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OUR IDENTITY IS ALSO IN OUR MOVEMENTS: A MULTIDISCIPLINARY APPROACH TO THE STUDY OF THE MECHANISMS OF STATIC AND DYNAMIC FACE RECOGNITION

PhD thesis by

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Abstract

Prosopagnosia is a relatively common deficit both in its acquired and developmental forms. However, no unitary protocols for its rehabilitation have been developed yet. Thus, a systematic review of the literature on the topic (Study 1) has been performed to understand the state of the art of rehabilitation of object agnosia and prosopagnosia. Results regarding developmental and acquired prosopagnosia reveal that treatments involving holistic perceptual processing of faces are the most effective and can be generalized to new views and perspectives of faces. However, few papers were obtained and new studies addressing the topic are required. This might be due to the scarce clarity about the mechanisms subserving face processing. Thus, we decided to deepen our understanding of the mechanisms subserving face recognition. First, we wanted to shed light on the relationship between facial expression and identity processing. The aim of study 2 was that of assessing whether there is a relation between individual face recognition abilities and facial expression processing. Indeed, according to models of face recognition, we recognize faces through two partially independent systems which process respectively, facial features and facial expressions. Based on that, we expected expressions, even when non-emotional to aid identity recognition. Results of study 2 showed that when the system used to process facial identity is deficient, dynamic facial expressions, even when non-emotional, help recognition. Study 3 aimed at deepening our understanding of the neural bases subserving the relation between facial expression and identity processing. To do so, we stimulated STS, preSMA and a Sham area with repetitive TMS while administering a task of identity matching of faces encoded through non-emotional facial expressions, rigid head movement or as neutral. Results of the Sham condition replicate findings from study 2 confirming that expressions, even when non-emotional, aid recognition in poor recognizers. Moreover, both preSMA and STS were implicated in identity processing: preSMA stimulation caused increased recognition

of faces encoded as neutral while STS stimulation caused increased recognition of faces encoded through a facial expression. To further explore the relation between expressions and identity recognition, Study 4 investigated Parkinson's Disease patients. Those patients are known to have reduced expression simulation and gave us a chance to study the role of expression simulation for identity recognition. Results reveal that PD patients do not show difficulties in recognizing static non-emotional facial expressions compared to Healthy Controls. Moreover, they do not show deficits in the recognition of identities of faces encoded through a dynamic non-emotional expression. These results hint at a reduced role of facial mimicry of expressions in facial identity processing, at least when is subserved by subcortical structures as in PD. Eventually, Study 5 was aimed at deepening our understanding of face recognition processes in patients with a lesion to the Posterior Cerebral Artery compared to healthy controls. Results of Study 5 reveal that face processing can be dissociated from mid-level perception and object perception. Besides, within face processing, it is possible to dissociate between different functions, such as perception and memory or matching and discriminating. Eventually, we were able to diagnose three of those patients with acquired prosopagnosia. Results of study 5 tell us that face processing is a complex function subserved by multiple overlapping systems, as shown by the frequent association between deficits; those systems are also at least partially separated from one another, as shown by the few observed classical dissociations.

The obtained results are of theoretical and clinical interest suggesting potential developments both for cognitive models and rehabilitative studies.

Riassunto

La prosopagnosia è un disturbo diffuso sia nella sua forma congenita che acquisita. Tuttavia, non esistono ancora protocolli unitari per la sua riabilitazione. Di consequenza, nello studio 1 è stata svolta una revisione sistematica della letteratura per avere una panoramica sugli studi esistenti sulla riabilitazione dell'agnosia per gli oggetti e della prosopagnosia. I risultati riguardanti la prosopagnosia mostrano che trattamenti basati su un'elaborazione olistica dei volti sono più efficaci e vengono generalizzati a nuove prospettive dei volti. Tuttavia, esistono pochi articoli sull'argomento ed è necessario sviluppare nuovi studi. Questo potrebbe essere dovuto alla poca chiarezza sui meccanismi di elaborazione dei volti. Per questo motivo, abbiamo deciso di approfondirli. Innanzitutto, abbiamo indagato la relazione tra espressioni facciali e riconoscimento di identità. L'obiettivo dello studio 2 era quello di indagare se c'è una relazione tra le abilità di riconoscimento individuali e l'elaborazione di espressioni facciali. Gli attuali modelli di riconoscimento volti postulano che elaboriamo i volti tramite due sistemi separati in interazione: uno elabora le caratteristiche del volto e l'altro le espressioni facciali. Di conseguenza, nello studio 2 ci aspettavamo di osservare una facilitazione delle espressioni sul riconoscimento di identità. I risultati confermano che espressioni facciali dinamiche non emotive aiutano nel riconoscimento di identità, ma solamente in chi ha delle scarse abilità di riconoscimento volti. L'obiettivo dello studio 3 era quello di approfondire le basi neurali di questa relazione. Per farlo, abbiamo stimolato STS, preSMA e un'area Sham con la TMS mentre i partecipanti dovevano riconoscere volti codificati tramite espressioni facciali dinamiche non emotive, un movimento rigido o come neutri. I risultati ottenuti nella condizione sham replicano quelli ottenuti nello studio 2: espressioni facciali non emotive aiutano il

riconoscimento di identità in scarsi riconoscitori. Inoltre, sia preSMA che STS si sono dimostrate implicate nel riconoscimento di identità dei volti: la stimolazione di preSMA ha causato un miglioramento nel riconoscimento di volti codificati come neutri, mentre la stimolazione di STS ha causato un miglioramento nel riconoscimento di volti codificati con espressione facciale. Per approfondire ulteriormente la relazione tra riconoscimento identità e espressioni, nello studio 4 abbiamo indagato queste abilità in pazienti con Malattia Di Parkinson, conosciuti per avere una ridotta capacità di simulare le espressioni. I risultati rivelano che pazienti con MDP non hanno difficoltà nel riconoscimento di espressioni facciali non emotive né nel riconoscimento di identità mediate da espressioni facciali non emotive. Questi risultati suggeriscono un ruolo ridotto della mimica facciale nel riconoscimento di identità, almeno quando mediato da strutture sottocorticali come nella MDP. Infine, lo studio 5 si poneva come obiettivo quello di studiare il riconoscimento volti in pazienti con lesione all'arteria cerebrale posteriore. I risultati rivelano che l'elaborazione dei volti può essere dissociata da funzioni percettive di medio livello e dall'elaborazione di oggetti. Inoltre, la memoria per i volti può essere dissociata dalla percezione dei volti, così come il loro matching può essere dissociato dalla loro discriminazione. Inoltre, tre di questi pazienti sono stati diagnosticati come prosopagnosici acquisiti. Dunque, l'elaborazione di volti è una funzione complessa mediata da diversi sistemi sovrapposti, come dimostrato dalle frequenti associazioni tra deficit. Tuttavia, questi sistemi sono anche almeno parzialmente separati come dimostrato dalle dissociazioni classiche osservate.

I risultati ottenuti sono di interesse teorico e clinico suggerendo potenziali sviluppi sia nella creazione di nuovi modelli cognitivi che nella progettazione di nuovi studi riabilitativi.

1. General introduction

1.1 Prosopagnosia

Prosopagnosia refers to a deficit in the recognition of both new and known faces (Corrow, Dalrymple & Barton, 2016). It can be the consequence of an acquired lesion (i.e., *acquired prosopagnosia*) or present since birth. In the cases where prosopagnosia is not acquired, some authors prefer to use the term "*congenital prosopagnosia*" (Palermo, Willis, Rivolta, McKone, Wilson & Calder, 2011; Bentin, DeGutis, D'Esposito & Robertson, 2007; Behrmann, Marotta, Gauthier, Tarr & McKeff, 2005; Carbon, Grüter, Weber & Lueschow, 2007), to define a genetic disorder (e.g., Cattaneo et al., 2016); while others use "*developmental prosopagnosia*" as a broader term aiming at referring both to congenital prosopagnosia and a deficit with its onset in the first period of infancy, due to other causes than genetic ones (Susilo & Duchaine, 2013). During this dissertation we will use both terms interchangeably. Although prosopagnosia has been extensively studied both in its developmental and acquired forms, no agreement on diagnostic criteria has been found yet. Some authors attempted to set generalizable criteria for diagnosis. For instance, Barton & Corrow (2016b) propose criteria for the diagnosis of developmental prosopagnosia that are a subjective impairment in daily-life face recognition combined with impaired performance in 2 or more tests of face familiarity (which can be both famous or non-famous faces). They also propose secondary criteria such as intact basic visual function, preserved familiarity of voices and names, exclusion of autism, and lesions to brain areas involved in face processing. Similar criteria were also proposed by Dalrymple and Palermo (2016). Congruent criteria were also set for acquired prosopagnosia: according to Albonico & Barton (2019), an acquired deficit in face recognition can be diagnosed with the sum of a coherent lesion on imaging, the patient's self-reported difficulty on face recognition

compared to pre-accident abilities, and poor performance on one or more tests of face recognition (see also Corrow et al., 2016).

Although tests for prosopagnosia diagnosis are numerous (Robotham & Starrfelt, 2018), there are no official tests for diagnosis (Geskin & Behrmann, 2018).

1.2 Rehabilitation of prosopagnosia

According to few epidemiological studies, prosopagnosia in its developmental form can be found in 2% of the population (Bowles et al., 2009; Kennerknecht, Ho & Wong, 2008), while difficulties in face recognition tests range from 21% to 80% of brain lesion patients, depending on the test (Valentine, Powell, Davidoff, Letson & Greenwood, 2006). Despite the incidence of difficulties in recognizing faces and their negative impact on social and daily life (Dalrymple et al., 2014), the rehabilitation for this disorder is still underinvestigated. Many studies on the topic are non-experimental (Clarke & Bindschaedler, 2005; Anderson & Rizzo, 1995; Raymond, 1996; Burns, 2004). Therefore, it is difficult to draw conclusions about the generalization of the results to different patients and settings.

A recent review has been published on the rehabilitation of prosopagnosia (Heutink, Indorf, & Cordes, 2019). The authors described both data on the rehabilitation of the deficits and articles giving suggestions for treatment without displaying any data. They analyzed the studies dividing them into compensatory and restorative approaches to rehabilitation (i.e., treatments aimed at a compensation of the lost function through other functions versus treatments aimed at restoring the missing function), concluding that, even though the data are too scarce to come to any conclusion, both these strategies seem to be useful in the case of object agnosia and compensatory approaches seem to work best for prosopagnosia. Moreover, the other two reviews on the rehabilitation of prosopagnosia came to the same conclusions. Bate and Bennets

(2014) stated that, for acquired prosopagnosia, compensatory treatments seem to be more effective. Still, they argue that understanding when restorative treatments are successful is crucial as the benefits are greater. Concerning developmental prosopagnosia, the authors conclude that there is not enough data to make inferences. DeGutis reached a similar conclusion: they found no evidence of the efficacy of restorative treatment on acquired prosopagnosia, while compensatory treatments seem to be effective in certain cases (DeGutis, Chiu, Grosso & Cohan, 2014). They pointed out the use of different successful treatment approaches in acquired prosopagnosia, such as galvanic vestibular stimulation (Wilkinson, Nicholls, Pattenden, Kilduff & Mildberg, 2008), and described rehabilitation of developmental prosopagnosia, discussing evidence of efficacy in both remedial and compensatory studies. We believe that one of the reasons why solid conclusions were not reached in the previous reviews could lay in the fact that there are several methodological limits in the published studies on the cognitive rehabilitation of agnosias. It is indeed often the case that studies are tailored to single patients and not replicable across clinical settings. The fact that there is a lack of studies regarding the rehabilitation of prosopagnosia might be due to several reasons, among which is a lack of clarity regarding the mechanisms subserving face recognition. That is why we think that deepening our understanding of such mechanisms might be helpful both in clarifying them from a theoretical point of view and for the development of new rehabilitation procedures.

1.3 Models of face recognition

One of the most influential models of face recognition is that of Bruce and Young (1986). Their model was primarily based on evidence from studies in the neuropsychological and cognitive domains. The authors postulate that we process faces both in a sequential and parallel way. At first, there is an initial analysis of facial features that is viewer-centered, where each feature is processed by specialized systems. It follows the phase called "structural encoding". In this

phase, the information goes from a "viewer-centered" representation to a viewpoint-independent one. Once a so-called "percept" is formed, our face recognition system compares it to stored information about faces. This information is called "face recognition units": it determines if a face is familiar or unknown. If a face is considered as familiar, semantic information about that person can be accessed through the "person identity node". It must be noted that one central assumption of this model is that identity and expression are processed separately. However, this view has been subsequently challenged (see paragraph below).

Subsequently, Haxby, Hoffmann, and Gobbini (2000) enriched this model by describing the neural systems involved in face processing. In particular, they describe a “Core system”, specifically selective for faces composed of the Fusiform Face Area (FFA), the Occipital Face Area (OFA), and the Superior Temporal Sulcus (STS), and an “Extended system”, involved in processing of familiarity of faces and located more diffusely in the brain. Within the core system, the OFA is involved in the early stages of face processing and then sends the information to FFA. FFA, in turn, is engaged in encoding the so-called invariant aspects of faces. By invariant aspects of faces, the authors refer to aspects of the structure of the face that are invariant across changes in expressions. OFA also sends inputs to the STS, deputed to processing the so-called changeable aspects of faces. By changeable aspects of faces, the authors refer to eye gaze, facial expressions, and those aspects that facilitate social communication. The authors emphasize the segregation between the invariant and changeable aspects of face processing, stating that otherwise, any change in facial expression would cause a change in identity. Later on, Haxby and Gobbini (2011) extend their model by deepening the description of the extended system. In particular, they point to brain areas involved in the representation of knowledge about the person (i.e., medial prefrontal cortex, temporoparietal junction, anterior temporal cortex, precuneus, and posterior cingulate), in the understanding of

actions (i.e., Inferior parietal & frontal operculum, intraparietal sulcus, and frontal eye fields) and in emotion processing (i.e., amygdala, insula).

Duchaine & Yovel (2015) further develop this model by stating that face recognition occurs through a ventral and a dorsal stream. The ventral stream processes information about faces starting with the processing of view-dependent representation in the OFA and view-symmetric representation in the FFA. Note that OFA is not the only entry point for perceptual information about faces as it was in previous models but works in parallel with other perceptual areas. Moreover, in this revised model, FFA does not appear to represent face familiarity, which is postulated to occur at a later stage. In addition, differently from previous models, FFA also processes information about changeable aspects of faces together with information about invariant aspects of faces. The dorsal stream mainly processes dynamic information about faces, and it is consequently implicated in social interactions.

1.4 The interplay between expression and identity recognition

As stated above, facial identity and expression processing seem to depend on partially overlapping systems. However, their level of interaction is still not clear. Some degree of separation between face identity and expression recognition has been observed in neuroimaging studies, such as PET (George et al., 1993), fMRI (Kesler et al., 2001; Gorno-Tempini et al., 2001; Winston et al., 2004), and ERP (Münte et al., 1998). Furthermore, interest in this issue has also arisen in the field of neuropsychology, where prosopagnosic individuals have been studied: a single dissociation between identity and expression recognition deficits was described both in acquired (Mattson, Levin & Grafman, 2000; Young et al., 1993) and congenital prosopagnosics (Bentin et al., 1999; Duchaine, Parker & Nakayama 2003; Jones & Tranel, 2001; Nunn, Postma & Pearson, 2001; Djouab et al., 2020), in the direction of preserved

expression with impaired identity recognition. Nevertheless, cases in which facial expression recognition is impaired along with its identity recognition have also been reported both for acquired (Humphreys, Avidan & Behrmann, 2007) and congenital prosopagnosia (Biotti & Cook, 2016).

Moreover, cases where a lesion causes a deficit solely limited to facial expressions are debated. This might be due also to the fact that in those clinical cases, it is often not clarified whether identity recognition is preserved (Bate & Bennets, 2015). Moreover, all these studies use emotional facial expressions, causing difficulty in interpreting the deficit (Calder & Young, 2005). We know from face recognition models that face identity and expression processing are integrated systems interacting with each other in the process of face recognition (Hinojsa, Mercado & Carretié, 2015; Calder and Young, 2005). In particular, O'Toole, Roark, and Abdi (2002) suggested that information about facial expressions carried by the STS (as in the model from Haxby, Hoffmann & Gobbini, 2000) can represent an alternative system for recognition of familiar faces in disturbed viewing conditions (see O'Toole & Roark, 2010 for an updated version). Moreover, in their revised model on face recognition, Duchaine & Yovel (2015) demonstrate that FFA has a role in both identity and expression processing, while the STS is mainly involved in processing expressions. Coherently with this model, a recent study demonstrated that the posterior STS shows stronger activation when seeing facial expressions (even without movement) than when seeing neutral faces and when seeing moving faces (even without any facial expression) with respect to static ones. The same preferences have not been shown in the FFA nor in the OFA (Bernstein et al., 2018). These results seem to be in line with the specificity of the STS for both facial expressions and inner facial motion. Considering the most recent models of face processing, we can hypothesize that when face recognition appears

as difficult, the information conveyed by both FFA and STS about facial expressions and that conveyed by STS about motion helps identity recognition.

Also, TMS studies bring evidence in favor of a specific neural network subserving facial expression and motion processing. In particular, TMS was shown to impair facial expression but not identity processing when delivered over OFA and the right Somatosensory Cortex (rSC) (Pitcher, Garrido, Walsh & Duchaine, 2008). In addition, Sliwinska and Pitcher (2018) showed that both right posterior STS (rpSTS) and left posterior STS (lpSTS) are implicated in emotional facial expression recognition. Moreover, rpSTS was shown to be involved in dynamic facial expression processing when stimulated with theta-burst transcranial magnetic stimulation (TBS) and measuring the effects through functional magnetic resonance imaging (fMRI) (Pitcher, Duchaine & Walsh, 2014). Interestingly, the authors found a role of rpSTS in facial motion processing even when the head was in motion but did not display any expression.

Moreover, a clinical population showing selective deficits in facial expression but not identity recognition is Parkinson's Disease patients (Argaud, Vérin, Sauleau & Grandjean, 2018). This deficit has been explained in terms of difficulties in social cognition which in turn bring to deficits in emotion processing (e.g., Wagenbreth et al., 2016). In addition, difficulties in facial expression processing in PD have also been linked to a lack in the possibility of simulating expressions for their understanding. This would be caused by classical symptoms of PD, such as amimia and facial bradykinesia (Prenger & MacDonald, 2018). Results of studies on PD patients add to our understanding of facial expression mechanisms by hypothesizing that expression processing also involves an embodied process of simulation of the expression for its understanding.

Two more aspects need to be clarified about facial expression processing: the role of emotions and the specific role of motion. As the first issue is concerned, the majority of the studies use emotional facial expressions (e.g., Righi et al., 2012; D'Argembeau et al., 2007; Humphreys, Avidan & Behrmann, 2007; Duchaine, Parker & Nakayama, 2003) making it difficult to disentangle the unique contribution of facial expressions on identity recognition at a perceptual level from that of emotional content in face processing. If we refer to O'Toole's model, we can say that the STS is involved in the elaboration of the social content of faces as well as the so-called "dynamic facial signatures", specific expressions involved in social interactions which can be considered as non-emotional facial expressions (O'Toole Roark and Abdi, 2002). Existing studies have demonstrated a facilitatory role of dynamic non-emotional expressions for unfamiliar face recognition in normal recognizers (Jesse & Bartoli, 2018); of static non-emotional expressions for unfamiliar face recognition in congenital prosopagnosics (Daini, Comparetti & Ricciardelli, 2014) and of static non-emotional expressions for famous face recognition in poor recognizers (Albonico, Malaspina & Daini, 2015).

As far as motion is concerned, the literature suggests a general "motion advantage" where faces encoded in motion are recognized more easily than those encoded as static (Schiff et al., 1986; Knight and Johnston, 1997; Lander et al., 1999; Lander & Butcher, 2015). Lander and colleagues (2004) described a patient, HJA, showing facilitation of motion in a task where he had to match identities, suggesting that neural mechanisms subserving motion of faces, such as the pSTS (as in Pitcher et al., 2011), might be involved in both identity and expression recognition (see also Bate & Bennets, 2015). In accordance with this claim, face identity recognition has been shown to be aided by seeing faces in motion both for unfamiliar (Pike et al., 1997; Knappmeyer et al., 2003; Lander and Bruce, 2003; Pilz et al., 2006; Lander and Davies, 2007; Butcher et al., 2011) and familiar faces (Knight and Johnston, 1997; Lander,

Christine & Bruce, 1999; Lander & Bruce, 2000; Lander et al., 2001) and in particular in poor viewing conditions (Knight and Johnston, 1997; Lander et al., 2001; Bennets et al., 2013). A motion advantage has been described especially for prosopagnosics (Steede et al., 2007; Bennets et al., 2015; Longmore and Tree, 2013; Xiao et al., 2014).

A way to understand the mechanisms behind face processing is through the investigation of patients with an acquired deficit in face processing. This might shed light on which processes contribute to face recognition.

1.5 Face recognition deficits: how specific are they?

As already seen above, several processes take part in face processing: however, the specificity and segregation of the single processes is not clear yet. A way to explore in detail the mechanisms behind face processing is to look at brain-lesioned patients and see whether specific functions are impaired together or singularly.

For example, it is not clear yet to what extent face perception and memory are separate as there are cases of both association and dissociation of deficits, both in acquired and developmental prosopagnosia (Barton et al., 2004; Dalrymple et al., 2011; Tippett et al., 2000; Dalrymple et al., 2014; Ulrich et al., 2017; Tian et al., 2020; Barton et al., 2004; Busigny et al., 2014; Dalrymple et al., 2011; Liu et al., 2021). Moreover, being impaired in one or more subtests of L-Post (Torfs, Vancleef, Lafosse, Wagemans & de-Wit, 2014), a test of low/mid-level perceptual processing is often used as an exclusion criterion in studies on congenital prosopagnosics, as face perception deficits are mostly dissociated from general perceptual deficits in developmental cases (e.g., Macaskill et al., 2021; Fry et al., 2020; Stumps et al., 2020). However, when patients with an acquired lesion are considered, the distinction between the two deficits is not as predictable; indeed, some low/mid-level deficits can be present

together with face processing deficits in acquired prosopagnosics (Monti et al., 2019). Moreover, it is not clear whether face processing involves specific mechanisms or mechanisms shared with other objects processing. As a matter of fact, a longstanding debate in the literature concerns whether face processing is subserved by a specific system, different from the one underlying object processing, or whether its apparent specificity is the result of our greater expertise for faces with respect to other categories (Kanwisher, 2000; McKone, Kanwisher & Duchaine, 2007; Young & Burton, 2018).

On the one hand, traditional domain-specific accounts of face processing claim the specificity of the Fusiform Face Area for face recognition (Kanwisher, McDermott & Chun, 1997), as part of a face recognition system (Haxby, Hoffman & Gobbini, 2000) as well as the existence of the ERP N170 component, which seems to be larger in response to faces than objects (Bentin, Allison, Puce, Perez & McCarthy,1996; Rossion et al., 2000; Carmel & Bentin, 2002). Neuropsychological data seem to confirm this account, as double dissociations of object agnosia and prosopagnosia deficits have been documented (Moscovitch et al., 1997; Geskin & Behrmann, 2018; Riddoch, Johnston, Bracewell, Boutsen & Humphreys, 2008). Moreover, another account in favor of the specificity hypothesis is that faces, compared to objects, are processed holistically (Robbins & McKone, 2007). This seems to be demonstrated by effects such as the inversion effect (i.e., a worsening of performance with inverted faces compared with upright ones - Yin, 1969), the composite effect (i.e., the alignment of two half faces belonging to different identities worsens performance in judgment on the single halves compared with misaligned trials - Young, Hellawell & Hay, 2013), and the part-whole effect (i.e., it is easier to discriminate individual facial features when presented within the entire face than when presented without the rest of the face - Tanaka & Farah, 1993; Kanwisher, 2000).

On the other hand, supporters of the expertise hypothesis brought evidence in favor of the

increased activation of the Fusiform Face Area and Occipital Face Area not only for faces but also for objects for which expertise has been acquired (Gauthier, Tarr, Anderson, Skudlarski & Gore, 1999; Gauthier, Skudlarski, Gore & Anderson, 2000; Burns, Arnold & Bukach, 2019). Moreover, the inversion effect has been found for non-face categories for which participants were experts (Diamond & Carey, 1986; Rezlescu, Chapman, Susilo & Caramazza, 2016), also in association with the N170 ERP component (Rossion et al., 2002). Additional accounts that favor the expertise hypothesis display evidence for a better memory for own-race faces than for other-race faces, explaining this as due to greater experience with own-race faces (Bukach, Gauthier & Tarr, 2006).

