (2.39)	18.8 (3.73)
DPCR	1.3 (0.67)	3.2 (0.42)

Table 4.1 Demographic and alexithymia profile of the low and high alexithymia groups. TAS-20: twenty-item Toronto Alexithymia Scale; DIF: difficulty in identifying feelings; DIF: difficulty in describing feelings; EOT: externally oriented thinking. SD: standard deviation. Low Alexithymia (n=10) and High Alexithymia (n=10) groups were obtained excluding the participants with discrepancy between TAS-20 and DPCR scores (cf. Material and Methods). DPCR: Alexithymia Module of Diagnostic Criteria for Psychosomatic Research scores.

Mood Questionnaire: LA and HA participants did not differ in all PANAS's affects rating, as reported in Table 4.2. Of particular relevance for the present investigation, the two groups did not differ in the rating of "afraid" (two independent

sample t-test: $t=0.4$, $df=18$, $p=0.69$), “enthusiastic” ($t=1.1$, $df=18$, $p=0.27$), “attentive” ($t=0.709$, $df=18$, $p=0.47$) and “interested” ($t=-0.87$, $df=18$, $p=0.39$) affects.

	HA (n=10)	LA (n=10)	t	p
Interested	3.5(0.4)	4 (0.3)	-0.87	0.39
Distressed	2.2(0.4)	1.8(0.2)	0.69	0.49
Excited	2 (0.2)	2 (0.2)	0	1
Upset	1.3 (0.2)	1.1 (0.1)	1.1	0.28
Strong	2.2 (0.4)	1.8 (0.3)	-1.3	0.2
Guilty	1.3 (0.2)	1 (0)	1.4	0.17
Scared	1.3 (0.2)	1.1 (0.3)	0.85	0.40
Hostile	1 (0)	1 (0)	0	1
Enthusiastic	3.1 (0.3)	2.5 (0.3)	1.1	0.27
Proud	2.7 (0.4)	2.2 (0.3)	1	0.29
Irritable	1.7 (0.3)	1.4 (0.2)	0.9	0.37
Alert	2.1 (0.4)	1.7 (0.3)	0.59	0.55
Ashamed	1.7 (0.3)	1.2 (0.1)	1.7	0.1
Inspired	2.2 (0.4)	1.6 (0.2)	1.2	0.21
Nervous	1.5 (0.2)	1.5 (0.2)	0	1
Determined	2.3 (0.4)	2.8 (0.4)	-1.3	0.31
Attentive	3.8 (0.3)	3.1 (0.4)	0.70	0.47
Jittery	1.9 (0.3)	1.4 (0.2)	1.13	0.27
Active	3.4 (0.3)	3.1 (0.4)	0.58	0.56
Afraid	1.3 (0.7)	1.2 (0.4)	0.4	0.69

Table 2. Participants' responses to PANAS (Positive and Negative Affect Schedule, Crawford and Henry, 2004). The PANAS consists of 10 positive and 10 negative affect items. Participants rated items on a five-point scale indicating to what extent they experienced certain mood states (1= very slightly or not at all; 5= extremely). Values denote the mean and standard error of the mean (in brackets).

Facial EMG:

Behavior: The ANOVA revealed a main effect of Group ($F[1,18]=4.8$; $p=0.04$; $\eta_p^2 = 0.21$) and of Stimulus ($F[3,54]=7.6$; $p=0.002$; $\eta_p^2 = 0.29$). The interaction Group x Stimulus is also significant ($F[3,54]=3.34$; $p=0.02$; $\eta_p^2=0.15$). The significant interaction

led us to analyze LA and HA subjects separately. In the LA group, the analysis reveals a significant main effect of Stimulus ($F[3,27]=6.74$; $p=0.001$; $\eta_p^2 = 0.42$). Post-hoc test show that, although the task is to respond to the gender of the faces, responding to fearful faces (mean 704.2 ± 28.2 sem. ms) is quicker than responding to happy faces (729.2 ± 30.2 ms; $p=0.04$) and neutral faces (732.3 ± 29.6 ms; $p=0.06$). Moreover, responding to an object (685.8 ± 32.4 ms) is faster than responding to a fearful (704.2 ± 28.2 ms, $p=0.13$), happy (729.2 ± 30.2 ms; $p=0.003$) and neutral (732.3 ± 29.6 ms; $p=0.003$) faces. In the HA group, the analysis reveal a significant main effect of Stimulus ($F[3,27]=5.37$; $p=0.004$; $\eta_p^2 = 0.37$). Post-hoc test show that, differently from LA participants, in HA there is no emotion modulation of RT. Responding to fearful faces (839.4 ± 34 ms) is not faster than responding to happy faces (822.9 ± 36.7 ms; $p=0.65$) or to neutral faces (788.9 ± 12.6 ms; $p=0.35$). Moreover, responding to objects is faster (706.7 ± 26.8 ms) comparing to responding to all the facial displays (all comparisons $p<0.03$).

Facial Reactions: The only significant main effect is Time ($F[4,72]=3.11$, $p=0.02$; $\eta_p^2 = 0.14$). The main effects of Group ($F[1,18]=0.45$, $p=0.5$; $\eta_p^2 = 0.02$), Muscle ($F[1,18]<0.001$, $p=0.99$; $\eta_p^2 < 0.001$) and Emotion ($F[1,18]=0.90$, $p=0.35$; $\eta_p^2 = 0.04$) are not significant. However, the critical interaction Group x Muscle x Emotion x Time is significant ($F[4,72]=6.54$, $p<0.001$; $\eta_p^2 = 0.26$). These results let us analyze the two muscles separately.

In the *Corrugator Supercilii*, the triple interaction Group x Emotion x Time is significant ($F[4,72]=2.83$, $p=0.03$; $\eta_p^2=0.13$). Post-hoc test reveal that, in LA participants, the activity of *Corrugator Supercilii* in fear condition show a peak of activation at 625 ms that is significantly higher compared to the activity of the same

muscle in happy condition ($p < 0.001$). In addition, in fear condition, activation at 625 ms is significantly different from all the others time intervals (all comparisons $p < 0.001$). On the contrary, no *Corrugator Supercilii* activity is detected in HA group during the observation of emotional faces.

In the *Zigomaticus Major*, the triple interaction Group x Emotion x Time is significant ($F[4,72]=5.73$, $p < 0.001$; $\eta_p^2 = 0.24$). Post-hoc test revealed that, in LA participants, the activity of *Zigomaticus Major* in happy condition show a peak of activation at 625 ms that is significantly higher compared to the activity of the same muscle in fear condition ($p < 0.001$). In addition, in happy condition, activation at 625 ms is significantly different from all the others time intervals (all comparisons $p < 0.001$). In HA participants the activity of *Zigomaticus Major* in happy condition show a peak of activation at 825 ms that is significantly higher compared to the activity of the same muscle in fear condition ($p < 0.001$). In addition, in happy condition, activation at 825 ms is significantly different from all the others time intervals (all comparisons $p < 0.001$).

Results are shown in *Figure 4.1*.

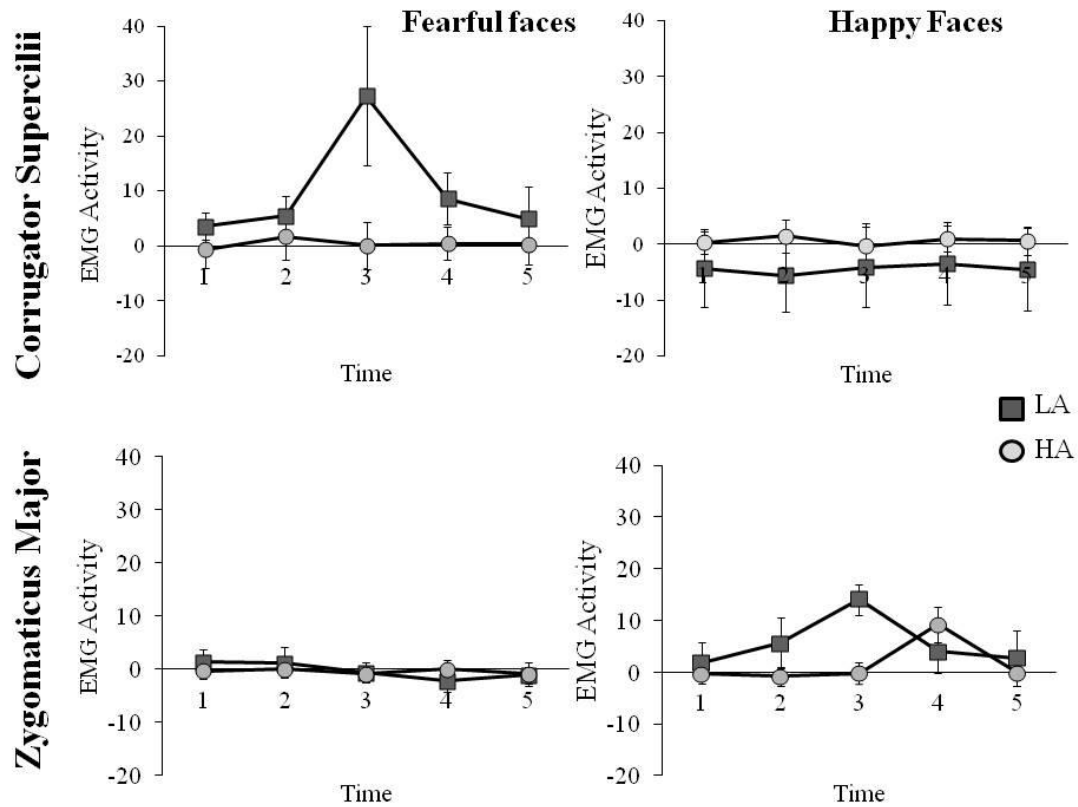


Figure 41. RFRs results. LA = Low Alexithymia group; HA = High Alexithymia group. EMG activity is expressed in $\mu V \cdot S$. Time refers to five different time intervals: 1 = EMG activity between 0 and 250 ms; 2 = EMG activity between 250 and 550 ms; 3 = EMG activity between 500 and 750 ms; 4 = EMG activity between 750 and 1000 ms; 5 = EMG activity between 1000 and 1250 ms after stimulus presentation. Error bars denotes SEM (Standard Error of the Mean).

Explicit Rating:

With respect to arousal ratings, a significant main effect of Stimulus ($F[3,54]=135.2$; $p<0.001$; $\eta_p^2 = 0.88$) was found. Positive and negative faces were rated as more arousing than neutral ones and objects ($p<0.001$ at post-hoc test). No differences between groups emerged. With respect to valence ratings, the statistical analysis revealed a significant effect of Stimulus ($F[3,54]=163.6$; $p<0.001$; $\eta_p^2 = 0.90$). Post-hoc tests revealed that valence ratings for happy faces (mean 7.23 ± 0.15 sem) were significantly higher than for fearful (mean: 2.6 ± 0.17 , $p<0.001$), neutral faces (mean: 4.3 ± 0.12 , $p<0.001$) and objects (mean: 4.9 ± 0.1 , $p<0.001$), whereas ratings for fearful

faces were lower than for neutral faces and objects (all comparisons $p < 0.001$). Again, no differences between groups emerged.

4.3 EXPERIMENT: Visceral reactions to emotional facial displays

4.3.1 Materials and Methods

Participants and protocol: The participants of the *Experiment 1* were asked to come a second time to perform the experiment. None of them refused to participate. The mean time elapsed between Experiment 1 and Experiment 2 was 183.15 (± 11.82) days. 10 LA and 10 HA subjects underwent this experiment. The experimental setting was the same as in Experiment 1 and the subjects were shown the very same Eckman faces of *Experiment 1* by means of the same procedures. The only difference in trial design was that in Experiment 2 the inter-trial interval was set to 10 sec instead of 20 ms.

Electrocardiographic (EKG) recordings and processing: EKG measurements were taken using non-polarizable Ag-AgCl electrodes attached to the left and right wrists referenced to the left mid-clavicle. Signals were recorded by a computer-based data acquisition system (Biopac MP150) and the corresponding software, AcqKnowledge (BIOPAC Systems Inc., Santa Barbara, CA). The signal was amplified $\times 100$ and digitized at 100 Hz. Only the series of consecutive heart beats starting 5 beats before the stimulus presentation and ending 5 beats after the stimulus presentation were considered for further analysis. At this point the continuous recordings were turned into all-or-nothing signals: QRS complexes were discriminated in the EKG recordings by triggering the R-wave peaks. The time interval between each consecutive QRS complex was then calculated (R-R interval) and its inverse value was calculated. Such value is an index of instantaneous HR and was multiplied by 60 in order to have a 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.

Statistical analysis: a mixed factor ANOVA on heart frequency (HF) using Group (two levels: LA and HA) as a between-subjects variable and Stimulus (three levels: fear, happy and neutral) and Time (two levels: Pre trigger and post trigger) as a within-subjects variables has been conducted. Neuman-Keuls post hoc test was used when necessary and the partial eta squared (η^2_p) was reported as an estimate of effect size.

Post-hoc correlations between electrophysiological measures and alexithymia scores: After evaluation of the main ad-hoc statistical analyses for each experiment, the existence of any linear correlation between TAS subscales and physiological measures was explored post-hoc. A series of regression analyses using as dependent variables the individual scores of each of the TAS-20 subscales (DIF, DDF and EOT) had been conducted.

To constrain the number of predictors (the physiological measures) the EKG data were further processed by computing the difference between the average post-stimulus HR and the average pre-stimulus (baseline) HR. The 3 resulting indexes (one for each facial display) were referred to as Δ HR. In this way the predictor variables were a total of 23 (3 EKG measures and 20 EMG measures corresponding to a 2 muscles x 2 emotions x 5 time bins structure). To keep the number of predictors reasonably low these were grouped into 5 clusters to perform separated Regression analyses. The clusters were: 1) the Δ HR in response to the happy, neutral or fearful faces (3 regressors), 2) the differential EMG from the corrugator muscle in response to happy faces at the 5 time-bins that were analyzed (5 regressors), 3) the 5 differential EMG

from the corrugator muscle in response to fearful faces at the 5 time-bins (5 regressors), 4) the differential EMG from the zygomaticus muscle in response to happy faces at the 5 time-bins (5 regressors) and 5) the differential EMG from the zygomaticus muscle in response to fearful faces at the 5 time-bins (5 regressors). In this way, 3 dependent variables were confronted with 5 clusters of regressors, ultimately producing a set of 15 separate regression analyses.

4.3.2 Results:

Heart frequency: The AVOVA reveal a main effect of Stimulus ($F[2,36]=4.2$, $p=0.02$; $\eta_p^2 = 0.18$), while the main effect of Group ($F[1,18]=0.55$, $p=0.46$; $\eta_p^2 = 0.02$) and Time ($F[1,18]<0.001$, $p=0.99$; $\eta_p^2 <0.001$) are not significant. However, the critical interaction Group x Stimulus x Time is significant ($F[2,36]=6.07$, $p=0.005$; $\eta_p^2 = 0.25$). These results let us analyze the two groups separately. In the LA subjects, the double interaction Stimulus x Time is significant ($F[2,18]=10.76$, $p=0.008$; $\eta_p^2 = 0.54$). Post-hoc test reveal that the pre trigger HF is not significantly different in the different type of stimulus presentation (mean 84.22 ± 0.5 sem for fear, 82.12 ± 0.9 for happy and 82.47 ± 0.7 for neutral, all $p>0.21$), whereas the post trigger HF is lower in fear (80.21 ± 1.6) than in happy (83.35 ± 0.8 , $p=0.03$) and neutral (84.87 ± 1.1 , $p=0.003$) faces. Moreover, only for fear there is a significant difference between HF pre and post trigger ($p=0.009$). In other terms, in LA participants the presentation of fearful faces determined a significant bradycardia, i.e. a deceleration in heart frequency, compared to baseline HR. On the contrary, in HA participants, no difference was found between the different type of stimulus presentation, as neither the interaction Stimulus x Time ($F[2,18]=1.26$, $p=0.30$; $\eta_p^2 = 0.11$), nor the main effect of Stimulus ($F[2,18]=2.7$,

$p=0.08$; $\eta_p^2 = 0.20$) and Time ($F[1,9]=0.02$, $p=0.88$; $\eta_p^2 = 0.01$) were significant. Results are shown in *Figure 2*.

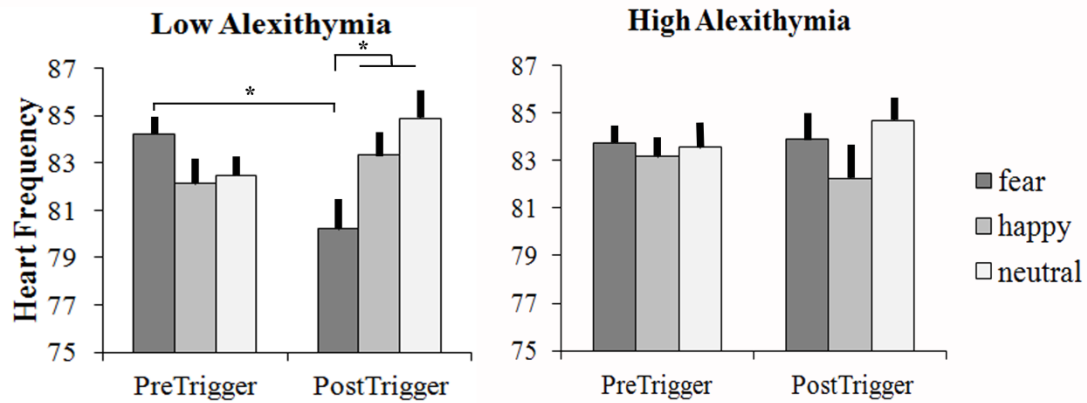


Figure 4.2. Heart Frequency results. Error bars denotes SEM (Standard Error of the Mean).

Correlations between electrophysiological measures and alexithymia scores: The full results of the 15 regression analyses are shown in Appendix A. The EKG parameters, i.e. the ΔHR , proved to be extraordinarily good predictors of the alexithymia scores. In particular, ΔHR to fearful faces was a significant predictor of all 3 sub-scores, DDF, DIF and EOT (all p -values <0.006). The polarity of the relation was positive, indicating that lesser fear-induced bradycardia predicted higher alexithymia symptoms. Similarly, the ΔHR to happy faces turned out to be a significant predictor of the EOT score only ($p=0.002$) with a negative relation, indicating that tachycardia in response to happy faces was associated with smaller scores of alexithymia symptoms.

The results of the 4 regression analyses on the facial EMG parameters indicated that the signal from the corrugator muscle 625 ms after the onset of a fearful stimulus and the signal from the zygomaticus 625 ms after the onset of a happy stimulus were both significant predictors of the DIF scores ($p=0.034$ and $p=0.032$ respectively) and

marginally significant predictors of the DDF scores ($p=0.07$ and $p=0.09$ respectively). The polarity of the correlation was negative, indicating that smaller congruent facial reactions predicted higher alexithymia symptoms. The only other significant effect was found in the 875 ms time bin, for the zygomaticus muscle when observing happy stimuli, which turned out to be a significant predictor of DDF scores. Interestingly, the polarity of the effect was positive, i.e. higher reactivity of the muscle in the 875 ms time bin was associated with higher scores of alexithymia symptoms.

4.4. Discussion

The main findings of the present study highlight a deficit of HA subjects in the production of emotional motor responses to implicitly processed emotional facial displays. In the first experiment HA participants demonstrated the absence of congruent RFRs in response to fearful stimuli, whereas RFRs in responses to happy stimuli although present were delayed, in comparison with LA participants. In the second experiment, a deficit in the non-communicative autonomic motor responses to fearful faces is reported, since the HA participants did not manifest a slowing in HR as did the LA participants. The present findings cannot be explained by the momentary emotional state, since the PANAS scores did not differ among groups (Niedenthal et al. 2001).

Dysfunction of communicative somatic emotional movements in HA.

RFRs to fearful and happy faces in LA participants appeared in congruent muscles at about 625 ms after stimulus presentation. These findings are in line with previous literature results in the general population, thus validating the appropriateness of the LA group as representative of the general population (Moody et al. 2007; McIntosh, 1996;

Niedenthal et al. 2001; Hatfield et al. 1992; Lundquist and Dimberg, 1995; McIntosh et al., 1994; Vaughan and Lanzetta, 1981). In contrast, HA subjects showed absence of significant responses to fearful faces in any of the two muscles but did show congruent RFRs to happy faces in the *zygomatici* muscles, but only at a later-than-normal time-bin (751-1000 ms). These results probably indicate that HA traits are associated with a malfunctioning but still present embodied communication system of affective states. The absence of RFRs to fear leaves little space to interpretation. Conversely, the presence of a congruent but abnormally delayed response to happiness is of greater interest. It indicates that HA subjects are probably still endowed with an intact embodied emotional communication system, only functionally different compared to that of LA subjects. The present data do not allow us to speculate on the nature of such functional difference, which could be supported by several factors such as a different threshold to emotional stimuli, a longer processing time or a faster than normal habituation to affective displays.

Recent theories of emotions suggest that observers imitate emotional expressions, and these imitated expressions entrain a feedback process, which in turn elicits a corresponding emotional state in the observer (Gallese, 2007; Goldman and Sripada, 2005). This is called “embodiment” of emotion (Niedenthal et al. 2001, Niedenthal, 2007). The emotional state then provides input that facilitates the emotion recognition process. Critically, when the somato-motor response to emotions is blocked, the emotions recognition is affected (Niedenthal et al. 2001). According with this latter study, it is possible to suggest that, in LA, the integrity of the embodied somato-motor aspect of emotions might help LA participants in emotion identification. In contrast, in

the HA, the embodiment mechanisms are altered, thus preventing these subjects to normally experience emotions.

It has been suggested that the degree to which an individual simulates another's emotions is predicted by the individual's empathy (Hussey and Safford, 2009), i.e. the ability to understand and resonate with another emotional state. Evidence suggests that highly emphatic individuals experience automatic imitation of other's facial expressions to a greater degree than less emphatic people (Chartrand and Bargh, 1999). In the light of this theoretical and empirical framework our data seem to indicate that subjects with HA traits are less efficient than LA subjects in the process of automatic pickup of others' emotions and amplifying and transmitting the emotional state to other conspecifics.

Dysfunction of non-communicative visceral emotional movements in HA.

The second findings of the current study are related to the visceral reaction to emotional faces. Consistent with prior studies (Neumann et al. 2004; Waldstein et al. 2002), our results did not yield any significant difference between groups in baseline levels of HR. Difference between groups emerged after the stimulus presentation, and only for fear. The results revealed a decrease of HR, i.e. bradycardia, after the presentation of a fearful face in LA subjects. In contrast, in HA participants, the fear-related bradycardia was absent, showing in this way a diminished visceral reaction to fearful faces. No between group differences emerged before and after the presentation of happy faces. Fear related bradycardia is considered to be a physiological component of a more complex freezing reaction (Azevedo et al. 2005; Hagenaaars et al. 2012; Roelofs et al. 2010), i.e. a cessation of locomotion that is characterized by a

parasympathetically dominated autonomic nervous system response that causes heart rate deceleration or bradycardia (Hermans et al. 2013). The freezing reactions is associated with sustained attentional processing of such stimuli (Libby et al. 1973; Lang and Davis, 2006), suggesting that attention is captured by negative valence stimuli in order to facilitate heightened attention to threat cues (Lang and Davis, 2006).

The findings of attenuated autonomic reactions to negative stimuli are consistent with prior research showing that alexithymia is associated with dampened physiological reactivity to such stimuli (i.e. Linden et al. 1996; Nemiah et al. 1997; Newton and Contrada, 1994; Wehmer et al. 1995; Neumann et al. 2004). Several studies demonstrated that higher alexithymia scores produced lower skin conductance and showed lower heart rate deceleration responses to emotional stimuli (Wehmer et al. 1995; Linden et al. 1996; Roedema and Simons, 1999), also if the emotional stimuli were presented below the level of consciousness (Pollatos et al. 2008). All these studies support the hypo-arousal model of alexithymia, according to which alexithymia is related to attenuated visceral responses in response to emotions (Neumann et al. 2004). The current findings further expanded the previous findings by showing that the hypo-arousal responses in alexithymia are not present only after inducing an emotional state in the participants (i.e. one's own emotion; Neumann et al. 2004), but also in response to facial displays (i.e. to others' emotion). Interestingly, autonomic responses are generally intended to benefit the subject only and are generally devoid of communicative significance. This finding moves the dysfunction of HA traits to a more general domain of emotion processing that goes beyond the communicative one.

HA subjects are not faster when implicitly processing fearful faces but are as efficient as LA subjects in explicit emotion recognition.

The behavioral data recorded during the EMG experiment showed that LA subjects were faster to respond to fearful faces than to neutral or happy faces, although the emotional task was implicit, i.e. the subjects were asked to respond to the gender of the seen faces. The fear facilitation effect did not emerge in HA participants. Notably, as witnessed by the analysis of explicit emotion/arousal ratings, the level of alexithymia influenced both the somatic and visceral reaction to affective facial displays, without affecting the explicit recognition of emotional faces, thus ruling out the hypothesis that the between groups differences on RFRs and HF results could depend on impaired explicit recognition of the emotional content of the faces. The arousal and valence rating did not differ between groups, a finding that is not new in alexithymia research (Grynberg et al. 2012).

Correlation between physiological data and Alexithymia scores.

The regression analysis between TAS subscales and RFRs revealed that RFRs to fear in the congruent muscle (*Corrugator Supercilii*) and RFRs to happiness in the congruent muscle (*Zygomatici* complex) at the physiologically expected time bin (501-750 ms) was a good predictor of the capacity to identify feelings (*inverse* relation with the Difficulty in Identifying Feelings - DIF score). This result is not surprising and is in line with our initial predictions. On the contrary, the RFRs in the *zygomaticus* complex to happy faces activity at 875 ms turned out to be good predictors of the difficulty in describing feelings (*direct* relation with the DDF scores). This apparently “paradoxical” result is easily explained and argues in favor of the importance of latency measurements

of RFRs. If maladaptive mechanisms in HA subjects manifest themselves as delayed RFRs, then its occurrence is an index of malfunctioning in the system and is expected to correlate positively with clinical symptoms.

The regression data, being produced post-hoc are weaker than the ANOVA results. They also potentially suffer from a bias due to the non-normal distribution of the alexithymia scores (very high and very low). However, given these limitations, they are very useful in suggesting that the RFRs were associated with the “affective” dimensions (DIF and DDF subscales), rather than to the “cognitive” dimension (EOT subscale), of alexithymia. These data are in accordance with previous findings, using other experimental paradigms, showing that the difficulty in implicit emotion recognition in alexithymia mainly correlates with affective rather than cognitive dimensions of alexithymia (Bermond et al. 2010; Pollatos et al. 2008, 2011).

A unifying interpretation

The results of RFRs, HR and RTs provided convergent evidence supporting diminished responses to emotional stimuli in HA participants compared to the LA. Interestingly, in all domains the deficit was more marked in response to fear displays rather than to happy faces. Indeed, *Experiment 3* found that HA participants did not automatically generate RFRs to fearful facial expression, they did not manifest the fear related bradycardia, and they did not show the fear related facilitation in RT. Instead, these phenomena were present in LA participants. These results might be explained by the amygdala hypo-functioning in alexithymia. The amygdala is a subcortical structure in the medial temporal lobe, and it is formed by different nuclei (Swanson and Petrovich, 1998). This structure is considered to be a key brain structure for emotional

processing (LeDoux, 2014; Adolphs, 2013), and it is known to be involved mainly in the processing of fear (Fusar-Poli et al. 2009; Vytal and Hamann, 2010), being an integral component of a continuous vigilance system, responding to unpredictable, novel and biologically relevant stimuli. Moreover, the amygdala plays a pivotal role in the early stage of facial expression processing (Calder et al. 2001; Zald, 2003; Liddell et al. 2005; LeDoux 2012). Critically, previous findings in alexithymia research revealed reduced amygdala reactivity in HA subjects in response to negative emotional stimuli (Kano et al. 2003; Kugel et al. 2008; Reker et al. 2010; Heinzl et al. 2010) as a consistent datum across studies (van der Velde et al. 2013).

The central nucleus of amygdala is an important output nucleus which has direct projection to a variety of anatomical areas that might be expected to be involved in many of the somatic and autonomic signs of fear (Davis and Whalen, 2001; LeDoux, 2012). Firstly, the central nucleus is indirectly connected to brainstem nuclei that mediate the production of affective facial postures (see Cattaneo and Pavesi, 2014). Indeed, electrical stimulation of the amygdala elicits facial movements, which mediate some of the facial expressions seen during fear reactions (Davis and Whalen, 2001; Gloor et al. 1981; Chapman et al. 1954) and a recent study suggests the involvement of amygdala in rapid facial reaction and embodiment of fearful faces (Harrison et al. 2010). Secondly, the amygdalar central nucleus projected to autonomic related centers, including the vagus nerve (LeDoux et al. 1988; Davis and Whalen, 2001; LeDoux, 2012), and modulate neurovegetative manifestations of emotions (Swanson and Petrovich, 1998; Amunts et al. 2005). Indeed, the vagus nerve is involved in bradycardia (Schwaber et al. 1982) and in freezing-like behavior (Davis and Shi, 2000; Terburg et al. 2012; Kalin et al. 2004) generation. In support of these studies, the

stimulation of the central nucleus of amygdala produces freezing and bradycardia (Chapman et al. 1954; Hermans et al. 2013); patients with amygdala lesions have been reported to have deficits in the fear conditioning (LeBar et al. 1995; Bechara et al. 1995) and lesions circumscribed to the central nucleus reduced freezing and bradycardia (Amorapanth et al. 2000). Finally, the present study also found a deficit in the embodiment of the somato-motor aspect of happiness in HA, as revealed by the delayed manifestations of RFRs. This is not surprising since the amygdala is also involved in positive emotions processing, although to a less extent compared to fear (Fusar-Poli et al. 2009; Vytal and Hamann, 2010), through direct projection to the nucleus accumbens in the ventral striatum, which is involved in positive affects (McDonald, 1991, 1992).

To sum up, *Experiment 3* and *Experiment 4* support the hypotheses of a deficit in the embodiment of the somato-motor aspect of fear (*Experiment 3*) and in the autonomic reactions to fearful faces (*Experiment 4*) in patients with high level of alexithymia. These results suggest that HA subjects experience difficulty in understanding emotions because they do not manifest the emotion related behaviors (RFRs and HF changes).

CHAPTER 5

Emotions and the body

“And I understood what he had been looking for! (...)

This water was something entirely different from ordinary nourishment. It was born from the walk under the stars, the singing of the pulley and the effort of my arms. It was good for the heart, like a gift”.

The Little Prince

Antoine de Saint-Exupéry

For the Little Prince, the water he drank in the desert is not only water because it had another meaning for him. For him, that water meant sacrifice, meant the walk under the stars. Similarly, an emotion is not only the bodily changes that accompanied it. An emotion is the meaning that people give to those changes. Alexithymic are known to be too much focused on their own bodily signals (Wise and Mann, 1994; Nyklicek and Vingerhoets, 2000; Nakao et al. 2002). They are also known to misinterpret their bodily signals (Wise and Mann, 1994; Nyklicek and Vingerhoets, 2000; Nakao et al. 2002; Wehmer et al. 1995). So the question is: how do they embody emotions whose characteristic is to induce strong bodily changes?

Experiment 1 showed that the embodiment of fear is defective in HA participants.

Thus, one may wonder if the results of *Experiment 1* could be limited to the emotion of fear or could be expanded to other negative emotions. This topic has been investigated in *Experiment 5*, where the embodiment of fear and disgust were compared between LA and HA participants.

5.1 Introduction

As introduced in Chapter 1, paragraph 1.2, an influential model of emotion processing (Adolphs, 2002a,b) suggests that, during face-to-face interaction, recognizing another's emotion depends on re-experiencing that emotion by activating a representation of it within one's own somatosensory system, which simulates how the other individual would feel when displaying a certain facial expression (Damasio, 1994; Adolphs et al 2000). Thus, the observed emotion is "embodied": we understand the facial expressions, and indeed the emotions of others, by activating similar emotions in ourselves (Niedenthal et al. 2007). Accordingly, neuroimaging studies have revealed that recognizing a facial expression of emotion in another person and experiencing that emotion in oneself involves overlapping neural circuits (Carr et al. 2003).

As already stated in *Experiment 1*, one way to study emotional embodiment is provided by a multisensory stimulation paradigm called emotional Visual Remapping of Touch (eVRT). The eVRT effect is thought to be evoked by means of a preliminary activation of somatosensory cortices when viewing fearful faces, resulting in facilitated processing of tactile information delivered to the participant's face (Cardini et al. 2012). Indeed, fearful faces are highly salient as they might alert the observer to a potential threat that needs to be identified so that a defensive, avoidance response may be prepared. Rapid recognition of such an emotion is therefore critical from an evolutionary perspective. This could be the reason why the enhancement of VRT emerges only with fearful facial expressions (Cardini et al. 2012).

Using the eVRT task, in *Experiment 1* it has been shown that the expected enhancement of the VRT effect with fearful faces, which is present in participants with

low levels of alexithymia (LA), is absent in participants with high levels of alexithymia (HA). HA participants are able to remap the observed touch onto their own face since they are more accurate at detecting the tactile stimuli delivered on their own face when they see a face being touched compared to being only approached. However, they do not show the specific effect of fear on VRT.

Like fear, disgust is a negatively valenced emotion and has been traditionally viewed as priming an avoidance response, since it serves the function of signaling ‘contaminating’ objects, behaviors or persons that are to be avoided (Rozin et al., 1999, 2000; Tybur et al. 2012). Disgust protects the body by discouraging contact with contaminating substances; likewise, moral and interpersonal disgust may protect the individual’s soul and identity, discouraging the endorsement of immoral actions (Rozin et al. 2000; Ciaramelli et al. 2013). Disgust is associated with the appraisal of being too close to something revolting, or to an indigestible object or idea (Lazarus, 1991), and it is characterized by the desire to refuse contact with the offending agent (Rozin et al. 2000). From this point of view, a disgusted face could potentially have the same function as a fearful face, i.e. enhancing vigilance to detect the source of a potential threat in the environment. Considering these functional similarities, one may predict that disgusted faces would have the same effect on VRT as fearful faces; i.e. an enhancement of the VRT effect relative to neutral faces in LA but not HA participants. We will refer to this hypothesis as the *functional* hypothesis.

