NEUROCOGNITIVE MECHANISMS OF THE EMOTIONAL CONTROL OF IMITATION

CANDIDATE

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SUBMITTED IN PARTIAL FULFILLMENT OF THE
REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY
AT
SCUOLA INTERNAZIONALE SUPERIORE DI STUDI AVANZATI (S.I.S.S.A)
TRIESTE, ITALY
February 26, 2010

“Dubium sapientiae initium”
Cartesio

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Abstract

Fear conditioning, facial expression recognition, autonomic activation, and emotional memory have been studied extensively during the last thirty years, yet the precise relationship between emotions and the action system has not been systematically explored. To our knowledge, the only human studies explaining this issue all employ the fear-potentiated startle reflex paradigm. In these studies, emotions are viewed as a preparatory state evoked by threat cues. This preparatory state is usually elicited by very unpleasant pictures, and is indexed by changes in the amplitude of electromyographic signals recorded from muscles involved in the startle reflex. Thus studies reported only covert responses and failed to find emotional modulations of overt voluntary action triggered by aversive stimuli.

In order to fill the gap in my dissertation I carried out 14 experiments to investigate the interplay between emotion and a particular subclass of overt actions: imitative processes.

In Study 1 (Experiments 1-4), subjects performed imitative and non imitative movements after emotional versus neutral primes. The main result was that only after the emotional prime and only for imitative movements reaction times were speeded up (what we called “Emotional Control Of Imitation, E.C.O.I. from now on). In Study 2 a computational model was developed in order to understand the functional mechanisms subserving the effects found in Study 1. By means of a biologically plausible

artificial neural network we were able to test 4 basic assumptions behind the E.C.O.I. In Study 3 using the ERP procedure I demonstrated that there is a differential cortical activity in the movement related potentials associated with the emotional imitative condition. In Study 4 I applied the ECOI paradigm to children affected by autism and showed that these children do not suffer from an *action-resonance mechanism* deficit, but they showed an impaired *emotional-resonance mechanism*.

In Study 5 by means of a fear conditioning technique I demonstrated that the emotional enhancement of imitation is a flexible mechanism that can be learned and unlearned according to environmental changes.

Overall, in my dissertation I was able to carry out evidences that a link between emotions and actions does exist.

Acknowledgments

I would like to thank all the people without whom this thesis could have not been possible.

First of all I would like to thank my supervisor, Raffaella Rumiati. She has constantly inspired and stimulated me in exploring fields originally unknown to me and, more importantly, she gave me the opportunity to learn several neuroscientific methods (EEG, fMRI, neuropsychology) and increase my expertise in doing research thanks to which I manage to get now a job at CiMeC/DiSCoF in Trento.

I would like to especially thank Paolo Brambilla for giving me the opportunity to study autistic children and to establish a fruitful collaboration. I would like also to thank Glyn Humphreys and Pia Roshtstein, for giving me the opportunity to learn fMRI and spending nine months at the Birmingham University Imaging Center. Unfortunately this study could have not been reported in this work because was not strictly related to the topic of my thesis. Nevertheless, it resulted in an important work for my PhD. I also would like to thank Rick Cooper for his precious supervision on the computational model presented in this thesis and all the people in the Udine hospital in which I ran my studies (EEG and patients). Special thanks go also to my collaborators who helped me during the years in collecting data (Danielle, Gioia, Joana, Roma and Tania). I would like to thank all my colleagues and friends at SISSA, in particular Alessandro, Alessio, Andrea, Antonio, Claudia, Corrado, Cristiano, Fabio, Gaz, Gioia, Linda, Liuba, Roma and Tania, and all the professors of the Cognitive Neuroscience sector, Alessandro Treves, Evan Balaban, Jacques Mehler, Mathew Diamond and Tim Shallice.

A special thank goes to my family who strongly supported me in these years of hard work with lovely and constant encouragements and patience.

Last but not least, I would like to thank all my friends around Italy: Daniele, Gabriele, Gustavo, Francesco, Manuela, from Taranto; Carmine, Francesco, Massi, Valentina from Trieste; Ale, Mira, Paola and Rosy from Padova; Alfonso, Cinzia and Daniele from Trentino.

Some of the experiments reported in the thesis have also been submitted elsewhere.

Specifically, the computational model presented in Chapter 3 has been published in Cognitive Systems Research [Grecucci, A., Cooper, R., and Rumiati, I.R. (2007). A computational model of action resonance and its modulation by emotional stimulation. Cognitive Systems Research, 8(3), 143-160], and partially in the Proceedings of the Seventh International Conference on Cognitive Modelling [Grecucci, A., Rumiati, I.R., and Cooper, R. (2006), In D., Fum, F., Del Missier, F., and A., Stocco (Eds.), Proceedings of the Seventh International Conference on Cognitive Modelling (ICCM-06). Trieste, Italy: Edizioni Goliardiche, 88-93]; the ERP experiment of Chapter 4 has been published in Archives italianne de biologie [Authors: Grecucci, A., Buiatti, T., Budai, R., Rumiati, I.R. (2009). The emotional control of imitation. ERP evidence. Archives italienne de Biologie 147(1-2), 37-49]; the study with autistic children of Chapter 5 has been recently submitted to Brain and Cognition [Grecucci, A., Rumiati, R., R. Siugzdaite, Londero, D., Fabbro, F., Brambilla, P. (submitted). Beyond the broken mirror: dissociating action resonance from empathy deficits in autistic children].

Chapter 1
Emotion and action: what has been missing?

"Emotions are action tendencies"
(Frijda, 1986)

1.1 Prologue

Humans seem to have a strong tendency to align their behavior with their fellows during social interactions (Lieberman, 2007). The relationship between emotion and imitation, although it has not been investigated until recently, has always been acknowledged. For instance, de Montaigne (1575), Adam Smith (1759), Nietzsche (1881), and Wittegenstein (1980), speculated on the association between the capacity to empathize and understand or reproduce others' behavior. The functional and neural mechanisms linking imitation to empathy, however, remain still unclear (Iacoboni, 2009). Only recently, a convergence between cognitive, developmental and social psychology on one hand, and neuroscience on the other, has happened.

1.1.1 On the links between emotion and action

While the concept of emotion is largely absent from contemporary theories of action, experts of action are concerned themselves with intentions, wants, purposes, desires, beliefs, plans, and volitions, and account for the nature of action by elaborating these theoretical constructs, they paid little attention to emotions. This is at odds with our common sense folk psychology, in which emotions and moods are frequently used for justifying actions and for predicting and explaining social behaviors.

The original word for emotion was “passion”. It comes from ancient Greek “pathos” and Latin “pati”, which means “passive” and “patient”. The passivity of emotion is usually in contrast with activity, the hallmark of action and agency.

Although not action themselves (Lang, Bradley, and Cuthbert, 1997), emotions influence action and typically emerge in circumstances where adaptive control is required (Ekman and Davidson, 1994). Emotions, therefore, are held to be functional products of Darwinian evolution, developed from primitive actions that facilitate the continued survival of living organism. The biphasic theory of emotions (Lang, 2000) posits that the broad array of emotions experienced and displayed by humans can be organized in two basic motivational systems: the appetitive for life sustaining stimuli, and the aversive for life threatening stimuli. When engaged, both systems affect the behaviour of humans.

Emotions can influence the generation of an action in two ways: the tendency and readiness to act, and the decision to act. Different emotions correspond to different patterns of action. For example, anger usually leads to aggression and retaliation; fear is involved in preparing for rapid escape from a dangerous situation. This led some psychologists to propose that emotions can be defined and identified by different sets of actions tendencies (Arnold, 1960; Frijda, 1986). Action tendencies are states of readiness to execute a given kind of action, involving both bodily arousal and psychological preparation following emotional appraisal. The connection between emotion and action tendency finds a natural

place in various appraisal theories of emotion (Lazarus, 1991). Emotional appraisal is a mental assessment of the potential harm or benefit of a situation relevant to one's personally significant concerns. From the appraisal theory perspective, action tendencies and readiness are natural consequences of a given emotional appraisal of how to cope with a situation. They are also shaped by evolution and adaptation: different emotions serve different survival related goals and therefore prepare the organism for adaptive behavioral responses to the current situation. The neglect of emotions in theories of action is untenable and is likely to be due to misunderstandings of the nature of emotion (Zhu and Thagard, 2002).

According to LeDoux (1996), there are two parallel pathways from emotional stimulus to motor response: the low road connecting directly the sensory thalamus to the amygdala and to the response, and the high road that connects these two systems passing through the cortex

The existence of a fast and direct pathway from the sensory thalamus to the amygdala suggests that emotional responses can act without consciousness and in a fast way in a cascade of automatic, endocrine, skeletomotor, and arousal systems to prepare the organism. These emotional responses are mediated by this fast and direct pathway and can be appropriately called *covert emotional reactions*. However, this is only part of the story for humans. In many circumstances, people can and indeed they are required to instantiate an *overt emotional action* to cope with an emotional stimulus. Little is known about how the human brain organizes the cognitive and control mechanisms which allow "the crucial shift from reaction to action" (LeDoux, 1996). LeDoux suggests that the prefrontal cortex and the basal ganglia are essential for the emotional action. The prefrontal cortex is considered as pivotal for planning decision-making and executive functions. On the other hand, the basal ganglia are heavily implicated in motor control and its interactions with the amygdala may be important in emotional action.

1.2 Attempts to understand the covert emotional *reaction*

In the past years, several studies were carried out to shed light on the motor reactivity following emotional stimulation. Historically, the first proof of the emotion-action coupling was the discovery of the so called fear-potentiated startle reflex. Recently using the transcranial magnetic stimulation, new data have been collected supporting the idea of the influence of emotions on the motor system.

1.2.1 The fear-potentiated startle reflex

The startle reflex refers to a diffuse skeletomotor response resulting from intense stimuli with rapid onset (e.g., a loud noise or an electrical shock). The eyeblink reflex is the first and most stable component of the startle pattern (Davis, 1986), easily measured by recording the electromyogram (EMG) activity of the muscle orbicularis oculi beneath both eyes: the stronger the startle response, the stronger the EMG activity of the eye blink muscle. Extensive animal research has demonstrated that the startle reflex is generated at the level of the brainstem and the neurophysiological pathways involved (in rats) are fairly well known (for a review, see Davis, 1992). The growing interest for the startle as a tool in the study of emotion is due to the fact that the primary startle circuit is not an isolated pathway, but can be modulated by higher brain structures such as the limbic system and the neocortex (Davis, 1992). Vrana, Spence, and Lang (1988) were the first to explore the idea that the startle magnitude can be modified by the ongoing emotional state. They argued that all emotions are primitively associated with a behavioural set favouring either avoidance (e.g., fear) or approach (e.g., happiness). In addition, they reasoned that the emotional response would be potentiated if there is a match of the emotional tone of the ongoing emotion and the emotional tone of a new stimulus; and conversely, they argued that a mismatch would result in an inhibition of the emotional response towards a new stimulus. Thus, the magnitude of the defensive startle reflex should be potentiated in the context of a negative emotional

state but attenuated in the context of a positive emotional state. In line with this, Vrana et al. (1988) have demonstrated that the startle response is reliably potentiated during perception of aversive stimuli (e.g., slides of mutilated bodies) and diminished during perception of pleasant stimuli (e.g., slides depicting nudes). In other words, a match of the (negative) affective tone of the probe (i.e., a sudden loud noise) with the foreground stimulus leads to a potentiation, whereas a mismatch results in an inhibition of the response.

1.2.2 Corticospinal excitability by stimulating the motor cortex

Increases in the corticospinal motor tract (CST) excitability to emotional stimuli have been demonstrated using TMS (Hajcak et al., 2007; Oliveri et al., 2003). Baumgartner and colleagues (Baumgartner, Willi, and Jäncke, 2007) found evidence for increased CST excitability in response to simultaneously presented emotional pictures. Oliveri and colleagues provided evidence that the supplementary motor cortex (SMA) may act as a neural gateway in conveying emotion-related information from the limbic system to the primary motor cortex for initiating action programs. They were able to analyze the excitability of the primary motor cortex (M1) after conditioning TMS of SMA, when subjects saw emotional and non-emotional visually cued movements (click a bottom with the mouse). They found that motor evoked potentials (MEP) were selectively enhanced in the visual-emotional triggered movement condition, compared with a control site condition and with visual-neutral guided movements. Schutter and colleagues (Schutter, Hofman, and Van Honk, 2008) clarified that the same corticospinal excitability can be found following the observation of fearful facial expressions. However, covert motor responses such as startle reflexes or increased spinal excitability may not be representative of the larger domain of overt actions. When we face a threat, this type of reflexes are only the first step; to effectively cope with imminent danger a more complex overt action must also be taken.

1.3 Attempt to understand the overt emotional action

To date, most studies investigating the links between emotions and overt actions have failed to find emotional modulation of overt voluntary action (Lemke, Fischer, Wendorff, Fritzer, Rupp, and Tetzlaff, 2005). For example, Lemke, Wendorff, Mieth, Buhl, Linnemann (2005) developed a paradigm to study simultaneously the influence of emotions on voluntary and involuntary movements. Subjects were shown IAPS positive, negative and neutral pictures. In order to view a slide, subjects had to press a button placed in front of them using their dominant hand. They were instructed to watch the slides as long as they needed to seize their contents. In order to switch a picture off, subjects had to press a second button that was placed in a standardized (60% of subjects arm length) distance to the first. While they were watching the pictures, an acoustic startle stimulus consisting of a 100-dB, 50-ms burst of white noise with instantaneous rise time was presented binaurally over headphones (onset of tone randomized 3-5.5 s following picture onset). The eye-blink component of the startle reflex was measured by recording EMG activity from the orbicularis oculi muscle beneath the left eye using Ag/AgCl surface electrodes. The reaching movements were recorded by an optoelectronic motion analysis system which consisted of two cameras equipped with infrared light emitting diodes (IRED) and video processors. These were attached to the wrist at the styloid process of the radius, the first joint and the nail of the index finger. For every movement different parameters were determined for velocity: duration of finger movement (DFM), duration of hand movement, peak velocity of finger. While for precision, the length of the finger's movement curve, and the length of the hand movement curve. The relative time to peak velocity of the finger and hand were used as parameters for the general performance of the movement. While Lemke and collaborators were able to reproduce the classic

modulation of SE amplitude for the unpleasant context, for which the magnitude increased during aversive stimuli; they failed to find any significant difference on the voluntary movements according to the emotional context. None of the above parameters were shown to be significantly modulated by the stimuli.

1.3.1 Compatibility between emotional words and the self

The links between action and emotions have been the focus of different studies employing the semantic compatibility between emotional words or between emotional self related words and the image of the self. In the spatial domain, an affective version of the Simon effect has been proposed (De Houwer and Eelen, 1998), in which participants were required to give a specific positive or negative response on the basis of a non-affective stimulus category. Despite being asked to ignore the irrelevant affective meaning of the stimulus, participants responded faster when the affective meaning of the stimulus was congruent with the required response (e.g., POSITIVE – positive valence), compared with the condition in which the meaning was incongruent (e.g. Negative valence). De Hower, Crombez, Baeyens, and Hermans (2001) used affective pictures half depicting man-made objects, and the other half natural objects (Experiment 2). Subjects were required to respond saying POSITIVE (or negative) to one category of pictures (for example "man-made objects). The same facilitated response for congruent irrelevant emotional dimension was obtained. In the Experiment 4 of the same study, the authors showed a facilitation of associating approach movement with positive stimuli, and avoiding movement with negative stimuli. In this experiment subjects moved a manikin upwards or downwards in the screen by pressing two keys when presented with adjectives or nouns (with an emotional valence to be ignored by the participants).

A similar facilitation effect was obtained also using an associative version of the affective Simon effect, by using the aversive conditioning technique (Beckers, De Houwer, and Eelen, 2002). Again, responses were guided according to the grammatical category of positive and negative target words: the matching between the irrelevant affective connotation of a target word and the affective valence of the effect produced by the required response resulted in significantly faster performance than non-correspondence.

To study the compatibility between emotional words and the image of the self Chen and Bargh (1999) exposed participants to valenced stimulus words, and they instructed on e group of participants to push a lever away from them if the stimulus word presented was positive and to pull the lever toward them if the stimulus word was negative; the second group received the opposite instructions. Results suggested that positive stimuli were associated with faster pulling of the lever toward the participant, whereas negative stimuli were associated with significantly faster responses when participants pushed the lever away from themselves. The authors speculated that speeded responses occurred when activation of defensive circuitry (withdrawal behaviour) was paired with pushing away the lever and when activation of appetitive circuitry (approach behaviour) was paired with pulling the lever toward the body. A second experiment in which half subjects were asked to just pull for both class of stimuli and the other half to push for the same stimuli, showed that negative compared with positive stimuli led to faster movements. This suggests that the activation of a defensive circuitry globally primes the human organism to move with greater haste. As such, when exposed to negative cues, speeded movement may increase survival rate, supporting the notion that such a functional relationship has an evolutionary advantage to the biological organisms. However, Rotteveel and Phaf (2004) showed that when the conscious evaluation of the stimuli and of the procedure was reduced, the effect was no longer present.

It may be that the effect found by Chen and Bargh (1999) is a voluntary manipulation generated by the subjects.

Coombes, Janelle and Duley (2005) developed a paradigm in which they measured both accuracy (absent in Chen and Bargh, 1999) and speed. Subjects performed a square-tracing task following neutral, pleasant and unpleasant IAPS pictures and they found that subjects were less accurate but faster in the unpleasant condition. In another study, Coombes, Cauraughand and Janelle (2006), required subjects to respond to an acoustic stimulus by initiating and then maintaining a sustained isometric bimanual contraction of the wrist and finger extensor muscles against two independents load cells, after seeing neutral, blank, pleasant and unpleasant pictures. The mean force generation was significantly stronger in the unpleasant condition

Taken together these findings can be explained as being due to a priming - consistency effect rather than being a real modulation of the motor response. The studies I have reviewed typically involve an irrelevant emotional dimensions to activate a congruent or incongruent dimension at the grammatical or semantic level of the stimulus. As De Houwer and colleagues argued, the facilitation and inhibition effects found in these experiments can be explained in terms of priming by means of the activation of the same semantic-conceptual area that facilitates or interferes with the two words or pairings of words-pictures (De Houwer and Eelen, 1998; De Houwer, Crombez, Bayens, and Hermans, 2001; Beckers, De Houwer and Eelen, 2002).

1.4 The social neuroscience perspective: A new look at the links between emotion and actions

Recent research has highlighted the importance of imitative behaviours in a large plethora of emotional and action phenomena such as empathy (Leslie, Johnson-Frey, and Grafton, 2004; Preston and de Waal 2002), the chameleon effect (Bargh, Chen, and Burrows, 1996), fear contagion (de Gelder,

Snyder, Greve, Gerard, and Hadjikhani, 2004), social cognition (Ochsner, 2004), autistic pathology (Dapretto, Davies, Pfeifer, Scott, Sigman, Bookheimer, and Iacoboni, 2006), related to the comprehension and sharing of emotions between subjects by means of motor feature detection or body language. Based on these premises, I suggest that imitative responses represent a good candidate to further investigating the links between voluntary action and emotions.

The discovery of mirror neurons drew an enormous attention to the motor structures. These neurons fire when the animal observes as well as executes an action (Rizzolatti and Craighero, 2004). They were discovered in the ventral premotor cortex area F5 in monkeys, and functional neuroimaging provides evidence that humans have similar mirror neurons. Authors suggested that mirror neurons are the basis of social cognition including emphatic processes (Gallese et al., 2004), claiming that *"the human brain is endowed with structures that are active both during the first- and third-person experience of actions and emotions…thus the understanding of basic aspects of social cognition depends on activation of neural structures normally involved in our own personally experienced actions or emotions".* This means that the understanding of others emotional states passes through a simulation of those states, simulation involving the facial, bodily and visceral aspects.

An example of this is the classic *Chameleon effect* (Chartrard and Bargh, 1999), by which the observer involuntary and automatically reproduces the same facial muscular mimicry when seeing a facial expression. This effect belongs to a general process called *emotional contagion*, by which our body becomes the interpersonal medium of communicating emotional states. Thus, empathy may occur via a mechanism of action representation that modulates and shapes emotional contents (Carr et al., 2003). Carr et al. (2003) for instance tested the hypothesis of the involvement of the same motor and emotional structures for imitating and observing emotional facial expressions. Their prediction was straightforward: if action representation mediation is critical to empathy and the understanding of the

emotions of others, then even the mere observation of emotional facial expression should activate the same brain regions of motor significance that are activated during the imitation of the emotional face expressions. Moreover, a modulation of the action representation circuit onto limbic areas via the insula predicts greater activity during imitation, especially in mirror areas for the overlapping of sensory input and motor planning. As predicted, the authors found that observation of emotional expressions robustly activated premotor and motor areas, that were strongly active in the imitation condition. Preston and de Waal (2002) have suggested that a motor resonance system could play a role in a perception-action model (PAM) of empathy, and that empathy is a part of a larger class of processes that depend on perception-action mechanisms. Activating our own motor representation could allow us also to activate motivations and intentions that are associated with those actions. This "resonance" with another individual can be viewed as a form of "empathy": not only we understand the goals of another person, but we experience their intentions and emotions (Kaplan and Iacoboni, 2006).

Leslie et al. (2003) asked subjects to watch movies of facial expressions (smile or frown) and hand movements (move index or middle fingers) while brain activity was recorded (fMRI). Three different conditions were implemented: passive viewing, active imitation and active motor control. They found evidence for a common cortical imitation circuit for both face and hand imitation, consisting of Broca's area, premotor areas, posterior temporo-occipital cortex, and cerebellar areas. The authors conclude that there may be a right hemisphere mirroring system that could provide a neural substrate for empathy. Unfortunately this experiment did not provide either reaction times or accuracy data, and therefore it is difficult to establish if emotions (empathy for faces) affect imitative tendencies.

The links between action and emotion have been studied also in the context of the emotional body language (EBL) (see de Gelder, 2006, for a review). De Gelder, Snyder, Greve, Gerard, and Hadjikhani (2004) were able to demonstrate that during the observation of emotionally body expressions, there is an

increase in the motor and body representation areas, probably due to the activation of emotion related areas such as the orbitofrontal cortex (OFC), the amygdala and the insula. Even if these studies showed a modulation of the motor related areas as a cause of emotional body specific primes, we still do not know if watching emotional postures in a model may lead us to imitate or empathize with him/her.

One could ask why there should be a link between imitation, which seems strictly related to the action domain and empathy, which is strictly related to the emotion domain. One possibility is that imitation facilitates social interactions, increases connectedness and liking, gets people closer to each other, and fosters mutual care (Iacoboni, 2009). If this is true, it follows that good imitators should also be good at recognizing emotions in other people, which in turn may lead to greater sense of empathy. Thus, this account would predict a correlation between the tendency to imitate others and the ability to empathize with them. This hypothesis was tested in a series of studies. Chartrand and Bargh (1999) asked subjects to choose pictures in a set of photographs. The cover story was that the researchers needed some of these pictures for a psychological test and wanted to know from the subjects which pictures they considered more stimulating. While subjects were choosing the pictures, a confederate was sitting in the same room with the real subject. The confederate pretended to be another subject who was also choosing good stimulating pictures. During the experimental sessions, some confederates deliberately rubbed their nose while the others shook their foot. Subjects were videotaped and their motor behaviour was measured. It was found that the real subjects unintentionally mimicked the motor behavior of the confederate with whom they were sharing the room. Those who shared the room with confederates who rubbed their nose, rubbed their nose more than did subjects who shared the room with confederates who shook their foot. Furthermore, subjects who shared the room with confederates who shook their feet, shook their foot more than did subjects who shared the room with confederates who

rubbed their nose. These results are in line with the idea that imitation is automatic and provide the necessary prelude to the following experiments.

A third experiment tested the hypothesis that the more people tend to imitate others, the more they are concerned with the feelings of other people. The setting of this third experiment was identical to the first experiment. The novel aspect of this last experiment was that the participants responded to a questionnaire that measured their empathic tendencies. The experiment found a strong correlation between the tendency to empathize and the amount of imitative behavior displayed by the participants. The more a subject imitated the confederate, the more that subject was an empathic individual (Chartrand and Bargh 1999). This result suggests that through imitation and mimicry, we are able to feel what other people feel (Iacoboni, 2009). By being able to feel what other people feel, we might also be able to respond compassionately to other people's emotional states (Eisenberg 2000, Tangney et al. 2007).

1.5 Problems

To date, studies have failed to detect the effect of emotions on voluntary action. This might have happened due to two reasons. First, in some studies the tasks did not involve real motor effects that emotions could modulate. TMS studies, the classic startle reflex, as well as the Emotional Body Language studies, did not involve any action to be performed, so their effect on muscle reactivity cannot be explained in terms of a real modulation of the motor system. The second reason is that some studies involved tasks that were not simply motor but involved visual or attentional components. For example, Bradley, Drobes and Lang (1996) found that in a simple button pressing after viewing emotional versus neutral pictures resulted in slow RTs for the emotional stimuli regardless of the valence. This may indicate the effect of attentional capture instead of motor modulation per se.

Another example comes from Lemke (Lemke et al., 2005) who asked subjects to perform movements without a clear motor task, meaning that there was not a task in which action planning or selection can be modulated by emotions.

Moreover, as in almost all the studies we discussed in the previous sections (e.g. experiments of Coombes) did not control for the arousal. It is difficult to disentangle the effect of a real emotional priming of a given motor processing from the mere augmentation of the motor excitability as a cause of arousal.

1.6 Aim of this thesis

The aim of this thesis is to show whether emotions do have an effect on voluntary action, and in particular whether they can influence imitation on their valence. Evolutionary pressure has led the nervous system to guarantee rapid and intense responses to negative dangerous harmful events (Carretiè, Albert, Lopez-Martin and Tapia, 2009). The hypothesis of a "*negative brain"* claiming that the evolution equipped the brain with a wide set of mechanisms *triggered by* and *to cope with* unpleasant or life threatening stimuli, has been proposed (Carretiè et al., 2009). The goal of these mechanisms is to produce rapid and efficient actions directed to avoiding or minimizing negative consequences associated with unpleasant stimulation just perceived. In the words of Carretiè and colleagues "the neural negativity bias led also to a motor negativity bias" (Carretiè et al., 2009), stemming from simple fight-flight mechanisms to very sophisticated motor behaviors.

Along this thesis I asked whether imitative responses belong to the class of *negatively-biased motor behaviors.*

In the present work, the functional mechanism and the neural basis of the emotional modulation of imitation will be discussed providing behavioral, computational, ERP and neuropsychological evidence for it. In particular I will dissect the mechanism by which imitation tendencies are facilitated and I will call this effect Emotional Control of Imitation (E.C.O.I. from now on).

I will demonstrate that the E.C.O.I. has the following features:

- Negative emotions prime imitative response, while this does not apply for positive emotions and non-imitative movements (Study 1, chapter 2);
- It relies upon specific action-resonance mechanisms and comes from the synergy between emotional and motor system (Study 2, chapter 3);
- It is associated with increased cortical movement-related activity and marked by specific Fear Potentiated Movement Related Potentials (Study 3, chapter 4);
- it is broken in abnormal population, namely children with autistic spectrum disorder (Study 4, chapter 5);
- even though it is probably hardwired in our brains, it can be also learned through experience (Study 5, chapter 6).

To overcome the problem of the arousal mentioned in the previous paragraph, I will use a paradigm that implements a control condition in which no effect of emotions should be detected (e.g. incompatible conditions). I will show that the effect of emotions is present only for compatible movements (where imitation takes place), while for incompatible movements is silent. If the E.C.O.I. is an effect of arousal, it should be detected in both conditions. Moreover, to see whether the E.C.O.I. follows the typical linear versus quadric trend, that respectively discriminate between valence and arousal effects, in a control experiment (Study 1), I will use also arousal balanced positive pictures. If

the E.C.O.I. is an effect of arousal, no difference between positive and negative pictures should be detected (but different compared with neutral pictures, showing a quadratic effect, emotional different from neutral, but no difference between emotions). If, on the contrary, the effect of emotions on imitation is valence specific and independent from arousal it should be visible only for one class of emotions (no difference between one kind of emotional and neutral pictures compared with the other type of emotion). I will provide evidence that the latter applies to the emotional control of imitation.

Chapter 2

The emotional control of imitation

2.1 Introduction

Observing actions performed by others induces in us a strong tendency to engage in the same actions ourselves, suggesting strong direct links between perception and action (see Prinz, Aschersleben & Koch, 2009, for a review). For example, we tend to whisper or speak louder when others do, scratch our head upon seeing someone else doing so, walk more slowly in the presence of elderly individuals, cycle faster after seeing a cycling race on TV (Dijksterhuis & Bargh, 2001). This is also known as the *chameleon effect* (Chartrand & Bargh, 1999) and refers to the tendency to mimic other individuals' behaviors without being aware of it. People indulge in mimicry because it can lead to the development of rapport (Chartrand, Maddux, & Lakin, 2005) but also to a number of positive consequences, including liking (Chartrand & Bargh, 1999), trust (Maddux, Mullen, & Galinsky, 2008), closeness to others (Ashton-James, van Baaren Chartrand, & Decety, 2007), and recovery from the experience of being excluded (Lakin, Chartrand, & Arkin, 2008). Others' actions affect one's own even when one acts alongside another person performing not the same but a complementary action (see the "social Simon effect", Sebanz, Knoblich and Prinz (2003).

Suggestions of a substantial overlap between motor and emotional contagion are offered by imaging investigations. Using fMRI, for instance, Carr *et al*. (2003) showed that imitation and observation of emotional facial expressions activated a largely similar network of brain areas. Within this network, there was greater activity during imitation, relative to observation of emotions, in motor as well as in emotional areas. Accordingly, our understanding of the emotions of others appears to be mediated by internal action representations. Recently, the investigation of the neural correlates of perception of emotional body expressions revealed an interplay between dynamic body postures and emotions (Grezes, Pichon & de Gelder 2006; Pichon, de Gelder, & Grezes 2008). The general finding is that motor areas and areas holding body representations are more activated when individuals observed emotional bodily expressions probably due to a modulation of emotion-related areas (see de Gelder, 2006, for a review). This empirical evidence alludes to a plausible interaction between involuntary motor tendencies and emotion, however there is no direct evidence to date that emotional contexts affect voluntary motor behaviour.

Lemke, Fischer, Wendorff, *et al.* (2005), for instance, studied the influence of emotions on involuntary (classic startle eye-blink reflex) and voluntary movements, measured by infrared kinematic tracking of the subjects' reaching hand movements. Viewing color slides of different emotional content produced the startle reflex, thus replicating previous results (Lang, Bradley, Cuthbert, & Patrick, 1993), but had no influence on voluntary movements. Viewing arousing stimuli, independently of their positive or negative valence, modulated the magnitude of motor evoked potentials (MEP) elicited by transcranial magnetic stimulation (TMS) delivered to the subjects' primary motor cortex (Hajcak et al., 2007). In a different study, fearful faces, but not happy or neutral one, were found to selectively increase TMS-elicited MEP magnitude (Schutter, Hofman & van Honk, 2008). Importantly, however, these studies showed influences of emotional processing only on involuntary behavior.

These different lines of research suggest that there are closer links between motor action and emotions than previously thought. To date a clear demonstration of their interplay has not been offered. However, this is an important fact to establish as the ability to generate adequate behavior depending on emotional contexts is a key component of social competence.

In the current study, we investigated the influence of the emotional context on action imitation. We used a modified version of the paradigm developed by Brass *et al.* (2001). In their study, subjects were pre-instructed to either lift or tap the index finger as soon as they saw a lifting or a tapping finger movement on the screen. In one block, subjects' response was always tapping, and in another it was always lifting, independently of whether the movement they saw was tapping or lifting. Brass *et al.* (2001) found that when seen and performed movements were the same (i.e. compatible trials), RTs were shorter than when they were different (i.e. incompatible trials). Brass et *al.* (2001) concluded that human adults have a strong tendency to imitate and suggested that action imitation can be examined as a special case of S-R compatibility.

2.2 Experiment 1

In Experiment 1, we modified the Brass *et al*.'s paradigm in two ways. First, we evoked an emotional context by presenting pictures with emotional significance prior to the stimulus movement. Second, subjects were told to begin their response when the movement of the animated hand was completed. Waiting until the prime movement has reached completion before beginning to respond is a standard procedure in imitation studies (see Rumiati & Tessari, 2007, for a review). To elicit an emotional context and observe its effect on subjects' imitative performance, negative, positive, or neutral pictures were presented as primes prior the stimulus movement to be imitated. The emotional prime pictures used in this and in the following experiment belong to categories whose meaning is

closely related to real life situations in which responding promptly is vital like, for example, attacks from conspecifics or non-conspecifics.

Emotional stimulation was expected to enhance the compatible effect (i.e., shorter RTs), and more specifically, negative primes were expected to produce a stronger effect than positive or neutral primes. It has been suggested that negative stimuli rapidly prepare the organism for actions associated with fight-flight behaviour, whereas positive stimuli, although they have inviting and rewarding properties, are less likely to induce the same response.

2.2.1 Methods

Participants. Twenty-one subjects, 11 male and 10 female, with mean age of 28.06 (SD ± 3.87) years, and mean education of 18.69 (SD ± 2.75) years, took part in the study. They were all right-handed, had normal or corrected-to-normal vision, were naïve to the study, and were paid for taking part to the study (three euros).