Further information in this regard comes from the literature regarding acquired prosopagnosia (AP). On the one hand, many studies report AP patients to be spared in recognition of within-class objects such as cars (Busigny, Graf, Mayer & Rossion, 2010; Schiltz & Rossion, 2006; Busigny & Rossion, 2010; Henke, Schweinberger, Grigo, Klos & Sommer, 1998; Rezlescu, Pitcher & Duchaine, 2012), birds, boats, chairs (Schiltz & Rossion, 2006) or vegetables (Henke et al., 1998). On the other hand, other studies report AP patients to have deficits in the recognition of the same type of within-class objects (Barton, Cherkasova, Press, Intriligator & O'Connor, 2004; Barton, 2008; De Haan & Campbell, 1991; Toftness, 2019). However, it might be that potential differences between face and within-category object processing are caused by a failure to benefit from experience with these types of objects and not a domain-general impairment in their recognition (Fry, Wilmer, Xie, Verfaellie & DeGutis, 2020). That is why some authors tested acquired prosopagnosics on novel objects for which neither patients nor control have previous experience. Also in this case, evidence is mixed with studies finding dissociation with face recognition (Rezlescu, Barton, Pitcher & Duchaine, 2014; Riddoch, Johnston, Bracewell, Boutsen & Humphreys, 2008) and others finding an association of deficits

(Gauthier, Behrmann & Tarr, 1999). In conclusion, the literature seems to shed heterogeneous results (Barton, Albonico, Susilo, Duchaine & Corrow, 2019).

1.6 Aims of the research project

Prosopagnosia is a diffuse and debilitating deficit both in its acquired and developmental forms. However, no unitary protocols for its rehabilitation have been developed yet. Several reviews exist on the rehabilitation of prosopagnosia: however, they do not reach solid conclusions due to the scarcity and heterogeneity of the studies present in the literature. That is why we decided to perform a systematic review (**Study 1**), including only those studies that are experimental. By doing that, we aimed to help researchers and clinicians willing to perform rehabilitation on such patients by providing them with replicable information. However, not many studies were present in the literature. This made us question the definition behind prosopagnosia itself. That is why we wanted to deepen our understanding of the mechanisms subserving face recognition. We think that deepening our understanding of the mechanisms behind face processing would reflect in the creation of new effective rehabilitation. Firstly, we wanted to shed light on the relationship between facial expression and identity processing. That is what brought us to study these abilities also in healthy participants (**Study 2**). The aim of study 2 was that of assessing whether there is a relation between individual face recognition abilities and facial expression processing. In fact, according to the models of face recognition described above, we would expect expressions to interact with features in identity recognition. However, the level of facilitation of expression on identity recognition is not clear yet. Deepening our understanding on that could help us in defining new rehabilitative protocols for prosopagnosics by creating rehabilitations focused on facial expressions use. This might be particularly helpful as prosopagnosics are often reported to be spared in facial expression processing. Thus, if it is true that facial expressions processing interact with identity processing, expressions might

represent a compensatory tool for identity recognition. Moreover, we wanted to deepen our understanding of the neural bases of the relation between facial identity and expression processing, and that was what motivated us to perform a TMS study on the topic (**Study 3**). In fact, the majority of studies investigate the neural bases behind facial expression recognition while we were interested in understanding the involvement of systems specific for processing facial expressions in the recognition of facial identity. Thus, the aim of study 3 was to understand the role of STS and preSMA in the use of facial expressions at the service of identity recognition. To the best of our knowledge, no studies on the role of STS and preSMA exist. Deepening our understanding of the role of these areas would be beneficial in updating existing models of face recognition. This might reflect in more effective treatments for prosopagnosia. In addition, we know from the literature that PD patients are often reported to be impaired in the simulation of expressions due to hypomimia and facial bradykinesia. Those patients give us a unique opportunity of studying the role of expression simulation in the recognition of facial identity. However, in the literature, those patients are reported to have deficits in the recognition of emotional expressions. Yet, it is not clear to what extent this deficit is due to problems in social cognition and to what extent it is due to a lack of the possibility of simulating expression due to motor disturbances linked to the disease. That was what motivated us to perform **Study 4**. The aim of study 4 was, first of all, to investigate whether PD patients show difficulties in the recognition of nonemotional facial expressions to disentangle the contribution of emotions from that of simulation of expression. Subsequently, we wanted to understand face recognition mechanisms in such patients and the interplay between expression and identity recognition in this clinical population. This study is particularly important for studying the role of expression simulation in identity recognition. This might extend existing face recognition models. Eventually, we wanted to clarify the mechanisms behind face processing by

understanding the level of independence of each process from the others. A unique opportunity to do so was to investigate the performance in different tests in patients with an acquired lesion to areas subserving face processing and general visual perception (**Study 5**). The aim of Study 5 was to understand the degree of separation between face processing and other functions (i.e., low/mid-level visual perception) and between face perception and nonface object perception. Moreover, we wanted to understand the level of independence of mechanisms within face processing (i.e., face perception and memory, face match, and non-match trials). Eventually, we wanted to investigate the prevalence of prosopagnosia in this group of patients. Studying patients based on their lesion site and not symptomatology represents a novelty in the literature, primarily focused on symptoms. Understanding the level of independence and interdependence of functions might help us in finding spared functions that might compensate for face processing deficits in patients with an acquired prosopagnosia deficit.

Each of the mentioned studies will be described separately: to ease the reader, the key concepts useful for the understanding of each study will be repeated in the introductions.

2[1]. Study 1: The rehabilitation of object agnosia and prosopagnosia: a systematic review

2.1. Introduction

Visual agnosia is a neuropsychological deficit characterized by impaired recognition of visually presented stimuli with preserved vision, semantic knowledge of the stimulus, language, and general cognition (Farah, 2004; Zihl & Kennard, 2003). Riddoch and Humphreys (1987) developed a model based on Lissauer's classification (1890) distinguishing apperceptive and associative forms of visual agnosia. Apperceptive agnosia refers to a deficit in the integration of different sensory attributes in a visual form, while associative agnosia refers to a lack in the attribution of meaning to the percept. Many forms of visual agnosia have been described (Farah, 2004): however, in this study, we will focus on visual object agnosia and prosopagnosia.

2.1.1. Object agnosia

Object agnosia specifically refers to objects (Humphreys & Riddoch, 1993; Moscovitch, Wincour & Behrmann, 1997). It can be apperceptive or associative (Riddoch & Humphreys, 1987). When considering the apperceptive form of visual agnosia, three further variants have been described. The first one is "Form agnosia" and refers to a lack in the analysis of the global configuration of a stimulus with preserved elaboration of its features. The second is "Integrative agnosia" and refers to impairment in the integration of single parts into a perceptual whole with preserved ability to recognize the single parts in isolation. The third is "Transformational

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agnosia", and is characterized by an impairment in the transformation process of a 3D object necessary to identify the object from non-canonical perspectives.

Although visual object agnosia has a very specific classification, mechanisms subserving object recognition are still not fully understood. Different models have been proposed on the functioning of object recognition. An important contribution in this area was apported by Elizabeth Warrington and Angela Taylor, who described patients with difficulties in naming objects seen from unconventional perspectives (Warrington & Taylor, 1973) and in matching them with the same object viewed from a conventional perspective (Warrington & Taylor, 1978). The authors' concept of conventional and unconventional perspective was vital for Marr in his description of object-centered representation of space (1982). According to the author, the visual representation of objects follows a sequential process. There is an initial viewer-centered representation in which the viewer encodes an object's basic visual and perceptual features and a second object-centered phase, in which the object is completely represented and recognized despite its view. Riddoch and Humphreys (1993) further developed the model with the idea that it is crucial to evaluate from a neuropsychological perspective the various stages involved in object recognition selectively. Those stages are the extraction of basic object dimensions, figure-ground segmentation, recognition through different points of view, representing what they define as "pre-categorical" processing, independent from knowledge of the object presented, and association of the stored knowledge of objects. Many standardized tests evaluate those different object recognition stages: however, their description is beyond the scope of the present article.

2.1.2. Prosopagnosia

The term prosopagnosia refers to an impairment of the ability to recognize both new and known faces (Corrow, Dalrymple & Barton, 2016). It can be a consequence of an acquired lesion (i.e., *acquired prosopagnosia*) or present since birth. For the second condition, there are authors who prefer to use the term "*congenital prosopagnosia*" (Palermo, Willis, Rivolta, McKone, Wilson & Calder, 2011; Bentin, DeGutis, D'Esposito & Robertson, 2007; Behrmann, Marotta, Gauthier, Tarr & McKeff, 2005; Carbon, Grüter, Weber & Lueschow, 2007). They use this term aiming at defining a disorder due to a genetic account (e.g., Cattaneo et al., 2016). However, we decided to use "*developmental prosopagnosia*" in the present study to refer broadly both to congenital prosopagnosia and deficits with onset in the first period of infancy and due to causes other than genetic ones (Susilo & Duchaine, 2013). Original models of face recognition postulate that we process faces sequentially (Bruce & Young, 1986). After an initial analysis of facial features, there is the stage called "structural encoding", where the information passes from a "viewer-centered" representation to a representation that is independent of the viewpoint. Once a so-called "percept" is formed, the face recognition system compares it to information about faces that is stored in the so-called "face recognition units". Those units determine whether a face is familiar or unknown. If a face is known, semantic information about that person is accessed through the "person identity node". Prosopagnosia can be the result of a disruption in one of those stages. Prosopagnosia can be caused, as discussed above, both to an acquired brain lesion or the atypical development of one or more of the face recognition subsystems (Susilo & Duchaine, 2013).

A longstanding debate in the literature regards whether face processing is mediated by a specific system, separate from that underlying object processing, or whether its observed specificity is the result of our greater expertise with faces compared to other object categories

(Kanwisher, 2000; McKone, Kanwisher & Duchaine, 2007; Young & Burton, 2018). On one hand, traditional domain-specific accounts of face processing claim the specificity of the Fusiform Face Area for face recognition (Kanwisher, McDermott & Chun, 1997), as part of a face recognition system (Haxby, Hoffman & Gobbini, 2000). Moreover, further evidence is attributed to the existence of the ERP N170 component, which has been reported to be larger in response to faces compared to objects (Bentin, Allison, Puce, Perez & McCarthy,1996; Rossion et al., 2000; Carmel & Bentin, 2002). This account has also been sustained by neuropsychological data as double dissociations of object agnosia and prosopagnosia deficits have been reported (Moscovitch et al., 1997; Geskin & Behrmann, 2018; Riddoch, Johnston, Bracewell, Boutsen & Humphreys, 2008). Moreover, another account favouring the specificity hypothesis is that faces, compared to objects, are processed holistically (Robbins & McKone, 2007). This is demonstrated by effects such as the inversion effect (i.e., a worst performance with inverted compared with upright faces - Yin, 1969), or the composite effect (i.e., the alignment of two half faces belonging to different identities worsens performance in judgment on the single halves compared with misaligned trials - Young, Hellawell & Hay, 2013), and the part-whole effect (i.e., it is easier to discriminate individual facial features presented within the entire face than without the rest of the face - Tanaka & Farah, 1993; Kanwisher, 2000). On the other hand, authors supporting the expertise hypothesis sustain their hypothesis with increased activation of the Fusiform Face Area and Occipital Face Area not only for faces but also for objects for which expertise has been acquired (Gauthier, Tarr, Anderson, Skudlarski & Gore, 1999; Gauthier, Skudlarski, Gore & Anderson, 2000; Burns, Arnold & Bukach, 2019). Moreover, the inversion effect described above has been reported for non-face categories for which the participants were experts (Diamond & Carey, 1986; Rezlescu, Chapman, Susilo & Caramazza, 2016). This effect has also been associated with the N170 ERP component

(Rossion et al., 2002). Additional accounts favouring the expertise hypothesis report evidence for a better memory for faces of the same race than for those of different races, explaining this as being due to greater experience with own-race faces (Bukach, Gauthier & Tarr, 2006). Although this debate has not yet been solved, it raises important points regarding the rehabilitation of face and object recognition. For example, one study claimed that rehabilitation of acquired prosopagnosia is impossible due to the specificity of brain areas subserving it (Coltheart, Brunsdon & Nickels, 2005). On the other hand, if face recognition is the result of expertise with one category, it is likely that training in the acquisition of expertise in a new category improves face recognition.

2.1.3. Rehabilitation of visual agnosia and prosopagnosia

Effective visual recognition is important to many daily tasks (Zihl, 2011). Object agnosia is a rare deficit (according to Zihl & Kennard, 2003 1-3% of brain-damaged patients): however, it might be underestimated (Tikhomirov, Konstantinova, Cirkova, Bulanov & Grigoryeva, 2019). Moreover, developmental prosopagnosia can be found in 2% of the population (Bowles et al., 2009; Kennerknecht, Ho & Wong, 2008). In addition, difficulties in face recognition tests range from 21% to 80% of brain lesion patients, depending on the test administered (Valentine, Powell, Davidoff, Letson & Greenwood, 2006). Difficulties in the recognition of objects and faces can negatively impact patients' social and daily life (Dalrymple et al., 2014). Yet, rehabilitation for visual-perceptual disorders is still in its infancy. Many studies about rehabilitation of visual recognition are non-experimental (Clarke & Bindschaedler, 2005; Anderson & Rizzo, 1995; Raymond, 1996; Burns, 2004). Consequently, it is difficult to draw conclusions about the generalization of the results to different patients and settings. The results did not offer the numbers for quantitative analysis (i.e., meta-analysis), but the presented studies can still direct future research towards promising treatments. A recent review was

published on the rehabilitation of visual agnosia and prosopagnosia (Heutink, Indorf, & Cordes, 2019). The authors described data on the rehabilitation of the deficits together with articles giving suggestions for treatment without reporting any data. The authors display the studies dividing them into compensatory and restorative approaches to rehabilitation. With compensatory rehabilitation the authors refer to treatments aimed at a compensation of the lost function through other functions, while with restorative treatments they refer to treatments aimed at restoring the missing function. Even if the data are too scarce to allow any conclusion, the authors conclude that both compensatory and restorative strategies seem to be useful for the rehabilitation of object agnosia. Moreover, compensatory approaches appear to work best for prosopagnosia. In addition, two previous reviews on the rehabilitation of prosopagnosia came to the same conclusions. Bate and Bennets (2014) stated that compensatory treatments appear to be more effective for acquired prosopagnosia. However, they argue that understanding when restorative treatments are successful would be helpful as the benefits are greater. When talking about developmental prosopagnosia, the authors conclude that data is not enough to make inferences. DeGutis et al. reached a similar conclusion as they found no evidence of the efficacy of restorative treatment on acquired prosopagnosia, while compensatory rehabilitations seem to work in certain instances (DeGutis, Chiu, Grosso & Cohan, 2014). Moreover, the authors underline the existence of different successful approaches to the treatment of acquired prosopagnosia, such as galvanic vestibular stimulation (Wilkinson, Nicholls, Pattenden, Kilduff & Mildberg, 2008). When considering developmental prosopagnosia, the authors discuss evidence of efficacy in both remedial and compensatory studies.

We found these reviews inspiring and used them as a starting point for conducting the present study. However, we believe that a reasons for which solid conclusions were not reached in the

previous reviews could be that there are several methodological limits. Namely, the fact that most studies are single cases, the fact that different tests were used for initial assessment and subsequent assessment of improvement, and the fact that there was a lack of assessment of mid-level visual functions. This is why we performed a new systematic review. Our aim was to help researchers and clinicians considering to perform rehabilitation on patients with agnosia and prosopagnosia with useful information for conducting their studies. To do so, differently from previous reviews, we decided to focus only on experimental studies addressing direct cognitive rehabilitation of visual object agnosia and prosopagnosia. When referring to "experimental studies", we mean studies using quantitative methods to assess the deficit and its improvement and using an experimental procedure as a treatment. This choice was made as, when conducting the literature search, we observed that many studies present non-experimental investigations or data that had already been used previously. This causes a difficulty for clinicians and researchers to replicate the results. In addition, we chose to include studies performed on adults only. This choice was made because rehabilitation is approached differently in children, and we wanted to consider a population that is uniform in an attempt to make the treatments generalizable. Only by doing this, can we try to be as specific as possible in reporting rehabilitation procedures that could be generalized. Moreover, by direct cognitive rehabilitation we mean what can be defined as restorative treatments, in other words those directly targeted at the rehabilitation of the recognition function and not at potential compensatory strategies. This choice was made for many reasons. Quite recently, Heutnik et al (2019) argued that compensatory strategies are effective in most cases, while restorative training produces mixed results. Nevertheless, restorative treatments may help in recovering the function by changing the neural organization subserving it (Raskin & Sohlberg, 2009). In addition, compensatory tools can reduce the effect of the restorative treatments.

Moreover, we believe that reviewing studies on the rehabilitation of both face and object recognition might be helpful in comparing the cognitive mechanisms involved in processing faces and objects. It must be noted that a certain degree of uncertainty in the level of separation between object agnosia and prosopagnosia still exists. Thus, a thorough investigation of the rehabilitation of those functions could represent a new element also in the understanding of the mechanisms subserving them. Given the paucity of published studies on the topic and the lack of standardized methods, the present review will not provide guidelines for the treatment of object agnosia and prosopagnosia. Its aim is that of providing a starting point for clinicians and researchers dealing with such patients in the hope of enhancing literature on the topic.

2.2. Methods

2.2.1. Search strategy

The Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines (Page et al., 2021) were followed. PsycINFO, ScienceDirect, and Pubmed were selected as databases and searched. The following search was used: “Rehabilitation” or “Treatment” or “Training” or “Perceptual Learning”, with each of the terms “Agnosia,” “Prosopagnosia,” “Visual Processing,” “Visual Perceptual Disorders,” “Visual Agnosia,” “Object agnosia,” “Apperceptive Agnosia,” “Associative Agnosia,” “Integrative Agnosia,” and “Form agnosia.” The search for peer-reviewed publications was conducted by two of the authors (SG and EP) independently. Articles were initially screened for their title, later for abstract and finally for the full text. The authors SG and EP discussed their choices after each of the phases described. Any conflict was solved by discussion with the supervision of RD and RC. Studies up to February 2022 were considered. The titles and abstracts of the results were screened to find the articles meeting our inclusion criteria. If sufficient information was not obtained through the title and abstract, the full text of the article was considered. The full texts of all articles meeting the inclusion criteria were obtained. The reference lists of the identified studies were also searched to identify potential additional items.

2.2.2. Study selection

Inclusion criteria were the following: a) studies describing empirical evidence for cognitive rehabilitation of visual agnosia and prosopagnosia; b) studies reporting the impairment as part of a global deficit or in conjunction with other impairments (we expected the literature to be too scarce only to consider “pure” impairments); c) articles published in English language. Exclusion criteria were the following: a) not human species studied; b) deficit caused by a psychiatric disorder or a disorder belonging to the autism spectrum; c) studies exclusively

dealing with cognitive domains other than visuoperceptual; d) studies only treating reading difficulties; e) studies describing the treatment of peripheral visual dysfunction, cortical blindness or visual field deficits; f) studies describing non-cognitive treatments; g) studies describing treatment of neurodegenerative diseases; h) unpublished data; i) studies describing compensative strategies as a treatment j) studies considering children or participants in their developmental age. Although we aimed to also include group studies as randomized controlled trials, in practice, most of the studies meeting our criteria considered single-case designs. No pre-registration was made for the following study. When missing data were found, the authors were reached via mail to retrieve it. Authors of three articles were contacted (Humphreys & Riddoch, 1994; Wilson et al., 1999; Zihl et al., 2011). Two of them replied (Wilson et al., 1999; Zihl et al., 2011), one (Zihl et al., 2011) was able to provide the necessary data.

2.2.3. Rating of quality of reporting

Most of the selected articles describe single-subject design studies. Thus, to evaluate the quality of reporting the Single-Case Experimental Design (SCED) scale (Tate, McDonald, Perdices, Togher, Schultz & Savage, 2008) was used. Three articles described case-control studies: in that case, the Critical Appraisal Skills Programme (CASP, available at https://casp-uk.net/casp-tools-checklists/) checklist was used (Ma, Wang, Yang, Huang, Weng & Zeng, 2020) to evaluate the quality of reporting. The authors SG and EP independently rated each paper. They gave a score of 1 if the item was clearly present throughout the article and 0 if it was not. Any incongruency was then discussed to agree on a final score. The total score ranged from 0 to 10 (items 2 to 11): higher scores represent higher quality of reporting. It must be underlined that scores are indicative and must not be taken as an evaluation of the article (Ma et al., 2020).

2.2.4. Risk of bias assessment

Risk of bias in the present review was assessed with the Risk Of Bias In Systematic Reviews (ROBIS) scale (Whiting et al., 2016). This scale is a tool comprising three phases: the first one is optional and is called the “assessment of relevance”. The second one is the “identification of concerns in the review process”, and the third is to “judge the risk of bias”.

2.3. Results

The selection process resulted in the inclusion of 17 studies. 7 studies concern single cases on the rehabilitation of object agnosia, 8 studies regard rehabilitation of acquired or developmental prosopagnosia. In addition, 2 studies describe training for both object agnosia and prosopagnosia. The articles considered were published between 1991 and 2019. Two studies were found by going through citations of other studies (Wilson, 1999; Davies-Thompson, Fletcher, Hills, Pancaroglu, Corrow & Barton, 2017). Below a description of results relative to object agnosia and prosopagnosia.

2.3.1 Risk of bias assessment

The assessment of Risk of bias resulted in a low risk of bias for the present review. For more details, please contact the corresponding author.

2.3.2. Object Agnosia

Two tables summarize the results obtained for the rehabilitation of visual object agnosia. As all the studies are single cases and patients present many differences, we filled Table 2.1 to summarize the localization and etiology (when reported) of the lesion of each patient and Table 2.2 to describe the characteristics of the studies. Below a discussion of each table separately can be found.

2.3.2.2. Lesions localization

Table 2.1 summarises the location of the lesions if specified. For each patient, each box corresponds to a cerebral area (Temporal lobe, Parietal lobe, Occipital lobe, Frontal lobe, Corpus Callosum, Basal Ganglia) of the right and left hemispheres respectively. Four patients (36%) underwent a Computerized Tomography (CT) scan (Behrmann et al., 2005; Rosenthal & Behrmann, 2006; Rosselli, Ardila & Beltran, 2001, Polanowska, Mandat, Laudanski, 2003) and two patients (18%) Magnetic Resonance Imaging (MRI) (Behrmann et al., 2005; Tanemura, 1999). Although heterogeneous with respect to the location and nature of the brain damage, the reported cases share common traits. Four out of six patients reporting data on lesions are reported to have a bilateral lesion and one (P2 in Zihl, 2011), was expected to have it (closed head injury and hypoxia). Patient SM (Behrmann et al., 2005) was described as having right damage. However, the circumstances of the injury (severe head trauma in a motor vehicle accident) cannot exclude a bilateral hemispheric involvement. Two patients (JW; P2 by Zhil 2011) suffered from anoxic encephalopathy (in P2 associated with a head injury). This condition is known for producing bilateral posterior cortical damage in the watershed areas; this in turn is associated with frequent involvement of the subcortical structures.

2.3.2.3. Studies description

A description of the studies that were included can be found in Table 2.2. Visual agnosia was assessed by using standardized neuropsychological assessments together with qualitative observations, and tests created ad hoc. Four studies describing five patients (45%) (Behrmann et al., 2005; Lev 2015; Rosenthal & Behrmann, 2006; Wilson, 1999) reported assessing the integrity of low-level visual processes. A control condition was established in the treatment of 5 (45%) of the patients described. This was done to address the possibility that improvement was due to a spontaneous recovery. In two studies (18%) (Humpreys & Riddoch, 1994;

Rosenthal & Behrmann, 2006), a control group was used as a baseline against which to compare the performance of patients. In one study describing two patients (18%) (Wilson, 1999), spontaneous recovery was checked for untrained items. Moreover, in another study describing two patients (18%) (Zihl, 2011) the author, after an initial assessment, introduced a waiting period of several weeks followed by another assessment to check whether spontaneous recovery occurred. The treatments differed from one other. Given the diversified nature of the studies, we decided not to use the distinction between compensatory and restorative rehabilitation. We divided treatments into those in which a generic rehabilitation was administered and those in which a specific rehabilitation was administered. By generic rehabilitation we meant a rehabilitation targeted to multiple cognitive functions (used for three patients (27%) Rosselli et al., 2001; Seniow et al., 2003; Tanemura, 1999), while with specific rehabilitation we intended a treatment targeted at the visual perceptual deficit. The latter consisted of exercises specifically targeting low-level visual functions in one case (9%) (Lev, 2015), visual perceptual abilities in another case (9%) (Humphreys & Riddoch, 1994), classification of simple (Rosenthal et al., 2006) and more complex (Behrmann et al., 2005) visual stimuli based on their visual features respectively in one study each (9%), a visual analysis of objects for two patients (18%) (Zihl, 2011), and labelling of drawings for the two remaining cases (18%) (Wilson, 1999).