However, the physiological underpinnings of disgust might suggest a different outcome on VRT in HA participants. For example, disgust tends to activate parasympathetic responses, reducing heart rate, blood pressure, and respiration (Ekman et al., 1983; Levenson et al. 1990). Conversely, fear usually swings these systems in the

opposite direction by stimulating sympathetic pathways, increasing heart rate and startle reflex (Levenson, 2003). Moreover, the facial configuration elicited by these two emotions are different as well: when subjects are exposed to a disgusting stimuli they manifest a smaller visual field and decrease in nasal volume and air velocity inspiration, while the opposite pattern was found when subjects are exposed to fearful stimuli (Susskind et al. 2008; Susskind and Anderson, 2008). A further difference between fear and disgust refers to their neural basis: the emotion of fear predominantly activates the amygdala (Vytal and Hamann, 2010; LeDoux, 2013; Johansen et al. 2010; Wilensky et al. 2006; Mobbs et al. 2009), whereas the emotion of disgust predominantly activates the insula (Vytal and Hamann, 2010; Anderson et al. 2003; Wright et al. 2004; Phillips et al., 1997; Wicker et al., 2003). Finally, disgust is often considered the most visceral of all the basic emotions (Harrison et al. 2010), eliciting peripheral bodily changes that facilitate the protection of the body from contaminating objects (Chapman and Anderson, 2012; Curtis, 2011), while fear enhances the vigilance to detect the source of a potential threat in the environment aiming to prepare the body to react to the external threats (LeDoux, 2013). Considering that alexithymic individuals are known to enhance the normal visceral phenomena accompanying emotional arousal (Wise and Mann, 1994; Nyklicek and Vingerhoets, 2000; Nakao et al. 2002) and the above reported physiological differences between the emotions of fear and disgust, one may predict that disgusted faces would have a different effect on VRT compared with fearful faces; i.e. an enhancement of the VRT effect relative to neutral faces in HA but not LA participants. We will refer to this second hypothesis as the *physiological* hypothesis.

To test these two alternative hypotheses, i.e. the *functional* hypothesis and the *physiological* hypothesis, participants with low and high levels of alexithymia took part in a tactile confrontation task (Serino et al. 2008; 2009): participants were touched on the left cheek, the right cheek, or both cheeks simultaneously, and were required to report the side of stimulation. To simulate tactile extinction (Bender, 1952; Ladavas, 2002), stimulus intensity on one cheek was stronger than on the other cheek. In line with previous results, the *a-priori* prediction was that, in trials of bilateral tactile stimulation, the stronger stimulus would frequently extinguish the weaker one (Serino et al., 2008; 2009). While performing the tactile confrontation task, participants watched a human face with a fearful expression, a disgusted expression, or a neutral expression (Eckman and Friesen, 1976) being touched or just approached, unilaterally or bilaterally, by one or two human fingers. Different results were predicted by the *functional* and *physiological* hypotheses.

5.2 EXPERIMENT 5: Dissociation between Emotional Remapping of Fear and Disgust

5.2.1 Material and Methods

Participants: 300 university students completed the 20-item Toronto Alexithymia Scale (TAS-20; Taylor et al. 2003). Individuals with high and low total TAS-20 scores (top quartile score >60; bottom quartile score <39) 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 et al. 2006), previously used in alexithymia research (Grandi et al. 2011), was used to further confirm the presence or absence of alexithymia. Moreover, due to the

high association between alexithymia and depression (Honkalampi et al. 2000; Hintikka et al. 2001), the Italian version of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al. 1997), mood disorders subscale, was used 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 DPCR were congruent. Three participants with a high TAS-20 score and a low DCPR score were discarded, as well as a participant with a high TAS-20 score who reported a high level of depression on the SCID. *Table 5.1* gives comparative information about the resulting low alexithymia (LA; n=20) and high alexithymia (HA; n=20) groups. All participants had equivalent educational backgrounds and were students at the University of Bologna.

	Low Alexithymia	High Alexithymia
n (male/female)	20 (5/15)	20 (5/15)
Age, mean (SD) (years)	22.6 (1.66)	21.8 (1.22)
<i>TAS-20</i>	<i>Minimum-maximum, mean (SD)</i>	
Total	26-39, 35.1 (3.5)	61-77, 65.5 (5.3)
DIF	8-18, 12.2 (2.6)	19-32, 24.9 (3.7)
DDF	5-12, 9.1 (2.2)	16-24, 19.9 (2.4)
EOT	10-16, 13.5 (2)	12-29, 20.4 (4.9)
DPCR	0-2, 1.1 (0.6)	3-5, 3.6 (0.7)

Table 5.1. Demographic and alexithymia profile of low and high alexithymia groups. TAS-20: twenty-item Toronto Alexithymia Scale; DIF: difficulty in identifying feelings; DDF: difficulty in describing feelings; EOT: externally oriented thinking. SD: standard deviation. Low Alexithymia (n=20) and High Alexithymia (n=20) groups were obtained by selecting volunteers with low or high total scores on the TAS-20. Participants with discrepant TAS-20 and DPCR scores were excluded (cf. Material and Methods). DPCR: Alexithymia Module of Diagnostic Criteria for Psychosomatic Research scores.

Sample size was determined a priori by conducting a power analysis using G*Power 3 (Faul et al. 2007). A small to medium effect size ($\eta_p^2 = 0.19$) was specified for group differences on viewing different emotional facial expressions, based on the results of *Experiment 1*. Within the chosen sample size and effect size, the power ($1 - \beta$) was approximately 0.90.

Emotional VRT: To test the ability of participants to remap others' emotions onto their own body, an indirect task was used: Emotional Visual Remapping of Touch (eVRT, Cardini et al. 2012).

Materials: Three human faces showing fearful, disgusted, and neutral facial expressions were chosen. Human faces were taken from the Pictures of Facial Affect dataset (Eckman and Friesen, 1976). Short (3000 ms) videos were created in Microsoft Power Point that showed each face on a black background being either touched or approached by one or two human fingers. A computer running C.I.R.O software (<http://www.cnc.unibo.psice.unibo/ciro>) displayed the visual stimuli and collected responses. Electro-tactile stimulation was delivered via two constant current electrical stimulators (DS7A, Digitimer) connected to two pairs of electrodes (Neuroline, AMBU), one on each side of the participant's face over the zygomatic arch.

Procedure: Following the staircase procedure used by Cardini and colleagues (2012), the detection rate of electro-tactile stimulation was set to nearly 100% on one cheek and to approximately 60% on the other. The cheek that received stronger electro-tactile stimulation (left or right) was counterbalanced between participants. Confirming correct calibration, the mean detection rate of bilateral tactile stimulation across all experimental conditions was 53.02% (SEM = $\pm 1.14\%$), and, when bilateral stimulation

was not correctly identified, errors mostly consisted of reporting unilateral stimulation on the stronger side ($M = 95.1\%$ of errors, $SEM = \pm 1.32\%$).

The experiment consisted of six blocks of VRT trials, two with neutral faces, two with fearful faces, and two with disgusted faces. Block order was counterbalanced between participants, and electro-tactile detection thresholds were re-calibrated between blocks. Each trial began with a face in the center of the screen and two fingers at the bottom of the screen on either side of the chin. One or both of the fingers then moved upward and either touched the cheek on the same side of the screen or touched a location about 5 cm lateral to the face before returning to the bottom of the screen. When the fingers reached the top of their trajectory (approximately 800 ms into the trial), electro-tactile stimulation was delivered to one or both of the participant's cheeks. Participants used a keyboard to indicate whether they felt touch on the left cheek, on the right cheek, or on both cheeks. They were instructed to respond as quickly and accurately as possible, and informed that the location of apparent touch on the cheeks of the observed face was non-informative about the touch on their own face. Each trial combined one of two types of tactile stimulation (unilateral or bilateral), one of two types of visual stimulation (unilateral or bilateral), and one of two types of finger trajectories (touch or no-touch), resulting in 8 trial types that were repeated 12 times each block for a total of 96 trials per block, presented in a random order. Only trials with both bilateral tactile stimulation and bilateral finger movement (touch or no-touch) were analyzed.

Direct Task: The direct emotion recognition task consisted of arousal and valence ratings of emotional facial expressions, as well as explicit emotion recognition and labeling. The very same Eckman faces used in the eVRT were presented to

participants: three different actors displaying fearful, disgusted and neutral expressions. Participants were asked to rate arousal and valence using a nine-point Likert scale, with 1 meaning “not very arousing” or “very negative emotion” and 9 meaning “highly arousing” or “very positive emotion.” Participants were also provided with a list of the six basic emotions, and they were required to label the emotion expressed in each Eckman face. Moreover, to assess whether the eVRT results could be related to individual differences in disgust sensitivity, the Disgust Scale (DS, [Olatunji et al. 2007](#)), a 32-item self-report questionnaire, was administered. The Disgust Scale is formed of two subscales that measure personal reactions to disgusting stimuli and personal evaluations of disgusting situations.

5.2.2 Results

eVRT task:

To investigate the effect of viewing emotional facial expressions on the VRT effect in alexithymia, the accuracy of HA and LA participants in identifying bilateral tactile stimulation was compared between trials in which both fingers touched or merely approached the observed face. An analysis of variance (ANOVA) was conducted on bilateral stimulation detection accuracy using Group (two levels: Low and High alexithymia) as a between-subjects variable, and Emotion (three levels: Fear, Disgust and Neutral) and Finger Trajectory (two levels: No-Touch and Touch) as within-subjects variables. Post-hoc comparisons (Duncan’s tests) were performed, when necessary, to compare single effects. The partial eta squared (η_p^2) was reported as an estimate of effect size ([Cohen, 1969](#)). The analysis revealed a significant main effect of Finger Trajectory ($F[1,38]=107.6$; $p<0.001$; $\eta_p^2 = 0.74$). Moreover, the critical triple

interaction Group x Emotion x Finger Trajectory was significant ($F[2,76]=14.7$; $p<0.001$; $\eta_p^2 = 0.28$). Consequently, ANOVAs on bilateral stimulation detection accuracy using Emotion and Finger Trajectory as within-subjects variables were performed separately for the LA and HA groups.

In the LA group, the analysis revealed a significant main effect of Finger Trajectory ($F[1,19]=60$; $p<0.001$; $\eta_p^2 = 0.76$). Moreover, the critical double interaction Emotion x Finger Trajectory was also significant ($F[2,38]=10.2$; $p=0.001$; $\eta_p^2 = 0.35$). Post-hoc tests showed that the Touch condition enhanced the accuracy of bilateral stimulation perception compared to the No-Touch condition for neutral faces (mean accuracy No-Touch: $57.7\% \pm 2.9\%$, mean accuracy Touch: $69.2\% \pm 2.6\%$, $p=0.002$), fearful faces ($57.6\% \pm 3.5\%$ vs $78.4\% \pm 3.4\%$, $p<0.001$) and disgusted faces ($60.4\% \pm 3\%$ vs $66.7\% \pm 3.8\%$, $p<0.001$). In the Touch condition, the accuracy of bilateral stimulation detection was higher for fearful faces ($78.4\% \pm 3.4\%$) than for disgusted faces ($66.7\% \pm 3.8\%$, $p<0.001$) or neutral faces ($69.2\% \pm 2.6\%$, $p<0.0001$), and there was no difference between accuracy for neutral faces ($69.2\% \pm 2.6\%$) and disgusted faces ($66.7\% \pm 3.8\%$). Moreover, in the No-Touch condition there were no differences in the accuracy of bilateral stimulation detection between the three emotions: fear ($57.6\% \pm 3.5\%$), disgust ($60.4\% \pm 3\%$) and neutral ($57.7\% \pm 2.9\%$). This is important for ruling out the possibility that the results are due to increased attention to fearful or disgusted faces compared to neutral faces.

In the HA group, the analysis revealed a main effect of Finger Trajectory ($F[1,19]=47.7$; $p<0.001$; $\eta_p^2 = 0.72$). Critically, the double interaction Emotion x Finger Trajectory was significant ($F[2,38]=5.7$; $p=0.006$; $\eta_p^2 = 0.23$). Post-hoc tests showed that the Touch condition enhanced the accuracy of bilateral stimulation perception

compared to the No-Touch condition for neutral faces (mean accuracy no touch: 59.5%±2.3%, mean accuracy touch: 68.3%±3%, $p<0.001$), disgusted faces (56.6%±1.7% vs 73.6%±2.1%, $p<0.001$), and fearful faces (59.5%±2.7% vs 66%±2.9%, $p=0.007$). In addition, in the Touch condition the accuracy of bilateral stimulation detection was higher for disgusted faces (73.6%±2.1%) than for fearful (66%±2.9%, $p=0.002$) or neutral faces (68.3%±3%, $p=0.02$), but there was no difference between accuracy for neutral faces (68.3%±3%) and fearful faces (66%±2.9%). Moreover, in the No-Touch condition there were no differences in the accuracy of bilateral stimulation detection between the three emotions: fear (59.5%±2.7%), disgust (56.6%±1.7%), and neutral (59.5%±2.3%). Again, this is important for ruling out the possibility that the results are due to increased attention to fearful or disgusted faces. Results are depicted in *Figure 5.1*.

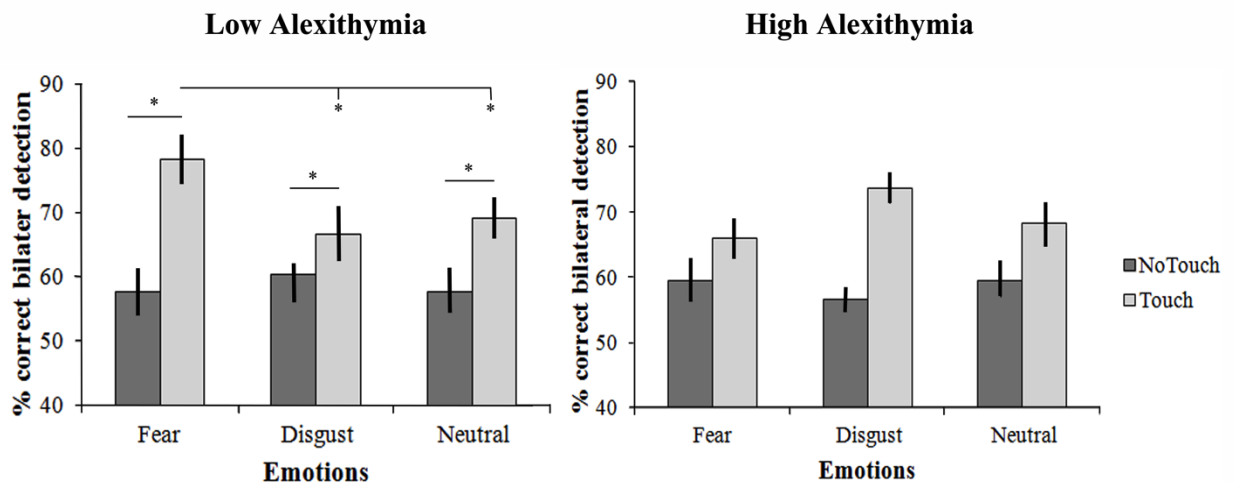


Figure 5.1. Results from the eVRT task in LA and HA participants, expressed as accuracy in detecting bilateral tactile stimulation while viewing movies showing either a fearful face, a disgusted face or a neutral face touched or just approached by two human fingers. Error bars show standard errors of the mean across LA and HA participants.

To further explore the differences between the LA and HA groups in emotional modulation of tactile perception, an emotional VRT index was calculated by taking the difference between accuracy of bilateral stimulation detection in the No-Touch and Touch conditions (Touch – No-Touch). Emotional VRT indices for each emotion were compared in LA and HA participants. An ANOVA was performed on VRT indices using Group (two levels: LA and HA) as a between-subjects variable and Emotion (three levels: Fear, Disgust and Neutral) as a within-subjects variable. The analysis revealed a significant Group x Emotion interaction ($F[2,76]=14.7$, $p<0.001$; $\eta_p^2 = 0.28$). Post-hoc tests in the LA group showed that the VRT index was bigger (i.e. bigger difference between No-Touch and Touch) when participants saw fearful faces ($20.8\% \pm 2.7\%$) than when they saw disgusted ($6.3\% \pm 2.3\%$, $p=0.001$) or neutral faces ($11.5\% \pm 2.4\%$, $p=0.007$), whereas there was no difference between neutral and disgusted expression conditions. On the contrary, in the HA group the VRT index was bigger when participants saw disgusted faces ($17\% \pm 1.7\%$) than when they saw fearful ($6.5\% \pm 3.3\%$, $p=0.003$) or neutral faces ($8.8\% \pm 1.9\%$, $p=0.01$), whereas there was no difference between neutral and fearful expression conditions. The results are shown in *Figure 5.2*.

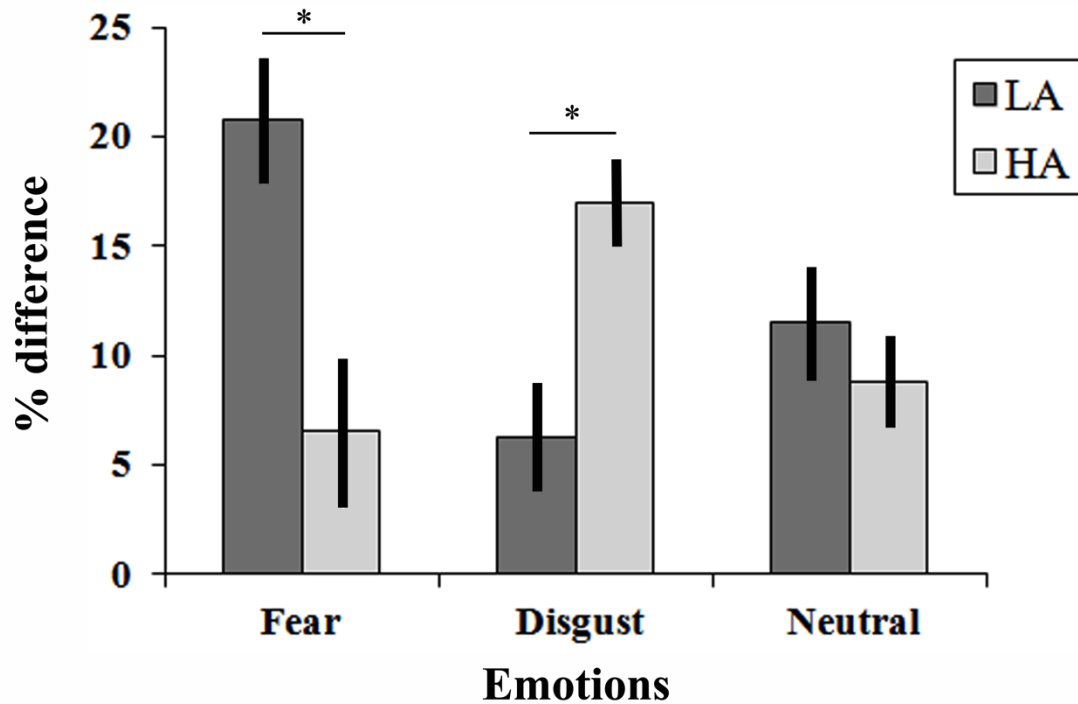


Figure 5.2. Results from the eVRT task in LA and HA participants, expressed as the difference in bilateral tactile detection accuracy between Touch and No-Touch conditions while viewing either a fearful face, a disgusted face or a neutral face. Error bars show standard errors of the mean across LA and HA participants.

Explicit Task: To investigate the explicit emotion recognition profile in alexithymia, LA and HA participants' explicit arousal and valence ratings of the emotional facial expressions were compared. Moreover, participants were asked to explicitly name the emotion expressed by each face. Mean arousal and valence ratings and the number of emotion naming errors were analyzed using mixed factors ANOVAs with Group (two levels: LA and HA) as a between-subjects variable and Emotion (three levels: Fear, Disgust and Neutral) as a within-subjects variable.

With respect to arousal ratings, a significant main effect of Emotion ($F[2,76]=642.4$; $p<0.001$; $\eta_p^2 = 0.94$) was found. Fearful (mean rate: 7.6 ± 0.11) and disgusted (7.1 ± 0.17) faces were rated as more arousing than neutral ones (1.6 ± 0.11 ,

$p < 0.001$), and fearful faces were rated as more arousing than disgusted ones ($p = 0.006$). The main effect of Group and the interaction between Group and Emotion were not significant. With respect to valence ratings, the analysis revealed a significant effect of Emotion ($F[2,76] = 193$; $p < 0.001$; $\eta_p^2 = 0.84$). Post-hoc tests revealed that valence ratings for fearful (mean rate: 2.4 ± 0.11) and disgusted faces (2.3 ± 0.12) were significantly lower than for neutral faces (4.7 ± 0.07 , $p < 0.001$), whereas no difference was found between fearful and disgusted face ratings. The main effect of Group and the interaction between Group and Emotion were not significant. With respect to explicit emotion recognition, a significant main effect of Group ($F[1,38] = 9$; $p = 0.004$; $\eta_p^2 = 0.2$) was found. HA participants made more errors in overall emotion identification compared to LA participants (0.98 ± 0.15 and 0.58 ± 0.18 errors, respectively, $p = 0.004$), but the interaction between Group and Emotion was not significant ($F[2,76] = 1.12$, $p = 0.33$; $\eta_p^2 = 0.02$).

Finally, to investigate whether eVRT findings could be related to individual differences in disgust sensitivity, LA and HA scores on the Disgust Scale and its subscales were compared by means of independent samples t-tests. LA and HA participants differed neither in their total DS score (mean 16.24 ± 0.65 and 15.32 ± 0.87 for LA and HA, respectively, $t = 0.77$, $df = 38$, $p = 0.44$, $\eta_p^2 = 0.01$), nor in their scores on the DS subscales (personal reaction subscale: 7.58 ± 0.38 and 7.64 ± 0.53 for LA and HA, respectively; $t = -0.08$, $df = 38$, $p = 0.93$, $\eta_p^2 = 0.002$; personal evaluation subscale: 8.59 ± 0.42 and 7.74 ± 0.46 for LA and HA, respectively; $t = 1.25$, $df = 38$, $p = 0.21$, $\eta_p^2 = 0.05$).

Altogether, these results suggest that the eVRT findings cannot be explained by differences in explicit emotion recognition and/or differences in disgust sensitivity between the groups.

5.2.3 Discussion of Experiment 5

In keeping with previous results (Serino et al. 2008, 2009), the present findings confirm that viewing a face being touched enhances tactile perception on one's own face, and that facial expressions of fear modulate this VRT effect (Cardini et al. 2012) in LA but not in HA participants (*Experiment 1*). The new finding of the present study is that, unlike fearful expressions, expressions of disgust modulate the VRT effect in HA but not in LA participants.

The present finding regarding eVRT for disgust cannot be explained in terms of a generic arousal effect induced by disgusted faces because the bilateral stimulation detection accuracy of the two groups did not differ in the No-Touch condition, nor could it result from differences in the perceived intensity of the emotional stimuli or sensitivity to disgusting stimuli between groups because the HA participants' arousal ratings and disgust sensitivity scores did not differ from those of the LA participants. The stronger VRT effect found in HA participants when the observed face showed a disgusted expression compared to a neutral or a fearful expression is in line with the *physiological* hypothesis. According to this hypothesis, this disgust-specific enhancement might reflect the different physiological responses induced by the experience of disgust and fear.

Indeed, consistent with its apparent origin in defending against the ingestion of contaminated food (Chapman and Anderson, 2012; Curtis, 2011), disgust is strongly

associated with visceral changes, decreasing sensory interactions with the environment and redirecting one's attention from the environment to internal (bodily) states (Vermeulen et al. 2009). The eVRT findings of heightened disgust remapping in HA suggest that alexithymics might exhibit increased responses to stimuli producing heightened “physical” and visceral sensations, and that HA might mainly rely on “physical” information while performing the multisensory stimulation paradigm. Indeed, alexithymia is classically characterized by an amplification of the normal visceral/somatic phenomena accompanying emotional arousal and to experience bodily sensations in an emotion-provoking situation (Karlsson et al. 2008). Moreover, alexithymics are more likely to be focused on those amplified bodily sensations. To account for this heightened focusing on bodily sensation, the “somatosensory amplification” hypothesis (Wise and Mann, 1994; Nyklicek & Vingerhoets, 2000; Nakao et al. 2002; Kano et al. 2007) has been proposed. In accordance with this hypothesis, and given that the emotion of disgust elicits strong visceral changes (Harrison et al. 2010), it is possible to hypothesize that the previously described results of eVRT modulation by disgusted faces might be due to the higher ability of HA participants in perceiving their own bodily signals.

To address this possibility, the same participants of *Experiment 5* underwent another experiment (*Experiment 6*), in which two indices of interoception ability have been evaluated: interoceptive sensibility and interoceptive sensitivity. In line with Garfinkel and Critchley (2013), interoceptive sensibility (ISb), defined as a dispositional tendency to be internally focused, has been evaluated using the Bodily Perception Questionnaire (BPQ) (Porges, 1993); while interoceptive sensitivity (ISt), defined as the objective accuracy in detecting internal bodily sensations, has been

evaluated using the Heartbeat Perception Task (Schandry, 1981). It was expected that HA would have higher ISb and ISt compared with LA participants.

Critically, the aim of *Experiment 6* was to investigate a possible association between the eVRT results and the interoceptive abilities. To this aim, firstly the eVRT results were correlated with the interoceptive results, with the *a priori* prediction that the two interoception indices would correlate with the eVRT results for disgust; then the ANOVAs on the eVRT indices were repeated using ISb and ISt as covariates, with the *a priori* prediction that including the ISb or the ISt as a covariate would eliminate the association between alexithymia and eVRT. Both these expected results would suggest that the differences in eVRT between HA and LA groups were due to HA participants' tendency to be focused on their own bodily signals (Wise and Mann, 1994; Nyklicek & Vingerhoets, 2000; Nakao et al. 2002; Kano et al. 2007).

5.3 EXPERIMENT 6: The role of interoception in embodiment of disgust.

In this second experiment, the hypotheses of whether LA and HA participants show different degrees of interoceptive sensibility (ISb) and sensitivity (ISt) and whether the degree of ISb and ISt correlates with eVRT effects were tested.

5.3.1 Materials and Methods

Participants: The participants from *Experiment 5* were asked to come back to participate in the second experiment. All 20 LA (5 male) and 20 HA (5 male) volunteers returned for the second experiment.

Interoceptive Sensibility (ISb): The Body Perception Questionnaire (BPQ) was adopted as a measure of interoceptive sensibility (ISb). The BPQ (Porges, 1993) is a 96-item self-report instrument that assesses body perception and interoceptive awareness on four subscales: i) awareness subscale: participants report how aware they are of their bodily processes (e.g. swallowing frequently); ii) stress response: participants imagine being in a very stressful situation and rate their bodily changes due to that situation (e.g. emotional problems such as more frequent feelings of depression, frustration, rage or anger); iii) autonomic nervous system reactivity: participants respond to statements about their autonomic nervous system reactions (e.g. ‘my heart often beats irregularly’); iv) stress style subscale: participants evaluate the manner in which they respond to stress (e.g. ‘I have difficulty speaking’). Each item is rated on a five-point Likert scale ranging from 1 (never) to 5 (always). The higher the score, the stronger the participant’s perception of bodily sensations and interoceptive awareness.

Interoceptive Sensitivity (ISt): The Heartbeat Perception Task was used as a measure of interoceptive sensitivity (ISt). For the heartbeat perception task, ECG 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 the corresponding software, AcqKnowledge (BIOPAC Systems Inc., Santa Barbara, CA). The heartbeat perception task was performed according to the Mental Tracking Method proposed by Schandry (1981), using four intervals of 25, 35, 45, and 55 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. IS_t was measured as a heartbeat perception score, calculated by taking the mean score across the four heartbeat perception intervals according to the following transformation: $1/4 \sum (1 - (|\text{recorded heartbeats} - \text{counted heartbeats}|) / \text{recorded heartbeats})$. 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 IS_t (Dunn et al. 2007; Herbert et al. 2007), has good test-retest reliability (up to .81), and correlates highly with other heartbeat detection tasks (Knoll and Hodapp, 1992).

5.3.2 Results

Interoceptive Sensibility (IS_b): Independent samples t-tests were conducted on the BPQ total and subscale scores of the HA and LA participants. The two groups significantly differed in total BPQ score (210.2 ± 9.82 and 262.3 ± 6.57 for LA and HA respectively, $t=-4.4$, $df=38$, $p<0.001$, $\eta_p^2 = 0.34$). Moreover, scores on each of the four subscales differed between groups (awareness subscale: 107.4 ± 7.22 and 137.9 ± 3.74 for LA and HA respectively, $t=-3.74$, $df=38$, $p=0.001$, $\eta_p^2 = 0.27$; stress response: 26.7 ± 1.86 and 33.7 ± 1.47 for LA and HA respectively, $t=-2.96$, $df=38$, $p=0.005$, $\eta_p^2 = 0.19$; autonomic nervous system reactivity: 46.4 ± 2 and 57 ± 2.9 for LA and HA respectively, $t=-2.91$, $df=38$, $p=0.006$, $\eta_p^2 = 0.18$; stress style subscale: 29.7 ± 0.95 and 33.6 ± 1.15 for LA and HA respectively, $t=-2.64$, $df=38$, $p=0.012$, $\eta_p^2 = 0.16$). After

correction for multiple comparisons, the differences between groups on the BPQ total score and the awareness subscale were still significant. Overall, those results reveal higher interoceptive sensibility in HA compared to LA participants.

Interoceptive Sensitivity (IS_t): Independent samples t-tests were conducted on the IS_t indices (heartbeat perception scores) of the LA and HA groups. The two groups significantly differed in heartbeat perception ability (0.63 ± 0.03 and 0.92 ± 0.01 for LA and HA respectively, $t=-7.7$, $df=38$, $p<0.001$, $\eta_p^2 = 0.61$), with HA participants showing higher IS_t than LA participants.

Link between IS_b and VRT performance: To investigate the link between IS_b and eVRT, Spearman correlations were performed across group between eVRT indices for the three emotions (neutral, fear and disgust) and the IS_b as measured by the total BPQ score. No correlations were significant (eVRT for fear and IS_b: $r=-0.26$, $p=0.1$; eVRT for disgust and IS_b: $r=0.21$, $p=0.18$; ; eVRT for neutral and IS_b: $r=-0.14$, $p=0.37$).

Link between IS_t and VRT performance: To investigate the link between IS_t and eVRT, Spearman correlations between eVRT indices for the three emotions (neutral, fear and disgust) and the IS_t index (heartbeat perception score) were performed across groups. There was a negative correlation between IS_t and eVRT for fear ($r=-0.38$, $p=0.01$), but a positive correlation between IS_t and eVRT for disgust ($r=0.5$, $p<0.001$). No correlation was found between IS_t and eVRT for neutral faces ($r=-0.09$, $p=0.57$). After correcting for multiple comparisons, the correlation between IS_t and disgust and IS_t and fear were still significant. In other words, the higher the IS_t score, the bigger the

eVRT index for disgust and the lower the eVRT for fear. This suggests that the eVRT effect for disgust and fear were linked to interoceptive signals. Results are illustrated in *Figure 5.3*.

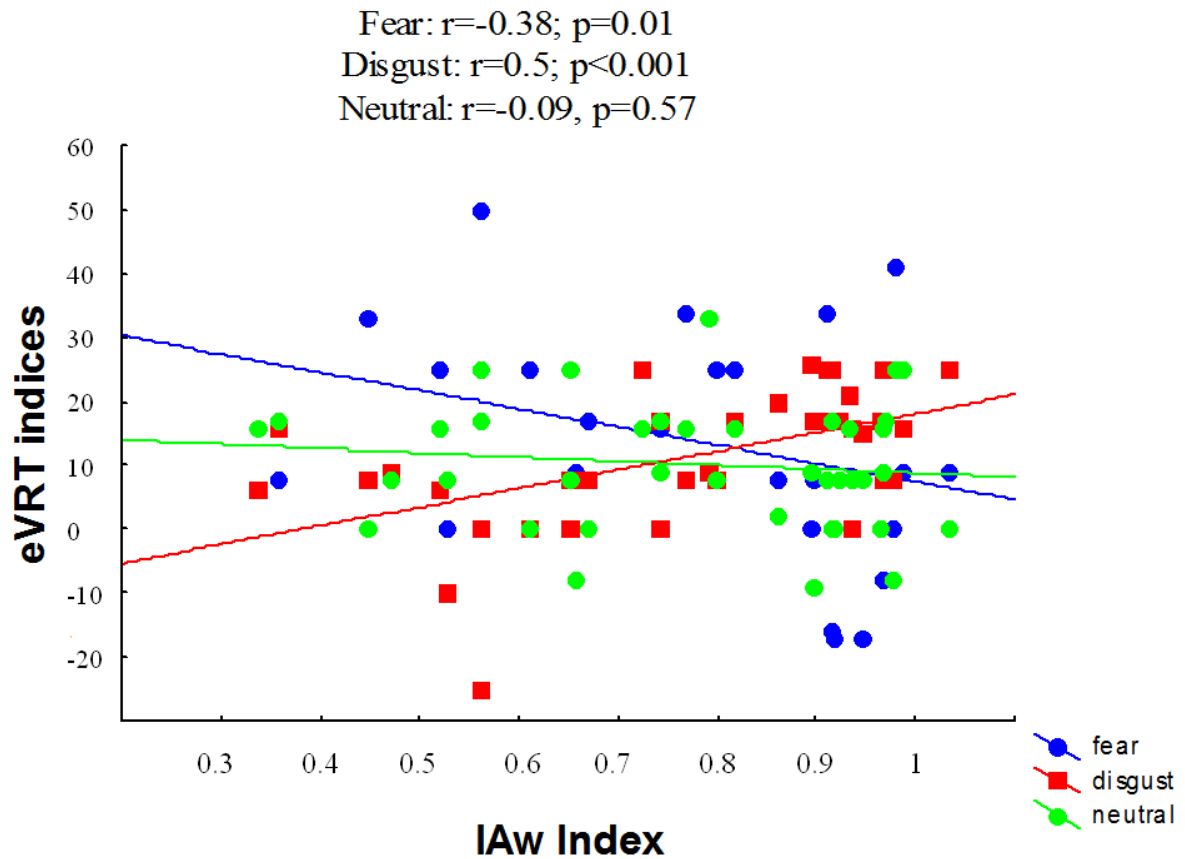


Figure 5.3. Correlations between eVRT indices for fearful, disgusted and neutral expressions and the IST index (heartbeat perception score).

Furthermore, according to the hypothesis that IST should predict the eVRT disgust effect in alexithymia, the IST index (heartbeat perception score) was added as a covariate to the ANOVA on bilateral stimuli detection accuracy with Group as a between-subjects variable and Emotion and Finger Trajectory as a within-subjects variables. With IST as a covariate, the main effect of Finger Trajectory was no more significant ($F[2,37]=0.28$;

$p=0.59$; $\eta_p^2 = 0.007$). Although the Group x Emotion x Finger Trajectory interaction was still significant ($F[2,74]=4.01$; $p=0.02$; $\eta_p^2 = 0.1$), when analyzing each alexithymia group individually, the interaction Emotion x Finger Trajectory was not significant either in the LA ($F[2,36]=0.99$; $p=0.38$; $\eta_p^2 = 0.05$) or in the HA ($F[2,36]=0.52$; $p=0.59$; $\eta_p^2 = 0.02$) group. This result indicates that the differences in eVRT between HA and LA groups were linked to the HA participants' heightened focus on their own body signals.