Stimuli and apparatus. Neutral (n = 50) and emotional (n = 50 positive and n = 50 negative) color photographs were used as primes, taken from the *International Affective Picture System* (IAPS, Lang & Öhman, 1988). The stimuli used in this study were chosen based on their arousal ratings (i.e., how much a stimulus activates the body) and valence ratings (i.e., how pleasant or unpleasant a stimulus is). The selected neutral pictures had a mean valence of 5.08 and arousal of 2.76, the negative pictures had a valence of 2.27 and mean arousal of 6.15, and the pleasant pictures had a mean valence rating of 7.73 and mean arousal of 6.05. That is, negative and positive primes were closely matched with respect to arousal ($t(1,49)=1,473$ $p= 0.231$) but differed substantially with respect to valence. Notably, arousal and valence were independent and did not covary (for negative $r=-0.179$ and for

neutral r=-0.118). Perceptual and semantic features of the stimuli were balanced across stimulus categories.

The experiment was controlled by a Pentium III computer, with 800 MHz and 512 MB RAM, graphic card Matrox G550 of 32 MB, and Monitor of 100Hz, and E-prime software was used for presenting the stimuli and for data acquisition.

Procedure. In each trial, first the prime picture (neutral, positive or negative) was presented for 1,500 ms on the computer screen, followed by a digitized video-sequence of five frames showing an animated hand presented for 1,105 ms. The first frame, identical for the tapping and lifting movements, lasted for 500 ms; the following three frames, lasted for about 40 ms each[1], and the last one, with the final position of the finger, lasted for 500 ms. The interstimulus interval was a blank screen that lasted for 1,500 ms (see Figure 1 (A)). Overall finger displacement was about 2° for the lifting and for the tapping movement. The animated hand mirrored the orientation of the subject's right hand, which was kept on a small platform on the table in front of her/him. The experiment contained 150 trials, 100 in the emotional and 50 in the neutral condition, presented in a random order. Subjects were told to perform the pre-instructed movement (i.e. tapping) when the presentation of the movement on the monitor was completed. Subjects' RTs were recorded from the onset of the last frame of the animated hand until they pressed the bar.

After signing the informed consent form, subjects began to perform a 20 trials training session to correct the hand position and the onset of the movement. Responses to these trials were excluded

[1] Due to hardware and video limits in the refresh rate , in order to have an image presentation of 40 ms, the e-prime time presentation for these frames was set to 35 ms.

from the analysis. At the end of the training session, subjects performed the experiment proper, for a total of 150 trials.

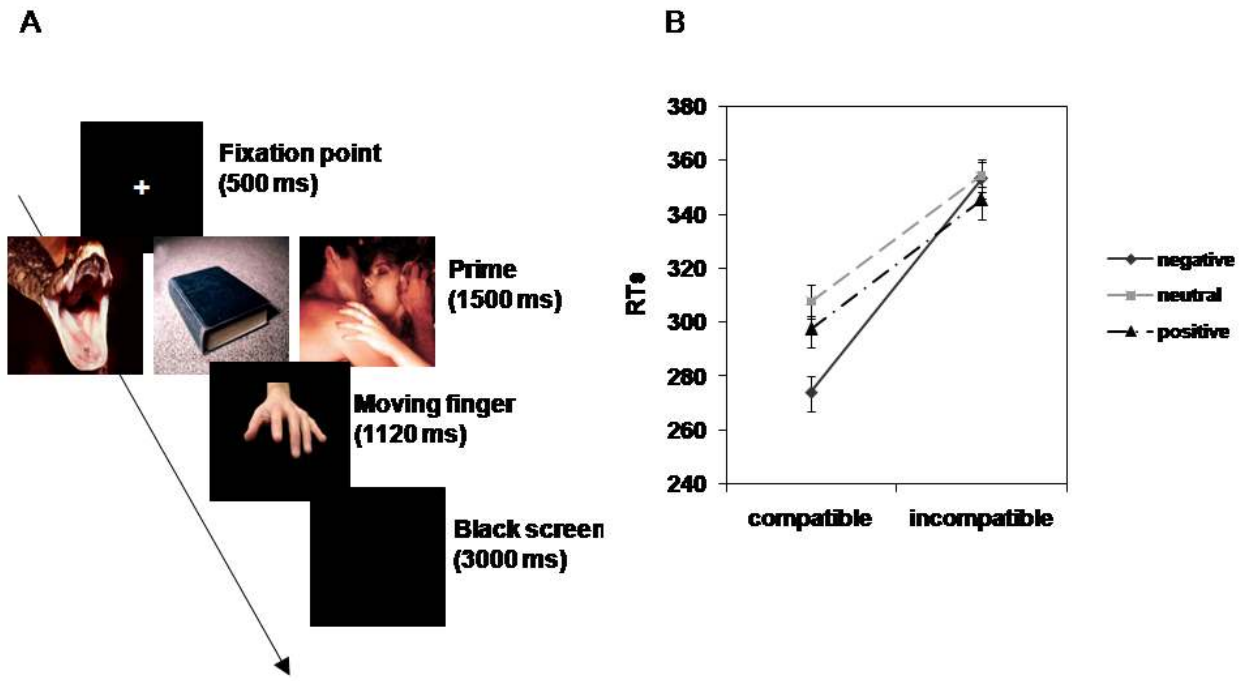

Figure 1.

Experiment 1. On the left the sequence of events. The emotional trigger lasts for 1500 ms., a neutral or an emotional picture (three examples of IAPS pictures are shown). Tapping movement execution of the preinstructed movement (tapping in one block, and lifting in the other). On the right, the results. Subjects are faster to respond after the negative emotional stimulation with respect to the neutral and the positive emotional condition. Error bars stand for standard errors.

Design. The independent within-subject variables were compatibility (compatible vs. incompatible) and prime (negative, neutral, positive). In a compatible trial, seen and performed movements were the same (i.e. tapping-tapping), and in an incompatible trial, seen and performed movements were different (i.e. lifting-tapping). The dependent variable was mainly RT, even though accuracy was analyzed, too.

2.2.2 Results

Participants' accuracy was very high (98%). These data entered a 2 x 3 ANOVA with compatibility (compatible vs. incompatible) and prime (negative, neutral, and positive), but none of the factors was significant (Compatibility, F(1,20)=0,29, p=0.59; Prime, F(1,20)=0,004, p=0,99; Interaction F(2,40)=0,017 p=0,98).

For RT analysis, we excluded RTs below and above 2 standard deviations as outliers. Table 1 shows mean RT as a function of compatibility and prime. A 2 x 3 analysis of variance (ANOVA) with compatibility (compatible vs. incompatible) and prime (negative, neutral, and positive) on RT revealed a main effect of compatibility, $F(1,20) = 17,15$, $p < 0.001$, and of prime, $F(1,20) = 3,46$, $p < 0.05$, but most importantly also an interaction of compatibility and prime, $F(2,40) = 6,74$, $p = 0.005$. T-tests (2-tailed) showed that inside the compatible condition negative trials significantly differed from positive trials ($t(20) = -3.138$, $p < 0.005$) and from neutral trials ($t(20) = 2.855$, $p < 0.010$), whereas RTs did not differ between positive or neutral trials ($t(20) = 0.128$, $p < 0.899$). Inside the incompatible condition the difference between negative vs neutral, negative vs positive and positive vs neutral did not reach significance (p>0.1). The differences between the primes inside the compatible condition compared with the absence of difference between the same primes inside the incompatible condition, resulted in different slopes (e.g. magnitudes in the compatibility effect) (79 ms in the negative condition, 46 in the

neutral and 47 in the positive). To test for these differences three T-Tests were run, one for each compatibility effect (difference between compatible vs. Incompatible trials). These returned to be significant (t(20)=-4,23 p=0.001 for the neutral trials, t(20)=-4,11 p=0.001 for the negative, and t(20)=-3,43 p=0.005 for the positive).See Figure 1 (B) and Table 1for details.

	Prime		
Condition	Negative	Neutral	Positive
Incompatible	353 (116)	354 (118)	345 (122)
Compatible	274 (76)	308 (101)	298 (88)
Compatibility Effect	79	46	47

Table 1. Mean RT (standard deviations) as a function of compatibility and prime in Experiment 1.

2.2.3 Discussion

The results of Experiment 1 supported the hypothesis that imitative tendencies, measured as compatibility effect, would be enhanced by emotional stimulation and that negative primes had a stronger facilitation effect than positive or neutral primes. The compatibility effect was larger when subjects were presented with negative primes than with positive or neutral primes. Moreover, inspection of RTs revealed that the increased compatibility effect after negative primes is largely due to shorter RTs on compatible trials, suggesting facilitation of compatible actions rather than increased interference in incompatible actions. Note also that the prime conditions were balanced with respect to arousal, suggesting that the facilitation effect was produced by the valence of the stimuli in the compatible condition.

Studies on animal behaviour report associations between anxiety disorders and alterations of systems sustaining defensive behaviour (Blanchard, Griebel, & Blanchard, 2001; Blanchard, Hynd, Minke, Minemoto, & Blanchard, 2001). Normally, viewing aversive stimuli can trigger a “fight-flight

system" (McNaughton & Corr, 2004) that allows animals to avoid incoming threads. However, anxiety gates the behavioural reactions to shift from active defensive behaviour (fight or flight) to inhibition and immobilization ("freezing") (Blanchard & Blanchard, 1988). These contrasting behavioural tendencies might have influenced the subjects' latencies in Experiment 1, with subjects with low-anxiety traits being faster in responding to negative stimuli, and subjects with high-anxiety traits being slower in responding to the same stimuli. However, in Experiment 1 we could not establish whether the subjects' performance was affected by anxiety as we did not collect any information concerning subjects' emotional reactivity.

2.3 Experiment 2

Experiment 2 was aimed to confirm the findings of Experiment 1 and to examine the potential role of affective traits such as negative affect and anxiety on the emotional modulation of the compatibility effect. To this end, we administered the PANAS (Positive Affect and Negative Affect Scale, Watson, Clark, and Tellegen, 1988) score to relate the size of the compatibility effect and its modulation to the anxiety level of the subjects. The Negative Affect scale of PANAS will be used to this aim since it correlates with the anxiety traits of subjects (Crawford and Henry, 2004). In this experiment, we employed only negative and neutral primes because Experiment 1 showed that the compatibility effect did not differ between neutral and positive primes.

2.3.1 Method

Participants. Twenty-three right-handed individuals (13 female), with a mean age of 24 years (SD ± 4) and 15 years of education (SD ± 3) took part in this experiment. They were all tested for the

level of negative affectivity (which correlates with anxiety traits) using the PANAS scale (Watson & Clark, 1984).

Stimuli, procedure, and design. Stimuli and procedure were the same as in Experiment 1, except for the following modifications. We employed only negative and neutral primes. The experiment was divided into two blocks. Each block contained 100 trials, 50 negative and 50 neutral primes. See Figure 2 for further details. Subjects were required to perform a pre-instructed movement, which was finger tapping in one block and finger lifting in the other block. Within each block, the order of trials with negative and neutral prime was randomized for each subject, whereas the order of the two blocks was counterbalanced across subjects. Subjects were told to begin their response at the offset of the prime movements. Due to the exclusion of positive primes, Experiment 2 had a 2(compatibility) x 2(prime) design. In a compatible trial seen and performed movements were the same (i.e. tapping-tapping or lifting-lifting), and an incompatible trial seen and performed movements were different (i.e. lifting-tapping or tapping-lifting). As the distinction between lifting and tapping responses was not critical, it was not maintained in the analysis.

2.3.2 Results

Two subjects were excluded because they reported to be under psychotropic treatments. Participants' accuracy was very high (98%). These data entered a 2 x 2 ANOVA with compatibility (compatible vs. incompatible) and prime (negative, neutral) but none of the factor were significant (Compatibility, F(1,20)=1,24, p=0.27; Prime, F(1,20)=0,120, p=0,73; Interaction F(1,20)=0,139 p=0,71). For RT analysis (see Table 2), RTs below and above 2 standard deviations were excluded. A 2 x 2 ANOVA on RTs with compatibility and prime (negative vs. Neutral) as within-subject variables obtained an effect of compatibility, $F(1,20) = 12.757$, $p < 0.005$, whereas the effect of prime just failed

to reach significance, $F(1,20) = 2.88$, p = 0.105. Most importantly however, like in Experiment 1, the interaction of compatibility and prime was significant, $F(1,20) = 4.849$, $p < 0.05$. A t-test showed that the compatibility effect was larger following negative primes than following neutral primes (61 ms vs. 39 ms; $t(20) = 2.202$, $p < 0.040$). See Figure 2 (B) and Table 2.

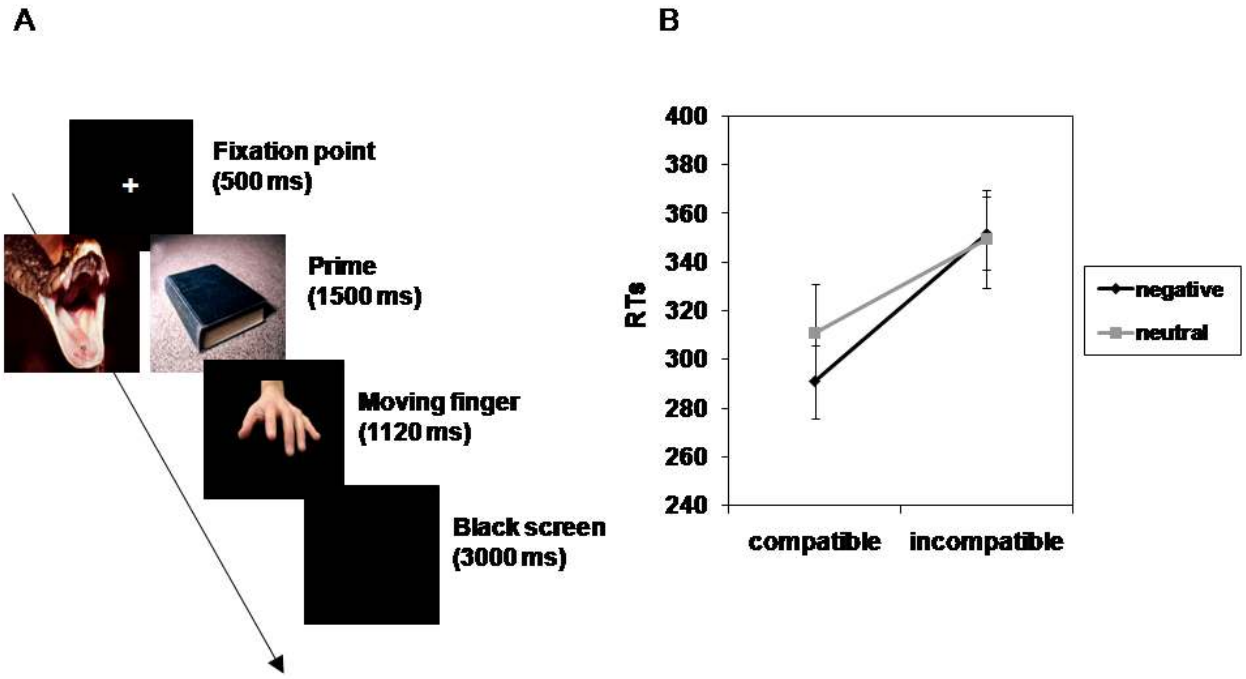

Figure 2.
Experiment 2. On the left the sequence of events. On the right, the results. Subjects are faster to respond after the emotional stimulation with respect to the neutral condition. Error bars stand for standard errors.

	Prime	
Condition	Negative	Neutral
Incompatible	352 (120)	349 (121)
Compatible	291 (121)	311 (120)
Compatibility Effect	61	39

Table 2. Mean RT (standard deviations) as a function of compatibility and prime in Experiment 2.

To explore the effect of anxiety on task performance, scores of the negative scale of the PANAS were entered as covariate with RTs, with the same design as the previous ANOVA. This returned a significant main effect of Prime ($F(1,18) = 5{,}691$, $p < 0.05$) and a trend for the Prime x PANAS interaction ($F(1,18) = 3{,}021$, $p = 0.099$), suggesting that anxiety may influence subjects' performance depending on the type of pictures that were presented with. To further explore the effect of anxiety, we also calculated two correlations between RTs for compatible negative and compatible neutral trials and the negative scale scores of PANAS. For negative trials, we found that the more anxious the subjects the slower they responded, the less anxious the subjects the faster they responded ($r = 0.44$, $p < 0.05$, 1-tailed); for incompatible trials there was no significant correlation between anxiety traits and performance ($r = 0.35$, $p = 0.124$, 1-tailed).

2.3.3 Discussion

The main finding in this experiment was the compatibility effect: subjects were faster in compatible than in incompatible trials. In Brass et al. (2001), subjects began responding while viewing the movement on the screen and the compatibility effect they observed may be caused by spatial correspondence between observed hand and subject hand. In the present experiment, subjects

responded only after the animated hand stopped its movement, a delay that is often used in imitation studies (see Rumiati & Tessari, 2007, for a review). Yet, even when participants reproduced a movement that was no longer visible, they still showed facilitation in the compatible trials.

This suggests that the imitation version of the SRC as shown in this study may rely on an action resonance mechanism. In fact, the interference between seeing a movement and performing the opposite one may derive from a "space overlap" between seen and executed actions, and not from a simple overlap between dimensions as the level of the stimulus nor between the stimulus and the response (see the computational model in the next chapter for a testing of this hypothesis). The second important result is that the response latencies to the emotional primes were shorter than those to the neutral primes. This effect is not specific for every action but it is specifically related to compatible/imitative movements. In fact, the two factors do interact ($p < 0.05$), and simple effect analysis showed that this effect is specific for the compatible condition and that it is related to the increase of the magnitude of the advantage in executing imitative movements versus non imitative movements in the emotional condition (the difference between compatible vs. incompatible emotionally primed movements is of 61 ms versus the 38 ms of the neutrally primed responses). The emotional context affects the motor system by increasing the cohesion between the organisms and their conspecifics, speeding up imitation. The emotional or neutral context does not affect the RTs for incompatible movements.

The results show that anxious participants responded more slowly in the emotional condition than non-anxious subjects. A positive correlation between larger RTs (slower response in the emotional relative to the neutral condition) was also found. This finding can be interpreted according to the McNaughton and Corr's model (2004). In normal (non anxious) subjects the hypothetical "fight-flight system" enhances motor responses to avoid aversive stimuli, while in subjects with anxiety a

"behavioural inhibition system" inhibits action. Probably the presence of anxiety in mammals gates the behavioural reactions to shift from the active defensive behaviour (fight or flight) to inhibition and immobilization (the so called "freezing"). However, the ANCOVA did not returned a significant interaction, so the role of negative affect and of anxiety in imitation remains an open issue.

Categories of stimuli and their emotional content (valence and arousal) affected the RTs as well. The pictures eliciting the strongest effect on RTs (from 40 to 70 ms.) depicted, in order of importance, mutilations followed by animal and human attack. If the defensive system is the product of evolutionary pressures to cope with aversive stimuli, and given that these aversive stimuli were principally attacks from organisms of non conspecific (animal attack from the human perspective) or conspecific (human attack), the activation of the defensive system should be maximally potentiated by these stimulus categories. Regarding the images of "mutilation", seeing dead and mutilated members of our own species must have a relevant impact on the defensive system since it is critical for the organism to get ready for a possible predator (that killed the members of its group) or an imminent danger.

2.4 Experiment 3

If the enhancement of the motor response following the emotional stimulation observed in Experiment 1 is specific to the imitation condition, in which the interaction between body parts is critical, then whether the model is human or not should make a difference. Some studies have already shown the so-called animacy effect for imitation, whereby normal subjects show a reduced imitative tendency when the movement is shown by a robotic model (Pierno et al**,** 2007; Press, Gillmeister and Heyes, 2006). The enhancement observed in the compatible condition after the emotional stimulation (Experiment 1), could be somewhat reduced when the stimulus is no longer represented by a finger.

Subjects reproduce what the model does when his/her action represent the best strategy, but for this identification between the model and the imitator might depend on the former being a human being. To test this hypothesis I required subjects to perform a tapping or lifting movement following the presentation of a dot moving upward or downward (with the same trajectory and size of the tip of the finger as shown in Experiment 1), in a neutral or emotional condition. In this way I controlled also for the spatial component of the task which is still maintained by the upward and downward movement of the white circle.

2.4.1 Method

Participants

18 subjects, 9 females, with mean-age of 26 years (SD ± 4) and 13 years of education (SD ± 5), took part in the study. All were right-handed, had normal or corrected-to-normal vision, were naïve to the study, and were paid for taking part to the experiment (3 euros). Subjects were interviewed to assess medical and psychological conditions, and were administered two tests to assess the 'negative affectivity' (PANAS by Watson & Clark, 1994). Anxious subjects were *a priori* excluded from the experiment. After signing the informed consent form, they began to perform 20 training trials and then the experiment.

Design and procedure

Design and procedure of Experiment 3 were exactly as in Experiment 2 except that the animated hand was replaced by a moving white dot with a circle of diameter of 1.5 cm. See Figure 3 (A) for details.

Stimuli

The same emotional stimuli as in Experiment 2 were used. The white dot was obtained with nine frames presented in sequence: one in the center, four in the space above, and 4 in the space below, in order to match the movement of the index finger of the animated hand.

2.4.2 Results

Participants' accuracy was of about 98%. These data entered a 2 x 2 ANOVA with compatibility (compatible vs. incompatible) and prime (negative, neutral), but none of the factor were significant (Compatibility, F(1,17)=0,004, p=0.94; Prime, F(1,17)=1,13, p=0,30; Interaction F(1,17)=0,15 p=0,69).

The ANOVA on RTs was computed with prime and compatibility as factors. The main factors of Compatibility returned to be significant (F(1,17)=18.721, p 0.001), though Prime F(1,17)3.871, p 0.066, and the Compatibility x Prime interaction F(1,17) = 0.284, p=0.6 were not. See Figure 3 (B).

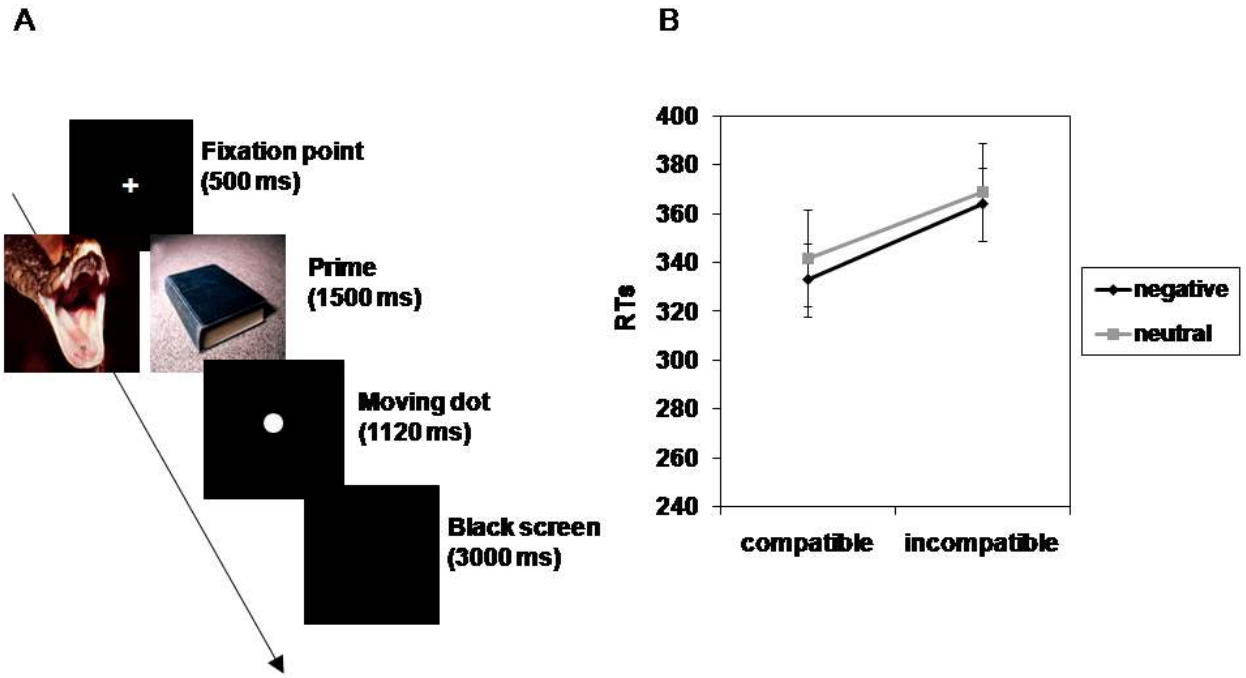

Figure 3.
Experiment 3. On the left the sequence of events. The moving finger was replaced by a moving dot. On the right, the results. No effect of emotions was detected as expected, though a smaller size of spatial compatibility was obtained.

2.4.3 Discussion

Experiment 3 was designed to test whether the effect found in experiments 1-2 of this chapter was due to the human interaction that occurs during imitation. In fact, visual observation of human actions provokes more motor activation than observation of robotic actions (Pierno et al., 2007; Press, Gillmeister and Heyes, 2006). Consistently with previous results, removing the animated hand from the paradigm used in Experiment 1 (substituted by a moving white dot), the effect of the facilitated imitative response following emotional stimulation disappeared, even though a compatibility effect was

maintained. This was probably due to the spatial component of the task that is hard to disentangle from the imitative task as shown by Heyes and Rey (2004).

2.5 Experiment 4

The effect found in Experiment 1 (emotional enhancement of motor response) may not be specific to imitation, but can be generalized to other tasks requiring a motor response. To test this hypothesis we required subjects to perform a simple reaction time task following the emotional stimulation using the same stimuli, timing and apparatus of Experiment 1. I aim to test whether this effect could be detected also with a different motor task. If this is not the case, probably the failure of previous researches was probably due to the task used to study this issue.

2.5.1 Method

Participants

Twenty five subjects, 12 males and 13 females, with mean-age of 24 years (SD ± 5) and 15 years of education (SD ± 3), took part in the study. All were right-handed, had normal or corrected-to-normal vision, were naïve to the study, and were paid for taking part to the experiment (3 euros). Subjects were interviewed to assess medical and psychological conditions, and were administered two tests to assess the 'negative affectivity' (PANAS by Watson & Clark, 1994). Anxious subjects were *a priori* excluded from the experiment. After signing the informed consent form, they began to perform 20 training trials and then the experiment.

Design and procedure

Design and procedure of Experiment 4 were exactly as in Experiments 2 except for the animated hand, since here subjects saw a red circle turning to green. In each trial there were the following events: the prime picture (neutral or unpleasant) was presented for 1500 ms on the computer screen, followed by a red circle that appeared in the centre of the screen for 1000 ms, at which point it became green lasting for 120 ms. Once the color changed the subjects executed the preinstructed movement (tapping and lifting). Between stimuli a blank screen was inserted for 1500 ms. See Figure 4 (A).

Before beginning the experiment, subjects performed 20 training trials which were excluded from the analysis. The experiment was divided into two blocks, the order of which was counterbalanced across subjects: in block 1, subjects were required to lift the index finger as soon as the red circle becomes green, after viewing neutral or negative emotional context images; in block 2, they were required to move the finger downwards (finger tapping) . Each block contained 100 trials, 50 for the emotional and 50 for the neutral condition.

Stimuli

The same emotional stimuli as in Experiment 1 were used. The red and the green circles had a diameter of about 2 cm. and they were presented in the centre of the screen.

2.5.2 Results

Accuracy (99%) entered a simple paired t-test and resulted to be not significant (t(1,24)= -0,216 P=0.830. A simple ANOVA was computed with Prime as the main factor that turn out not to be significant ($F(1,24)=1,587$, $p = 0.220$). See Figure 4 (B).

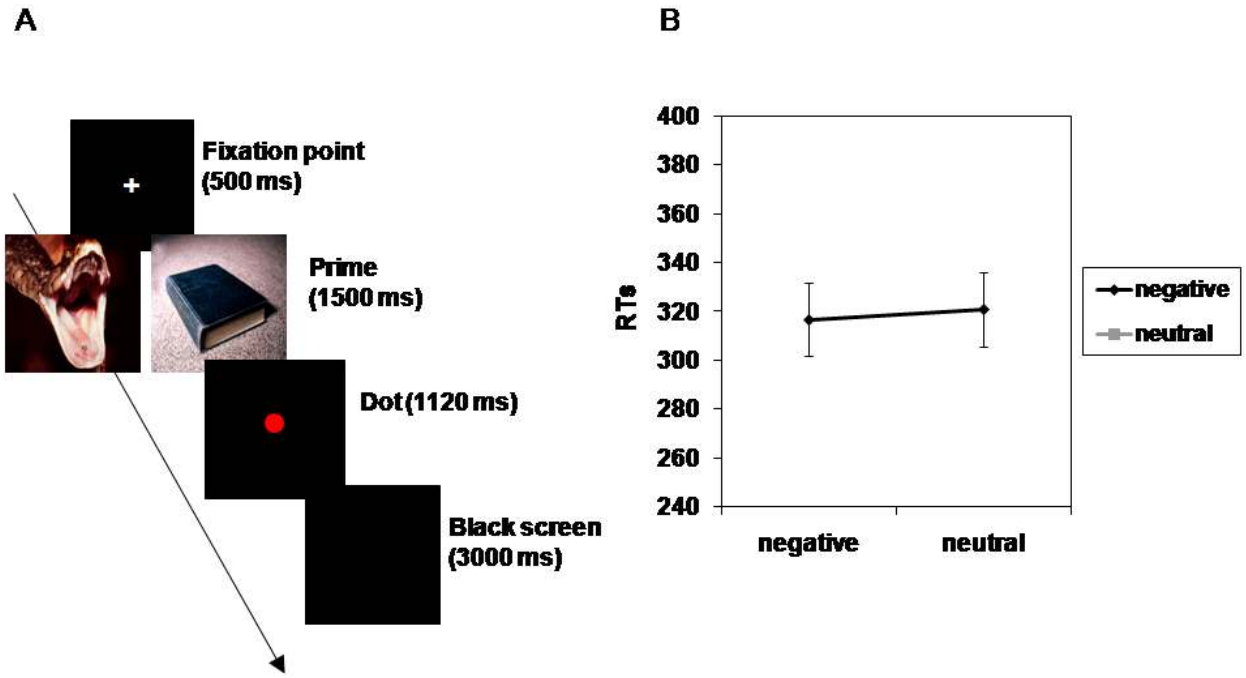

Figure 4.
Experiment 4. On the left the sequence of events. The moving finger was replaced by a still green dot that becomes red, in a simple reaction times task. On the right, the results. No effect of emotions was detected.

2.5.3 Discussion

In Experiment 4, subjects performed a simple reaction times task following the emotional stimulation with the same stimuli, timing and apparatus as in Experiment 2, to test whether the effect found in Experiment 2 was specific to imitation or is a general response of the motor system

No differences were found between the RTs in the emotional and those in the neutral condition. We can conclude that the emotional enhancement of motor performance is confined only to the

stimulus response compatibility or imitation domain (since only the compatible movements significantly differ, see Experiment 1), and not to the action domain in general (since the simple motor task of Experiment 4 fails to detect any significant difference).

An alternative view is to consider the effect of emotional enhancement of performance as a more general effect concerning the whole action domain which is detectable only with certain kind of tasks (like the imitation task of Experiment 1), that act like a "magnifying glass" through which the emotional enhancement of action long failed to find in behavioural experiments, becomes visible.

I hypothesize that (aversive) if emotions enhance the areas involved in the task (Simpson et al., 2000; Paradiso et al., 1999, in this case consider the motor areas), there is a double modulation of the motor system in the task employed in Experiment 1, the first coming from the enhancement of the perceived movement representation, and the second from the enhancement of the representation of the to be executed movement.

2.6 Experiment 5

The present experiment was designed to test whether the effect found in Experiment 2 was also extendible to an on-line imitation paradigm (e.g. imitating while observing the movement). In the previous experiments subjects started their movements at the offset of the movement (off line imitation). In contrast, in the original Brass paradigm (Brass et al, 2001) subjects performed their movement while observing the animated hand.

To test for an online imitation we asked subjects to perform their movement at the same time of the observed movement, using the same design, stimuli and apparatus of Experiment 1.

2.6.1 Method

Participants

18 subjects, 9 females, with mean-age of 23 years (SD ± 2) and 14 years of education (SD ± 2), took part in the study. All were right-handed, had normal or corrected-to-normal vision, were naïve to the study, and were paid for taking part to the experiment (3 euros). Subjects were interviewed to assess medical and psychological conditions, and were administered two tests to assess the 'negative affectivity' (PANAS by Watson & Clark, 1994). Anxious subjects were *a priori* excluded from the experiment. After signing the informed consent form, they began to perform 20 training trials and then the experiment.

Design and procedure

Design and procedure of Experiment 5 were exactly as in Experiment 2.

Stimuli

The same emotional stimuli as in Experiment 2 were used.

2.6.2 Results

Data entered a 2 x 2 ANOVA with compatibility (compatible vs. incompatible) and prime (negative vs.neutral), but none of the factor were significant (Compatibility, $F(1,17)=2,216$, $p=0.155$; Prime, $F(1,20)=3,663$, $p=0,95$; Interaction $F(1,20)=1,590$ $p=0,224$).