Importantly, the duration of the treatment was not specified for two patients (18%) (Tanemura, 1999; Humphreys & Riddoch, 1994). In the remaining studies treatment duration varied, ranging from a period of 15 days (P1 from Zihl, 2001) to 1 year (Seniow, 2003). Improvement after training was measured by monitoring the accuracy of the task in the case of seven patients (64%) (Behrmann et al., 2005, Humphreys & Riddoch, 1994; Rosenthal & Behrmann, 2006; Wilson, 1999; Zihl, 2011). Alternatively, improvement was measured by administering the

same neuropsychological tests used pre-treatment for four patients (36%) (Lev et al., 2015; Rosselli et al., 2001; Seniow et al., 2003, Tanemura, 1999). Rehabilitation was effective for all patients. In those using specific stimuli that were created ad-hoc, generalization was assessed as effective for three patients (27%) (Behrmann et al., 2005; P1 and P2 from Zihl, 2011). It was not effective in 2 cases (18%) (Paula from Wilson, 1999; Humphreys & Riddoch, 1994), and partially effective in one case (9%) (Jenny from Wilson, 1999). A follow-up evaluation was performed for five patients (45%) (Lev et al., 2015; Rosenthal et al., 2006; P1 and P2 from Zihl, 2011; Jenny from Wilson, 1999), all of which showed consistency in results over time.

2.3.2.4. Quality of reporting

SCED scale scores can be found in Table 2.2. It must be noted that one study could not be evaluated due to insufficient information (Humphreys & Riddoch, 1994). When looking at the assigned scores, we noted their heterogeneity. Among many factors, this can be attributed to many cases of missing information. For some cases, however, missing data could be retrieved from the authors (e.g., Zihl, 2011). Thus, the results of the quality of reporting ratings are difficult to compare. A characteristic that stood out is that the authors did not account for inter-rater reliability in evaluating post-treatment scores, nor were the indicators of improvement independent of the data used to assess the pre-treatment baseline. Two items worth considering in our discussion of object agnosia have different scores on the SCED scale. The first is the replication of the results which was assessed across time for Rosenthal et al., 2006 and across patients (Zihl 2011), and the second is the observation of evidence for generalization of the results which was assessed in Behrmann et al., 2005; Lev et al., 2015 and Paula from Wilson et al., 1999.

2.3.3. Prosopagnosia

Results obtained for prosopagnosia are summarized in three tables. Studies concerning acquired and developmental prosopagnosia will be discussed separately. The studies were classified as perceptual or mnemonic treatments following the distinction from Davies-Thompson et al. (2017). The authors define mnemonic treatments as treatments referring to interventions aimed at associating faces to specific semantic information and perceptual treatments as based on the perceptual analysis of faces.

2.3.3.1.1 Acquired prosopagnosia

Table 2.3 presents the results from the rehabilitation of acquired prosopagnosia. It must be noted that the deficit differs greatly from one study to another. Two studies (29%) describe patients with a semantic impairment accompanying prosopagnosia (De Haan, Young & Newcombe, 1991; Francis, Riddoch & Humphreys, 2002). Four studies (57%) describe patients having other visual perceptual deficits in addition to prosopagnosia (De Haan et al., 1991; Francis et al., 2002; Behrmann et al. 2005; Zihl, 2011). Finally, two studies (29%) describe cases of relatively "pure" prosopagnosia (Polster & Rapcsak, 1996; Davies-Thompson et al., 2017). A control condition was used in four (57%) studies (Davies-Thompson et al., 2017; Francis et al., 2002; Powell, Letson, Davidoff, Valentine & Greenwood, 2008; Zihl, 2011). Three studies (43%) report perceptual treatments (Davies-Thompson et al., 2017; Behrmann et al. 2005; Zihl, 2011). Two studies (29%) describe a mnemonic treatment (De Haan et al., 1991; Francis et al., 2002). Finally, two studies (29%) compare mnemonic and perceptual rehabilitations (Polster & Rapcsak, 1996; Powell et al., 2008). Improvement is reported to be poor for mnemonic treatments and better for perceptual ones, except for one case (Polster & Rapcsak, 1996). The duration of the treatment was reported for 5 studies (71%) and ranged

from a period of approximately two weeks (Powell et al., 2008; Zihl, 2011) to a period of four months (Behrmann et al. 2005). Follow-up assessment was reported in three studies (43%) (De Haan et al., 1991; Davies-Thompson et al., 2017; Francis et al., 2002), and improvement was maintained in two studies (29%) (Davies-Thompson et al., 2017; Francis et al., 2002).

2.3.3.1.2 Lesions localization

Table 2.4 reports the location of the lesions in the described cases of acquired prosopagnosia. For each patient, each box corresponds to a cerebral area (Temporal lobe, Parietal lobe, Occipital lobe, Frontal lobe, Corpus Callosum, Basal Ganglia) of the right and left hemispheres.

The right hemisphere is always involved, particularly the temporal lobe (temporal lobe involvement is assumed in P2, Zihl et al., 2011). For six cases, the lesion also extends to the occipital lobe. In four out of seven patients, the damage is bilateral, while temporo-occipital involvement is reported in three cases.

2.3.3.1.3 Ratings of quality of reporting

For acquired prosopagnosia, quality of reporting was assessed using the SCED and CASP scales, depending on whether the design was a single case or not. Thus, the results cannot be compared with each other. A factor that stands out when performing the CASP scale (Davies-Thompson et al., 2017) is that in the item relating to the evaluation of potential confounding factors in the design and/or the analyses, the response is “can’t tell”. This is mirrored in the SCED items with respect to the inter-rater reliability and independence of assessors, always resulting in a “no”. The items relating to replication and generalization gave mixed results as happened for object agnosia. Neither replication nor generalization was reported in four studies (Polster & Rapcsak, 1996; De Haan et al., 1991; Francis, Riddoch & Humphreys, 2002; Powell et al., 2008). Generalization, but not replication, was reported in two studies (De Gutis, Bentin, Robertson & D’Esposito, 2007; Behrmann et al., 2005), while replication across subjects was partially obtained in one study (Zihl, 2011).

2.3.3.2.1 Developmental prosopagnosia

Criteria for diagnosing developmental prosopagnosia have not been discussed until recently (Barton & Corrow, 2016; Dalrymple & Palermo, 2016). Thus, the inclusion criteria for the selected studies are heterogeneous. A control condition was planned for all the selected studies. All the studies describe perceptual treatments. The duration of the training ranged from two weeks (De Gutis et al., 2007) to eleven weeks (Corrow et al., 2019). Improvement occurred for all the patients described. A follow-up was carried out in two of the three studies (De Gutis et al., 2014; Corrow, Dalrymple & Barton, 2019) but maintenance of improvement was reported only in one case (Corrow et al., 2019).

2.3.3.2.2 Ratings of quality of reporting

Quality of reporting in developmental prosopagnosia studies was evaluated as high. however, for both object agnosia and acquired prosopagnosia, inter-rater reliability and independence of assessors were not assessed. Generalization but not replication, assessed using the SCED scale, was reported in De Gutis et al., 2014. However, it must also be noted that both Corrow et al. (2019) and DeGutis et al. (2014) adapted a paradigm that had been used previously to a new population. In this sense, we can consider that these two articles are a replication of previous results. Even if this specific item is not present in the CASP scale, it is important to note that generalization was also marginally present in Corrow et al. (2019) and present for untrained stimuli and task formats, although not for untrained views in DeGutis et al. (2014).

2.4. Discussion

The purpose of the present article was to review systematically rehabilitation studies present in the literature, providing experimental evidence for current cognitive rehabilitations for visual object agnosia and prosopagnosia. Particularly, we aimed at providing information useful for authors willing to carry on a cognitive rehabilitation of visual object agnosia and prosopagnosia. We focused on original studies performed on adults using quantitative methods to assess the deficit and its improvement and by using an experimental procedure as a rehabilitation. Moreover, the present study aimed to focus on the cognitive mechanisms subserving object agnosia and prosopagnosia, still discussed in the literature. The PRISMA method was used, and seven articles were selected for object agnosia, eight articles for prosopagnosia, and two articles for both deficits. Results obtained from the quality of reporting of the studies are taken into consideration in the discussion of findings.

2.4.1. Object agnosia

Different treatments for visual agnosia emerged from the selected articles. We separated them into generic and specific treatments, where generic refers to those rehabilitation interventions that were not primarily aimed at the recovery of object recognition, but targeting several cognitive functions, and those which are specifically targeted to a cognitive function (see Table 2.2). Even if not primarily aimed at a recovery in visual agnosia, generic treatments caused an improvement in object recognition. This result has a clinical validity; however it is challenging to interpret it in terms of generalization to other patients. Indeed, the improvement of visual agnosia could be attributed to many indivisible uncontrolled factors.

When considering specific treatments, the authors of the remaining articles attempted to treat visual object recognition deficits in many ways. Patients impaired in low-level visual functions

were rehabilitated with basic visual perceptual tasks and categorization of simple visual stimuli. Some showed an improvement in the specific task (Rosenthal & Behrmann, 2006), other showed an improvement extending to visual object recognition (Lev et al., 2015) and maintained over time. When both low- and high-level visual impairments occur, it is challenging to isolate the single contribution of the specific deficits. However, it is possible that treating low-level visual functions leads to improvement in higher visual processes. This is likely not only because it trains low-level functions themselves, but also because it may trigger plasticity at a higher cortical level (Ahissar & Hochstein, 2004). Humphreys and Riddoch (1994) put together different approaches and found out that grouping, object recognition, and pattern identification led to an improvement which was specific only to the trained items. Wilson (1999) focused on naming line drawings (Paula) and to copying and visually analysing them (Jenny), finding an improvement in the trained items for both the trained patients (Paula and Jenny) and a generalization to other similar objects for one patient (Jenny). These improvements were maintained over time for one patient (Jenny). Behrmann et al. (2005) performed a rehabilitation based on categorizing unknown stimuli (i.e., greebles) differing for their internal features. This rehabilitation was effective and generalized to object recognition. Zihl in 2011 treated two patients with visual object agnosia through a visual analysis of photographs of objects. This treatment resulted in an improvement in the trained stimuli and in a generalization to other objects. The treatments these two authors performed are quite different. First, Behrmann et al. (2006) used unknown stimuli, while Zihl (2011) used known objects. Moreover, Behrmann performed a categorization task, while Zihl was focused on visual analysis. Nevertheless, both rehabilitations require a visual analysis of the object parts with the difference that in one case (Zihl, 2011) this is explicit, while in the other (Behrmann et al., 2005) this is functional to the categorization. Using spared analysis of the object parts to

rehabilitate object recognition is consistent with a study by Behrmann et al. (2006) describing two patients. One patient (S.M.) who was the same integrative agnosic patient receiving the rehabilitation with Greeble stimuli, was impaired in detecting the spatial arrangement between object parts rather than a change of a single part. Thus, it is likely that an analysis of object parts may accounts for the success of a treatment based on feature discrimination, provided that the parts are not too numerous (Behrmann & Williams, 2007).

Because the localization of the lesion is considered, the patients who were reported with object agnosia suffered from widespread and severe brain damage. This in turn makes it challenging to identify the neural basis of the neuropsychological disorder. One feature that needs to be underlined is that damage is bilateral and mostly in posterior regions. In addition, brain lesions were generally not described in depth. Furthermore, the possible relationship between lesion localization and cognitive symptoms was not discussed. Eventually, neuroimaging techniques were less advanced at the time when the cases were reported. Nevertheless, the observed low interest in the lesion data is likely to reflect a general "cognitive" approach to rehabilitation.

It must be added that a developmental form of object agnosia has also been described (Germine, Cashdollar, Düzel & Duchaine, 2011). As it was found that treatments that were useful for developmental prosopagnosia were also suitable for use in the acquired cases, we suspect that the same treatment used for acquired object agnosia would also work in such cases. However, as the literature available is limited, it is difficult to draw any definite conclusion.

2.4.2. Prosopagnosia

In the case of prosopagnosia, both acquired and developmental cases were described.

In the case of acquired prosopagnosia, the pattern of brain lesions always involved the right hemisphere. Therefore, according to the brain imaging data reported in the considered articles

for the present review, right hemisphere damage appears to be the condition necessary for developing prosopagnosia. More specifically, a lesion encompassing the right occipitotemporal regions and presumably including the right fusiform gyrus. Although acquired prosopoagnosia is associated with lesions confined to the occipito-temporal regions of the right hemisphere (De Renzi et al., 1994), a left-handed prosopoagnosic patient with a lesion in the left occipital and face areas (Barton, 2008) has been also documented. It should be acknowledged, however, that this patient, additionally to visual perceptual deficits, also presented difficulties in accessing semantic and autobiographical information conveyed by name, which is generally of left hemispheric competence. Thus, the author concluded that the disorder of the described patient reflects an abnormal lateralization of visuoperceptive functioning, rather than reversed lateralization of functions. Different possible rehabilitation for both acquired and developmental prosopagnosia were described in the selected articles. That is why we divided them into mnemonic and perceptual treatments (following the distinction from Davies-Thompson et al., 2017).

A completely perceptual treatment was administered to all congenital prosopagnosic patients described in the studies examined, and in four of the papers on acquired prosopagnosis. Perceptual treatments involved analysis of visual features (Zihl, 2011), face matching (Davies-Thompson et al., 2017; Corrow et al., 2019), face discrimination (De Gutis et al., 2007; DeGutis et al., 2014), and categorization non-face stimuli (Behrmann et al., 2005). Perceptual treatment was found to be effective, but only when using face-like stimuli. Moreover, it generalized to new faces and different views only when discrimination or matching tasks were applied. The analysis of single features was not effective.

A mnemonic treatment was applied in two papers on acquired prosopagnosics with a little improvement in one case (Polster & Rapcsak, 1996) and a big improvement in the other

(Francis et al., 2002). However, the latter describes a patient with a prevalent mnemonic rather than perceptual deficit.

Finally, a combination of the two types of treatments was applied in two studies on acquired prosopagnosics (Polster & Rapcsak, 1996; Powell et al., 2008). In one case, there was a marginal improvement after mnemonic/semantic treatment which, however, did not generalize to new views of faces (Polster & Rapcsak, 1996). In the other case, the other the prosopagnosic patient only benefited from a perceptual analysis of the single parts (Powell et al., 2008).

In summary, perceptual treatments consisting of discrimination or matching faces were effective, while mnemonic treatments were effective in a patient with a clear mnemonic impairment (Francis et al., 2002). It must be noted that both acquired and developmental prosopagnosia can manifest with apperceptive, associative, or amnesic deficits (Davies-Thompson, Pancaroglu & Barton, 2014). Thus, the efficacy of mnemonic or perceptual types of treatment inevitably depends on the nature of prosopagnosia and on the presence of associated deficits. Therefore, tests to understand prosopagnosia subtypes better are crucial to address treatments tailored to specific patients.

It must be noted that similar treatments resulted as effective for both acquired and developmental forms of prosopagnosia.

Developmental prosopagnosics, differently from acquired prosopagnosics, might have found a way to compensate for the deficit since it was present from birth (Behrmann & Avidan, 2005). Yet, perceptual treatments seem to be effective despite the prosopagnosia subtype.

Another issue arising from this review is that perceptual treatments need face-like stimuli to be effective. Coherently with that, authors who used non-face-like stimuli to train an acquired prosopagnosic noticed a worsening of the patient's performance with faces (Behrmann et al., 2005). According to the authors, a possible interpretation of this result can be that there is a

competition between objects for which we are experts. As explained by the authors, it could be the case that face and greeble processing rely on shared psychological and neural systems and, once greebles are trained, these mechanisms become less specific for faces (Behrmann et al., 2005). Indeed, it has been observed that objects for which we are experts trigger holistic processing, similar to that used for faces (Gauthier, Curran, Curby & Collins, 2003). Thus, it is likely that as greebles became objects of expertise, they triggered holistic processing; this, in turn may have competed with the processing of faces. On the other hand, as suggested by the authors, it could be that greebles were not an adequate stimulus to trigger holistic processing but triggered part-based processing instead. Therefore, if this is the case, it might be that stimuli capable of triggering holistic processing, if used, would be equally effective compared to faces as a treatment of prosopagnosia.

2.5. General discussion

Visual object agnosia and prosopagnosia are exhausting cognitive deficits that may lead to life-changing outcomes (Riddoch & Humphreys, 1987). Yet, protocols for their neuropsychological rehabilitation are lacking, leaving such patients with their visual recognition impairment. This may be partially accounted for by the fact that cognitive mechanisms subserving object and face recognition are still not clear. This review aimed at discussing the cognitive mechanisms of face and object recognition. Moreover, it was aimed at guiding clinicians managing agnosic patients and, at the same time, address future research on this matter. By taking into consideration recent reviews on the rehabilitation of agnosia or prosopagnosia (Heutink et al., 2019; Bate & Bennets, 2014; De Gutis et al., 2014), we performed a new review only selecting the original experimental studies regarding adults on the rehabilitation of visual object agnosia

and prosopagnosia. The overarching aim of the present review was that of obtaining a comparison between the approaches towards these two deficits.

The definition of "associative agnosia" includes a wide variety of deficits (Farah, 2004). It emerges from the literature that treatments using semantic memory result in improvement in patients with an associative/semantic deficit (e.g., De Haan et al., 1991; Wilson, 1999), while a perceptual training does not (De Haan et al., 1991). Conversely, when the deficit is perceptual in nature, perceptual treatments are more effective (e.g., Powell et al., 2008). However, it is challenging to consider semantic and mnemonic treatments of the agnosic deficit. Moreover, the material on the topic is scarce. Thus, we did not focus on semantic nor mnemonic treatments of both object agnosia and prosopagnosia.

When considering perceptual treatments, a first question that arises regards the differences between object and face processing. Understanding mechanisms underlying these two processes might be fundamental to address specific treatments and vice versa.

On one hand, there is still no agreement in the literature as to whether faces are special stimuli because of their social relevance or if we process them separately from objects (Young & Burton, 2018). If the former interpretation is true, as expertise with faces results from our exposure to faces in our lifetime, we would expect a prosopagnosic having trouble in learning to discriminate exemplars belonging to any new category for which he/she has acquired expertise. This, however, was not observed in the study by Duchaine et al. (2004) where a severe prosopagnosic could learn to recognize greebles within ten sessions (Duchaine, Dingle, Butterworth & Nakayama, 2004).

The same happened when looking at two acquired prosopagnosics in a study by Rezlescu et al. (2014) showing improvement in greeble but not face learning (Rezlescu, Barton, Pitcher &

Duchaine, 2014). Rezlescu and colleagues (2014) interpreted their results in terms of domain specificity of face processing, Duchaine et al. (2004) maintained that greebles did not elicit expertise, and the improvement of the patient could be explained by him using intact object-recognition mechanisms. The latter interpretation is coherent with the results of Behrmann et al. (2005). Their patient (S.M.) learned to classify greebles; this in turn was reflected in an improvement in object recognition mechanisms. Coherently, we could also explain the competition that was observed between greebles and faces. It might be that greebles were never processed holistically. Consequently, learning greebles was not able to train the type of perceptual processing needed for faces.

Bukach et al. (2012) trained L.R., an acquired prosopagnosic patient, using greebles and found that he could learn to classify them (Bukach et al., 2012). However, he needed more sessions than the controls: the authors interpreted this result as an abnormal perceptual processing strategy. They concluded that his face recognition impairment derived from a general difficulty in object recognition. Particularly, both developmental and acquired prosopagnosia can be reported in concomitance with a more general object processing deficit (Geskin & Berhmann, 2018). Moreover, patients with both acquired object agnosia and prosopagnosia were found to share abnormal crowding (Strappini, Pelli, Di Pace & Martelli, 2017; Sand, Robotham, Martelli & Starrfelt, 2018). Thus, a clear dissociation between the two deficits and their respective rehabilitations was rather difficult. Consequently, more rehabilitation studies are required.

Independently from the ongoing specificity-expertise debate, it emerges from the present review that face and object recognition benefit from different trainings. Particularly, holistic training of face processing resulted in better face recognition abilities (DeGutis et al., 2007; DeGutis et al.; Davies-Thompson et al., 2017; Corrow et al., 2019). Moreover, it appears that eyes, eyebrows, and to a lesser extent the mouth, are critical regions for face discrimination.

Moreover, prosopagnosics rely quantitatively less on those features with respect to poor recognizers (Tardif et al., 2019). This can be attributed to the fact that the eyes and the mouth (even though to a lesser extent) represent configurations (Caldara, Schyns, Mayer, Smith, Gosselin & Rossion, 2005; DeGutis, Cohan, Mercado, Wilmer & Nakayama, 2012). On the other hand, when looking at results from our review, object recognition benefits from part-based training (Behrmann et al., 2005; Zihl, 2011). It must be noted that exception to this distinction is represented by the study of Powell et al. (2008).

While examining the articles found in this review, it became clear that there are many aspects to consider in planning new rehabilitative studies on visual object agnosia and prosopagnosia to guide researchers and clinicians. First, the fact that there is a lack of a unitary account explaining the deficits reflects an eclectic clinical assessment. Patients are often described only qualitatively. We believe that it is vital to assess visual recognition impairment more systematically. It is crucial for a thorough assessment to consider low-level visual disturbances. This must be done to exclude the possibility that a recognition deficit is the result of a peripheral visual deficits (Bauer, 2006) or low/middle vision deficits. Only then a specific assessment of visual object agnosia and prosopagnosia can be carried out (e.g., Riddoch & Humphreys, 1993; Vancleef et al., 2015; Warrington & James, 1991). When considering prosopagnosia, it is important to evaluate object recognition ability. This may allow us to observe whether some treatments are more effective in patients showing selective deficits for faces or shared deficits for faces and objects. A further consideration on neuropsychological assessment is the urge to specify if patients suffer from an apperceptive or an associative deficit. In the studies reviewed hereby, this distinction could not be made, and this was due to heterogeneity of evaluations. We believe it may be important for readers to know which rehabilitation worked on which specific subsample of patients. In addition, it is important to find a control condition.

Otherwise, it becomes difficult to know whether the improvement was due to spontaneous recovery or rehabilitation.

Another issue that should be considered is generalization. The first aim of neuropsychological rehabilitation should be that the patient improves not only in the trained tasks but also in other related tasks (Humphreys & Riddoch, 1994) and, ultimately, in everyday life. Consequently, it is important to assess the patient's generalization and daily functioning to assess the efficacy of the treatment. For the same reason, it is important to plan a follow-up evaluation to determine whether the progress is maintained over time.

From the current literature analysis, it emerged that many rehabilitations are carried out by following a trial-and-error approach. This in turn causes a clinical improvement specific for the treated patient. Consequently, these approaches are not suitable to be extended to other patients. Thus, it is evident that it is impossible to carry out a generalizable rehabilitation of visual object agnosia and prosopagnosia without referring to a cognitive theory. A novel account on visual recognition deficits can be found in the work from Strappini et al. (2017). The authors, through 14 visual recognition tests, hypothesize a parallel between the performance of patients with visual agnosia and that of healthy individuals tested in the condition of peripheral vision. Peripheral vision of complex images is constrained by visual crowding, defined by the authors as "the failure to identify a simple object (like a letter) because of surrounding clutter." The authors hypothesize that this phenomenon drives object recognition difficulties in a domain-general fashion. We believe that comparing classic and novel theories on visual object agnosia and prosopagnosia through the implementation of different assessment tools and rehabilitation programs may help in shedding light on the mechanisms underlying the deficits.

In conclusion, two treatments specific for object agnosia were shown to be effective. One using categorization of greebles (based on their parts) while the other using a part-based analysis of objects. Thus, a task that involves an analysis of object parts might be the best choice in rehabilitating object agnosia. When considering apperceptive prosopagnosia, treatments based on holistic perceptual processing (categorization or matching) of faces appear to be the most effective and have also been shown to generalize to new views and perspectives of faces. Thus, it appears that a holistic analysis of face-like stimuli is effective for both in the rehabilitation of acquired and developmental prosopagnosia. However, literature on object agnosia and prosopagnosia rehabilitation is still circumscribed and new studies addressing the topic are needed.

Tables

	Right hemisphere						Left hemisphere						Etiology
	T	P	O	F	CC	BG	T	P	O	F	CC	BG	
SM (Bermann et al., 2005)	*				*	*							Head injury
JW (Rosenthal et al., 2006)		*	*						*				Anoxic encephalopathy
XX (Rosselli et al., 2001)		*	*					*	*				Fat embolism
DE (Seniow et al., 2003)	*	*	*					*	*				Gunshot
YY (Tanemura, 1999)								*	*	*	*		Stroke
P1 in: Zihl, 2011	*		*				*		*				Stroke
P2 in Zihl, 2011	Closed head trauma with severe chronic hypoxia; presumably frontal or frontotemporal involvement for the trauma and biparietal involvement for hypoxia												

Table 2.1. Localization of lesion in patients with object agnosia (when reported in the study): the asterisk indicates the damaged area.
abbreviations: T = temporal; P = parietal; F = Frontal; CC = Corpus Callosum; BG = Basal Ganglia

Authors	SCED score (0-10)	Deficit(s)	Control condition	Treatment description/dependent variables of assessment	Treatment duration	Results of treatment	Follow up (if present)
Behrmann et al. 2005 Patient SM	7	**Preserved**: - low-level visual processing; - matching of objects from different viewpoints or along a foreshortened axis. **Impaired** performance in: - BNT[1]; - discrimination of exemplars within a single category; - recognition of photographs of a famous individual; - BFRT[2].	None for behavioral training. fMRI: 2 control groups matched for age and education as the baseline.	**Specific treatment**: Categorization of unknown stimuli (Greebles). **Improvement:** accuracy and RTs for each session. **Generalization:** accuracy for non-trained greebles, objects, and faces. fMRI acquisition pre- and post-training.	4 months (twice a week).	Significant improvement for accuracy and RTs over session. Generalization to untrained greebles and objects but worsening of performance with faces. fMRI: face-selective voxels activated more by greebles and less by faces	No follow up.
Humphreys & Riddoch 1994 Patient HJA	-	**Preserved:** - draw objects from memory; - accurate descriptions of visual attributes of objects. **Impaired**: - object recognition (object decision task; - semantic categorization; - naming objects; - situations where segmentation of a stimulus from a complex background is needed.	For the first experimental condition: one young and one age-matched control subject.	**Specific treatment**: grouping (1), object identification (2), and pattern recognition (3) tasks. **Improvement:** accuracy and RTs (1), test-stimuli created ad hoc (2), accuracy (3). **Generalization**: accuracy for non-trained items.	Not fully specified.	RT decrease (1); good item-specific learning for line drawings but not for photographs (2); learning specific to trained items (3).	No follow up.
Lev et al. 2015 Patient LG	5	**Impaired**: - low-level vision (visual acuity, crowding, lateral interactions, stereoacuity); -mid-level vision (contour detection threshold, perceptual organization: L-POST); - high-level vision: BORB[3], VOSP[4], BFRT[2], HVOT[5].	No control condition.	**Specific treatment**: contrast detection, lateral masking. **Improvement:** pre- vs. post-treatment scores of neuropsychological tests.	9 months (3 times a week).	Improvement in low, mid, and partially high-level visual functions.	Follow up after 4 years: performance within the norm in a subset of cognitive tests.