5.3.3 Discussion of Experiment 6

The critical findings of *Experiment 6* were that the interoceptive sensitivity correlates with the eVRT effect for disgusted faces, and that, if the influence of the interoceptive sensitivity were removed from the eVRT analysis, the eVRT effect for disgusted faces was no longer present. Importantly, the significant association between ISt and the eVRT effect for disgust suggests that the greater the ability to perceive bodily (i.e. cardiovascular) signals, the stronger the remapping of others' disgusted expressions onto oneself. By contrast, no correlation was found between ISt and the eVRT indices for fearful and neutral expressions. These results indicate that the eVRT results for disgust are strictly associated with the interoceptive abilities of participants, i.e. with their tendency to be focused on their own bodily signals and, as a consequence, being more able to detect them. These results support the hypothesis that HA might rely on “physical” and bodily information while performing the multisensory stimulation paradigm, since they exhibit increased responses to stimuli producing heightened “physical” and visceral sensations.

Furthermore, these results confirm the “somatosensory amplification” hypothesis (Wise and Mann, 1994; Nyklicek and Vingerhoets, 2000; Nakao et al. 2002; Kano et al. 2007; Wehmer et al. 1995) and are in accordance with our *physiological* hypothesis, stating that the physiological underpinnings of disgust may play a crucial role in modulating the activity of somatosensory cortices during eVRT with disgusted faces.

A secondary result of *Experiment 6* is that HA participants showed higher interoception, both ISb and ISt, than LA participants. These findings are in line with recent work using BPQ (ISb) (Ernst et al. 2013), but not with other results from the heartbeat perception task (ISt) (Herbert et al. 2011). However, the discrepancy between the previous heartbeat perception study and the present results might be due to methodological differences, for instance, in the participant inclusion procedure. Indeed, in Herbert et al.’s (2011) study only the TAS-20 was used, whereas in the present study an highly selective procedure was adopted, involving the TAS-20, a semi-structured interview (DCPR) and excluding participants with clinical depression. As a consequence, the present study has the advantage of a higher specificity in participant selection. Moreover, the statistical analyses conducted were different as well. Contrarily to the current study, in which we conducted direct comparisons on two groups of participants including participants scoring in the upper or lower quartile of TAS-20, Herbert et al. (2011) included all the participants regardless of their TAS-20 score and performed a regression analysis between TAS-20 score and heartbeat perception task. As a consequence, their participants TAS score had a small variance, since few real alexithymic subjects had been included.

5.4 Discussion

The present studies investigated the mechanisms of embodiment of emotions in people with impaired ability to identify and describe one's own emotional state, such as alexithymia. In particular, the aim was to understand whether the emotion embodiment difficulties exhibited by alexithymic individuals when observing fearful faces (*Experiment 1*), were also present during the observation of disgusted faces. Accordingly, the way in which these two negatively valenced emotions, namely fear and disgust, were embodied in alexithymic subjects has been investigated by means of the eVRT paradigm. Two possible effects of disgusted and fearful expressions on eVRT performance have been hypothesized. On the one hand, the *functional* hypothesis highlighted the similarity between fearful and disgusted expressions in signaling a potential threat in the environment. Because the remapping mechanism is automatically evoked in order to understand the nature of the threat signaled by the emotional expression (Cardini et al, 2012), the functional hypothesis suggested that, like the eVRT effect with fearful faces, viewing touch on a disgusted face should enhance tactile perception on the face in LA but not in HA participants, because of the latter's difficulty in remapping threat-indicative expressions, such as fear onto the self. This remapping might be useful in priming avoidance reactions, which is a shared function between fear and disgust.

On the other hand, the *physiological* hypothesis emphasized the distinct physiological underpinnings (i.e. autonomic reactions, facial configuration, neural basis) for fear and disgust. Indeed, compelling evidence has shown that disgust is the most embodied and visceral of all basic emotions (Harrison et al., 2010), since the observation of disgusting stimuli, both objects and faces, elicits strong visceral and

physiological reactions (Curtis, 2011), different from those evoked by fear. Moreover, individuals with higher alexithymia scores have a tendency to rely on, or to amplify their physical symptoms (Kano et al., 2007), and exhibit “hypersensitivity” and increased neural response to stimuli accompanied by a ‘physical’ context, such as somatosensory or sensorimotor processes (Moriguchi and Komaki, 2013). Based on these premises, the physiological hypothesis predicted that viewing touch on a face showing a disgusted expression should enhance perception of tactile stimuli on the face in people with HA but not in people with LA.

The results of *Experiment 5* are in line with the *physiological* hypothesis, as they reveal a clear dissociation between the remapping of fearful and disgusted expressions in low and high alexithymic participants. Indeed, while the emotion of fear is remapped in LA but not in HA participants, the opposite pattern is true for the emotion of disgust.

In the following paragraphs, the results concerning LA and HA participants will be discussed separately.

In LA participants, the remapping of fearful expressions replicates the findings of previous studies that used the same paradigm in normal populations (Cardini et al., 2012), and in participants with low level of alexithymia (*Experiment 1*), namely an enhancement of the VRT effect when participants viewed fearful expressions, probably reflecting a fear-related preactivation of the fronto-parietal network and related somatosensory cortices involved in the VRT mechanism (Cardini et al., 2011). The absence of enhanced VRT when LA participants viewed disgusted expressions, relative to neutral expressions, further corroborates the previous hypothesis that the emotion-related enhancement of the VRT effect is specific to fearful expressions in non-alexithymic individuals (Cardini et al., 2012), since this enhancement is not elicited by

positive emotional expressions (i.e. happiness: [Cardini et al., 2012](#); *Experiment 1*), or by other negative emotional expressions, such as anger ([Cardini et al., 2012](#)), and disgust (present results).

The most interesting findings of the present study concern the HA group, however. The results of eVRT with fearful expressions in HA participants replicate those of our previous study using the same paradigm in alexithymia (*Experiment 1*): the expected enhancement of the VRT effect when viewing fearful faces is absent in HA participants. This result has been interpreted as an inability of HA participants to remap the fearful expressions of others onto their own somatosensory system. This is probably due to a hypoactivation of the amygdala in alexithymia ([van der Velde et al. 2013](#)), which might fail to preactivate somatosensory cortices.

The novel results of the current study show that alexithymic participants have an abnormally strong response to disgust, manifested as enhanced disgust remapping compared to LA participants. The physiological hypothesis stated that alexithymic participants, which are mainly focused on their own physical, rather than mental, states ([Wise and Mann, 1994](#); [Nyklicek & Vingerhoets, 2000](#); [Nakao et al. 2002](#); [Wehmer et al. 1995](#)), might show an enhancement of the VRT effect for emotional stimuli that are accompanied by strong physical and visceral sensations. Specifically, the enhanced modulation of eVRT by disgusted faces has been hypothesized to be related to the higher ability of HA participants in perceiving and reporting their own bodily signals ([Karlsson et al. 2008](#); [Ernst et al. 2013](#)). Thus, *Experiment 6* has been conducted, in which participants' interoceptive abilities, namely interoceptive sensitivity and sensibility, were correlated with their eVRT performance and compared between LA and HA participants. The results of this second experiment corroborated the *a-priori* hypothesis

that HA might mainly rely on physical and bodily information while performing the multisensory stimulation paradigm. The interoceptive abilities of HA participants resulted tightly linked to the eVRT performance for disgusted faces. The association between interoception and eVRT results suggests that the greater the ability to perceive bodily (i.e. cardiovascular) signals, the stronger the remapping of others' disgusted expressions onto oneself.

Previous findings showed that environmental stimuli trigger emotional responses that are associated with an impoverished conscious experience of emotion in HA individuals. Instead, HA individuals exhibit the tendency to experience physical sensations in response to emotion-provoking stimuli (Karlsson et al. 2008; Kano et al. 2007), thereby suggesting a different mode of emotion processing in alexithymia. The present data suggest that this phenomenon might be more pronounced for emotions associated with stronger bodily changes, namely the disgust, thereby explaining the higher eVRT for disgust in HA subjects observed here. Our results have crucial implications for alexithymia research, since they reveal that negative emotions are abnormally embodied in HA individuals, but with important differences between emotions. Indeed, while the embodiment of the emotion of fear is defective (*Experiment 1*), the embodiment of the emotion of disgust is heightened. Previous research has demonstrated that HA subjects may perceive signals from the body in an aberrant manner, and may be more aroused by interoception of unpleasant stimuli than LA individuals, (see Kano and Fukudo, 2013, for a review). Consistent with these studies, and with the somatosensory amplification hypothesis of alexithymia, the present results of *Experiment 5* indicate that alexithymic individuals manifest an abnormal

embodiment of disgust, a basic emotion characterized by greater interoceptive and physiological arousal.

Previous studies suggest that the VRT effect is evoked by means of a preactivation of somatosensory cortices when viewing touch on others' face (Cardini et al., 2011, 2012). Likewise, the abnormal eVRT effect while observing disgusted faces may be induced by an enhancement of somatosensory cortices activation. Indeed, the emotion of disgust has been found to preferentially activate somatosensory and insular cortices (Anderson et al. 2003; Wright et al. 2004; Phillips et al., 1997; Wicker et al., 2003). The reciprocal interactions between the insula and the somatosensory cortices are particularly relevant because they suggest that bodily perception may be enhanced by the experience of disgust. Accordingly, previous neuroimaging findings in alexithymia research have shown increased insular and somatosensory reactivity in response to negative stimuli in HA participants (Karlsson et al. 2008; Frewen et al. 2008; Heinzl et al. 2010). Thus, the strict relation between the insula and the somatosensory cortices, and their greater activation in alexithymia can account for how the disgusted faces may increase VRT effect in HA, but not LA, participants. Moreover, the insula is known to play a pivotal role in interoception (Craig, 2002, 2003, 2004, 2009, 2011; Critchley et al. 2004; Zaki et al. 2012), which represents an important finding of the current study (i.e. the higher interoceptive abilities of the HA group compared to the LA group). Interestingly, a recent work showed that alexithymia and increased interoceptive abilities are closely associated with increased glutamate-mediated excitatory transmission in the insula (Ernst et al. 2013), thereby suggesting that this brain region may be crucially implicated in the interconnection between increased awareness of bodily responses and alexithymic features. Thus, previous neuroimaging findings,

together with the disgust-related enhancement of the VRT effect and the increased interoceptive abilities reported here in alexithymic individuals, converge to support the hypothesis that people with HA are more likely to be focused on their bodily states and direct physical sensations than people with LA, possibly due to aberrant insular and somatosensory activation in responses to emotion-evoking events (Karlsson et al. 2008; Frewen et al. 2008; Heinzel et al. 2010).

It is worth noting that the current results of higher interoceptive abilities in alexithymia appear to be *prima facie* inconsistent with previous studies indicating that interoceptive accuracy and subjective emotional experience are strictly interdependent (Pollatos et al., 2005, 2011, Critchley et al., 2004), and that the same neural regions, particularly the anterior insula, are involved in appraisal of emotions and bodily physiology (Craig, 2003, 2009, 2011; Terasawa et al. 2013). However, although strongly linked, the precise contribution of interoceptive accuracy and physiological responses to conscious awareness of emotion still remains controversial (Gendron and Barrett, 2009; Critchley and Harrison, 2013). In fact, interoceptive accuracy can be necessary but not sufficient for the conscious appraisal of emotions. For instance, according to the theoretical construct of emotion proposed by Lane and Schwartz (1987), emotional awareness can be graded in different ‘levels’, and awareness of physiological responses are graded in the lower level. On this view, people with alexithymia rely on a lower, physiological level of emotional awareness, thereby stagnating at the level of bodily sensations. In other words, although being able to detect their own visceral changes, alexithymic participants may fail to link these visceral signals to higher levels of emotional processing. In this way, the emotion-evoking event is perceived only at the “physical” level, devoid of any emotional implication. Thus, the

current results did not contradict, but rather expand, previous literature, supporting the hypothesis of a dissociation between interoceptive accuracy and emotional awareness in alexithymia.

As a final consideration, the current results suggest that HA individuals might be particularly vulnerable to disgusting stimuli, which, above all, would enhance their tendency to develop psychiatric and psychosomatic symptoms (Lumley et al. 1996). Supporting this hypothesis, alexithymia is a risk factor for the so called “pathologies of the disgust system”, i.e. pathologies in which the responses to disgusting stimuli are disproportionate to actual risk (Davey, 2011), causing maladaptive behavior, which interfere with the ability to lead a normal life. Among these pathologies it is worth noting the presence of the obsessive compulsive disorders (contamination obsessions, Roh et al. 2011), eating disorder (disgust for the bodily self; Harvey et al 2002, Kessler et al. 2006), some symptoms of schizophrenia (Schienle et al. 2003, Cedro et al. 2001) and hypochondriasis (Davey and Bond, 2006, Wise et al. 1990).

To summarize, the present findings highlight a dissociation between the remapping of fear and disgust in people with HA and suggest that the heightened abilities of HA, compared with LA, participants in embodying the emotion of disgust could be associated with their predisposition to abnormally detect internal bodily sensations. Thus, the emotional profile of HA individuals on the eVRT task has been hypothesized to be related to their abnormal predisposition to be focalized on their internal bodily signals reporting the body’s physiological state, and to their tendency to experience emotions in a “physical” way. Finally, these results on HA might be due to an enhancement of insular activity during the perception of disgusted faces.

CHAPTER 6

Testing the functional contribution of the Amygdala

“<<If I were to order a general to fly from one flower to another like a butterfly, or to write a tragedy, or to change himself into a sea-bird, and if the general did not carry out the order, which one of us would be at fault?>>
<<It would be you>> said the little prince firmly.
<<Exactly. One must demand of each and every one what he or she is capable of>>.”

The Little Prince
Antoine de Saint-Exupéry

The king of the asteroid 325 was wise when he told the Little Prince that one must demand of each and every one only what he or she is capable of. Alexithymic individuals have been proven to have difficulties in embodying emotions. Hence, they cannot be asked to behave normally in social environments, where the decoding of the emotions of the other is essential for the proper functioning. Indeed, *Experiments 1* and *5* revealed a deficit in the embodiment of the somatic aspects of fear in participants with high level of alexithymia (HA). *Experiment 3* showed a deficit in the embodiment of the motor aspects of fear in HA. These deficits have been found, in *Experiment 4*, to be accompanied by an absence of the fear induced bradychardia normally observed during the observation of a fearful expression.

The question to be asked is: why? Why do alexithymic individuals manifest a deficit in the embodiment of fear? Taken together, the results of *Experiments 1, 3, 4* and *5* suggest that the above reported results might be explained by an amygdala hypo-

activation in response to fearful faces in alexithymia. To test the hypothesis of the possible role of the amygdala in the embodiment abilities of the somatic aspect of fear, the performance at the VRT paradigm of participants with amygdalar lesions were compared with those of two different groups of controls.

6.1 Introduction

Facial expressions represent a powerful nonverbal display of emotion, signaling valuable information about other people's intentions and inner states, in order to provide appropriate responses in social interactions. Understanding others' emotions is thought to involve a mechanism of internal representation, where the emotional state of the other is simulated in one's own sensory system (Goldman and Sripada, 2005). This embodied emotion simulation relies on the activation of a distributed sensorimotor network, involving premotor, somatosensory, insular and anterior cingulate cortices and the amygdalae (Carr et al., 2003; Wicker et al., 2003; Singer et al., 2004; Jabbi et al., 2007).

In particular, the somatosensory cortices appear to actively participate in the recognition of visually presented emotional facial expressions, as revealed by fMRI studies showing increased BOLD responses in the somatosensory cortex during emotional expression judgments (Winston et al., 2003). Moreover, data from patients with focal brain lesions revealed that lesions of the right somatosensory cortices are associated with impairments in the recognition of emotional faces (Adolphs et al., 2000). Crucially, it has been demonstrated that disrupting the somatic simulation of emotions by targeting the right somatosensory cortex with repetitive transcranial magnetic stimulation (rTMS) compromises the ability to recognize several visually presented emotional expressions (Pitcher et al., 2008). However, the level of internal

somatic representation in somatosensory cortices for the recognition of emotional faces might vary in response to different emotional expressions (Hussey and Safford, 2009). Indeed, Pourtois et al. (2004) showed that single pulse TMS to the right somatosensory cortex selectively interferes only with recognition of fearful expressions, suggesting a preferential involvement of the somatosensory cortex in processing fear. Such preferential activation in the somatosensory cortex for the recognition of fearful faces might represent an evolutionary adaptive feature, critical for survival. Indeed, the presence of a fearful face might signal a potential threat in the environment requiring a rapid and appropriate defensive response. The present study was designed to investigate whether the preferential activation of somatosensory cortices in response to fearful faces might depend on the activity of the amygdala. Since pioneering physiological studies on animals (Kapp et al., 1979; 1992), the amygdala has been suggested to be a necessary component of the neural circuits involved in the acquisition and the expression of learned fear responses (for recent reviews: LeDoux, 2014; Adolphs, 2013). Indeed, because of its broad connectivity with cortical sensory and prefrontal cortices (Amaral et al. 1992; Young et al., 1994), the amygdala represents a subcortical structure ideally situated to influence cognitive functions in reaction to emotional stimuli (Phelps, 2006). More specifically, the amygdala has been widely reported to be a core-face selective region (Mende-Siedlecki et al., 2013; Todorov, 2012), responding to a variety of emotional facial expressions (Anderson et al., 2003), but especially relevant in processing fearful faces, as suggested by neuro-imaging meta-analysis (Phan et al., 2002; Vytal & Hamann, 2010; Fusar-Poli et al., 2009), animal models (Davis et al., 1994), single-unit recordings (Maren, 2001) and lesional studies (Adolphs et al., 1994).

To test the contribution of the amygdala to the preferential activation of the somatosensory cortices in response to threat, a group of patients with unilateral lesions to the amygdala following temporal lobectomy, a control group of patients with lesions in the extra-temporal regions and a group of healthy participants were tested in a multisensory paradigm called Visual Remapping of Touch (VRT; [Serino et al., 2008](#)). In VRT, viewing a face being touched, compared to merely being approached by fingers, increased the detection of near-threshold tactile stimuli on one's own face. This visually evoked tactile enhancement relies on an increased activity in a fronto-parietal network of pre-motor cortices and somatosensory cortices (SI/SII; [Cardini et al., 2011](#)), responsible for processing tactile information ([Macaluso, 2006](#)), suggesting a mechanism of remapping of the tactile sensation seen on the body of others onto one's own somatosensory system ([Blakemore et al., 2005](#); [Ebisch et al., 2008](#)). Notably, the VRT effect is modulated by the emotional content of the seen face ([Cardini et al., 2012](#)), with participants showing an enhanced performance in tactile discrimination when viewing touch towards a fearful face, compared to neutral or happy faces. This specific enhancement for fearful faces in VRT has been attributed to a preliminary activation of the somatosensory system in response to threat, probably due to a partial overlap between the networks sub-serving visuo-tactile interactions ([Cardini et al., 2011](#)) and embodied emotion recognition ([Keysers et al., 2004](#)). The preparatory activation of the somatosensory cortex might facilitate the processing of tactile information delivered on the participant's face while viewing touch towards a fearful face. Indeed, due to the highly adaptive value of a rapid recognition of fear, participants could be more prone to remap the fear-specific information onto their own somatosensory system. Since the activity of the amygdala is responsible for updating the relevance of the features in the

environment and signaling sources of potential harm (Jacobs et al., 2012), the involvement of this subcortical structure in the emotional modulation of VRT seems plausible. Viewing a fearful face being touched and concurrently perceiving touch on one's own face represents an ambiguous event, in which the amygdala could signal the existence of a potential common threat, requiring a prioritized sensory analysis. A disruption of the preferential activation of the somatosensory cortex and, therefore, no enhancement in VRT when viewing touch towards fearful faces is expected in patients with lesions to the amygdala, while patients with lesions not involving the amygdala and healthy controls are expected to show the typical fear-related VRT enhancement. However, patients with lesions to the amygdala, similarly to healthy controls and patients with lesions not involving the amygdala, are expected to show the typical visual remapping patterns with neutral and happy faces.

6.2. EXPERIMENT 7: Disruption of the fear enhancement of tactile perception after Amygdala lesions

6.2.1 *Material and Methods*

Participants: All the participants were right-handed, had normal or corrected-to-normal vision, reported normal touch, and were naïve to the purposes of the experiment. They all gave informed consent to participate and the study was conducted according to the Declaration of Helsinki (BMJ 1991; 302: 1194) and the Local Ethical Committee.

Patients with lesions to the amygdala (AMG): Seven patients (P1-P7; see table 6.1) were selected after complete amygdalohippocampectomy. They had suffered mesial temporal lobe epilepsy in either the left (n:5) or right hemisphere (n:2) from low

grade glial tumors (dysembryoplastic neuroepithelial tumor, ganglioglioma, and oligodendroglioma). At the time of the study the age of patients ranged between 18 and 23 years (20.8, st.dev: 1.7). Epilepsy onset ranged between 1 and 12 years of age (mean: 6.8, st.dev: 3.9). Mean age at surgery was 14 years (st.dev: 4.3, range: 10-21 yr) and mean duration of epilepsy was 7.8 years (st.dev: 6.6, range: 1-18). All patients were seizure free for more than 2 years (mean time of seizure-free: 6.5 years, st.dev: 4.5, range: 2-12) and were treated with one or two AED in a stable regimen.

Patients with extra-temporal lesions (EXTRA-TEMPORAL): Seven patients (P8-P14; see table 6.1) were selected after extra-temporal resective surgery. Lesions involved the right frontal lobe (n:3), and the third ventricle (n:4). Lesion etiology was low grade tumors (meningioma, oligodendroglioma, papilloma, astrocytoma). At the time of the study the age of patients ranged between 19 and 29 years (mean: 24.6, st.dev: 4). Epilepsy onset ranged between 3 and 16 years of age (mean: 12, st.dev:4). Mean age at surgery was 13 years (st.dev: 5, range: 3-21) and mean duration of epilepsy was 9 months (st.dev: 7, range: 2-24 months). All patients were seizure free for more than 2 years (mean time of seizure-free: 7.8 years, st.dev: 4.8, range: 2-14) and were treated with one or two AED in a stable regimen.

Healthy control participants: Seven age-matched participants took part in the study (mean age: 20.7; age range: 19-25). All the participants had no history of epilepsy and neurological or psychiatric diseases.

Neuropsychological assessment: A formal neuropsychological assessment, including cognitive level (i.e. IQ), attention and executive functions, memory, visuo-spatial and visuo-motor functions was carried out after surgery at the time of the present

study. Assessment was performed with standardized instruments validated on an Italian sample (WISC III, Wechsler, 1991; Spinnler and Tognoni, 1987; Tressoldi et al., 2005). All patients showed normal IQ (AMG: mean: 88, st.dev: 13; EXTRA-TEMPORAL: mean: 91, st.dev: 6) and normal neuropsychological functions, excepting for patients P2 and P4 who showed defective verbal memory. The presence of psychiatric symptoms was evaluated with Symptom Check List -SCL-90 (Derogatis 1994) and Child Behavioral Checklist -CBCL (Achenbach et al. 2008). Mood and anxiety were assessed with Beck depression Inventory-II (BDI-II, Beck et al. 1996) and State-Trait Anxiety Inventory (STAI I and II, Spielberger et al. 1980). All patients showed normal emotional well-being, with the exception of patients P3 and P4 who presented mild symptoms of anxiety and depression.

Lesion analysis: Mapping of brain lesions was based on the most recent clinical CT or MRI. Lesions were traced on the T1-weighted template MRI scan from the Montreal Neurological Institute provided with the MRICron software (Rorden and Brett, 2000; Rorden et al., 2007). Left lesions were traced on the right hemisphere. Lesion volumes were computed for patients whose images were available (6 in the AMG and 6 in the EXTRA-TEMPORAL group) and the extents of the lesions were compared between the group of patients, revealing no significant differences between patients with lesions to the amygdala (2498 ± 55682 sem mm^3) and with extra-temporal lesions (14280 ± 1660 sem mm^3 ; $t(10) = -1.67$; $p = .12$].

Case	Sex	Age	Age at surgery	Epilepsy onset (years)	Epilepsy duration (months)	Seizure free (years)	Lesion site
P1	M	21	18	12	72	3	L MT
P2	F	22	10	8	12	12	L MT
P3	F	23	21	3	216	2	R MT
P4	F	19	16	1	180	3	L MT
P5	F	22	10	5	60	12	L MT
P6	F	18	11	10	12	7	R MT
P7	M	21	17	9	108	4	L MT
P8	F	29	15	15	8	14	L FL
P9	M	24	15	15	12	9	R TV
P10	M	19	12	12	10	6	R FL
P11	F	28	3	3	24	13	TV
P12	M	23	14	14	8	9	L PL
P13	M	18	16	16	4	2	R PG
P14	M	23	21	16	60	2	R FL

Table 6.1. Summary of clinical, demographic and lesional data. M: male; F: female; L: left; R: right. MT= Medial Temporal lobe. FL= Frontal lobe. TV= third ventricle. P= Parietal lobe. PG=pituitary gland.

As showed in *Figure 6.1*, although all patients in the AMG group showed damage that included the amygdala, areas adjacent to the amygdala (e.g. temporal pole, fusiform gyrus, hippocampus and parahippocampus) were also damaged to some degree. In contrast, patients with extra-temporal lesions revealed lesions in the frontal lobe (n=3), in the occipito-parietal regions (n=1), in the regions surrounding the third ventricle (n=2) and in the pituitary gland (n=1).

Notably, patients in both groups revealed no lesions to the somatosensory cortices.

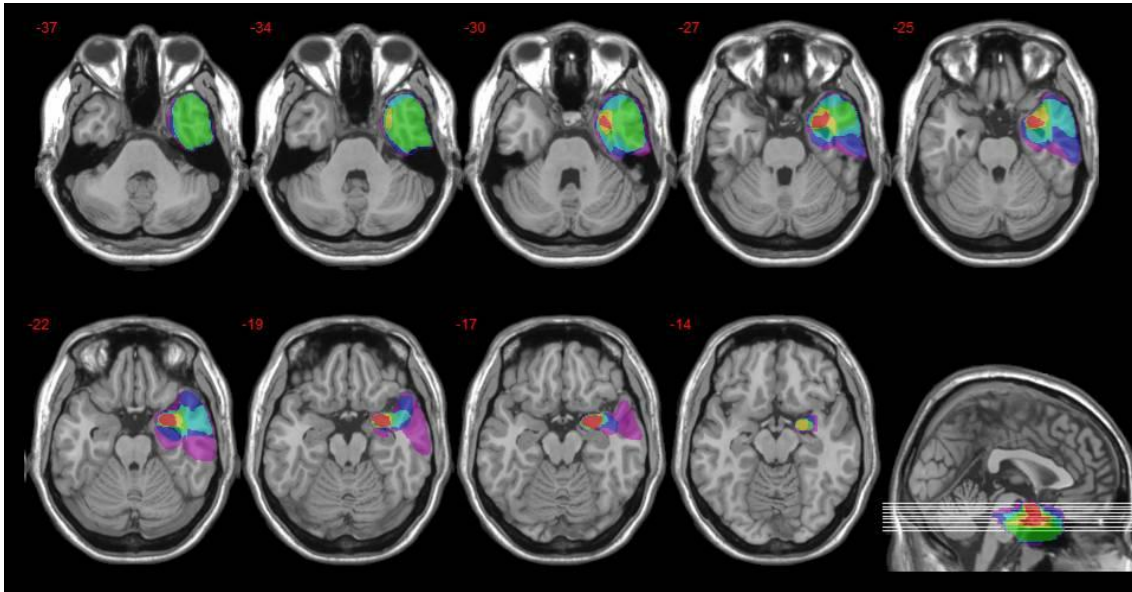


Figure 6.1. Location and overlap of brain lesions. The image shows the lesions of the AMG patients projected on axial slices of the standard MNI brain. The level of axial slices has been marked by white lines on the sagittal view of the brain.

eVRT task: Tactile stimuli were delivered by two constant current electrical stimulators (DS7A, Digitimer), via two pairs of neurological electrodes (Neuroline, AMBU) placed on the participant's right and left cheeks. For half of the participants in each group, the tactile stimulus on the left cheek was set to be more intense than that on the right cheek, and vice-versa for the other half. Prior to the experiment, each participant looked at static, neutral faces while the intensity of the electrical stimuli was calibrated with a staircase procedure to a threshold detection rate of 100% for the stronger stimulus and 60% for the weaker. As a confirmation of correct calibration of the stimuli, the mean accuracy for unilateral strong and unilateral weak stimuli was $89\% \pm 4\%$ (mean \pm s.e.m.) and $73\% \pm 5\%$, respectively (paired sample t -test: $p < 0.003$) [$88\% \pm 5\%$ and $79\% \pm 7\%$ in AMG, $84\% \pm 10\%$ and $78\% \pm 10\%$ in EXTRA-TEMPORAL and $96\% \pm 1\%$ and $61\% \pm 7\%$ in CONTROL; $F(2,18)=1.13$; $p=.34$ for

stronger stimuli and $F(2,18)=1.83$; $p=.19$ for weaker stimuli]. This stimulus calibration results in a tendency for participants to fail to report the weaker stimulus during trials with bilateral stimulation. Mean accuracy for bilateral tactile detection was $54\% \pm 3\%$ ($52\% \pm 6\%$ in AMG group, $51\% \pm 7\%$ in EXTRA-TEMPORAL group and $57\% \pm 7\%$ in CONTROLS; $F(2,18)=0.23$; $p=.8$). Errors consisted mostly of reporting the side of the stronger stimulus: the mean probability of reporting the side of the stronger stimulus in the case of errors during bilateral stimulation was $91\% \pm 3\%$ ($95\% \pm 2\%$ in AMG group and $82\% \pm 9\%$ in EXTRA-TEMPORAL and $95\% \pm 2\%$ in CONTROLS; $F(2,18)=2.12$; $p=.15$).

The visual stimuli and experimental procedure was identical to the one adopted in Chapter 3, *Experiment 1*. Visual stimuli consisted of video trials presented on a 17" computer screen placed in front of the participant at a distance of about 60 cm. Faces with neutral, fearful or happy expressions were presented in three different counterbalanced blocks. The neutral faces used in the experiment were different from the faces used in the calibration procedure. The faces were static black-and-white pictures selected from the Pictures of Facial Affect (PFA) database (Eckman and Friesen, 1976). Overall, 6 different actors (3 males and 3 females) were used. Male participants were presented with images of male actors, while female participants were presented with images of female actors. The faces were presented as a central, static image in the background of the movie. In the foreground, two fingers were initially positioned on the lower part of the screen, one on the right and one on the left side. During the movie the fingers moved towards the face and then returned back to their initial position. In different trials, the finger-motion followed one of two trajectories: in the Touch condition, the fingers touched the cheeks of the shown face, in the same

position where tactile stimulation on the participants' cheeks was administered. In the No-Touch condition, the fingers stopped about 5 cm alongside the face (see Figure 2). In different trials, either the finger on the right, on the left or both fingers moved. Visual and tactile stimuli were synchronized so that when the fingers reached the peak of their trajectory a tactile input was delivered to the participant's face. Each movie lasted a total of 2700 ms, and tactile stimulation was delivered at 700 ms from movie onset.

A PC running C.I.R.O. software (www.cnc.unibo.psice.unibo/ciro) was used to control the presentation of the stimuli and record the responses. Each experiment consisted of 6 counterbalanced experimental blocks of the tactile confrontation tasks, two blocks for each facial emotion. Stimuli comprised a combination of the two types of tactile stimulation (unilateral left or right and bilateral), the two types of visual stimulation (unilateral left or right and bilateral) and the two finger movement trajectories (Touch and No-Touch). Each block consisted of 48 trials, presented in a random order. A pause of 3 minutes, during which tactile thresholds were recalibrated, was interposed between blocks.

The Eckman 60 Faces Test: After eVRT, participants underwent The Eckman 60 Faces Test, investigating the recognition of six basic emotions (anger, disgust, fear, happiness, sadness and surprise) from the Eckman and Friesen series of Pictures of Facial Affect ([Eckman and Friesen, 1976](#)). In the test, photographs of the faces of 10 models (six female and four male) were presented. Each model was presented in six different poses, corresponding to each of the six basic emotions. Different models and emotions were interleaved. Each face was presented on an A4 sheet with six labels of basic emotions below the photograph. The participants were required to verbally

indicate the appropriate label describing the facial expression presented. The maximum score was 10 for each basic emotion (for a total score of 60).

6.2 Results

eVRT task: In order to investigate the effect of the emotional content of the viewed face on the VRT effect, subjects' accuracy in responding to bilateral tactile stimuli was compared when both fingers did or did not touch the faces. An ANOVA on bilateral stimulation detection accuracy was conducted using Group (AMG, EXTRA-TEMPORAL, CONTROL) as a between-subjects variable, and Emotion (Fear, Happy and Neutral) and Finger Trajectory (No Touch and Touch) as within-subjects variables. Post-hoc comparisons (Newman-Keuls test) were performed, when necessary, to compare single effects. The analysis revealed a significant main effect of Finger Trajectory ($F[1,18]=74.67$; $p<0.001$), with a significantly higher bilateral detection accuracy in the Touch condition ($65.87\% \pm 9.5\%$), compared to the No Touch ($52.77\% \pm 8.3\%$). Moreover, the critical triple interaction Group x Emotion x Finger Trajectory was significant ($F[4,36]=5.56$; $p=0.001$). Consequently, an ANOVA on bilateral stimulation detection accuracy using Emotion and Finger Trajectory as within-subjects variables was performed on each group, separately.

In the AMG group (Figure 6.2 a), the analysis revealed a significant main effect of Finger Trajectory ($F[1,6]=7.89$; $p=0.03$), with a significantly higher bilateral detection accuracy in the Touch condition ($68.52\% \pm 7.3\%$), compared to the No Touch ($60.52\% \pm 8.1\%$). Moreover, the critical double interaction Emotion x Finger Trajectory was also significant ($F[2,12]=4.89$; $p=0.02$). Post-hoc tests showed that the Touch condition enhanced the accuracy of bilateral stimulation perception in comparison to the

No Touch condition for neutral faces (No Touch: 62.14%±5.9% vs Touch: 80%±8.5%, $p=0.001$), but not for happy (No Touch: 62%±7.7% vs Touch: 64.49%±7.4%, $p=0.57$) and fearful (No Touch: 57.28%±7.21% vs Touch: 61%±7%, $p=0.36$) faces. Moreover, whereas in the No Touch condition there was no difference in the accuracy of bilateral stimuli detection between the three emotions (fear vs happy: 57.28%±7.2% vs 62%±7.7%, $p=0.61$; fear vs neutral: 57.28%±7.2% vs 62.14%±5.9%, $p=0.45$; happy vs neutral: 62%±7.7% vs 62.14%±5.9%, $p=0.96$), in the Touch condition the accuracy of perception of bilateral stimuli was higher for neutral faces than for happy or fearful faces (all $ps < 0.003$).