2.6.3 Discussion

In this Experiment I tested whether the effect of emotions on imitation was detectable even in case of an online imitation following Brass et al (2001) study. Unfortunately, this manipulation failed in reproducing the E.CO.I. effect. This may be due to the fact that subjects knew in anticipation the movement to perform (e.g. preinstructed) and that they started without taking into consideration the movement of the animated hand. This strongly reduced the imitative advantage effect (no effect of compatibility) as well as the emotional effect (no effect of Prime).

2.6 General discussion

In this study I evaluated the influence of the emotional context on action and in particular that of a fearful scenario (as seeing pictures of mutilations, dangers or animal and human attacks) on a compatibility/imitative task as revealed by reaction times. I proved for the first time, that the emotional and the social contexts significantly affected the normal imitative tendency humans show when primed by a model.

In Experiment 1, subjects were faster in performing a motor task involving an imitative version of the stimulus-response compatibility effects after the negative emotional stimulation (pictures taken from IAPS) only. Positive pictures did not affect the performance and behaved in the same way as neutral pictures. One problem that could rise with this experiment is that to balance the arousal of the positive picture we were forced to use several erotic pictures which can have distracted subjects from the execution of the task. We are currently replicating this experiment using emotional facial expressions which do not have this problem. Unfortunately, the interaction was not significant and even if simple effects ANOVA showed that the only difference was between the neutral and the fearful compatible movements, I cannot conclude that the two factors were interacting. By this experiment I

was able also to exclude that the effect found in Experiment 1 is simply related to the arousal of the stimuli which are believed to improve the performance. Obviously arousal plays a role in the effect found, but it's not limited to this. Experiment 2 was carried out to replicate Experiment 1, controlling for possible confounding factors such as psychological and medical conditions of the subjects that could have clouded the results. Since positive pictures were not affecting the response and were problematic in their interpretation, we decided to take them apart and to focus our attention to neutral versus fearful only pictures. The results in this experiment showed a significant interaction between imitative movements and the emotional context: subjects were faster in responding in the compatible imitative trials only in the emotional condition. Moreover, the size of this effect significantly correlated with the arousal and valence dimension of the emotional stimuli: the most threatening stimuli yield to faster responses. This size effect is probably due to the parsimonious principle according to which the biological organisms are subjected to: the force of the output should be a function of the intensity of the stimulation, the stronger the input the stronger the response. A further analysis showed that subjects with low-level anxiety were faster to imitate (larger difference between emotional and neutral conditions), while high-level anxiety participants were slower to imitate (smaller differences between the two conditions). This seems a direct consequence of anxiety because it is supposed to slow down overt responses by freezing the behavior and increasing the attention to the environment. Several psychiatric disorders include alterations of motor behaviour as symptoms, like for instance the motor inhibition in anxiety disorders (McNaughton and Corr, 2004), motor slowing in depressed patients (Lemke et al., 2000), or impulsive behaviour in borderline personality disorders. Two control experiments were ran to see what happens if instead of the response compatibility task, the subjects are required to perform a spatial reaction times task not involving imitation (Experiment 3) or a simple reaction times task (Experiment 4).

In Experiment 3, the moving hand was substituted with a moving dot keeping constant the same timing and movement of Experiment 1 (the dot replicated exactly the same positions of the finger tip). Even if a smaller compatibility effect was found, probably for the spatial interference associated with the moving dot, no effect of emotions was detected. I concluded that the effect found in Experiment 1 and 2 is specific for the action domain, and not belonging to aspecific spatial effects. It might also be that an interaction with human is necessary to show the E.C.O.I.

In Experiment 4 a simple reaction time task was carried out in which subjects were required to perform a tapping or a lifting after a cue (red circle turning to red), without observing compatible or incompatible movements. The failure to reproduce the effect found in Experiment 1 is probably due to the fact that the compatibility task used in Experiment 1 is more sensitive to the simple reaction time task used in Experiment 4. The effect found in Experiment 1 is specific to the imitation domain and not generalizable to other kinds of actions.

Finally, Experiment 5 failed in reproducing the E.C.O.I. effect in an online imitation paradigm.

In agreement with recent studies (de Gelder, 2006) I can hypothesize that emotions can influence action by a bottom-up enhancement of the ventral and dorsal stream, which are sensible to the spatial and motor properties of movements. In particular several physiological studies found increased activation in cortical visual areas when processing emotional stimuli in respect to neutral ones (Simpson et al., 2000; Pardiso et al., 1999). Junghoefer et al., (2001) in an ERP study found the greater dipole strength for emotional visual stimuli in the occipital and occipito-parietal cortical areas. The parietal cortex is involved in action programming. The amygdalar activation can “boost” activation in this area and produce facilitation in the processing of the movements to execute. This issue has been explored in a computational model in Study 2. It is known from animal studies that electrical stimulation of the amygdala is responsible for the release of a complex pattern of behavioural and

autonomic responses which resemble those elicited by the natural context that allow animals to respond to threatening stimuli (Misslin, 2003; Amaral, et al., 2002; Davis, 1992). This structure, together with the *bed of the stria terminalis* (the rostral extension of the amygdala), seems to control the activation of the hypothalamic-pituitary-adrenal axis (Herman and Cullinan, 1997). This fast and phylogenetically primitive neural pathway alerts the organism and prepares it for action according to the evolutionary preparedness hypothesis (Seligman, 1971). The motor system is enhanced probably through the backprojections of the amygdala similar to what has been found in the visual areas (Amaral, et al., 2002; Davis, 1992).

Magnuson and Gray (1990), hypothesized that the release of norepinephrine onto motoneurons via amygdala activation of the locus coeruleus, or via amygdalar projections to serotonin-containing raphe neurons, could lead to enhanced motor performance during a state of fear, because these neurotrasmitters both facilitate the excitation of motoneurons (Davis, 1992; McCall and Aghajanian, 1979; White and Neuman, 1980). Actually there are no proves of these hypothesis, but we know something of how the amygdala interacts with the posterior areas of the brain, that can tell us something about our experiment.

Several authors (Tellegen, 1985; Shaver et al., 1987; Ortony et al., 1988; Lang et al., 1998) have argued that emotion's motivational organization has a biphasic structure: pleasant emotions are associated with an appetitive system associated with approach; unpleasant emotions engage the defensive motivational system, primary associated with withdrawal, escape and attack. In this study we wanted to explore the defensive system in its proper behavioural features. In the light of these experiments we strongly believe that negative emotions are involved in *action readiness* to avoid or to face a threatening stimulus. From an evolutionary point of view it seems not plausible that brain evolved modules to worsen the performance of an organism as previously discovered in various

psychological, psychophysiological and imaging experiments (Keil et al., 2005; Lemke et al., 2005; Northhoff et al., 2004; Hartikainen et al., 2000; Tipples and Sharma, 2000; Isenberg et al., 1999, Drevets and Raiche, 1998). Our conviction is that, using experiments with less cognitive interference, and more related to the motor system, researchers would be able to find an advantage of action readiness caused by emotional activation.

In conclusion, we have seen that negative emotions which are supposed to engage the defensive motivational system (Lang et al., 1997), trigger the subject to respond faster. This enhancement of action was theoretically supposed by all the evolutionary theories of emotions, but it was very difficult to prove its existence by a series of problems discussed previously. With this experiment we started shedding some light on this important evolutionary function.

Chapter 3

A computational model of action resonance and its modulation by emotional stimulation

3.1 Introduction

In the previous chapter we provided evidence of the influence of emotions on action. The aim of this study in this chapter is to understand the mechanisms underlying both the imitation facilitated response (as a particular case of a broader Stimulus-Response Compatibility effect), and its enhancement by emotional stimulation.

Stimulus-Response Compatibility (SRC) effects have been extensively studied within the spatial domain. In a choice reaction time task, participants are faster to respond when the stimulus and the appropriate response key are on the same side of the body than when they are on opposite side, even when stimulus location is irrelevant to the response. This effect, known also as Simon effect (Simon, 1969), persists when different effectors are used (hands or feet), and even when hands are crossed (so the left hand operates the right response key, and the right hand operates the left response key, see Kornblum, 1992; 1994). More recently, SRC effects have been shown to arise in the context of

imitation. Brass, Bekkering, and Prinz (2001) found that participants were faster when performing the same finger movement (e.g., lifting or tapping) as the seen movement, relative to when the seen and the response movement were different. This result was interpreted as deriving from the activation of the representation of the observed movement which interferes with the activation of the representation of the to-be-executed movement (Brass et al., 2001). Grecucci, Koch and Rumiati (submitted) modified Brass et al.'s (2001) paradigm by requiring subjects to begin their response after the observed movement, and found an imitation effect comparable with the SRC effect of Brass et al. (2001) (Experiment 1). Importantly, this imitation response can be modulated by negative emotional state (Grecucci, et al., submitted): subjects were faster when they responded after seeing unpleasant emotional pictures compared with neutral pictures. The aim of the present study is to investigate the computational mechanisms underlying the SRC effect in the context of imitation and its emotional modulation.

3.1.1 Direct mapping and action resonance

Kornblum and colleagues (Kornblum, 1992, 1994; Kornblum et al., 1990; Kornblum and Lee, 1995) have proposed a "dimensional overlap" model to account for a series of effects in which there is an overlap of some dimensions at a given processing level. According to this model, behind these effects, there are two types of dimensional overlap: stimulus-stimulus (SS) overlap – similarity between two stimulus dimensions (such as color and meaning in the Stroop task), or stimulus-response (SR) overlap – similarity between a stimulus dimension and a response dimension (such as spatial position of the stimulus and spatial position of the response effector in the Simon task). In the action domain, the motor version of the SRC effect has been related to the *direct-matching hypothesis* of Prinz (1990) – also called action resonance mechanism hypothesis (Iacoboni et al., 1999) – according to

which the observation of somebody executing an action leads to the activation of an internal motor representation in the observer. This representation may then be used to execute (i.e., imitate) the same action. Moreover, observing an action facilitates its execution because perception and action planning share a common representational code (e.g., Prinz, 1990; 1997). This formulation derives directly from the ideomotor theory of James (1890) and Greenwald (1970), and is supported by more recent behavioural (Brass et al., 2000; Prinz, 2002), brain imaging, and neurophysiological studies (for a review, see Heyes, 2001). As to the last point, neurons have been found in area F5 (di Pellegrino et al., 1992; Gallese et al., 1996) and in the inferior parietal lobule (Fogassi, Ferrari, et al., 2005) of the monkey brain discharging not only when the animal performs an action but also when it observes the same action. Altogether, these neurons constitute the so-called Mirror Neurons System (MNS). Transcranial Magnetic Stimulation (TMS; Fadiga, Fogassi, Pavesi and Rizzolatti, 1995) and imaging findings (Iacoboni et al., 1999) have also been taken as evidence of a human analogue of the MNS observed in the monkey brain. Action observation appears to activate a complex network of areas (occipital, parietal and temporal and premotor cortices) that overlap with the network activated by action planning and execution (Buccino et al., 2001; Iacoboni et al., 1999; Nishitani and Hari, 2002). Moreover some imaging studies suggest the existence of a somatotopic organisation for observed movements in the premotor cortex (Buccino et al., 2001; Wheaton et al., 2004). Thus action observation seems to share not only the same computations but also the same neural basis as action production (for a review, see Brass and Heyes, 2005).

Iacoboni et al. (1999) scanned subjects using fMRI while they were performing a task adapted from Brass et al. (2001), and found that the blood oxygenation level (BOLD) of the left inferior frontal cortex and the right superior parietal lobule correlated with the observation condition only, and that this background activity was potentiated when they had to imitate the observed

movement as it summed up with the activity of the observed movement. These authors argued that there is in the brain an action resonance mechanism that directly maps a kinematic description of the observed action onto an internal motor representation of the same action.

To our knowledge, this hypothesis has not previously been tested in a formal model. What remains to be explained is why executing a movement is facilitated by prior observation of the same movement (as in imitation), and why it is relatively more difficult to reproduce incompatible movements relative to the one just observed. We suggest that the imitation version of the SRC derives from a response-response (RR) overlap.

3.1.2 Computational accounts of SRC and related effects

To date there are two main computational attempts to explain SRC effects. The first was produced by Zorzi and Umiltà (1995), who proposed a neural network model of the Simon effect. In this work the Simon effect arises from the conflict between discrepant stimulus and response spatial codes in the response system by means of a competitive mechanism, implemented via lateral inhibition. The spatial code (which refers to the left or right position of the cues, with one node for each position) and the non-spatial code (i.e., the nature of the two cues to be discriminated, with again one node for each type) converge in the response system (which consists of two competing nodes), where they may conflict. In the case of incompatible trials, the position of the node inhibits the incongruent but correct node response.

The second model was proposed by Zhang, Zhang and Kornblum (1999). It attempts to explain all SRC effects as derived from the Simon task, as well as further SRC effects that may occur in the Stroop and flankers tasks. This model is based upon the dimension overlap model, assuming that stimuli and responses are represented in terms of dimensions and features and that the concept of

dimensional overlap is crucial. The stimulus and response are represented respectively by the input and the output modules, each of which consist of units, representing different features. An intermediate layer represents abstract concepts (such as the name of the color in the Stroop task), and projects to the output (response) layer by means of specific "control lines" that represent prewired (and weighted) SR mappings. Mutual inhibition allows nodes within the same module to compete. The model assumes that with the SS overlap the two modules at the input layer converge onto a common module at the intermediate level causing eventual conflicts for opposite features implemented in the input layer (such as the word "RED" written in green ink). In case of SR overlap, the conflict arises from the connections between the intermediate layer and the output layer.

Zorzi and Umiltà's (1995) model of the Simon effect does not generalize directly to the imitation SRC effects found by Brass et al. (2001) and Rumiati and Grecucci (submitted). Spatial cues have very little influence on the effect observed in these studies. We argue that the imitation version of SRC (Rumiati and Grecucci, submitted), as well as the interference effects reviewed by Blakemore and Frith, (2005), cannot be accommodated within these models as they emphasize a conflict mechanism at the stimulus level or at the stimulus-response level. In contrast, we believe that the interference phenomena observed in the context of imitation could be better explained by exploring the motor programming or the response level. There exist other models of SRC related effects, such as the Cohen and Huston (1994) model of Stroop task, however they are rather remote from the classes of phenomena discussed here, and are based on the same principles at the core of Zhang et al.'s model (Zhang, Zhang and Kornblum, 1999).

In the action domain, Oztop and Arbib (2002) produced a computationally explicit model of the circuitry for visually guided grasping of objects (the MNS1 model) in order to explain the role of mirror neurons in grasping. They used a feed forward neural network with one hidden layer which was

trained to map between a representation of the visual history of an object/hand interaction and a representation of the action being performed. The latter was identified with the "action recognition schema", a theoretical construct which itself was identified with the mirror neurons in F5. The learning task of the MNS1 model involves back propagation error driven learning in which the output produced by the network, the "mirror response", is compared to the target, the "motor program". The network has to change its weights to approximate its output vector to the appropriate target vector corresponding to the type of grasp. The MNS1 model has not been applied to the imitation task in which the motor SRC effect has been demonstrated. Critically for the current context, Oztop and Arbib (2002) did not implement a specific action resonance system. Thus, the MNS1 model operates in a strictly feed-forward manner with no recurrent connections. There is therefore no sense in which representations evoked in the model's output layer may resonate with, or experience interference with, representations of existing actions.

3.1.3 Emotion and the motor system

There are many outstanding questions concerning the interaction of emotions with the motor system. It is not yet well understood what happens in the motor areas of the brain when we are exposed to aversive stimuli, or how we can "read" emotions of others by means of the information conveyed by whole-body postures. According to the perspective we favour (Frijda, 1987; Ohman et al., 2001), emotions evolved in order to improve the adaptation of the organism in its environment by providing it with fast harm avoidance. More precisely, aversive stimuli cause the engagement of a hypothetical defensive system whose function is to trigger the actions that help to avoid the danger.

So far most psychological and psychophysiological studies have failed to find a modulation of overt action triggered by aversive stimuli that activate the defensive motivational system (Lemke et al.,

2005). One exception is the study of Rumiati and Grecucci (submitted), as reported in chapter 2, in which subjects were asked to look at very unpleasant or neutral picture and then to perform a tapping or lifting movement after the observation of a compatible or incompatible movement. Rumiati and Grecucci (submitted) found that when the emotional stimulation was followed by an explicit motor task, participants were faster to respond, possibly due to the engagement of a defensive system which causes the enhancement of action – a mechanism selected by evolution for survival reasons. These results cannot be explained as a priming effect, since the emotional pictures served only to elicit an emotional state in the subjects, and were not related in any way with the response (tapping or lifting).

The emotional modulation of action due to aversive stimuli is not the only link between action and emotions. Recent data from imaging studies has highlighted the influence of emotions on action production and understanding (e.g., in emotional contagion, the tendency to feel and express the mood of others, and the chameleon effect, the unconscious tendency to mimic the facial and postural expressions of others; see Carr et al., 2003, and Leslie et al., 2004), and conversely the importance of action for emotion understanding (e.g., in empathy and social cognition; see de Gelder et al. 2004; Hadjikhani and de Gelder, 2003). Thus, Preston and de Waal (2002) have suggested that a motor resonance system could play a vital role in a perception-action model (PAM) of empathy. According to this theory, the same premotor neurons that are involved in the generation of facial expressions are also involved in recognizing those of others (Leslie et al., 2004). Imaging studies seem to support the existence of an "emotional action resonance system" which activates for both emotional (face) expression and observation (Carr et al., 2003). Moreover Leslie et al. (2004) suggested that a common imitation circuit would be active during both face and hand imitation in a task which combined hand imitation with face emotional expression imitation (in a similar fashion to the Rumiati and Grecucci experiment in which emotional visual stimuli and hand stimuli to be imitated were combined).

3.1.4 Emotion and task demands

Evolutionary theories of emotions (Lang, Davis and Ohman, 2000) hold that emotions trigger action in the case of aversive stimulation, but as the mammalian species evolved, the primate brain became equipped with a more sophisticated control system able to guide, by planning and also by inhibition, action in a more sophisticated way. However, it is not yet known how the emotional behavioural repertoire interacts with this higher level cognitive control of action. There is in fact a fracture in the literature about how emotions can modulate task performance in laboratory settings: on one hand there are data supporting the "synergistic view" (Ito, Larsen, Smith, and Cacioppo, 1998; Sato, Kochiyama, Yoshikawa, and Matsumura, 2001; Simon-Thomas, Role, and Knight, 2005), according to which emotional stimulation facilitates task performance (as demonstrated by faster RTs); on the other there are data supporting the "competitive view" (Drevets and Raichle, 1998; Northoff, Heinzel, Bermpohl, Niese, Pfenning, Pascual-Leone, and Schlaug, 2004) according to which emotional stimulation impairs performance (as demonstrated by slower RTs).

We suggest that this fracture can be resolved by considering the role of task instructions in the experimental setting. In particular, we suggest that if the task is very demanding in terms of cognitive resources, RTs will slow down because of interference between emotional stimuli (which capture resources via automatic processes, see Ohman, 1991), and cognitive instructions, while if the task is less taxing (as the motor task of Rumiati and Grecucci, submitted), and the subject does not have to keep in mind complicated instructions, an advantage in processing after emotional stimulation is shown (as suggested by evolutionary theories of emotions, see Grecucci, Koch and Rumiati, submitted).

3.1.5 The current study

The current chapter addresses the above issues by presenting a computational model of an SRC effect in the context of imitation and its modulation by negative emotional state (Grecucci, Cooper, Rumiati, 2007). The model accounts for the main effect of compatibility as found by Brass et al. (2001) and Rumiati and Grecucci (submitted) and the interaction of compatibility and negative emotional state. In addition, by varying task demands, the model illustrates how both competitive and synergistic processing following negative emotional stimulation may arise. The remainder of the paper begins with a discussion of the main assumptions of the model and their implementation followed by a detailed description of the model and a number of simulation studies. The simulations demonstrate the criticality of each assumption and the effect of varying task demands. The general discussion considers the implications of the modelling work with respect to both the theoretical arguments and the related existing models discussed above. Some anatomical considerations are also discussed.

3.2 Basic assumptions of the model and their implementation

The model we will present here is based on four key assumptions concerning the neural mechanisms underlying the motor version of the SRC effect and its modulation by emotional state. This section presents and motivates those assumptions and discusses their implementation in the model. Three assumptions concern the basic motor SRC effect in a fixed (neutral) emotional state, and we shall consider them first. The fourth assumption concerns the modulation of the motor SRC effect by negative emotional state.

The ideo-motor theory proposes that observation and execution of an action share the same neural representations and, supposedly, overlapping brain regions (Greenwald, 1970). Accordingly, executing an action activates an internal motor representation in the observer, which in turn is then used to execute (i.e., imitate) the same action. Several behavioural studies (Blakemore and Frith, 2005;

Brass et al., 2001; Prinz, 2002) have demonstrated that movement execution is faster when preceded by observation of a congruent movement than when it is not. Furthermore, neuroimaging studies (Decety and Grèzes, 1999; Grèzes and Decety, 2001) have demonstrated that the passive observation of action is sufficient to activate the same motor areas involved in actual execution. Blakemore and Frith (2005) argued that the interference for incompatible movements in these tasks may reflect interference within a common neural network (a resonance mechanism) that encodes both the observed and the executed movements. In our simulation the assumption that this resonance mechanism may have a possible neural candidate in the "mirror" system (di Pellegrino et al., 1992) is implemented by having the same layers of units activated for both the *observation* of a movement and the *execution* of the same movement. We will refer to this as the "Action Schema" layer.

The second assumption of the model is that *observing a movement facilitates the subsequent execution of the same movement*, i.e., produces a *priming effect*. Thus, *viewing* an action (e.g., tapping) activates the neurons of the brain region corresponding to that action; the activation of this region will facilitate the subsequent *execution* of the same action as the region will be still slightly active. Within an artificial neural network, priming can be implemented by an "activation-based" memory. Activation-based memory refers to the persistence of a slight state of activation in the artificial neurons of a region previously activated. This contrasts with "weight-based" memory, i.e. memory that is permanently stored in the weights of the units (though subjected to modification by experience), or in the synapses in the real brain (O'Reilly and Munakata, 2000). Our simulation adopts the activation-based memory approach. Thus, viewing an action (e.g., tapping) activates the units of the region corresponding to that action which in turn will facilitate a subsequent flow of activation in the same areas for the execution of the same movement (on compatible trials). This region is facilitated in becoming re-excited because of the residual prior activation. Iacoboni (2005) proposed that a motor

resonance system (possibly implemented via mirror neurons) may be active during both action observation and action execution, and that this neural activity sums additively during imitation (Iacoboni et al., 1999). The overlap of neural activity for compatible imitation may be the basis of this motor priming effect. This mechanism is implemented in our system with a graded activation spreading function of the units and by bidirectional connectivity that enables representations to remain active even in the absence of externally derived excitation (O'Reilly and Munakata, 2000). In sum, the state of activation of units following action observation carries over and affects the activation of units during subsequent action execution.

The third assumption is that *the activation of a representation inhibits other "similar" representations*. This refers to the competition for activation between near regions of the cortex and is implemented in the model with intra-layer competition through *k-winners-take-all* (kWTA) dynamics (Majani, Erlarson and Abu-Mostafa, 1989), which causes dynamic competition between k-units and the other units within a given layer. A kWTA function ensures that no more than *k* units out of *n* total in a layer are active at any given time (O'Reilly and Munakata, 2000). Within the model presented here, this is obtained by computing a uniform level of inhibitory current for all units in the layer, providing a computationally effective approximation to biologically plausible inhibitory dynamics. (See Appendix for further details). In sum, an incompatible movement is more difficult to perform because seeing a movement (e.g., tapping) activates the corresponding region (which we shall call "schema 1"), while performing a different movement (e.g., lifting) requires the activation of the region corresponding to that second movement (which we shall call "schema 2"). This region competes with the region for tapping (schema 1) which is still slightly activated and still inhibiting the lifting region (schema 2). For this reason there is a disadvantage when executing incompatible movements.

The above three assumptions, i.e., a common layer representing both observed and to-be-

executed movements, persisting activation of the observed movement, and the inhibition dynamic between observed schemas and the actual schema to be executed, will all affect the network performance. Specifically, we hypothesise that these three assumptions jointly account for the facilitation of action in the compatible trials and the impairment of action in the incompatible trials.

We also hypothesize that the emotional modulation of the SRC effect in imitation is sustained by the amygdala. This structure responds to threatening, aversive inputs with fear reaction outputs (Misslin, 2003). While it is still not clear how the amygdala influences action, it has been argued that threatening stimuli might have priority as a result of an evolutionary optimisation of processing stimuli that might pose an immediate threat to survival (Taylor and Fragopanagos, 2005). It has also been suggested that the amygdala alerts the organism by "boosting" a better and deeper encoding of relevant stimuli (Anderson and Phelps, 2001), and several physiological studies have found increased activation and greater dipole strength in occipital and temporal areas when processing emotional stimuli compared with neutral ones (Junghöfer et al., 2001; Pardiso et al., 1999; Simpson et al., 2000). It seems that the core computational processing of the amygdala is the enhancement of the areas involved in a task. Our hypothesis is that emotions can influence action by enhancing the regions involved in action representation and execution. Within the model this hypothesis was implemented by introducing extensive reciprocal bidirectional connections between units in the amygdala layer and those in the action representation layer, forming a closed and reverberating loop between these areas (Amaral, Price, Pitkanen, and Carmichael, 1992).

3.3 Description of the model

3.3.1 Network architecture

The model (see Figure 1) is a multi-module neural network comprising five layers. In the input layer both the movement and the emotional information associated with a trial is encoded. This layer consists of a matrix of 2 x 7 units with one set of units representing observed actions (tapping and lifting) and another executed actions (also tapping and lifting). The input layer is connected to an "action representation" layer (where sparse representations of movements are developed). This layer consists again of a matrix of 2 x 7 units with one set of units representing observed actions and another one executed actions. The action representation layer is connected, in turn, to an "action schema" layer (where observation and execution are effectively collapsed into schemas for each action). This layer consists of a matrix of 2 x 10 units in which the two movements are coded: one representation for the observed and executed tapping, and one for the observed and executed lifting).

The action representation layer is then connected to the output layer (only 2 units). The input layer also feeds into an "amygdala" layer (2 x 7 units), which is trained to detect emotionally marked stimuli and which, when such stimuli are detected, boosts the action representation layer. All layers are bi-directionally connected except for the input to action representation layer and the input to amygdala layer. The bidirectional connections provide the model with powerful attractor dynamics in which the contributions of each layer are balanced during action observation and execution to reach an attractor state.

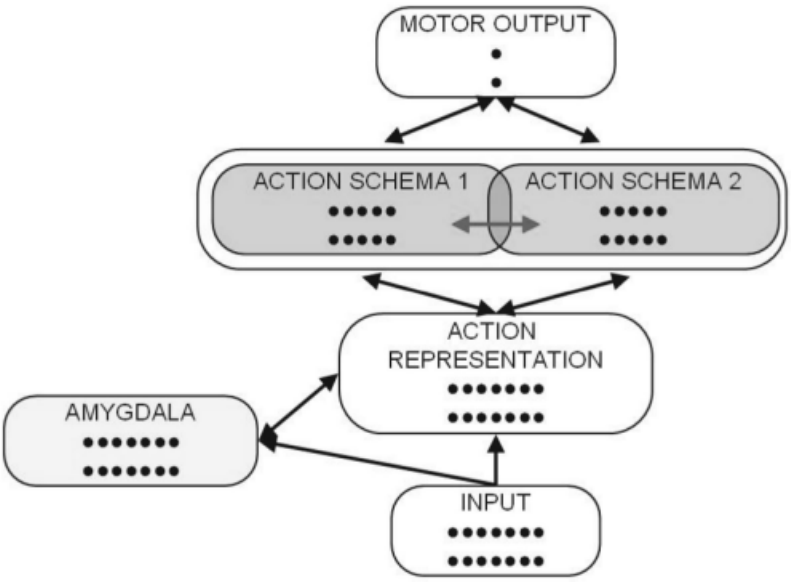

Figure 1
Architecture of the network. The inputs from the retina are sent in parallel to two routes: A) the visuo-motor stream consisting of ACTION REPRESENTATION and then the two ACTION SCHEMA to analyze the type of the movement B); the "AMYGDALA" which responds to the emotional trials with a boost in activation of the ACTION-REPRESENTATION/ACTION-SCHEMA modules.

We postulate two separate layers for action implementation because we assume that there are at least two separate neural representations of actions; one more conceptual (semantic), the other one in which the actual motor program is implemented (Rumiati, Zanini, Vorano, and Shallice, 2001). We test the relevance of this assumption below by demonstrating that the SRC effects do not occur when only one layer is used for action representation.

The choice of 2 x 7 units for the main layers of the net was chosen to allow implementation of topographic properties across layers. Within the PDP++ modelling environment, in which the model was developed, this was done by means of a specific connection type: the OnetoOnePrjnSpec for all layers except for those between the action schema and the action representation layer, for which we used the TesselPrjnSpec. This latter type of projection leads to tessellated (repeated) patterns. The

combination of connection types create “receptive field” like patterns which respect the spatial position of the sending units into the receiving ones across layers.

3.3.2 Stimulus and response encoding

To simplify the implementation of the task, the input module is divided into four regions: the upper part codes for the lifting movement, the lower part for the tapping; the observed movement is coded in the left column (lifting in the upper part, and tapping in the lower part), while the executed movement is coded in the right column (again lifting in the upper part, and tapping in the lower part). A separate unit of the input module encodes the emotional valence of the trial, as described further below. Thus, in the compatible neutral trials, say a lifting, a unit will be activated in the left-upper part of the input module during observation of the movement, while a unit in the right-upper part will be activated to signal the execution of the same movement. For the incompatible trials, the two active units (one for the observation, the other for execution) will occupy two different regions of the input module (one in the upper part for the lifting, the other in the lower part for the tapping).

The emotional valence of the stimuli is represented in the input layer by a unit which is initialized to a value of 1.0 for emotionally marked trials and 0.0 for the neutral trials. In the behavioural experiment, emotional stimulation (emotionally neutral or emotionally negative pictures) was presented prior to action observation on each trial. In this simulation for simplicity the emotional stimulus is presented “in parallel” with the action observation, in the sense that the two movements (a tapping and a lifting) are labelled with this “emotional-valence-unit”. As described below, in the trained network activation of this unit causes activation of the amygdala module.

The output is represented by two units: one that functions as a “no go” signal when the network just observes the movement, and one as a “go” signal to execute the action when the network

is required to execute the movement. A single trial proceeds as follows. First, an input unit corresponding to an observed action is activated. The network then settles (as a result of training, as described below) into a stable state with the "no-go" output unit activated. An action is then presented to the execute units of the input module, signalling that the network is to execute the given action. The network's response time is the number of cycles between presentation of this input and activation of the "go" output unit.

3.3.3 Learning algorithm

The type of network is a multi-modular network which shows attractor dynamics restricted to the execution of each trial by means of recurrent connections between each module. Learning and activation dynamics employed O'Reilly's LEABRA (Learning in an Error-driven and Associative, Biologically Realistic Algorithm: O'Reilly, 1998) formalism. This algorithm was selected for its biological plausibility and its flexibility in implementing large scale cognitive but biologically well-grounded models. It combines principles from several network architectures and learning algorithms, including those of binary and continuous Hopfield network (Hopfield, 1982; 1984), the deterministic Boltzmann Machine network (Ackley, Hinton, and Sejnowski, 1985), the interactive activation and competition network (McClelland and Rumelhart, 1981), and the GRAIN approach (Movellan and McClelland, 1994).

LEABRA is based on a point-neuron activation function (that models the basic electrophysiological properties of real neurons in producing an output activity based on net input excitation), with *k*-Winners-Take-All inhibition (that achieves sparse distributed representations and competing emerging mechanisms by means of inhibitory interconnections, similar to the effect of real inhibitory interneurons) and a plausible version of error-driven learning (performed using a variant of

contrastive Hebbian learning, similar to the Boltzmann machine: Ackley et al., 1985). Within the LEABRA approach, a network settles in two phases: a plus phase, where the output units are clamped at their target values and a minus phase, where the network's actual output is produced. Weight changes are based on the difference in synaptic activation across these two phases:

where x_i and y_j are the activations of unit i in layer x and unit j in layer y respectively.

In addition, weight adjustments include a Hebbian term (so weights between correlated units tend to be increased) and a mechanism to ensure that all weights are bounded between zero and one.

3.3.4 Training

The network learned to represent the movements in the action representation layer with a localist code: one unit per tapping observed, one unit per tapping executed, one unit per lifting observed, and one per lifting executed. This symbolic representation was obtained in the action representation layer with a k-Winners-Take-All algorithm with $k = 1$ (so one unit wins against the others), while in the input layer it was obtained by simply initializing it with single-unit coding.

In the action schema layer the network learned to develop only one representation for each movement, regardless of whether it was an observed movement or an executed movement (thus implementing the action resonance mechanism). A k-Winners-Take-All dynamic was implemented within this layer with $k = 4$. This resulted in the two movements (tapping observed/executed and lifting observed/executed) being represented by two orthogonal groups of four units, with each group competing for activation.

The network was trained in two separate sessions: in the first session it was trained on the

basic stimulus-response compatibility task, while in the second session the amygdala layer was trained to respond to fearful stimuli. Two separate training sessions were used to ensure that the model was able to perform the basic stimulus-response compatibility task in the absence of aversive stimulation.