Rosenthal et al.2006 Patient JW	5	**Preserved**: - visual acuity; - color-orientation adaptation; - coarse shape discrimination; - color parallel search; - object imagery and memory; - binocular visual-motor coordination. **Impaired**: - fine orientation discrimination; - contrast sensitivity; - figure-ground segregation; - shape segmentation; - gestalt grouping; - contour integration; - symmetry judgement; - object, faces and letter recognition; - aspect ratio discrimination; - fine shape discrimination; - parallel search for orientation; - monocular visual-motor coordination.	Four healthy controls matched for gender, age, and education.	**Specific treatment**: classification learning of stripe stimuli. **Improvement:** accuracy in the trained task. **Generalization**: not assessed.	2 months (8 sessions).	Improved in learning to classify stimuli despite the persistent deficit in perceiving the simple individual stimuli.	Follow up after 6 months: same results as post-treatment.
Rosselli et al. 2001 Unknown name patient	4	**Impaired**: - abilities involved in Balint's syndrome; - reading: alexia without agraphia (letter reading, word reading); - faces recognition (famous people photographs); - visual agnosia for schematized objects (recognition of schematized figures, recognition of overlapped figures); - memory capacity (WMS[6] MQ).	No control condition.	**Generic treatment:** eye movements, convergence, word reading, writing, visuokinetic functioning, visual search, trail making. **Improvement:** pre- vs. post-treatment scores of neuropsychological tests.	1 year (twice a week).	Improvement, particularly for tests sensitive to scanning deficits (TMT, word reading) and measuring simultanagnosia (ROCF-copy). Significant increase in the patient's everyday life with his return to work.	No follow up.
Seniow et al. 2003 Patient DE	5	**Impaired**: - performance in non-verbal IQ of WAIS[7]; - visual Perception and Memory: BVRT[8].	No control condition.	**Generic treatment:** paper and pencil exercises for visual analysis and synthesis: computer training for verbal memory, visuoperceptual, visuospatial, and constructive functions; occupational therapy.	1 year (twice a week).	Gradual improvement of performance in all affected cognitive domains.	No follow up.

				Improvement pre- vs. post-treatment scores of neuropsychological tests and GOS[9].			
Tanemura 1999 Unknown name patient	2	**Preserved** (VPTA[10]) - discrimination of shapes; - naming colors. **Borderline** (VPTA[10]): - selection of colored pencil; - naming pictures. **Impaired** (VPTA[10]): - changes in visual experience; - naming object and picture of situations; - familiar and unfamiliar face recognition.	No control condition.	**Generic treatment**: improvement of visual perception using the kinesthetic sense. **Improvement:** pre- vs. post-treatment scores to neuropsychological tests.	Not specified.	Improvement of performance for object agnosia, picture agnosia, and pure alexia but not for prosopagnosia.	No follow up.
Zihl 2011[1] Patient 1 Patient 2	4	**Preserved:** - DS[11]; - WAIS[7] logical memory and logical reasoning; - AAT[12] without visual items Visual tests were not applied; - sustained and focused attention (behavioral level).	Visual agnosia persisted in P1 for 15 weeks without significant recovery.	**Specific treatment** in three steps involving analysis of photographs of visual objects belonging to four visual categories. **Improvement**: accuracy. **Generalization:** new visual categories.	15 days (30 sessions).	Improvement in the trained object class. Generalization occurred for objects.	Follow up after 6 months: further improvement in object recognition.
	4	**Preserved**: - DS[11]; - AAT[12] without visual items. **Impaired:** - WAIS[7] logical memory.	Visual agnosia persisted in P2 for 22 months without significant recovery.		28 days (112 sessions).		
Wilson 1999 Patient Paula	4	**Preserved:** - visual field; - visual acuity; - memory for Faces; - auditory agnosia was excluded.	The patient was checked for spontaneous recovery in	**Specific treatment**: line drawings were shown to the patient and named once. Not labelled drawings served as control. **Improvement:** accuracy.	6 months (10 sessions).	Improvement in the trained stimuli but no generalization to the untrained ones nor to the alternative versions of the trained stimuli.	No follow up (Paula's father wouldn't let her).

Patient Jenny		**Impaired**: - object recognition: better performance with real objects (improved if objects were rotated) than toy objects and animals (lowest scores); - GNT[13]; - Warrington and Taylor's unusual views - Oldfied Wingfield drawings.	non-trained items.	**Generalization:** alternative examples of objects for which she had learned names.			
	6	**Preserved**: - oral description of the visual appearance of objects; - pattern detection; - visual matching; - language (naming errors were visual). **Impaired**: - visual field (slightly) - Recognition of everyday objects (not helped by rotation nor touch) - Identification of photographs and line drawings - WAIS[7] picture completion, - Reading - Face recognition - Memory	No control condition.	**Specific treatment**: naming objects Jenny couldn't identify, copying the objects, visual analysis. **Improvement:** accuracy. **Generalization:** alternative stimuli.	3 times a week for one hour, one session with the psychologist (part of which dedicated to object recognition). Sometimes sessions became 2 if there also was the memory group running (which she attended for 9 weeks, 5 days a week).	Improvement in real object recognition, pictures, and line drawings. Improvement in everyday life perceptual abilities Generalization to other objects or drawings provided these were similar to those whose names had been given.	Duration of follow up not specified: further improvement.

Table 2.2. Studies description.

[1] Boston Naming Test
[2] Benton Faces Test
[3] Birmingham Object Recognition Battery
[4] Visual Object Space Perception
[5] Hooper Visual Organization Test
[6] Wechsler Memory Scale
[7] Wechsler Adult intelligence Scale
[8] Benton Visual Retention Test
[9] Glasgow Outcome Scale
[10] Visual Perception Test for Agnosia

[11] Digit Span
[12] Aachner Aphasie Test
[13] Graded Naming Test

Authors	SCED/ CASP	Deficit	Control Condition	Treatment description/ dependent variables of assessment	Treatment duration	Results of treatment	Follow-up
Polster & Rapcsak, 1996 *N = 1*	SCED: 5	**Preserved**: - verbal and semantic memory - facial gender and emotional expression discrimination - identical faces matching **Impaired**: - famous faces recognition - learning of new faces (WRMT[1]) - matching different views of a face (BFRT[2])	not specified	**Perceptual and mnemonic treatments**: attention to facial features; personality trait judgements; attention to distinctive features of the face; learning of identity-specific semantic information **Improvement** measured through accuracy Generalization to new views of a face was assessed	Not specified	Improvement after personality traits judgements and learning of identity-specific semantic information These results did not generalize to new views of faces	No follow-up
De Haan, Young & Newcombe, 1991 *N = 1*	SCED: 4	**Preserved**: - visual acuity and colour perception **Impaired**: - Performance subtest (WAIS[3]) - Long term memory (WMS[4], ROF[5]) - contrast sensitivity - emotional facial expression recognition, race and gender discrimination - face matching - recognition of familiar faces - poor within class recognition (e.g. flowers) - object recognition	not specified	**Mnemonic treatment**: repeated overt identification of familiar faces and presentation of famous faces in semantic categories **Improvement** was measured through accuracy	Not specified	No improvement for the repeated overt identification of familiar faces Little improvement for 5 of the 6 categories presented	Two months later he had returned to baseline on the categories test
Davies-Thompson et al., 2017 *N = 10*	CASP: 7	**Preserved**: - visual acuity - object recognition and memory **Impaired**: - subjective face recognition - famous faces recognition - at least one of CMFT[6] or WRM[1]	Control task: british television series of their choice	**Perceptual treatment**: face matching task **Improvement**: same six online assessment tests used for initial characterization + neuropsychological and neuroimaging assessment **Generalization**: to untrained view, and to untrained expression was evaluated within the six assessment tests	11 weeks	Improvement in the discrimination of trained faces, some benefits in daily life Generalization to new views and expressions and untrained faces although effects on neuropsychological tests were minimal	Effects persisted for at least three months.

Francis, Riddoch & Humphreys., 2002 *N = 1*	SCED: 5	**Preserved**: - Early perceptual abilities - BFRT[2] - Executive abilities (WCST[8]) **Impaired:** - General Memory (WMS[4]) - Semantic memory - Raven's progressive matrices - Picture naming test BORB[7] - Famous faces identification through vision	Control task: no treatment	**Mnemonic treatment**: face/name learning of familiar people with recall of semantic information and imagery; comparison of semantic treatment with simple name retrieval or no treatment Improvement was measured through accuracy	first treatment: seven two-hour sessions of therapy second treatment: five two hour sessions over a fortnight	face/name learning of familiar people with recall of semantic information and imagery improved the number of recognized faces; Improvement bigger after the semantic condition with respect to the others	1 week after the end of second treatment : effect maintained
Powell et al., 2008 N = 20 brain injured patients N = 1 Acquired Prosopagnosic	SCED: 5	Brain-injured patients: - **Preserved** screening tests of visual perception - **Impaired** in face learning test among other heterogeneous cognitive impairments Acquired prosopagnosic patient	Control group: 12 brain-injured patients comparable to the 20 tested were used as a control group: the same 4 face sets as the experimental group were administered, all in simple exposure task	**Perceptual and mnemonic treatments**: 4 facesets in 4 different conditions: simple exposure, caricaturing, semantic association, part recognition Improvement: measured through accuracy of recognition of faces presented with the above four methods.	4 one-hour sessions over a period of two weeks	Experimental group benefited for the 3 experimental conditions compared to the simple exposure; control group did not show differences between facesets. Pure prosopagnosic patient showed uniquely benefit of part recognition training.	No follow-up
Behrmann et al. 2005	SCED: 6	**Preserved**: - low-level visual processing; - matching of objects from different viewpoints or along a foreshortened axis. **Impaired** performance in: - BNT[9]; - discrimination of exemplars within a single category; - recognition of photographs of a famous individual; - BFRT[2].	None for behavioral training. fMRI: 2 control groups matched for age and education as the baseline.	**Perceptual treatment**: Categorization of unknown stimuli (Greebles). **Improvement:** accuracy and RTs for each session. **Generalization:** accuracy for non-trained greebles, objects, and faces. fMRI acquisition pre- and post-training.	4 months (twice a week).	Worsening of performance with faces. fMRI: face-selective voxels activated more by greebles and less by faces	-

Zihl 2011[1] *N = 2*	SCED: 4	**P1: Preserved:** - DS[10]; - WAIS[3] logical memory and logical reasoning; - AAT[11] without visual items Visual tests were not applied; - sustained and focused attention (behavioral level). - identification of facial expression **Impaired:** - identification of gender and age based on facial information	Visual agnosia persisted in P1 for 15 weeks without significant recovery.	**Perceptual treatment:** analysis of visual features **Improvement**: accuracy. **Generalization:** not assessed for faces.	15 days (30 sessions).	Improvement in accuracy of age, gender and discrimination. Improvement in familiar faces discrimination	-
	SCED: 4	**P2. Preserved**: - DS[10]; - AAT[11] without visual items. - identification of facial expression - visual field, treated - colour discrimination, treated **Impaired:** - WAIS[3] logical memory. - identification of gender and age based on facial information - visual field, treated - colour discrimination, treated	Visual agnosia persisted in P2 for 22 months without significant recovery.	**Perceptual treatment** analysis of visual features **Improvement**: accuracy. **Generalization:** not assessed for faces.	28 days (112 sessions)	Improvement in accuracy of age, gender and discrimination. Lack of improvement with familiar faces even after additional training	-

Table 2.3. Studies description.

[1] Warrington Recognition Memory Test
[2] Benton Facial Recognition Test
[3] Wechesler Adult Intelligence Scale
[4] Wechesler Memory Scale
[5] Rey-Osterrieth Figure
[6] Cambridge Memory Faces Test
[7] Birmingham Object Recognition Battery
[8] Wisconsin Card Sorting Test
[9] Boston Naming Test
[10] Digit Span
[11] Aachner Aphasie Test

	Right hemisphere						Left hemisphere						Etiology
	T	P	O	F	C C	B G	T	P	O	F	C C	B G	
PH (DeHaan et al., 1991)	*		*				*		*				trauma
NE (Francis, Riddoch & Humphreys, 2002)	*												Herpes Simplex
WJ (Powell et al., 2008)	*		*				*		*				stroke
RJ (Polster & Rapcsak, 2008)	*		*										stroke
SM (Behrman et al., 2005)	*		*										stroke
P1 (Zihl, 2011)	*		*				*		*				stroke
P2 (Zihl, 2011)	Closed head trauma with severe chronic hypoxia; presumably frontal or frontotemporal involvement for the trauma and biparietal involvement for hypoxia												

Table 2.4. Localization of the lesion in the seven patients with acquired prosopagnosia.

Authors	SCED/ CASP	Selection criteria	Control Condition	Treatment description/ dependent variables of assessment	Treatment duration	Results of treatment	Follow - up
De Gutis et al., 2007 *N = 1 (MZ)*	SCED: 7	**Impaired**: - famous faces recognition - tests of visual memory for faces but not for words - unfamiliar face matching	Control group: 6 days of the same training in participants with normal face recognition	**Perceptual treatment**: face discrimination task on the basis of spacing among internal components **Improvement**: RT and accuracy in the training task, neuropsychological tests, ERP N170 component and fMRI **Generalization**: faces were changed each day of training	- first training: one week; - 105 days interval; - second training: 1 week; - unsupervised training for 140 more days	Improvement in RT and accuracy of face discrimination; ERP N170 normally selective to faces differently from before-treatment; no changes in face-selective regions measured by fMRIchanges in coherence between OFA and FFA; generalization to neuropsychological tests and everyday life	Effects faded after several weeks. N170 lacked face selectivity after 90 days. However, re-learning was faster.
Corrow et al., 2019 *N = 10*	CASP:8	**Impaired** in at least two of: - difference between memory for faces and words (WRMT[1]) - impaired performance in CMFT[2] - impaired performance in ONFT[3] - impaired score in a famous face recognition test	Control task: british television series of their choice	**Perceptual treatment**: face matching task (same as Davies-Thompson 2017) **Improvement**: same six online assessment tests used for initial characterization + neuropsychological and neuroimaging assessment **Generalization**: to untrained view, and to untrained expression was evaluated within the six assessment tests	11 weeks	Improvement in perceptual sensitivity for faces; generalization to new views and new expressions of the trained faces; several subjects reported improvement in everyday life; marginal generalization to new faces; modest improvements in neuropsychological tests.	Effects persisted for at least three months.
DeGutis, Cohan & Nakayama, 2014 *N = 24*	CASP: 9	**Impaired**: - face recognition (self reported) - impaired performance in CMFT[2]	Control group: waiting period of 15 days	**Perceptual treatment**: face discrimination task on the basis of spacing among internal components (same as De Gutis 2007) **Improvement and generalization**: online assessment test battery involving front-view face discrimination, face discrimination from different view-points, holistic face processing, self reported everyday improvement in face processing	3 week	Improvement in tests of front-view face matching and holistic face processing, generalization to different stimuli and task formats but not to different view-point rotations; improvement in self-reported face recognition	No follow-up

Table 2.5. Developmental prosopagnosia studies.

[1] Warrington Recognition Memory Test
[2] Cambridge Memory Faces Test
[3] Old/New Faces Tes

Figures

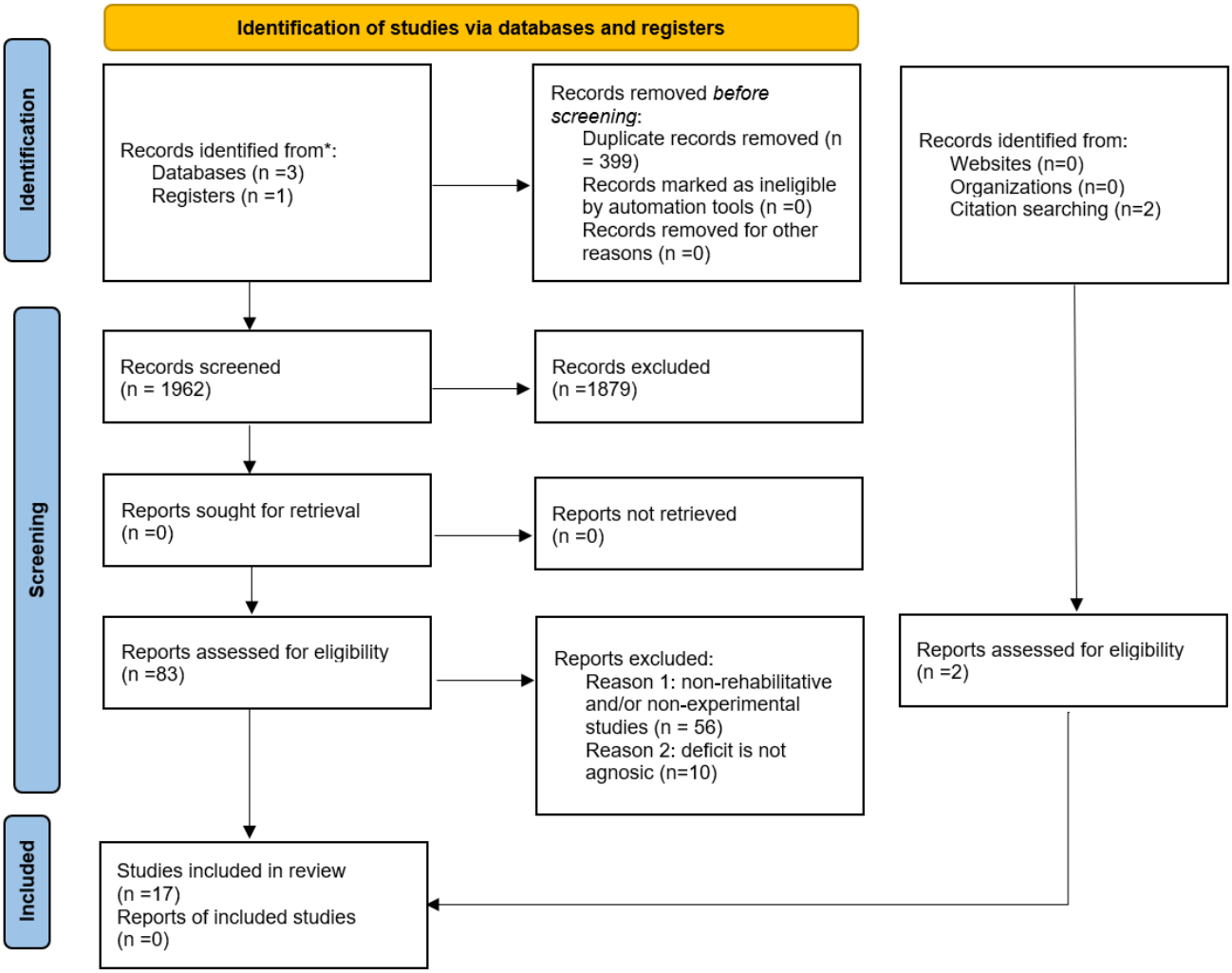

Figure 2.1. PRISMA flow diagram: Overview of the steps of the literature search (Page et al., 2020).

3. Study2[2]: Dynamic Non-Emotional Facial Expressions Help Face Recognition In Poor Recognizers

3.1 Introduction

Traditional models of face recognition postulate that faces are processed through two systems: one for the recognition of the identity and the other for the recognition of their facial expressions. Indeed, in their original model, Bruce and Young (1986) postulated that face identity and expression processing are separate. Successive behavioural findings seem to support this view: in particular, Ellis and colleagues (1990) performed a series of studies on repetition priming whose results show that decisions about facial expressions are not affected by the familiarity of faces, suggesting separate mechanisms for the two processes. In addition, Young and colleagues (1986) showed that in an expression matching task, reaction times were not different for familiar with respect to unfamiliar faces while this was true for identity matching. More recent models of face recognition describe the neural systems involved in face processing (Haxby, Hoffmann & Gobbini, 2000; Haxby & Gobbini, 2011). In particular, they describe a "Core system", specifically selective to faces composed of the Fusiform Face Area (FFA), the Occipital Face Area (OFA), and the Superior Temporal Sulcus (STS), and an "Extended system", involved in processing the familiarity of faces and located more diffusely in the brain. Within the core system, encoding of the so-called invariant aspects of faces is subserved by the FFA. On the other hand, processing of what is defined as changeable aspects of faces, comprising facial expressions, is mediated by the STS. Thus, this model seems to be in line with the distinction between identity and expression processing postulated by Bruce and Young in 1986. Some degree of separation between face identity and expression recognition has further been observed in neuroimaging studies, such as PET (George et al., 1993), fMRI

[2] This study is in preparation with Roberta Daini

(Kesler et al., 2001; Gorno-Tempini et al., 2001; Winston et al., 2004), and ERP (Münte et al., 1998). Furthermore, interest in this issue has also arisen in the field of neuropsychology where individuals with deficits in face recognition (i.e., prosopagnosics) have been studied: a single dissociation between identity and expression recognition deficits was described both in acquired (Mattson, Levin & Grafman, 2000; Young et al., 1993) and congenital prosopagnosics (Bentin et al., 1999; Duchaine, Parker & Nakayama 2003; Jones & Tranel, 2001; Nunn, Postma & Pearson, 2001; Djouab et al., 2020), in the direction of preserved expression with impaired identity recognition. Nevertheless, cases in which facial expression recognition is impaired along with its identity recognition have also been reported both for acquired (Humphreys, Avidan & Behrmann, 2007) and congenital prosopagnosia (Biotti & Cook, 2016). Moreover, cases where a lesion causes a deficit solely limited to facial expressions are debated; this might be due also to the fact that in those clinical cases, it is often not clarified whether identity recognition is preserved (Bate & Bennets, 2015). Moreover, all these studies use emotional facial expressions, causing difficulty in the interpretation of the deficit (Calder & Young, 2005). The uncertainty on the degree of separation of those two anatomical and functional systems might be, at least partially, overcome if we look at face identity and expression processing as integrated systems interacting with each other in face recognition (Hinojsa, Mercado & Carretié, 2015; Calder and Young, 2005). In particular, O'Toole, Roark, and Abdi (2002) suggested that information about facial expressions, carried by the STS (as in the model from Haxby, Hoffmann & Gobbini, 2000), can represent an alternative system for recognition of familiar faces in disturbed viewing conditions (see O'Toole & Roark, 2010 for an updated version).

Moreover, in their revised model on face recognition, Duchaine & Yovel (2015) demonstrate that FFA has a role in both identity and expression processing, while the STS is mainly

involved in the elaboration of expressions. Coherently with this model, a recent study demonstrated that the posterior STS shows stronger activation when seeing facial expressions (even without movement) than when seeing neutral faces and when seeing moving faces (even without any facial expression) with respect to static ones. The same preferences have not been shown in the FFA nor in the OFA (Bernstein et al., 2018). These results seem to be in line with the specificity of the STS for both facial expressions and facial inner motion. Considering the most recent models of face processing, we can hypothesize that, when face recognition appears as difficult, the information conveyed by both FFA and STS about facial expressions and that conveyed by STS about motion helps identity recognition. Two more aspects need to be clarified on this facilitation: the role of emotions and the specific role of motion. As the first issue is concerned, the majority of the studies use emotional facial expressions (e.g.: Righi et al., 2012; D'Argembeau et al., 2007; Humphreys, Avidan & Behrmann, 2007; Duchaine, Parker & Nakayama, 2003) making it difficult to disentangle the unique contribution of facial expressions on identity recognition at a perceptual level from that of emotional content in face processing. If we refer to O'Toole's model, we can say that the STS is involved in the elaboration of the social content of faces as well as the so-called "dynamic facial signatures", specific expressions used in social interactions which can be considered as non-emotional facial expressions (O'Toole Roark and Abdi, 2002). As such, we expect a facilitation of facial expressions on recognition despite their emotional content. However, only a few studies exist on the matter. Existing studies have demonstrated a facilitatory role of dynamic non-emotional expressions for unfamiliar face recognition in normal recognizers (Jesse & Bartoli, 2018); of static non-emotional expressions for unfamiliar face recognition in congenital prosopagnosics (Daini, Comparetti & Ricciardelli, 2014) and of static non-emotional expressions for famous face recognition in poor recognizers (Albonico, Malaspina & Daini, 2015).