In the EXTRA-TEMPORAL group (Figure 6.2 b), the analysis revealed a main effect of Finger Trajectory ($F[1,6]=34.97$; $p=0.001$), with a significantly higher bilateral detection accuracy in the Touch condition (65%±6.4%), compared to the No Touch (50%±8.2%). Critically, the double interaction Emotion x Finger Trajectory was significant ($F[2,12]=7.25$; $p=0.008$). Post-hoc tests showed that the Touch condition enhanced the accuracy of bilateral stimulation perception in comparison to the No-Touch condition for neutral faces (No Touch: 51%±5.1% vs Touch: 62.8%±7.9%, $p=0.01$) and for fear (No touch: 48.57%±6.3% vs Touch: 74.57%±8.9%, $p<0.001$), but not for happy faces (No touch: 50.4%±6.5% vs Touch: 57.6%±6.4%, $p=0.16$). Moreover, whereas in the No Touch condition there was no difference in the accuracy of bilateral stimuli detection between the three emotions (fear vs happy: 48.57%±6.3% vs 50.4%±6.5%, $p=0.61$; fear vs neutral: 48.57%±6.3% vs 51%±5.1%, $p=0.78$; happy vs neutral: 50.4%±6.5% vs 51%±5.1%, $p=0.87$), in the Touch condition the accuracy of perception of bilateral stimuli was higher for fearful faces than for neutral or happy faces (all $ps < 0.007$).

In the CONTROL group (Figure 6.2 c), the analysis revealed a main effect of Finger Trajectory ($F[1,6]=43.27$; $p<0.001$), with a significantly higher bilateral detection accuracy in the Touch condition ($64.09\% \pm 7.1\%$), compared to the No Touch ($47.81\% \pm 7.9\%$). Critically, the double interaction Emotion x Finger Trajectory was significant ($F[2,12]=13.59$; $p<0.001$). Post-hoc tests showed that the Touch condition enhanced the accuracy of bilateral stimulation perception in comparison to the No-Touch condition for neutral faces (No Touch: $48.1\% \pm 6.2\%$ vs Touch: $63\% \pm 8.8\%$, $p=0.003$) and for fear (No touch: $46\% \pm 8.5\%$ vs Touch: $74.85\% \pm 8.5\%$, $p<0.001$), but not for happy faces (No Touch: $49.2\% \pm 9.5\%$ vs Touch: $54.4\% \pm 8.8\%$, $p=0.13$). Moreover, where in the No Touch condition there was no difference in the accuracy of bilateral stimuli detection between the three emotions (fear vs happy: $46\% \pm 8.5\%$ vs $49.2\% \pm 9.5\%$, $p=0.58$; fear vs neutral: $46\% \pm 8.5\%$ vs $48.1\% \pm 6.2\%$, $p=0.52$; happy vs neutral: $49.2\% \pm 9.5\%$ vs $48.1\% \pm 6.2\%$, $p=0.73$), in the Touch condition the accuracy of perception of bilateral stimuli was higher for fearful faces than for neutral or happy faces ($p<0.003$).

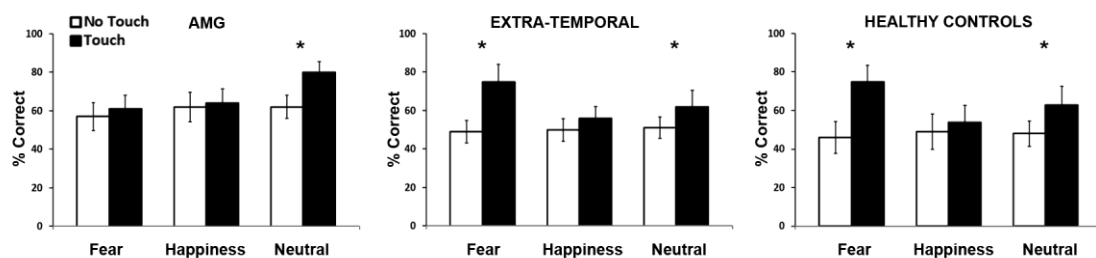


Figure 6.2. Results for the eVRT in AMG patients (A) and EXTRA-TEMPORAL patients (B) and HEALTHY CONTROLS (C). Accuracy in detecting bilateral tactile stimulation while viewing either a fearful face, a happy face or a neutral face that could be touched or just approached by two human fingers. Error bars show standard errors of the mean.

To further explore the differences between the groups in the VRT performances with emotional faces, an emotional VRT index was calculated, subtracting the accuracy of bilateral stimuli detection in the No Touch condition from the Touch condition (VRT index = Touch – No Touch). The participants' emotional VRT index for each emotion was compared. An analysis of variance (ANOVA) on VRT indices was performed using Group (AMG, EXTRA-TEMPORAL, CONTROL) as a between-subjects variable, and Emotion (Fear, Happy and Neutral) as a within-subjects variable. The analysis revealed a significant Group x Emotion interaction ($F[4,36]=5.49$, $p<0.001$). The post-hoc test showed that the VRT index for fear was smaller (i.e. smaller difference between no touch and touch) in AMG ($3.85\pm 2.1\%$) than in EXTRA-TEMPORAL ($26\pm 2.5\%$, $p=0.03$) and CONTROL ($28.85\pm 3.3\%$, $p=0.01$), whereas no difference was detected between EXTRA-TEMPORAL and CONTROL ($p=0.66$). On the contrary, the VRT index for happy and neutral did not differ between the three groups (all $p>0.6$).

Finally, a correlation analysis between lesions, volume of patients (AMG and EXTRA-TEMPORAL group) and the VRT index for fearful faces was performed, to test the possibility that reduced VRT effects for fearful faces could be due to bigger lesions in the group of patients with lesions to the amygdala. The result of the correlation was not significant ($r=-0.08$, $p=0.80$), supporting the assumption that the VRT for fearful faces is not driven by lesion size.

The Eckman 60 Faces Test: The total scores of the participants were compared with Kruskal-Wallis ANOVA, using group (AMG, EXTRA-TEMPORAL, CONTROL) as a factor. No significant difference between groups was found [$H(2,21)=2.05$; $p = 0.36$; AMG: 51 ± 2.64 ; EXTRA-TEMPORAL: 51 ± 1.41 ; CONTROL: 54 ± 1.5]. In

addition, to investigate the presence of deficits in the recognition of specific categories of emotional faces, the scores of the participants for each single emotion were compared with Kruskal-Wallis ANOVA, using group (AMG, EXTRA-TEMPORAL, CONTROL) as a factor. The results revealed no significant differences between the groups in each of the emotions (all p s $>$. 2).

Voxel-based Lesion-Symptom Mapping: The relation between brain tissue damage and the behavioural performance was investigated on a voxel by voxel basis, using the Voxel-based Lesion-Symptom Mapping technique (VLSM, Bates et al., 2003) on traced brain lesions of 12 out of 14 patients (6 in the AMG and 6 in the EXTRA-TEMPORAL group). This analysis allows identifying regions associated with a specific behaviour (i.e. VRT for fearful faces) and playing a causal role in task performance (Gläscher et al., 2010).

Patients' VRT index (Touch – No Touch) for fearful faces was entered in the Non-Parametric Mapping software (NPM, Rorden et al., 2007; free available at <http://www.cabiatl.com/mricro/npm/>), which compares performance of patients with damage with performance of patients without damage to the amygdale at each voxel in the brain using the nonparametric Brunner-Munzel (BM) rank-order test (Brunner and Munzel, 2000). Only voxels affected in at least 20% of cases were included in the analysis. The alpha level of significance was set at $p < 0.05$ and was corrected for multiple comparisons by using the False Discovery Rate (FDR) 5% threshold. Additionally, an extent threshold of 50 voxels was used (see also Gläscher et al., 2010).

The VLSM analysis revealed that the cluster associated with reduced VRT index for fearful faces was located in the amygdala. The analysis reported that the centre of

the mass was located at the coordinate 36, 2, -25, referring to the right amygdala (cluster size 349 voxels, maximum BM z score 2.72). These results confirm that the amygdala has a causal role in decreasing VRT for fearful faces.

6.3. Discussion

The present findings on patients with unilateral lesions of the amygdala suggest that the preferential activation of the somatosensory cortices in response to fear relies on the activity of this subcortical structure. In line with previous studies, the results on healthy participants and control patients with lesions not involving the amygdala confirm that viewing a neutral face being touched increases tactile perception on one's own face, compared to viewing the same face just being approached, showing a typical Visual Remapping of Touch effect (VRT; [Serino et al., 2008](#)). In addition, an enhanced VRT for fearful faces, compared to neutral and happy faces, was found, consistent with previous studies ([Cardini et al., 2012](#)). More interestingly, the results revealed that after unilateral lesions to the amygdala, following temporal lobectomy, although patients demonstrated the standard VRT effect for neutral faces, the typical enhancement in the VRT for fearful faces disappeared, suggesting a critical role of the amygdala in modulating somatosensory activity in response to threat.

The VRT effect is a visuo-tactile interaction where the touch viewed on a face is remapped onto one's own somatosensory system and relies on the activation of a fronto-parietal network, involving pre-motor and somatosensory cortices ([Cardini et al., 2011](#)). VRT has been shown to be modulated by the presentation of emotional faces: more precisely, VRT is enhanced when viewing a fearful face, compared to neutral, happy or angry faces ([Cardini et al., 2012](#)). The emotional modulation of VRT is thought to

depend on a simulating mechanism by which viewing others' emotions generates an internal representation of that emotion within one's own somatosensory system (Adolphs et al., 2000). In particular, the specific enhancement of VRT for fearful faces could rely on a preliminary activation of the somatosensory cortex in response to threat (Cardini et al., 2012), consistently with evidence showing critical role of the somatosensory cortex in fear processing (Adolphs et al., 1996; Pourtois et al., 2004). Thus, recognizing fearful faces might require a stronger internal somatic representation, due to the relevance of faces expressing fear as a signal of potential danger. The new findings of the present study suggest that the greater somatic preparation related to fearful faces might depend on the activity of the amygdala. Indeed, patients with selective lesions to the amygdala report no significant differences in tactile perception when viewing a fearful face being touched compared to simply being approached by fingers, demonstrating a lack of any preferential activation of the somatosensory cortex for fear. However, these patients show a typical increase in tactile perception when viewing touch on a neutral face, compared to viewing the same neutral face just being approached by fingers, therefore suggesting a significant effect of visual remapping of touch with neutral faces. In other words, after amygdala lesions, the ability of remapping the tactile sensations observed on a seen face onto one's own sensory system (i.e. visual remapping of touch) is generally preserved, but the typical enhancement of this effect in presence of fearful faces is disrupted, in line with the hypothesis that amygdala lesions might interfere with the preferential activation of the somatosensory cortices in response to threat.

Fearful faces represent a typical stimulus activating the amygdala, providing information about the presence of a threat, without necessarily indicating the source of

that threat (Whalen, 1998). Moreover, viewing a fearful face being touched and concurrently perceiving touch on one's own face represents a potential harm arising from the immediate environment. In healthy participants and patients with lesions not involving the amygdala, this subcortical structure, being an integral component of a continuous vigilance system, responding to unpredictable, novel, ambiguous and biologically relevant stimuli, signals the potential tactile threat to the somatosensory cortices, in order to understand the source of the threat. This mechanism is disrupted when the amygdala is lesioned and, therefore, no enhancement in VRT effect can be observed. The modulatory effect of the amygdala on the somatosensory cortices could rely on amigdalo-cortical projections terminating in somatosensory fields of SII, as reported by electrophysiological studies on primates (Amaral and Price, 1984). Indeed, due to its broad connectivity with cortical and subcortical structures (Amaral et al., 1992; Young et al., 1994), the amygdala can influence distant brain areas, thus changing the functional pattern of activation to fearful faces in regions distant to the amygdala itself. In line with this hypothesis, recent findings showed that amygdala lesions disrupt the typical BOLD enhancement for fearful faces in the extra-striate cortex and early occipital areas, suggesting a distant functional influence of the amygdala on connected and structurally intact sensory cortices and providing a plausible neural substrate for the prioritization of fearful stimuli in perception (Vuilleumier et al., 2004).

Notably, in the present study, the lesions to the amygdala disrupt the enhancement for fearful faces in the somatosensory cortex, without affecting explicit recognition of emotional faces, thus ruling out the hypothesis that the lack of VRT for fearful faces could depend on impaired recognition of the emotional content of the faces. Indeed, patients with lesions to the amygdala performed similarly to healthy controls and

patients with lesions not involving the amygdala at the Eckman 60 Faces Test, where explicit recognition of 60 emotional faces is required, showing no impairment in the recognition of specific emotions, including fear. Although after damage to the amygdala (mainly bilateral and congenital) the subjective experience of negative emotions (Tranel et al., 2006), the intensity and similarity judgments of emotional faces (Adolphs et al., 1994; 1996; Anderson et al., 2000; Siebert et al., 2003; McClelland et al., 2006) and social signals (Adolphs et al., 2002) might be altered, recent findings report no effects of amygdala lesions (especially unilateral) on recognition of emotional faces (Adolphs et al., 1995; Hamann et al., 1996; Siebert et al., 2003; Vuilleumier et al., 2004; Graham et al., 2007; Tsuchiya et al., 2009), in line with the present findings.

The lack of VRT effect (i.e. no difference between touch and no touch) in the presence of happy faces both in patients (with and without lesions to the amygdala) and in healthy controls is consistent with previous results (Cardini et al., 2012) and could represent a by-product of the motor resonance phenomenon. Indeed, observing emotional faces could elicit spontaneous mimicry (Dimberg et al., 2000); more specifically, when observing happy faces, a spontaneous contraction of the zygomaticus major muscle occurs. *Experiment 3 (Chapter 4)* revealed that healthy participants, presented with images of happy faces selected from the same database used in the present experiment (Eckman and Friesen, 1976), showed a peak of activation in the zygomaticus major muscle 625 ms after stimulus presentation. As a consequence, the rapid muscle activity elicited by happy faces could have interfered with the discrimination of the electro-tactile stimuli placed on the cheeks, delivered at 700 ms, during the present experimental task, supporting the hypothesis that the lack of VRT effect for happy faces could depend on motor mimicry.

To sum up, the present findings suggest the existence of a cooperative mechanism between the amygdala and the somatosensory cortices, in which the amygdala can signal potential threat to the somatosensory cortices, resulting in a prioritized tactile analysis. This mechanism could represent the neural counterpart of the typically observed fear-related enhancement in the VRT effect. Indeed, after amygdala lesions no enhancement in VRT when viewing touch towards fearful faces is observed.

CHAPTER 7

Are alexithymics impaired in face perception or in emotion perception conveyed by faces?

“<<Please draw me a sheep>> said the Little Prince. Once again, I made another drawing. But it was rejected too, like the previous ones. <<This one is too old. I want a sheep that will live for a long time>>. My patience ” had run out then (..) so I scribbled a drawing. And I explained: <<That is only the box. The sheep you asked for is inside.>>” But I was very surprised to see the face of my little judge lighting up: <<this is exactly the way I wanted it>>.”

The Little Prince
Antoine de Saint-Exupéry

Accordingly to Antoine de Saint-Exupéry, a sheep is not simply a sheep. For instance, a sheep could be an old sheep, or a sick sheep. In the same way, a face is not simply a face. For instance, a face could be a happy face or a fearful face. The adjectives we attribute to things make things different. In normal conditions, to perceive a sheep, or a face, is easy. However, to perceive automatically and quickly the secondary features of the sheep or the emotions conveyed by a face is not so easy, and only the Little Prince has been able to perceive the sheep through the box.

7.1. Introduction

In the previous experiments (Experiments 1, 3, 4 and 5), we provided convergent results supporting the hypothesis that alexithymic participants exhibit a deficit in emotional embodiment, by using highly homogeneous stimuli, i.e. emotional faces. This

choice has been made with the aim of removing a possible confound in the results due to the heterogeneity of the stimuli. Experiment 8 has been specifically designed to investigate if participants with high and low level of alexithymia also differ in early perceptual processes involving the structural encoding of emotional faces (Eimer and Holmes, 2002; Bentin and Deouell, 2000; Itier and Taylor, 2004).

Indeed, the topic of face perception is of utmost relevance, since human faces represent a biologically salient category of stimuli, whose efficient perception is crucial for social life. The perceptual processing of faces 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 the single features, as suggested for example by the inversion effect, a phenomenon in which faces presented upside-down are more difficult to recognize compared to inverted objects (Adolphs et al. 2002).

At the electrophysiological level, event-related potentials (ERPs) in response to faces showed a prominent negative deflection at occipito-parietal electrodes peaking in a range between 150-180 ms after stimulus presentation. This negative waveform is called N170 and is commonly thought to reflect early perceptual processes involving the structural encoding of faces (Eimer and Holmes, 2002; Bentin and Deouell, 2000; Itier and Taylor, 2004). The N170 component is hypothesized to arise primarily from the fusiform gyrus and superior temporal sulcus (STS) (Haxby et al. 2000; Itier and Taylor, 2004). Moreover, it can be readily distinguished from the ERP response to other classes of stimuli, for instance objects (Herrmann et al. 2005). In addition, the N170 has also been described as a component modulated by emotions (Pizzagalli et al. 2002; Batty and Taylor, 2003; Ashely et al. 2004; Blau et al. 2007) although the results in literature are non-conclusive (see Eimer and Holmes, 2002; Eimer et al. 2003 for an

absent emotional modulation). Specifically, the N170 has been consistently reported to present an increased amplitude when observing fearful faces (Batty and Taylor, 2003; Blau et al. 2007; Carlson and Reinke, 2010; Miyoshi et al. 2003; Pegna et al. 2008, 2011; Lee et al. 2007; Lyn and Salisbury, 2008), while its amplitude is not modulated when observing happy faces (Batty and Taylor, 2003; Caharel et al. 2005; Krombholz et al. 2007; Lyn and Salisbury, 2008). Results regarding other emotions are inconsistent; for instance, disgust has been reported to modulate N170 amplitude in Caharel et al. (2005) but not in Batty and Taylor (2003). Finally, the N170 emotional modulation frequently observed in healthy participants is absent in participants with psychiatric disorders involving emotional processing, i.e. schizophrenia (Ibanez et al. 2012; Lee et al. 2007; Lyn and Salisbury, 2008; Feuerriegel et al. 2014).

Moreover, at a later stage of perceptual representation, typically around 350 ms after stimulus onset), salient emotional faces are known to modulate the amplitude of the early posterior negativity (EPN), which reflects a stimulus-driven attentional capture, in which relevant stimuli are selected for further processing (Sato et al. 2001; Schupp, et al., 2004a; Frühholz et al., 2011; Calvo and Beltran, 2014). Like the n170, the EPN amplitude has been reported to be most pronounced for threat related expressions (e.g. Shupp et al. 2004; Rellecke et al. 2011).

In alexithymia, despite limited ERP data, recent studies have documented an early deficit for visual encoding of emotional stimuli (Pollatos and Gramann, 2011). Indeed, this study demonstrates a hampered perception of emotionally salient stimuli at an early stage in the time course of visual processing. The attenuation of basic emotional process in alexithymia starts as early as 120 ms, as reflected in reduced P1 amplitudes for negative valenced stimuli. Critically, the authors used the International

Affective Picture System (IAPS, [Lang et al. 1999](#)) stimuli as emotional stimuli. The IAPS is a standardized and well-characterized collection of visual images designed to evoke either neutral, positive, or negative emotional states ([Lang et al., 1999](#)). Pictures in the IAPS vary with respect to two primary dimensions: affective valence and arousal. Negative pictures included images such as frightening animals and mutilated human bodies, happy pictures included scenes of parties, people smiling and interacting, while neutral pictures depicted daily necessities such as tableware and books. Consequently, [Pollatos and Gramann's \(2011\)](#) ERP analysis was not specific either for faces (since IAPS stimuli included emotional scenes) or for emotions (since the analyses were valence specific instead of emotion specific).

Thus, although the N170 and EPN modulation by emotions has been widely studied in healthy participants, it is still unclear whether its modulation by emotions conveyed by faces is present also in participants with subclinical emotional difficulties, such as Alexithymia. Therefore, the present study was designed to investigate, using the high-temporal resolution of the ERP technique, whether the stage of structural encoding of faces, as reflected in the N170 component, and the visual selective attention processing, as indexed by the subsequent EPN component, are influenced by the emotion-related information represented in faces, in high and low alexithymic participants. To this end, an electroencephalogram (EEG) was recorded in HA and LA participants performing a visual task in which they were presented with pictures of objects or faces with neutral or emotional (fearful, happy or disgusted) expressions.

In keeping with previous evidences ([Pizzagalli et al. 2002](#); [Batty and Taylor, 2003](#); [Ashely et al. 2004](#); [Blau et al. 2007](#)), a N170 and EPN modulation by emotions was expected in LA participants. In particular, fear face was expected to mainly

enhance both the N170 and EPN amplitude compared with neutral expression and other emotional expression, in line with previous studies (Batty and Taylor, 2003; Miyoshi et al. 2003). In contrast, this specific fear modulation of N170 and EPN was expected to be absent in HA participants, in accordance with the early processing results obtained in alexithymia using IAPS images (Pollatos and Gramann, 2011) and in accordance with the N170 results on psychiatric population characterized by some emotional difficulties (please see Feuerriegel et al. 2014 for a review). Moreover, these hypothesized results would partially explain the deficit in the embodiment of fear found in Experiments 1, 3, 5 and 6.

7.2 EXPERIMENT 8: n170 emotional modulation in alexithymia

7.2.1 Material and Methods

Participants selection: As described in Chapter 2, participants were included in the study if i) they had no history of neurological, major medical or psychiatric disorder and ii) they do not show subclinical or clinical depression at the Italian version of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al. 1997); iii) their scores at two different tests to assess alexithymia were congruent (the self report questionnaire in the Toronto Alexithymia Scale –TAS-20- ,Taylor et al. 2003 and the alexithymia module of the structured interview for the Diagnostic Criteria for Psychosomatic Research –DCPR- Mangelli et al. 2006). Subjects were included in the high alexithymia (HA) group if their TAS-20 score fell above 61, while they were included in the low alexithymia (LA) group if their TAS-20 score fell below 39. The TAS-20 has a three factor structure, namely: Difficulty Identifying Feelings (DIF); Difficulty Describing Feelings (DDF); Externally Oriented Thinking (EOT).

238 students were screened using TAS-20. Based on TAS-20 scores, 25 HA were initially recruited. 3 of them were subsequently excluded, since the DCPR score did not confirm their alexithymia level. 1 subject was excluded due to a high level of depression based on SCID-I. One additional subject was excluded as he was previously diagnosed as suffering from bipolar disorder. The final HA sample included 20 subjects. 19 age and gender matched LA subjects were included in the study. *Table 7.1* reported the alexithymia and sociodemographic features of the sample.

	Low Alexithymia	High Alexithymia
n (male/female)	19 (5/14)	20 (6/14)
Age, mean (SD) (years)	25.3 (1.48)	24.05 (1.08)
<i>TAS-20</i>	<i>Minimum-maximum, mean (SD)</i>	
Total	23-39, 33.3 (4.5)	61-80, 65.6 (5.3)
DDF	7-15, 9.2 (2.2)	14-24, 19.8 (2.9)
DIF	6-15, 10.7 (2.7)	15-31, 24.1 (3.9)
EOT	9-17, 13.4 (2.5)	13-31, 21.7 (4.7)
DPCR	0-2, 1.4 (0.7)	3-5, 3.7 (0.7)

Table 7.1. Demographic and alexithymia profile of low and high alexithymia groups. TAS-20: twenty-item Toronto Alexithymia Scale; DIF: difficulty in identifying feelings; DDF: difficulty in describing feelings; EOT: externally oriented thinking. SD: standard deviation. Low Alexithymia (n=19) and High Alexithymia (n=20) groups were obtained excluding the participants with discrepancy between TAS-20 and DPCR scores (cf. Material and Methods). DPCR: Alexithymia Module of Diagnostic Criteria for Psychosomatic Research scores.

The included participants were also asked to respond to the Kellner's Emotional Inhibition Scale (EIS; [Kellner, 1987](#)), to investigate their emotional management profile, and to the Interpersonal Reactivity Index (IRI; [Davis, 1983](#)), to investigate their empathy profile.

Stimuli. The stimuli were presented on a PC running Presentation software (Version 0.60; www.neurobs.com) and consisted of centrally presented gray-scale photographs of 80 objects and 80 faces in front view taken from the Karolinska Directed Emotional Faces (KDEF) picture library ([Lundqvist et al. 1998](#); [Goeleven et al. 2008](#)). Ten male and ten female actors exhibit a neutral, fearful, disgusted and happy expression each, for a total of 80 faces stimuli, 20 for each emotion. Stimuli were resized to a visual angle of 8.23° height and 9.83° width and presented on a black background at a viewing distance of 90 cm.

EEG recording. The 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 ([Rellecke et al. 2013](#)). Vertical and horizontal electro-oculogram (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 0.01-100 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.

Experimental Task: The experimental session was run in a sound-attenuated and dimly lighted room. Participants seated in a relaxed position on a comfortable chair in front of a 17" PC monitor (refresh rate 60 Hz). Prior to the experiment a short practice session was administered to familiarize the participants with the task.

Each trial started with a central fixation period (500 ms), followed by the stimulus (1000 ms). The inter-stimuli interval randomly varied from 800, 1000 o 1200 milliseconds (Thierry et al. 2007). Participants were asked to keep their gaze fixed on the central fixation and decide whether the presented stimulus was an object or a face. In this way, the participants were not aware of the true intent of the study, and the task was implicit, in accordance with previous experiments (Experiments 1, 3, 4, 5). Responses had to be given by pressing the left and right Alt keys with the left and right index fingers, respectively. The stimulus-response mapping was counterbalanced across participants. Eye movements were controlled throughout the task with electro-oculography (EOG). Participants performed eight blocks in an experimental session. Each block contained 160 trials (80 objects and 80 faces) presented in random order, resulting in a total of 1280 test trials for each subject. The experiment lasted for approximately an hour and thirty minutes, and small breaks were allowed between blocks if the participants so desired.

Explicit rating task: After the EEG experiment, participants were asked to explicitly rate the arousal and valence of the emotional stimuli. The very same faces and objects used in the EEG experiment were presented to participants. Participants were requested to rate arousal using a nine point Likert scale, with 1 meaning “not very arousing” and 9 meaning “highly arousing”. Participants were requested to rate valence using a nine point Likert scale, with -4 meaning “very negative emotion” and +4 meaning “very positive emotion.”

Data analysis.

ERP data analysis: ERP data were analyzed using custom routines in MatLab 7.0.4 (The Mathworks, Natic, MA, USA) and EEGLAB 5.03 (Delorme and Makeig,

2004; <http://www.sccn.ucsd.edu/eeglab>). Segments of 200 ms before and 800 ms after 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. In addition, epochs contaminated with large artifacts were identified using two methods from the EEGLAB toolbox (Delorme et al., 2007): 1) An epoch was excluded whenever the voltage on an EOG channel exceeded 100 μ V to remove epochs with large EOG peaks; 2) An epoch was excluded whenever the joint probability of a trial exceeded five standard deviations to remove epochs with improbable data. Remaining blinks and horizontal artifacts were corrected by an eye movement correction procedure (Automatic Artifact Removal Toolbox Version 1.3; http://www.germangh.com/eeglab_plugin_aar/index.html; Gratton et al., 1983). The remaining epochs were averaged separately for each participant and each condition. The N170 amplitude was quantified as the mean amplitude in a time window of 130-200 ms post stimulus presentation. Scalp topographies for the N170 component were calculated as mean amplitude in a time window of 130-200 ms post stimulus presentation. In addition, EPN was calculated as the mean amplitude in a time window of 300-400 ms post-stimulus presentation.

Both N170 peak amplitude and EPN mean amplitudes were analyzed with a mixed ANOVA with Group (two levels: HA and LA) as a between subjects variable and Hemisphere (two levels: right and left), Electrodes (three levels: P, PO, O) and Stimulus (five levels: fear, happy, disgust, neutral faces and objects) 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.

Behavioral analysis: although participants were not required to respond to the emotion conveyed by faces, it is interesting to explore whether an emotion related difference in reaction time was present in HA and LA participants. To this aim, a mixed ANOVA on reaction times was performed, using Group (two levels: HA and LA) as a between subjects variable, and Stimuli (5 levels: objects, neutral faces, fearful faces, disgusted faces and happy faces) as a within subject variable. Newman-Keuls post-hoc test was used when necessary.

Explicit rating analysis: to investigate the explicit emotion recognition profile in alexithymia, LA and HA subjects' explicit arousal and valence ratings of the objects and emotional facial expressions were compared. Mean arousal and valence ratings for each group of subjects were calculated separately for the four stimulus conditions (fear, happy, neutral and objects) and submitted to mixed factors ANOVAs with Group (two levels: LA and HA) as a between-subjects variable and Stimulus (five levels: objects, neutral faces, fearful faces, disgusted faces and happy faces) as a within-subjects variable. Newman-Keuls post-hoc test was used when necessary.

Correlations: Finally, possible correlations between n170 emotional modulation and participants' emotional profile have been explored. To this aim, the ERP peaks associated with vision of affective facial displays (fear, happy and disgust) were normalized by subtracting from them the value of the ERP peak associated with vision of the neutral faces. In this way, indices for each emotion were created: emotional index = (peak amplitude for emotion – peak amplitude for neutral). These emotional indices have been correlated with the TAS, IRI and the EIS total scores and subscales scores.

The results were corrected for multiple comparisons. These analyses were performed on HA and LA groups separately.

7.2.2 Results

Self Report questionnaires

Table 7.2 summarizes the differences between the two groups in the self report questionnaire and provides the statistical data. Data were analyzed by means of the two independent t-tests, with multiple comparison correction. In brief, regarding emotion management revealed by EIS, HA groups had higher scores on the EIS verbal inhibition and disguise of feelings subscales, but not on the timidity and self-control subscale. These data are in line with previous literature ([Grandi et al. 2011](#)). Interestingly, given the different scores between the HA and LA groups in two out of the four EIS subscales, our results pointed out that alexithymia and emotional inhibition are only partially overlapping constructs. Regarding the empathy profile, revealed by the IRI, the HA subjects scored significantly different from LA subjects in two out of four subscales. In particular, they referred less ability to fantasize compared to the LA subjects and they seemed less able to take the perspectives of other people. These data provide convergent evidence in support of a difficulty in managing emotions in social relationships in HA subjects.

These features in EIS and IRI profile, decrease the likelihood of having a false positive in the HA group and a false negative in the LA group.

	Low Alexithymia	High Alexithymia	t	p
EIS total score	25.4 (5.9)	38.1 (6.4)	-6.401	<0.001*
EIS verbal inhibition	4.4 (2.3)	9.4 (2.6)	-6.188	<0.001*
EIS timidity	6.0 (2.6)	7.9 (2.9)	-2.112	0.042
EIS disguise of feelings	5.2 (2.9)	10.5 (2.1)	-6.436	<0.001*
EIS self-control	9.8(1.9)	10.2 (2.5)	-0.633	0.531
IRI total score	76.4 (7.1)	57.2 (12.4)	2.811	0.008
IRI empathy concern	20.3 (3.9)	17.8 (5.2)	1.644	0.109
IRI fantasy	22.8 (2.9)	19.7 (3.7)	2.960	0.005*
IRI perspective taking	21.5 (4.1)	14.8 (4.3)	4.932	<0.001*
IRI personal distress	11.6 (5.0)	14.8 (3.4)	-2.308	0.027

Table 7.2: Self report questionnaires results. EIS: Emotional Inhibition Scale; IRI: Interpersonal Reactivity Index. Values denotes mean (standard deviation). t = independent sample t-test; p = p-value. Asterisk denotes comparisons still significant after correction for multiple comparisons.

ERP results

N170 peak: The topography of both LA and HA group revealed that the peak amplitude was more pronounced in the electrodes O1,O2, PO7, PO8, P7, P8. Consequently, the peak amplitude of in those electrodes was submitted into an ANOVA using Group (two levels: LA and HA) as between subject variable and Hemisphere (two levels: right and left), Electrodes (three levels: O, PO and P) and Stimulus (two levels: faces, objects) as within subjects variables. The analyses revealed a main effect of Stimulus ($F[1,37]=200,7$, $p<0.001$, $\eta_p^2 = 0.84$). Post hoc test showed that the n170 for faces ($-7.48 \mu\text{V}$) was much more negative than the n170 for objects ($-2.62 \mu\text{V}$, $p<0.001$), regardless of the Group. This confirmed the presence of n170 specific for faces. Moreover, the analysis revealed a main effect of Electrodes ($F[2,74]=44.28$, $p<0.001$, $\eta_p^2 = 0.54$), indicating that the peak amplitude in P electrodes (mean -6.2 ± 0.76 s.e.m. μV) was bigger than those in PO ($-5.24\pm 0.83 \mu\text{V}$, $p<0.001$) and O

electrodes ($-3.77 \pm 0.78 \mu\text{V}$, $p < 0.001$). As a consequence, the same ANOVA on peak amplitude was carried only on P electrodes (Batty and Taylor 2003) and only on faces, in order to investigate the different effects of emotional content on the N170.

This analysis revealed a main effect of Stimulus ($F[3,111]=24$, $p < 0.001$, $\eta_p^2 = 0.39$). Neither the interaction Group x Hemisphere ($F[1,37]=0.34$, $p=0.56$, $\eta_p^2 = 0.08$), nor the triple interaction Group x Stimulus x Hemisphere ($F[3,111]=2.3$, $p=0.08$, $\eta_p^2 = 0.06$) were significant. Critically, the interaction Group x Stimulus is significant ($F[3,111]=22.9$, $p < 0.001$, $\eta_p^2 = 0.37$). This significant interaction allow to perform two separate analysis, involving the factors Group and Stimulus, one for each Group.

In the ANOVA performed on LA group, the main effect of Stimulus is significant ($F[3,54]=24.7$, $p < 0.001$, $\eta_p^2 = 0.57$). Post hoc test showed that the peak amplitude for neutral faces ($-9.03 \pm 0.92 \mu\text{V}$) was significantly less negative compared to the peak amplitude for fear ($-10.16 \pm 0.91 \mu\text{V}$, $p < 0.001$), for disgust ($-10.39 \pm 0.97 \mu\text{V}$, $p < 0.001$), and for happiness ($-9.67 \pm 0.93 \mu\text{V}$). Additional differences within emotions emerged: both the emotions of disgust and fear resulted to be significantly different from the emotions of happiness ($p=0.006$ and $p < 0.001$ for fear and disgust, respectively), but no difference between fear and disgust emerged ($p=0.18$). Results are shown in *Figure 7.1*.