During stimulus-response compatibility training, the network was presented with the following pairs of input-output associations: 1) an observed movement in the input layer and the "no go" unit in the output layer; then an executed *compatible* movement in the input layer and the "go" unit in the output layer; 2) an observed movement in the input layer and the "no go" unit in the output layer; then an executed *incompatible* movement in the input layer and the "go" unit in the output layer. All configurations were presented in each training epoch and the network was trained for 1000 epochs, by which time the sum-square error had fallen to less than 0.02.

The amygdala module was trained to respond to the emotionally marked stimuli presented in the input layer. Thus, the connections between the input and amygdala units were trained with two patterns. In the first, the emotion input unit and all amygdala units were set to 1.0, in the second the same units were set to 0.0. For the purposes of this study we consider the capacity of the module to discriminate a stimulus as aversive to be an innate and hard-wired property. Hence we were not interested in how the amygdala module becomes able to recognize a stimulus as emotionally marked. The role of this layer is to send its activation to the action representation layer, only in emotional trials. This additional activation should help units overcome the threshold faster, resulting in a deeper basin of attraction and fewer processing cycles for settling of the network.

3.3.5 Analysis of performance

Simulating performance on a single trial involves presenting a stimulus, corresponding to an observed action, to the input layer of the model and allowing it to settle into a stable (attractor) state. A

stimulus corresponding to an *executed* action is then presented to the input layer and the number of cycles taken by the model to settle to a stable state is recorded as the key dependent variable. For emotionally valent trials, the emotion unit in the input layer was also set, as described above.

Bidirectional propagation of activation constrains the flow of information between layers, ensuring that all modules contribute to the model's final attractor state. We assume that the number of cycles needed for the network to reach the attractor state, is proportional to the time required by human subjects to process the task. Additional time is required by human subjects to process the stimuli and execute the response, but we assume that these times are constant across conditions and hence that differences in the number of cycles for the network to settle may be directly related to differences in subject reaction times.

3.3.6 Anatomical observations

The action resonance mechanism, represented in our model by the action schema layer in which the representations for observed and executed movements are collapsed in the same representation (i.e., same units active) is hypothesized to be localised in the right temporo-parietal lobe in which, according to Iacoboni et al. (1999), a kinaesthetic copy of a movement is formed during movement observation. This is then used during action execution. While the precise map of the analogue for humans of the mirror system of monkeys (in F5) is not yet well understood, several studies have reported the overlapping of regions implemented in our model. The action representation is hypothesised to be in the more temporal areas, in which the semantics of the movement is coded. Hypothetically the Superior Temporal Sulcus may be the site for biological movement recognition (Jellema and Perrett, 2002). The amygdala module corresponds anatomically to the Amygdaloid complex which, in the case of emotionally valent stimuli, enhances the other layers' activation (the

action representation module). The amygdala module together with the action representation module may be considered as a unitary *emotional action resonance system* that specifically activates in all the situations involving action during emotional stimulation in a social context (such as empathy, social contagion, etc.).

3.4. Simulations

3.4.1 Simulation Experiment 1: Normal Performance

3.4.1.1 Methods

The aim of simulation Experiment 1 was to replicate the results obtained by Rumiati and Grecucci (submitted) concerning stimulus response compatibility and the modulation of action execution by negative emotional state. On the basis of the above assumptions, we reasoned that the network would be able to reproduce both the stimulus compatibility effect and the emotional modulation of action execution. The network was tested for 30 blocks, with each block consisting of four simulated trials: compatible-neutral, incompatible-neutral, compatible-emotional, incompatible-emotional. Noise added to the membrane potential within the PDP++ environment was set to 0.0005.[2]

3.4.1.2 Results

Mean response times (in cycles) are plotted for each condition in Figure 2 (left), while the

[2] Some level of noise is required within the LEABRA environment to resolve competition between representations within a layer (O'Reilly, 1998). The main effects of compatibility and trigger reported below are still present at higher levels of noise (e.g., at the LEABRA default level of 0.005), though with higher levels of noise the interaction between those factors is weaker.

human data is reproduced in Figure 2 (right). Comparison of the model's behaviour with human performance on the task reveals several qualitative similarities. The model captures the basic SRC effect in the emotionally neutral condition, and the modulation of the effect by prior aversive stimulation is in the correct direction (i.e., in both the model and the human data responses are generated more quickly following aversive emotional stimulation). The primary effects obtained in the empirical studies were replicated. Thus, a repeated measures 2 x 2 ANOVA with "Compatibility" (compatible versus incompatible) x "Trigger" (emotional versus neutral) as factors found significant main effects of "Compatibility" ($F(1, 29) = 580.453$, $p < 0.001$) and "Trigger" ($F(1, 29) = 396.664$, $p < 0.001$), and a significant interaction ($F(1, 29) = 54.988$, $p < 0.01$).

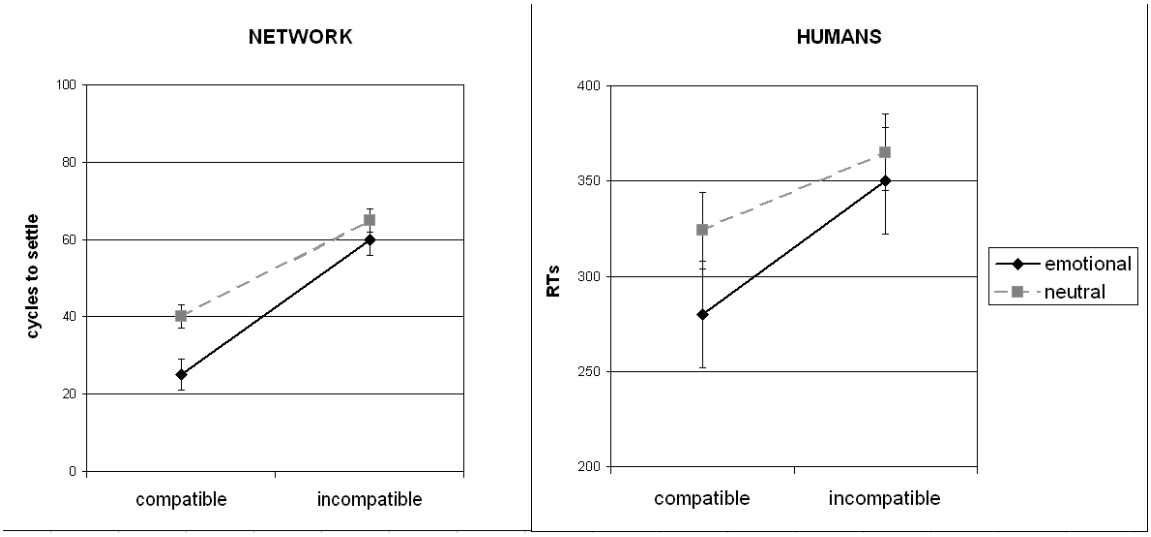

Figure 2
The model and human performance for both neutral and emotional conditions for compatible and incompatible trials. The model is able to reproduce the Stimulus Response Compatibility effect and the emotional enhancement of action found by Rumiati and Grecucci (in preparation).

3.4.1.3 Discussion

A strong stimulus response compatibility effect was found: responses were generated for the compatible trials (tapping after tapping) more quickly (i.e., fewer cycles were required for the network to settle) than for the incompatible trials (lifting after tapping). An effect of prior emotional stimulation was also found, with responses being generated more quickly to stimuli in the emotionally marked condition than in the neutral condition, similar to that shown in the behavioural experiments (see Figure 2). In the emotionally marked condition, activation of the amygdala units causes the network to settle to its final attractor state more quickly (i.e., in fewer cycles). According to the hypothesis we favour (Rumiati and Grecucci, submitted), emotional stimuli enhance motor performance in dangerous situations, where a faster response of the organism is necessary to escape the threatening stimulation. This simulation seems to support our interpretation. Another link between actions and emotions may be the mechanism through which emotional bodily expressions of an individual are translated into emotions of another individual. One possibility is that this phenomenon is mediated by the resonance of the body posture of the observed in the observer. de Gelder et al. (2004), for instance, have hypothesized that the integrated activity of the motor and the emotional system may constitute a mechanism for fear contagion and for fear preparation of action responses following the observation of fearful body expressions or dangerous stimuli. Our simulation supports this hypothesized mechanism for faster action production in the case of fear related action observation.

3.4.2. Testing the assumptions of the model

Each of the assumptions underlying the model was tested to demonstrate their roles in reproducing the effects found in the human data.

Simulation experiment 2a: First assumption

The first assumption (that the same area is activated both for observation and execution of the same movement) was tested by removing the layer in which the resonance mechanism is implemented (the action schema layer), and by sparing the layer with distinguished representations for observed and executed movements (the action representation layer). This layer was reconnected directly to the output layer. The network was retrained with the same input-output associations of Experiment 1 for 1000 epochs. Once trained, the network's performance on emotionally neutral trials was tested for 10 epochs. The effect of compatibility was no longer found (see Figure 3a). In fact there was no difference between compatible and incompatible trials ($t(9) = 0.790$, $p = 0.44$).

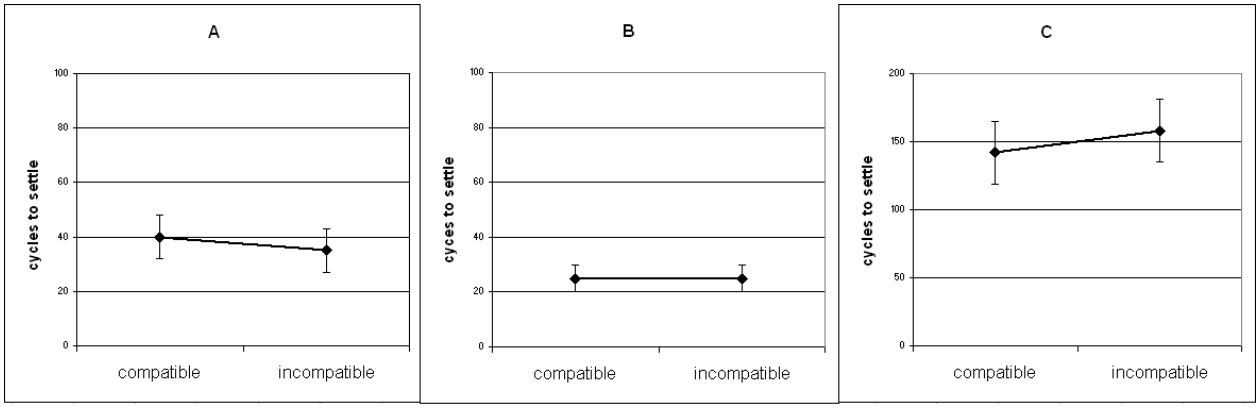

Figure 3
Testing the assumptions of the model. Figure 3A depicts the behaviour of the model when the Action Schema layer is removed. The representation for the observed and executed movements are collapsed within this layer, and the SRC effect disappears when the layer is removed. Figure 3B shows the performance of the model without the activation based memory (which is responsible for the priming effect). Again, the SRC effect is no longer reproduced. Figure 3C shows the models behaviour when k, a parameter of the kWTA mechanism, is reduced, thus testing the third assumption of the model. The SRC effect in this case is substantially reduced.

Simulation experiment 2b: Second assumption

The second assumption (concerning the priming effect) was tested by changing the parameter which integrates over time the membrane potential. In optimal conditions, neurons integrate in a graded fashion the current contributions (i.e., the excitatory activation for the to-be-executed movement) with the previous ones (i.e., the persisting activation of the just-observed movement). The persisting activity of previous contributions constitutes the neural basis of the priming effect (O'Reilly and Munakata, 2000). When the degree of persistence was reduced (in the PDP++ environment this was done changing the *dt_vm* parameter that controls the rate of change for membrane potentials from 0.05 to 0.005) the network no longer showed the compatibility effect ($t(9) = 0.826$, $p = 0.80$, see Figure 3b).

Simulation experiment 2c: Third assumption

The third assumption (inhibition of "nearby" regions that represent similar movements) was tested by lesioning the action schema layer by reducing the value of *k* in the *k*-Winners-Take-All function in the action schema layer. In PDP++ this was done by changing the *kwta.t* parameter for that layer from 4 to 0.1. This parameter controls the level of inhibition for all units within a layer. With *k*WTA in operation, within-layer inhibition is adjusted on every processing cycle such that exactly *k* units are activated above threshold. Setting k to below 1 effectively lesions a layer. In this case, even though activation was passed to the output layer, excitation within the schema layer was not sufficient to overcome the within-layer inhibition and hence high-quality (i.e., distinctive) representations could not be build up in the action schema layer. The rationale behind this manipulation was that without strongly activated nearby representations there should be no competition between different action schemas during incompatible trials.

The network was then tested without being retrained. As hypothesized the difference between compatible and incompatible trials decreased and was no longer significant ($t(9) = 1.156$, $p = 0.28$, see Figure 3c).

Simulation experiment 2d: Fourth assumption

Since, by hypothesis, in the emotional condition the activation of the amygdala should enhance the activation of the other layers, we tested if the activation of the action representation units varied as a function of the amygdala units' activation by monitoring the sending activation of the amygdala and the action representation modules. We tested the original network for 30 epochs while recording the sending activation of the two layers. We computed the sending activation as the sum of the activation of the "on" units for each trial, and calculated the average for each epoch. Thirty average values were obtained for each layer. The results are plotted in Figure 4.

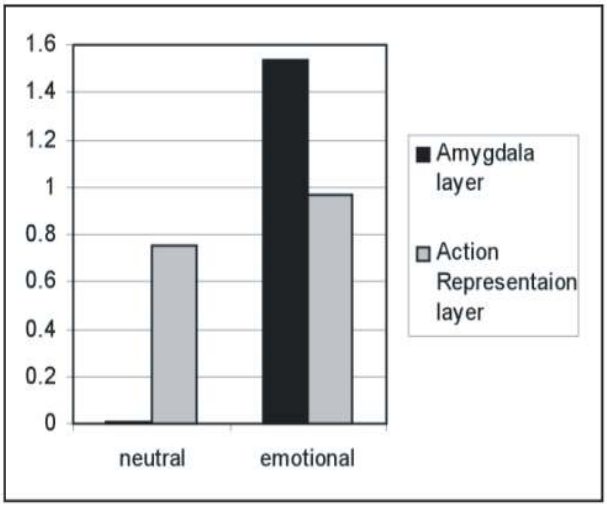

Figure 4
The activation of the amygdala enhances the activation of the Action Representation layer. This results in faster RTs for the emotional condition, for both the compatible and incompatible movements as seen in Figure 2.

A linear regression was computed with the dependent variable being the average activation of the units in the action representation layer and the predictor variable being the average sending activation of the amygdala layer units. The sending activation of the action representation layer was found to depend significantly on the sending activation of the amygdala layer ($\beta = 0.455$, $p < 0.001$).

3.4.2.1 Discussion

The simulations reported in this section have tested the four key assumptions underlying the model and demonstrated that in the context of our system the assumptions each play a critical role in reproducing the empirical effects investigated in the present paper.

The result of the simulation in Experiment 2a supports the *action resonance mechanism* (that the representation for an observed movement is utilized to execute the same movement), because when the network is required to use two different representations – one for the observed movement and one for the executed movement (as stored in the action representation layer) – then the compatibility effect is no longer reproduced.

Phenomena of stimulus-response compatibility suggest a direct relationship between perceptual and action domains (Prinz, 1990). According to a recent view, the Mirror Neuron System may be a putative neural basis of this resonance mechanism (Iacoboni, et al., 1999). In our simulation the action schema layer "resonates" both when the network observes the movement and when it executes the same movement. If this layer is bypassed the network is still able to reproduce the task (executing a movement after observing the compatible or incompatible movement), but the SRC effect is no longer reproduced.

Oztop and Arbib (2002) proposed a computational model of grasping in which there is a

separate neural module for the Mirror Neuron System. When performing *a grasp and object axes mismatch experiment*, the network fails to respond; therefore there is no data to compare our results with in order to ascertain whether the incompatibility effect is the product of an integrated action resonance system (as in our model) or is caused by a separate module for action resonance (Oztop and Arbib, 2002). In their model there was no real implementation of some mirror or resonance mechanism. The authors employed a feedforward network with one hidden layer in which they coded the associations between *object-affordance* and *hand-state* information and an output layer corresponding to mirror neurons that activates for grasping but without a mirror or resonance mechanism. Our simulation suggests that in order to reproduce, for instance, the behavioral results of a SRC task, or interference effects, there is need for an action resonance mechanism (i.e., a mechanisms in which only one representation is used for both the observed and the executed movement). Our model supports the results attributed to the mobilization of mirror neurons, but without assuming a brain module exclusively dedicated to a mirror function. Instead, our model assumes that action and perception converge into a common system sharing one common code (Prinz, 1997).

We have seen that if an action resonance mechanism exists in the brain, and if the motor system is geared up to execute movements while simultaneously observing different or opposite movements, this should result in interference (Blakemore and Frith, 2005). The results from Simulation Experiment 2c are is in accord with the behavioral results of studies using the interference paradigm of Kilner et al. (2003), who had subjects make sinusoidal movements with their right arm at the same time as observing congruent and incongruent movements. The results showed that the variance of the arm trajectory of subjects was greater in the incongruent condition that in the congruent condition. This may be explicable by assuming a common network in which 1) similar actions are coded which compete by reciprocal inhibition (the second assumption of our model), and 2) action observation primes action

execution which may interfere with the ongoing incongruent movement (the first assumption of our model). We can tentatively conclude that, in order to reproduce the stimulus response compatibility in our neural network system (and *mutatis mutandis* in the brain), three elements are necessary: i) a region of the cortex which activates both for the observation and for the execution of the same movement; ii) lasting neuronal activation which results in temporary memory and reactivation facilitation; and iii) strong competition for activation between cell assemblies representing two "close" actions (a mechanism similar to "contention scheduling" as hypothesized by Norman and Shallice (1986)).

The above considerations are similar to the ones proposed by Zorzi and Umiltà (1995) in their computational study of the Simon effect and by Zhang and colleagues (Zhang, Zhang, and Kornblum, 1999). They suggested that the Simon effect (and other compatibility effects) arises from an automatically activated task-irrelevant representation that activates a response which, in turn, competes (via a response selection mechanism) with the task-relevant response. In our simulation the previously activated representation for the observed movement competes with the other representation for the incompatible executed movement. The activation of the first representation is enhanced by the observation of the movement and it cannot be suppressed, nor can its effect on the following action production, as demonstrated by the RT results in the incompatible trials (in the SRC effect). In the same way, while performing a spatial task, it is not possible to suppress the automatically activated spatial representation even if it is task irrelevant (the Simon effect). In the Zorzi and Umiltà model, the response system incorporates a competitive mechanism via lateral inhibition (similar to the mechanism underlying k-WTA in our model) in which the spatial information (left and right position codes, which correspond to two distinct nodes) and the non-spatial stimulus to be discriminated attributes (again represented by two nodes) converge. When performing the task, the input activates both the spatial and the non-spatial attributes which affects the time to reach the final state of the network. In the case of

incompatible trials, the time to settle is longer, similar to what happens in our model, even though our model is more concerned with the motor system and its peculiarities (such as inhibiting related actions). However, some important differences must be stressed. First of all, with respect to the model proposed by Zhang, Zhang and Kornblum (1999), we must notice that our successful simulation of RT human data comes from the interference inside the motor module, not between the input and the output module as hypothesized by previous stimulus-response compatibility models. Another advantage of our model is in the biological plausibility of the algorithm employed by our network, and last but not least the fact that our model can account also the emotional modulation of the compatible condition, which is the novelty of this study.

The results of simulation experiment 2d relate directly to several imaging studies. In a PET study of the processing of emotional facial expressions, Morris et al. (1998) found that the amygdala activation "explains" the increase in rCBF in the occipito-temporal cortex. Likewise, in our simulation the amygdala activation "explains" the activation of the motor representational layers (i.e., the action representation and action schema layers). The activation of the amygdala units increases with the increasing of the sending activation of the action representation units. As a result the network settles more quickly, reproducing the behavioural data (Rumiati and Grecucci, submitted). In another imaging study, Bradley et al. (2003) found an increase in the strength of the BOLD signal when viewing aversive stimuli with respect to viewing neutral pictures. Emotions may influence action by enhancement of the motor areas, as has been found for the sensory areas (Anderson and Phelps, 2001; Bradley et al., 2003; Junghöfer et al., 2001; Pardiso et al., 1999; Simpson et al., 2000). Our results support this increase in the "strength of activation" for the action representation layer following aversive emotional stimulation but they are not able to disambiguate if this modulation is: 1) task specific, that is only the area involved in the task is enhanced (Rumiati and Grecucci, submitted); or 2)

more general, that is the emotional systems enhance the whole cortical arousal through the cholinergic neurotransmitter (Kapp et al., 1992). In support of the first hypothesis, the imaging study by de Gelder et al. (2004) found a fear-related activation in areas dedicated to action representation and motor areas in a task involving the observation of fearful body expressions.

3.4.3 Simulation Experiment 2: The conflict between cognitive task and emotional stimulation

The interaction between emotional stimulation and task demands is still poorly understood. From an evolutionary point of view the emotional action guidance systems precede the more sophisticated higher level planning of the primate brain. It is interesting to see how a later mechanism (higher-action planning), "implanted" on a ready functioning brain (based on the reptilian emotional automatic behaviour repertoire; MacLean, 1990), interfaces and interacts with it. Some of us have shown that voluntary action (pre-instructed finger movement) may be modulated by emotional stimulation (Rumiati and Grecucci, submitted), and Simulation Experiment 1 has shown how this may occur computationally. But what happens in the case of a dangerous situation when we are required to perform the right action respecting some environmental constraints? How might emotions affect the motor system when we are required to plan action instead of performing only a speeded response? The primitive defensive behavioural repertoire would fail to choose the right response in a complex situation where a stereotypical automatic response would be inappropriate.

This issue has typically been studied by requiring subjects to perform some cognitive tasks (spatial, attentional, semantic etc.) after emotional stimulation (visual, auditory or somatic). The results until now have been confusing and divided into opposite factions. On the one hand there are authors

sustaining a "competitive view" of emotions (Drevets and Raichle, 1998; Northoff, Heinzel, Bermpohl, Niese, Pfenning, Pascual-Leone, and Schlaug, 2004), on the other there are those who support a "synergistic view" (Ito, Larsen, Smith, and Cacioppo, 1998; Sato, Kochiyama, Yoshikawa, and Matsumura, 2001; Simon-Thomas, Role, and Knight, 2005). The first approach claims that emotions, in particular those with a negative value, degrade cognitive (and motor) functions. This view is based in part on increased RT following emotional negative stimulation compared to neutral stimulation in attentional, spatial and linguistic tasks (Keil et al., 2005; Hartikainen et al., 2000; Isenberg et al., 1999). It is also supported by neuroimaging studies showing an inverse relationship between brain regions involved in negative emotions and cognitive tasks (Northhoff et al., 2004; Drevets and Raiche, 1998). The second view argues that negative emotions may enhance cognitive processing (Gray et al., 2002; Ohman et al., 2001; Sato et al., 2001; Ito et al., 1998), resulting in shorter RTs when performing a task. The picture is further complicated by studies that have found no effect of emotional stimulation on RTs (e.g., Simon-Thomas et al., 2005).

How might these discrepancies in experiments between "synergistic", "competitive" and "no-effects" of emotional stimulation on cognition be explained? We suggest that the effect of emotional stimulation on a specific cognitive task depends on the balance between the *emotional load* and the *cognitive load* in terms of, for instance, instructions to be kept in mind to execute the specific cognitive task. If the instructions are many or complex, the cognitive component of the task will compete for resources and conflict with the emotional part causing impairment in performance (i.e., slower RTs, as predicted by the "competitive view"). On the other hand, if the task has a strong emotional component and fewer or simple instructions (such as a preinstructed finger movement, as in the experiment of Rumiati and Grecucci, submitted), enhancement of performance is observed (i.e., faster RTs, as predicted by the "synergistic" view). This second case is what was demonstrated in Simulation

Experiment 1, in which motor performance following aversive emotional stimulation was faster.

Some studies suggest that the "site" of the conflict between emotional and cognitive parts of the task may be the Ventro-Medial Pre-Frontal Cortex (VMPFC), which serves as the crossroad between the amygdala and the Dorso-Lateral Pre-Frontal Cortex (DLPFC). The DLPFC is known to play a fundamental role in task-maintenance, sustaining the instructions to execute a task, whereas the VMPFC is held to mediate between the emotional information from the amygdala and the higher executive instructions from the DLPFC. Imaging studies have found that there is a "crossed interaction" through inhibitory reciprocal connections between these two regions (Drevets and Raichle, 1998; Northoff et al., 2004; Yamasaki et al., 2002; but see also Mayberg, 1997). We suggest that this mutual inhibition can explain why in certain experiments emotional stimulation impairs performance while in others it enhances it. Moreover this DLPFC layer resembles the Supervisory Attentional System proposed by Norman and Shallice (1986), for its role in top-down modulation of the emotional (amygdala layer) and the action subsystems.

To summarize, when the task of an experiment is low in cognitive load (corresponding to low activation of DLPFC) and high in emotional load (corresponding to high activation of the amygdala-VMPFC complex), a synergistic contribution of emotional stimulation will be detected (i.e., RT will be faster). In contrast, when the task is characterized by a high cognitive load (corresponding to high activation of the DLPFC), emotional stimulation will interfere and conflict with the cognitive part of the task, leading to slower RTs. Simulation Experiment 2 was designed to test this hypothesis.

3.4.3.1 Method

We augmented the model with a new layer corresponding to a DLPFC module (see Figure 5) whose function was to hold the task instructions, whatever they might be. This module was superimposed on the network and played the role of a modulatory top-down influence. We

implemented the “active maintenance” function of DLPFC (O’Reilly and Munakata, 2000), with units which have a self sustained activity that do not spread out over time (represented by the circles with arrows for each unit in Figure 5). In particular, for each trial all units of the DLPFC module become active and remain in a state of sustained firing. In PDP++ this was achieved by creating a LeabraContextLayer, which implements a Simple Recurrent Network context layer. The main feature of this module is that it maintains activation over time via excitatory self-connections. The connectivity of this module is bidirectional with the amygdala module, the action representation layer and with the output layer, and unidirectional from the input layer. This was connected to the action representation layer and not to the action schema layer as the DLPFC is thought to disrupt the emotional effect which takes place in the amygdala layer and the action representation layer, rather than the action schema layer (see paragraph 4.2.4). (Consistent with this, connecting it to the action schema layer was found to interfere with the compatibility effect). The connections between the DLPFC layer and the amygdala and action representation layers were set to be bidirectional. The network was not retrained. Rather, based on the features of this particular type of layer within the PDP++ environment, we just connected it to the other layers. The desired activity level over the entire layer was computed using the percentage of units active in the layer. This activity does not decay over time (i.e., decay was set to zero). The resulting sending and incoming activation from DLPFC to the target modules caused a change in the attractor dynamic of the target layers. The final state of the units was no longer reached through the contributions of both the particular input pattern and of the learned weights, but instead it was also affected by the self-sustained activity of the PFC layer.

The number of units active in the DLPFC was varied to see how “task demand load” affects performance. We can imagine that each active unit represents an instruction to hold in mind in order to execute the task. Eight different architectures were tested. The first had a DLPFC module consisting of

only 1 unit, the second was a matrix of 2 x 1 units, the remainder had more (2 x 2, 2 x 3, 2 x 4, 2 x 5, 2 x 6 and 2 x 7 units).

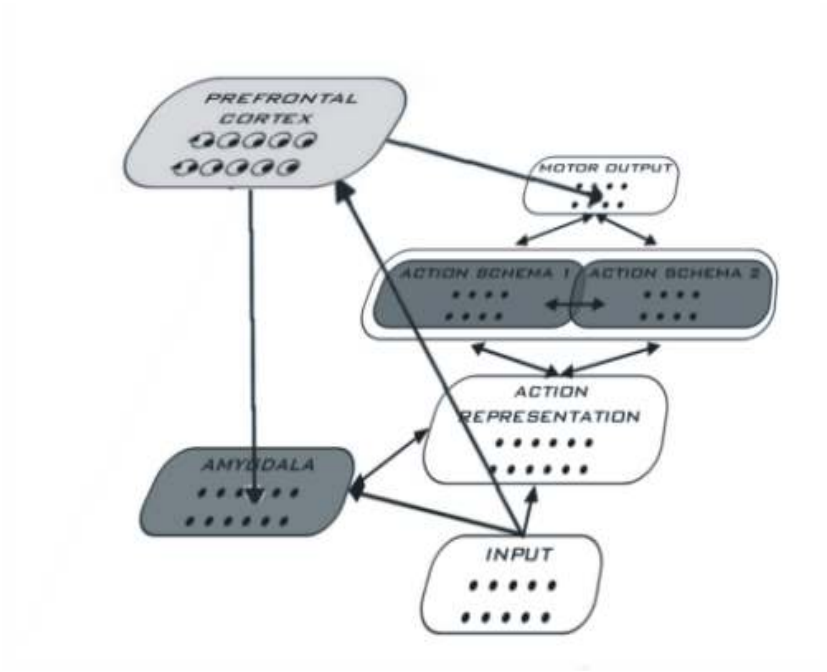

Figure 5
To simulate the effect of the interference between cognitive tasks and emotional stimulation we implemented a layer corresponding to the (dorso-lateral) prefrontal cortex in which the units have the particularity of self sustaining their activity. This property may be the basis of task maintenance. Our suggestion is that task maintenance can influence the emotional modulation of the SRC found in Experiment 1, by hypothesizing that when the task load is not taxing in terms of cognitive resources (and so few units are active in the PFC layer), the synergistic effect of emotion will be clear (shorter RTs for task execution, left side of the graph). When task demands are taxing (and so many units are active in the PFC), the competitive effect appears and task performance reverses with respect to the neutral condition (larger RTs, on the right of the graph).

Within the extended model, our prediction was that sustained activation of the DLPFC, representing the "task-instructions-to-be-held-in-mind", should conflict with the amygdala, the action representation layer and output layer processing, and result in interference and disruption of the "emotional" computation. For high numbers of units active in the DLPFC, we hypothesised that this would lead to a slowdown in the time taken to reach the final state, reproducing the typical

"competitive" result of the behavioural experiments cited above. By hypothesis the size of the DLPFC will affect the dynamics of the system to different degrees. In the case of few units in the DLPFC, activity in the amygdala should still be able to affect the network's performance (through the action representation layer) in hastening the reaching of the final attractor state in the emotionally valent condition. However, with many active units in the DLPFC, the self sustaining activity of this layer should increase excitation throughout the network, particularly when the amygdala is also active (i.e., in the emotionally valent condition). Under these conditions it should take longer for competition (e.g., within the action schema layer) to be resolved, and hence longer for the network to settle.

3.4.3.2 Results

The different networks were each tested for 10 simulated blocks. Figure 6 shows the resultant behaviour. Only the compatible trials from both the neutral and the emotional condition are reported. We see that as the number of active DLPFC units increases, the behaviour of the network changes: at first the network is faster for the emotional condition as found in Simulation Experiment 1 (in accord with the synergistic view), but when the DLPFC size is increased, the tendency reverses and the network becomes slower to respond to the emotional trials, simulating the results of the competitive view. In fact there is a significant positive correlation between the number of units active in the PFC layer and the cycles to settle in the emotional condition ($r = 0.929$, $n = 8$, $p < 0.001$, one-tailed). While for the neutral trials there is no significant correlation ($r = –0.195$, $n = 8$, $p = 0.322$, one-tailed). Thus in the neutral condition the number of units active in the PFC layer does not affect the network's behaviour. This applies also for the incompatible trials for both emotional ($r = -0.038$, $n = 8$, $p > 0.05$, one-tailed), and for the neutral trials ($r = 0.007$, $n = 8$, $p > 0.05$, one-tailed). The reason why no significant effects are found in the incompatible condition for both primes can be explained considering

that incompatible trials requires more computational resources by the whole system. This may have hidden the eventual influence of the DLPFC module stimulation.

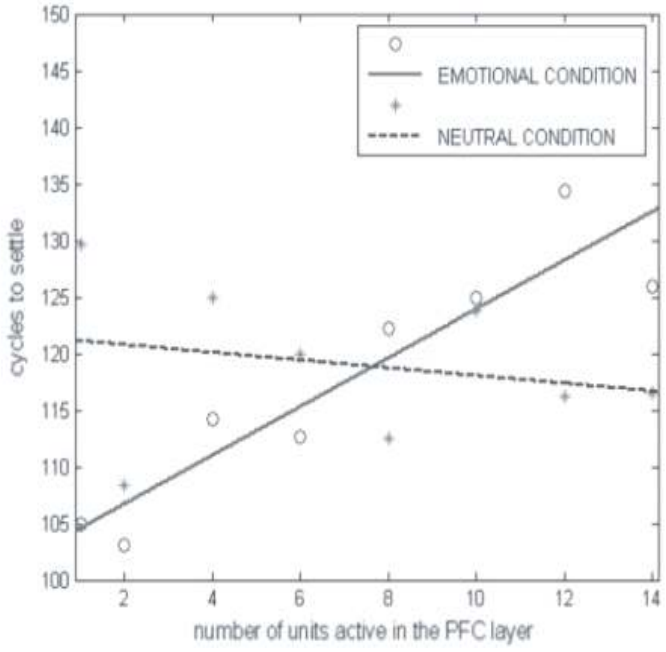

Figure 6
A scatter plot of settling time against number of active units in the simulated PFC layer for the neutral and emotional conditions. In the emotional condition, settling times are significantly positively correlated with the number of active units in the PFC layer, while no such relation holds in the neutral condition.