As far as motion is concerned, the literature suggests a general "motion advantage" where faces encoded in motion are recognized more easily than those encoded as static (Schiff et al., 1986; Knight and Johnston, 1997; Lander et al., 1999; Lander & Butcher, 2015). Lander and colleagues (2004) described a patient, HJA, showing facilitation of motion in a task where he had to match identities, suggesting that neural mechanisms subserving motion of faces such as the pSTS (as in Pitcher et al., 2011) might be involved in both identity and expression recognition (see also Bate & Bennets, 2015). In accordance with this claim, face identity recognition has been shown to be aided by seeing faces in motion both for unfamiliar (Pike et al., 1997; Knappmeyer et al., 2003; Lander and Bruce, 2003; Pilz et al., 2006; Lander and Davies, 2007; Butcher et al., 2011) and familiar faces (Knight and Johnston, 1997; Lander, Christine & Bruce, 1999; Lander & Bruce, 2000; Lander et al., 2001) and in particular in poor viewing conditions (Knight and Johnston, 1997; Lander et al., 2001; Bennets et al., 2013). A motion advantage has been described, especially for prosopagnosics (Steede et al., 2007; Bennets et al., 2015; Longmore and Tree, 2013; Xiao et al., 2014).

Based on these premises, in Experiment 1 we aimed at investigating the role of dynamic non-emotional facial expressions in face recognition by contrasting the recognition of faces encoded through dynamic non-emotional facial expression, a dynamic rigid head movement, or as neutral static stimuli and relating it with individual face recognition abilities. Moreover, to disentangle the specific contribution of motion on the facilitation of facial expressions on identity recognition, in Experiment 2 we presented the same experimental paradigm to a new sample of participants but with the removal of motion (i.e., using static expressions, static rotated heads, and static neutral faces).

3.2 Experiment 1

3.2.1 Methods

3.2.1.1 Participants

Twenty-five healthy young adults (8 males, mean age=24.24, sd=3.24) participated in the experiment. The numerosity of the sample was calculated a priori and set to 28 participants. It was calculated using the software Gpower (parameters: Power=.80, α=.05, medium effect size =.25) for an analysis of variance with 3*1 design (3 conditions per one group). After collecting data from 25 participants, a preliminary analysis was run. It emerged that analyzing data through a mixed-effects model explained a bigger proportion of variance than using a standard Analysis of Variance (ANOVA). To inspect whether participants' numerosity was already sufficient, a power calculation for mixed-effects models was performed (following Brysbaert & Stevens, 2018), revealing that power was already above .80 and effect size was already above .25.

For this reason, data collection was stopped at this point. Exclusion criteria were low-level visual disturbances or a history of neurological or psychiatric disease, based on self-report. All participants were volunteers and provided their consent. The study was approved by the Ethical Committee of the University of Milano-Bicocca (protocol number RM-2020-361).

3.2.1.2 Stimuli

Stimuli were created by selecting 60 identities (30 males) from the Chicago Face Database (Ma, Correll & Wittenbrink, 2015), matched for attractiveness and trustworthiness. Once identities were selected, an avatar was created for each of them using the program Character Creator 3. 10 avatars (5 males) were morphed to assume 6 non-emotional facial expressions, and 25 captures were taken, each one with a different and growing level of intensity of the

expressions. The 25 captures were presented in rapid succession (every 60 ms) to simulate a movement. A pilot study was conducted to ensure that expressions were non-emotional. To do so, the same identities were morphed to have the six emotional facial expressions (Ekman & Friesen, 1971) and were presented together with the non-emotional facial expressions using the program Inquisit 6 in its online version (https://www.millisecond.com/). 96 participants took part in the pilot study (78 females, mean age=34.36): each participant saw a subset of 30 faces and had to rate their level of arousal on a scale from 1 to 7. Participants rated significantly lower arousal scores for non-emotional facial expressions than for emotional ones ($p<.001$). Only one non-emotional expression elicited comparable arousal to the emotional expressions and was excluded, so the final number of non-emotional expressions was 5.

In addition to the non-emotional expressions, 10 avatars (5 males) were morphed to produce a rigid movement of the head on the three rotational axes. This served as a movement control condition. In fact, stimuli displayed a rigid movement, but the facial expression remained neutral. As happened for the expressions, 25 captures were taken for as many degrees of rotation and presented in rapid succession. To ensure that both expressions and rigid movements were indeed perceived as in motion, a pilot study was run on the same 96 participants who participated on the pilot study on emotions. Participants were asked to rate the level of perceived movement on a scale from 1 to 7. Results confirm that stimuli were perceived as in motion (mean=5.2; sd=2.2). Eventually, 10 identities (5 males) were created as neutral and static. Moreover, the static version of all identities was created, and 30 more static neutral identities (15 males) were created to serve as distractors. All images were on a greyscale to avoid recognition driven by color cues. For an example of the three categories of stimuli, see Figure 3.1.

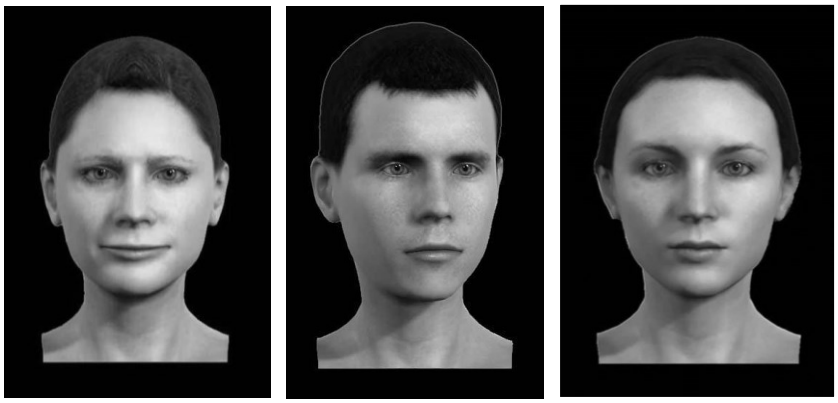

Figure 3.1 Examples of the stimuli. On the left is one of the validated non-emotional expressions, in the centre a rigid head movement around the horizontal axis, and on the right a neutral expression. Non-emotional facial expressions and rigid movement stimuli were dynamic, composed by a succession of 25 images (one each 60ms), while neutral faces were static.

3.2.1.3 Procedure

The experiment was structured as follows. Participants were presented with three blocks in randomized order. Each block belonged to one of the three conditions (i.e., non-emotional facial expression, rigid movement of the head, or neutral) and was divided into two parts. In the first part, a fixation cross was presented for 500 ms at the centre of the screen. It was followed by the face presented in the experimental condition belonging to the block. Faces in the expression and rigid movement conditions were presented as a sequence of 25 frames, one each 60 ms for a total of 1500 ms. Each face was shown two times to reach a total exposure to each face of 3000 ms (see Daini et al., 2014). Faces were presented twice because presenting a single face for 3000 ms created a movement that was too slow and therefore unnatural. To create movement at a more natural speed, faces were shown twice for 1500 ms. For the neutral

static condition, the faces were presented for 1500 ms two times to maintain the same exposure scenario as for the other conditions. After viewing each face twice, participants were asked to make a male/female decision to be sure that participants were processing the faces (Bindermann, Burton & Jenkins, 2005). Once the 10 faces belonging to the experimental condition were judged, the second part began, where the neutral static version of each face was presented for 3000 ms together with the distractors, and participants were asked to say whether they already saw that face or not. For an example of the experimental procedure, see Figure 3.2 or a demo version of the experiment at the following OSF link: https://osf.io/6p24r/.

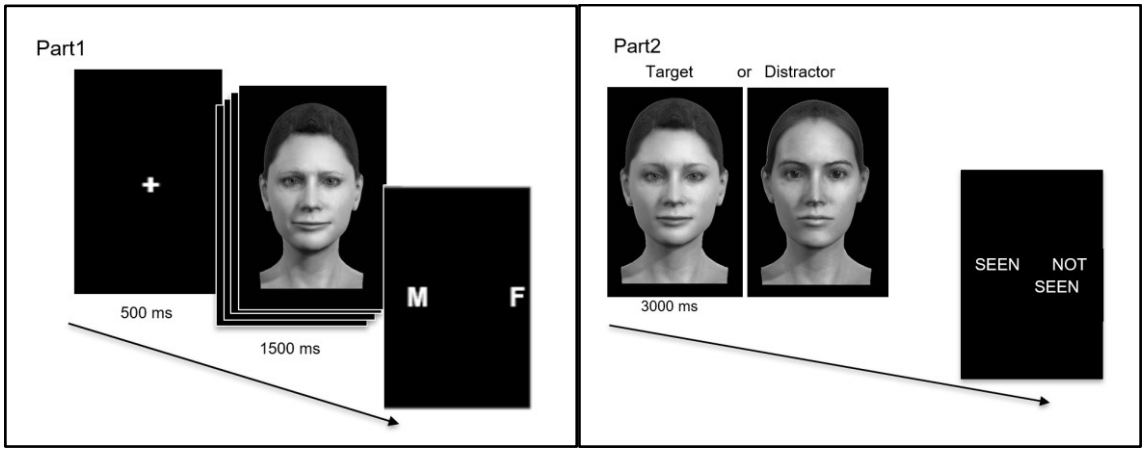

Figure 3.2 An example of the experimental procedure. Each block was divided into two parts: participants saw a fixation cross for 500 ms, followed by the stimulus in the first part. Each stimulus was presented twice for 1500 ms. After the stimulus, the letters M and F appeared on the screen, and the participants had to indicate whether the seen stimulus was a male or a female. After seeing the 10 identities belonging to the running block, participants were administered part 2. In this part, participants saw a neutral static face which could be the neutral static version of the already seen stimulus or a distractor for 3000 ms, and had to decide whether they already saw that face.

In addition to the experimental procedure, three tests were administered to participants. The Cambridge Face Memory Test (CFMT, Duchaine & Nakayama, 2006), a test of famous faces recognition (FFRT, Malaspina et al., 2017), and a test of famous places recognition (FPRT, Cattaneo et al., 2016). The Cambridge Face Memory Test and the famous faces recognition test were performed to assess participants' performance in face recognition and detect potential prosopagnosics (Dalrymple & Palermo, 2016). The famous place recognition test was used as a control for impairments in the recognition of categories other than faces.

Order of administration of the experimental procedure and tests was randomized across participants.

3.2.2 Results

Data were analyzed using the software Jamovi 1.6.15. Scores in the Cambridge Face Memory Test ranged from a minimum of 45 and a maximum of 72 (mean=59.8, sd=6.71). D'prime scores (Heeger & Landi, 1997) were calculated using the software R 4.0.4, package Psycho (Version 0.6.1) and ranged from a minimum of 0 to a maximum of 3.38 (mean=1.48, sd=.69). A linear mixed model was run where the dependent variable was the d-prime score obtained by participants; random factors were the single participants; the fixed factor was the experimental condition (i.e., face shown with non-emotional facial expression, with rigid head movement, or as neutral). Moreover, the Cambridge Face Memory Test, the Famous Faces Recognition Test, and the Famous Places Recognition Test were used as moderators of the fixed effect (as in Passarelli, Masini, Chiorri, Nurcis, Daini & Bracco, 2022). Effect sizes for the main effects and interactions reported below were calculated with the R software 4.0.4, using the package "effectsize".

A significant main effect of condition was found ($F(2,42)=5.43$, $p=.008$, $\eta^2_p=.21$): mean dprime score for, respectively, the expression, movement, and neutral conditions are 1.59 (sd=0.62), 1.51 (sd=0.72) and 1.34 (sd=0.72). However, none of the post-hoc comparisons resulted significant neither with Bonferroni, Holmes nor with Tukey corrections. Main effects and post hoc are not always aligned and Tian and colleagues (2018) suggested interpreting cases as the just described one as false alarms. Most interestingly, a significant interaction was found between condition and Cambridge Face Memory Test ($F(2,42)=4.69$, $p=.01$, $\eta^2_p=.18$). Results concerning the interaction are displayed in Figure 3.3. Simple effects analyses revealed a significant and positive slope for Movement ($t(56.9)=2.179$, $p=.03$) and Neutral ($t(56.9)=3.247$, $p=.002$) conditions while the slope relative to the Expression condition did not reach significance ($t(56.9)= -.443$, $p=.66$). In sum, face recognition abilities seem to predict

recognition of faces encoded as neutral or with rigid movement while faces encoded through non-emotional facial expressions were equally recognized in poor and good recognizers.

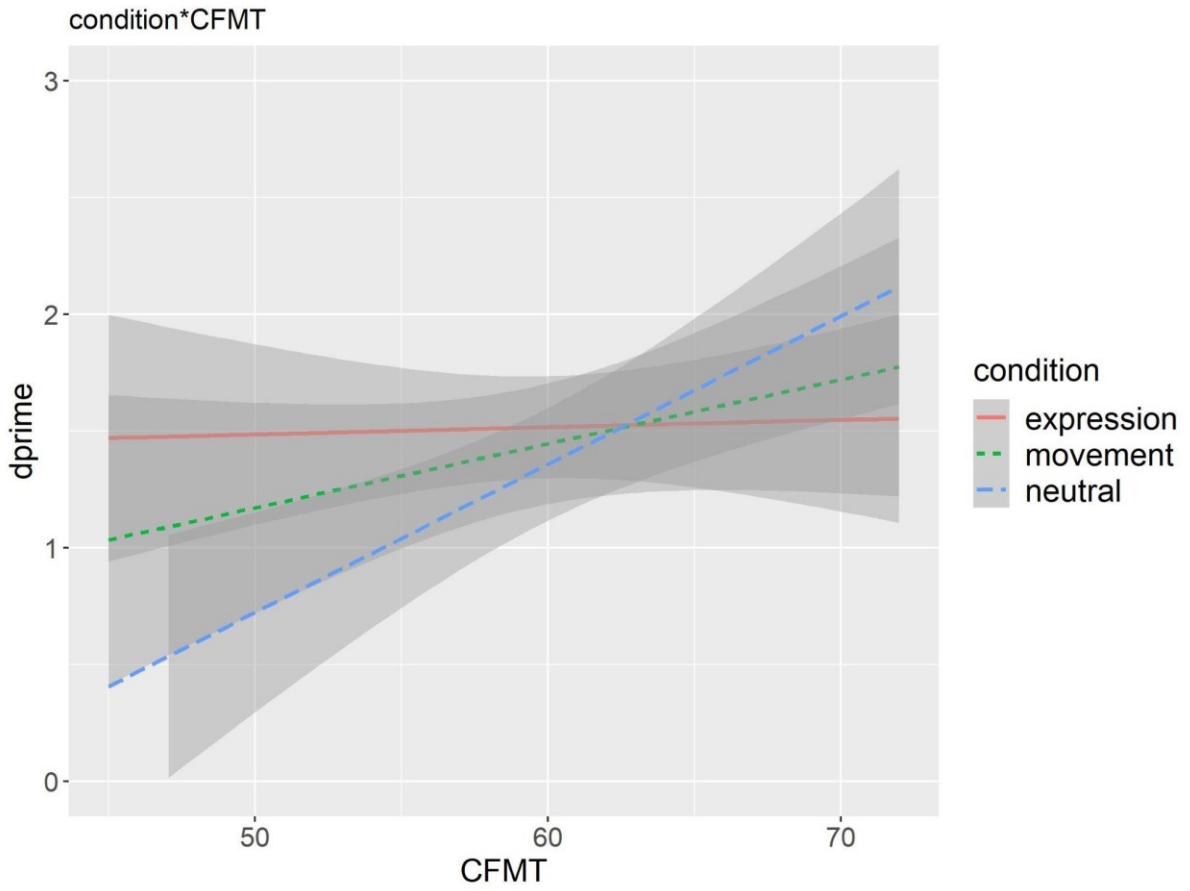

Figure 3.3: In the graph, the relationship between face recognition abilities and the condition of presentation of faces on dprime scores are represented. Faces encoded as neutral or with a rigid head movement are recognized significantly better as face recognition abilities improve. This does not happen for faces encoded through non-emotional facial expressions, which are equally recognized by poor and good recognizers.

To further explore whether there was a significant effect of facilitation of facial expressions in identity recognition for poor recognizers, simple effects were calculated where CFMT was set as a moderator and the condition as the simple effect variable. CFMT scores were therefore divided into those below 1 sd from the mean (low performers: mean CFMT=53), mean ones

(medium performers: mean CFMT=59.8), and the ones scoring 1 sd above the group mean (high performers: mean CFMT=66.5). What emerged is that within low performers faces encoded through non-emotional facial expressions were recognized significantly better than those encoded as neutral (t(2,42)=-3.24; *p*=.002); a trend towards significance was also observed for expressions with respect to rigid movement (t(2,42)=-1.89; *p*=.06). On the contrary, no significant differences were observed for medium and high performers (see Figure 3.4). In sum, it appears that non-emotional facial expressions indeed aid face identity recognition, but only for poor recognizers (i.e. low performers in CFMT).

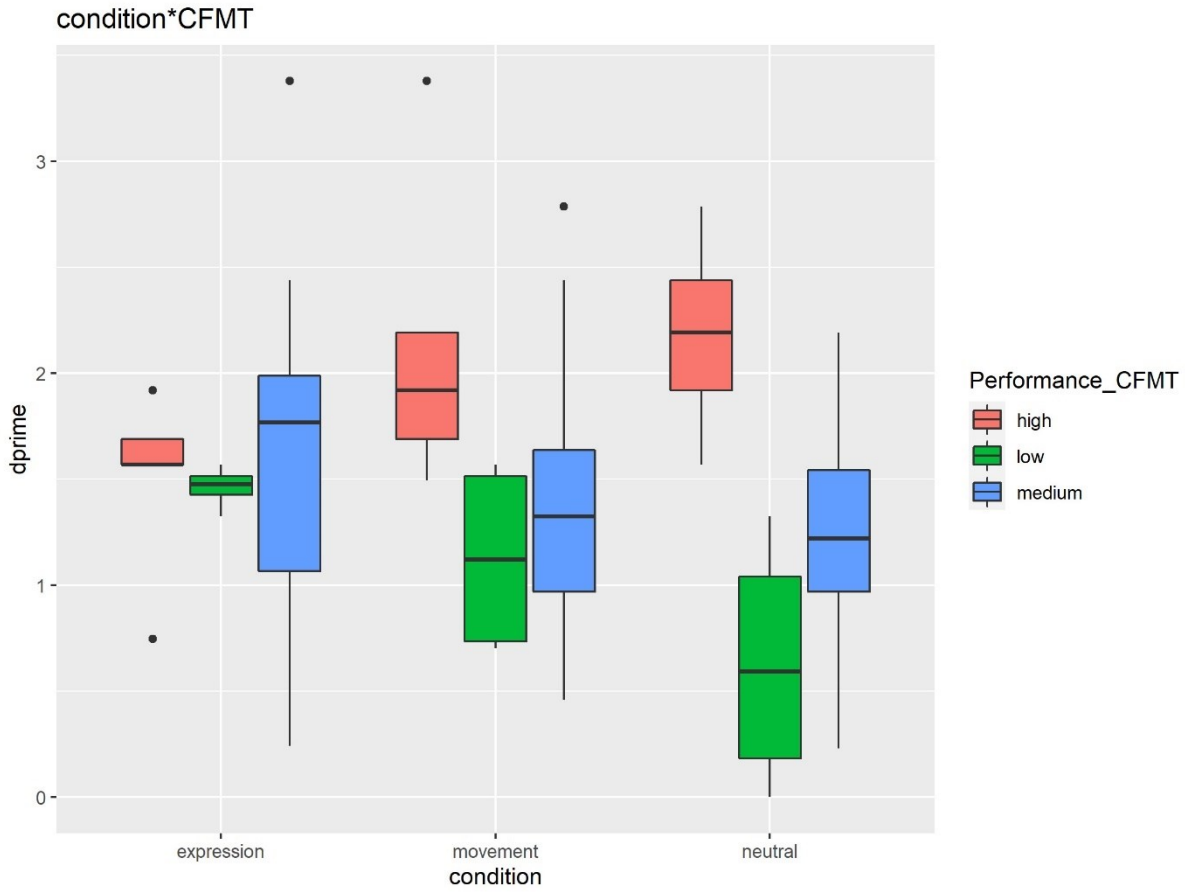

Figure 3.4: The relationship between condition and face recognition abilities on dprime scores is represented. Low performers: participants with face recognition abilities below 1 standard deviation from the mean; mean performers: participants with face recognition

abilities within the mean; high performers:participants with face recognition abilities above 1 standard deviation from the mean.

Interaction between condition and Famous Faces Recognition Test was not significant even though it showed a trend towards significance ($F(2,42)=3.07$, $p=.057$, $\eta^2_p=.13$). Finally, the interaction between condition and Famous Places Recognition Test was not significant, as expected ($F(2,42)=.16$, $p=.77$, $\eta^2_p=.01$). Thus, it appears that poor recognizers recognize faces encoded through facial expressions as accurately as good recognizers while they are significantly worse in recognizing faces encoded through rigid movement or as neutral. To clarify the relative contribution of facial expression and that of movement in these results, we decided to perform a second experiment removing movement.

3.3 Experiment 2

3.3.1 Methods

3.3.1.1 Participants

A new sample of 25 healthy young adults (9 males, mean age=23.88) underwent the second version of the experiment. Exclusion criteria were low-level visual disturbances or a history of neurological or psychiatric disease, based on self-report. All participants were volunteers and provided their consent. The study was approved by the Ethical Committee of the University of Milano-Bicocca (protocol number RM-2020-361).

3.3.1.2 Stimuli

Stimuli and procedure were the same morphed avatars as in Experiment 1, with the difference that non-emotional facial expressions and rigid head movement were not presented as dynamic. For non-emotional facial expressions, stimuli were created as a succession of 5 captures with a different and growing level of intensity of the expression. Each capture was presented for 600 ms for a total of 3000 ms. To make sure that no movement was elicitated by the succession of the 5 images, they were interleaved by a grey mask with the silhouette of a head matched to the face for luminance. The mask stayed on the screen for 1000 ms after each image. 5 captures for as many different rotation angles in progression were selected for the rigid head movement condition and presented for 600 ms each interleaved by the same grey mask with the same method as for non-emotional facial expressions. Images in the neutral static condition were also presented five times interleaved by the grey mask as in the other conditions. For an example of the stimuli see Figure 3.5.

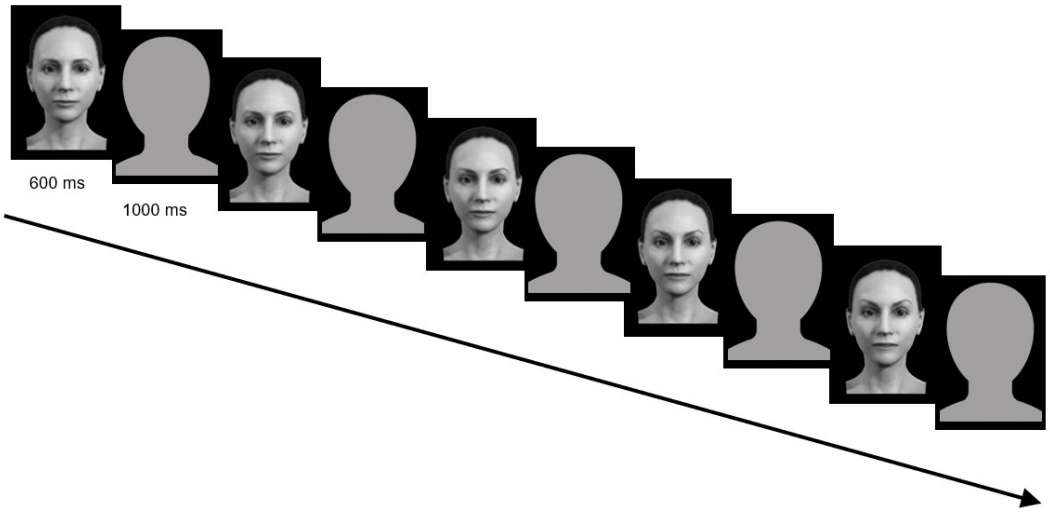

Figure 3.5 Example of stimulus presentation in Experiment 2.

3.3.1.3 Procedure

The structure of the procedure remained the same as in Experiment 1 (see Figure 3.2). The only difference between the two was that in Experiment 2 each identity was shown only once. The total exposure to each face remained the same (3000 ms).

3.3.2 Results

An independent sample t-test was performed to check that data from the faces and monuments recognition tests were comparable across the two groups of participants. The range of scores was not different for the two groups in Cambridge Face Memory Test (t(48)=-.352, *p*=.726), in the Famous Faces Recognition Test (t(48)=-.561; *p*=.577) nor in the Famous Places Recognition Test (t(48)=-.746; *p*=.459).