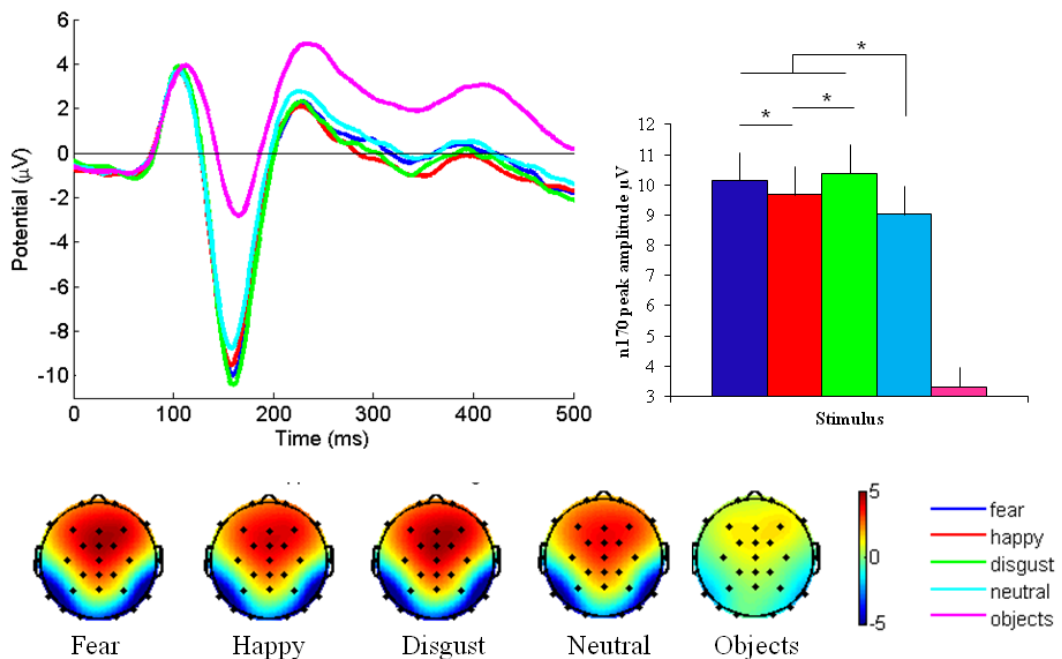


Figure 7.1. *n170* results in LA participants. In the left side, ERPs elicited by different stimuli. The images represent the *n170* waveform amplitude over P7 and P8. In the right side, the histogram represent the mean waveform peak amplitude as a function of Stimulus. Error bars denotes standard error of the mean. In the bottom row, the scalp topographies of the most negative peak (in blue) at the time window of 130-200 ms following stimulus onset.

In the ANOVA performed on HA group, the main effect of Stimulus was significant ($F[3,57]=21.37$, $p<0.001$, $\eta_p^2 = 0.52$). Post hoc test showed that the peak amplitude for neutral faces ($-8.80\pm 0.99 \mu\text{V}$) and for happy faces ($-8.66\pm 1.03 \mu\text{V}$) were more negative than the peak amplitude for fearful ($-8.08\pm 1 \mu\text{V}$, all comparisons $p<0.001$) and more positive than the peak amplitude for disgusted ($-9.23\pm 1.01 \mu\text{V}$, all comparisons $p<0.005$) faces. Moreover, the peak amplitude for disgust was significantly more negative than peak amplitude for fear (-9.23 ± 1.01 vs -8.08 ± 1 , $p<0.001$). It is worth noting that in this case, contrarily to the LA subjects, the emotion of fear

produces a less negative n170 amplitude compared with the neutral, happy and disgusted faces. In addition, the emotion of disgust produced the most negative n170 amplitude comparing to all the other faces. Results are shown in *Figure 7.2*.

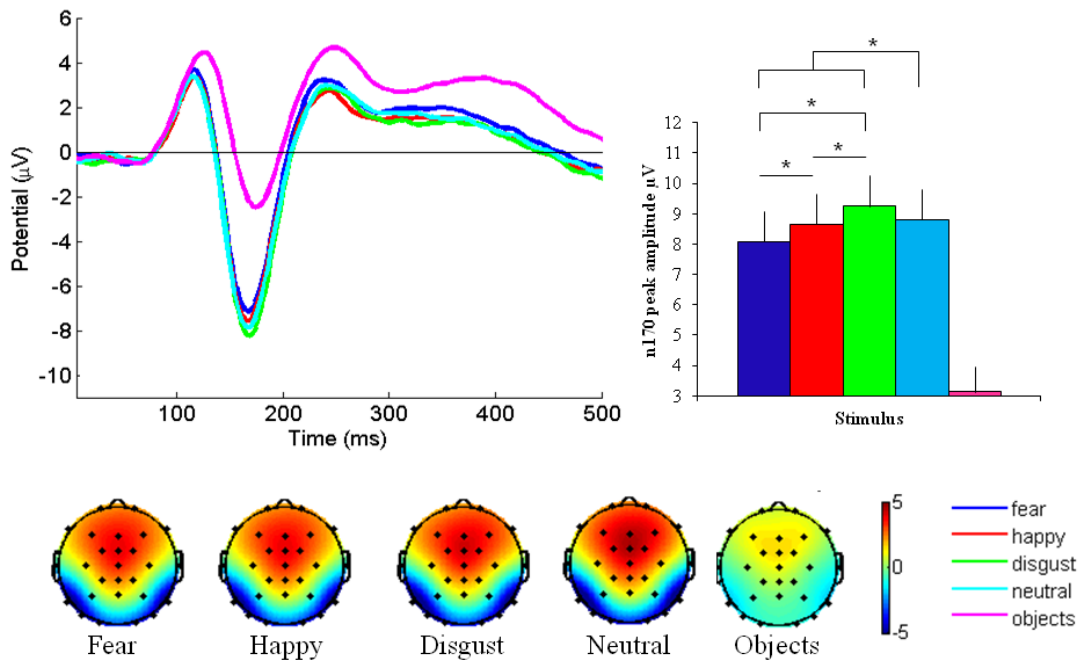


Figure 7.2. n170 results in LA participants. In the left side, ERPs elicited by different stimuli. The images represent the n170 waveform amplitude over P7 and P8. In the right side, the histogram represent the mean waveform peak amplitude as a function of Stimulus. Error bars denotes standard error of the mean. In the bottom row, the scalp topographies of the most negative peak (in blue) at the time window of 130-200 ms following stimulus onset.

N170 latency: An ANOVA on n170 peak latency was carried out using Group (two levels: LA and HA) as between subjects factor and Hemisphere (two levels: right and left) and Stimulus (four levels: fear, happy, disgust, neutral) as within subjects factors. The analysis revealed a significant triple interaction Group x Hemisphere x Stimulus ($F[3,111]=2.7$, $p=0.04$, $\eta^2 = 0.06$). Thus, LA and HA group will be analyzed in two separate ANOVA using Hemisphere and Stimulus as within factor.

The ANOVA on LA group revealed a main effect of Stimulus ($F[3,54]=6.7$, $p<0.001$, $\eta^2 = 0.27$). Post hoc test showed that responding to happy faces (161.8 ± 1.59 ms) is faster than responding to fearful (164.1 ± 1.9 ms, $p=0.001$) and disgusted faces (163.8 ± 1.75 ms, $p<0.002$). No other differences between faces emerged. The main effect of Hemisphere ($F[1,18]=0.54$, $p=0.46$, $\eta^2 = 0.01$) and the interaction Hemisphere x Stimulus ($F[3,54]=0.40$, $p=0.75$, $\eta^2 = 0.02$) were not significant.

The ANOVA on HA group revealed a significant interaction Hemisphere x Stimulus ($F[3,57]=2.99$, $p=0.03$, $\eta^2 = 0.13$). Post hoc test showed that within each hemisphere there was no significant difference among the stimuli (all $p>0.32$). However, n170 has a larger latency in the right hemisphere compared to the left one in responding to disgusted (168.3 ± 1.1 for the left hemisphere vs 171.4 ± 1.2 for the right hemisphere, $p=0.01$) and neutral stimuli (167.7 ± 1.13 for the left hemisphere vs 170.9 ± 1.4 for the right hemisphere, $p=0.009$). For the emotion of happiness (168.8 ± 1.5 for the left hemisphere vs 171.4 ± 1.4 for the right hemisphere, $p=0.06$) and fear (170.2 ± 1.09 for the left hemisphere vs 170.1 ± 1.2 for the right hemisphere, $p=0.91$), no between hemisphere differences emerged.

EPN: The ANOVA on mean amplitude using Group (two levels: LA and HA) as between subjects factor and Hemisphere (two levels: right and left) and Stimulus (two levels: faces, objects) as within subjects factors revealed a significant main effect of Stimulus ($F[1,37]=61.1$, $p<0.001$, $\eta^2=0.62$). Post hoc test showed that the EPN mean amplitude was smaller for objects (2.05 ± 0.40 μV) than for faces (0.37 ± 0.39 μV , $p<0.001$). Thus, an ANOVA was performed on faces only, using Group (two levels: LA and HA) as between subjects factor and Hemisphere (two levels: right and left) and Stimulus (four levels: fear, happy, disgust, neutral) as within subjects. The analysis

revealed a main effect of Stimulus ($F[3,111]=9.24, p<0.001, \eta^2=0.19$). Post hoc test highlighted that the neutral faces (mean amplitude $0.51\pm 0.27 \mu\text{V}$) induced a smaller (i.e. more positive) EPN compared with the emotion of disgust ($0.19\pm 0.28 \mu\text{V}, p=0.002$) and happiness ($0.17\pm 0.39 \mu\text{V}, p=0.004$) and no difference between neutral and fearful faces emerged ($0.51\pm 0.27 \mu\text{V}$ vs $0.60\pm 0.27 \mu\text{V}, p=0.38$). The variable Group did not interact with the variable Stimulus ($F[3,111]=2.51, p=0.07, \eta^2=0.06$). Results are depicted in *Figure 7.3*, left side.

However, the interaction Group x Hemisphere was significant ($F[1,37]=8.28, p=0.006, \eta^2=0.18$). Thus, the effect of hemisphere has been assessed in the two groups separately. In the LA the main effect of Hemisphere is not significant ($F[1,18]=0.96, p=0.33, \eta^2=0.04$), thus revealing a similar EPN amplitude in the two hemisphere ($0.25\pm 0.21 \mu\text{V}$ and $-0.19\pm 0.17 \mu\text{V}$ for the left and right hemisphere, respectively). On the contrary, in the HA the main effect of Hemisphere is significant ($F[1,19]=8.69, p=0.008, \eta^2=0.31$), due to the EPN amplitude being smaller in the right ($1.49\pm 0.53 \mu\text{V}$) than in the left ($-0.09\pm 0.21 \mu\text{V}, p=0.008$) hemisphere. Results are depicted in *Figure 7.3*, right side.

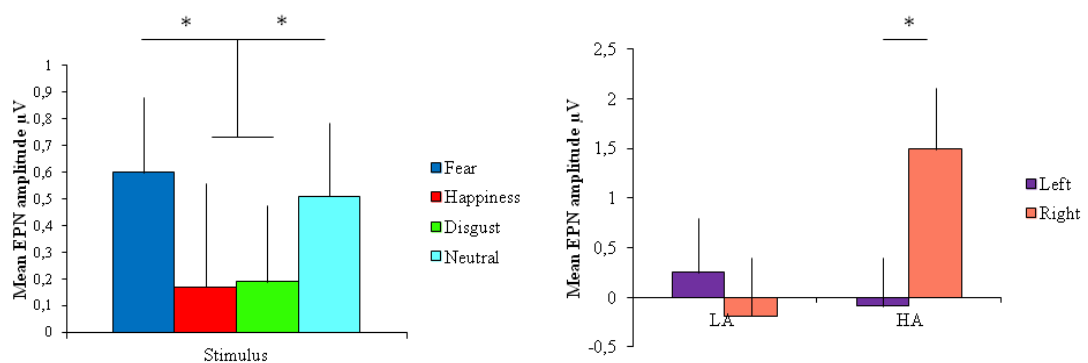


Figure 7.3. EPN results. On the left side, the overall effect of Stimulus on the EPN mean amplitude is shown. On the right side, it is possible to appreciate the contribution of the two hemisphere to the EPN amplitude, in LA and HA group separately. Error bars denotes s.e.m.: standard error of the mean.

Behavioral results: reaction times

The ANOVA on RT of correct responses revealed a main effect of Stimulus ($F[4,148]=3.91$, $p=0.004$, $\eta^2 = 0.5$), denoting that RT were slower when responding to an object (mean 487.6 ± 9.25 ms) than when responding to a faces, regardless the emotion expressed by faces (mean 474.8 ± 10.03 ms for fearful faces, 474.3 ± 10.01 ms for happy faces, 477.6 ± 10.39 ms for disgusted faces, 476.6 ± 10.3 ms for neutral faces, all comparison with objects $p<0.009$). No differences in RT between different faces emerged (all $p>0.69$). The main effect of Group ($F[1,37]=1.69$, $p=0.2$, $\eta^2 = 0.04$) and the interaction Group x Stimulus ($F[4,148]=1.4$, $p=0.23$, $\eta^2 = 0.03$) were not significant. The ANOVA was then repeated on faces only. The main effects of Group ($F[1,37]=1.9$, $p=0.17$, $\eta^2 = 0.04$) and Stimulus ($F[3,111]=1.3$, $p=0.27$, $\eta^2 = 0.03$) were not significant. The interaction Group x Stimulus ($F[3,111]=0.24$, $p=0.86$, $\eta^2 = 0.01$) was not significant as well.

Correlations

Correlation between n170 and TAS-20 subscales. In the LA group, the disgust index correlated with the DIF subscale ($R=0.58$, $p=0.008$). However, this significant correlation did not survive after multiple comparison correction. In the HA group, the fear index correlated with the DIF subscale ($R=0.70$, $p=0.0005$) and is still significant after multiple comparison correction. This denotes the the bigger the difficulty in identifying feeling, the bigger the difference between fear and neutral waveform. Please note that in HA subjects the expression of fear induced a less negative n170 waveform. Please see *figure 7.4*, left side.

Correlation between n170 and EIS. In the LA group, the fear index correlated with the Timidity subscale ($R=0.46$, $p=0.04$), and the disgust index with the EIS total

score ($R=-0.54$, $p=0.01$) and with the Self control subscale ($R=-0.57$, $p=0.009$). No one of these correlations were still significant after multiple comparisons correction. In the HA group, the fear index correlated with the EIS total score ($R=0.74$, $p=0.0001$) and with the self control subscale ($R=0.58$, $p=0.006$). The correlation between fear index and EIS total score is still significant after multiple comparisons correction. Please see *figure 7.4*, right side.

Correlation between *n170* and *IRI*. In the LA group, the disgust index correlated with the personal distress subscale score ($R=0.52$, $p=0.02$). In the HA group, the fear index correlated with the empathic concern subscale score ($R=0.50$, $p=0.02$). Both these correlations were no more significant after multiple comparison correction.

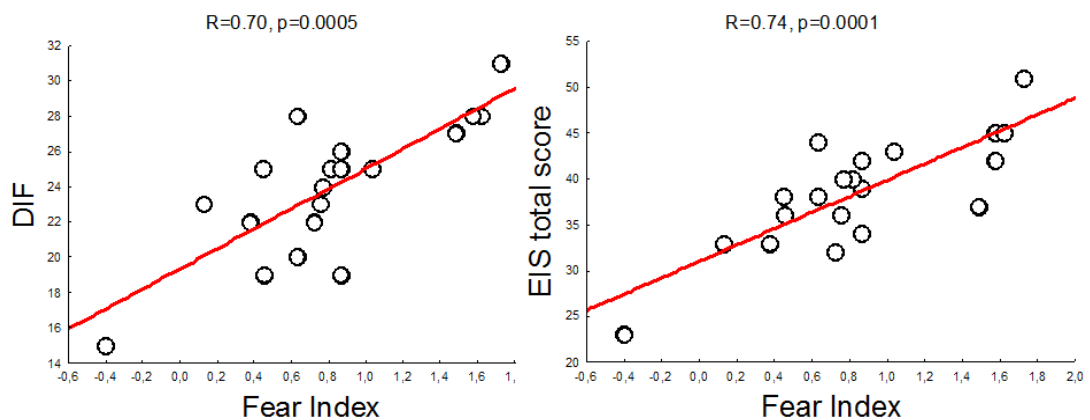


Figure 7.4. Correlations between Fear index and Difficulty in identify subscale and Emotional Inibition total score in High Alexithymia Participants.

Explicit rating results

The following analysis were performed using Group (two levels: LA and HA) as between subjects variable and Stimulus (four levels: fear, happy, disgust and neutral) as within subjects variable.

The analysis was, a significant main effect of Stimulus ($F[3,93]=3045$; $p<0.001$; $\eta_p^2 = 0.99$) was found. Fearful (mean rate: 7.9 ± 0.03 s.e.m.) and disgusted (7.8 ± 0.07) faces were rated as more arousing than the happy (6.8 ± 0.07 , $p<0.001$) and neutral faces (0.71 ± 0.06 , $p<0.001$), while fearful faces rating did not differ from the disgusted ones ($p=0.3$). Happy faces are more arousing than neutral faces ($p<0.001$). The main effect of Group ($F[1,31]=0.01$; $p=0.91$; $\eta_p^2 = 0.01$) and the interaction between Group and Stimulus ($F[3,93]=1.42$; $p=0.24$; $\eta_p^2 = 0.02$) were not significant.

With respect to the valence ratings, a significant main effect of Stimulus ($F[3,111]=4669$; $p<0.001$; $\eta_p^2 = 0.99$) was found. Fearful (mean rate: -3.08 ± 0.03) and disgusted (-3.17 ± 0.04) faces were rated as more negative than happy (3.06 ± 0.04 , $p<0.001$) and neutral faces (-0.17 ± 0.04 , $p<0.001$), while fearful faces rating did not differ from the disgusted ones ($p=0.13$). Happy faces were rated as more positive than all the other faces (all comparisons $p<0.001$). The main effect of Group ($F[1,37]=0.94$; $p=0.33$; $\eta_p^2 = 0.01$) and the interaction between Group and Emotion ($F[3,111]=1.66$; $p=0.17$; $\eta_p^2 = 0.02$) were not significant.

7.3 Discussion

Seeing images of faces elicits a robust negative deflection peaking at 170 ms post-stimulus onset (N170) reflecting the early structural encoding of these stimuli (Itier and Taylor, 2004; Bentin and Deouell, 2000; Bentin et al. 1996), and a subsequent relative

negativity (EPN) indexing attentional engagement to salient stimuli (Schupp et al., 2006; Olofsson et al., 2008). Both these components are modulated by the emotion conveyed by the face (Eimer and Holmes, 2007; Rellecke et al. 2013), in particular the emotional content of faces is reflected by the heightened amplitudes of the waveforms (i.e. the waveforms are more negative). The results of *Experiment 8* revealed the presence of the n170 modulation by emotions in participants with low alexithymia (LA) but not in participants with high alexithymia (HA), reflecting between-group differences in the visual encoding of faces. On the contrary, at the later stage of visual processing, no between-group differences emerged in the emotional modulation of EPN, reflecting LA and HA similarity in attention engagement. Moreover, in the behavioral data, the HA participants' performance did not differ from the LA's one, either in the reaction time to different emotional faces, or in the explicit evaluation of emotional arousal and valence of the stimuli, in accordance with previous experiments.

In details, with respect to the structural encoding of faces, the results on n170 of *Experiment 8* in LA subjects revealed that both the emotions of fear and disgust are able to modulate the early stage of the visual encoding of faces, as reflected by heightened negativity of n170 in fear and disgust compared to neutral and happy faces. No differences between the emotions of fear and disgust emerged. This is in accordance with previous evidences showing the larger n170 amplitude for negative emotions compared with positive ones (Batty and Taylor, 2003; Blau et al. 2007; Carlson and Reinke, 2010; Miyoshi et al. 2003; Pegna et al. 2008, 2011; Lee et al. 2007; Lyn and Salisbury, 2008, Caharel et al. 2005). Also in the HA group there is a modulation of emotion on the n170 amplitude, but while the emotion of disgust induced larger n170 amplitude compared to neutral ones, as in the LA group, the emotion of fear induced a

smaller n170 amplitude compared to neutral, happy and disgusted faces; thus highlighting an encoding problem of fearful faces in HA. Interestingly, in the HA group only, the smaller the fear induced n170 amplitude, the higher the participant's experience alexithymia related difficulties, namely difficulty in identifying feelings (DIF TAS-20 subscale) and emotional inhibition (EIS total score). Moreover, encoding difficulties in the HA group is also highlighted by the bigger latency of their n170 for disgusted and neutral faces.

Electrophysiological studies suggest that the N170 component represents the process of extraction of the relevant perceptual properties of the human faces for categorization (Eimer and Holmes, 2002; Bentin and Deouell, 2000; Eimer et al. 2011) and is considered the earliest component indexing structural features of faces. However, the n170 also discriminates the emotion conveyed by the observed faces. This emotional modulation of n170 has consistently been associated with threat related emotions, which elicit a larger amplitude of n170 compared to neutral faces (Batty and Taylor, 2003; Blau et al. 2007; Carlson and Reinke, 2010; Miyoshi et al. 2003; Pegna et al. 2008, 2011; Lee et al. 2007; Lyn and Salisbury, 2008; Caharel et al. 2005) and positive emotional faces (Caharel et al. 2005; Krombholz et al. 2007; Lyn and Salisbury, 2008). This is exactly what has been found in LA participants. Such emotional modulation of the structural encoding might reflect an adaptive mechanism, in which the perceptual representation of potentially threatening stimuli is enhanced. Similarly, neuroimaging studies showed that observing both fearful and disgusted faces enhance the activation of the fusiform gyrus and inferior parietal lobule (Fusar-Poli et al. 2009a; Vytal and Hamann, 2010) from which the N170 is originated (Haxby et al. 2000; Itier and Taylor, 2004). Importantly, fearful faces in particular, and disgusted faces in lesser extent, are

known to enhance activation in the amygdala (Fusar-Poli et al. 2009a; Vytal and Hamann, 2010), the key subcortical structure for signaling fear and potential threat (LeDoux, 2014; Adolphs, 2013). Therefore, the enhanced N170 over the right parietal electrodes might reflect a distant functional influence of the amygdala on interconnected visual cortices, useful for effective perceptual processing of threat signals (Vuilleumier et al., 2004).

The new findings of *Experiment 8* revealed that the emotional modulation of n170 observed in LA individuals is present in participants with high alexithymia as well. However, while the emotion of disgust induced larger n170 amplitude compared to neutral ones, as in the LA group, the emotion of fear induced a smaller n170 amplitude compared to neutral, happy and disgusted faces, highlighting in this way an encoding problem of fearful faces in HA. This inverse fear induced modulation of n170 seems to be specifically linked to the alexithymia profile. Indeed, the smaller the fear induced n170 amplitude the higher the difficulty in identifying feeling (DIF subscale) and the total emotional inhibition score (EIS) of HA participants. These strong correlations between electrophysiological findings and behavioral measures further support the conclusion that the current results are tightly related to participant's difficulties in dealing with the emotion of fear. Indeed, the results on DIF suggest that the smaller the fear induced n170 amplitude reflected the difficulties of HA participants in recognizing this emotion. This finding is perfectly in line with the existent literature, since the DIF subscale has been previously found to correlate with other ERP component in the alexithymic group (Maier et al. submitted; Pollatos et al. 2008, see also *Experiments 1, 3 and 4*). This suggests that the DIF TAS-20 subscale is the more sensitive in highlighting HA difficulties. Moreover, the data on EIS suggest that the disruption of

the process of fear encoding might also be due to participants' emotional inhibition. The EIS questionnaire has been previously used in alexithymia research (Grandi et al. 2011), but has never been correlated with behavioral or electrophysiological data in alexithymia.

In keeping with the above suggested hypothesis, the smaller n170 amplitude induced by the emotion of fear in alexithymia might reflect the disruption of the distant functional influence of the amygdala on visual cortices. This hypothesis allows us to explain the observed significant differences between the n170 evoked by the emotion of fear and disgust. Indeed, in normal subjects, the amygdala becomes activated in the presence of threats, thus enhancing the visual processing in order to better perceive the threat signals (Vuilleumier et al., 2004). Critically, the amygdala has been found to be consistently hypoactivated in response to negative stimuli in alexithymia (van der Velde et al. 2013, for a meta-analysis). The amygdala conveys information about threat related emotions, but it is especially relevant in processing the emotion of fear (Fusar-Poli et al. 2009a; Vytal and Hamann, 2010; LeDoux, 2012, 2014), as suggested by neuro-imaging meta-analysis (Phan et al., 2002; Vytal and Hamann, 2010; Fusar-Poli et al., 2009a), animal models (Davis et al., 1994), single-unit recordings (Maren, 2001) and lesional studies (Adolphs et al., 1994). This preferential amygdalar activation in response to fearful stimuli explains why a reduced amygdalar modulation on visual cortices might be related to the smaller n170 amplitude for fear, but a normal (i.e. larger) n170 amplitude for disgust, compared to the neutral ones, observed in HA participants.

Interestingly, at a later stage of visual processing (i.e. after 300 ms post-stimulus onset), the EPN component emotional modulation did not differ between groups. However, since the interaction between group and stimulus is really close to the

significance ($p=0.07$), this might be due to the fact that the sample size might be too small to be able to highlight the effect. Thus, the current results regarding the absence of EPN amplitude between groups should be considered with caution. More interestingly, the EPN result was enhanced (i.e. more negative) for disgusted and happy stimuli compared with neutral ones in both groups. The EPN represents a relative negativity for emotional stimuli (Schupp et al., 2006). Such an emotional modulation indexes a stimulus-driven attentional capture of salient emotional stimuli, which might reflect the degree of attention needed for recognition of relevant signals (Oloffson et al., 2008). Previous studies have shown increases 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). Notably, the results did not show a significant difference between fearful and neutral faces. The absence of fear related EPN modulation is somehow surprising, but the effect seems to be robust. Further research on LA and HA participants are needed in order to further investigate and better understand this result. Moreover, among the negative emotions, the fear related RPN is smaller than the disgusted related ones. A recent study showed that the EPN amplitudes were augmented for both disgusting and threatening relative to neutral images, though significantly more for disgust (Wheaton et al. 2013), suggesting that disgusting stimuli might modulate this component to a larger extent than fearful stimuli.

Notably, since in the LA group no difference between the two hemispheres emerged in the EPN amplitude, the current results also highlight that in the LA participants the process of attentional engagement to salient stimuli is carried out similarly by both the hemispheres. This is in accordance with previous studies that

found no support for the hypothesis of right-lateralization of emotional processing (Fusar-Poli et al. 2009b for a meta-analysis). However, it is interesting to note that this is not true in HA participants, where the smaller EPN amplitude in the right hemisphere compared to the left one might reflect a difficulty of the right hemisphere in carrying out the attentional engagement to salient stimuli, such as emotional stimuli. This indicates that, in HA, at the later stages of the visual process the right hemisphere is less efficient in engaging attentional resources for the recognition of salient emotional stimuli. This is in line with early neurobiological theories of alexithymia, which suggest a dysfunction of the right cerebral hemisphere (Larsen et al. 2003 for a review).

Overall, these results of *Experiment 8* further corroborate the results of previous experiments (please see *Experiments 1, 3, 4* and *5*) that the emotional processing difficulties in HA might be specific for fear. Moreover, the current results also suggest that the anomalies of fear processes in HA individuals starts as early as with the visual encoding of faces, as reflected by the fear induced smallest n170 amplitude. However, at a later stage of visual processing, alexithymic participants exhibit a normal attention engagement, as revealed by the absence of group differences in the EPN amplitude. The EPN results, however, are still preliminary and need to be further explored by increasing the sample size. However, at this later stage, alexithymic difficulties emerged when the activities of the two hemispheres were directly compared. Indeed, in HA subjects only, the right hemisphere is less able than the left one in directing the attention onto salient stimuli.

In conclusion, such an highly efficient and specialized structural encoding and subsequent attentional engagement for fearful faces, which seems to mark a powerful

tool to infer information about goals, intentions and emotions of others, and represents an adaptive mechanism useful to facilitate social communication is impaired in alexithymia. This could account for the difficulties experienced by these individuals in everyday life.

CHAPTER 8

General Discussion

“The proof that the little prince really existed was that he was enchanting, that he laughed and that he wanted a sheep. Now when you want a sheep, it proves that you exist”.

The Little Prince
Antoine de Saint-Exupéry

The proof that Little Prince really existed is that he laughed and he wanted a sheep. In other words, he felt and expressed emotions and he was able to use his emotions to make choices. He expressed the desire of having a sheep and he missed his rose. Due to this latter feeling, the Little Prince decided to come back to his planet. Although humans being are not usually able to see sheep throughout the box, they are similar to the Little Prince in the way they feel and communicate their own emotions. Indeed, humans beings are social animals, and the ability to communicate the deep feelings, both verbally and bodily, are indispensable to behave correctly in a social context. Moreover, humans should also understand the emotion of others, in order to adapt continuously to their social signals. They do that by “embodying” the emotions of others, i.e. re-experiencing, re-creating the emotions of others on their own self (Niedenthal, 2007; Niedenthal et al. 2010).

However, the current dissertation revealed that alexithymic individuals are lacking in these abilities. Although the research on primary alexithymia is still at its beginning, alexithymic individuals are known to experience difficulties in dealing with their own

and others' emotions. Please refer to Chapter 2 for a synthetic review of the existent literature. Critically, to date, there are no studies investigating the ability of alexithymic individuals in embodying the emotions they see expressed by others. Therefore, it remains unclear whether the emotional difficulties of alexithymic individuals are also manifested in a deficit in embodying other's emotion. In particular, to date there are no studies investigating their embodiment abilities during emotion observation conveyed by human faces.

In the present thesis, different paradigms and techniques falling within the field of affective neuroscience have been employed in order to test a possible deficit in the embodiment of emotions in alexithymia while subjects were requested to observe faces expressing different emotions. However, during the main experimental paradigms, participants were never asked to respond to each emotion in a specific way. Indeed, the tasks selected were always indirect, i.e. the participants were asked to focus their attention on something other than emotions, for example the tactile stimulation delivered on their cheeks, or the gender of the observed faces. Importantly, participants were selected using a highly selective procedure involving the screening of possible alexithymia by means of the Toronto Alexithymia Scale (TAS-20, [Taylor et al. 2003](#)) and a subsequent confirmation of the presence of alexithymia by means of the Alexithymia Subscale of the Diagnostic Criteria for Psychosomatic Research (DCPR, [Mangelli et al. 2006](#)). Additionally, participants were excluded if they manifested clinically relevant signs of depression, as assessed using the SCID ([First et al. 1997](#)). Finally, the EIS ([Kellner, 1987](#)), IRI ([Davis, 1983](#)) and BPQ ([Porges, 1993](#)) were often administered to further investigate the included participant's emotional profile. Notably, the specific emotional (EIS), empathy (IRI) and bodily perception (BPQ) profile are

widely different between participants with low and high level of alexithymia, thus decreasing the possibility of participants' misclassification.

Summarizing, the current thesis has at least three strengths: first, the same experimental stimuli have been used in all the experiments, thus making the results of different experiments comparable. Secondly, the task was always indirect, so the stimuli were consciously perceived but the participant's attention was deliberately diverted from the emotional content of the stimuli. Thirdly, the participants' selection procedure has ensured that they were as homogeneous as possible, and this has been further confirmed by the EIS, IRI and BPQ results. Notably, in the only study testing patients with brain lesions, the selection procedure is highly selective as well. This resulted in few patients screened in for the study, but in a highly homogeneous sample.

Moreover, three emotions have been studied in the current dissertation, namely fear, disgust and happiness. The emotion of fear and disgust were chosen as emotions of interest to be studied in order to investigate whether the alexithymics' difficulties in dealing with negative emotions could be expanded also to the embodiment of these emotions. The results of fear are highly consistent across the different experiments, since they clearly showed a deficit of HA subjects in processing the signals of fear conveyed by faces, both at the perceptual level and at the somato-sensory and sensory motor levels. On the contrary, the results on disgust are still not conclusive, since they showed a normal perceptual encoding of disgust, coupled with heightened activity of the somato-sensory system in HA compared with LA participants. However, the emotion of disgust has never been studied in alexithymia, and in the current dissertation the disgusted faces have been used in two experiments only. Thus, the results and conclusion should be considered as explorative. Finally, the emotion of happiness has

been initially selected as control condition. However, the results of the experiments conducted in the current thesis revealed interesting results for happiness, too. Indeed, HA participants seemed to show difficulties also in the processing of happiness. However, these difficulties are not manifested in an absence of the effect, but in a delayed one in the sensory-motor system.

It is important to note that in every experiment performed, no between group emerged in the explicit rating of emotions arousal and valence. Furthermore, no between group emerged in the evaluation of cognitive functions (*Experiment 2*), thus ruling out the hypothesis that a different neuropsychological profile might account for the obtained results.

In the following paragraphs, the main findings of the implicit task will be summarized for each emotion, separately. Finally, a unifying interpretation will be provided.

8.1 Alexithymia and happiness:

Although initially selected as a control, positive valence emotion, happiness, has been found to be an interesting emotion in alexithymia. Indeed, the overall results on happiness revealed a defective perceptual encoding of this emotion, coupled with a slight difficulty at the level of the communicative somato-motor system in high alexithymia compared with low alexithymia participants.

In detail, in order to investigate if the early components of emotional processing are altered in alexithymia, the perceptual structural encoding of happiness and the following stimulus-driven attentional capture were explored by means of electroencephalography. In HA participants, the early perceptual processes of faces

when they manifest the emotion of happiness resulted to be defective. Indeed, while in LA participants the emotion of happiness induced a larger n170 compared to the neutral faces, this was not true in HA individuals (*Experiment 8*). On the contrary, at later stage of perceptual representation, the happiness driven attentional capture in HA subjects was comparable to those of LA participants, since in both groups the emotion of happiness induced a larger EPN compared to the neutral faces (*Experiment 8*). Probably this correct attention allocation compensates for the earlier deficit in the structural encoding of the features of happiness, thus accounting for the results on the somato-sensory and somato-motor system in HA, which are almost normal, with the only exception of a slight delay in communicative motor responses to happiness.

In particular, the results regarding HA abilities to embody the emotion of happiness in their own somato-sensory system, which was studied by means of the VRT paradigms, revealed normal somato-sensory reactions to happiness. Indeed, the results revealed the presence of the VRT effect when HA but not when LA participants saw happy faces being touched compared to being just approached by fingers. These results were interesting, since in the VRT paradigms the electrodes delivering tactile stimulation are placed upon the *Zygomaticus Major*, which is known to be involved in the spontaneous synchronization of the somato-motor system with the seen emotion. Thus, the lack of VRT effect in the presence of happy faces in LA (*Experiment 1*) and in healthy participants ([Cardini et al., 2012](#)) could represent a by-product of the motor resonance phenomenon. Indeed, the rapid muscle activity elicited by happy faces could have interfered with the discrimination of the electro-tactile stimuli placed on the cheeks. The unexpected presence of the VRT effect in HA seemed to suggest the absence of motor resonance phenomenon for happiness in these subjects.

Interestingly, *Experiment 3*, investigating the embodiment of emotion in the somato-motor system, specifically studied the motor resonance by measuring the RFRs in response to happiness. The results revealed that HA participants did manifest rapid facial reactions in response to happy faces, however, their reaction is belated (750-1000 ms) compared to those of LA's participants (500-750 ms) (*Experiment 3*). These results likely indicate that HA traits are associated with the presence, although delayed in time, of the embodiment of the motor aspect of this emotion. On the contrary, the emotion of happiness did not have any influence either in LA and HA group on the non communicative (i.e. visceral, cardiovascular) motor system, as revealed by *Experiment 4*. The results on the somato-motor system (*Experiment 3*) might also partially explain the results on the somato-sensory system (*Experiment 1*), i.e. the absence of the VRT effect in LA but not in HA participants. Indeed, in the VRT task the electrical/tactile stimulation is provided at 700 ms after stimulus presentation. In LA participants, the RFRs for happiness were manifested between 500 and 750 ms after stimulus presentation and thus are likely to disrupt the perception of the near-threshold stimulation in the VRT task. At variance, in the HA participants, the RFRs for happiness are delayed in time, and they are manifested between 750 and 1000 ms after stimulus presentation, and thus they happened after the electrical stimulation provided in the VRT task.