3.4.3.3 Discussion

In Simulation Experiment 3 we tested the hypothesis that task demands and emotional activation interfere. If the task is very demanding in terms of cognitive resources, RTs will slow down because of interference between emotional stimulation and task instructions. If the task is less taxing (as the motor task of Rumiati and Grecucci, submitted), and the subject does not have to keep in mind

complicated instructions, an advantage following an emotionally valent trigger is shown. Anatomical data suggest that this conflict is grounded in an interaction between the amygdala/VMPFC and the DLPFC.

Drevets and Raichle (1998) found what they referred to as a "cross-modal" interaction in the rCBF between the amygdala/VMPFC and the DLPFC. A similar interaction occurs in our model between the DLPFC module and the amygdala module. This causes the disruption of the emotional effect found in Simulation Experiment 1 when unbalancing the load of the DLPFC contribution (increasing the number of units) to the other layers. As the size of DLPFC increases the effect found in Simulation Experiment 1 reverses reproducing the typical results found in behavioural studies (Keil et al., 2005; Hartikainen et al., 2000; Isenberg et al., 1999). We were also able to reproduce the "null hypothesis" derived from the experiment of Simon-Thomas et al. (2005) which found no difference between the emotional versus the neutral condition. By our simulation the difference from the latter and the former relies on this balance between the cognitive load and the emotional stimulation of the experiment. In the study of Simon-Thomas et al. (2005), which showed no effect of emotional stimulation, the cognitive task (a modified Stroop task) was not so demanding; in contrast the tasks used, for instance, by Hartikainen et al. (2000) were more complex and demanding (e.g., a hemispheric visual discrimination task).

3.5. General discussion

In this study we investigated the cognitive mechanisms of the stimulus-response compatibility effect in the context of imitation, and its modulation by negative emotional state using a biological plausible artificial neural network model. The aim of Simulation Experiment 1 was to reproduce both the stimulus-response compatibility effect and the effect of emotion on action

performance as founding Experiment 1 (chapter 2). We were able to demonstrate that the network is able to reproduce the SRC effects and its emotional modulation found in behavioural experiments by virtue of four mechanisms including the action resonance layer, the facilitation effect in compatible trials, and the inhibitory dynamics between different action representations. Moreover we demonstrated that the enhancement of this effect following emotional stimulation may arise from the system settling into the attractor state more quickly caused by the sending activation of the emotional detector layer (which stands for the amygdala). In Simulation Experiment 2 we applied the model to the long standing debate between synergistic and competitive views of cognition/emotion interaction. We demonstrated that SRC effects can be modulated by task demands: when instructions are taxing in terms of cognitive resources, the emotional stimulation results in the network taking longer to settle into an attractor state.

More specifically, in Experiment 1 the network was capable of reproducing both effects. When the network "executed" the same movement previously as had been observed, the response attractor was reached more quickly (i.e., the network required fewer cycles to settle, and hence responded in shorter reaction times) than when executing the incompatible movement, as found in behavioural studies (Brass et al., 2001; Rumiati and Grecucci, submitted).

In an informal sense, the fact that we have a strong tendency to spontaneously imitate explains the compatibility effect. In theoretical terms, the direct-matching hypothesis (that observing an action activates the corresponding representation in the observer, which may then be used to execute that movement) explains well why we are facilitated in performing compatible movements. But this hypothesis has proved to have difficulties in explaining why we are slowed in performing incompatible movements. A possible neurally plausible explanation of this slowing in RT is that cortical representations of similar actions may produce interference. Thus, when observing an action, a specific cortical region may be activated. An activation of this region causes inhibition of the regions

representing similar actions. When we are then required to execute not the seen movement but one similar, the representation of this action would, following observation of the related action, be inhibited. This dynamic is implemented in our system by a *k*-Winners-Take-All algorithm. In addition, as a result of the activation of the observed movement, the same region is facilitated in becoming re-activated for the execution of this movement via activation-based memory. In the case of compatible stimulus-response assignments, stimuli will prime the correct response as a result of this action resonance mechanism, whereas in incompatible assignments, this priming will refer to the incorrect response and therefore lead to interference (Prinz, 1997). The incompatible trials of our simulation reproduce this interference effect too. There is now a growing body of evidence suggesting that, in humans, some brain regions are activated both during action generation and observation of actions of others (Decety et al., 1997; Hari et al., 1998; Rizzolatti et al., 1996). This activation has been shown to be very specific in topographic terms (Buccino et al., 2001). Our model takes into account all these data. The resonance mechanism is necessary for reproducing the compatibility and the interference effects found in behavioural experiments and our simulations support the claim that there is motor contagion by which the motor system of the observer is primed to produce the observed movement (Blakemore and Frith, 2005).

In Simulation Experiment 1 we also saw that the network was faster in reaching the final state following emotional stimulation. Emotions may influence action by a bottom-up enhancement of the sensory and motor areas producing an increase in the "strength of activation" (i.e., increase of the signal) with respect to viewing neutral pictures (Bradley et al., 2003). Our results support this increase in the strength of activation for the action representation layer as a result of the emotional stimuli. According to Whalen (1998) and Davis and Whalen (2001), the amygdala may be especially involved in increasing vigilance. This function may be related to a "fear module" (Ohman and Mineka, 2001)

implemented in the amygdala, which serve as a preparedness trigger to avoid dangerous stimuli. The amygdala is known to be involved in many functions, from facial expression perception to the valuation of negative stimuli, prosodic interpretation and many others, but it seems that its *common computational profile* (Sander, et al., 2005) may be this general activation of sensory, attentional and motor processes. Indeed, several imaging studies (Amaral and Price, 1984; Bradley et al., 2003; Lane et al., 1997; Morris et al., 1998) have shown that for aversive stimuli the amygdala enhances activation of the occipital-parietal and temporal cortices.

Another mechanism by which the amygdala can influence action is the release of norepinephrine onto motor neurons via lateral extended amygdala activation of the locus coeruleus, or via projections to serotonin that could enhance motor performance during a state of fear, because both norepinephrine and serotonin facilitate excitation of motor neurons (Davis and Whalen, 2001). The core mechanisms implemented in the model presented here (*the sending activation which excites the other layers)* may be viewed as the computational effect of this neuromodulation. The excitatory effect of the amygdala for action is similar to the excitatory effect for attention reproduced by Taylor and Fragopanagos (2005) in several neural network simulations. In their model the amygdala layer is reciprocally connected with the attentional systems, similarly to as in our model, but with somewhat different results. Whereas the result of our simulation shows the modulation of performance (faster RTs when task demands are low, but slower RTs when task demands are high), in Taylor and Fragopanagos' (2005) simulation the activation of the amygdala results in a worsening of performance, possibly due to the interaction with more cognitive parts of the network.

The modulation of processing resulting from activation of the amygdala may be task specific in the sense that the area enhanced is only the one involved in the task or it may be an aspecific effect of increased cortical arousal. If the first hypothesis is true, we suppose that in the brain there would be

an “emotional action resonance mechanism” which activates in emotional conditions while interacting in a social context to facilitate the sharing of emotions (e.g., empathy, the chameleon effect, etc.) and group dynamics (such as common responses as hypothesized by social contagion). Recent studies (see for example de Gelder et al., 2004) point the finger at the relation between action and emotions in recognizing emotional bodily expression. It was hypothesized that this link may be somewhere between the mirror system and the limbic system in order to explain how we can understand emotions of others by simply seeing bodily expressions of the *receiver* and conversely how emotions can be translated into informative bodily expressions in the *sender*. It seems that an action resonance mechanism directly connected to the emotional structures of the brain (probably via the insula, see de Gelder et al., 2004) might be responsible for these primitive and non-conscious transmissions of states between agents-in-relation (Leslie et al., 2004). If we consider the evolutionary role of emotions as a way of communicating in a fast and efficient way before the appearance of structured language, we can argue that there must be also an efficient and fast mechanism for emotion understanding in animals receiving the emotional information of the animal who is feeling that emotion. Otherwise the evolutionary argument of the supposed communicative advantage of emotions would be no longer reasonable. The results of this simulation take into account also why we are facilitated in recognizing the emotions of others by assuming this emotional modulation of the resonance mechanism by which we can understand others’ mimes and imitate them. Our model provides a first attempt to ground this emotional action resonance system on empirical facts (the reproduction of the behavioural results of Rumiati and Grecucci, submitted) but also takes into account recent experimental findings related to these issues (Carr et al., 2003; de Gelder et al., 2004; Hadjikhani and de Gelder, 2003; Leslie et al., 2003).

In Simulation Experiment 2 we tested the interaction between the emotional stimulation and

the task instructions. Having to hold in mind task instructions (corresponding to sustained activation of the PFC layer) interferes with the processing of emotional stimuli, slowing down RT. This interaction has been previously implemented by Taylor and Fragopanagos (2005), but they reproduced the results predicted only by the "interference" hypothesis, without addressing the issue of why certain experiments show an enhancement effect (as predicted by the "synergistic view") and others an inhibitory effect ("competitive view"). We addressed this issue by showing that in normal conditions, when unpleasant emotions are processed without cognitive additional tasks, they improve performance yielding faster reactions to threatening stimuli (Simulation Experiment 1 and *smaller-size-DLPFC* simulations of Simulation Experiment 3). When using more demanding cognitive tasks, emotional stimulation worsens performance since it overloads the system (as shown in the *larger-size-DLPFC* simulations of Simulation Experiment 3). We hypothesized that this critical interaction may be implemented at the crossroad between the amygdala and the DLPFC, in the VMPFC. We have also seen that there is a cross-modal interaction between the amygdala and DLPFC, resulting from the inhibitory connections (possibly mediated by VMPFC). This cross-modal interaction may result from specific mechanisms evolved for survival reasons: in a dangerous situation it is better to use emotional systems and for other situations it is better to use more cognitive strategies and suppress emotional responses.

Chapter 4
Psychophysiological mechanisms of fear guided imitation

4.1 Introduction

The present experiment was designed to examine possible electroencephalographic correlates of the basic effect found in Chapter 2 (Experiment 2) and to test by using previously characterized cortical movement-related potentials to see if the neural signal differs in the emotional condition with respect to the neutral one. According to the computational model presented in Chapter 3 we predicted that during the execution of movements following the emotional prime, enhanced cortical activity (as found in the action representation layer of the model, assumption 4) should be found in two movement-related components: the Readiness Potential (RP) and the motor potential (MP), the first preceding the execution of the movement, and the second accompanying it.

The RP is a negative wave starting 1 sec before the movement itself having the site of maximum activity in the precentral area extending to the parietal and frontal regions, contralateral to the movement (Shibasaki, Barret, Halliday, and Halliday, 1980). This potential is thought to result from activity in large frontal motor areas, and in primary and secondary sensory areas extending more parietally (Green,

Arnold, Rozhkov, Strother, and Garrot, 2003). Two main epochs of the RP have been distinguished, an earlier symmetrical component followed by a lateral asymmetric one that starts about 500 ms before movement onset (Dirnberger, Fickel, Lindinger, Lang and Jahanshahi, 1998). Our analysis will use only this second phase of the RP which some authors refer to as the "negative slope" (Shibasaki, et al., 1980). The Motor potential (MP) is the last stage of the motor related potentials characterized by the maximum negative peak accompanying the executed movement. Its scalp distribution is more asymmetrical (contralaterally) and fronto-central (Shibasaki, et al., 1980) and it typically accompanies the execution of the movement from the muscle burst to the end of the movement. In addition to these potentials we also measured the EMG signal recorded from the arm executing the movement.

In order to index the stimulus-driven emotional engagement of the subjects, we analyzed the Late Positive Potential (LPP), a large and long-lasting positive deflection over occipito-parietal sensors (Sabatinelli, Lang, Keil, and Bradley, 2007; Shupp, Junghofer, Weike, and Hamm, 2004). This potential has been previously used as a physiological marker for the allocation of perceptual and attentional resources to emotionally- relevant stimuli, and as an index of the engagement of basic defensive and appetitive circuits in the brain (Lang et al., 1997). This positive shift usually begins as early as 350 ms after the onset of emotional picture exposure (Schupp, Cuthbert, Bradley, Cacioppo, Ito, and Lang, 2000) and reaches its peak at about 400-700 ms after stimulus onset. A second point to address is what happens in the compatible non-imitative trials. The computational model (Chapter 2) led us to conclude that compatible movements are easier and faster because of an action resonance mechanism (assumption 1), and that incompatible movements are more difficult and slower because of a cortical competition between close but different motor representation (assumption 3). These fine grained mechanisms cannot be tested by means of ERP procedure. However, it might be that another mechanism comes into play in the execution of incompatible movements.

Recent evidence suggests that simply observing an action seems to evoke a tendency to execute that action (Brass, Bekkering and Prinz 2001), but since they are not always adaptive in everyday situations, they are usually inhibited (Brass, Zysset, and von Cramon, 2001). Inhibition is postulated to be a mechanism by which prefrontal cortex (PFC) exerts its effects on subcortical and posterior cortical regions to implement executive control (Aron, Robbins, and Poldrack, 2004; Miller, and Cohen, 2001). Regardless of the particular task or domain, dorsolateral prefrontal cortex (DLPFC), inferior frontal gyrus (IFG) and dorsal anterior cingulated cortex (ACC) are consistently activated when subjects are engaged in tasks involving active control of response (Duncan and Owen, 2000). In particular, the IFG seems to be critical in inhibiting motor response when they are not context or task appropriate (Aron et al., 2004).

More related to the inhibition of imitation responses, neuropsychological case studies shed light on this imitation tendency after frontal damage. Brass, Derrfuss, von Cramon and von Cramon (2003), reported that frontal patients displayed significantly stronger imitative response tendencies than a group with nonfrontal lesions (the so called "echopractic behavior", Luria, 1966; 1980 also known as "imitation behaviour" Lhermitte, Pillon and Serdaru, 1986).

To investigate the neural structures involved in the inhibition of imitative responses, Brass, et al. (2001), carried out an fMRI experiment in which subjects executed a simple preinstructed finger movement (tapping), while viewing compatible (tapping) or incompatible (lifting) movements. When contrasting the incongruent with the congruent trials, the right middle frontal gyrus (MFG), the banks of the superior frontal sulcus, as well as the right superior frontal gyrus (SFG), the right anterior parietal cortex, the posterior precuneus bilaterally and the parieto-occipital sulcus were found activated. The argued that these areas are associated with the conflict between the externally triggered imitative response, and the intention of the to-be-executed willed action (Brass, et al., 2001). They also

claimed that prefrontal cortex (especially the MFG) plays a role in inhibiting the externally triggered imitative response. If this mechanism is true, we should find prefrontal related cortical activity in the waves associated with the movement execution.

Interestingly, given the time constraints imposed by the fMRI the dynamic between the regions involved remains unaddressed. To claim that the prefrontal cortex activates to inhibit the motor response, the temporal dynamic of their activation should be explored. What's the temporal dynamic between these regions? Who is the "leader" and who is the "follower"? Who is the "inhibitor" and who is in turn "inhibited". To shed light on the time course of activation in these different regions, we carried out an analysis on compatible versus incompatible trials of the neutral condition of the original experiment.

4.2 Methods

Participants

Fifteen subjects (seven males) all right-handed, with normal or corrected-to-normal vision (mean age: 25 years; education: 16 years), took part in the study after providing written informed consent. They were naïve to the study and were paid 10 euros for their participation. Subjects were interviewed to assess their medical and psychological condition, and were administered two tests to assess anxiety-related personality traits that potentially affect reactions to emotional stimuli: 'negative affectivity' (PANAS, Watson and Clark, 1994) and 'behavioural inhibition/activation' (BIS/BAS scale, Carver and White, 1994). All subjects scored within the normal range, so no analysis of these questionnaires will be reported. Prior to the experiment proper, subjects performed a familiarization phase (20-trials, later excluded from the analysis). Those who did not perform the task adequately were asked to perform an

additional 20 trials to reach standard performance. Subjects were monitored via a unidirectional mirror during the experiment.

Stimuli

Neutral and emotional pictures and short movies of a moving hand were employed as stimuli. Two sets of pictures from the International Affective Picture System (IAPS, Lang, Bradley, and Cuthbert, 2005) were used as primes. The IAPS, developed to provide a set of normative emotional stimuli for experimental investigations of emotion, contains a large set of emotionally-standardized, color photographs distributed in a two-dimensional affective space formed by "valence" and "arousal" ratings. In this experiment we used only negative and neutral pictures based on previous work that suggests that unpleasant negative pictures significantly facilitated imitation (Rumiati *et al*. submitted). Negative pictures, which were generally judged to be very unpleasant, had a mean valence of 2.28, and mean arousal of 6.05; neutral stimuli had a mean valence of 5.08 and mean arousal of 2.76. Fear-relevant stimuli depicted dangerous objects, animal attacks, human attacks, scenes of calamities and accidents, bloody scenes, human and animal mutilations, self-defence, pollution and corpses, known to elicit defensive reactions. Perceptual (from simple objects to complex images) and semantic features (grouped in four categories: objects, animals, humans and landscapes) of the stimuli were balanced across conditions. A total of 100 pictures (50 neutral and 50 negatively emotional) were selected. Since each picture was repeated twice, subjects saw a total of 100 negative and 100 neutral pictures.

To control for perceptual differences in terms of complexity, brightness and colors, between the emotional and neutral pictures, we analyzed the "entropy" of the intensity of the images (used to characterize their texture), the "inertia" (mean contrast between each pixel and its neighbour over the whole image), the "uniformity" (summation of squared elements in the grey level co-occurrence

matrix), the "homogeneity" (closeness of the distribution of elements in the grey level co-occurrence matrix to the same matrix diagonal), the brightness and the percentage of the three fundamental colours using functions provided in the Matlab Image Processing Toolbox. Each stimulus was analyzed independently, and the values of the two sets (neutral vs. emotional) were compared using ANOVA. Of these parameters, only Percentage of Green ($F(1,98) = 9.776$, $p < 0.01$) and Percentage of Red ($F(1,98) = 3.829$, $p = 0.053$) differed significantly or nearly-significantly between the two sets of stimuli (Entropy $F(1,98) = 2.06$, $p = 0.155$; Inertia $F(1,98) = 0.66$, $p = 0.42$; Uniformity $F(1,98) = 0.88$, $p = 0.35$; Homogeneity $F(1,98) = 2.29$, $p = 0.133$; Brightness $F(1,98) = 1.14$, $p = 275$; Percentage of Blue: $F(1,98) = 0.659$, $p = 0.419$). However, perceptual features such as color, brightness, and compression format do not appear to be relevant for predicting the salience (measured with subjective ratings) of these kinds of pictures (Buodo, Sarlo, and Palomba, 2002). The differences in green and red color percentage should therefore have little direct effect on the ERP motor components measured in this study, especially since these occur later than the visual evoked potentials affected by color parameters**.**

After each prime (emotional or neutral), subjects saw the movement of an animated hand. The animated hand mirrored the participant's right hand which was kept on the table in front of them. The video sequence consisted of five frames, the first of which showed the index finger in a middle, resting position, identical for both movements (lifting and tapping), which remained visible for 300 ms; the subsequent three frames flashed for about 40 ms each. The last frame contained an image of the final finger position and remained on the screen for 210 ms (Figure 1).

Experimental Procedure

All procedures were approved by the SISSA ethical research committee. Participants sat in a recliner in a sound-attenuated, dimly lit room. Scalp and peripheral electrodes (EMG and EOG) were

applied, and computerized task instructions were given. To prevent excessive eye-blink artefacts, participants were also asked to blink their eyes as much as possible while viewing the fixation point and to avoid eyeblinks during other parts of the trial. A Pentium IV computer controlled the experiment (E-prime software) and the data acquisition. The visual stimuli were projected on a 19-inch white screen located approximately 1.1 m from the participant's eyes (≈ 22° of visual angle).

Each trial consisted of the following events (Figure 1): first a fixation point appeared, lasting for 200 ms, followed by a black screen for 200 ms. Then the prime pictures were presented for 1 second, followed by a digitized video-sequence of the animated hand for 600 ms, at the end of which subjects were required to execute the pre-instructed movement. The images of the moving hand were positioned in the centre of the screen, and were of approximately life size (13x16 cm); IAPS pictures covered the whole screen. Each experiment contained 200 trials, 100 in the emotional and 100 in the neutral condition.

The type of stimulus (i.e. lifting or tapping) was varied within each block in a compatible or incompatible fashion. The subjects then performed the finger-tapping task while either compatible or incompatible finger movements were shown (lifting and tapping). All IAPS pictures were randomized, as well as the coupling of compatible-incompatible events with the pictures across trials.

Subjects had a maximum of 3000 ms to respond, after which a black screen was presented for another 3000 ms as an interstimulus interval. Subjects' reaction times were recorded from the onset of the last frame of the animated hand until they pressed the bar. During training trials, participants were instructed to perform the movement as accurately as possible according to the following procedure: the hand was positioned on a platform, with all the fingers in a resting position except for the index finger which was rigidly kept up 6.5 cm upon the bar of the computer keyboard. The required movement was a

downward motion of the index finger of about 60 degrees, comparable to the one observed on the screen.

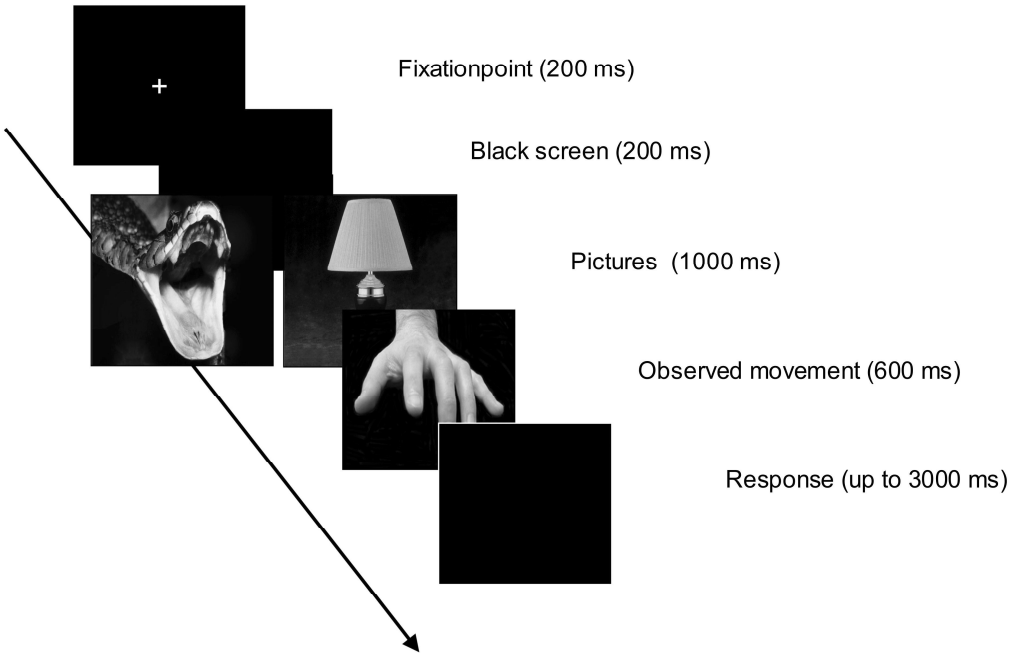

Figure 1. The experimental procedure.
The same procedure of Experiment 2 from Chapter 2, was applied except for slight changes in the timing of each event, to better fit an ERP procedure.

Apparatus and Data Analysis

Electrophysiological data were recorded from the scalp using a 32-channel system (Neuroscan), with two channels used for the oculogram (HEOG and VEOG), and one for the Electromyogram (EMG). Nonpolarizable Ag/AgCl electrodes were positioned in a standard precabled cap (10/20 international system). Scalp impedances for each electrode were kept below 5 kΩ. The electroencephalogram (EEG) was recorded continuously with a sampling rate of 1000 Hz, with the left mastoid as a reference and the right mastoid as ground; signals were acquired with a bandwidth of 200 Hz (12 db impedance). The signal was filtered offline (bandpassed between 0.05 Hz - 30 Hz). A baseline correction was applied by

subtracting the mean of the signal from 100 ms before the fixation point to the stimulus onset from the recording of each trial.

The active EMG electrode was positioned on the extensor digitorum muscle of the arm performing the movement and referenced to an electrode placed 3 cm proximally on the same arm. The EMG channels were acquired with a bandwidth of 1500 Hz. Horizontal EOG (HEOG) was recorded bipolarly from the outer canthi of both eyes, while the Vertical EOG (VEOG) was recorded by placing the electrodes on an imaginary vertical line traversing the pupil of the eye, with the lower electrode 14 mm above the upper border of the eyebrow. The surface electrodes were filled with conductive gel after abrasion of the corresponding skin.

The signals were epoched and analyzed using Scan 4.2 software (EMS s.r.l.). Averaged data were statistically analyzed in SPSS, with further analyses run in Matlab 7.14 (graphics and interpolations). Raw EEG epochs were first visually inspected for artefact rejection. Trials with horizontal and vertical eye blink artefacts were manually rejected; the number of eliminated trials was less than 20% of all trials.

Repeated measures ANOVAs were used to test for differential cortical processing of the emotional conditions, with the Bonferroni-corrected t-test used for post-hoc tests. Individual waveform analyses of each sensor served to identify the temporal and spatial characteristics of the signal. Based on this exploratory analysis, we then planned further analyses in which data were collapsed across previously established meaningful dimensions. After an exploratory analysis, the scalp was divided into three regions (region of interest, ROI): anterior, central and posterior; each of these regions was subdivided in turn into left, mid and right areas. The electrodes belonging to each region were averaged, giving nine scalp regions-of-interest (three electrodes per region, except two temporal regions including four electrodes). See Table 1 for details.

REGION	LATERALITY	ELECTRODES
ANTERIOR	LEFT	F1, F3, FC5
	MID	FP1, FP2, Fz
	RIGHT	F8, F4, FC6
CENTRAL	LEFT	T3, C3, CP1, CP5
	MID	FC1, FC2, CZ
	RIGHT	T4, C4, CP2,CP6
POSTERIOR	LEFT	T5, P3, 01
	MID	PO3, PO4, PZ
	RIGHT	T6, P4, PO4

Table 1. Electrode groupings.

Trials were divided into two time windows of interest, one covering the emotional stimulation, the other concerning the motor part of the task. For the emotional part of the task (from fixation point to emotional / neutral pictures), *stimulus locked* epochs were extracted from 100 ms before until 1400 ms after fixation point onset, covering the presentation of the IAPS pictures. The late-positive potential (LPP), a large positive deflection lasting for the duration of the emotional image over occipito-parietal sensors (Sabatinelli, Lang, Keil, and Bradley, 2007;Shupp, Junghofer, Weike, and Hamm, 2004), was scored for mean activity in the time interval of 400 - 800 ms after picture onset (corresponding to 800 ms – 1200 ms after fixation point onset). The LPP has previously been used as a physiological marker for the allocation of perceptual and attentional resources to emotionally- relevant stimuli, and as an index of the engagement of basic defensive and appetitive circuits in the brain (Lang *et al*., 1997).

The motor part of the task (movement observation and execution) involved *response-locked* epochs extracted from 1000 ms before to 200 ms after muscle burst onset, using the standard back-

averaging procedure usually employed to study motor related potentials. This procedure comprised the following steps: the EMG was full-wave rectified, and trigger signals indicating the beginnings of the movement (defined by the onset of electromyogram compound muscle action potential (CMAP) activity), were inserted by visually reviewing the continuous recordings for each trial. A threshold level (30% of the peak value) was defined to mark the beginning of each CMAP in a non-biased way. This indicated the onset of movement-related cortical activity, calculated by averaging the signal across trials from the CMAP backward.

For statistical purposes the resulting signal was further divided into two different movement-related potentials: the Readiness Potential (RP), scored as mean activity in the time interval ranging from -500 ms to 0 ms before the trigger, and the Motor Potential (MP), scored as mean activity from 1 ms to 200 ms after the trigger. Separate average waveforms were calculated for each condition (compatible versus incompatible and emotional versus neutral), for each subject and for each electrode.

The RP is a negative wave starting 1 sec before the movement, which has a site of maximum activity in the precentral area extending to the parietal and frontal regions, contralateral to the movement (Shibasaki, Barret, Halliday, and Halliday, 1980). This potential is thought to result from activity in frontal motor and more parietally-extending primary and secondary sensory areas (Green, Arnold, Rozhkov, Strother, and Garrot, 2003). Two main epochs of the RP have been distinguished, an earlier symmetrical component followed by a lateral asymmetric one that starts about 500 ms before movement onset. This analysis will use only the second phase of the RP, which some authors refer to as the "negative slope" (Shibasaki *et al*., 1980). The Motor potential (MP) occurs in the latter phase of motor-related potential activity, and is characterized by the maximum negative peak accompanying the executed movement. Its scalp distribution is more asymmetrical and fronto-central (Shibasaki *et al*., 1980) and it typically accompanies the movement execution from the muscle burst to the end of the

movement. Being the main focus of this chapter looking for differences between emotions and neutral trials inside the compatible condition, where imitation takes place, data were analyzed using ANOVA including Category (emotion vs. neutral), Region (Anterior, Central, Posterior) and Laterality (Left, Mid, Right), as factors for each of the three time windows (covering the LPP, the RP and the MP). These hypotheses driven analyses were necessary to explore the more important effects I was interested in. After having showed that the main effects reported in Study 1 were replicated, I looked at their neural markers following the crucial questions.

The behavioural data were collected from the keyboard of the computer projecting the stimuli. Reaction times were analyzed using an ANOVA with all fifteen subjects, with two within-subjects factors, Compatibility (compatible vs. incompatible movements), and Category (neutral vs. emotional). Only trials with correct responses were included in the analysis; trials with response times below and above two standard deviations from the mean were excluded.

4.3 Results

4.3.1 Behavioral Data

Performance accuracy was high (97%), and ANOVA revealed main effects of Compatibility ($F(1,14) = 19.325$, $p < 0.001$) and of Prime ($F(1,14) = 6.859$, $p < 0.05$). Subjects were faster responding in the compatible condition and after the negative emotional trigger; the interaction of Compatibility and Prime was significant ($F(1,14) = 5.369$, $p < 0.05$) (Figure 2). Within compatible trials, post-hoc tests showed that the emotional and neutral trials differed significantly in compatible trials ($F(1,14) = 8.607$, $p < 0.05$). These difference did not reach significance in incompatible trials ($F(1,14) = 0.403$, $p = 0.532$). Post hoc analyses showed that inside compatible trials the emotional vs neutral trials significantly differ ($F(1,14) = 8.607$, $p < 0.05$), while inside the incompatible trials they do not ($F(1,14)$

= 0.403, p = 0.532). Post-hoc analyses also showed that the compatible trials were significantly different from the incompatible trials in both the neutral ($F(1,14) = 6.120$, $p < 0.05$) and the emotional condition ($F(1,14) = 9.680$, $p < 0.01$). This replicates the pattern of effects previously found in Experiment 2, Chapter 2.

Subjects obtained scores within the normal range on negative and positive affectivity (as scored by the PANAS self administered questionnaire), suggesting that they were not affected by any emotional disorders that could have affected the measures employed in this study.

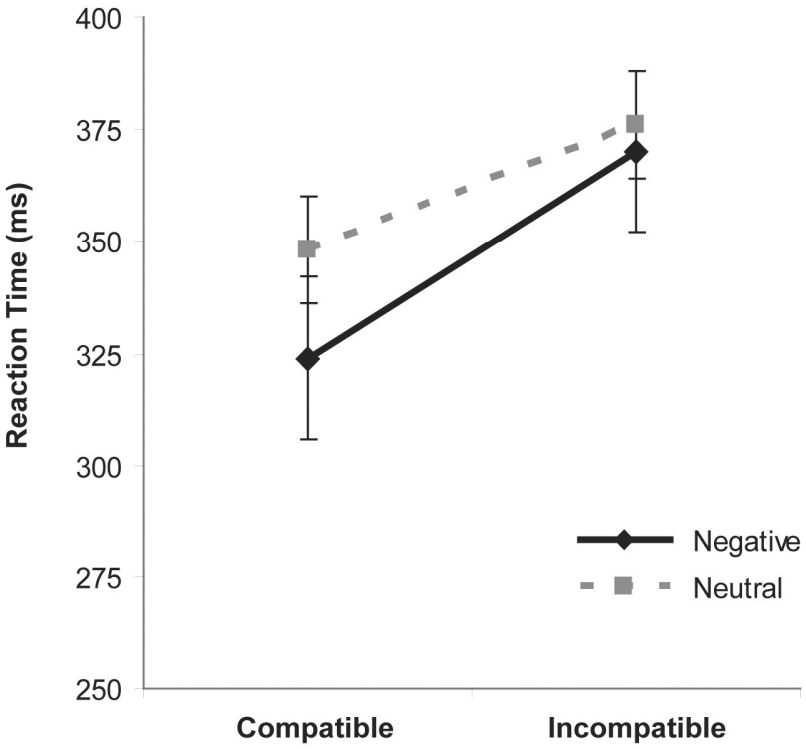

Figure 2. Behavioural results
Subjects were faster responding after emotional stimuli than after neutral stimuli in the compatible condition. Subjects were also faster in the compatible condition than in the neutral condition (a response compatibility effect).