The same analyses as Experiment 1 were performed for the second experiment. We performed a linear mixed model analysis with d-prime as the dependent variable, participants as a random

factor, and condition as a fixed factor. Moreover, we entered CFMT, FFRT, and FPRT as moderators of the fixed factor. However, in this experiment, the results of experiment 1 were not replicated. No main effect of condition was observed ($F(2,42)=0.51$, $p=.60$, $\eta^2_p=.02$) nor interaction between condition and scores in the Cambridge Face Memory Test ($F(2,42)=.55$, $p=.58$, $\eta^2_p=.02$), between condition and Famous Faces Recognition Test ($F(2,42)=.41$, $p=.67$, $\eta^2_p=.02$) and between condition and Famous Places Recognition Test ($F(2,42)=.61$, $p=.55$, $\eta^2_p=.03$).

We performed a correlation matrix merging scores of participants from both experiments 1 and 2 on the CFMT, FFRT and FPRT to investigate the reciprocal relations. Interestingly, results show that CFMT scores are positively correlated with FFRT (Pearson's r = .329, $p=.02$); FFRT scores are positively correlated with FPRT (Pearson's r = .451, $p=.001$) while almost no correlation was observed between CFMT and FPRT (Pearson's r =-.118, $p=.416$)

3.4 Discussion

Experiment 1 was aimed at verifying whether dynamic, non-emotional facial expressions can help unfamiliar face recognition, with respect to dynamic rigid head movement or static faces. In addition, it aimed at verifying whether individual face recognition abilities affect this relationship. Young participants were administered an experimental procedure where they had to recognize faces presented through non-emotional facial expression, rigid head movement, or as neutral. Moreover, they were administered tests to assess their face recognition abilities. Results confirm that faces encoded through facial expressions are equally recognized in poor and good recognizers, while poor recognizers are significantly better at recognizing faces encoded with facial expression than those encoded with rigid head movement or as neutral. Thus, it seems like there is a facilitatory role of dynamic non-emotional facial expressions in

face identity recognition, but only for poor recognizers. We argue that good recognizers already have the ability to accurately recognize faces based on their features, without needing to compensate via dynamic facial expressions.

Within poor recognizers, no facilitation was observed for rigid movement of the head. Medium and good recognizers did not show any difference in the recognition of faces encoded through the three modalities. Modern accounts of face processing postulate that facial expression and identity processing are separate yet interacting systems (O'Toole et al., 2002; Haxby et al., 2000; Fox et al., 2009; Fitousi & Wenger, 2013; Duchaine & Yovel, 2015). The results of the present experiment seem to favor these accounts: participants poor in face recognition tests lack the structural encoding of faces and are likely to use the route dedicated to the processing of facial expressions as compensation for identity recognition. Aside from the CFMT, also tests of famous faces recognition have been used as indicators of difficulties in face processing (Kress & Daum, 2003). If this kind of test is a good predictor of face recognition abilities, we would observe an interaction of FFRT with the experimental condition in Experiment 1, as was observed for the CFMT. However, we just observed a trend towards significance. We argue that this might be due to the fact that even developmental prosopagnosics show covert recognition of famous faces (Barton, Cherkasova & O'Connor, 2001; Eimer, Goslin & Duchaine, 2012). Therefore, it is possible that the famous faces recognition test used in the present study was less effective in detecting impairment in face recognition ability in poor recognizers compared to the CFMT. Another reason could lay in the fact that the validity of famous faces recognition tests is often limited as it is difficult to define faces that are famous and familiar to all participants (Grüter, 2011). However, we believe that this was not our case as scores in CFMT and FFRT showed a significant and positive correlation, meaning that they are both, to some extent, assessing face recognition. Thus, we conclude that probably, the lack

of significance of the interaction of FFRT with the experimental condition is due to the fact that the task itself is with unfamiliar faces, and as such, the only significant effect of moderation is that of CFMT, which evaluates unfamiliar face recognition.

Experiment 2 was designed to clarify the role of motion in the facilitation observed in Experiment 1. In fact, if non-emotional facial expressions were able to boost facilitation for poor recognizers by themselves, we would observe facilitation also in Experiment 2, where images are presented as static. However, this facilitation was not observed in Experiment 2, suggesting a specific contribution of motion in the results of Experiment 1. In the literature, a general "motion advantage" is described (Butcher & Lander, 2017): motion has been shown to facilitate learning of new faces (Xiao et al., 2014; Butcher, Lander, Fang, & Costen, 2011; Lander & Bruce, 2003; Pike et al., 1997) as well as familiar faces (O'Toole et al., 2002; Johnston & Edmonds, 2009; Roark et al., 2003). In particular, motion in face processing can be rigid and non-rigid/elastic (the two terms are hereby used interchangeably). Rigid motion is defined as a movement of the head where the 3D structure of the face does not vary (e.g., nodding), while non-rigid motion refers to the changes of internal components of a face that we can observe when a person is talking or displaying a facial expression (Knappmeyer, Thornton, & Bülthoff, 2003). Moreover, according to O'Toole (2002), there are two separate yet not mutually exclusive hypotheses on motion advantage: the "representation enhancement hypothesis" suggests that we benefit from elaborating moving faces thanks to perceptual structure-from-motion processes helping in building a more complete three-dimensional structure of the face. The "supplemental information hypothesis", on the other hand, suggests that moving faces are more informative as dynamic identity signatures are processed in addition to the invariant features of faces.

Results of our experiment on low performers seem to be in line with the supplemental information hypothesis. In fact, from the representation enhancement hypothesis perspective, if motion aids in building a three-dimensional representation of the face, we should have observed facilitation in recognition for both the rigid motion and the non-emotional facial expression condition. On the contrary, we only observed a facilitatory effect of non-emotional facial expression. This is more likely due to the fact that when learning new faces, poor recognizers use information conveyed from non-rigid motion in addition to structural information and this is the reason why they show facilitation. This explanation better fits the supplemental information hypothesis.

The fact that we did not find facilitation of non-emotional facial expression presented without movement on recognition is in contrast with a previous study finding this facilitation (Daini et al., 2014). However, Daini and colleagues (2014) presented this experiment to congenital prosopagnosics while our participants were distributed along a continuum of face recognition abilities within the normal limits. As a consequence, the facilitation observed by Daini and colleagues (2014) in participants with more pronounced difficulties in feature processing, could have not emerged in our sample of participants because they show widespread scores in face recognition tests. Moreover, it could be that facilitation conveyed by facial expressions is itself placed along a continuum, going from the highest facilitation with dynamic motion to less facilitation with static expressions.

The present results may also have interesting implications for the rehabilitation of face processing impairments. Few studies have been successful in the rehabilitation of face recognition impairments through training of holistic processing in both congenital and acquired prosopagnosia (Davies-Thompson et al., 2017; De Gutis et al., 2007; De Gutis et al., 2014; Corrow et al., 2019). Non-rigid facial movement (e.g. non-emotional facial expressions) for

familiar faces has been shown to be processed as a whole, while it is less clear what is its processing with respect to unfamiliar faces (Pipers et al., 2021; Xiao et al., 2012; Xiao et al., 2013). If non-rigid facial movement can trigger holistic processing, training with non-emotional facial expressions might improve holistic processing in prosopagnosics. Moreover, it could also represent a compensatory non-impaired route to face recognition (O'Toole et al., 2002).

In conclusion, from the present study emerges that non-emotional facial expressions have a facilitatory role in face recognition, but only for poor recognizers and only when presented in motion. These results are in line with the existence of two separate but interacting systems for face recognition: one for identity and one for facial expressions (O'Toole et al., 2002). As the system used for facial identity is poor, facial expressions become more important as a compensatory mechanism for face recognition. Moreover, a facilitatory role of non-rigid motion for unfamiliar face recognition is observed. These results are crucial both from a theoretical point of view and from a rehabilitative perspective. In fact, non-emotional dynamic facial expressions might represent powerful stimuli for the rehabilitation of face recognition impairments. The following study aims at deepening neural bases implicated in the relation between expressions and identity processing.

4. Study 3[3]: The role of preSMA and STS in face recognition: a Transcranial Magnetic Stimulation (TMS) study

4.1 Introduction

Face recognition is a complex ability mediated by multiple systems widespread throughout the brain. Those systems can be roughly divided into a "core system" composed of the Fusiform Face Area (FFA), the Occipital Face Area (OFA), and the Superior Temporal Sulcus (STS) and an "extended system", more distributed in the brain (Haxby, Hoffmann & Gobbini, 2000; Haxby & Gobbini, 2011). Haxby and colleagues (2011) propose the core system to be specific for face stimuli, while the extended one regards face familiarity. If we consider the system selective for faces, we can further distinguish the relative contribution of certain areas to specific aspects of face recognition. Specifically, O'Toole, Roark, and Abdi (2002) propose that information regarding facial expressions is mainly carried by the STS, while featural information is conveyed by a more ventral system, subserved by the FFA (see O'Toole & Roark, 2010 for an updated version). Moreover, the authors suggest that both systems are implicated in facial identity recognition. This is because the system mediated by the STS and that mediated by the FFA are separate yet interact in the recognition of faces (Hinojosa, Mercado & Carretié, 2015; Calder and Young, 2005; Duchaine & Yovel, 2015). In support of the interaction between the two pathways for face recognition, several studies demonstrated that FFA indeed shows a response to facial expressions (Ganel, Valyear, Goshen-Gottstein & Goodale, 2005; Xu & Biederman, 2010; Jiahui, Yang & Duchaine, 2020). On the other hand, STS has been shown to be more selective for faces seen with an expression, even when static,

[3] The present study is currently under review in *Cognitive, Affective and Behavioural Neuroscience* in collaboration with Carlotta Lega, Angelica De Sandi and Roberta Daini

compared to neutral ones (Bernstein et al., 2018). In addition, several studies show how the dorsal system for face recognition mediated by STS is more selective for dynamic than static faces. As a matter of fact, STS, contrarily to FFA, was shown to be more strongly activated when seeing facial motion than seeing faces as static (Fox, Iaria & Barton, 2009; Pitcher, Dilks, Saxe, Triantafyllou & Kanwisher, 2011). These studies show results that are also in line with the implication of STS in processing biological motion (Grossman et al., 2000). However, whether there is a differential activation of STS for different types of motion remains to be understood. In fact, facial motion can be roughly divided into rigid and elastic motion. Rigid motion is defined as a movement of the head where the 3D structure of the face does not vary (e.g., nodding). In contrast, non-rigid motion refers to the changes in the internal components of a face that we can observe when a person is talking or displaying a facial expression (Knappmeyer, Thornton, & Bülthoff, 2003).

TMS studies bring evidence in favor of a neural network subserving facial expression and motion processing. In particular, TMS was shown to impair facial expression but not identity processing when delivered over OFA and the right Somatosensory Cortex (rSC) (Pitcher, Garrido, Walsh & Duchaine, 2008). In addition, Sliwinska and Pitcher (2018) showed that both right posterior STS (rpSTS) and left posterior STS (lpSTS) are implicated in emotional facial expression recognition. Moreover, rpSTS was shown to be involved in dynamic facial expression processing when stimulated with theta-burst transcranial magnetic stimulation (TBS) and measuring the effects through functional magnetic resonance imaging (fMRI) (Pitcher, Duchaine & Walsh, 2014). Interestingly, the authors found a role of rpSTS in facial motion processing even when the head was in motion but did not display any expression.

The described studies show evidence in favor of an implication of STS in facial expression and facial motion processing. However, it remains to be seen what its interaction is with areas deputed to the analysis of face identity. In the literature, a general "motion advantage" is described. It refers to the effect for which faces encoded as in motion are recognized more easily than those encoded as static (Schiff et al., 1986; Knight and Johnston, 1997; Lander et al., 1999; Lander & Butcher, 2015). Lander and colleagues (2004) described a prosopagnosic patient, HJA, showing facilitation of motion in a task where he had to match identities, suggesting that neural mechanisms subserving motion of faces, such as the pSTS (as in Pitcher et al., 2011), might be involved in both identity and expression recognition (see also Bate & Bennetts, 2015). In accordance with this claim, face identity recognition has been shown to be aided by seeing faces in motion both for unfamiliar (Pike et al., 1997; Knappmeyer et al., 2003; Lander and Bruce, 2003; Pilz et al., 2006; Lander and Davies, 2007; Butcher et al., 2011) and familiar faces (Knight and Johnston, 1997; Lander, Christine & Bruce, 1999; Lander & Bruce, 2000; Lander et al., 2001) and in particular in poor viewing conditions (Knight and Johnston, 1997; Lander et al., 2001; Bennetts et al., 2013). A motion advantage has been described especially for prosopagnosics (Steede et al., 2007; Bennetts et al., 2015; Longmore and Tree, 2013; Xiao et al., 2014). Moreover, a case of a patient with a lesion to the rpSTS was demonstrated to have difficulties in unfamiliar face matching (Sakurai, Hamada, Tsugawa & Sugimoto, 2016). Aside from STS, the pre-Supplementary Motor Area (preSMA) is also implicated in the facial expression processing circuit. This is because preSMA is involved in general motor mimicry (Johnston et al., 2013; Hardwick et al., 2018). Moreover, it emerges from the literature that it shows specificity for faces: consequently, it has been hypothesized that it is part of an extended mirror neuron system that is supposed to participate in facial expressions recognition (Van der Gaag et al., 2007). In line with this hypothesis, in a study, the

authors disrupted the activity of left preSMA by using TMS stimulation, and this caused a decrease in facial happiness recognition. The authors attributed this effect to an embodied cognition account (Rochas et al., 2013). It must be underlined that studies on pre-SMA and faces mostly refer to emotional facial expressions (van der Gaag et al., 2007; Kircher et al., 2013; Rymarczyk et al., 2018). Studies comparing emotional and non-emotional facial expressions find preSMA to be more strongly activated for emotional than nonemotional facial expressions (van der Gaag et al., 2007; Kircher et al., 2013).

So, based on the described literature, not only is FFA implicated in both facial identity and expression processing, but also STS is suggested to have a role in identity recognition. Thus, the first aim of the present study was that of deepening our understanding of the contribution of STS on face identity recognition. To do so, we stimulated it with TMS in healthy participants while viewing faces as static and neutral, in motion with a rigid head movement, or in motion with non-emotional facial expression (i.e., non-rigid movement). Subsequently, a neutral and static face appeared, and participants were asked whether it was the same as the just-seen one. We used rigid and non-rigid (i.e., non-emotional facial expressions) motion to clarify further the role of facial expressions and rigid motion in identity recognition. The reason why we used non-emotional facial expressions is that most studies use emotional facial expressions. This makes it difficult to disentangle the unique contribution of facial expressions on identity recognition at a perceptual level from that of emotional content in face processing. The second aim of the present study was to explore the role of the left preSMA in the face recognition circuit. In fact, the activity of lpreSMA has been attributed to an embodied cognition account where it is suggested to be implicated in the simulation of expressions for their comprehension. Thus, we decided to stimulate this area using TMS when participants looked at the same stimuli described above: faces seen as neutral, rigid head motion, and non-emotional facial

expressions. Participants had to indicate whether the face they were seeing in front of them was the same.

In conclusion, if it is true that the system for facial expressions processing and that for recognizing identities interact and that STS and SMA are implicated in facial expression processing, we expect their stimulation to affect facial identity recognition.

4.2 Methods

4.2.1 Participants

34 healthy right-handed participants participated in the study (25 females, mean age = 23.61, sd = 3.67). Before the study, they were screened for any TMS contraindications following Rossi and colleagues' guidelines (2021). All participants were eligible for TMS use. They also provided written informed consent. The study was in line with the principles of the Declaration of Helsinki and was approved by the ethics committee of the University of Milano-Bicocca.

4.2.2 Stimuli

Stimuli were 60 avatars created through the software Character Creator 3 (https://www.reallusion.com/character-creator/) starting from 60 identities taken from the Chicago Face Database (Ma, Correll & Wittenbrink 2015). The identities were selected to be comparable in age and attractiveness based on the validation scores present in the Chicago Face Database. Once avatars were created, half of them were assigned to the target group and the other half to the distractor group. Stimuli in the target group were morphed to belong to three experimental conditions: neutral, non-emotional facial expression, or rigid head movement. 10 avatars (5 females) belonged to the neutral condition and were not modified. 10 avatars (5 females) were morphed to assume 6 non-emotional facial expressions. To do so, 25 captures were taken, each with a different and growing level of intensity of the expressions. The 25 captures were presented in rapid succession for a total of 250 ms to simulate a movement. A pilot study was conducted to ensure that expressions were non-emotional and details about it can be found in Study 2.

10 avatars (5 females) were morphed to produce a rigid head movement on the three rotational axes. This served as a movement control condition. In fact, the stimuli displayed a rigid movement, but the facial expression remained neutral. As for the expressions, 25 captures were taken for as many degrees of rotation and presented in rapid succession. A pilot study was run to ensure that both expressions and rigid movements were perceived as in motion.

Moreover, the static version of all identities was created, and 30 more static neutral identities (15 females) were created to serve as distractors. Each target stimulus was matched to its distractor both for gender and similarity. All images were on a grayscale to avoid recognition driven by color cues. Moreover, to avoid effects due to perceptual matching and not to mechanisms specific for faces, targets and distractors differed by 10% in their dimensions. Each pair of target and distractor was presented twice: once, the target was 10% bigger than the distractor, and the second time it was the reverse in counterbalanced order. To see an example of the three categories of stimuli, see Figure 4.1.

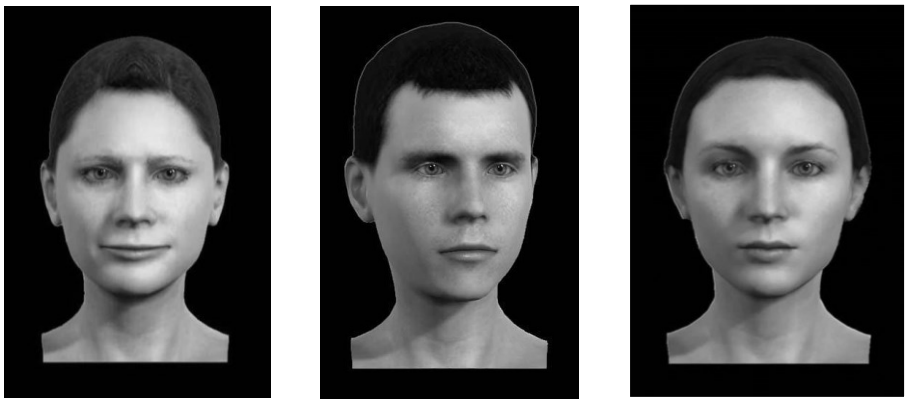

Figure 4.1 Examples of the stimuli. On the left is one of the validated non-emotional expressions, on the center is a rigid head movement around the horizontal axis, and on the right is a neutral expression. Non-emotional facial expressions and rigid movement stimuli were dynamic, composed of a succession of 25 images, while neutral faces were static.

4.2.3 Procedure

The experimental task was structured as follows: participants saw a fixation cross for 500 ms, and then a target face presented for 250 ms. In synchrony with the target face, the TMS stimulation was delivered. Afterward, a neutral version of the target face or a distractor was presented, and participants had to indicate whether the second face presented had the same identity as the target face. To do so, they were instructed to press the key "o" or "p". Response keys were counterbalanced across participants. The same experimental paradigm was presented four times, one for each stimulation condition (see details below) in counterbalanced order. Before starting with the TMS stimulation, participants underwent three practice trials. A schematic representation of the procedure is represented in figure 4.2. Aside from the experimental procedure, participants were also administered two tests of face recognition: the Cambridge Face Memory Test (Duchaine & Nakayama, 2006) and the Cambridge Face Perception Test (Duchaine, Germine & Nakayama, 2007). These tests were always administered before the experimental task.

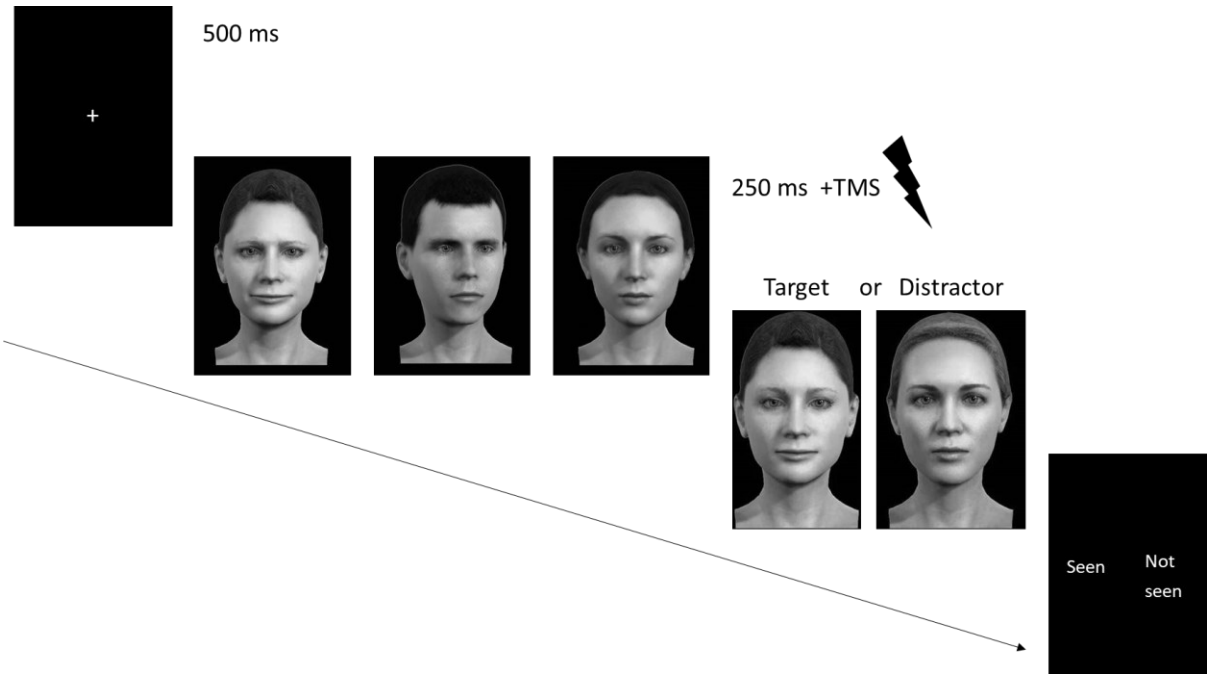

Figure 4.2 Representation of the experimental procedure. Each trial was composed of a fixation cross lasting 500 ms after which participants saw a face in one of the three conditions (expression, movement, or neutral) for 250 ms. During the face presentation, TMS was delivered over one of the stimulation sites. Right afterward, participants saw another face, which was always static and neutral, and had to answer whether it was the same as the just seen one.

4.2.4 Transcranial Magnetic Stimulation

TMS stimulation was delivered using a Magstim Rapid^2 stimulator connected to a 70 mm butterfly coil. The intensity of the stimulation was calculated for each participant according to the individual resting Motor Threshold (rMT). The resting motor threshold (rMT) was determined using a software-based "adaptive method" developed by Awiszus (2003) (Motor Threshold Assessment Tool, version 2.0: http://www.clinicalresearcher.org/ software.htm). Any visible muscle twitch was entered in the software as a "valid response." During the experiment, TMS was delivered at 100% of the individual rMT (mean intensity 51.02% (sd = 4.30) of the maximum stimulator output). The rMT was defined as the minimum TMS stimulation intensity needed to elicit evoked observed movements in at least 50% of trials (Rossini et al., 1999) and was calculated as follows. Three TMS pulses were delivered at 10 Hz, starting at the onset of the target stimuli (see Figure 4.2). These stimulation parameters have been previously demonstrated effective in modulating the underlying cortical activity (Cohen Kadosh et al., 2010; Saad and Silvanto, 2013), also considering specifically face processing studies (Pitcher, 2014). Targeted sites were rpSTS, lpreSMA, and two sham stimulation conditions. The first sham stimulation condition was an active stimulation over the vertex. However, considering that the vertex is anatomically close to the lpreSMA, we

introduced a second sham stimulation where we positioned the coil over the vertex but flipped it. In this case, no active stimulation was delivered on the scalp.

The two sites of active TMS stimulation were localized using stereotaxic navigation (Softaxic, EMS). This was done on estimated individual MRIs acquired through a 3D warping process by fitting an MRI template obtained in high resolution to the participant's scalp model and craniometric points.

Neuro-navigation used anatomical Talairach coordinates (Talairach and Tournoux, 1988) obtained by converting the MNI coordinates of the sites of interest. Coordinates for rpSTS were obtained from Sliwinska & Pitcher (2018), while those for lpreSMA were acquired from Rochas and colleagues (2013). The coordinates were x=53, y=-38, z=12 for rpSTS, and x=-6, y=15, z=58 for lpreSMA.