Summarizing, the results of the current dissertation on the emotion of happiness suggest that alexithymic individuals manifest some difficulties in the processing of happiness. These difficulties are clear at the level of the perceptual encoding of faces. However, although this latter process is altered in alexithymia, HA participants

correctly allocated the attention on happy faces, and this probably compensates for the earlier deficit in the structural encoding of the features of happiness. Indeed, at later stages of emotional processing, no particular difficulties emerged in the embodiment of happy related information in the somato-sensory and in the somato-motor systems. The only intriguing datum is the slower communicative motor system of HA participants in response to happiness, as revealed by the delayed rapid facial reaction in response to this emotion, which seems to suggest that the difficulties of HA participants in the embodiment of happiness is not revealed by an absence of the effect, but rather in delayed HA reactions. Further studies are needed to investigate the possible link between the early processing deficit and the following delayed motor reactions in response to happiness in HA.

8.2 Alexithymia and disgust:

The current dissertation also provided preliminary results on the ability of HA participants to deal with the emotion of disgust. The only clear difference between groups emerged for the embodiment of disgust at the somato-sensory level. Although the findings are really interesting, they should be considered with caution because they are explorative. Indeed, the emotion of disgust has been used in two experiments only (*Experiments 5 and 8*) and was never been used in alexithymia research before.

At the level of the perceptual encoding, a slight difference between LA and HA groups emerged. Indeed, even if in both groups the emotion of disgust improves the structural encoding of faces, as revealed by the larger amplitude of the disgust induced n170 amplitude compared to the neutral faces induced n170 amplitude (*Experiment 8*), in HA only the n170 peak for disgust was delayed in time in the right hemisphere

compared with the left one (*Experiment 8*). This between-hemisphere difference, which is present in the HA group only, might indicate that the right hemisphere is slightly less efficient, i.e. slower, in the perceptual encoding of the fear related information. This is in line with early neurobiological theories of alexithymia, which suggest a dysfunction of the right cerebral hemisphere (Larsen et al. 2003 for a review).

Even more interesting, the emotion of disgust strongly influenced the somato-sensory system in HA participants but not in LA ones. Indeed, this emotion produced an abnormally high VRT effect, i.e. an enhancement of tactile perception when participants saw faces being touched compared to being just approached by fingers, in HA group only (*Experiment 5*). This suggests, in HA participants, an abnormally high embodiment of disgust at the somato-sensory level. These results are likely to reflect the strong visceral changes induced by the experience of disgust (Eckman et al., 1983; Page, 1994; Susskind et al. 2008), and HA predisposition to abnormally detect internal bodily sensations (Wise and Mann, 1994; Nyklicek and Vingerhoets, 2000; Nakao et al. 2002). To test this possibility, *Experiment 6* was performed, in which participants were asked to evaluate their own heart frequency at rest. This task is known as “heartbeat perception task” and provided an index of interoceptive ability, namely the ability to perceive signals from one own body (Schandry, 1981). As expected, HA participants have been found to have an higher interoception compared to LA ones. Critically, the interoceptive index strongly correlated with the somato-sensory embodiment of disgust. These data support the hypothesis that the embodiment profile at the somato-sensory level of HA individual might be related to their tendency to exhibit increased responses to stimuli producing heightened “physical” and visceral sensations, and that HA might rely mainly on “physical” information while performing the multisensory stimulation

paradigm. This is in accordance with the notion that HAs tend to experience bodily sensations in an emotion-provoking situation (Karlsson et al. 2008; Lane et al. 2004).

Summarizing, the results of the current dissertation on the emotion of disgust suggest that alexithymic individuals manifest some difficulties in the processing of disgust. These difficulties are nuanced at the level of the perceptual encoding of faces. Indeed, they are manifested only in a slight delay on which the right hemisphere elaborated the disgusting information conveyed by the observed faces. On the contrary, the abnormality of the disgust processing in alexithymia is very clear at the level of the embodiment of this emotion in the somato-sensory system. Indeed, the emotion of disgust is over-embodied in alexithymics compared with the low alexithymic participants. This might reflect the intrinsic characteristics of both the emotion of disgust (i.e. which induces strong bodily and visceral reactions) and alexithymic subjects (i.e. which are highly focalized on visceral changes). Further studies are needed to investigate if the over reactivity to disgust observed in HA somato-sensory system is present also at the level of somato-motor system, both the communicative and non communicative ones.

8.3 Alexithymia and fear:

The main result of the current dissertation regards the embodiment of fearful information. Indeed, data from *Experiments 1,3,4* and *5* provided convergent evidence supporting that the mechanism of the embodiment of fear is widely defective in alexithymia, both in the somato-sensory and in the sensory-motor (communicative and non communicative) systems. Furthermore, the problems of HA individuals in the

processing of fearful information seems to start as early as the perceptual encoding of faces.

In detail, in *Experiment 8* the perceptual encoding of faces and the subsequent stimuli-driven attentional capture processes were investigated. To test the possibility that HA might be impaired in these early processes, participants were asked to observe neutral faces, emotional faces and objects and categorize the seen stimuli as face or object, while their electroencephalography was recorded. Subsequently, two waveforms have been analyzed: the n170, reflecting the structural encoding of face process, and the EPN, reflecting a stimulus-driven attentional capture. Interestingly, results revealed a disruption in the early perceptual process of faces in alexithymia. Indeed, in HA the fear-induced n170 is smaller than the neutral-induced n170, whereas in LA participants the opposite pattern emerged, i.e. the fear-induced n170 is larger than the neutral-induced n170. These results strongly correlated with the difficulty in identifying feelings using the TAS-20 subscale, further supporting the strict relationship between the electroencephalographic results and participant's alexithymia profile. Contrarily, no fear-induced modulation of EPN amplitude compared to neutral-induced EPN amplitude emerged in both groups, indicating that the participants of both groups did not allocate more attention to fearful stimuli compared to the neutral ones. These latter results should, however, be considered with caution, given that the EPN results are close to the statistical significance. Further studies are needed to clarify the possible link between the early perceptual deficit and the embodiment alteration in alexithymia.

Experiments 1 and *5*, aimed to explore the embodiment of fear in the somato-sensory system. To this end, the Visual Remapping of Touch (VRT) paradigm has been used, in which seeing a face being touched improves detection of near-threshold tactile

stimulation concurrently delivered to one's own face. Participants' behavioral performance at the VRT task while they saw fearful, a control emotion (i.e. happiness in *Experiment 1* and disgust in *Experiment 5*, see further discussion) and neutral faces were compared. Results of both *Experiment 1* and *5* revealed that the VRT effect in HA subjects was not influenced by the fearful content of the observed faces. On the contrary, in LA participants, the fear conveyed by faces produced an enhancement of the VRT effect, confirming previous data on normal populations (Cardini et al. 2012). Thus, the fear related information seems not to be remapped on the somato-sensory system of HA participants. The robustness of this result is also emphasized by the negative correlation between the VRT index for fear and the difficulty in identifying feelings in *Experiment 1*, which further corroborates the hypothesis that the lack of the VRT effect in HA subjects is due to an affective dimension of alexithymia. The results of these experiments provided clear and consistent evidence that alexithymia is associated with difficulties in remapping /embodying the emotion of fear onto the observer's own somato-sensory system.

To investigate whether the embodiment deficit for fear in HA is circumscribed to their inability to re-experience the emotion of fear in their own somato-sensory system only or, on the contrary, are expanded to the sensory motor system, the motor aspects of the embodiment of fear have been explored in Chapter 4. To the aim, the degree of which participants produced rapid facial reactions (RFRs) in *Corrugator supercilii* and *Zygomaticus Major* muscles congruent with the facial expressions displayed in observed faces, i.e. fear and happiness respectively, has been measured in *Experiment 3*. The findings demonstrated the absence of congruent RFRs in response to fearful stimuli in HA but not in LA participants, highlighting a deficit of HA subjects in the production

of emotional motor responses to implicitly processed emotional facial displays, i.e. a deficit in embodiment process at the somato-motor level. These results supported the hypothesis of a widespread deficit in the embodiment of fear in HA participants. Similarly to *Experiment 1*, the results of RFRs for fear strongly correlates with the participants' difficulty in identifying feelings, an affective dimension of alexithymia. In addition, in order to understand if this emotional motor deficit of HA subjects is circumscribed to the social/communicative domain or is related to a more general domain of self-experience of emotion, in *Experiment 4* a non-communicative emotional movement, namely a visceral (cardiovascular) response, i.e. the instantaneous variations of heart rate (HR), was investigated, in response to fearful, happy and neutral faces. Results indicate that the fearful stimuli did not influence the physiological arousal in HA participants, as revealed by the absence of the fear related bradycardia, which was instead clearly present in LA. Thus, since both the communicative (RFRs) and non-communicative (HF) motor reactions to fearful stimuli are defective in HA, it is possible to conclude that HA are impaired in the general self-experience of the emotion of fear, involving the whole somato-motor system.

Taken together, the results of *Experiment 1, 5, 3 and 4* showed that HA subjects clearly experience an impairment in the embodiment of the fearful information, coupled with a deficit in the fear associated visceral responses. These results suggest that HA difficulties in understanding other's fear are strictly related to a deficit in the embodiment of the fear related information conveyed by faces. In other words, these HA difficulties are associated to their inability to manifest the emotion related somato-motor and visceral fear related responses.

Summarizing, the main result of the current dissertation refers to the widely reported deficit in the embodiment of the emotion of fear at the level of the somato-sensory and sensory motor system in HA, coupled with an absence of fear induced visceral changes and with a disruption of the early perceptual encoding of fearful faces. Importantly, these results are linked to the individual's difficulty in identify feelings, as measured by the DIF TAS-20 subscale. Theories of embodiment of emotions suggest that this "re-experience" of the emotions of others in one's own body elicits a corresponding emotional state in the observer (Gallese, 2007; Goldman and Sripada, 2005). This felt emotional state helps the observer in emotional understanding and in the implementation of appropriate behaviours. However, when the embodiment of emotion is blocked or altered, the emotions' understanding is affected (Niedenthal et al. 2001). The results of the current dissertation consistently revealed an alteration in the embodiment of multiple aspects of fear in HA participants, thus preventing these subjects from normally experiencing emotions.

8.4 A unifying interpretation: the amygdalar dysfunction

The results of the Experiments conducted in the current dissertation provided convergent evidences that the pattern of fear induced changes in the perceptual encoding, in the somato-sensory system and in the somato-motor system (both the communicative and the non communicative ones) is widely and consistently altered in HA participants. This support the hypothesis of a diminished response to fearful stimuli in HA participants compared to the LA.

As already suggested in the previous Chapters, the consistency and the strength of these results might be explained by the amygdala hypo-functioning in alexithymia,

which has been showed in HA by previous research in response to negative stimuli (Kano et al. 2003; Kugel et al. 2008; Reker et al. 2010; Heinzl et al. 2010) as a consistent datum across studies (van der Velde et al. 2013). Indeed, the amygdala plays a pivotal role in the early stage of facial expression processing (Calder et al. 2001; Zald, 2003; Liddell et al. 2005; LeDoux 2012), and is known to be involved in emotional processing (LeDoux, 2014; Adolphs, 2013), in particular of fearful information (Fusar-Poli et al. 2009; Vytal and Hamann, 2010).

To test the hypothesis of the possible role of the amygdala on the somato-sensory system, i.e. in the embodiment abilities of the somatic aspect of fear, in *Experiment 7* the performance at the VRT paradigm of participants with amygdalar lesions were compared with those of two different groups of controls, namely patients with extratemporal lesions and healthy participants. The results of *Experiment 7* revealed that patients with amygdalar lesions manifested a disruption of the fear specific enhancement of the VRT effect, contrarily to both the control groups, in which the normal VRT effect for fear is detected. The similarities of the behavioral findings on fear related VRT effect on the somato-sensory system of alexithymic participants and patients with amygdalar lesions support the hypothesis that the fear specific difficulties repeatedly observed in the current dissertation in HA participants might be explained by an amygdalar hypo-functioning in response to fearful stimuli in alexithymia.

According to LeDoux (1996), the information about fear reached the amygdala through two different routes. The first one bypasses conscious processing and allows for immediate reactions to fearful stimuli (Tamietto and de Gelder, 2010, LeDoux et al. 1996; Garrido et al. 2012) and consist of a direct projection from the thalamus to the amygdala. This route is also known as the “low road” for fear responses (LeDoux, 1996,

Day-Brown et al. 2010; Garrido et al. 2012; Morris et al. 1999), and allows a crude but very fast processing of fearful information. The alternative route is known as the “high road” pathway, which routes the incoming information through sensory cortex, allowing for fear processing that, while slower, is conscious, more detailed and integrated with higher level cognitive processes (LeDoux et al. 1996; Garrido et al. 2012).

Previous investigations of the functional processing of emotional information (although not specifically for fear) in alexithymia revealed an hypo-activation of the amygdala even if the emotional stimuli were presented below the level of consciousness (Reker et al. 2010; Kugel et al. 2008). Since the stimuli did not reach the consciousness, these studies indirectly suggested that the “low road” of emotional processing might be altered in alexithymia. Critically, these studies also reported that alexithymic’s explicit rating of valence did not differ from the rating of the control group (Reker et al. 2010; Kugel et al. 2008), thus suggesting that when the emotional content of the stimuli is consciously perceived and cognitively processed to be explicitly evaluated, alexithymic’s performance is normal. These results indirectly seem to suggest that the “high road” might be spared and functional in alexithymia.

Critically, in the current dissertation, all the experiments adopted an indirect task, in which the stimuli were consciously perceived but the participant’s attention was deliberately diverted from the emotional content of the stimuli. In each experiment, participants were asked to explicitly rate the arousal and valence of the stimuli after completing the implicit task. The results of the experiment were concordant in the finding of an alteration of HA responses in the implicit tasks, as supported by the electroencephalographic, autonomic and embodiment measurements. On the contrary,

in the explicit rating of arousal and valence, which required the emotions to be cognitively processed to be carried out, no differences between groups emerged.

This consistent dissociation between indirect measures (which are consistently impaired in alexithymia) and explicit measures (which are consistently spared in alexithymia) are of greater interest, since it seems to suggest a possible alteration of the “low road” to the amygdale for the fear processing, coupled with a spared “high road” functionality. Indeed, when the attention is diverted from the emotional content of the stimuli, this is processed non-consciously and in a fast and crude way. This hypothesis fully explains the results obtained in the current thesis on the emotion of fear.

However, between-groups differences in the somato-sensory system activity in response to disgusted faces emerged as well. Please refer to paragraph 8.2 for details. Although the processing of the emotion of disgust is known to mainly rely on insular activation (Phillips et al., 1997; Wicker et al., 2003), recent studies revealed that the amygdala is involved in the processing of disgust as well (Fusar-Poli et al. 2009; Vytal and Hamann, 2010 for meta-analysis of imaging data; Sarinopoulos et al. 2006). Interestingly, amygdala and insula seem to be part of the same brain network (Bebko G et al. 2015; Denny et al. 2014), whose activity is modulated by personality trait (Baur et al. 2013). Thus, it could be hypothesized that the absence of the distant influence of the amygdala activity on the insula functionality could result in an insular hyper-activation, which in turn affect participants somato-sensory reactivity to disgusted stimuli.

Finally, the present results on HA also found a slight difficulty in the perceptual encoding and somato-motor features of happiness. Please refer to paragraph 8.1 for details. These results are explained by the hypothesis of the hypo-functioning of the “low road” to the amygdala during the processing of emotions, too. Indeed, the

amygdala is also involved in positive emotions processing, although to a lesser extent compared to fear (Fusar-Poli et al. 2009; Vytal and Hamann, 2010), through direct projection to the nucleus accumbens in the ventral striatum, which is involved in positive affects (McDonald, 1991, 1992).

Summarizing, the hypothesis of the hypo-functioning of the “low road” to amygdala in alexithymia during emotional processing could account for the results of all the three different emotions. Indeed the amygdala is critical for the processing of fearful information, and its functional alteration fully explains the consistent and robust results presented in the current thesis. However, the amygdala is also known to be involved in other emotions processing, and this could account for the current results on disgust and happiness.

8.5 Final consideration about the term “Alexithymia”

Considering the studies reviewed in Chapter 2, paragraph 2.4, and the results presented in the current thesis, it is evident that the term “*Alexithymia*” can no longer be considered adequate to definite this construct in evolution. Indeed, the difficulty in the verbal expression of their own emotions is not the core problem of alexithymic individuals. Recent literature on alexithymia and the experiments presented in the current thesis provided evidences that this personality trait seems to be related to an early (*Experiment 8*, perceptual encoding) emotional processing deficit, to an alteration of physiological signals (*Experiment 4*, non communicative motor reactions) and to an embodiment deficit (*Experiments 1,3,5*, somato-sensory and communicative sensory-motor system) which prevent these subjects to correctly perceive emotions.

Thus, the definition “alexithymia” has two main limitations:

- 1) The prefix “a-” is privative, indicating an absence of something, in this particular case of words (lexis) for emotions (thimos). It is accepted worldwide that alexithymia, which is a subclinical phenomenon and a personality trait, and not a psychiatric disorder (Taylor et al. 1991, 2000), is not characterized by an ABSENCE of such abilities, but only by a difficulty. According to that, the use of the privative prefix has to be considered inappropriate.
- 2) The term “*Alexithymia*” only refers to the difficulties in the verbal expression of emotions, whereas alexithymia is characterized by a marked difficulty in earlier stages of emotion processing, as already stated. It could be assumed that these early difficulties in emotional processing contribute to later labeling troubles (Pollatos and Gramann, 2011). Thus, the term “*alexithymia*” no longer encapsulates the essence of this subclinical phenomenon, since the problem in emotional labeling is not the only or the major problem of these individuals.

Here, a new term is proposed, which aims to cope with the above reported limitation of the term “Alexithymia”. In order to maintain the Greek etymology of the term, the new term proposed is “*DISEPONETIMIA*”, from the Greek *dis* ($\delta\iota\varsigma$ = altered), *ekponeo* ($\epsilon\kappa\pi\omicron\nu\epsilon\omega$ = achieve) and *thimos* ($\theta\upsilon\mu\omicron\sigma$ = emotion), i.e. altered realization, achievement of emotions. This new term, using the prefix *dis-* instead of *a-*, would focalize the attention on the difficulties rather than the absence of an ability. Moreover, it would be related not only to the emotional labeling, but instead to all the processes involved in emotion perception, creation and recognition.

8.6 Future directions

The current data suggest a pervasive deficit in the embodiment of the emotion of fear in alexithymia. Although the results of *Experiments 1,3,4,5 and 8* provided convergent and clear results supporting this conclusion, future studies are needed to investigate more deeply if the observed difficulties are also present using other physiological indices, for instance skin conductance. Moreover, it will be interesting to investigate how the embodiment of these deficits are reflected in the daily life or in more ecological situations. Furthermore, as suggested in paragraph 8.4, the difficulties specific for fear are likely to be related to an amygdalar hypo-functioning in response to fear in these participants. An alteration in the function of the amygdala has already been showed in alexithymia (see [van der Velde et al. 2013](#) for a meta-analysis), but studies investigating the structural morphology of the amygdala are still missing. It would be interesting to understand if alexithymic's amygdalae are normal in structure but not in function or if, otherwise, their amygdalae are structurally abnormal. A possible *a-priori* hypothesis might be that in alexithymia the amygdala is smaller compared to LA, particularly in the central nucleus. Indeed, the central nucleus of the amygdala is an important output nucleus which has direct projection to a variety of anatomical areas that might be expected to be involved in many of the somatic and autonomic signs of fear ([Davis, Whalen, 2001](#); [LeDoux, 2012](#); [LeDoux et al. 1988](#)).

The current results about the heightened embodiment of disgust, although preliminary, are interesting as well. Future studies are needed to deeper investigate the embodiment of disgust in alexithymia and the alexithymic visceral and autonomic reaction to disgusted stimuli using different paradigms. Moreover, it will be interesting

to investigate whether the reported heightened embodiment of disgust might be related to an increased risk to developing the psychopathologies of the disgust system in alexithymia. Finally, as suggested in chapter 5, the enhancement of the VRT effect for disgusted faces might be due to an abnormally high insular activation in response to disgust, which, in turn, influences the activity of somatosensory cortices, on which the VRT effect relies. An alteration in the function of insula has been shown in alexithymia, providing contrasting results (please see [Karlsson et al. 2008](#); [Heinzel et al. 2010](#); [Frewen et al. 2008](#); [Ernst et al. 2014](#) for insular hyperactivation, but [Reker et al. 2010](#); [van der Velde et al. 2013](#); [Baird et al. 2010](#) for insular hypoactivation). As for the amygdala, studies investigating the structural morphology of the insula are still missing. It would be interesting to understand if alexithymic's insulae are normal in structure but not in function or if, otherwise, their insulae are structurally abnormal. A possible *a-priori* hypothesis might be that in alexithymia the anterior insula, which is the brain region responsible for the conscious emotions ([Gu et al. 2013](#); [Craig, 2011](#) for recent reviews) and empathy ([Engen and Singer, 2013](#); [Bernhardt and Singer, 2012](#) for recent reviews), is smaller than those of LA participants, in accordance with alexithymia being defined as difficulty in conscious experience of emotions ([Taylor et al. 1991](#)). On the contrary, the posterior insula, in which physical features of interoception are processed ([Craig 2009](#)) might be bigger in alexithymia, thus accounting for the results of *Experiment 6*.

Finally, future studies are also needed to confirm the conclusion of the current thesis, i.e. that alexithymic individuals exhibit a deficit in emotional embodiment which is specific for fear. Indeed, the current results regarding the abnormally high embodiment of disgust needs further support. Moreover, the emotions of anger and

sadness were not investigated in the current thesis. The reason is that anger is not a imitative but a reactive emotion (i.e. the vision of an anger face does not induce anger in the observer, but fear), and that sadness is considered to be a less arousing emotion compared to the other negative emotions. However, it is not possible to conclude with certainty that alexithymia is a specific problem in dealing with fear until both anger and sadness have been specifically tested.

References

- Achenbach, T.M., Becker, A., Döpfner, M., Heiervang, E., Roessner, V., Steinhausen, H., and Rothenberger, A. (2008). Multicultural assessment of child and adolescent psychopathology with ASEBA and SDQ instruments: research findings, applications, and future directions. *Journal of Child Psychology and Psychiatry*. 49(3): 251–275.
- Adolphs, R., Tranel, D., Damasio, H. and Damasio, A. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature*, 372(6507), 669-72.
- Adolphs, R., Tranel, D., Damasio, H. and Damasio, A.R. (1995). Fear and the human amygdala. *The Journal of Neuroscience*, 15(9), 5879-91.
- Adolphs, R., Damasio, H., Tranel, D. and Damasio, A.R. (1996). Cortical system for the recognition of emotion in facial expressions. *Journal of Neuroscience*. 16(23); 7678-7687.
- Adolphs, R., Damasio, H., Tranel, D., Cooper, G. and Damasio, A.R. (2000). A role for somatosensory cortices in the visual recognition of emotion as revealed by three-dimensional lesion mapping. *Journal of Neuroscience*. 20(7); 2683-2690.
- Adolphs, R. (2002a). Recognizing emotion from facial expressions: psychological and neurological mechanisms. *Behavioral and Cognitive Neuroscience Reviews*. 1(1); 21-62.
- Adolphs, R. (2002b). Neural system for recognizing emotion. *Current Opinion in Neurobiology*. 12; 21-62.
- Adolphs, R., Baron-Cohen, S. and Tranel, D. (2002). Impaired recognition of social emotions following amygdala damage. *Journal of Cognitive Neuroscience*. 14(8), 1264-74.
- Adolphs, R. (2013). The biology of fear. *Current Biology*, 23(2):R79-93.
- Aftanas, L.I., Varlamov, A.A., Reva, N.V. and Pavlov, S.V. (2003). Disruption of early event-related theta synchronization of human EEG in alexithymics viewing affective pictures. *Neuroscience Letters*. 340(1); 57-60.
- Aftanas, L.I. and Varlamov, A.A. (2007). Effects of alexithymia on the activity of the anterior and posterior areas of the cortex of the right hemisphere in positive and negative emotional activation. *Neuroscience Behavioral Physiology*. 37(1); 67-73.

- Aleman, A. (2005). Feelings you can't imagine: towards a cognitive neuroscience of alexithymia. *Trends in Cognitive Science*. 9(12):553-5.
- Allen, L.A., Gara, M.A., Escobar, J.I., Waitzkin, H., and Silver, R.C. (2001). Somatization: a debilitating syndrome in primary care. *Psychosomatics*. 42(1):63-7.
- Allen, L.B., Qianm L., Tsaom, J.C., Hayes, L.P. and Zeltzer, L.K. (2011). Depression partially mediates the relationship between alexithymia and somatization in a sample of healthy children. *Journal of Health Psychology*. 16(8):1177-86.
- Alvarez, G. and Fuentes, P. (1994). Source Recognition of facial expression in diverging socioeconomic levels. *Brain and Cognition*. 25(2):235-9.
- Amaral, D.G., Price, J.L., Pitkanen, A. and Carmichael, S.T. (1992). Anatomical organization of the primate amygdaloid complex, in J.P. Aggleton, (ed), *The Amygdala: Neurobiological Aspects of Emotion, Memory, and Mental Dysfunction* (pp. 1-66), New York: Wiley-Liss.
- Amaral, D.G. and Price, J.L. (1984). Amygdalo-cortical projections in the monkey (*Macaca fascicularis*). *Journal of Comprehensive Neurology*, 230(4), 465-96.
- Amorapanth, P., LeDoux, J.E. and Nader, K. (2000). Different lateral amygdala outputs mediate reactions and actions elicited by a fear-arousing stimulus. *Nature Neuroscience*.3(1):74-9.
- Amunts, K., Kedo, O., Kindler, M., Pieperhoff, P., Mohlberg, H., Shah, N.J., Habel, U., Schneider, F. and Zilles, K. (2005). Cytoarchitectonic mapping of the human amygdala, hippocampal region and entorhinal cortex: intersubject variability and probability maps. *Anatomy Embryology (Berl)*. 210(5-6):343-52.
- Anderson, A.K., Christoff, K., Panitz, D., De Rosa, E. and Gabrieli, J.D. (2003). Neural correlates of the automatic processing of threat facial signals. *Journal of Neuroscience*, 23(13), 5627-33.
- Anderson, A.K., Spencer, D.D., Fulbright, R.K. and Phelps, E.A. (2000). Contribution of the anteromedial temporal lobes to the evaluation of facial emotion. *Neuropsychology*, 2000, 14(4), 526-36.
- Ashley, V., Vuilleumier, P., and Swick, D. (2004). Time course and specificity of event-related potentials to emotional expressions. *Neuroreport*. 15(1):211-6.

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

Bach, M., de Zwaan, M., Ackard, D., Nutzinger, D.O. and Mitchell, J.E. (1994). Alexithymia: relationship to personality disorders. *Comprehensive Psychiatry*. 35;239-243.

Bagby, M.R., Quilty, L.C., Taylor, G.J., Grabe, H.J., Luminet, O., Verissimo, R., De Grootte, I., and Vanheule, S. (2009). Are there subtypes of alexithymia? *Personality and Individual Differences*. 47: 413–418

Balconi, M., Vanutelli, M.E., and Finocchiaro, R. (2014). Multilevel analysis of facial expressions of emotion and script: self-report (arousal and valence) and psychophysiological correlates. *Behavioral Brain Function*. 10(1):32.

Bastiaansen, J.A., Thioux, M. and Keysers, C. (2009). Evidence for mirror systems in emotions. *Philosophical Transaction of the Royal Society London B Biological Science*. 364(1528):2391-404.

Bates, E., Wilson, S.M., Saygin, A.P., Dick, F., Sereno, M.I., Knight, R.T. and Dronkers, N.F. (2003). Voxel-based lesion-symptom mapping. *Nature Neuroscience*, 6, 448-450.

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

Baur, V., Hänggi, J., Langer, N., and Jäncke, L. (2013). Resting-state functional and structural connectivity within an insula-amygdala route specifically index state and trait anxiety. *Biological Psychiatry*. 73(1):85-92.

Bavelas, J. B., Black, A., Lemery, C. R., and Mullett, J. (1986). "I show how you feel": Motor mimicry as a communicative act. *Journal of personality and social psychology*. 50(2): 322-329.

Beadle, J.N., Paradiso, S., Salerno, A., and McCormick, L.M. (2013). Alexithymia, emotional empathy, and self-regulation in anorexia nervosa. *Annals of Clinical Psychiatry*. 25(2):107-20.

Beaumont, W. (1933). Experiments and observations on the gastric juice and the physiology of digestion. Plattsburg, FP Allen.

Bebko, G., Bertocci, M., Chase, H., Dwojak, A., Bonar, L., Almeida, J., Perlman, S.B. Versace, A., Schirda, C., Travis, M., Gill, M.K., Demeter, C., Diwadkar, V., Sunshine, J., Holland, S., Kowatch, R., Birmaher, B., Axelson, D., Horwitz, S., Frazier, T., Arnold, L.E., Fristad, M., Youngstrom, E., Findling, R., and Phillips. M.L. (2015). Decreased amygdala-insula resting state connectivity in behaviorally and emotionally dysregulated youth. *Psychiatry Research*. 2015. 231(1):77-86.

Becerra, R., Amos, A., and Jongenelis, S. (2002). Organic alexithymia: a study of acquired emotional blindness. *Brain Injury*. 16(7):633-45.

Bechara, A., Tranel, D., Damasio, H., Adolphs, R., Rockland, C. and Damasio, A.R. (1995). Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science*. 269: 1115-1118.

Beck, A.T., Steer, R.A. and Brown, G.K. (1996). *Manual for the Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.

Bentin, S., Allison, T., Puce, A., Perez, E., and McCarthy, G. (1996). Electrophysiological studies of face perception in humans. *Journal of Cognitive Neuroscience*. 8, 551–565.

Bentin, S., and Deouell, L. Y. (2000). Structural encoding and identification in face processing: ERP evidence for separate mechanisms. *Cognitive Neuropsychology*. 17, 35–55.

Bermond, B. (1997). Brain and alexithymia. In A. Vingerhoets, F. van Bussel, & J. Boelhouwer (Eds.), *The (non)expression of emotions in health and disease* (pp. 115–129). Tilburg: Tilburg University Press.

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

Bernhardt, B.C., and Singer, T. (2012). The neural basis of empathy. *Annual Review Neuroscience*. 35:1-23.

Bender, M.B. (1952). *Disorders of perception*. Springfield, IL: Charles C. Thomas.

Bird, G., Silani, G., Brindley, R., White, S., Frith, U., and Singer, T. (2010). Empathic brain responses in insula are modulated by levels of alexithymia but not autism. *Brain*. 133(Pt 5):1515-25.

Blakemore, S.J., Bristow, D., Bird, G., Frith, C. and Ward, J. (2005). Somatosensory activations during the observation of touch and a case of vision-touch synaesthesia. *Brain*. 128(Pt7); 1571-1583.

Blau, V.C., Maurer, U., Tottenham, N., and McCandliss, B.D. (2007). The face-specific N170 component is modulated by emotional facial expression. *Behavioral and Brain Function*. 2007; 3: 7.

Bodini, B., Mandarelli, G., Tomassini, V., Tarsitani, L., Pestalozza, I., Gasperini, C., Lenzi, G.L., Pancheri, P., and Pozzilli, C. (2008). Alexithymia in multiple sclerosis: relationship with fatigue and depression. *Acta Neurologica Scandinavica*. 118(1):18-23.

Bogdanov, V.B., Bogdanova, O.V., Gorlov, D.S., Gorgo, Y.P., Dirckx, J.J., Makarchuk, M.Y., Schoenen, J., and Critchley, H. (2013). Alexithymia and empathy predict changes in autonomic arousal during affective stimulation. *Cognitive & Behavioral Neurology*. 26(3):121-32.

Bogousslavsky, J. (2005). Guillaume Apollinaire, the lover assassinated. In Bogousslavsky, J. e Boller, F. (eds) *Neurological Disorders in Famous Artists. Frontiers of Neurology and Neuroscience*. 19, 1-8.

Borsci, G., Boccardi, M., Rossi, R., Rossi, G., Perez, J., Bonetti, M., and Frisoni, G.B. (2009). Alexithymia in healthy women: a brain morphology study. *Journal of Affective Disorders*. 114(1-3):208-15.

Breiter, H.C., Etcoff, N.L., Whalen, P.J., Kennedy, W.A., Rauch, S.L., Buckner, R.L., Strauss, M.M., Hyman, S.E. and Rosen, B.R. (1996). Response to habituation of the human amygdale during visual processing of facial expression. *Neuron*. 17, 875-887.

Brunner, E. and Munzel, U. (2000). The nonparametric behrens-fisher problem: asymptotic theory and a small-sample approximation. *Biometrical Journal*, 42, 17-25.

Bydlowski, S., Corcos, M., Jeammet, P., Paterniti, S., Berthoz, S., Laurier, C., Chambry, J., and Consoli, S.M. (2005). Emotion-processing deficits in eating disorders. *International Journal of Eating Disorder*. 37(4):321-9.

Cacioppo, J.T., Petty, R.E., Losch, M.E. and Kim, H.S. (1986). Electromyographic activity over facial muscle regions can differentiate the valence and

intensity of affective reactions. *Journal of Personality and Social Psychology*. 50(2):260-8.

Caharel, S., Courtay, N., Bernard, C., Lalonde, R., and Rebaï, M. (2005). Familiarity and emotional expression influence an early stage of face processing: an electrophysiological study. *Brain & Cognition*. 59(1):96-100.

Calder, A.J., Lawrence, A.D. and Young, A.W. (2001). Neuropsychology of fear and loathing. *Nature Review Neuroscience*. 2(5):352-63.

Calvo, M. G., and 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 emotion: A critical examination and an alternative theory. *American Journal of Psychology*. 59, 106-124.

Cardini, F., Costantini, M., Galati, G., Romani, G.L., Ladavas, E. and Serino, A. (2011). Viewing one's own face being touched modulates tactile perception: an fMRI study. *Journal of Cognitive Neuroscience*. 23(3):503-513.

Cardini, F., Bertini, C., Serino, A. and Ladavas, E. (2012). Emotional Modulation of Visual Remapping of Touch. *Emotion*. 12(5); 980-7.

Carlson, J.M., and Reinke, .K.S. (2010). Spatial attention-related modulation of the N170 by backward masked fearful faces. *Brain & Cognition*. 73(1):20-7.

Caretti, V., Porcelli, P., Solano, L., Schimmenti, A., Bagby, R.M., and Taylor, G.J. (2011). Reliability and validity of the Toronto Structured Interview for Alexithymia in a mixed clinical and nonclinical sample from Italy. *Psychiatry Research*. 187(3):432-6.