4.3.2 Analysis of ERP data

4.3.2.1 Late positive potential

Emotional pictures elicited a larger Late Positive Potential than neutral pictures (starting from 400 ms after picture onset, not shown), consistent with previous findings using IAPS images (Schupp *et al*., 2000). Mean amplitudes from the time window of interest (400 - 800 ms after picture onset, where LPP is visible) were entered into the analysis. Greenhouse-Geisser adjustment was applied to correct for sphericity violations. The size of the LPP was modulated as a function of the emotional content of pictures ($F(1,14) = 9.778$, $p < 0.01$). Emotional and neutral picture trials also had differing scalp distributions ($F(2,28) = 60.476$, $p < 0.001$); and different lateralization patterns ($F(2,28) = 12.393$, $p < 0.001$). The only significant interaction was ROI x Lateralization ($F(4,56) = 8.379$, $p < 0.001$) [non-significant interactions included Prime x ROI ($F(2,28) = 0.503$, $p = 0.61$), Prime x Lateralization ($F(2,28) = 0.766$, $p = 0.48$), and Prime x ROI x Lateralization ($F(4,56) = 1.931$, $p = 0.122$)]. Post-hoc contrasts revealed significant differences between emotional and neutral conditions in seven out of nine regions on the scalp (see Table 1): left posterior ($p < 0.001$), mid posterior ($p < 0.001$), right posterior ($p < 0.001$), left central ($p < 0.05$), mid central ($p < 0.05$), right central ($p < 0.01$), and left anterior ($p < 0.05$). These data indicate that stimulus processing was modulated by the emotional content of the stimuli.

A differential early modulation of ERP between emotional and neutral trials was also visible, starting from about 70 ms after picture onset. This component, known as Early Posterior Negativity (Schupp *et al*., 2004), was not formally analyzed because the focus of this study was not on emotional content *per se*, but rather on how emotion modulates motor responses.

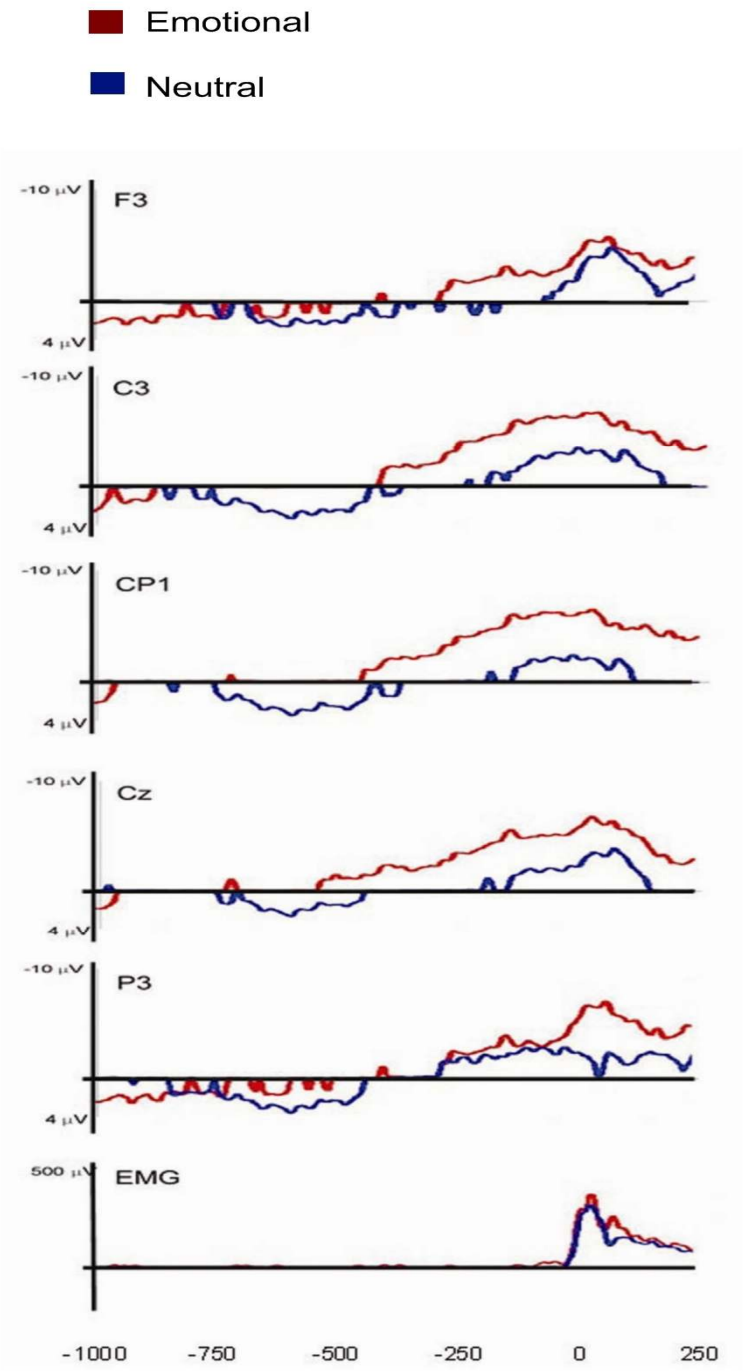

Figure 3. ERP for the emotional vs. neutral trials.

The grand average ERP waveforms for the emotional vs. neutral compatible conditions, showing the Readiness and Motor potentials. Sensors contralateral to the hand used for the motor response were selected based on enhanced RP and MP amplitudes in the emotional condition.

Motor-related Potentials

Statistical analyses were conducted separately according to whether responses occurred in the compatible or the incompatible movement condition, because our previous analyses indicated that reaction times after viewing emotional vs. neutral stimuli differ significantly only in the compatible movement condition.

4.3.2.2 Analyses from the compatible movement condition

Readiness potential data

In the compatible condition, the amplitude of the RP (the wave associated with the preparation of the movement) increased in the emotional condition (see Figure 3, on the left), consistent with the hypothesis of a fear-potentiated overt motor response after aversive emotional stimulation. The amplitude of the RP was significantly different between emotional and neutral stimuli ($F(1,14) = 6.753$, $p < 0.05$), and emotional and neutral trials were associated with different scalp distributions ($F(2,28) = 8.814$, $p < 0.005$) and different lateralization patterns ($F(2,28) = 6.320$, $p < 0.05$). Only the ROI x Prime interaction was significant ($F(2,28) = 4.423$, $p < 0.05$) [non-significant interactions included ROI x Lateralization ($F(4,56) = 0.765$, $p = 0.55$), Prime x Lateralization ($F(2,28) = 1.862$, $p = 0.170$), and Prime x ROI x Lateralization ($F(4,56) = 1.107$, $p = 0.36$)]. After application of Greenhouse-Geisser adjustment, post-hoc contrasts revealed significant differences between emotional and neutral conditions at three out of nine regions of the scalp (see Table 1 - left central ($p < 0.001$), mid central ($p < 0.001$) and left posterior ($p < 0.05$)). These findings suggest that motor planning is modulated by the emotional content of prior visual stimulation. However, it is possible that the differences seen at posterior sites could also be related to the stimulation provided by a moving hand.

Motor potential data

The MP (the wave accompanying the execution of the movement) in the emotional condition was associated with a stronger negative shift relative to the neutral condition as shown in Figure 3 (left), consistent with the hypothesis of a fear-potentiated overt motor response following negative emotional stimulation. The amplitude of the MP was different between emotional and neutral trials ($F(1,14) = 14.750$, $p < 0.005$), but neither the scalp distributions ($F(2,28) = 1.953$, $p = 0.168$) nor lateralization patterns ($F(2,28) = 0.475$, $p = 0.628$) were different. Only the Prime x ROI interaction was significant ($F(2,28) = 4.138$, $p < 0.01$) as well as the three-way interaction Prime x ROI x Lateralization ($F(4,56) = 7.386$, $p < 0.001$), [non-significant interactions included ROI x Lateralization ($F(4,56) = 1.056$, p = 0.389), Prime x Lateralization ($F(2,28) = 0.483$, $p = 0.623$)]. After Greenhouse-Geisser adjustment, post-hoc contrasts revealed significant differences between emotional and neutral conditions in seven out of nine regions of the scalp (see Table 1- left posterior ($p < 0.05$), mid posterior ($p < 0.05$), right posterior ($p < 0.05$), left central ($p < 0.01$), mid central ($p < 0.01$), and left and right anterior ($p < 0.01$)). These results indicate that motor execution is also modulated by the emotional content of the prime. The MP also has a larger effect size between conditions than the sequentially earlier RP, suggesting that the increases in motor potentials are not a simple carryover effect of the LPP, in which case a larger RP (earlier) relative to the MP (later) should have been observed.

4.3.2.3 C3 and EMG latency analysis

The peak latencies of the EMG (the signal recorded from the extensor digitorum of the arm performing the movement), and of C3 (the site near the contralateral motor areas most involved in the execution of the movement) were also analyzed. These measurements were taken from 0 to 3000 ms in order to preserve the real timing of events. Wilcoxon matched-pairs sign-rank tests were used to assess

the significance of the signal latency differences between emotional and neutral trials. The EMG latencies differed significantly (z = -2,478, N-ties= 13, $p < 0.05$, two-tailed), with the muscle burst arriving 25 ms earlier in the emotional relative to the neutral condition, while C3 latencies did not differ (z = -1,584, N-ties = 13, p = 0.113, two-tailed). Additional analyses were run for mean peak amplitude in the EMG signal, which did not differ significantly (z = -1,542, N-ties = 13, p = 0.81, two-tailed).

4.3.2.4 Analyses from the incompatible movement condition

No significant emotional modulation of the amplitude of the Readiness Potential ($F(1,14) = 1.897$, p = 0.206) was found (Figure 4), but scalp and lateralization pattern effects were significant (respectively $F(2,28) = 9.134$, $p < 0.005$, and $F(2,28) = 4.829$, $p < 0.05$). The interactions were not significant (Prime x ROI $F(2,28) = 0.724$, p = 0.5; Prime x Lateralization F(2,28) = 2.272, p = 0.116; ROI x Lateralization $F(4,56) = 1.893$, p = 0.136), nor was the triple interaction ($F(4,56) = 1.454$, $p = 0.239$). The modulation by emotion was not significant during the time window of the Motor Potential ($F(1,14) = 1.924$, $p = 0.232$), nor was the lateralization pattern distribution ($F(2,28) = 3.457$, p = 0.9), but the scalp distribution was significant ($F(1,14) = 6.012$, $p < 0.05$). The two-way and three-way interactions were not significant [Prime x ROI $F(2,28) = 0.585$, $p = 0.582$; Prime x Lateralization $F(2,28) = 3.646$, $p = 0.082$; ROI x Lateralization $F(4,56) = 0.801$, $p = 0.573$, Prime x ROI x Lateralization $F(4,56) = 2.810$, $p = 0.144$].

An ANOVA, with Greenhouse-Geisser correction, on the incompatible and compatible trials in the neutral condition confirmed that the motor potentials significantly differed between the two sets of trials ($F(1,14) = 6,066$, $p < 0.05$); these were associated with different scalp distributions ($F(2,28) = 7,918$, $p < 0.005$), and a trend for different lateralization patterns ($F(2,28) = 2,926$, p = 0.075). No interaction was found to be significant. The motor-related potentials showed a positive deflection in the

incompatible condition, while in the compatible condition we observed the typical negative deflection associated with the preparation and execution of the movement (the readiness and motor potentials).

The scalp was further analyzed for the following electrodes: Fp1 (left prefrontal), F3 (left frontal), C3 (left central), P3 (left parietal), Fz (mid frontal), Cz (mid central), Pz (mid parietal), Fp2 (right prefrontal), F4 (right frontal), C4 (right central), P4 (right parietal). Post-hoc contrasts revealed significant differences between compatible and incompatible conditions in five out of 11 electrodes, Fp1 ($p < 0.01$), F3 ($p < 0.05$), F4 ($p < 0.05$), C4 ($p < 0.05$), P4 ($p < 0.05$). These results indicate that there was a cortical response modulation during the incompatible condition.

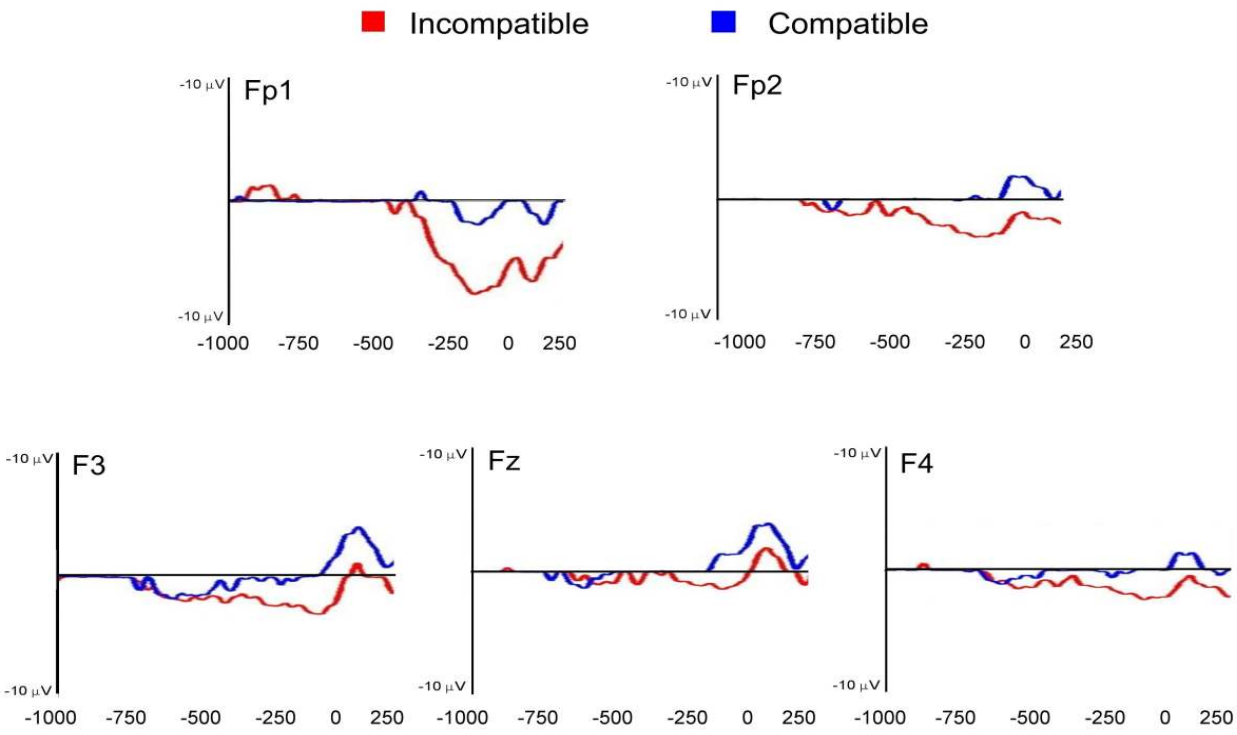

Figure 4. ERP for the incompatible vs. compatible trials.
The grand average ERP waveforms over frontal regions for incompatible and compatible conditions during neutral stimuli.

Motor and perceptual enhancement

To test whether activity associated with the LPP could account for the enhancement of potentials over motor areas when subjects saw emotional pictures in the compatible trials, the correlation of the peak amplitudes of the three potentials for each subject (LPP, RP, MP) was compared using Pearson partial correlations (Table 2). There was a nonsignificant positive trend between RP and MP magnitudes, when controlling for the correlation of each with LPP (r = 0.637, n =10, p = .065, two-tailed). However, there were no significant correlations between LPP and RP magnitudes controlling for MP (r = 0,248, n = 10, p = 0.519, two-tailed), nor between LPP and MP magnitudes controlling for RP (r = -0,041, n = 10, p = 0.917, two-tailed). Since a general increase in arousal should have produced positive correlations between the two motor potentials and LPP, this analysis suggests that a single common mechanism such as arousal could not be responsible for the pattern of brain potential results obtained in this study.

	LPP	**RP**	**MP**
LPP	-	.2488 (10) p = .519	-.0406 (10) p = .917
RP	.2488 (10) P = .519	-	.6376 (10) p = .065
MP	-.0406 (10) p = .917	.6376 (10) p = .065	-

Table 2. Partial correlation matrix between Late positive potential (LPP), Readiness potential (RP), and Motor potential (MP). The Pearson product-moment correlation coefficient (above the diagonal), partial correlation coefficient (below the diagonal), degrees of freedom and 2-tailed significance are reported.

4.4 Discussion

The purpose of this study was to examine the influence of negative emotions on overt voluntary action and its electroencephalographic correlates. The enhancement of action as a consequence of emotional stimulation is a part of many theories of emotions (Fanselow, 1994; Frijda, 1987; Lang *et al*., 1997; McNaughton, and Corr, 2004; Ohman and Mineka, 2001; Mineka, and Ohman, 2002), but experimental studies to date have failed to convincingly detect it (Lemke *et al*., 2005; Grecucci, Cooper and Rumiati, 2007; Rumiati *et al*., submitted).

The present study provides evidence for enhanced imitative responses in the emotional condition relative to the neutral condition, as indexed by Readiness potential and Motor potential amplitude. The Readiness potential was strongly augmented in the emotional condition over the central electrodes contralateral to the hand performing the movement (see electrodes FC1, C3, Cz, CP1 in Figure 3). The signals from the two conditions began to differentiate starting about -750 ms before the muscle burst.

The potentiation of the RP, a signal thought to reflect intentional planning and preparation of movements prior to their execution (Kristeva-Feige, Rossi, Feige *et al.*, 1997), may be interpreted as a stronger preparation to act after fearful stimulation, in accordance with previous theories (Frijda, 1987). The Motor Potential represents final cortico-spinal outflow (Kristeva-Feige *et al*., 1997); it also increased in the emotional condition, suggesting that motor responses are potentiated after emotionally-negative stimulation. To our knowledge, this is the first demonstration of the effect of emotions on imitation. In fact, results speak clearly in favour of a selective modulation of compatible only trials (where imitation takes place) compared with incompatible trials. With analogy to the Fear-Potentiated Startle Reflex (referring to covert action), these Fear-Potentiated Movement-Related Potentials (FPMPs) affect overt action. This negative enhancement of the motor-related potentials cannot be explained as a carryover effect of the LPP (a signal with a positive polarity) – if the RP was affected by the LPP, it

should have been less negative with respect to the neutral condition, and not more negative as was found.

Since the LPP usually lasts for the duration of the emotional stimulus (Bradley *et al*., 2003), its effect is likely to have been minimal when subjects started their movements. The lack of any significant difference in the motor-related potentials for the incompatible condition further suggests that the increased negativity of the RP and MP cannot be explained as an effect of the positive wave elicited by the emotional stimuli. Positively-valenced pictures do not show any effect on RTs (Grecucci *et al*., submitted), and EMG amplitudes were not modulated by emotions, which indicates that this effect is not related to any general increase of the arousal.

Taken together, these results confirm the hypothesis that after negative emotional stimulation there is a larger engagement of resources for motor programming and execution, and demonstrates that there is an important modulation of voluntary actions when subjects are required to respond after processing motivationally-relevant stimuli.

This study also suggests that inhibitory effects of imitative responses can be seen both at a behavioural and at a neural level. Executing a movement incompatible with the one previously observed causes an interference that leads to longer response latencies (Figure 2). Brass *et al*. (2001b) postulated a need for inhibiting the natural tendency to imitate movements, an interpretation which this work strongly supports. The classic negativity associated with movement-related potentials is also smaller for incompatible movements, which may be a physiological correlate of the slower RTs observed in this condition (Figure 4).

Chapter 5

Emotional resonance versus action resonance deficits in autistic children

5.1 Introduction

The aim of the present chapter is to verify whether the paradigm presented in this thesis can be fruitfully extended to study of neuropsychiatric syndromes such as autistic developmental generalized disorders. It has been argued that children with autism are characterized by both emotional and imitational deficits. Since the IAPS pictures used in the previous chapters are not adequate for being used with children, I reverted to facial expressions. Facial expressions do elicit the same emotions in the observer, so they can be used as primes in the same way of IAPS. This changing in the experimental set gives us the opportunity to extend to other kind of emotional primes the paradigm developed in Study 1.

The imitation difficulties of children with autistic spectrum disorder (ASD) have been considered the core deficit of this pathology (Avikainen, Wohlschlager, Liuhanen, Hanninen, and Hari, 2003; Rogers, 1999; Rogers, Bennetto, McEvoy, and Pennington, 1996; Williams *et al*., 2001), even though in some studies no evidence was found (e.g. Aldridge, Stone, Sweeney, and Bower, 2000;

Beadle-Brown and Whiten, 2004; Carpenter, Pennington, and Rogers, 2001; D'Entremont and Yazbek, 2007; Hamilton, Brindley, and Frith, 2007; Pierno, Mari, Lusher, and Castiello, 2008). This inconsistency may be due to the fact that these studies used different methodologies and that emotional and relational factors intervened in the relationship between the autistic individuals and the model to imitate. Although not all patients suffering from autism show imitation difficulties, a theory of autism was proposed, called "the broken mirror" theory (Iacoboni and Dapretto, 2006; Ramachandran and Oberman, 2006; Williams, Whiten, Suddendorf, and Perrett, 2001; Rizzolatti and Fabbri-Destro, in press)), according to which autism is due to a dysfunction of the action resonance mechanism. This would correspond in essence to the so-called mirror neuron system (MNS) that, when damaged, is said to lead to an inability to understand and reproduce actions (imitation) (Iacoboni *et al*., 1999; Rizzolatti and Fabbri-Destro, in press), emotions (empathy) (McIntosh *et al.,* 2006), and intentions of others (theory of mind) (Frith and Frith, 2006). This "action resonance" deficit may, in turn, give rise to a corollary of social and relational dysfunctions. Recently an fMRI study proposed that ASD children have an abnormal activity in MNS (Dapretto *et al*., 2006). The human MNS, comprising the inferior frontal gyrus and parietal cortex bilaterally, has been proposed to be active both when actions are executed and observed, particularly during imitation (e.g. Iacoboni *et al*., 1999).

Neuroscientific studies tried to discover which brain areas of the MNS need to be damaged or altered in order to give rise to abnormal imitation in autism. Unfortunately, studies that aimed at detecting a malfunctioning MNS in ASD have too led to inconsistent findings (Bird *et al*., 2008), probably due to the complexity of this pathology and its variety of symptoms.

Using MEG, Avikainen, Kulomaeki, and Hari (1999) found preserved motor cortex excitability in ASD and control participants when they observed simple hand movements, suggesting a normal MNS activity in the ASD group. In addition, the MNS deficits in autism have been associated with

different brain areas. While Dapretto *et al*. (2006) found that individuals with ASD show normal activity in the parietal mirror area but reduced activity in the inferior frontal gyrus, Williams *et al.* (2006) reported exactly the opposite pattern of results, i.e. normal activity in the inferior frontal gyrus and reduced activity in the parietal mirror area.

A malfunctioning of the MNS in ASD would not be sufficient to explain why these individuals perform poorly in most imitative tests as well as in tests evaluating empathy, and theory of mind. In fact, imitative performance typically encompasses a broad range of cognitive, motivational and praxic abilities, involving perceptual processing of complex stimuli, executive function, attentional control, motor control, theory of mind, language, and the comprehension of social cues (e.g. Rogers, Hepburn, Stackhouse, Spare and Wehner, 2003; Leighton *et al*., 2008). Therefore, impaired social abilities in autism might reflect not only a malfunctioning understanding and reproduction of the intentions of others' actions (i.e. the "broken mirror" theory), but also a wide range of emotional and social mechanisms. We argue that in order to explain all the deficits in ASD children an impairment of the mechanisms that code emotional values derived from any social situation must be hypothesized as a complement of the broken mirror theory.

In agreement with what has been suggested by others (Gillberg, 1992; Greimel et al., in press), we shall call this "emotional resonance deficit" as an extension of the "action resonance deficit". We define *emotional resonance* as the mechanism by which one can understand, empathize and feel the emotion expressed by a model by resonating with him. Recent brain imaging studies on pain perception (but the same seems to apply for disgust, fear and other emotions) have shown overlapping activation patterns when subjects feel their own emotions and observe the same emotions in others (Royet et al. 2003; Singer, 2006; Morrison et al., 2004; Botvinick et al., 2005; Lamm et al., 2007; Ochsner et al., 2008).

Neuroimaging studies show that when we observe sensations or emotions felt by others, as well as when experiencing these sensations and emotions ourselves, we activate common circuits (see Bastiaansen, Thioux and Keysers, 2009, for a recent review). This clearly suggests that seeing someone else experiencing touch, disgust or pain triggers much more in us than a purely theoretical, disembodied interpretation of other people's mental states. Moreover, witnessing someone experiencing an emotion or a sensation is associated with a pattern of activity in our brain that embodies their actions, sensations and affective states (Bastiaansen et al., 2009).

According to the "emotional resonance deficit" hypothesis, autistic subjects can be considered emotionally dysregulated and blind to the environmental cues that normally trigger, guide, and sustain cognitive and motor processes. Evidence in support of this hypothesis comes from several studies that investigated the processing of emotional stimuli such as emotional facial expressions (McIntosh, Reichmann-Decker, Winkielman, and Wilbarger, 2006; Baron-Cohen, 1993; Dawson, Webb, Schellenberg, Aylward., Richards, Dager and Friedman, 2002; Frith, 1989; Hobson, Ouston, and Lee, 1989). Children with autism also showed poor performance on facial imitation tasks (Rogers *et al*., 2003), automatic emotional mimicry (McIntosh, Reichmann-Decker, Winkielman, and Wilbarger, 2006), matching emotional expressions (Celani, Battacchi and Arcidiacono, 1999; Hobson, Ouston, and Lee, 1988; Hobson *et al*., 1989; Loveland, Tunali Kotoski, Chen, Brelsford, Ortegon, and Pearson, 1995), and on attending to the emotional expressions of others (Sparks *et al*., 2002). Moreover, functional differences between an autistic group and a control group in right frontal regions have been reported when subjects performed an emotional imitation task (Dapretto *et al*., 2006). Autistic children were also found to have significantly increased amygdala volume compared with typical and developmentally delayed children (see Brambilla et al., 2003; 2004 for review), suggesting that the

anatomical system involved in fear processing may be abnormal at an early age. These findings seem to be consistent with the "emotional resonance deficit" hypothesis.

The aim of the present study was to test which of the two above mentioned hypotheses (action resonance defits vs emotional resonance defits) better explains the imitative disorders in ASD children theory. In the Experiment, we assessed the ability to imitate bodily movements performed by a neutral or by a fear-facial expression model and in a further assessment we evaluated their ability to name and imitate neutral and fear facial expressions. The prediction was straightforward. If the "emotional resonance deficit" hypothesis is correct, we predict ASD children to be impaired in the emotional compared with the neutral condition, but to normally perform the imitative task itself. If this is the case, impaired imitative performance as showed in previous studies involving strong social components might be interpreted as affected by the poorly controlled emotional factors. The emotional resonance deficit hypothesis can explain all the social deficits as well as imitation deficits observed in previous studies (Avikainen, Wohlschlager, Liuhanen, Hanninen, and Hari, 2003; Rogers, 1999; Rogers, Bennetto, McEvoy, and Pennington, 1996; Williams *et al*., 2001), suggesting that ASD are characterized by an emotional defective system failing to correctly modulate the motor system and imitative abilities.

5.2 The Experiment

In the present experiment, we tested the imitative tendencies of neutral and emotional models by using the paradigm developed in the previous chapters, and originally proposed by Brass, Bekkering and Prinz (2001), using which we showed a modulatory effect of emotional pictures on imitative responses. In this paradigm, subjects were pre-instructed to tap in one block, and to lift in another, independently of whether the movement they saw was tapping or lifting. RTs were faster when seen

and performed movements (i.e. compatible trials: tapping-tapping or lifting-lifting) were the same than when they were different (i.e. incompatible trials: tapping-lifting or lifting-tapping). In addition, observed tapping or lifting movements were preceded by an unpleasant, a pleasant or a neutral picture. The emotional content of primes affected the compatibility between seen and performed movements, with compatible – imitative movements being faster after negative than after neutral primes (see Study 1). This effect has been interpreted as a product of an empathic mechanism which facilitates the identification with the emotion and the behaviour of a model.

This task has been employed in different methodologies and manipulations, and has been proven to be efficient in detecting imitative tendencies (Brass, Derfuss and von Cramon, 2005) and their relation to the emotional meaning of the context or of the model performing the action (Study1 and 3).

While in Study 1 and 3 the emotional pictures were taken from the International Affective Picture System (IAPS, Lang, Bradley, and Cuthbert, 1997), in the present study we employed emotional and neutral faces. This paradigm, in which the face expression and the hand movement are presented serially, was preferred because a pilot study demonstrated that, in this condition, subjects pay attention to both events, whereas, when they are presented simultaneously, subjects may ignore one or the other.

We predicted that ASD children would perform differently from normal controls in the emotional compared with the neutral condition. In particular, we predicted that controls' reaction times would be faster in the emotional relative to the neutral condition in imitating a model (Study 1 and 3). By contrast, ASD children's performance is not expected to change across the two conditions, thus supporting our hypothesis that autism is characterized, among other factors, by an impaired emotional resonance mechanism.

5.2.1 Method

Participants

Fifteen high-functioning Autistic children (14 Males and 1 Female, mean age = 7.28, SD ± 1.86) assessed for autism severity (CARS) and I.Q. (WIPPSI and WISC according to their age), and fifteen (14 Males and 1 Female, mean age = 7.24, SD ± 1.83) typically developing children matched 1:1 for age, sex and handedness were tested. The two groups did not differ in I.Q. levels ($p>0.05$). All participants in the ASD group had previously received a diagnosis in accordance to the DSM-IV standard criteria validated by consensus meeting of a child psychiatrist and a child psychologist. The mean score on the Children autistic rating scale (CARS) was of 41.64 (SD ± 5.06). The experiment reported here was part of a wider project in which the same children were assessed also for their ability to imitate neutral transitive and intransitive gestures, as well as meaningless gestures (see Carmo *et al*., in preparation).. The experiment was performed with the approval of local ethical committee, and parents signed an informed consent. Children were carefully introduced to the experimenters and to the experimental set up; they were also given rewards (small toys and candies) to facilitate their collaboration and to maximize their performance.

Stimuli

Forty neutral colour pictures, taken from the NimStim Set (Tottenham et al., 2009), depicting half male faces and half female faces, were used as primes. Only the stimuli correctly rated at 95% confidence in a previous pilot study were used. The hand movement consisted of five frames depicting the finger of left hand in a specular position.

Design and procedure

In each trial, a neutral or negative facial expression was presented for 500 ms on the computer screen, followed by a digitized video sequence of five frames of an animated hand presented for 1.105 ms; at the end of which participants were instructed to perform the pre-instructed movement. A blank screen that lasted for 3 sec was then presented (see Figure 1). Each block contained 80 trials, 40 emotional and 40 neutral, of which half were compatible and half were incompatible. Subjects were required to execute an index finger tapping with their right hand and were asked to begin their responses only at the offset of the prime movements. The order of compatible and incompatible movements was counterbalanced across subjects.

The type of observed movement (i.e. lifting or tapping) varied within each block in a compatible or incompatible fashion. Subjects performed the finger tapping movement while both randomly compatible and incompatible finger movements were shown (lifting and tapping). Compatible trials were those in which they performed a tapping after seeing a tapping movements, and incompatible trials when they performed a tapping after seeing a lifting movement. This paradigm allows assessing implicitly the imitative tendency. To contain the cognitive load and the length of the experiment which may affect performance in children, we used only one type of movement to be performed by subjects (tapping). In the previous chapters I was able to show that subjects behave in the same way relative to compatibility effects. The present experiment was controlled by a Dual duo core laptop, with 2 GHz and 4 MB RAM, graphic card Matrox G550 of 128 MB, and 15 inches Monitor of 100Hz, and the E-prime software was used for stimulus presentation and for data acquisition. After parents signing the informed consent form, subjects began to perform a 20 trials training session after which they performed the experiment proper.