4.2.5 Statistical analyses

Data were analyzed using a linear mixed effects model (Baayen, Davidson & Bates, 2008). F-test was used to test statistical significance with the Satterthwaite approximation of degrees of freedom. All the analyses were conducted using R (R Development Core Team, 2016) and the *nlme* package (Pinheiro et al., 2017). Post hoc tests were conducted using the R-package *emmeans* (Russel, 2019) and applying the Bonferroni–Holm correction for multiple comparisons.

The dprime index (Macmillan & Creelman, 1990) was calculated through the package *psycho* (Makowski, 2018) for each stimulation area, respectively, for each condition and each participant. To check that our two sham conditions did not differ, we performed an independent sample t-test on dprime scores of the vertex active condition and the vertex sham one (T = 0.608, df = 201.86, p = 0.544). Given that the two conditions did not shed different

dprime scores, we decided to average them together to have a unique sham condition in the analyses.

4.3 Results

The TMS stimulation site (STS, preSMA, Sham), the condition of presentation of faces (facial expression, rigid movement, neutral), and their interaction were entered as fixed factors in a linear mixed model predicting dprime values. CFMT and CFPT scores were entered as covariates. Moreover, single participants and face identities were entered as random intercepts. This analysis revealed a significant main effect of TMS stimulation ($F_{(2,16312)}$=64.362, p<.0001) with higher dprime scores for preSMA stimulation condition (estimated marginal mean = 2.55) compared to Sham (estimated marginal mean = 2.49) and STS (estimated marginal mean = 2.47) stimulation. Post hoc test revealed a significant difference between preSMA and Sham stimulation (t=7.429, p<.0001), between STS and Sham stimulation (t=3.712, p=.0006), and between preSMA and STS stimulation (t=11.141, p<.0001). From this analysis also emerged a main effect of condition ($F_{(2,16312)}$=1861.776, p<.0001) with higher dprime scores for the neutral condition (estimated marginal mean = 2.65) with respect to expressions (estimated marginal mean = 2.62) and movement (estimated marginal mean = 2.24). Post hoc test revealed a significant difference between expression and movement condition (t=50.716, p<.0001), expression and neutral condition (t=-4.029, p=.0002), and movement and neutral condition (t=-54.745, p<.0001). From the analysis also emerged a significant interaction between TMS stimulation and the condition of presentation of faces ($F_{(4,16312)}$=163.754, p<.0001). A graphical representation of the interaction is represented in Figure 4.3. Post hoc test revealed that Sham stimulation significantly differs in the neutral condition from preSMA (t=12.037, p<.0001) and STS (t=3.856, p=.0042) stimulations. Moreover, it differs in the movement (t=15.599, p<.0001) and in the expression (t=-13.025, p<.0001) conditions from STS. STS and preSMA stimulations differ in the expression condition (t=-11.356, p<.0001), in the movement condition (t=14.760, p<.0001), and in the neutral condition (t=15.893, p<.0001).

If we look at the post hoc results regarding each single stimulation area, results reveal that within Sham stimulation, there is a significant difference between the expression and movement conditions (t=18.903, *p*<.0001), between the expression and neutral conditions (t=-4.497, *p*=.0002) and between movement and neutral conditions (t=-23.401, *p*<.0001). Within the STS stimulation, there is a significant difference between the expression and neutral condition (t=12.384, *p*<.0001), between the expression and movement condition (t=47.527, *p*<.0001), and movement and neutral conditions (t=-19.545, *p*<.0001). Eventually, within the preSMA stimulation, there is a significant difference between the movement and neutral conditions (t=-36.276, *p*<.0001), between movement and expression conditions (t=21.412, *p*<.0001), and neutral and expression conditions (t=-14.865, *p*<.0001).

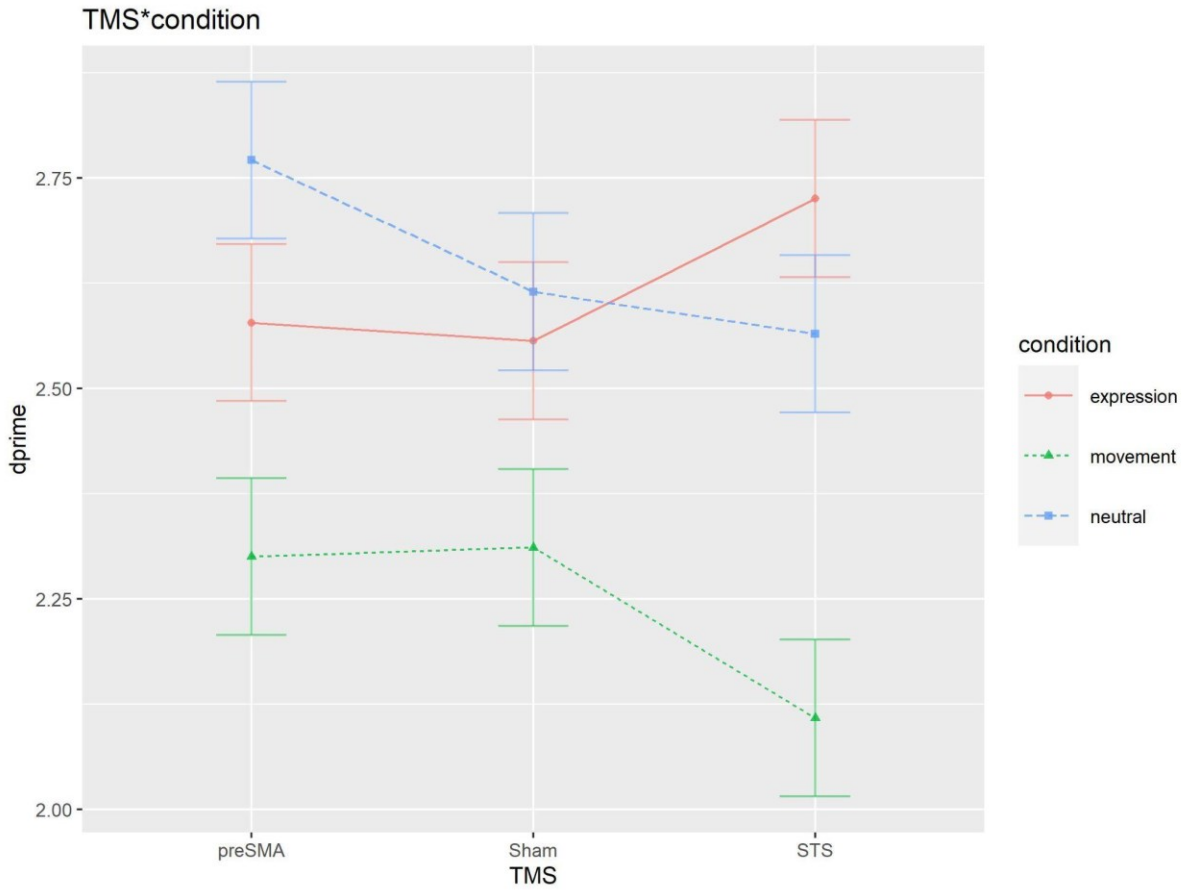

Figure 4.3 Graphical representation of the interaction between TMS stimulation and conditions of presentation of faces.

The covariate CFMT had a significant effect on results ($F_{(1,31)}$=7.478, p=.0102), while the effect of CFPT was not significant ($F_{(1,31)}$=0.5779, p=.4529).

As the CFMT covariate was significant, we conducted an additional analysis. For each stimulation area, we fitted a linear mixed model where the interaction between the condition of presentation of stimuli and CFMT was entered as a fixed factor, and single participants and face identities were entered as random intercepts.

When considering only the Sham stimulation data, the model revealed a significant interaction between condition and CFMT ($F_{(2,4076)}$= 15.279, p<.0001). Post hoc test revealed that dprime significantly grows when CFMT scores grow, but this happens only for the neutral (CFMT $trend_{(32)}$ = 0.0178, lower CI =0.004, upper CI = 0.032) and movement (CFMT $trend_{(32)}$ = 0.0173, lower CI =0.003, upper CI = 0.032) conditions. When considering preSMA stimulation data, a significant interaction between condition and CFMT was also found ($F_{(2,4076)}$= 58.251, p<.0001). Post hoc test revealed that dprime significantly grows when CFMT grows for neutral (CFMT $trend_{(32)}$ = 0.0286, lower CI =0.0089, upper CI = 0.0482), movement (CFMT $trend_{(32)}$ = 0.0332, lower CI =0.0135, upper CI = 0.0528), and expression (CFMT $trend_{(32)}$ = 0.0201, lower CI =0.00053, upper CI = 0.0398) conditions. Eventually, when looking at the STS stimulation, there is again a significant interaction between the condition and CFMT ($F_{(2,4076)}$= 6.87, p=001). However, none of the post hoc tests resulted as significant. A graphical representation of this analysis is represented in Figure 4.4.

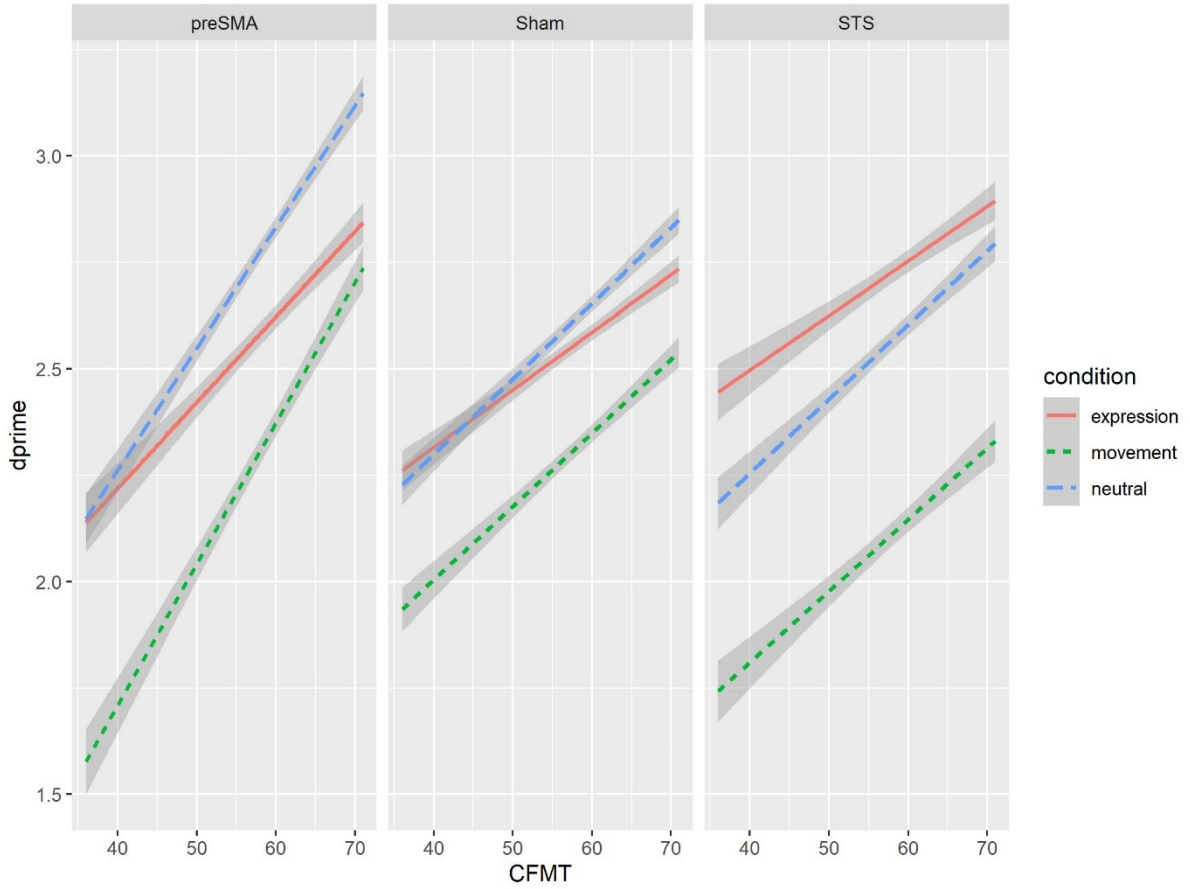

Figure 4.4. Interaction between CFMT scores and condition of presentation of faces on dprime scores during the three stimulation conditions.

4.4 Discussion

Face recognition is a complex ability mediated by multiple systems widespread throughout the brain (Haxby & Gobbini, 2011). The core system for face processing can be divided into a so-called ventral system which processes structural aspects of faces and is mainly subserved by the Fusiform Face Area, and a dorsal system responsible for facial expression and facial movement processing, mediated by the Superior Temporal Sulcus (Haxby et al., 2000). Moreover, when talking about facial expressions processing, other systems, such as the pre-Supplementary Motor Area, are implicated in an embodied cognition process of simulation of expressions for their understanding (van der Gaag et al., 2007). Systems for facial expression and facial identity processing interact with one another; however, it is not clear yet to what extent (O'Toole et al., 2002).

The present study was aimed at investigating the role of STS and preSMA in recognizing faces in different static/dynamic conditions. To do so, 34 participants underwent repetitive TMS stimulation over rpSTS and lpreSMA and two sham conditions while looking at faces encoded through dynamic non-emotional facial expressions, rigid head movements, or as neutral. Subsequently, they had to recognize the same faces just seen in their neutral and static versions among distractors. Results revealed an effect of the TMS stimulation site on dprime scores in both stimulation sites (i.e., larger dprime scores for preSMA stimulation compared to Sham and STS). Moreover, there was also an effect of the condition of the presentation of stimuli. In this case, dprime scores were higher when faces were encoded as neutral than when they were encoded with a facial expression and through a rigid head movement. Even though we varied the dimension of faces to prevent a perceptual matching of target and response faces when presented as neutral, it is possible that this condition resulted as easier because the stimulus and the response were presented in the same form (i.e., static and neutral). On the other hand, the very presence of the movement could constitute a burden of elaboration on a characteristic

that in this particular task is not helpful for those who already have a ceiling performance. Moreover, faces were presented very quickly (250 ms), and it might be that faces presented through rigid head movement were harder to recognize because there was not enough time to process them when moving on the horizontal and vertical axes.

Our most interesting results come from the interaction between TMS stimulation and the condition of the presentation of stimuli. Sham condition revealed no differences between the static condition and the facial expression one and a worse performance with the head movement condition. STS stimulation induced a further decrease in the head movement condition and a significant increase in face recognition within the facial expression condition. Instead, preSMA stimulation improved the static condition. The first result indicates a direct role of STS in encoding head and facial movement, extending previous literature indicating the involvement of STS in motion perception and biological motion processing. The head movement result is likely due to the fact that STS, when stimulated, prevents the processing of moving faces. Contrary to what happens with rigid head movement, when looking at scores relative to the expression condition, STS stimulation caused a significant improvement in scores. This was not in line with our hypothesis as, being the TMS stimulation inhibitory and being STS involved in facial expression processing (Haxby et al., 2000), we would have expected this score to decrease, as happened with rigid head movement. This result can have different interpretations.

A first interpretation concerns methodological reasons: the effect of repetitive TMS on STS is not necessarily inhibitory but state-dependent. Here we have assumed that repetitive TMS has an inhibitory effect, however, there are cases where a repetitive TMS stimulation causes facilitation in emotional facial expression processing (Candidi, Stienen, Aglioti & de Gelder, 2011). The authors of that study argue that TMS pulses act on state-dependent cells; this, in

turn, might have excitatory or inhibitory effects based on the baseline activation of that specific subset of cells. This baseline activation might differ depending on the type of facial movement presented (i.e., rigid head movement or facial expressions). Although this might be the case of our results, the reported explanation is merely speculative, and more studies on the topic might shed light on it. Specifically, studies where there is a co-registration between TMS and EEG would be particularly helpful in disentangling this matter. This because it might be that facial expressions triggers specific brain activation (Dzhelyova, Jacques & Rossion, 2017) and this might explain why STS stimulation differently affects rigid head movement when compared with facial expressions.

A second possible explanation of the present result might have to do with the nature of the present study. Indeed, the main difference between the present and previous studies is that the present study is an identity recognition task. Conversely, the existing literature we referred to presents facial expression recognition tasks. A study by Skiba and Vuilleumier (2020) highlighted how STS is implicated in the analytic processing of local visual information composing a facial expression. In the present study, however, we asked participants to recognize facial identity, which is mediated by holistic/configural processes (Richler & Gauthier, 2014) and not by analytic/featural processes. Thus, it might be that being STS implicated in analytic/featural processing, its inhibition through TMS stimulation caused a decrease in featural processing in favor of holistic processing. This might have, in turn, caused facilitation in identity processing due to the fact that this process relies on holistic processing. Although plausible, also the just presented explanation remains speculative.

A final explanation of the result relative to STS regards the temporal dynamics of face recognition. There are studies analyzing the activity of different areas in the face processing circuit. Such studies find that different areas are implicated in processing different facial

aspects at different moments (Pitcher, 2014). Particularly, specific temporal dynamics in processing facial expressions have been reported (Li, Richardson & Ghuman, 2019). Based on these studies, we might assume that stimulating STS in the phase of encoding does not affect the inner facial movements processing. It might be that to affect this process, stimulation in a different phase is needed. This might be coherent with the results of Sliwinska & Pitcher, (2018), who stimulated STS in the phase of recognition of the expression rather than during its encoding and found a decrease in performance. In addition, it must be noted that previous TMS studies on the role of STS in facial expression processing use tasks of expression recognition (Sliwinska & Pitcher, 2018), while in the present study, we wanted to study identity recognition when mediated by an expression. Thus, it might be that the temporal dynamics of the activation of STS are different when at the service of the identity recognition system with respect to the expression recognition one, which might drive our result.

When looking at scores relative to the preSMA stimulation, the static condition is improved. We think that an explanation related to the temporal dynamics of the activation of preSMA in the face processing circuit might be more plausible. Further studies are needed to clarify the temporal dynamics aspect. It might be interesting to use TMS in combination with EEG to have information on the temporal activation of areas during the task.

Eventually, as the covariate CFMT resulted as significant, we decided to deepen our understanding of the relationship between individual face recognition abilities and the recognition of faces encoded in the three presentation conditions within each stimulation area. In the Sham condition, as the CFMT score increases (indicating a better face recognition ability), dprime increases, indicating that a better face recognition ability is associated with a better performance in the experimental face recognition task. However, this happens for neutral and movement conditions but not expression conditions. This means that poor recognizers

show dprime scores in the expression condition comparable to good recognizers. This result can be interpreted with the facilitation of facial expressions in identity recognition for poor recognizers. In support of this interpretation, the literature reports that facial expressions facilitate identity recognition in congenital prosopagnosics both for unfamiliar (Daini, Comparetti & Ricciardelli, 2014) and familiar (Albonico, Malaspina & Daini, 2015) faces.
This relation changes when preSMA is stimulated: in this case, scores relative to all three conditions increase when the CFMT score increases. Thus, if we follow the interpretation we gave for this relation in the Sham stimulation, in this case, the facilitation of facial expressions for identity recognition in poor recognizers is canceled when preSMA is stimulated. Again, this result is in line with the literature on the role of preSMA in an embodied simulation account for facial expression processing (van der Gaag et al., 2007). If preSMA is modulated through TMS, it fails to perform its simulation role for facial expression processing, thus causing a stop in its facilitation at the service of identity recognition. When STS is stimulated, the interaction results as significant; however, none of the post hoc tests resulted as significant.

To sum up, the present results indicate that STS and preSMA have a role in facial identity recognition. STS, when stimulated, caused a decrease in the recognition of faces encoded through a rigid head movement. This result is in line with the literature on its role in biological motion processing. STS stimulation also caused an increase in recognition of faces encoded through facial expressions with respect to the Sham stimulation. This, even if it was not in line with the expectations of a decrease in expression processing, paralleling that of rigid movement processing, suggests a role of STS not only in facial expression processing, but also in face recognition. PreSMA, when stimulated, caused an increase in recognition of faces encoded as neutral. Moreover, it eliminated the facilitatory role of facial expressions in identity recognition

in poor recognizers observed in the Sham stimulation condition. The result of an implication of STS in the recognition of the identity of faces encoded through a facial expression represents a novel contribution to our understanding of the circuit mediating facial identity and expression processing. Indeed, results reported so far only investigated the contribution of STS to expression processing neglecting its role in identity recognition. Also, the result of preSMA implication in the face processing circuit represent a step forward in extending the existing models on face processing.

5. Study4[4]: The role of Parkinson's Disease in recognizing facial expressions and identities

5.1 Introduction

Parkinson's disease is usually associated with a range of symptoms, which can be motor (Rodriguez-Oroz et al., 2009; Ruiz, Catalan & Carril, 2011) and non-motor (Jellinger, 2015). Non-motor symptoms are often overlooked; however, it has been reported that at least one non-motor symptom is present in 100% of patients (Pfeiffer, 2016), impacting patient's quality of life (Tolosa, Santamaria, Gaig & Compta, 2010). Among non-motor symptoms are also affective and cognitive symptoms (Poletti, De Rosa & Bonuccelli, 2012), often associated with problems in social cognition (Palmeri et al., 2017).

Within the social cognition domain, an ability that has often been studied is recognizing emotional facial expressions (e.g., Borg et al., 2012; Heller et al., 2018). Deficits have been observed in the identification, discrimination, and recognition of emotional facial expressions for patients with Parkinson's disease when compared to healthy controls (Argaud, Vérin, Sauleau & Grandjean, 2018; Gray & Tickle-Degnen, 2010). However, several inconsistencies have been reported, such as in the selectivity for the recognition of specific emotions. Authors who investigated this found that there were emotions that could be specifically impaired; however, different authors found heterogeneous results. In particular, there were authors found specific difficulty in recognizing anger and fear (Martins, Muresan, Justo & Simao, 2008), anger and disgust (Sprengelmeyer et al., 2003), happiness and disgust (Wagenbreth, Wattenberg, Heinze & Zaehle, 2016), or disgust alone (Suzuki, Hoshino, Shigemasu &

[4] The present study is in preparation with Elisa Urso, Aurora Colombo, Cecilia Perin, Matilde Menghini and Roberta Daini

Kawamura, 2006). Other authors, on the other hand, found general impairment in emotion recognition in Parkinson's disease (Alonso-Recio, Martín-Plasencia, Loeches-Alonso, & Serrano-Rodríguez, 2014; Enrici et al., 2015; Lin, Tien, Huang, Tsai & Hsu, 2016).

Several interpretations have been given for this deficit in PD patients. It has been linked to neural substrates of emotion processing, which may be impaired due to the disease. In fact, the emotions more consistently impaired in the mentioned studies are the so-called negative emotions (Argaud et al., 2018), and the authors often report these emotions to be specifically impaired due to their neural substrates. For example, disgust seems to be subserved by insula and striatum, strictly connected with basal ganglia, the system impaired in PD (Wagenbreth et al., 2016; Sprengelmeyer et al., 2003). Coherently, when anger and fear are selectively impaired, Martins and colleagues (2008) conclude that the loss of dopamine in the brain due to the disease causes a lack of recognition of anger and fear.

Regarding the other emotions that have been selectively found impaired, authors interpret their results only speculatively (Wagenbreth et al., 2016). Finally, it must be noted that not all studies investigate the recognition of all six basic emotions; some only investigate one or two specific emotions (e.g., Marneweck & Hammond, 2014). Thus, results about selective impairments must be interpreted cautiously.

Another possible interpretation of the emotion recognition deficit in PD is that it might be linked to impairments in emotion expressivity (Prenger & MacDonald, 2018). This interpretation relies on the embodied simulation theory, according to which emotion recognition is enhanced by internal simulations of the observed expressions. These simulations occur when viewing an emotional facial expression which in turn activates the corresponding emotion in the viewer (Argaud et al., 2018). Deficits in emotional expressivity have been attributed to different causes. On the one hand, symptoms commonly observed in PD patients

are amimia, hypomimia, or facial bradykinesia (Bowers et al., 2006; Bologna, Fabbrini, Marsili, Defazio, Thompson & Berardelli, 2013; Aarsland, Ballard, McKeith, Perry & Larsen, 2001) which in turn cause reduced facial expressiveness. On the other hand, it might be that reduced facial expressiveness is caused by a malfunction of the mirror neuron system in PD patients compared to controls (Pohl et al., 2017). Whatever the cause, it seems that blocking facial mimicry impacts emotion recognition. As a matter of fact, several studies find decreased mimicry in PD patients compared to controls (Livingstone, Vezer, McGarry, Lang & Russo, 2016; Kang, Derva, Kwon & Wallraven, 2019). This, in turn, has been linked to impairment in emotion recognition for this clinical population (Prenger & MacDonald, 2018; Marneweck, Palermo & Hammond, 2014; Jacobs, Shuren, Bowers & Heilman, 1995; Ricciardi et al., 2017; Argaud et al., 2016). It must be noted that also in this case, there is heterogeneity in the results as there are studies finding a reduction in the ability to mimic only certain emotions (Oberman, Winkielman & Ramachandran, 2007; Chuang et al., 2022).