Carr, L., Iacoboni, M., Dubeau, M.C., Mazziotta, J.C. and Lenzi, G.L. (2003). Neural mechanisms of empathy in humans: a relay from neural system for imitation to limbic areas. *Proceedings of the National Academy of Sciences*. 100(9): 5497-5502.

Cattaneo, L., and Rizzolatti, G. (2009). The mirror neuron system. *Archives of Neurology*. 66(5):557-60.

Cattaneo, L., and Pavesi, G. (2014). The facial motor system. *Neuroscience and Biobehavioral Review*. 38:135-59.

Cedro, A., Kokoszka, A., Popiel, A., and Narkiewicz-Jodko, W. (2001). Alexithymia in schizophrenia: an exploratory study. *Psychological Report*. 89(1):95-8.

Chapman, H.A. and Anderson, A.K. (2012). Understanding disgust. *Annals New York Academy Science*. 1251: 62-76.

Chapman, W.P., Schroeder, H.R., Geyer, G., Brazier, M.A., Fager, C., Poppen, J.L., Solomon, H.C. and Yakovlev, P.I. (1954). Physiological evidence concerning importance of the amygdaloid nuclear region in the integration of circulatory function and emotion in man. *Science*. 120(3127):949-50.

Chapman, W.P., Schroeder, H.R., Guyer, G., Brazier, M.A.B., Fager, C. and Poppen, J.L.(1954). Physiological evidence concerning the importance of the amygdaloid nuclear region in the integration of circulating function and emotion in man. *Science*. 129: 949-950.

Chartrand, T.L. and Bargh, J.A. (1999). The chameleon effect: the perception-behavior link and social interaction. *Journal of Personality and Social Psychology*. 76(6):893-910.

Ciamarelli, E., Sperotto, R., Mattioli, F. & di Pellegrino, G. (2013). Damage to the ventromedial prefrontal cortex reduces interpersonal disgust. *Social, Cognitive & Affective Neuroscience*. 8: 171-180.

Cohen, J. (1969). *Statistical power analysis for the behavioral sciences*. San Diego, CA: Academic Press.

Colombo, L., Sartori, G. and Brivio, C. (2002). Stima del quoziente intelletivo tramite l'applicazione del TIB (Test di intelligenza Breve). *Giornale Italiano di Psicologia*. 3:613-637

Craig, A.D. (2002). How do you feel? Interoception: the sense of the physiological condition of the body. *Nature Review Neuroscience*. 3(8):655-66. Review.

Craig, A.D. (2003). Interoception: the sense of the physiological condition of the body. *Current Opinion in Neurobiology*. 13(4):500-5. Review.

Craig, A.D. (2004). Human feelings: why are some more aware than others? *Trends in Cognitive Sciences*. 8(6):239-41. Review.

Craig, A.D. (2009). How do you feel--now? The anterior insula and human awareness. *Nature Review Neuroscience*. 10(1): 59-70

Craig, A.D. (2011). Significance of the insula for the evolution of human awareness of feelings from the body. *Annals of the New York Academy of Science*. 1225:72-82. Review.

Crawford, J.R. and Henry, J.D. (2004). The positive and negative affect schedule (PANAS): construct validity, measurement properties and normative data in a large non-clinical sample. *British Journal of Clinical Psychology*. 43(Pt 3):245-65.

Critchley, H.D., Wiens, S., Rotshtein, P., Ohman, A., and Dolan, R.J. (2004). Neural systems supporting interoceptive awareness. *Nature Neuroscience*. 7(2):189-95.

Critchley, H.D., and Harrison, N.A. (2013). Visceral influences on brain and behavior. *Neuron*. 77(4):624-38.

Curtis, V. (2011). Why disgust matters. *Philosophical Transactions of the Royal Society*. 366; 3478-3490.

Dahlenburg, B. and Spitzer, C. (2005). Major depression and stroke in Caspar David Friedrich. In Bogousslavsky, J. e Boller, F. (eds) Neurological Disorders in Famous Artists. *Frontiers of Neurology and Neuroscience*. 19, 112-120.

Damasio, A.R. (1994). *Descartes' Error: emotion, rationality and the human brain*. New York: Grosset/Putnam.

Davey, G.C., and Bond, N. (2006). Using controlled comparisons in disgust psychopathology research: the case of disgust, hypochondriasis and health anxiety. *Journal of Behavioral Therapy Experimental Psychiatry*. 37(1):4-15.

Davey, G.C. (2011). Disgust: the disease-avoidance emotion and its dysfunctions. *Philosophical Transactions of the Royal Society B: Biological Society*. 366(1583):3453-65.

Davis, M.H. (1983). Measuring individual differences in empathy: evidence from a multidimensional approach. *Journal of Personality and Social Psychology*.44:113-126.

Davis, M. (1994). The role of the amygdala in emotional learning. *International Review Neurobiology*, 36, 225-66.

Davis, M. and Whalen, P.J. (2001). The amygdale: vigilance and emotion. *Molecular Psychiatry*. 6: 13-34.

Davis, M. and Shi, C. (2000). The amygdala. *Current Biology*. 10(4): R131.

Day-Brown, J.D., Wei, H., Chomsung, R.D., Petry, H.M., and Bickford, M.E. (2010). Pulvinar projections to the striatum and amygdala in the tree shrew. *Frontiers in Neuroanatomy*. 4:143.

de Vente, W., Kamphuis, J.H., and Emmelkamp, P.M. (2006). Alexithymia, risk factor or consequence of work-related stress? *Psychotherapy Psychosomatics*. 2006;75(5):304-11.

Deborde, A.S., Berthoz, S., Godart, N., Perdereau, F., Corcos, M., and Jeammet, P. (2006). Relations between alexithymia and anhedonia: a study in eating disordered and control subjects. *Encephale*. 32(1 Pt 1):83-91.

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

Demartini, B., Petrochilos, P., Ricciardi, L., Price, G., Edwards, M.J., and Joyce, E. (2014). The role of alexithymia in the development of functional motor symptoms (conversion disorder). *Journal of Neurology Neurosurgery & Psychiatry*. 85(10):1132-7.

Denny, B.T., Fan, J., Liu, X., Guerreri, S., Mayson, S.J., Rinsky, L., New, A.S., Siever, L.J., and Koenigsberg, H.W. (2014). Insula-amygdala functional connectivity is correlated with habituation to repeated negative images. *Social Cognitive & Affective Neuroscience*. 9(11):1660-7.

Derogatis, L.R. (1994). *Symptom Checklist-90-R: Administration, scoring, and procedures manual (3rd ed.)*. Minneapolis, MN: National Computer Systems.

De Saint-Exupéry, A. (1943). *The Little Prince*. *Wordsworth Classics Editions*.

Dimberg, U. (1982). Facial reactions to facial expressions. *Psychophysiology*. 19(6):643-7.

Dimberg, U. and Thunberg, M. (1998). Rapid facial reactions to emotional facial expressions. *Scandinavian Journal of Psychology*. 39; 39-45.

Dimberg, U., Thunberg, M. and Elmehed, K. (2000). Unconscious facial reactions to emotional facial expressions. *Psychological Science*. 11(1): 86-89.

Dimberg, U. and Petterson, M. (2000). Facial reactions to happy and angry facial expressions: evidence for right hemisphere dominance. *Psychophysiology*. 37(5):693-6.

Dimberg, U., Thunberg, M. and Grunedal, S. (2002). Facial reactions to emotional stimuli: automatically controlled emotional responses. *Cognition and Emotion*. 16 (4), 449–471.

Dunn, B. D., Dalgleish, T., Ogilvie, A. D., & Lawrence, A. D. (2007). Heartbeat perception in depression. *Behaviour Research and Therapy*. 45, 1921–1930.

Ebisch, S.J., Perrucci, M.G., Ferretti, A., Del Gratta, C., Romani, G.L. and Gallese, V. (2008). The sense of touch: embodied simulation in a visuotactile mirroring mechanism for observed animate or inanimate touch. *Journal of Cognitive Neuroscience*. 20(9); 1611-1623.

Eckman, P. and Friesen, W.V. (1976). Pictures of facial affect. Consulting Psychologists Press, Palo Alto, CA.

Ekman, P., Levenson, R.W., and Friesen, W.V. (1983). Autonomic nervous system activity distinguishes among emotions. *Science*. 221(4616):1208-10.

Eimer, M., and Holmes, A. (2002). An ERP study on the time course of emotional face processing. *NeuroReport*. 13, 427–431.

Eimer, M. and Holmes, A. (2007). Event-related brain potential correlates of emotional face processing. *Neuropsychologia*. 45, 15-31.

Eimer, M., Holmes, A., and McGlone, F.P. (2003). The role of spatial attention in the processing of facial expression: an ERP study of rapid brain responses to six basic emotions. *Cognitive Affective and Behavioral Neuroscience*. 3(2):97-110.

Engen, H.G., and Singer, T. (2013). Empathy circuits. *Current Opinion in Neurobiology*. 23(2):275-82. Review.

Ernst, H., Key, J.D., and Koval, M.S. (1999). Alexithymia in an adolescent with agenesis of the corpus callosum and chronic pain. *Journal of American Academy of Child and Adolescent Psychiatry*. 38(10):1212-3.

Ernst, J., Boker, H., Hattenschwiler, J., Schupbach, D., Northoff, G., Seifritz, E. & Grimm, S. (2013). The association of interoceptive awareness and alexithymia with neurotransmitter concentrations in insula and anterior cingulate. *Social, Cognitive & Affective Neuroscience*. 9(6):857-63.

Falkenberg, I., Bartels, M. and Wild, B. (2008). Keep smiling! Facial reactions to emotional stimuli and their relationship to emotional contagion in patients with

schizophrenia. *European Archives of Psychiatry and Clinical Neuroscience*. 258(4):245-53.

Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39, 175–191.

Fehr, Fred S., and John A. Stern. "Peripheral Psychological Variables and Emotion: The James-Lange Theory Revisited." *Psychological Bulletin* 74 (1970): 411-24.

Feuerriegel, D., Churches, O., Hofmann, J., and Keage, H.A. (2014). The N170 and face perception in psychiatric and neurological disorders: A systematic review. *Clinical Neurophysiology*.

First, M.B., Spitzer, R.L., Gibbon, M. and William, J.B.W. (1997). SCID-I. Structured Clinical Interview for DSM-IV Axis I Disorders. Versione italiana a cura di Mazzi F, Morosini P, De Girolamo G, Lussetti M, Guaraldi GP (2007). *Giunti O.S.*

Fitzgerald, M. and Bellgrove, M.A. (2006). The overlap between alexithymia and Asperger's syndrome. *Journal of Autism and Developmental Disorders*.36(4):573-6.

Froni, F. and Semin, G.R. (2011). When does mimicry affect evaluative judgment? *Emotion*. 11(3):687-90.

Franken, I.H., Van Strien, J.W., and Nijs, I.M. (2006). Effect of hedonic tone on event-related potential measures of cognitive processing. *Psychiatry Research*. 142(2-3):233-9.

Franz, M., Schaefer, R., Schneider, C., Sitte, W., and Bachor, J. (2004). Visual event-related potentials in subjects with alexithymia: modified processing of emotional aversive information? *American Journal of Psychiatry*. 161(4):728-35.

Frewen, P.A., Dozois, D.J., Neufeld R,W. and Lanius, R.A. (2008). Meta-analysis of alexithymia in posttraumatic stress disorder. *Journal of Traumatic Stress*. 21(2): 243-6.

Fridlund, A.J. and Cacioppo, J.T. (1986). Guidelines for human electromyographic research. *Psychophysiology*. 23(5):567-89.

Friedman, D.P., Murray, E.A., O'Neill, J.B. and Mishkin, M. (1986). Cortical connections of the somatosensory fields of the lateral sulcus of macaques: evidence for

a corticolimbic pathway for touch. *Journal of Comprehensive Neurology*. 252(3):323-47.

Friedlander, L., Lumley, M.A., Farchione, T. and Doyal, G. (1997). Testing the alexithymia hypothesis: physiological and subjective responses during relaxation and stress. *Journal Nervous Mental Disorders*. 185(4):233-9.

Frijda, N.H. (2009). Emotion experience and its varieties. *Emotion Review*. 1:264-71.

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

Fukunishi, I., Sei, H., Morita, Y. and Rahe, R.H. (1999). Sympathetic activity in alexithymics with mother's low care. *Journal of Psychosomatic Research*. 46(6):579-89.

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. and Politi, P. (2009a). Functional atlas of emotional faces processing: a voxel-based meta-analysis of 105 functional magnetic resonance imaging studies. *Journal of Psychiatry Neuroscience*. 34(6), 418-32.

Fusar-Poli, P., Placentino, A., Carletti, F., Allen, P., Landi, P., Abbamonte, M., Barale, F., Perez, J., McGuire, P., and Politi, P.L. (2009b). Laterality effect on emotional faces processing: ALE meta-analysis of evidence. *Neuroscience Letters*. 452(3):262-7.

Gallese, V. (2007). Embodied simulation: from mirror neuron systems to interpersonal relations. *Novartis Foundation Symposium*. 278:3-12.

Gallese, V. and Sinigaglia, C. (2011). What is so special about embodied simulation? *Trends in Cognitive Sciences*. 15(11):512-9.

Garfinkel, S.N. & Critchley, H.D. (2013). Interoception, emotion and brain: new insight link internal physiology to social behavior. *Social Cognitive and Affective Neuroscience*. 8:231-234.

Garrido, M.I., Barnes, G.R., Sahani, M., and Dolan, R.J. (2012). Functional evidence for the dual route to amygdale. *Current Biology*. 22(2): 129-34.

Gendron, M., and Barrett, L.F. (2009). Reconstructing the Past: A Century of Ideas About Emotion in Psychology. *Emotion Review*. 1(4):316-339.

Gläscher, J., Rudrauf, D., Colom, R., Paul, L.K., Tranel, D., Damasio, H. and Adolphs, R. (2010). Distributed neural system for general intelligence revealed by lesion mapping. *Proceedings of the National Academy of Sciences*, 107, 4705-4709.

Gloor, P., Olivier, A. and Quesney, L.F. (1981). The role of the amygdala in the expression of psychic phenomena in temporal lobe seizures. In: Ben-Ari Y (ed). *The Amygdaloid Complex*. Elsevier/North Holland: New York, pp 489-507.

Goeleven, E., De Raedt, R., Leyman, L., and 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., and Hooker, C.I. (2013). Distinct associations of insula and cingulate volume with the cognitive and affective dimensions of alexithymia. *Neuropsychologia*. 53:284-92.

Goldman, A.I. and Sripada, C.S. (2005). Simulationist models of face-based emotion recognition. *Cognition*, 94(3), 193-213.

Grabe, H.J., Spitzer, C., and Freyberger, H.J. (2004). Alexithymia and personality in relation to dimensions of psychopathology. *American Journal of Psychiatry*. 161(7):1299-301.

Graham, R., Devinsky, O. and Labar, K.S. (2007). Quantifying deficits in the perception of fear and anger in morphed facial expressions after bilateral amygdala damage. *Neuropsychologia*, 45(1), 42-54.

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

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

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

Gronwall, D.M. (1977) Paced auditory serial-addition task: a measure of recovery from concussion. *Perceptual & Motor Skills*. 44(2): 367-73.

Grynberg, D., Chang, B., Corneille, O., Maurage, P., Vermeulen, N., Berthoz, S. and Luminet, O. (2012). Alexithymia and the processing of emotional facial expressions

(EFEs): systematic review, unanswered questions and further perspectives. *PLoS One*. 7(8):e42429.

Gu, X., Hof, P.R., Friston, K.J., and Fan, J. (2013). Anterior insular cortex and emotional awareness. *Journal of Comparative Neurology*. 521(15):3371-88. Review.

Hagenaars, M.A., Stins, J.F. and Roelofs, K. (2012). Aversive life events enhance human freezing responses. *Journal of Experimental Psychology: General*. 141, 98-105.

Halberstadt, J., Winkielman, P., Niedenthal, P.M., and Dalle, N. (2009). Emotional conception: how embodied emotion concepts guide perception and facial action. *Psychological Sciences*. 20(10):1254-61.

Ham, B.J., Lee, M.S., Lee, Y.M., Kim, M.K., Choi, M.J., Oh, K.S., Jung, H.Y., Lyoo, I.K., and Choi, I.G.(2005). Association between the catechol O-methyltransferase Val108/158Met polymorphism and alexithymia. *Neuropsychobiology*. 52(3):151-4.

Hamann, S.B., Stefanacci, L., Squire, L.R., Adolphs, R., Tranel, D., Damasio, H. and Damasio, A. (1996). Recognizing facial emotion. *Nature*, 379(6565), 497.

Harrison, N.A., Morgan, R. and Critchley, H.D. (2010). From facial mimicry to emotional empathy: a role for norepinephrine? *Social Neuroscience*. 5(4):393-400.

Harrison, N.A., Gray, M.A., Gianaros, P.J., and Critchley, H.D. (2010). The embodiment of emotional feelings in the brain. *The Journal of Neuroscience*.30(38):12878-84.

Harvey, T., Troop, N.A., Treasure, J.L., and Murphy, T. (2002). Fear, disgust, and abnormal eating attitudes: a preliminary study. *International Journal of Eating Disorders*. 32(2):213-8.

Hatfield, E., Cacioppo, J. T. and Rapson, R. (1992). Primitive emotional contagion. In: M. S. Clark (Ed.), *Emotion and social behavior. Review of personality and social psychology*, Vol. 14 (pp. 151–177). Thousand Oaks, CA: Sage Publications, Inc.

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

Haviland, M.G., Hendryx, M.S., Shaw, D.G., and Henry, J.P. (1994). Alexithymia in women and men hospitalized for psychoactive substance dependence. *Comprehensive Psychiatry*. 35(2):124-8.

Heaton, R.K., Chelune, G.J., Talley, J.L., Kay, G.G. and Curtis, G. (2008). WCST forma completa revisionata. Adattamento italiano a cura. In: di MC, Harday MG, Carta, Cabras PL, editors. Firenze: Giunti OS.

Heinzel, A., Schafer, R., Muller, H.W., Schieffer, A., Ingenhag, A., Northoff, G., Franz, M. and Hauntzel, H. (2010). Differential modulation of valence and arousal in high-alexithymic and low-alexithymic individuals. *Neuroreport*. 21:998-1002.

Henry, J.D., Bailey, P.E., von Hippel, C., Rendell, P.G. and Lane, A. (2010). Alexithymia in Schizophrenia. *Journal of Clinical and Experimental Neuropsychology*. 32(8); 890-7.

Henry, J.D., Phillips, L.H., Crawford, J.R., Theodorou, G. and Summers, F. (2006). Cognitive and psychological correlates of alexithymia following traumatic brain injury. *Neuropsychologia*. 44: 62-72.

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

Herbert, B.M., Herbert, C. and Pollatos, O. (2011). On the relationship between interoceptive awareness and alexithymia: is interoceptive awareness related to emotional awareness? *Journal of Personality*. 79; 5-21.

Hermans, E.J., Henckens, M.J.A.G., Roelofs, K. and Fernandez, G. (2013). Fear Bradycardia and activation of the human periaqueductal grey. *NeuroImage*. 66: 278-287.

Herrmann, M.J., Ehlis, A.C., Ellgring, H., and Fallgatter, A.J. (2005). Early stages (P100) of face perception in humans as measured with event-related potentials (ERPs). *Journal of Neural Transmission*. 112(8):1073-81.

Hess, U. and Bourgeois, P. (2010). You smile–I smile: Emotion expression in social interaction. *Biological Psychology*. 84, 514–520.

Hess, U. and Fischer, A. (2013). Emotional mimicry as social regulation. *Personality and Social Psychological Review*. 17(2):142-57.

Hintikka, J., Honkalampi, K., Lehtonen, J. and Viinama, H. (2001). Are Alexithymia and Depression Distinct or Overlapping Constructs? A Study in a General Population. *Comprehensive Psychiatry*. 42 (3):234-239.

Hinz, S. (1968). (ed.) Caspar David Friedrich in Briefen un Bekenntnissen. MUnchen, Rogner & Bernhard.

Höistad, M. and Barbas, H. (2008). Sequence of information processing for emotions through pathways linking temporal and insular cortices with the amygdala. *Neuroimage*. 40(3):1016-33.

Homack, S., Lee, D. and Riccio, C.A. (2005). Test review: Delis-Kaplan executive function system. *Journal of Clinical and Experimental Neuropsychology*. 27(5):599-609.

Honkalampi, K., Hintikka, J., Tanskanen, A., Lehtonen, J. and Viinamaki, H. (2000). Depression is strongly associated with alexithymia in the general population. *Journal of Psychosomatic Research*. 48; 99-104.

Horney, K. (1952). The paucity of inner experiences. *American Journal of Psychoanalysis*. 12: 3-9.

Huang, M.F., Yeh, Y.C., Tsang, H.Y., and Chen, C.S. (2010). Alexithymia associated with bilateral globus pallidus lesions after carbon monoxide poisoning. *Kaohsiung Journal of Medical Science*. 26(6):333-6.

Hussey, E. and Safford, A. (2009). Perception of facial expression in somatosensory cortex supports simulationist models. *The Journal of Neuroscience*, 29(2), 301-2.

Hyer, L., Woods, M.G., Summers, M.N., Boudewyns, P. and Harrison, W.R. (1990). Alexithymia among Vietnam veterans with posttraumatic stress disorder. *Journal of Clinical Psychiatry*. 51(6):243-7.

Ibáñez, A., Riveros, R., Hurtado, E., Gleichgerrcht, E., Urquina, H., Herrera, E., Amoruso, L., Reyes, M.M., and Manes, F. (2012). The face and its emotion: right N170 deficits in structural processing and early emotional discrimination in schizophrenic patients and relatives. *Psychiatry Research*. 195(1-2):18-26.

Itier, R.J. and Taylor, M.J. (2004). Source analysis of the n170 to faces and objects. *NeuroReport*. 15:1261-1265.

Jabbi, M., Swart, M. and Keysers, C. (2007). Empathy for positive and negative emotions in the gustatory cortex. *NeuroImage*. 34(4); 1744-53.

Jacobs, R.H., Renken, R., Aleman, A. and Cornelissen, F.W. (2012). The amygdala, top-down effects, and selective attention to features. *Neuroscience and Biobehavioral Review*, 36(9), 2069-84.

Johansen, J.P., Tarpley, J.W., LeDoux, J.E., and Blair, H.T. (2010). Neural substrates for expectation-modulated fear learning in the amygdala and periaqueductal gray. *Nature Neuroscience*. 13(8):979-86.

Johnstone, B., Callahan, C.D., Kapila, C.J. and Bouman, D.E. (1996). The comparability of the WRAT-R reading test and NAART as estimates of premorbid intelligence in neurologically impaired patients. *Archives of Clinical Neuropsychology*. 11(6):513-9.

Kalin, N.H., Shelton, S.E. and Davidson, R.J. (2004). The role of the central nucleus of the amygdala in mediating fear and anxiety in the primate. *The Journal of Neuroscience*. 24(24):5506-15.

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

Kano, M., Hamaguchi, T., Itoh, M., Yanai, K., and Fukudo, S. (2007). Correlation between alexithymia and hypersensitivity to visceral stimulation in human. *Pain*. 132(3):252-63.

Kano, M., Mizuno, T., Kawano, Y., Aoki, M., Kanazawa, M., and Fukudo, S. (2012). Serotonin transporter gene promoter polymorphism and alexithymia. *Neuropsychobiology*. 65(2):76-82.

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

Kapp, B.S., Frysinger, R.C., Gallagher, M. and Haselton, J.R. (1979). Amygdala central nucleus lesions: effect on heart rate conditioning in the rabbit. *Physiological Behavior*, 23(6), 1109-17.

Kapp, B.S., Whalen, P.J., Supple, W.F. and Pascoe, J.P. (1992) Amygdaloid contributions to conditioned arousal and sensory information processing. in J.P. Aggleton, (ed), *The Amygdala: Neurobiological Aspects of Emotion, Memory, and Mental Dysfunction* (pp. 229-254), New York: Wiley-Liss.

- Karlsson, H., Naatanen, P. and Stenman, H. (2008). Cortical activation in alexithymia as a response to emotional stimuli. *The British Journal of Psychiatry*. 192: 32-38.
- Kellner, R. (1987). A symptom questionnaire. *Journal of Clinical Psychiatry*. 48: 268–273.
- Kelman, N. (1952). Clinical aspects of externalized living. *American Journal of Psychoanalysis*. 12: 15-23.
- Kessler, H., Schwarze, M., Filipic, S., Traue, H.C., and von Wietersheim, J. (2006). Alexithymia and facial emotion recognition in patients with eating disorders. *International Journal of Eating Disorders*. 39(3):245-51.
- Keysers, C., Wicker, B., Gazzola, V., Anton, J.L., Fogassi, L. and Gallese, V. (2004). A touching sight: SII/PV activation during the observation and experience of touch. *Neuron*. 42(2); 335-346.
- Knoll, J. F., & Hodapp, V. (1992). A comparison between two methods for assessing heartbeat perception. *Psychophysiology*, 29, 218–222.
- Kokkonen, P., Karvonen, J.T., Veijola, J., Laksy, K., Jokelainen, J., Jarvelin, M.R. and Joukamaa, M. (2001). Prevalence of socio-demographic correlates of alexithymia in a population sample of young adults. *Comprehensive Psychiatry*. 42;471-476.
- Komaki, G. (2006). Impaired self-awareness and theory of mind: an fMRI study of mentalizing in alexithymia. *Neuroimage*. 32(3):1472-82.
- Kooiman, C.G. (1998). The status of alexithymia as a risk factor in medically unexplained physical symptoms. *Comprehensive Psychiatry*. 39(3):152-9.
- Kreitler S. (2002). The psychosemantic approach to alexithymia. *Personality and Individual Differences*. 33, 393–407.
- Krombholz, A., Schaefer, F., and Boucsein, W. (2007). Modification of N170 by different emotional expression of schematic faces. *Biological Psychology*. 76(3):156-62.
- Kugel, H., Eichmann, M., Dannlowski, U., Ohrmann, P., Bauer, J., Arolt, V., Heindel, W. and Suslow, T. (2008). Alexithymic features and autonomic amygdale reactivity to facial emotion. *Neuroscience letters*. 435; 40-44.
- Làdavas, E. (2002). Functional and dynamic properties of visual peripersonal space. *Trends in Cognitive Sciences*, 6, 17–22.

Lamberty, G.J. and Holt, C.S. (1995). Evidence for a verbal deficit in alexithymia. *Journal of Neuropsychiatry Clinical Neuroscience*. 7: 320-324.

Lane, R.D., and Schwartz, G.E. (1987). Levels of emotional awareness: a cognitive-developmental theory and its application to psychopathology. *American Journal of Psychiatry*. 144(2):133-43.

Lane, R.D., Sechrest, L., Reidel, R., Weldon, V., Kaszniak, A. and Schwartz, G.E. (1996). Impaired verbal and nonverbal emotion recognition in alexithymia. *Psychosomatic Medicine*. 58(3):203-10.

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

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

Lang, P.J., Bradley, M.M., and Cuthbert, B.N. (1999). International Affective Picture System (IAPS): Instruction manual and affective ratings. Technical Report A-4, Center for Research in Psychophysiology. University of Florida, Gainesville/Florida.

Larsen, J.T., Norris, C.J. and Cacioppo, J.T. (2003). Effects of positive and negative affect on electromyographic activity over the *Zygomaticus major* and *corrugators supercilii*. *Psychophysiology*. 40; 776-785.

Larsen, J.K., Brand, N., Bermond, B., and Hijman, R. (2003). Cognitive and emotional characteristics of alexithymia: a review of neurobiological studies. *Journal of Psychosomatic Research*. 54(6):533-41.

Latzman, R.D. and Markon, K.E. (2010). The factor structure and age-related factorial invariance of the Delis-Kaplan Executive Function System (D-KEFS). *Assessment*. 17(2):172-84.

Lazarus, R.S. (1991). Emotion and adaptation. *New York, NY: Oxford University press*.

LeBar, K.S., LeDoux, J.E., Spencer, D.D. and Phelps, E.A. (1995). Impaired fear conditioning following unilateral temporal lobectomy in humans. *The Journal of Neuroscience*. 15: 6846-6855.

LeDoux, J.E., Iwata, J., Cicchetti, P. and Reis D.J. (1988). Different projections of the central amygdaloid nucleus mediate autonomic and behavioral correlates of conditioned fear. *Journal of Neuroscience*. 8(7): 2517-2529.

LeDoux, J. (1996). Emotional networks and motor control: a fearful view. *Progress in Brain Research*. 107:437-46. Review.

LeDoux, J. (2012). Rethinking the emotional Brain. *Neuron Perspective*. 73: 653-676.

LeDoux, J.E. (2013). The slippery slope of fear. *Trends in Cognitive Science*. 17(4):155-6.

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

Lee, B.T., Lee, H.Y., Park, S.A., Lim, J.Y., Tae, W.S., Lee, M.S., Joe, S.H., Jung, I.K. and Ham, B.J. (2011). Neural substrates of affective face recognition in alexithymia: a functional magnetic resonance imaging study. *Neuropsychobiology*. 63:119-124.

Lee, S.H., Kim, E.Y., Kim, S.R., Im, W.Y., Seo, H. S., Han, S.W., Park, Y.M. and Kim, H. (2007). Facial Affect perception and Event related potential n170 in schizophrenia: a preliminary study. *Clinical Psychopharmacology and Neuroscience*. 5(2): 76-80.

Lesser, I.M. (1981). A review of the alexithymia concept. *Psychosomatic Medicine*. 43:531-43

Levenson, R.W., Ekman, P., and Friesen, W.V. (1990). Voluntary facial action generates emotion-specific autonomic nervous system activity. *Psychophysiology*. 27(4):363-84.

Levenson, R.W. (2003). Blood, sweat, and fears: the autonomic architecture of emotion. *Annals of New York Academy of Science*. 1000:348-66.

Libby, W.L., Lacey, B.C. and Lacey, J.I. (1973). Pupillary and cardiac activity during visual attention. *Psychophysiology*. 10, 270-294.

Liddell, B.J., Brown, K.J., Kemp, A.H., Barton, M.J., Das, P., Peduto, A., Gordon, E. and Williams, L.M. (2005). A direct brainstem-amygdala-cortical 'alarm' system for subliminal signals of fear. *NeuroImage*. 24(1):235-43.

- Linden, W., Lenz, J.W. and Stossel, C. (1996). Alexithymia, defensiveness and cardiovascular reactivity to stress. *Journal of Psychosomatic Research*. 41(6):575-83.
- Liu, Y., Keil, A., and Ding, M. (2012). Effects of emotional conditioning on early visual processing: temporal dynamics revealed by ERP single-trial analysis. *Human Brain Mapping*. 33(4):909-19.
- Loas, G., Fremaux, D., Boyer, P. (1997). Anhedonia and alexithymia: distinct or overlapping constructs. *Perception & Motor Skills*. 84(2):415-25.
- Loas, G., Dhee-Perot, P., Chaperot, C., Fremaux, D., Gayant, C., and Boyer, P. (1998). Anhedonia, alexithymia and locus of control in unipolar major depressive disorders. *Psychopathology*. 31(4):206-12.
- Loas, G., Otmani, O., Lecercle, C. and Jouvent, R. (2000). Relationships between the emotional and cognitive components of alexithymia and dependency in alcoholics. *Psychiatry Research*. 96:63-74.
- Lumley, M.A., Stettner, L. and Wehmer, F. (1996). How are alexithymia and physical illness linked? A review and critique of pathways. *Journal of Psychosomatic Research*. 41(6):505-18.
- Lundquist, L. O. and Dimberg, A. (1995). Facial expressions are contagious. *Journal of Psychophysiology*, 9, 203–211.
- Lundqvist, D., Flykt, A., and Öhman, A. (1998). The Karolinska Directed Emotional Faces - KDEF. CD ROM from Department of Clinical Neuroscience, Psychology section: Karolinska Institutet, ISBN 91-630-7164-9.
- Lynn, S.K., and Salisbury, D.F. (2008). Attenuated modulation of the N170 ERP by facial expressions in schizophrenia. *Clinical EEG & Neuroscience*. 39(2):108-11.
- Lyvers, M., Jamieson, R., and Thorberg, F.A. (2013). Risky cannabis use is associated with alexithymia, frontal lobe dysfunction, and impulsivity in young adult cannabis users. *Journal of Psychoactive Drugs*. 45(5):394-403.
- Malacuso, E., Frith, C.D. and Driver, J. (2005). Modulation of human visual cortex by crossmodal spatial attention. *NeuroImage*. 26; 414-425.
- MacLean, P.D. (1949). Psychosomatic diseases and “the visceral brain”. *Psychosomatic Medicine*. 11:338-353.
- Malacuso, E. (2006). Multisensory processing in sensory-specific cortical areas. *Neuroscientist*, 12(4), 327-38.

Maier, M.E., Scarpazza, C., Filogamo, R. and Làdavas, E. (submitted). Performance monitoring is related to processing internal affective states.

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

Mantani, T., Okamoto, Y., Shirao, N., Okada, G. and Yamawaski, S. (2005). Reduced Activation of Posterior Cingulate Cortex during imagery in subjects with high degrees of alexithymia: a functional magnetic resonance imaging study. *Biological Psychiatry*. 57; 982-990.

Marchesi, C., Fontò, S., Balista, C., Cimmino, C. and Maggini, C. (2005). Relationship between alexithymia and panic disorder: a longitudinal study to answer an open question. *Psychotherapy and Psychosomatics*. 74; 56-60.

Maren, S. (2001). Neurobiology of Pavlovian fear conditioning. *Annual Review Neuroscience*, 24, 897-931.

Martin, J.B., and Pihl, R.O. (1986). Influence of alexithymic characteristics on physiological and subjective stress responses in normal individuals. *Psychotherapy & Psychosomatic*. 45(2):66-77.

McClelland, S., Garcia, R.E., Peraza, D.M., Shih, T.T., Hirsch, L.J., Hirsch, J. and Goodman, R.R. (2006). Facial emotion recognition after curative nondominant temporal lobectomy in patients with mesial temporal sclerosis. *Epilepsia*, 47(8), 1337-42.

McDonald, P.W. and Prkachin, K.M. (1990). The Expression and Perception of Facial Emotion in Alexithymia: a pilot Study. *Psychosomatic Medicine*. 52; 199-210.

McDonald, A.J. (1991). Topographical organization of amygdaloid projections to the caudatoputamen, nucleus accumbens, and related striatal-like areas of the rat brain. *Neuroscience*. 1991;44(1):15-33.

McDonald, A.J. (1992). Projection neurons of the basolateral amygdala: a correlative Golgi and retrograde tract tracing study. *Brain Research Bulletin*. 28(2):179-85.

McIntosh, D. N., Druckman, D. and Zajonc, R. B. (1994). Socially induced affect. In: D. Druckman & R. A. Bjork (Eds.), *Learning, remembering, believing: Enhancing human performance* (pp. 251–276, 364–371). Washington, DC: National Academy Press.

McIntosh, D. N. (1996). Facial feedback hypotheses: Evidence, implications, and directions. *Motivation and Emotion*, 20, 121–147.