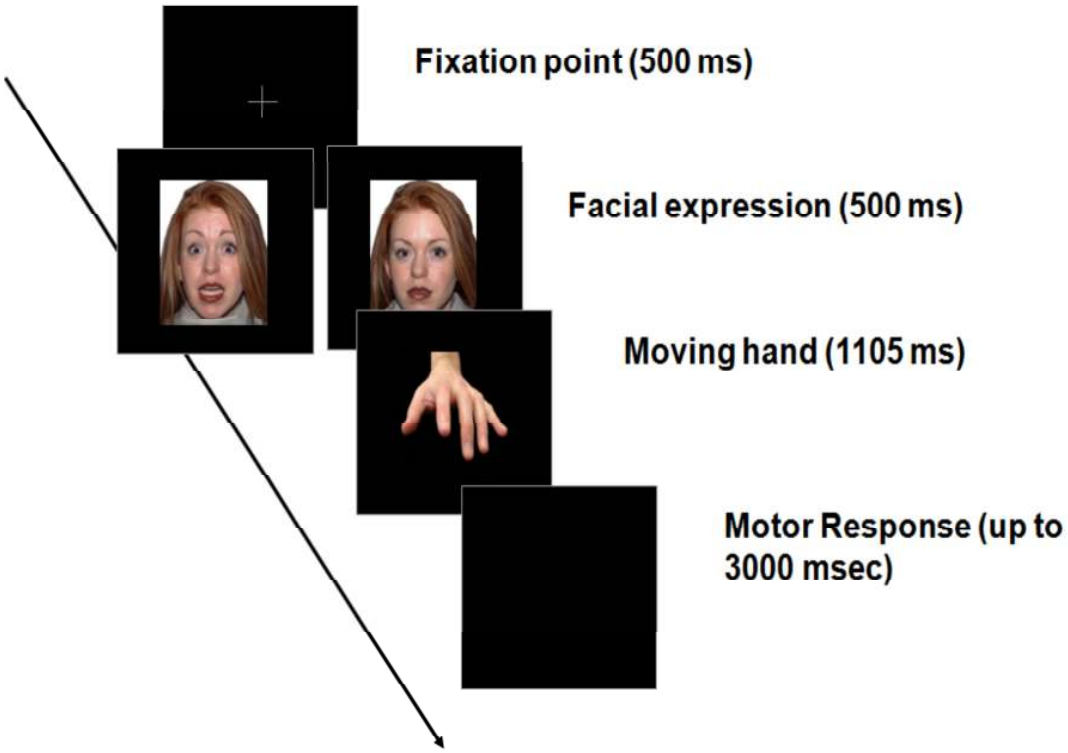

Figure 1. The task
The procedure employed in the Experiment: After a fixation point, the neutral or fear facial expressions of the model were presented for 500 ms. The animated hand performing randomly a finger tapping or lifting was presented at the end of which subjects performed the pre-instructed tapping movement.

Data analysis

A mixed ANOVA with Compatibility (compatible vs. incompatible movements) and Model (neutral vs. emotional negative) as within-subject factors, and Group as a between-subject factor was computed. In order to explore single effects inside each group, two-ways ANOVA and simple effects ANOVA were also computed.

5.2.2 Results

The mixed ANOVA returned a significant main effect of Compatibility ($F(1, 28)= 18.99$, $P < 0.001$) and a significant Model x Group ($F(1, 28) = 4.44$ $P < 0.05$) interaction. No other main effect or interaction was found to be significant (all P values > 0.1). Driven by our hypothesis of resonance deficits in the ASD children absent in CTRL, we run two separated ANOVAs, one for each group, with Compatibility and Model as factors. For the Control group, the two main effects were significant ($F(1,14) = 16.79$, $P < 0.001$ and $F(1,14) = 9.73$, $P < 0.01$ respectively), while for the ASD group only the Compatibility factor was significant ($F(1,14) = 6.07$, $P < 0.05$). Both groups showed normal imitative effects, with RTs in the compatible trials being faster than in the incompatible trials; however, ASD failed to show faster responses in emotion relative to neutral contexts as controls did. See Figure 2 for details.

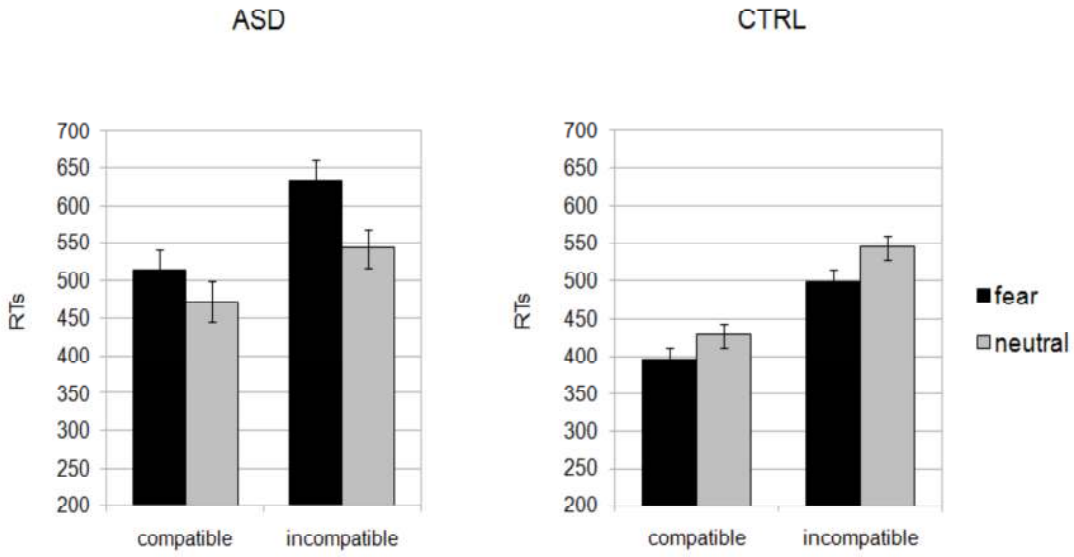

Figure 2. Reaction times results
In the Experiment, both groups showed normal imitative performance in the neutral condition, with compatible (imitative) movements being faster than incompatible movements. While controls showed also the effect of

emotions on motor responses (faster RT), ASD children failed to show this effect (they were slower instead of being faster).

Unfortunately the interaction for both groups was not significant ($F(1,14) = 1.448$, $P < 0.249$ for autistic and ($F(1,14) = 0.265$, $P < 0.61$ for CTRL).

5.2.3 Discussion

The results from this experiment are consistent with the "emotional resonance deficit" hypothesis. In fact, while both groups showed the compatibility effect, with RTs being faster in the compatible than in the incompatible trials, ASD children's RT performance resulted particularly impoverished in the emotional condition. No statistical difference was found in this population even if ASD were slower in the emotional relative to the neutral condition, while healthy children in the emotional condition were faster. This is in line with results coming from previous chapters. We propose that the ASD children in this study have a deficit in understanding and recognizing facial emotional expressions. In fact, no significant difference was found between RTs in the emotional and those in the neutral condition. To prove whether this interpretation was correct we assessed ASD children ability to recognize the emotional facial expressions shown by the model performing the hand movement.

Unfortunately, the interaction between model and compatibility for both groups did not reach significance. This contrasts with the results coming from adults as shown in the previous chapters. I think this should be considered a developmental issue. Taken separately, imitation and emotional processes look similar to adults (different neutral vs emotional trials, as well as different compatible vs. incompatible trials). What is missing is clearly the interaction between these two processes: the emotional on one side and the imitative on the other. One can speculate that during infancy they are

separated and do not interact, but, during development they start working in a synergetic way. This may come from a gradual improvement in the social abilities that come learned in their interaction with the others. Being a lack in the literature on these issues, this consideration remains at a very speculative level.

5.3 Further assessment

When viewing emotional expressions, adults quickly and spontaneously activate congruent facial muscles (i.e. they smile to a smile and scowl to a scowl). Automatic mirroring occurs even when expressions are presented without instructions to mimic (Dimberg, 1982) or when they are presented subliminally (Dimberg, Thunberg, and Elmehed, 2000). Automatic facial mimicry facilitates social interaction, including interpersonal rapport, emotional contagion and recognition (Lundquist and Dimberg, 1995; McIntosh, 1996; McIntosh, Druckman, and Zajonc, 1994; Niedenthal *et al*., 2005). Although the importance of rapid, automatic emotional mimicry for normal social functioning individuals has been theorized, it has not been yet understood whether this mimicry is affected in autism (Moody and McIntosh,2006). There is evidence that these different aspects concerning the processing of emotional faces might be impaired in ASD (Baron-Cohen, 1993; Dawson, Webb, Schellenberg, Aylward, Richards, Dager, and Friedman, 2002; Frith, 1989; Hobson, Ouston, and Lee, 1989). For instance, some studies documented an impaired ability to explicitly imitate facial expressions in autism (Rogers, Hepburn, Stackhouse and Wehner, 2003; Williams, Whiten, Suddendorf and Perrett, 2001), especially for fear and other basic negative emotions (Ashwin, et al., 2006).

To test for the possible presence of deficient recognition and imitation of facial expressions, ASD and control children were asked to name 60 neutral, fear and happy facial expressions and to imitate them. If ASD children had an impaired emotional resonance mechanism they should have

difficulties only in imitating emotional facial expressions but they should be able to recognize and name them.

5.3.1 Methods

Stimuli

In addition to the faces used in the Experiment, 20 happy facial expressions of the same models were also used to have a more appropriate baseline for imitation of fear facial expressions.

Procedure

Subjects were asked to name facial expressions (naming task), and to imitate them (imitation task). Sixty stimuli were presented in a random order for unlimited time until response was provided. The same monitor and computer used in the Experiment were also used in this assessment. If the participant could not name or imitate a given stimulus, the following stimulus was presented. The dependent variable was their accuracy on performing the tasks. We were not able to record subject's performance due to logistic constraints. An observational facial coding system was then used. Each response was scored online by two experimenters who judged and scored the children's facial expressions seen. Subjects had to show a complete reproduction of both mouth and eyes typical movements associated with fear and happy expressions. Experimenters were previously trained to detect and score as objectively as possible the facial expressions following standard facial expressions coding systems such as the Facial Action Coding System by Ekman and Friesen (1978). Special attention was paid to known relevant action units (and associated muscles) responsible for each of the two facial expressions such as the inner brown raiser (Levator palpebrae superioris), the upper lid raiser

(frontalis pars medialis) for eyes, and the jaw drop (Masseter, relaxed Temporalis and internal Pterygoid) for mouth for the fear expressions, and the cheek raiser (Orbicularis oculi, pars orbitalis) and the lid tightener (Orbicularis pars parpebralis) for eyes and the lip corner puller (Zygomaticus major) and the cheek puffer (Levator anguli oris) for mouth in the happy expression. The two experimenters were sitting on the two sides of the participants to detect the facial muscles movements.

5.3.2 Results

Accuracy rates were entered in a mixed ANOVA with Model (neutral, fear, happy) and Task (naming, imitation) as within-subject factors, and Group (ASD, CTRL) as a between-subject factor.

The main effect of Model ($F(1,28) = 4.59$, $P < 0.01$), the Group x Task interaction ($F(2,56) = 5.57$, $P < 0.05$, the Task x Model interaction ($F(2,28) = 5.43$, $p<0.01$) and the three-way Group x Task x Model interaction ($F(2,56) = 3.69$, $P < 0.05$) were found to be significant. Two separated ANOVAs were run for each group: for the control group neither the simple effects not the interaction were found to be significant (all $P > 0.05$), while for the ASD group the main effect of Task $(1,14) = 4.9$, $P < 0.05$, Model ($F(1,14) = 3.97$, $P < 0.05$) as well as the interaction Model Task ($F(1,14) = 6,13$, $P < 0.01$) were found significant. To further explore this latter result, t-tests were run. ASD children were significantly better at imitating neutral than fear expressions ($t(14)= 3.3$, $P < 0.01$), and happy than fear expressions ($t(14) = 2.32$, $P < 0.05$), whereas no differences in naming the three types of expressions were found ($P > 0.05$). Interestingly, only naming fear versus neutral facial expressions significantly differed ($t(14)= 3.6$, $P < 0.001$. These results are plotted in Figure 3).

A correlation between response time in the compatible fear expression condition (from the experiment) and the accuracy in imitating the same expressions was computed. This returned a high

correlation between how the subjects were good at imitating the fear expression and how fast their response was in the bodily imitation task ($r = -0.54$, $P < 0.05$).

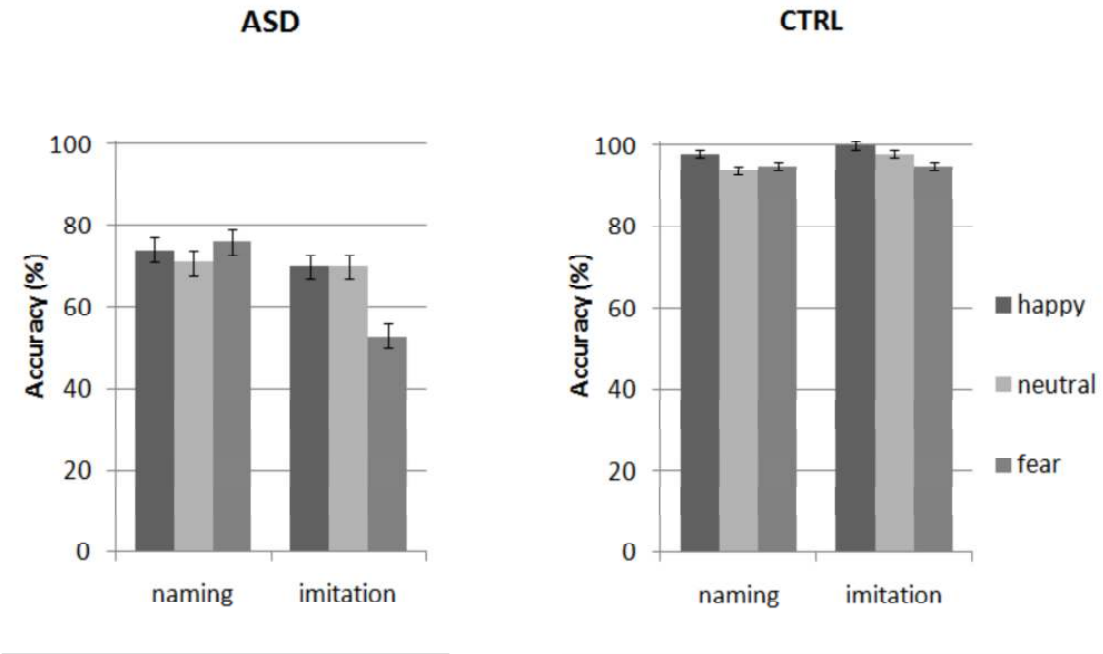

Figure 3. Further assessment
While controls performed accurately both on naming and imitation tasks, showing no differences between emotional conditions, ASD children showed a selective deficit at imitating fear expressions only, although they were still able to name them all and to imitate the neutral and happy ones correctly. See the text for details.

5.3.3 Discussion

All subjects could recognize facial emotional expressions correctly. Similarly to controls, ASD children were proficient in performing the imitation task when the model was neutral and happy, suggesting that the putative action resonance mechanism is intact in this population. However, unlike controls, ASD children had difficulties at imitating facial fear expressions, thus suggesting that their problem was caused by an emotional resonance deficit specific for fear. The fact that they were good at naming all expressions suggests that ASD children's reduced imitation ability is not due to their inability to understand the expressions per se. These findings are also consistent with the "emotional resonance deficit" hypothesis.

Recent studies have documented emotion recognition impairments mainly in the perception of negative emotions, especially fear (Baron-Cohen *et al.*, 2000; Dawson *et al*., 2004; Welchew *et al*., 2005; Ashwin *et al*., 2006, 2007; Corden *et al*., 2006; Humphreys *et al.*, 2007; Hadjikhani *et al.,* 2009). This may be due to morphological abnormalities (Nacewicz et al., 2006; Shumann et al., 2004), or in the functionality (Baron-Cohen et al., 2000; Piggot et al., 2004; Wang et al., 2004) of the amygdala.

5.4 General Discussion

In this study we aimed at extending the "broken-mirror" theory that explains ASD deficits coming from "action-resonance deficits" (Iacoboni and Dapretto, 2006; Ramachandran, and Oberman, 2006; Williams, Whiten, Suddendorf, and Perrett, 2001) to the specific domain of emotions suggesting the hypothesis of an "emotional resonance deficit". Beside damage to the action resonance mechanism whose putative substrate is the MNS the "emotional resonance deficit" hypothesis predicts that these children have impaired recognition and imitation of actions with an emotional meaning.

The data reported in the present study are in support of this hypothesis. In the Experiment, we showed that autistic children performing an imitative task showed an abnormal pattern in the emotional

response. However, both groups were faster when they performed imitative (compatible) movements than non imitative (incompatible) movements in the neutral condition, showing intact imitative tendencies. The overall imitative advantage was of 109 ms for controls and 104 ms for ASD (regardless of the type of emotion).

Previous studies documented a *generalized* imitation impairment in the autistic population (Williams *et al*., 2004) as well as spared imitation (Bird *et al*., 2007). Therefore, whether or not a particular voluntary imitation task presents a challenge for individuals with ASD may depend on other factors, like, for example, the emotions expressed by the experimenter or in the testing situation itself. In fact, the only difference between ASD and controls comes from the emotional condition only: while controls were faster in responding following a fear model, autistic were not. We propose that the "emotional resonance deficit" hypothesis explains better the emotional and social deficits of ASD children.

Another difference between our study and those showing impaired performance depends on the type of task used. While in our study (see also Bird *et al*., 2007 for similar results and task) an implicit imitation task was used (in the experiment subjects were not actually told to imitate), the other studies employed an explicit imitation task (except the one from Bird *et al*., 2007).

To control for possible deficits in recognizing emotional expressions in autistic children that may explain for their abnormal performance in the experiment, in the further assessment we verified whether they could name and imitate neutral, fear and happy facial expressions. We found that, relative to controls, ASD children showed a selective deficit at imitating fear facial expressions; in contrast, no differences in recognizing/naming emotional relative to neutral or happy facial expressions were observed between the two groups.

Our findings support the interpretation that ASD children have a deficient ability to resonate with the emotional facial expression of others (as suggested by the lack of an effect of the fear compared with the neutral condition in the experiment), but also a strong deficit in voluntary reproducing the facial mimicry (further assessment). This is in contrast with the observations of McIntosh *et al*. (2006) who used electromyography to record from facial muscles to obtain implicit measure of mimicry. They found that adults with autism did not show automatic mimicry of emotional facial expressions, but typical adults showed this mimicry effect even if when they were required to explicitly mimic the expressions, no differences were found (McIntosh *et al.*, 2006). The considerable difference of age of the children who took part in our and in their study might explain the different results. Compared with controls, adult autistics may have learned how to cognitively control facial expressions at an older age. Interestingly, imitation of finger movements primed by emotions (the experiment) and imitation of emotional facial expressions (further assessment) highly correlated, suggesting that bodily imitation and facial imitation rely upon the same empathic-resonant mechanism.

This deficit may explain why autistics have severe problems in social contexts and in engaging in any relationship. Several authors have argued that automatic mimicry facilitates social functioning, including interpersonal rapport, fast learning and understanding of other minds (Decéty and Chaminade, 2003, Lakin, and Chartrand, 2003). During development, a mimicry deficit could impair a child's ability to grasp others' emotions, and if such a deficit occurred early, it could impair the child's ability to form self–other correspondences, perhaps contributing to autism (Rogers, 1999).

At a more speculative level, Adolphs, Damasio, Tranel and Damasio (1996) proposed that, during development, the child acquires the link between the facial expression of fear and the personal experience of fear. This would require a neural structure that can perceive a specific facial expression, as well as a structure that can link the facial expression (encode and retrieve) to the affective

experience of that specific facial expression. Aggleton and Young (2000) also suggested that the amygdala might be critical for making such associations between a specific stimulus and the affective experiences intrinsically associated with that stimulus. Compared with typical and developmentally delayed children, autistic children were found to have significantly increased amygdala volume, in excess of increased cerebral volume (Nacewicz *et al*, 2006; Shumann *et al*., 2004). This suggests that the anatomical system involved in fear processing may be abnormal at an early age.

Concluding, autism is a disorder characterized by specific impairments in processing social and emotional information (e.g. Baron-Cohen, Tager-Flusberg and Cohen, 1993; Dawson, Meltzoff, Osterling, Rinaldi and Brown, 1998), with early-appearing impairments evident in social orienting (Dawson, Toth, Abbott, Osterling, Munson, Estes and Liaw, 2002), joint attention (Dawson, Meltzoff, Osterling and Rinaldi, 1998), responses to the emotional displays of others (Sigman, Kasari, Kwon and Yirmiya, 1992) and face recognition (Dawson, Carver, Meltzoff, Panagiotides, McPartland and Webb, 2002; Klin, Sparrow, de Bilt, Cicchetti, Cohen and Volkmar, 1999). These social impairments, some of which are apparent from the first months of life (Osterling, Dawson and Munson, 2002), suggest that autism is related to early dysfunction of brain circuitry involved in social cognition (Baron-Cohen, Ring, Bullmore, Wheelwright, Ashwin andWilliams, 2000).

The “emotional resonance deficit” hypothesis can account for such deficits.

Chapter 6
Fear conditioning effects on imitation

6.1. Introduction

After having shown in the previous chapter that emotional facial expressions can enhance compatible-imitative tendencies, in the present study I aimed at investigating whether neutral facial expressions associated with an unpleasant stimulation may have a similar effect.

A key question that remains to be addressed is whether this facilitation is produced only by hard-wired emotional stimuli or whether it can be extended, through experience, to neutral or novel stimuli associated with an emotional context, conveyed, for example, by the facial expression of someone near us. The ability to associate emotional significance to a stimulus has been studied extensively using the classical Pavlovian conditioning (see LeDoux, 1995, for a review). When a neutral stimulus is paired with an aversive unconditioned stimulus (US), the neutral stimulus becomes conditioned (CS) and elicits a fear response (conditioned response, CR), normally elicited by the US (Estes and Skinner, 1941; McAllister and McAllister, 1971). Once the neutral stimulus has become conditioned, it elicits both autonomic and behavioural responses (e.g. Ohman, 1979; Hodes et al., 1985; Grillon et al., 1991). In Experiment 1 we tested whether, using the classic fear conditioning paradigm, conditioned neutral facial expressions can influence performance on the same imitative task. If this is

the case, I can speculate on the fact that even if the ECOI is probably hardwired, and coming from natural selection as a more adaptive mechanism, it can be learned through experience for previously considered neutral stimuli that change their valence in time. Finally, in Experiment 2, we will test for extinction of the learned association.

6.2 Experiment 1: fear conditioned imitation

Since I was able to show that emotional facial expressions do affect imitative tendencies (Study 4, chapter 5), I wanted to test also if we can associate a neutral facial expression to an emotional context so that we learn to modulate our imitative tendencies according to it. The classic fear-conditioning paradigm was applied.

This experiment included a pre-conditioning phase (which served as a baseline), an intermediate fear-conditioning phase, a post-conditioning phase (which served as a test) (see Figure 1), an extinction phase and a further test phase. A fear conditioning procedure was applied during the intermediate phase and involved pairing white noise bursts (fear eliciting unconditioned stimulus) to half of the stimuli used in the pre- and post-conditioning phases. We predicted that subjects would transfer the fear reaction (unconditioned response, UR), earlier associated with the noise shock (unconditioned stimulus, US), to half of the pictures (CS), which provoke a fear reaction, or conditioned response (CR), just as the US do. Similarly to what has been found in other (Study 1 and 3), in which negative emotional pictures primed imitative responses, here the conditioned neutral facial expressions were expected to speed up the subjects' imitation performance. On the other hand, no differences in imitation were expected following the presentation of non-conditioned stimuli in the pre- and the post-conditioning phases.

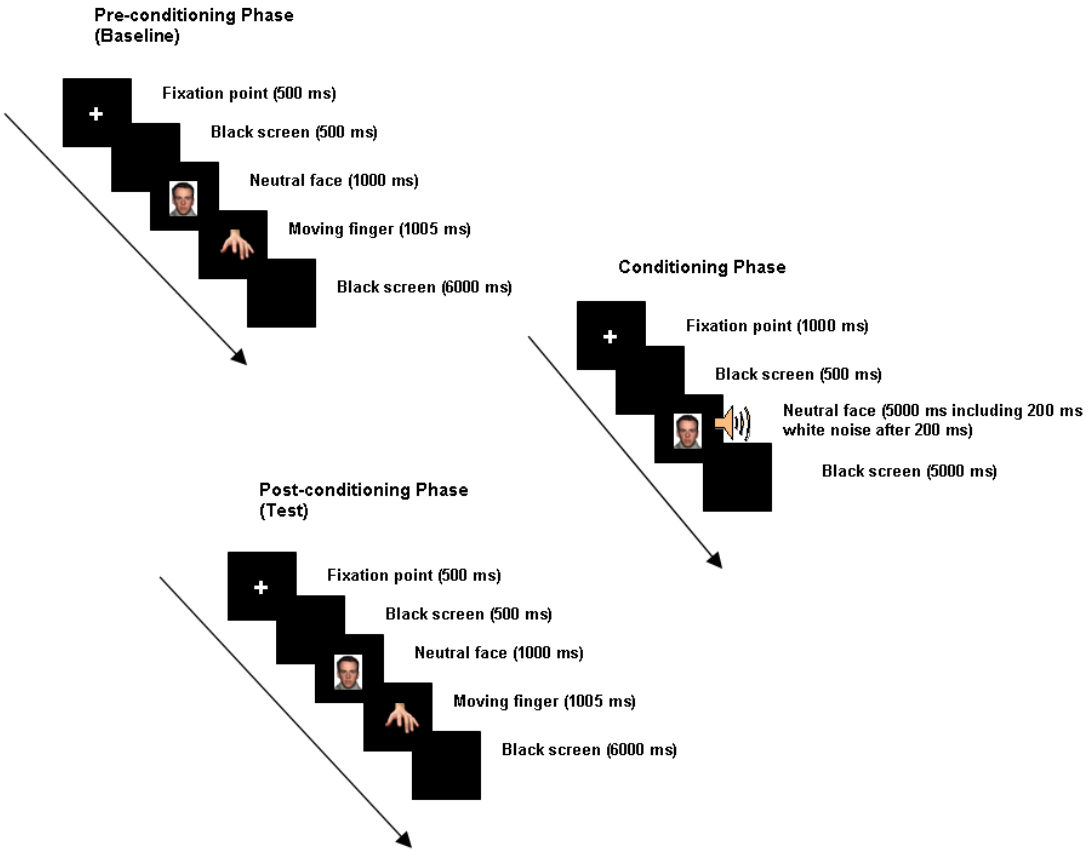

Figure 1. Experiment 1. A fear conditioning paradigm was applied. In a Pre-conditioning phase subjects were tested with the same paradigm of Study 5, but using only neutral facial expressions. In the Conditioning phase, subjects were shown half of the pictures of Phase 1 paired with an aversive stimulus (a white noise). In the Post-Conditioning Phase subjects were retested with the same paradigm of Phase 1.

In the second retest, we checked for a possible extinction of the fear-conditioned response after one month (extinction phase). If a CS is presented repeatedly in absence of the US to a fear-conditioned subject, the amplitude and frequency of the conditioned fear are gradually expected to decrease (Kim and Jung, 2006): this process is known as extinction. After performing Experiment 1, subjects are likely to see many faces that are not associated with the unconditioned stimulus (i.e. fear eliciting noise); thus, after a reasonable period of time, the imitative response that has been enhanced through

fear-conditioning would be extinguished. If the learned enhanced imitation is subjected to extinction I can speculate that imitative mechanisms are notably flexible and that can provide an efficient way of responding to an ever changing environment by means of regulating social learning mechanisms.

6.2.1 Methods

Participants

Twenty-two subjects (13 females), with mean-age of 22 years (SD ± 4) and 13 years of education (SD ± 4), took part in the study after providing written informed consent. They were all right-handed, had normal or corrected-to-normal vision, and were paid five euros for taking part to the study. Subjects were administered with two tests that assess anxiety traits of personality which may affect the way they react to emotional stimuli by measuring the 'negative affectivity' (PANAS scale by Watson and Clark, 1994). They signed the informed consent after been introduced to the experimental set-up and procedure.

Stimuli

Thirty neutral colour pictures, taken from the NimStim Set depicting half male faces and half female faces, were used as primes. Only the stimuli rated as neutral at 95% confidence previous pilot study were used. Each face identity was repeated twice in each experimental phase, for a total of six times. After each prime, subjects saw the video-sequence of an animated hand that consisted of five frames, the first of which showed the index finger in a middle, resting position, that was identical for both movements (lifting and tapping), and remained visible for 500 ms; the subsequent three frames flashed for about 40 ms each. The last frame of the video-sequence contained a picture of the final finger position and remained on the screen for 500 ms. Overall displacement of the finger was about 2°

for the lifting and for the tapping movement. The animated hand was the left one, while the participant responded with the right hand which was kept on a platform in front of her/him.

Design and procedure

All procedures were approved by the SISSA ethical research committee. The sequence of events and procedure of the three phases (pre-conditioning, conditioning and post-conditioning) of Experiment 2 are described in Figure 2 (see also the legend). The same procedure and instructions were given to participants for the first and the third phase. For the intermediate phase, subjects were required to look at the pictures which were visible for 5 sec, while they listened to the white noise burst (the unconditioned stimulus) for half of the stimuli. The gender of subjects and the gender of the conditioned faces were balanced (half male subjects conditioned for female and half for male faces, the same for female subjects). The unconditioned stimulus was an 80 dB binaural white-noise burst presented via headphones after 200 ms from the onset of face presentation. In a pilot study we established that a 80 dB noise (controlled by a Voltcraft SL-200, with a range of measurement from 30 to 130 dB, resolution 0.1, and a frequency range from 31.5 to 8000 Hz) was sufficient to elicit a fear subjective and autonomic response without being perceived as painful for the subjects. We used a delay conditioning paradigm in which the presentation of the unconditioned did not overlap with the conditioned stimuli. After 200 ms of stimulus presentation, the white-noise burst was delivered. The post-conditioning phase was identical to pre-conditioning phase, except that in the former, half of the faces had been conditioned.

This experimental design allowed comparing the target and non-target faces after the conditioning (test phase), as well as comparing the faces in the pre- and post- conditioning phases.

Participants were asked to perform a preinstructed movement (i.e. tapping of the index finger) after observing either a tapping or a lifting index movement. RTs measured the time from stimulus offset until key-pressing. To facilitate the position and the movement of the hand, and to prevent muscle fatigue, a rigid platform was built to best positioning the hand. Subjects performed a total of 180 trials (60 for each phase).

Prior to the experiment proper, subjects performed 20 training trials with the supervision of the experimenter who carefully monitored the execution of the correct movement. After the experiment, they were debriefed and asked whether they felt anxious or scared during the experiment.

In Phase 3 (post-conditioning), 14 out of 22 subjects who took part in Phase 1 (pre-conditioning) were tested, using the same procedure and stimuli as in the post-conditioning phase, and only RTs were recorded. Additionally, a Retest phase was run after one month of the original experiment in order to control for eventual extinction of the conditioned stimuli.

A Pentium III computer, with 800 MHz and 512 MB RAM, graphic card Matrox G550 of 32 MB, and Monitor of 100Hz, controlled the experiments and the E-prime software was used for stimuli presentation and data acquisition.

6.2.2 Results

Two separate ANOVAs were performed, one on the RTs for the pre-conditioning session and one for the post-conditioning session with 2 Target (conditioned faces vs. not-conditioned faces) x 2 Compatibility (compatible vs. incompatible) as within-factors.

Preconditioned. None of the effects was found to be significant (Compatibility, $F(1,21)=2,389$, $p=0.161$; Target,$F(1,21)=0,471$, $p=0.5$; Compatibility X Target, $F(1,21)=0,0001$, $p=0,97$) (see Figure 3 left).

Postconditioned. There was a significant main effect of Compatibility ($F(1,21)=4,559$, $p < 0.05$) (281 ms vs. 308 ms, see Figure 3 right), as well as a significant Compatibility and X Target interaction ($F(1,21)=6,127$, $p<0.05$), while the factor Target was not significant ($F(1,21)=2,070$, $p=0.165$). To further explore the interaction, I performed separate single ANOVAs. Only the difference between target and non-target faces in the compatible movement was significant ($F(1,21)=6,449$, $p<0.05$), with target faces being faster than non target faces, thus suggesting that the fear conditioning brought about the compatibility effect. The difference between compatible and incompatible in the post-conditioning session was also significant ($F(1,21)=8,771$, $p<0.01$), confirming that the effect of fear conditioning was specific to the imitative process and not a general effect (no significant difference was found between the incompatible movement for the target and non-target faces in the pre nor in the post-conditioning sessions). See Figure 2 for details.

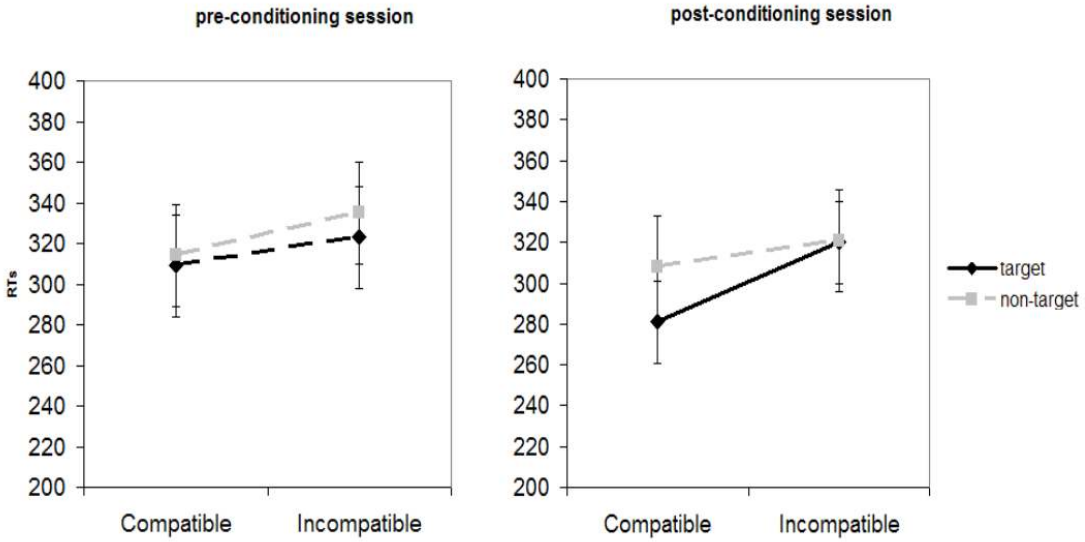

Figure 2.
Results from Experiment 1 are shown. On the left, the results from the Pre-conditioning session are reported. No difference was found between faces to be conditioned in the Conditioning phase and the ones not to be conditioned. On the right, results from the Post-conditioning session are reported showing difference between conditioned versus not conditioned faces.