McIntosh, D.N. (2006). Spontaneous facial mimicry, liking and emotional contagion. *Polish Psychological Bulletin*. 37(1): 31-42.

McIntosh, D.N., Reichmann-Decker, A., Winkielman, P. and 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., Roux, S., and Batty, M. (2013). Early visual ERPs are influenced by individual emotional skills. *Social Cognitive and Affective Neuroscience*. 9(8):1089-98.

Mende-Siedlecki, P., Verosky, S.C., Turk-Browne, N.B. and Todorov, A. (2013). Robust selectivity for faces in the human amygdala in the absence of expressions. *Journal of Cognitive Neuroscience*, 25(12), 2086-106.

Meng, X.L., Rosenthal, R. and Rubin, D.B. (1992). Comparing Correlated Correlation Coefficients. *Psychological Bulletin*. 111(1); 172-175.

Meganck, R., Vanheule, S., Desmet, M., and Inslegers, R. (2009). Does the 20-item Toronto Alexithymia Scale measure alexithymia? A study of natural language use. *Psychological Report*. 105(3 Pt 1):945-56.

Mériaux, K., Wartenburger, I., Kazzner, P., Prehn, K., Lammers, C.H., van der Meer, E., Villringer, A. and Heekeren, H.R. (2006). A neural network reflecting individual differences in cognitive processing of emotions during perceptual decision making. *NeuroImage*. 33(3):1016-27.

Messina, A., Beadle, J.N. and Paradiso, S. (2014). Towards a classification of alexithymia: primary, secondary and organic. *Journal of Psychopathology*. 20:38-49.

Miyoshi, M., Katayama, J., and Morotomi, T. (2003). Face-specific n170 component is modulated by facial expressional change. *NeuroReport*. 15(5), 911-914.

Mobbs, D., Marchant, J.L., Hassabis, D., Seymour, B., Tan, G., Gray, M., Petrovic, P., Dolan, R.J., and Frith, C.D. (2009). From threat to fear: the neural organization of defensive fear systems in humans. *The Journal of Neuroscience*. 29(39):12236-43.

Moody, E.J., McIntosh, D.N., Mann, L.J. and Weisser, K.R. (2007). More than mere mimicry? The influence of emotion on rapid facial reactions to faces. *Emotion*. 7(2): 447-457.

Morel, S., Ponz, A., Mercier, M., Vuilleumier, P., and George, N. (2009). EEG-MEG evidence for early differential repetition effects for fearful, happy and neutral faces. *Brain Research*. 1254, 84–98.

Moriguchi, Y., Ohnishi, T., Lane, R.D., Maeda, M., Mori, T., Nemoto, K., Matsuda, H., and Komaki, G. (2006). Impaired self-awareness and theory of mind: an fMRI study of mentalizing in alexithymia. *Neuroimage*. 32(3):1472-82.

Moriguchi, Y., Ohnishi, T., Decety, J., Hirakata, M., Maeda, M., Matsuda, H., and Komaki, G. (2009). The human mirror neuron system in a population with deficient self-awareness: an fMRI study in alexithymia. *Human Brain Mapping*. 30(7):2063-76.

Moriguchi, Y., and Komaki, G. (2013). Neuroimaging studies of alexithymia: physical, affective, and social perspectives. *Biopsychosocial Medicine*. 7(1):8.

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.

Morris, J.S., Ohman, A. and Dolan, R.J. (1998). Conscious and unconscious emotional learning in the human amygdale. *Nature*. 393;467-470.

Mueller, J., Alpers, G. and Reim, N. (2006). Dissociation of rated emotional valence and Stroop interference in observer-rated alexithymia. *Journal of Psychosomatic Research*. 61; 261-269.

Nakao, M., Barsky, A.J., Kumano, H., and Kuboki, T. (2002). Relationship between somatosensory amplification and alexithymia in a Japanese psychosomatic clinic. *Psychosomatics*. 43(1):55-60.

Nemiah, J.C., and Sifneos, P.E. (1970). Psychosomatic illness: a problem in communication. *Psychotherapy and Psychosomatics*. 18(1):154-60.

Nemiah, J. C. and Sifneos, P. E. (1970). Affect and fantasy in patients with psychosomatic disorder. In: Hill OW, editor. *Modern Trends in Psychosomatic Medicine*, vol. 2. New York: Apple-ton-Century-Crofts. 26-34.

Nemiah, J.C., Freyberger, H. and Sifneos, P.E. (1976). Alexithymia: a view of the psychosomatic process. In: Hill OW (ed.), *Modern Trends in Psychosomatic Medicine*, vol 3. *Butterworths, London*, pp. 430-439.

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.

Newton, L.T. and Contrada, R.J. (1994). Alexithymia and repression: contrast in emotion-specific coping styles. *Psychosomatic Medicine*. 56, 457-462.

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

Niedenthal, P.M., Brauer, M., Halberstadt, J.B. and Innes-Ker, A.H. (2001). When did the smile drop? Facial mimicry and the influences of emotional state on the detection of change in emotional expression. *Cognition & Emotion*. 15(6): 853-864

Niedenthal, P.M., Barsalou, L.W., Winkielman, P., Krauth-Gruber, S., and Ric, F. (2005). Embodiment in attitudes, social perception, and emotion. *Personality and Social Psychological Review*. 9(3):184-211.

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

Niedenthal, P.M., Mermillod, M., Maringer, M. and Hess, U. (2010). The Simulation of Smiles (SIMS) model: Embodied simulation and the meaning of facial expression. *Behavioral Brain*. 33(6):417-33.

Nittono, H., Shibuya, Y., and Hori, T. (2007). Anterior N2 predicts subsequent viewing time and interest rating for novel drawings. *Psychophysiology*. 44(5):687-96.

Nowakowski, M.E., McFarlane, T., and Cassin S. (2013). Alexithymia and eating disorders: a critical review of the literature. *Journal of Eating Disorders*. 1:21.

Nyklíček, I., and Vingerhoets, A.J. (2000). Alexithymia is associated with low tolerance to experimental painful stimulation. *Pain*. 85(3):471-5.

Oberman, L.M., Winkielman, P. and Ramachandran, V.S. (2007). Face to face: blocking facial mimicry can selectively impair recognition of emotional expressions. *Social Neuroscience*. 2(3-4):167-78.

Olatunji, B.O., William, N.L., Tolin, D.F., Abramowitz, J.S., Sawchuk, C.N., Lohr, J.M. & Elwood, L.S. (2007). The disgust scale: item analysis, factor structure, and suggestions for refinement. *Psychological Assessment*. 19(3): 281-297.

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

Papciak, A.S., Feuerstein, M., and Spiegel, J.A. (1985). Stress reactivity in alexithymia: decoupling of physiological and cognitive responses. *Journal of Human Stress*. 11(3):135-42.

Pasley, B.N., Mayes, L.C. and Schultz, R.T. (2004). Subcortical discrimination of unperceived objects during binocular rivalry. *Neuron*. 42; 163-172.

Paradiso, S., Vaidya, J.G., and McCormick, L.M. Aging and alexithymia: association with reduced right rostral cingulate volume. *American Journal of Geriatric Psychiatry*. 16:760-9.

Parker, P.D., Prkachin, K.M. and Prkachin, G.C. (2005) Processing of facial expressions of negative emotion in alexithymia: the influence of temporal constraint. *Journal of Personality*. 73(4):1087-107.

Parker, J.D., Taylor, G.J., and Bagby, R.M. (2003). The 20-Item Toronto Alexithymia Scale. III. Reliability and factorial validity in a community population. *Journal of Psychosomatic Research*. 55(3):269-75.

Parker, J.D.A., Taylor, G.J., Bagby, R.M. and Acklin, M.W. (1993). Alexithymia in panic disorder and simple phobia: a comparative study. *American Journal of Psychiatry*. 150;1105-1107.

Pegna, A. J., Landis, T., and 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., and Khateb, A. (2011). Early ERP modulation for task-irrelevant subliminal faces. *Frontiers in Psychology*, 2, 88.

Phan, K.L., Wager, T., Taylor, S.F. and Liberzon, I. (2002). Functional neuroanatomy of emotion: a meta-analysis of emotion activation studies in PET and fMRI. *NeuroImage*, 16(2), 331-48.

Phelps, E.A. (2006). Emotion and cognition: insights from studies of the human amygdala. *Annual Review Psychology*, 57, 27-53. Review.

Phillips, M.L., Young, A.W., Senior, C., Brammer, M., Andrew, C., Calder, A.J., Bullmore, E.T., Perrett, D.I., Rowland, D., Williams, S.C., Gray, J.A., and David,

AS. (1997). A specific neural substrate for perceiving facial expressions of disgust. *Nature*. 389(6650):495-8.

Pitcher, D., Garrido, L., Walsh, V. and Duchaine, B.C. (2008). Transcranial magnetic stimulation disrupts the perception and embodiment of facial expressions. *The Journal of Neuroscience*, 28(36), 8929-33.

Pizzagalli, D.A., Lehmann, D., Hendrick, A.M., REGARD, M., Pascual-Marqui, R.D., Davidson, R.J. (2002). Affective judgments of faces modulate early activity (approximately 160 ms) within the fusiform gyri. *NeuroImage*. 16(3 Pt 1):663-77.

Pollatos, O., Kirsch, W., and Schandry, R. (2005). On the relationship between interoceptive awareness, emotional experience, and brain processes. *Brain research. Cognitive brain research*. 25(3):948-62.

Pollatos, O., Schubo, A., Herbert, B.M., Matthias, E. and Schandy, R. (2008). Deficits in early emotional reactivity in alexithymia. *Psychophysiology*. 45; 839-846.

Pollatos, O. and Gramann, K. (2011). Electrophysiological evidence of early processing deficits in alexithymia. *Biological Psychology*. 87;113-121.

Pollatos, O., Werner, N.S., Duschek, S., Schandry, R., Matthias, E., Traut-Mattausch, E. and Herbert, B.M. (2011). Differential effects of alexithymia subscales on autonomic reactivity and anxiety during social stress. *Journal of Psychosomatic Research*. 70: 525-533.

Porcelli, P. and Rafanelli, C. (2010). Criteria for Psychosomatic Research (DPCR) in the medical setting. *Current Psychiatry Report*. 12:246-254.

Porcelli, P. and Sonino, N. (2007). Psychological Factors Affecting Medical Conditions: A New Classification for DSM-V. *Advances in Psychosomatic Medicine*. Basel, Karger. vol 28, pp I–X.

Porges, S. (1993) Body Perception Questionnaire (Laboratory of Developmental Assessment, University of Maryland).

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

Preston, S.D. and de Waal, F.B. (2002). Empathy: Its ultimate and proximate bases. *Behavioral Brain Sciences*. 25(1):1-20.

Pourtois, G., Sander, D., Andres, M., Grandjean, D., Reveret, L., Olivier, E. and Vuilleumier, P. (2004). Dissociable roles of the human somatosensory and superior

temporal cortices for processing social face signals. *European Journal of Neuroscience*, 20 (12), 3507-15.

Rabavilas, A.D. (1987). Electrodermal activity in low and high alexithymia neurotic patients. *Psychotherapy & Psychosomatic*. 47(2):101-4.

Raven, J.C. (1947). Progressive matrices series sets A, AB, B: board and book form. London: Lewis.

Raven, J.C. (1981). Standard Progressive Matrices- Manuale *Organizzazioni Speciali*, Firenze.

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

Rellecke, J., Palazova, M., Sommer, W., and Schacht, A. (2011). On the automaticity of emotion processing in words and faces: event related brain potentials from a superficial task. *Brain & Cognition* . 77(1): 23-32.

Rellecke, J., Sommer, W., and Schacht, A. (2013). Emotion effects on the n170: a question of reference? *Brian Topography*. 26: 62-71.

Rey, G., Knoblauch, K., Prévost, M., Komano, O., Jouvent, R., and Dubal, S. (2010). Visual modulation of pleasure in subjects with physical and social anhedonia. *Psychiatry Research*. 176(2-3):155-60.

Rinn, W.E. (1984). The neuropsychology of facial expression: a review of the neurological and psychological mechanisms for producing facial expressions. *Psychological Bulletin*. 95(1):52-77.

Rizzolatti, G., Fabbri-Destro, M., and Cattaneo, L. (2009). Mirror neurons and their clinical relevance. *Nature Clinical Practice Neurology*. 5(1):24-34.

Roedema, T.M. and Simons, R.F. (1999). Emotion-processing deficit in alexithymia. *Psychophysiology*. 36(3):379-87.

Roelofs, K., Hagenars, M.A. and Stins, J. (2010). Facing freeze: social threat induces bodily freeze in humans. *Psychological Science*. 21(11): 1575-1581.

Roh, D., Kim, W.J., and Kim, C.H. (2011). Alexithymia in obsessive-compulsive disorder: clinical correlates and symptom dimensions. *Journal of Nervous Mental Disorder*. 199(9):690-5.

Rorden, C., Bonilha, L. and Nichols, T.E. (2007). Rank-order versus mean based statistics for neuroimaging. *NeuroImage*, 35, 1531-1537.

Rorden, C. and Brett, M. (2000). Stereotaxic display of brain lesions. *Behavioral Neurology*, 12, 191-200.

Rozin, P., Lowery, L., Imada, S. & Haidt, J. (1999). The CAD triad hypothesis: a mapping between three moral emotions (contempt, anger, disgust) and three moral codes (community, autonomy, divinity). *Journal of personality and social psychology*. 76, 574-586.

Rozin, P., Haidt, J. & McCauley, C.R. (2000). Disgust. In M. Lewis & J.M. Haviland (Eds.). *Handbook of emotions* (2nd ed., pp. 575-594). New York, NY: Guilford Press.

Ruesch, J. (1948). The infantile personality. *Psychosomatic Medicine*. 10: 134-144.

Sah, P. and Lopez De Armentia, M. (2003). Excitatory synaptic transmission in the lateral and central amygdala. *Annals of New York Academy Sciences*. 985:67-77.

Salminen, J.K., Saarijarvi, S., Aarela, E., Toikka, T. and Kauhanen, J. (1999). Prevalence of alexithymia and its association with socio-demographic variables in the general population of Finland. *Journal of Psychosomatic Research*. 46;75-82.

Sarinopoulos, I., Dixon, G.E., Short, S.J., Davidson, R.J., and Nitschke, J.B. (2006). Brain mechanisms of expectation associated with insula and amygdala response to aversive taste: implications for placebo. *Brain Behavioral Immunology*. 20(2):120-32.

Sato, W., Kochiyama, T., Yoshikawa, S., and 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.

Schachter, S. and Singer, JE. (1962). Cognitive, social and physiological determinants of emotional state. *Psychological Review*. 69: 379-99.

Schachter, S. (1964). The interaction of cognitive and physiological determinants of emotional state. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 1). New York: Academic Press.

Schaefer, R., Schneider, C., Tress, W., Franz, M., 2007. Cortical augmenting in alexithymic subjects after unpleasant acoustic stimulation. *Journal of Psychosomatic Research*. 63, 357–364.

Schandry, R. (1981). Heart beat perception and emotional experience. *Psychophysiology*. 18 (4): 483-8.

Schienle, A., Schäfer, A., Stark, R., Walter, B., Franz, M., and Vaitl, D. (2003). Disgust sensitivity in psychiatric disorders: a questionnaire study. *Journal of Nervous Mental Disorder*. 191(12):831-4.

Schultz, R.T. (2005). Developmental deficits in social perception in autism: the role of the amygdale and fusiform face area. *International Journal of Developmental Neuroscience*. 23; 125-141.

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

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

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

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

Schwaber, J.S., Kapp, B.S., Higgins, G.A. and Rapp, P.R. (1982). Amygdaloid and basal forebrain direct connections with the nucleus of the solitary tract and the dorsal motor nucleus. *The Journal of Neuroscience*. 2(10):1424-38.

Serino, A., Pizzoferrato, F. and Ladavas, E. (2008). Viewing a face (expecially one's own face) being touched enhances tactile perception on the face. *Psychological Science*. 19(5);434-9.

Serino, A., Giovagnoli, G. & Lådavas, E. (2009) I feel what you feel if you are similar to me. *PLoS One*. 4(3): e4930.

Siebert, M., Markowitsch, H.J. and Bartel, P. (2003). Amygdala, affect and cognition: evidence from 10 patients with Urbach-Wiethe disease. *Brain*, 126(Pt 12), 2627-37.

Sifneos, P.E. (1973). The prevalence of “alexithymic” characteristics in psychosomatic patients. *Psychotherapy and Psychosomatics*. 22;255-262.

Singer, T., Seymour, B., O'Doherty, J., Kaube, H., Dolan, R.J. and Frith, C.D. (2004). Empathy for pain involves the affective but not sensory components of pain. *Science*. 303(5661):1157-62.

Singer, T., Critchley, H.D. and Preuschoff, K. (2009). A common role of insula in feelings, empathy and uncertainty. *Trends of Cognitive Sciences*. 13(8):334-40.

Spalletta, G., Pasini, A., Costa, A., De Angelis, D., Ramundo, N., Paolucci, S., and Caltagirone, C. (2001). Alexithymic features in stroke: effects of laterality and gender. *Psychosomatic Medicine*. 63(6):944-50.

Spielberger, C.D., Vagg, P.R. and Barker, L.R. (1980) The factor structure of the State-Trait Anxiety Inventory. In: I.G. Sarason, C.D. Spielberg, (eds), *Stress and anxiety*, New York: Hemisphere/Wiley.

Spinnler, H. and Tognoni, G. (1987). Standardizzazione e taratura italiana di test neuropsicologici. *The Italian Journal of Neurological Science*. 8(6), 1-20.

Spitzer, C., Brandl, S., Rose, H.J., Nauck, M., and Freyberger, H.J. (2005). Gender-specific association of alexithymia and norepinephrine/cortisol ratios. A preliminary report. *Journal of Psychosomatic Research*. 59(2):73-6.

Stainbrook, E. (1952). Psychosomatic medicine in the nineteenth century. *Psychosomatic Medicine*. 14: 211-227.

Stone, L.A., and Nielson, K.A. (2001). Intact physiological response to arousal with impaired emotional recognition in alexithymia. *Psychotherapy & Psychosomatic*. 70(2):92-102.

Sturm, V.E., and Levenson, R.W. (2011). Alexithymia in neurodegenerative disease. *Neurocase*. 17(3):242-50.

Suslow, T., and Junghanns, K. (2002). Impairments of emotion situation priming in alexithymia. *Personality and Individual Differences*. 32, 541–550.

- Susskind, J.M., Lee, D.H., Cusi, A., Feiman, R., Grabski, W., and Anderson, A.K. (2008). Expressing fear enhances sensory acquisition. *Nature Neuroscience*. 11(7):843-50.
- Susskind, J.M., and Anderson, A.K. (2008). Facial expression form and function. *Communicative & Integrative Biology*. 2008;1(2):148-9.
- Swanson, L.W. and Petrovich, G.D. (1998). What is the amygdala? *Trends in Neuroscience*. 21(8):323-31.
- Swart, M., KorteKaas, R. and Aleman, A. (2009). Dealing with feelings: characterization of trait alexithymia on emotion regulation strategies and cognitive-emotional processing. *Plos One*. 4(6); e5751.
- Tamietto, M., and de Gelder, B. (2010). Neural bases of the non-conscious perception of emotional signals. *Nature Review Neuroscience*. 11(10):697-709.
- Taylor, G.J. (1984). Alexithymia: concept, measurement, and implications for treatment. *American Journal of Psychiatry*. 141(6):725-32.
- Taylor, G.J., Parker, J.D., and Bagby, R.M. (1990). A preliminary investigation of alexithymia in men with psychoactive substance dependence. *American Journal of Psychiatry*. 147(9):1228-30.
- Taylor, G.J., Bagby, R.M. and Parker, J.D. (1991). The alexithymia construct. A potential paradigm for psychosomatic medicine. *Psychosomatics*. 32;153-164.
- Taylor, G.J., Parker, J.D.A., Bagby, R.M. and Bourke, M.P. (1996). Relationship between alexithymia and psychological characteristics associated with eating disorders. *Journal of Psychosomatic Research*. 41; 561-568.
- Taylor, G.J., Bagby, R.M. and Parker, J.D. (1997). Disorders of affect regulation. Alexithymia in medical and psychiatric illness. *Cambridge University Press, Cambridge*.
- Taylor, G.J., Bagby, R.M. and Parker, J.D. (2003). The 20-Item Toronto Alexithymia Scale. IV. Reliability and factorial validity in different languages and cultures. *Journal of Psychosomatic Research*. 55:277-283.
- Taylor, G.J., and Bagby, R.M. (2004). New trends in alexithymia research. *Psychotherapy & Psychosomatics*. 73(2):68-77.

Taylor, G.J. (2010). Affects, trauma, and mechanisms of symptom formation: a tribute to John C. Nemiah, MD (1918-2009). *Psychotherapy & Psychosomatics*, 79(6):339-49.

TenHouten, W.D., Hoppe, K.D., Bogen, J.E., and Walter, D.O. (1986). Alexithymia: an experimental study of cerebral commissurotomy patients and normal control subjects. *American Journal of Psychiatry*, 143(3):312-6.

Terasawa, Y., Shibata, M., Moriguchi, Y., and Umeda, S. (2013). Anterior insular cortex mediates bodily sensibility and social anxiety. *Social Cognitive & Affective Neuroscience*, 8(3):259-66.

Terburg, D., Morgan, B.E., Montoya, E.R., Hooge, I.T., Thornton, H.B., Hariri, A.R., Panksepp, J., Stein, D.J. and van Honk, J. (2012). Hypervigilance for fear after basolateral amygdala damage in humans. *Translational Psychiatry*, 2:e115.

Thierry G, Martin CD, Downing P, and Pegna AJ. (2007). Controlling for interstimulus perceptual variance abolishes N170 face selectivity. *Nature Neuroscience*, 10(4):505-11.

Thom, N., Knight, J., Dishman, R., Sabatinelli, D., Johnson, D. C., and 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):849-60.

Todarello, O., Porcelli, P., Grilletti, F. and Bellomo, A. (2005). Is alexithymia related to negative symptoms of schizophrenia? A preliminary longitudinal study. *Psychopathology*, 38(6):310-4.

Todorov, A. (2012). The role of the amygdala in face perception and evaluation. *Motivation and Emotion*, 36(1), 16-26.

Torres, S., Guerra, M.P., Lencastre, L., Miller, K., Vieira, F.M., Roma-Torres, A., Brandão, I., and Costa, P. (2015). Alexithymia in anorexia nervosa: the mediating role of depression. *Psychiatry Research*, 225(1-2):99-107.

Tranel, D., Gullickson, G., Koch, M. and Adolphs, R. (2006). Altered experience of emotion following bilateral amygdala damage. *Cognitive Neuropsychiatry*, 11(3), 219-32.

Tressoldi, P.E., Vio, M., Gugliotta, M., Bisiacchi, P.S. and Cendron, M. (2005) *Batteria di valutazione neuropsicologica per l'età evolutiva (BVN 5-11)*. Trento: Erickson.

Tsuchiya, N., Moradi, F., Felsen, C., Yamazaki, M. and Adolphs, R. (2009). Intact rapid detection of fearful faces in the absence of the amygdala. *Nature Neuroscience*, 12(10), 1224-5.

Tybur, J.M., Lieberman, D., Kurzban, R., and DeScioli, P. (2013). Disgust: evolved function and structure. *Psychological Review*. 120(1):65-84.

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

van der Velde, J., Servaas, M.N., Goerlich, K.S., Bruggeman, R., Horton, P., Costafreda, S.G. and Aleman, A. (2013). Neural correlates of alexithymia: A meta-analysis of emotion processing studies. *Neuroscience Biobehavioral Review*. 37(8):1774-85.

Van't Wout, M., Aleman, A., Bermond, B. and Kahn, R.S. (2007). No words for feelings: alexithymia in schizophrenia patients and first-degree relatives. *Comprehensive Psychiatry*. 48(1):27-33.

Vaughan, K. B. and Lanzetta, J. T. (1980). Vicarious instigation and conditioning facial expressive and autonomic responses to a model's expressive display of pain. *Journal of Personality and Social Psychology*, 38, 909—923.

Vermeulen, N., Luminet, O. and Corneille, O. (2006). Alexithymia and the automatic processing of affective information: evidence from the affective priming paradigm. *Cognition & Emotion*. 20 (1): 64-91.

Vermeulen, N., Godefroid, J. & Mermillod, M. (2009). Emotional modulation of attention: fear increases but disgust reduces the attentional blink. *Plos One*. 4 (11): e7924.

Vuilleumier, P., Richardson, M.P., Armony, J.L., Driver, J. and Dolan, R.J. (2004). Distant influences of amygdala lesion on visual cortical activation during emotional face processing. *Nature Neuroscience*, (11), 1271-8.

Vytal, K. and Hamann, S. (2010). Neuroimaging support for discrete neural correlates of basic emotions: a voxel-based meta-analysis. *Journal of Cognitive Neuroscience*, 22(12), 2864-85.

Whalen, P.J. (1998). Fear, Vigilance, and Ambiguity: Initial Neuroimaging Studies of the Human Amygdala. *Current Direction in Psychological Science*, 7, 177-188.

Wehmer, F., Brejnak, C., Lumley, M.A. and Stettner, L. (1995). Alexithymia and physiological reactivity to emotion-provoking visual scenes. *Journal of Nervous Mental Diseases*. 183, 351-357.

Wexler, B.E., Levenson, L., Warrenburg, S. and Price, L.H. (1994). Decreased perceptual sensitivity to emotion-evoking stimuli in depression. *Psychiatry Research*. 51(2):127-38.

Wheaton, M.G., Holman, A., Rabinak, C.A., Macnamara, A., Proudfit, G.H., Phan, K.L. (2013). Danger and disease: electrocortical responses to threat- and disgust-eliciting images. *International Journal of Psychophysiology*. 90(2):235-9.

Wicker, B., Keysers, C., Plailly, J., Royet, J.P., Gallese, V. and Rizzolatti, G. (2003). Both of us disgusted in My insula: the common neural basis of seeing and feeling disgust. *Neuron*. 40(3); 655-64.

Wild, B., Erb, M. and Bartels, M. (2001). Are emotions contagious? Evoked emotions while viewing emotionally expressive faces: quality, quantity, time course and gender differences. *Psychiatry Research*. 102(2):109-24.

Wilensky, A.E., Schafe, G.E., Kristensen, M.P., and LeDoux, J.E. (2006). Rethinking the fear circuit: the central nucleus of the amygdala is required for the acquisition, consolidation, and expression of Pavlovian fear conditioning. *The Journal of Neuroscience*. 26(48):12387-96.

Williams, C., and Wood, R.L. (2010). Alexithymia and emotional empathy following traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*. 32(3):259-67.

Winkielman, P., Berridge, K.C., and Wilbarger, J.L. (2005). Unconscious affective reactions to masked happy versus angry faces influence consumption behavior and judgments of value. *Personality & Social Psychological Bulletin*. 31(1):121-35.

Winston, J.S., O'Doherty, J. and Dolan, R.J. (2003) Common and distinct neural responses during direct and incidental processing of multiple facial emotions. *NeuroImage*, 20(1), 84-97.

Wise, T.N., Mann, L.S., Hryvniak, M., Mitchell, J.D., and Hill, B. (1990). The relationship between alexithymia and abnormal illness behavior. *Psychotherapy and Psychosomatics*. 54(1):18-25.

Wise, T.N., and Mann, L.S. (1994). The relationship between somatosensory amplification, alexithymia, and neuroticism. *Journal of Psychosomatic Research*. 38(6):515-21.

Wright, P., He, G., Shapira, N.A., Goodman, W.K., and Liu, Y. (2004). Disgust and the insula: fMRI responses to pictures of mutilation and contamination. *Neuroreport*. 15(15):2347-51.

Yehuda, R., Steiner, A., Kahana, B., Binder-Brynes, K., Southwick, S.M., Zelman, S., and Giller, E.L. (1997). Alexithymia in Holocaust survivors with and without PTSD. *Journal of Trauma Stress*. 10(1):93-100.

Young, M.P., Scannell, J.W., Burns, G.A. and Blakemore, C. (1994). Analysis of connectivity: neural systems in the cerebral cortex. *Review Neuroscience*, 5(3), 227-50.

Yu, S., Li, H., Liu, W., Zheng, L., Ma, Y., Chen, Q., Chen, Y., Yu, H., Lu, Y., Pan, B. & Wang, W. (2011). Alexithymia and personality disorder functioning styles in paranoid schizophrenia. *Psychopathology*. 44(6):371-8.

Zaki, J., Davis, J.I., and Ochsner, K.N. (2012). Overlapping activity in anterior insula during interoception and emotional experience. *Neuroimage*. 62(1):493-9.

Zald, D.H. (2003). The human amygdala and the emotional evaluation of sensory stimuli. *Brain Research Review*. 41(1):88-123.

Zeitlin, S.B., McNally, R.J., and Cassiday, K.L. (1993). Alexithymia in victims of sexual assault: an effect of repeated traumatization? *American Journal of Psychiatry*. 150(4):661-3.

Zhu, X.Z., Wang, X.Y., Huang, T., Yao, S.Q. and Tang, H.B. (2006). A comparative study of Wisconsin Card Sorting Test in individuals with different degrees of alexithymia. *Chinese Journal of Clinical Psychology*. 14: 132-133.

Zoccolotti, P., Pizzamiglio, L., Pittau, P.A. and Galati, G. (1994). Batteria di test per l'esame dell'attenzione. Roma. PSYTEST; 1994.

APPENDIX

Dependent variable:	ddf			dif			eot		
	R ²	F (df)	p	R ²	F (df)	p	R ²	F (df)	p
model	0.377	1.693 (5)	.201	0.389	1.784 (5)	0.181	0.415	1.984 (5)	0.144
single predictors' coefficients	Beta	t (df)	p	Beta	t (df)	p	Beta	t (df)	p
corrugator_fear_125	-0.436	-0.84 (16)	0.415	-0.392	-0.763 (16)	0.458	-0.836	-1.663 (16)	0.119
corrugator_fear_375	0.917	1.637 (16)	0.124	1.028	1.853 (16)	0.085	0.547	1.008 (16)	0.331
corrugator_fear_625	-0.740	-1.963 (16)	0.070	-0.876	-2.347 (16)	0.034*	-0.571	-1.562 (16)	0.141
corrugator_fear_875	-0.227	-0.434 (16)	0.671	-0.126	-0.244 (16)	0.811	0.542	1.068 (16)	0.304
corrugator_fear_1125	0.037	0.1 (16)	0.922	0.04	0.11 (16)	0.914	-0.384	-1.083 (16)	0.297

Table A1: Results of linear regression with corrugator EMG activity in response to fearful faces as predictors. * denotes statistical significance.

Dependent variable:	ddf			dif			eot		
model	R²	F (df)	p	R²	F (df)	p	R²	F (df)	p
	0.102	0.319 (5)	0.893	0.207	0.729 (5)	0.613	0.115	0.364 (5)	0.865
single predictors' coefficients	Beta	t (df)	p	Beta	t (df)	p	Beta	t (df)	p
corrugator_happiness_125	-0.48	-0.741 (16)	0.471	-0.635	-1.044 (16)	0.314	0.346	0.539 (16)	0.599
corrugator_happiness_375	0.95	0.945 (16)	0.361	1.642	1.736 (16)	0.104	0.594	0.595 (16)	0.562
corrugator_happiness_625	-0.881	-0.907 (16)	0.38	-1.227	-1.344 (16)	0.2	-0.356	-0.369 (16)	0.718
corrugator_happiness_875	0.237	0.317 (16)	0.756	-0.121	-0.172 (16)	0.866	-0.313	-0.422 (16)	0.68
corrugator_happiness_1125	0.355	0.729 (16)	0.478	0.486	1.059 (16)	0.307	-0.401	-0.829 (16)	0.421

Table A23: Results of linear regression with corrugator EMG activity in response to happy faces as predictors.

Dependent variable:	ddf			dif			eot		
model	R²	F (df)	p	R²	F (df)	p	R²	F (df)	p
	0.129	0.414 (5)	0.831	0.114	0.362 (5)	0.866	0.27	1.034 (5)	0.436
single predictors' coefficients	Beta	t (df)	p	Beta	t (df)	p	Beta	t (df)	p
zygomaticus_fear_125	-0.424	-0.8 (16)	0.437	-0.401	-0.751 (16)	0.465	-0.752	-1.549 (16)	0.144
zygomaticus_fear_375	0.324	0.521 (16)	0.61	0.186	0.297 (16)	0.771	0.106	0.187 (16)	0.854
zygomaticus_fear_625	-0.376	-0.624 (16)	0.543	-0.16	-0.263 (16)	0.796	0.166	0.301 (16)	0.768
zygomaticus_fear_875	0.459	0.937 (16)	0.365	0.401	0.811 (16)	0.431	0.323	0.72 (16)	0.483
zygomaticus_fear_1125	-0.063	-0.111 (16)	0.913	0.067	0.117 (16)	0.908	0.005	0.01 (16)	0.992

Table A3: Results of linear regression with zygomaticus EMG activity in response to fearful faces as predictors.

Dependent variable:	ddf			dif			eot		
model	R²	F (df)	p	R²	F (df)	p	R²	F (df)	p
	0.67	5.677 (5)	0.005	0.638	4.936 (5)	0.008	0.288	1.13 (5)	0.389
single predictors' coefficients	Beta	t (df)	p	Beta	t (df)	p	Beta	t (df)	p
zygomaticus_happiness_125	0.21	0.395 (16)	0.699	0.033	0.06 (16)	0.953	-0.287	-0.368 (16)	0.718
zygomaticus_happiness_375	-0.394	-0.826 (16)	0.423	-0.199	-0.4 (16)	0.695	-0.008	-0.011 (16)	0.991
zygomaticus_happiness_625	-0.441	-1.801 (16)	0.093	-0.609	-2.376 (16)	0.032*	-0.483	-1.341 (16)	0.201
zygomaticus_happiness_875	0.948	2.981 (16)	0.01*	0.73	2.194 (16)	0.046*	0.212	0.455 (16)	0.656
zygomaticus_happiness_1125	-0.657	-1.265 (16)	0.226	-0.264	-0.486 (16)	0.634	0.083	0.109 (16)	0.915

Table A4: Results of linear regression with zygomaticus EMG activity in response to happy faces as predictors. * denotes statistical significance.

Dependent variable:	ddf			dif			eot		
model	R²	F (df)	p	R²	F (df)	P	R²	F (df)	p
	0.430	4.018 (3)	0.026	0.422	3.892 (3)	0.029	0.605	8.154 (3)	0.002
single predictors' coefficients	Beta	t (df)	p	Beta	t (df)	P	Beta	t (df)	p
EKG_happy	-0.149	-0.772 (16)	0.452	-0.159	-0.817 (16)	0.426	-0.486	-3.025 (16)	0.008*
EKG_neutral	-0.442	-2.093 (16)	0.053	-0.357	-1.681 (16)	0.112	-0.309	-1.76 (16)	0.097
EKG_fear	0.661	3.194 (16)	0.006*	0.676	3.245 (16)	0.005*	0.643	3.733 (16)	0.002*

Table A5: Results of linear regression with EKG parameters as predictors. * denotes statistical significance.