6.2.3 Discussion

In Experiment 1, test phase, seeing neutral stimuli previously conditioned to a fear response led subjects to respond faster in the compatible-imitative condition compared with the incompatible condition. This imitation advantage occurred only in the compatible, but not in the incompatible movements, with post-conditioning responses being on average 37 ms faster than pre-conditioning responses. In the incompatible condition, no differences were found between the pre-conditioning and post-conditioning phase (see Figure 2).

For some reason the compatibility effect was not significant in the preconditioning phase. Being such a robust effect as shown along this thesis, this is something to be explained. I think that this happened because of the lacking of emotional stimuli (subjects saw only neutral faces), that made this phase too much monotonous and subjects less attentive to the task.

6.3. Experiment 2: extinction

Subjects were asked to perform the experiment one month later, in order to be tested for the presence of the fear-conditioning effect on their imitation responses or of its extinction. If a conditioned stimulus is presented repeatedly in absence of the unconditioned stimulus to a fear-conditioned subject, the amplitude and frequency of the conditioned fear gradually decreases (Kim and Jung, 2006). This process is called extinction.

Given this, we should expect that the experimentally induced fear conditioned imitative response (conditioned response) found in Experiment 1 should get extinguished in a short period of time if the faces (conditioned stimulus) is no longer associated with the threatening noise (the fear eliciting unconditioned stimulus).

6.3.1 Methods

The same paradigm used in the pre-conditioning / post-conditioning phase was used here. Only 14 subjects underwent the second experiment.

6.3.2 Results

A repeated measure ANOVA was performed with Target and Compatibility as factors. The main factor of Compatibility resulted to be significant ($F(1,12)=3,759$, $p<0.05$), while Conditioning ($F(1,12)=0,050$, $p=0,826$ n.s.) and the interaction ($F(1,12)=0,005$ $p=0,944$ n.s.) were not (see Figure 3).

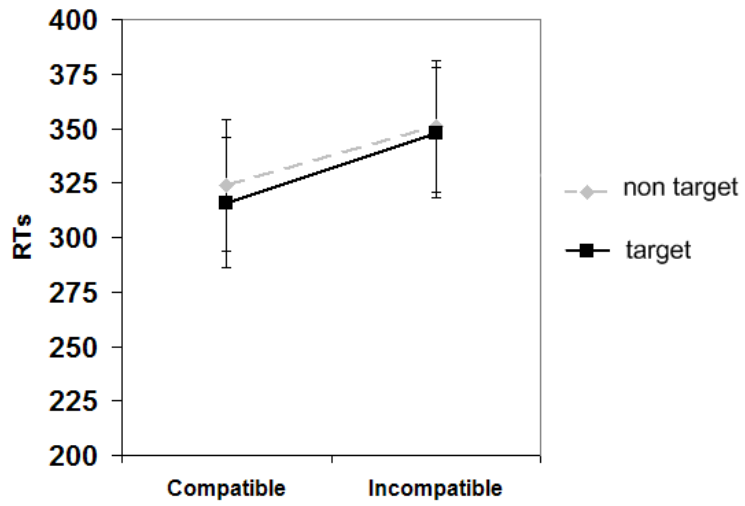

Figure 3.
Results from the retesting phase after one month. The effect of the conditioning has extinguished itself. The difference between target and non-target faces is no longer shown.

6.3.3 Discussion

In this experiment we showed that after one month since Experiment 1 the effect of the conditioned faces disappeared as an effect of the extinction.

According to a recent view the extinction is believed to represent a form of new learning rather than a forgetting of a previously established association. Since our subjects after Experiment 1 have surely been exposed to many faces without being associated to the unconditioned stimulus (fear eliciting noise), we can assume that after a reasonable period of time (one month), the acquired fear conditioned enhanced imitative response would be extinguished. The aim of this experiment was to test this hypothesis.

6.4 General Discussion

In the present study I was able to show that the enhancement of the imitation response found in the previous chapter, does not seem to be confined to evolutionary relevant stimuli such as emotional facial expressions, animal and human attacks and so on, but can be acquired by association with an emotional stimulation.

Experiment 1 showed that fear conditioned stimuli prompt faster response only for compatible movements with respect of the pre-conditioning session and of the unconditioned stimuli of the same post conditioning session. Instead, Experiment 2 showed that after one month the conditioned stimuli have spontaneously lost their power to elicit the effect of Exp. 1. In fact, the conditioned faster imitative response is now extinguished.

It is important to emphasize that even if fear conditioning has been largely studied in humans, this is the first time that the imitative behaviour is shown to be conditioned.

In rats, typical fear conditioned response include freezing (Blanchard and Blanchard, 1969, 1972; Bolles, 1970; Fanselow, 1984; LeDoux et al., 1986), enhancement of musculature reflexes (e.g. potentiated startle) (Brown et al., 1951; Davis, 1997), analgesia (or decreased pain sensitivity) (Fanselow, 1986; Helmstetter, 1992), ultrasonic distress vocalization (Blanchard et al., 1991; Lee and Kim, 2004), and in both humans and animals alterations in autonomic nervous system activities (heart rate, blood pressure, respiration rate, skin conductance, corrugator supercilii) (Iwata et al., 1986; Kapp et al., 1979; Stiedl and Spiess, 1997). To my knowledge, the present study is the first proof of the effect of fear conditioning in the domain of imitation.

Experiment 2, extends our results by showing that the imitation response can be modulated according to environmental changes. The same imitation response can be beneficial in a given time at a certain conditions but no longer if something changes. Being imitation an adaptive mechanism by which we can learn new behaviors it can be modulated by environmental constraints to permit a more flexible behavior.

A large body of evidence from lesion, pharmacological and neurophysiological studies point to the amygdala as the key neural system subserving fear conditioning (see Kim and Jung 2006, for a critical review). Anatomically, the amygdala receives sensory inputs from diverse areas of the brain (e.g. thalamus, neocortex, hippocampus) and sends projections to various autonomic and somatomotor structures thought to mediate specific fear responses (e.g. bed nucleus of the stria terminalis for activating stress hormones, periacqueductal gray matter for freezing, lateral hypothalamus for sympathetic activation) (LeDoux, 1996). It has been shown that sensory information enters the amygdala through its basal and lateral nuclei (BLA) (Aggleton, 2000; LeDoux, 1996) where the pairing *conditioned stimulus* (for example the neutral faces) – *unconditioned stimulus* (white noise burst) association takes place. In particular, it has been shown that lesions of the medial division of the medial

geniculate nucleus (mMGN) of the thalamus, which relays auditory information to the amygdale (LeDoux et al., 1990), block the formation of noise-foot shock (LeDoux et al., 1986). Human neuropsychological studies (LaBar et al., 1995) as well as fMRI studies (Knight et al., 1999; LaBar et al., 1998) confirmed the importance of the amygdale for fear conditioning. The BLA then send the information to the central nucleus of the amygdala (CeA), which is believed to be the main output to the structures cited above for instantiating the cascade effect of fear.

I speculate that this output is also sent to the cortical frontal motor areas in which the imitation-compatibility effect comes from. In fact, in Chapter 4, we were able to show that using the same task but with emotional evolutionary relevant stimuli, the Readiness Potential associated to the preparation and execution of the movement was found to be increased (more negative deflection with respect to the neutral condition). Another important region that plays a great role in associating a given stimulus to a fear response is hippocampus. It has been shown in animal studies that disruption of the hyppocampi (but also to the insular cortex) impairs fear conditioning (Anagnostars et al., 1999; kim and Fanselow, 1992; Maren et al., 1997; Shi and Davis, 1999).

With respect to the extinction process explored in Experiment 2, the prefrontal cortex (PFC) has been proposed as an important brain area. It is known that PFC has inhibitory projections to many subcortical regions, such as the amygdala and hypothalamus (Fuster, 1997). In fact, stimulating the PFC the amygdaloid neurons get inhibited (Quirk et al., 2003; Rosenkranz and Grace, 2002), and reduces conditioned fear responses of the animal (Milad and Quirk, 2002; Milad et al., 2004). Similar inhibitory effect has been found also in humans (Kim et al., 2003; Ochsner et al., 2002). Lesion studies confirm the importance of PFC in extinguishing the fear conditioned response (Morgan et al., 1993).

In conclusion, in the present chapter I demonstrated that fear conditioned neutral faces trigger movements increasing our spontaneous imitation tendency in a modified SRC task. This mechanism of

emotional action triggering has been supposed by evolutionary theories as a survival mechanism to cope with imminent danger in case of life threatening stimuli. This has been extensively shown with the technique of fear conditioning which involves the pairing of a neutral stimulus with an aversive unconditioned stimulus (US), following which the neutral stimulus becomes conditioned (CS) to elicit a fear response (CR), normally elicited by the US.

The power of this mechanism relies on the fact that a useful adaptive mechanism such as imitation can be further improved if modulated by emotional cues, giving humans a flexible tool to improve the adaptation.

Chapter 7
Further comments and open questions

7.1 Key results of the present thesis: toward a model of a *negative bias for imitation*

This dissertation investigated the neurocognitive mechanisms of the *emotional control of imitative responses or empathy for actions in emotional contexts*.

Evolutionary pressure has led the nervous system to guarantee rapid and intense responses to negative dangerous harmful events (Carretiè, Albert, Lopez-Martin and Tapia, 2009). The hypothesis of a "*negative brain"* claiming that the evolution equipped the brain with a wide set of mechanisms *triggered by* and *to cope with* unpleasant or life threatening stimuli, has been proposed (Carretiè et al., 2009). This set of mechanisms act at multiple stages: from input selection and facilitation of incoming relevant stimuli, to evaluation dedicated modules to assess risk and danger, and last but not least to output processes to guide informed and efficient behavior. Psychological research has shown that this negativity bias is manifested through diverse response systems, including those related to cognitive, emotional and social behavior (Cacioppo and Gardner, 1999; Taylor, 1991). Biased neural mechanisms of perception and attention have been shown in a plethora of studies (for a recent review see Lang and Bradley, in press). The goal of these mechanisms is to produce rapid and efficient actions directed to avoiding or minimizing negative consequences associated with unpleasant stimulation just perceived.

In the words of Carretiè and colleagues “the neural negativity bias led also to a motor negativity bias” (Carretiè et al., 2009), stemming from simple fight-flight mechanisms to very sophisticated motor behaviors.

Along this thesis I asked whether imitative responses belong to the class of *negatively-biased motor behaviors.*

In order to accomplish this aim, I carried out five studies in which participants were asked to perform covert imitative movements following emotional contexts or emotioned models’ facial expressions. Several methodologies (behavioral, computational, ERP techniques as well as neuropsychology), and different populations of subjects (adult vs. children and normal vs. abnormal) were used in order to explore and dissect this function.

The key results are:

Study 1 (Chapter 2): Subjects were asked to perform tapping movements following neutral versus emotional contexts (IAPS pictures depicting different threatening situations). Reaction times were recorded and participants resulted being faster in performing imitative movements relative to non imitative movements only following the emotional stimulation (interaction was significant at $p<0.05$). Moreover the more emotional are these pictures (according to standardized affective ratings), the larger the effect (faster RTs) (Experiment 1). The effect was only for negative emotional pictures and did not extended to positive (arousal balanced pictures). This confirmed that the effect was not only an effect of the subjects being aroused by the emotional content of the stimuli, but also a true valence effect, limited to negative aversive stimuli. I argued that this enhanced imitation for only negative situations might be an important evolutionary mechanism by which an action already started by others in the

environment can be copied (that is imitated) without spending precious time to program and to start a new one. Wasting time in programming a new action can be risky because of its time consumption (commonly this will be expressed by the sentence "be quick or be dead"). To assess if this was an effect related with imitative tendencies, two control experiments were run, in which we manipulated the presence of the imitation factor in the task (varying the presence of the observed movement or the timing of subjects onset response), but no effect of the interaction between emotions and imitation was found. In another experiment, we controlled for the anxiety levels of the participants and we found that high level anxiety subjects were slowed down in their imitative responses. These data fit with psychological hypothesis about the role of the anxiety in freezing behavioral responses to better assess for dangers in the environment. A series of further experiments showed that the ECOI does not extend to other action domains (Experiments 3 and 4). Lastly I showed that the effect disappear using an online imitation paradigm.

I also performed an exp in which I used an online imitation paradigm (Experiment 5). This allowed me to test whether the time course of negative emotions affected compatibility as in an offline paradigm, but I failed in finding such effect. This might be because the short time in which subjects are supposed to imitate does not allow negative emotions to affect the motor system. I concluded that the E.C.O.I. needs a minimum time of processing to show the negatively-biased advantage.

Study 2 (Chapter 3): an artificial model of the basic findings of Study 1 was built in order to explore the computational mechanisms underlying the effect of emotions on imitation. The main structures hypothesized as producing the E.C.O.I. effect were implemented in a biologically plausible artificial

neural network consisting in several layers (each of which represents a dedicate brain module), and three assumptions were tested:

1. Action resonance mechanism
2. Facilitated response for similar actions (motor priming)
3. Inhibition for different actions

The first assumption is strictly connected with the ideo-motor theory that proposes that observation and execution of an action share the same neural representations and, supposedly, overlapping brain regions (Greenwald, 1970). Accordingly, executing an action activates an internal motor representation in the observer, which in turn is then used to execute (i.e., imitate) the same action. In our simulation the assumption that this resonance mechanism may have a possible neural candidate in the "mirror" system (di Pellegrino et al., 1992) is implemented by having the same layers of units activated for both the *observation* of a movement and the *execution* of the same movement.

The second assumption of the model is that *observing a movement facilitates the subsequent execution of the same movement*, i.e., produces a motor *priming effect*. Thus, *viewing* an action (e.g., tapping) activates the neurons of the brain region corresponding to that action; the activation of this region will facilitate the subsequent *execution* of the same action as the region will be still slightly active. Within an artificial neural network, this was implemented by an "activation-based" memory. Thus, viewing an action (e.g., tapping) activates the units of the region corresponding to that action which in turn will facilitate a subsequent flow of activation in the same areas for the execution of the same movement (on compatible trials).

The third assumption is that *the activation of a representation inhibits other "similar" representations*. This refers to the competition for activation between near regions of the cortex and is

implemented in the model with intra-layer competition through *k-winners-take-all* (kWTA) dynamics (Majani, Erlarson and Abu-Mostafa, 1989), which causes dynamic competition between k-units and the other units within a given layer.

The above three assumptions, i.e., a common layer representing both observed and to-be-executed movements, persisting activation of the observed movement, and the inhibition dynamic between observed schemas and the actual schema to be executed (different from the observed), all affected the network performance.

We also hypothesize that the emotional modulation of the SRC effect in imitation is sustained by an emotional alert system supposingly the amygdale in the real brain. It has been suggested that the amygdala alerts the organism by "boosting" a better and deeper encoding of relevant stimuli (Anderson and Phelps, 2001). Our hypothesis is that emotions can influence action by enhancing the regions involved in action representation and execution. Within the model this hypothesis was implemented by introducing extensive reciprocal bidirectional connections between units in the amygdala layer and those in the action representation layer, forming a closed and reverberating loop between these areas (Amaral, Price, Pitkanen, and Carmichael, 1992).

After being trained, the model was tested and successfully reproduced the main findings of Study 1, that is, the emotional enhancement for compatible-imitative responses. Precise neural predictions were given, including the activation of an emotional system, which speeded up the reaching of the final state of the network (the so called "attractor") in the emotional imitative condition only which explains the behavioral performance of the subjects of Study 1.

Study 3 (Chapter 4): an Event Related Potential study was carried out to test for the neurocognitive hypothesis generated by the computational simulation of Experiment 1.

In particular, we tested for the prediction that an emotional activation of dedicated structures of the brain (supposingly the amygdale) will cause an increased activation of the neurons associated with movement preparation and execution. A behavioural paradigm similar to Experiment 2 of Study 1, was used and 15 subjects were tested while their scalp activity was recorded with a 32 channels EEG system. This technique was used because of the possibility to look at specific brain waves associated with the preparation (readiness potential) and the execution (motor potential) of movements. Our hypothesis (derived hypothetically by Study 1 and more formally by Study 2) was that associated with faster response of subjects performing imitative movements following emotional contexts, there should be an increased brain activity in the motor regions of the brain responsible for the movement instantiation. As predicted, an increased signal for both the preparation and the execution of the movement was found in the form of more negative (which is to be read as stronger) readiness potentials and movement related potentials. The former indicates stronger preparation and programming of the movement to be performed and the latter indicates a more powerful execution. We called these brain waves "Fear potentiated movement related potentials" (FPMRP).

Study 4 (Chapter 5): In this study the paradigm developed in Study 4, Experiment 1, was applied to a particular class of patients, namely, children with autistic spectrum disorder (ASD). This class of patients was selected because of their known problems in both imitation of actions and facial expressions. I was able to show that these patients contrary to previous experiments have no problems in imitation, but only in the response associated to the emotional condition: while normal controls performed faster the emotional movements (as the adult subjects in the previous experiments), the ASD did not differ in the two conditions. I argued that beside their preserved imitative abilities (preserved

action resonance mechanism as shown in the assessment), these children suffer from an empathic deficit (emotional resonance mechanism), which leads to abnormal social behaviors.

Study 5 (Chapter 6): Experiments in this chapter were designed to see if the emotional modulation of imitation is something hardwired in our brain or can be learned through experience. Based on the classic fear conditioning procedure I was able to demonstrate that even neutral faces previously associated with an emotional stimulus (conditioning phase by means of a white noise burst) are able to trigger the same modulation as shown in Study 1. Moreover, after one month the same subjects were retested and I found that the effect found in Experiment 1 was no longer present, that is the effect of enhanced imitation acquired through conditioning extinguished.

Thus, data described here are in favour of a dedicated neurocognitive mechanism for which imitation in emotional contexts or of emotioned models comes facilitated (faster RTs). This mechanism relies on a linkage between emotional and motor structures, as manifested by increased neural signal in motor related potentials following emotional stimuli only. Moreover, this mechanism can be learned through experience and shows temporal flexibility in accordance with changes in the environment. Finally, I showed that this mechanism can undergo dysfunction in a certain class of patients suffering from specific empathic-social deficits (ASD children). All this data are in support of my hypothesis of imitation as one of the negatively-biased motor mechanisms.

7.2 Further comments on the link between empathy and imitation

Emotions considered as a functional products of Darwinian evolution, developed from primitive (re)actions that facilitate the continued survival of living organism. One particular class of action I decided to investigate is imitation because of its social importance in connecting people's behaviour. Several theories of emotions argued that every emotion is associated with a particular kind of motor patter (for example, fear leads to flight, see Lang, Bradley, and Cuthbert, 1997). One of the closest links between the action and emotions domain is empathy. Empathy, as a form of mixed emotional-and-motor resonance as shown by different studies (Leslie, Johnson-Frey, and Grafton, 2004; Preston and de Waal 2002). In fact, empathy let us to bodily resonate with the model and to understand and reproduce its inner state by means of sharing of emotions between subjects by means of motor feature detection or body language (de Gelder, 2009; Pfeifer, Scott, Sigman, Bookheimer, and Iacoboni, 2006). Emotional imitation can be considered as a particular form of empathy. Someone called it sensorimotor empathy (Blakemore and Frith, 2005).

This is strictly related with the so called *emotional contagion*, by which our body becomes the interpersonal medium of communicating emotional states. Thus, empathy may occur via a mechanism of action representation that modulates and shapes emotional contents (Carr et al., 2003). Preston and de Waal (2002) have suggested that a motor resonance system could play a role in a perception-action model (PAM) of empathy, and that empathy is a part of a larger class of processes that depend on perception-action mechanisms. As shown in the computational model (Study 2), this mechanism of common perception and action (*action schema layer* in my model) is what let the artificial biologically plausible neural network to reproduce the advantage in performing a just performed action (that is to imitate). What facilitates this resonance according to my model, is the activation of the emotional

system which triggers the reaching of the final state corresponding to the reproduction of the observed action (imitation). This is shown behaviourally in faster RTs for imitative trials in the emotional condition throughout the whole thesis, and neurally by stronger movement related potentials (Study 3). This was predicted by the computational model for which the activation of the *amygdala layer* enhances the mean activation of the units of the *schema layer*.

On one side, boosted activations in the motor system during imitation have been found by some authors. For example Leslie and colleagues (Leslie et al., 2003) asked subjects to watch movies of facial expressions (smile or frown) and hand movements (move index or middle fingers) while brain activity was recorded (fMRI). Three different conditions were implemented: passive viewing, active imitation and active motor control. They found evidence for a common cortical imitation circuit for both face and hand imitation, consisting of Broca's area, premotor areas, posterior temporo-occipital cortex, and cerebellar areas. On the other, the work of de Gelder (2006) has shown in several experiments that the perception of emotional body language leads to motor system enhancement as well.

Taken together, these results show a stronger modulation of the motor system for both imitation and emotional actions. Unfortunately the paradigm used in these experiments (as for example in de Gelder experiments) lack of a quantitative measure at the behavioural level to claim that there is a true modulation of the motor system.

The advantage of my paradigm is that it gives us quantitative measure of the motor system (RTs) to catch for differences in imitative vs. non imitative responses in the neutral and in the emotional condition. It might be that the increased BOLD of the motor system found by de Gelder as well as increased cortical spinal excitability in TMS studies (Hajcak et al., 2007; Oliveri et al., 2003) while seeing emotional bodies or expressions are associated with faster motor and imitative responses as in our experiments, but we do not know.

7.3 Further comments on the arousal question.

One critique that can be moved to the paradigm I used is that the effect found in these studies can be explained as coming from a general arousal effect. This hypothesis can be formulated in this way: the improved response found in all the experiments reported here comes from a simple energization of the motor system which is not specific to the imitation domain. That is, the effect is only an arousal effect and does not represent a dedicated mechanism for facilitating the imitative behaviour in emotional contexts.

Since the *negative brain hypothesis* builds on the fact that arousing-positive stimuli do not elicit defensive mechanisms, and that, valence is the only thing triggering this set of behaviours, I will put some arguments to sustain that E.C.O.I. is valence dependent and arousal independent.

To begin with, we must notice that the effect of faster responses (as well as stronger event related potential, Study 3), is restricted only for compatible-imitative and not for incompatible-non imitative movements. Almost all the previous experiments done in the field did not control for the arousal (see for example Coombes experiments, in which there was only a motor condition modulated by emotions without an identical control condition which was not affected), so it is difficult to disentangle the effect of a real emotional priming of some kind of motor schemas from the mere augmentation of the motor excitability as a cause of arousal.

One can say that incompatible movements are more difficult because of their conflicting nature between observed and executed action and this may hide the effect of emotion on them. Even if this is true, this does not speak against my hypothesis that evolution gave humans this mechanism of

enhanced response selective for imitative responses in emotional context to facilitate empathy and the so called social glue between members of the same group doing something together. This might have led to stronger cooperation increasing the probability to survive.

A second argument comes from Experiment 1 of Study 1 in which I showed that the effect is specific for negative emotional pictures and not for arousal-balanced positive pictures. If the effect was specific for arousal I should have found it also in the positive condition. This is not the case. One can say that in Experiment 2 of Study 4 (the one using happy, fear and neutral facial expressions) we found this effect also for happy faces (which are kind of positive stimuli as pleasant IAPS pictures). My feeling is that pleasant IAPS pictures and happy facial expressions are very different between each other, and this difference shows another important aspect of my paradigm which is connected to empathy. The fact that I did not find any effect using pleasant IAPS pictures comes from the fact that subjects did not have anything to empathize with, because the content of these pictures was related to pleasant stimuli such as food, sport and erotic scenes. By contrast, unpleasant pictures were related with dramatic scenes (mourning, crying, harmed and suffering people, illness, contagion, car accident, human and animal aggressions) which lead to empathic responses. Consider also that the arousal and the valence dimension do not correlate for the IAPS stimuli chosen in this thesis.

Instead, happy and fear facial expressions share the property of triggering an implicit mechanism of motor and emotional identification with them. As a result of this empathy, subjects showed faster and augmented EEG signal in case of imitative following emotional contexts and expressions.

A third observation comes from Experiment 3, Study 1, in which we found that people with anxiety traits show slower instead of faster imitative responses. Anxiety is a function of arousal, and anxious people have stronger arousal activation in emotional unpleasant contexts. If the effect E.C.O.I

was an arousal only effect, I should have seen even faster motor response in these subjects, but this is not what data show.

A fourth observation comes from Study 3, in which we found no correlation between the late positive potential (LPP) which is associated with the increased cortical activation during emotional pictures and the readiness (RP) and motor potential (MP) which are associated with the motor planning and execution. If the effect was due to an increased of the cortical arousal (because of the perception of emotional pictures) we should have found continuity between the two signals. This did not happen, and the two signals seems independent from each other. Moreover, please consider that LPP is a positive wave while RP/MP are negative waves. If the first was influencing the second and the third we must have seen less negative potential in the emotional condition instead of observing an increased negativity.

Additional observations come from experiments or data reported here. First in an experimented not reported here we used angry versus fear and neutral facial expressions, but I found the effect only for fear (as shown in this thesis in Study 4), but not for angry faces even if they are arousing as fear faces, but, subjects are not supposed to empathize with them (usually we react to an angry man, instead of empathizing with him). The last observation is derived from the literature about the fear potentiated startle reflex (an augmentation of the motor reflexes after emotional unpleasant only stimuli) which is not found for arousal-balanced pleasant pictures.

All these observations speak against the arousal only hypothesis. However, arousal is one of the two components of emotions (valence being the other), and I think it is impossible to have a negative emotions (valence) without feeling activated (arousal). Both components play a role in the experience of emotion. Even if the E.C.O.I. is true emotional effect and not only an aspecific energization effect, arousal for sure plays a role in it. My feeling is that it can explain the size of the effect but not the effect

itself.

7.4 Future developments and limitations of the current work.

Several questions can be formulated for future research along with some limitations of the current work. Based on the computational investigation of Study 2, I would like to further explore the neural bases of the E.C.O.I. If my speculations are correct, I will see the involvement of the amygdala in triggering the motor system. One future question is if our task is associated to the increased activation in the same areas found by other authors (supplementary motor area, premotor area, temporo-parietal regions, orbitofrontal cortex, amygdala and insula, de Gelder, 2006). Lacking of an fMRI study, the anatomical considerations behind the emotional control of imitation remain at a very speculative level.

Another important question is if the E.C.O.I. is specific for biological motion or extends to non-biological movements. The well known finding that observing a robot making incongruent movements had no significant effect on executed movement (Craighero, Bello, Fadiga and Rizzolatti, 2002; (Kilner, Paulignan and Blakemore, 2003), might be a consequence of the brain processing biological and non-biological movements differently. The ability to distinguish between biological and non-biological movement develops early: 3-month-old babies are able to discriminate between displays of moving dots that depict biological motion and displays in which the same dots move randomly (Bertenthal, 1993). This suggests that the detection of biological motion becomes hardwired in the human brain at an early age. One future research will be aimed at testing this hypothesis in my experiments. Is the E.C.O.I. present also when observing non-biological robotic movements? Unfortunately, I did not have the possibility to test this topic. My prediction is that this should not happen for the considerations reported

in study 1. It will be intriguing also to extend this human vs robot paradigm to ASD children who show selective preference for inanimate objects.

Another important issue to be explored in the next future is the distinction between spatial vs. anatomical imitation. Is there a difference between mirror-image imitation (e.g., the imitator's right side corresponds to the model's left side, as I did in my experiments) and anatomical imitation (e.g., the imitator's right side corresponds to the model's right side)? According to the goal-directed theory of imitation (GOADI, e.g., Bekkering, Wohlschlager, and Gattis, 2000; Wohlschlager, Gattis, and Bekkering, 2003) the imitator first deconstructs the observed action into a hierarchy of abstract goals (not body movements), then uses this representation as a basis for reconstructing the action.

Imitation is sensitive to the observer's representation of the goals of the action. Moreover, children (Bekkering et al., 2000; Gleissner, Meltzoff, and Bekkering, 2000; Schofield, 1976; Wapner and Cirillo, 1968) as well as adults (Avikainen, Wohlschlager, Liuhanen, Hanninen, and Hari, 2003; Ishikura and Inomata, 1995) show larger preference for mirror-image imitation over anatomical imitation. This may arises because mirror-image actions are most strongly triggered through the mirror neuron system located in this same frontal area (Koski, Iacoboni, Dubeau, Woods, and Mazziotta, 2003).

Another question belongs to the clinical aspects of this effect. Many classes of patients suffer from problems in both emotions and imitation, and it will be interesting applying my paradigm to these clinical populations. For example, Parkinson disease patients have been reported to show blunted emotional reactivity for visual stimuli while recording the startle reflex (augmented in normal people as a consequence of the emotional stimulation) (Miller et al., 2006). My paradigm can be fruitful applied to them. As their neural defect implies damage to the dopaminergic system and compromised basal ganglia functionality, these patients can extend our knowledge of the neural bases of my experiments.

Beside neurological patients, psychiatric patients can also be an interesting population to use for their well known social and emotional deficits.

Some limitations of the present study must be pointed out. One clear limitation is that IAPS pictures used in Study 1 & 3, are largely heterogeneous and very complex and are not well controlled stimuli. To overcome this problem, I would like to use only facial expressions in my future studies.

Another limitation is that following my results I could not separate between different kinds of negative emotions. This is because IAPS pictures do not make distinctions between different emotions. Further experiments using separate emotional triggers (such as emotional facial expressions) will shed light on this point.

7.5 Conclusions

This project aimed to investigate the neurocognitive mechanism of the emotional control of imitation. A detailed dissection of this function by a variety of approaches (i.e. behavioral, EEG, computational modeling, and cognitive neuropsychology) and different populations (children and adults, normal and abnormal) was used to pursuit our aim at both the cognitive level and neural level of investigation. Results of various experiments support the hypothesis that a dedicated mechanism for facilitated imitative response in emotional context or of an emotioned model does exist. In the same way facial expressions prime facial mimicry we showed that bodily movements (and not non biological movements) executed in an emotional context do prime "body mimicry" as shown by faster RTs in imitative/compatible movements.

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Appendix: Technical details of the computational model (Study 2)

The algorithm used in this simulation is LEABRA, as developed by O'Reilly (1996a). This appendix provides a brief review of its basic operation. For a comprehensive LEABRA reference manual and for further details, see O'Reilly (1996a, 1998) or O'Reilly and Munakata (2000).

1) Point Neuron Activation Function

LEABRA uses a point neuron activation function that models the electrophysiological properties of real neurons, though it simplifies the geometrical structure to a single point. The membrane potential V_m is updated as a function of ionic conductances with reversal potentials:

$$\Delta V_m = \sum_j g_j(t)\overline{g}_j(E_j - V_m(t))$$

where *j* ranges over excitatory, inhibitory and leak inputs.

Activation communicated to other cells is thresholded by a sigmoidal function of the membrane potential.

2) k-Winners-Take-All implementation

kWTA is implemented by setting a uniform level of inhibitory current (g_i) in the equation

above) for all units within a layer:

$$g_i = (1 - q).g^{\Theta}_{k+1} + q.g^{\Theta}_{k}$$

where q is a parameter for setting the inhibition between an upper bound of the net excitation of the k^{th} most active unit and the lower bound of the net excitation of the $(k + 1)^{th}$ most active unit. These boundary inhibition values are computed as a function of the level of inhibition necessary to keep a unit right at threshold:

$$g_i^{\Theta} = \frac{g_e^* \bar{g}_e (E_e - \Theta) + g_l \bar{g}_l (E_l - \Theta)}{\Theta - E_i}$$

where g_e is the excitatory net input, g_l is the leak net input and Θ is the threshold membrane potential.

3) Learning algorithm

Within LEABRA weights are adjusted according to a combination of a symmetric version of the GeneRec algorithm (O'Reilly, 1996b) and a Hebbian learning term adjusted via a variant of Oja normalization (Oja, 1982). Weights are further subject to soft-bounding, so that they remain within the range of 0 to 1.

GeneRec involves the network settling in two phases (the plus phase and the minus phase), with inputs and outputs clamped in the plus phase but only inputs clamped in the minus phase. The GeneRec weight change term is then computed as a simple difference of pre and post synaptic activation across the two phases:

$$\Delta w_{ij} = \left(x_i^+ y_j^+\right) - \left(x_i^- y_j^-\right)$$

This term is combined in a weighted average with the Hebbian learning term adjusted, as

mentioned above, with a variant of Oja normalization:

$$\Delta w_{ij} = y_j \left(x_i - w_{ij} \right)$$

4) Learning procedure

The learning procedure can be decomposed into three stages:

a) Settling

I. Initialize all units

II. Apply external patterns

b) For each cycle

I. Compute excitatory net input

II. Compute kWTA

III. Compute point neuron activation function (combining I and II)

c) Update weights

I. Compute error driven weight changes

II. Compute Hebbian weight changes

III. Compute net weight (combining I & II)

IV. Adjust weights according to III.

The emotions aren't always immediately subject to reason,
but they are always immediately subject to action"
William James