In conclusion, the topic of emotional facial expression recognition in Parkinson's Disease has been studied extensively: however, there is still controversy about the interpretations. A way to shed light on it is by investigating expression recognition using non-emotional expressions. In this way, we could disentangle the contribution of a deficit in general emotional processing in PD (Péron, Dondaine, Le Jeune, Grandjean & Vérin, 2012) from that of an expression simulation deficit. To the best of our knowledge, few studies exist on non-emotional facial expressions. One investigated facial expression processing, also including non-emotional expressions (Derya, Kang, Kwon & Wallraven, 2019), and concluded that there were no differences in expression recognition in PD patients compared with controls. This result might be a clue towards a deficit in PD patients specifically linked to emotion processing rather than in the simulation of expressions for their understanding. On the other hand, it could be linked

to expression simulation, but to a lesser extent than for emotions. Conversely, another study investigated ERP in PD patients in response to both emotional and non-emotional expressions. The authors divided PD patients into LPD (i.e., patients with disease insurgence on the right hemisphere causing initial symptoms on the left side) and RPD (i.e., patients with disease insurgence on the left hemisphere causing initial symptoms on the right side). They found that LPD but not RPD were impaired in dynamic expression processing compared to controls (Garrido-Vásquez, Pell, Paulmann, Sehm & Kotz, 2016), hinting at a predominant role of expression simulation.

Moreover, while many studies report difficulties in recognizing emotional facial expressions in PD, only a few display data on identity processing. Some authors find difficulties specific to unfamiliar identity recognition measured by both the Benton Face Recognition test (Benton, Sivan, deS, Varney & Spreen, 1983; Sprengelmeyer et al., 2003) and the Cambridge Face Memory Test (Duchaine & Nakayama, 2006; Marneweck et al., 2014). Others find difficulties in configural processing and link them, at least partially, to emotion recognition (Narme, Bonnet, Dubois & Chaby, 2011; Cousins, Pettigrew, Ferrie & Hanley, 2021). However, it is unclear whether there are deficits specific to the disease or if the results obtained are also a consequence of aging and developing Parkinson's Disease Dementia (Ho et al., 2020).

Thus, the aim of Experiment 1 was to investigate whether there were differences in recognition of non-emotional facial expressions and identities in PD patients compared to healthy controls. If the observed deficit in PD for the recognition of emotional expressions is mainly caused by the difficulty with emotions, we would expect them not to show difficulties in recognition of non-emotional facial expressions. On the other hand, if the deficit in emotional expression recognition is mainly caused by the mimicry component, we would expect our patients to show the deficit.

Moreover, it must be noted that models of face recognition postulate that we recognize faces using two separate yet interacting systems. In particular, a ventral system implicated in the recognition of structural aspects of faces subserved mainly by the Fusiform Face Area and Occipital Face Area, and a dorsal system, involved in processing facial expressions and subserved mainly by the Superior Temporal Sulcus (O'Toole, Roark & Abdi, 2002; Duchaine & Yovel, 2015; Haxby, Hoffmann & Gobbini, 2000). Those systems interact with each other in the recognition of facial identity. Thus, if the deficit in recognizing emotional expressions is caused by a problem specific for expressions, we would expect those patients also to show difficulties in recognizing face identity when faces are presented with an expression. That is why we designed Experiment 2, a study where participants needed to recognize faces presented as neutral, with a non-emotional facial expression, and with a rigid head movement. As a matter of fact, if it is true that PD patients have deficits in the recognition of facial expressions and if it is true that the dorsal system for face recognition is involved in the recognition of the identity of faces encoded through an expression, we would expect PD patients to have difficulties in recognizing faces encoded through an expression but not as neutral. We used non-emotional facial expressions to further investigate the role of expressions by separating it from that of emotional processing.

5.2 Experiment 1

5.2.1 Aim

The first aim of Experiment 1 was to deepen our understanding of the mechanisms involved in facial expression recognition in patients with Parkinson's Disease. To do so, we tested them through a non-emotional expression recognition test and compared them with healthy controls. The second aim of Experiment 1 was to investigate facial identity recognition in PD. To do so, we also administered a task of identity recognition to our patients and a group of healthy controls.

5.2.2 Methods

5.2.2.1 Participants

Twenty-two patients and twenty-four control participants took part in the study. The sample size was determined through the software Gpower (parameters: power = .90, α= .05, medium effect size= .25) for a 2*2 (2 experimental conditions * 2 groups) design.

22 patients (12 females, mean age = 62,09; sd = 9,42) were recruited through the institute "Istituti Clinici Zucchi". Twenty-four patients were initially collected: however, two had to be excluded for being uncooperative and not finishing the experimental tasks. Patients with PD met clinical criteria for mild to moderate idiopathic PD (Hoehn & Yahr Stage 1–3). Motor disability was measured through the Unified Parkinson's Disease Rating Scale (UPDRS). All individuals with PD were taking medications for their motor symptoms and were in the "on" state during testing.

Table 5.1 displays the demographic and clinical characteristics of the PD group and control group.

Exclusion criteria for the patient group included: non-corrected visual or auditory problems; history of alcohol or drug abuse; DBS implant; neurological (other than PD) or psychiatric

illness; a deficit in a screening test for cognitive functioning (i.e., MoCa, Conti, Bonazzi, Laiacona, Masina & Coralli, 2015); history of traumatic brain injury or intracranial surgical operations.

24 control participants (11 females, mean age = 61,74; sd = 9,60) were recruited through caregivers. Exclusion criteria for the control group included not having a neurological or psychiatric illness; never suffering from traumatic brain injury; not having a deficit in a cognitive screening test (i.e., MoCa, Conti et al., 2015). All participants were native Italian speakers. Moreover, all participants provided written informed consent and the study was approved by the Ethics Committee of the University of Milan-Bicocca. All the procedures used in the study were in accordance with the Declaration of Helsinki.

	PD	HC	T	*p*
Mean age, year (SD)	62.09(9.42)	61.74(9.60)	-0.17	.86
Mean education, year (SD)	11.41(4.03)	12.29(4.46)	1.01	.32
Mean MoCa (SD)	23.86(2.27)	24.37(3.06)	0.92	.36
Mean BFRT (SD)	47.43(4.48)	48.71(3.79)	1.47	.15

T*able 5.1* Demographics of PD patients in comparison with HCs.

5.2.2.2 Stimuli

Stimuli were 10 avatars (5 females) created starting from the Chicago Face Database (Ma, Correll & Wittenbrink, 2015) through the software Character Creator 3. All avatars were rendered to be without hairs to prevent a facilitatory effect due to the hair cue in the recognition task (see the procedure section). For each of the 10 identities, 10 facial expressions were created. Those expressions were previously validated to be nonemotional in a pilot task on an independent sample of participants. An example of stimuli can be found in Figure 5.1.

Figure 5.1 Example of two male and two female identities showing 4 of the 10 non-emotional facial expressions created for the present experiment.

5.2.2.3 Procedure

After signing the informed consent, participants were administered a cognitive screening test (i.e., MoCa, Conti, Bonazzi, Laiacona, Masina & Coralli, 2015). After doing that, they were administered the Benton Facial Recognition Test (BFRT, Benton & Van Allen, 1968). Parkinson's disease patients were also administered the Questionnaire for impulsive-compulsive disorders in Parkinson's Disease (QUIP-RS) (Weintraub, Mamikonyan, Papay, Shea, Xie & Siderowf, 2012) and, when a caregiver was available, the Neuropsychiatric Inventory (NPI) (Cummings, Mega, Gray, Rosenberg-Thompson, Carusi & Gornbein, 1994).

All participants were then administered the two experimental tasks in counterbalanced order. The first experimental task was an identity recognition task. Participants looked at a face for 5 seconds and, right afterward, were presented with two faces. They were asked to indicate which of them represented the same person just seen. The two alternatives presented different facial expressions compared to the target, but one represented the same identity. An example of a trial can be seen in figure 5.2. The second experimental task was the expression recognition task. In this case, participants looked at a face for 5 seconds. Right afterward, they were presented with two alternatives and had to say which one represented the same facial expression. Identity changed from target to recognition, but one of the two alternatives represented a face with the same facial expression. An example of an expression recognition trial is represented in figure 5.3.

In the case of PD patients, the time when they last took their pharmacological therapy was noted as well as the year and month (when possible) when the first symptoms emerged.

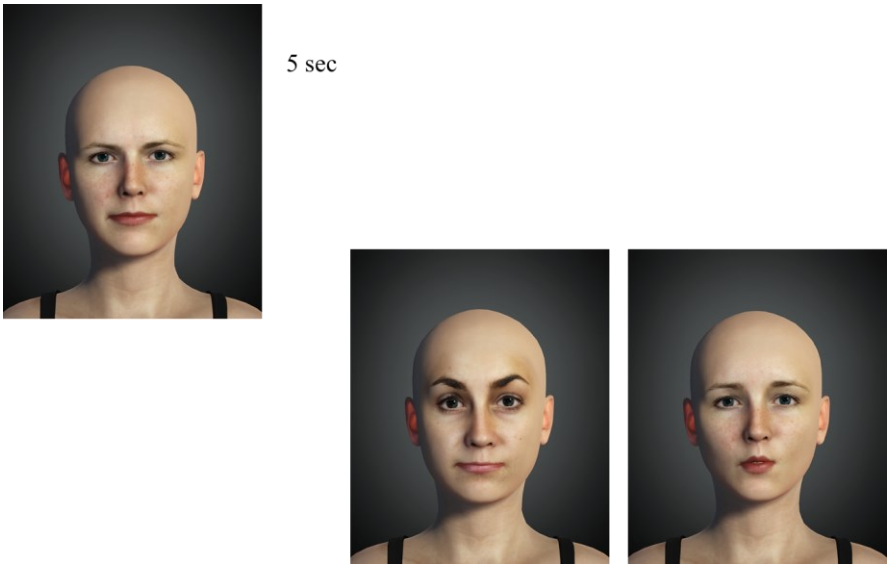

Figure 5.2. An example of an experimental trial from the identity recognition task.

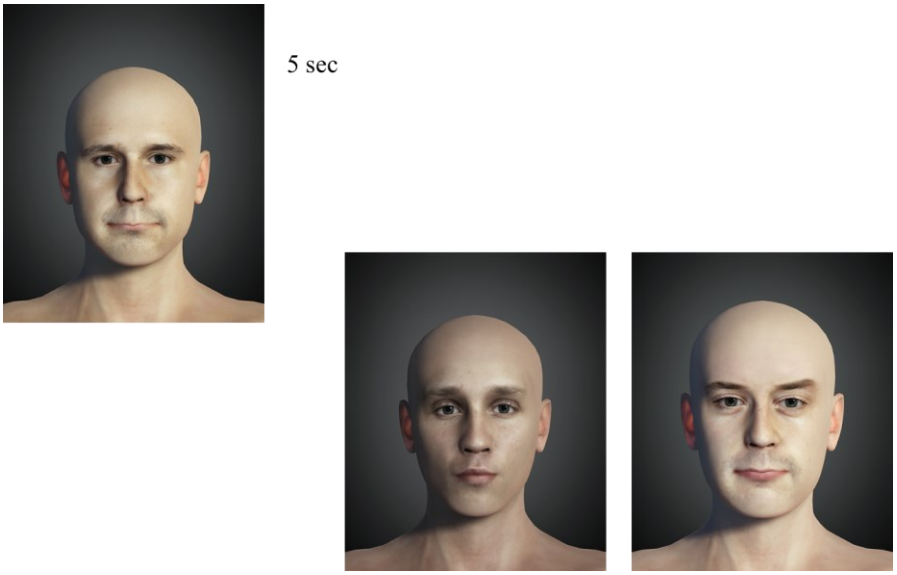

Figure 5.3 An example of an experimental trial from the expression recognition task.

5.2.3 Results

Statistical analyses were performed through the software R (version 4.0.4) and the packages *nlme* (Pinheiro et al., 2017), and *emmeans* (Russel, 2019).

Patients and controls were compared on age, education, and scores in the MoCa and BFRT tests by means of independent samples t-test. Results indicate that patients and controls did not differ in age, education, MoCa, or BFRT (see *Table 5.1*).

The dependent variable that was analyzed was the accuracy in the two tasks (Expressions and Identity recognition tasks). Analyses were performed excluding an outlier control participant scoring an accuracy of 0.55 in recognizing expressions and 0.97 in recognizing identities. We hypothesized that this control participant might have a selective deficit in recognizing facial expressions compared to identity recognition. For this reason, she was excluded from the analyses. Accuracies were analyzed through a linear mixed effect model where condition (i.e., expression or identity) and the group (i.e., HC or PD) were entered as fixed factors, and age, education, MoCa, and Benton were entered as covariates. Single subjects were entered as random factors. Results revealed a main effect of condition ($F_{(1,43)}$=30.899, p<.0001). Post-hoc tests revealed that the expression task ($emmean_{(39)}$ = 0.818) was harder on average than the identity one ($emmean_{(39)}$ = 0.902; t ratio = -5.445, p < .0001). However, no interaction between the condition and the group was found ($F_{(1,43)}$=2.295, p=.137). Moreover, the age covariate had a significant effect on accuracies ($F_{(1,39)}$=6.155, p=.0175) as well as the BFRT covariate ($F_{(1,39)}$=6.875, p=.0124). A simple regression with accuracy as the dependent variable and age as the independent variable revealed that the relation between these two variables was negative (estimate = -0.0029, T = -2.512, p=.0138); that is, the older the participants, the lower the accuracies. Moreover, a simple regression with accuracy as the dependent variable and BFRT as the independent variable revealed that the relation between these two variables was positive

(estimate = 0.0066, T = 2.523, p = .0134) that is, the higher the score in BFRT the higher the accuracy in the two experimental tasks.

Additionally, the relation between accuracy and the time when the last medication was taken was investigated in the PD group through a simple linear regression where accuracy was the dependent variable and the time since the last assumption was the independent variable. Results reveal no effect of the time since the last assumption on accuracies ($F_{(1)}$=0.612, p=.4384). Eventually, the relation between the distance from the insurgence of the disease and accuracies was investigated through a simple linear regression where accuracy was the dependent variable and the distance from the insurgence the independent variable. Results reveal no effect of the distance from the insurgence of the disease on accuracies ($F_{(1)}$=0.919, p=.3433).

5.3 Experiment 2

5.3.1 Aim

The aim of study 2 was to understand the mechanisms behind facial identity recognition in PD. In particular, we wanted to understand whether PD patients show differences in processing faces with a non-emotional expression. To do so, we tested them and a group of healthy controls in a task where they had to recognize the identity of faces encoded as neutral, with a non-emotional expression, or as neutral.

5.3.2 Methods

5.3.2.1 Participants

Twenty-four patients and twenty-five control participants took part in the study. The sample size was determined through the software Gpower (parameters: power = .95, α= .05, medium effect size= .25) for a 3*2 (3 experimental conditions * 2 groups) design.

24 patients (10 females, mean age = 67,33; sd = 9.95) were recruited through the institute "Centro Parkinson Bignami". Motor disability was measured through the Unified Parkinson's Disease Rating Scale (UPDRS). Table 5.2 displays the demographic and clinical characteristics of the PD group and control group.

Exclusion criteria for the patient group included non-corrected visual or auditory problems; history of alcohol or drug abuse; DBS implant; neurological (other than PD) or psychiatric illness; a deficit in a screening test for cognitive functioning (i.e., MoCa, Conti, Bonazzi, Laiacona, Masina & Coralli, 2015); history of traumatic brain injury or intracranial surgical operations.

25 control participants (18 females, mean age = 64,64; sd = 9.53) were recruited through caregivers. Exclusion criteria for the control group included: not having a neurological or psychiatric illness; never suffering from traumatic brain injury; not having a deficit in a

cognitive screening test (i.e., MoCa, Conti et al., 2015). All participants were native Italian speakers. Moreover, all participants provided written informed consent and study was approved by the Ethics Committee of the University of Milan-Bicocca. All the procedures used in the study were in accordance with the Declaration of Helsinki.

	PD	HC	T	*p*
Mean age, year (SD)	67.33(9.95)	64.79(9.67)	-1.48	.14
Mean education, year (SD)	12.79(3.45)	14.17(3.97)	1.66	.10
Mean MoCa (SD)	23.67(2.88)	24.96(2.40)	3.19	.0017
Mean BFRT (SD)	42.54(3.78)	44.37(4.30)	2.97	.0035

Table 5.2

5.3.2.2 Stimuli

Stimuli were created by selecting 60 identities (30 males) from the Chicago Face Database (Ma, Correll & Wittenbrink, 2015), matched for attractiveness and trustworthiness. Once identities were selected, an avatar was created for each of them using the program Character Creator 3. 10 avatars (5 males) were morphed to assume 6 non-emotional facial expressions, and 25 captures were taken, each one with a different and growing level of intensity of the expressions. The 25 captures were presented in rapid succession (every 60 ms) to simulate a movement. A pilot study was conducted to ensure that expressions were non-emotional. In addition to the non-emotional expressions, 10 avatars (5 males) were morphed to produce a rigid head movement on the three rotational axes. This condition served as a movement control condition. In fact, the stimuli displayed a rigid movement, but the facial expression remained

neutral. As for the expressions, 25 captures were taken for as many degrees of rotation and presented in rapid succession. To ensure that both expressions and rigid movements were indeed perceived as in motion, a pilot study was run. Eventually, 10 identities (5 males) were created as neutral and static.

Moreover, the static version of all identities was created, and 30 more static neutral identities (15 males) were created to serve as distractors. All images were on a greyscale to avoid recognition driven by color cues. For an example of the three categories of stimuli, see Figure 5.4.

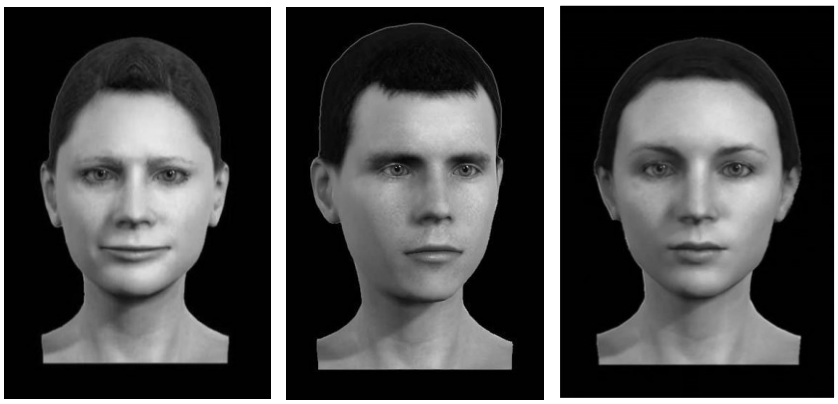

Figure 5.4 Examples of the stimuli. On the left is one of the validated non-emotional expressions, a rigid head movement around the horizontal axis, and a neutral expression on the right. Non-emotional facial expressions and rigid movement stimuli were dynamic, composed by a succession of 25 images (one each 60ms), while neutral faces were static.

5.3.2.3 Procedure

Both groups underwent cognitive evaluation through MoCa (Conti et al., 2015), the Benton Face Recognition Test (BFRT, Benton et al., 1983), and an experimental procedure which is described below.

The experimental procedure was structured as follows. Participants were presented with three blocks in randomized order. Each block belonged to one of the three conditions (i.e., non-emotional facial expression, rigid head movement, or neutral) and was divided into two parts. In the first part, a fixation cross was presented for 500 ms at the center of the screen. It was followed by the face presented in the experimental condition belonging to the block. Faces in the expression and rigid movement conditions were presented as a sequence of 25 frames, one each 60 ms for a total of 1500 ms. Each face was shown two times to reach a total exposure to each face of 3000 ms (see Daini, Comparetti & Ricciardelli, 2014). Faces were presented twice because presenting a single face for 3000 ms created a movement that was too slow and, therefore, unnatural. To create movement at a more natural speed, faces were shown twice for 1500 ms. For the neutral static condition, the faces were presented for 1500 ms two times to maintain the same exposure scenario as for the other conditions. After viewing each face twice, participants were asked to make a male/female decision to be sure that participants were processing the faces (Bindermann, Burton & Jenkins, 2005). Once the 10 faces belonging to the experimental condition were judged, the second part began, where the neutral static version of each face was presented for 3000 ms together with the distractors. Participants were asked to say whether they already saw that face or not. For an example of the experimental procedure, see Figure 5.5 or a demo version of the experiment at the following OSF link: https://osf.io/6p24r/.

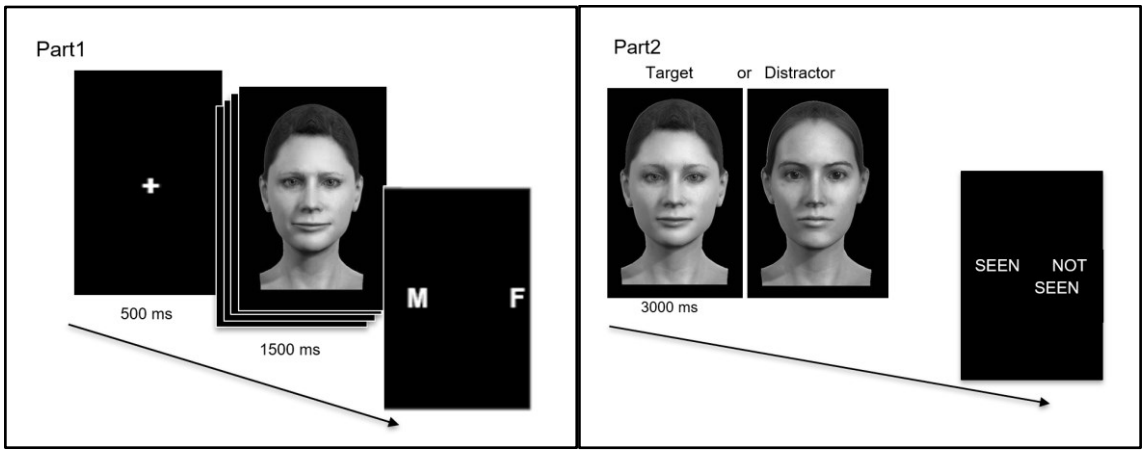

Figure 5.5 An example of the experimental procedure. Each block was divided into two parts: participants saw a fixation cross for 500 ms, followed by the stimulus in the first part. Each stimulus was presented twice for 1500 ms. After the stimulus, the letters M and F appeared on the screen, and the participants had to indicate whether the seen stimulus was a male or a female. After seeing the 10 identities belonging to the running block, participants were administered part 2. In this part, participants saw a neutral static face which could be the neutral static version of the already seen stimulus or a distractor for 3000 ms, and had to decide whether they already saw that face.

In addition to this, PD patients were administered the Questionnaire for impulsive-compulsive disorders in Parkinson's Disease (QUIP-RS) (Weintraub, Mamikonyan, Papay, Shea, Xie & Siderowf, 2012) and, when a caregiver was available, the Neuropsychiatric Inventory (NPI) (Cummings, Mega, Gray, Rosenberg-Thompson, Carusi & Gornbein, 1994).

4.3.3 Results

Statistical analyses were performed through the software R (version 4.0.4) and the packages *lme4* (Bates et al., 2009), *lmertest* (Kuznetsova, Brockhoff & Christensen, 2015), *emmeans* (Russel, 2019), *singcar* (Rittmo & McIntosh, 2021), and *psycho* (Makowski, 2018).

Patients and controls were compared on age, education, scores in the MoCa and BFRT tests by means of independent samples t-test. Results indicate that patients and controls did not differ in age, or education. However, they differed in MoCa raw scores, and BFRT raw scores (see *Table 2*).

The dependent variable considered was the d'prime score (Heeger & Landi, 1997), and it was calculated for each participant for each condition (i.e., non-emotional expression, rigid head movement, or neutral). D'prime scores were entered in a linear mixed-effect model as the dependent variable. Group (PD or control) and condition (non-emotional facial expression, rigid head movement, or neutral) were entered as fixed factors. Moreover, age, education, MoCa, and BFRT scores were entered as covariates. Single subjects were entered as random factors. Results reveal a main effect of condition ($F_{(2,92)}$=3.510, p = .0340). Post-hoc tests revealed that there was a significant difference between movement and neutral conditions (T ratio = -2.644, p=.0289). No other contrasts resulted as significant. A graphical representation of d'prime scores relative to each condition can be found in figure 6. No effect of group was observed ($F_{(1,42)}$=0.237, p = .6291). In addition, none of the covariates resulted as significant.

In order to consider hypomimia data for PD patients, we performed the same analysis only on the PD group. Particularly, we performed a linear mixed effects model where we entered dprime scores as the dependent variable and the presentation condition as a fixed factor. Moreover, we added age, education, MoCa, and Benton as covariates. In addition, the score relative to the UPDRS hypomimia question was added. It must be noted that we could not obtain this score for all of the participants as for some of them, it was impossible for the

neurologist to perform the UPDRS due to time constraints. Thus, this analysis was performed on a subsample of 20 patients. Results reveal a main effect of condition ($F_{(2,36)}$=4.085, p = .0252). Post hoc tests revealed that faces encoded with a rigid movement were recognized to a poorer extent both when compared to neutral faces (T ratio = -2.514, p = .0299) and non-emotional expressions (T ratio = 2.436, p = .0299). Interestingly, the UPDRS hypomimia score also significantly affected dprime scores ($F_{(1,14)}$=4.73, p = .0473).

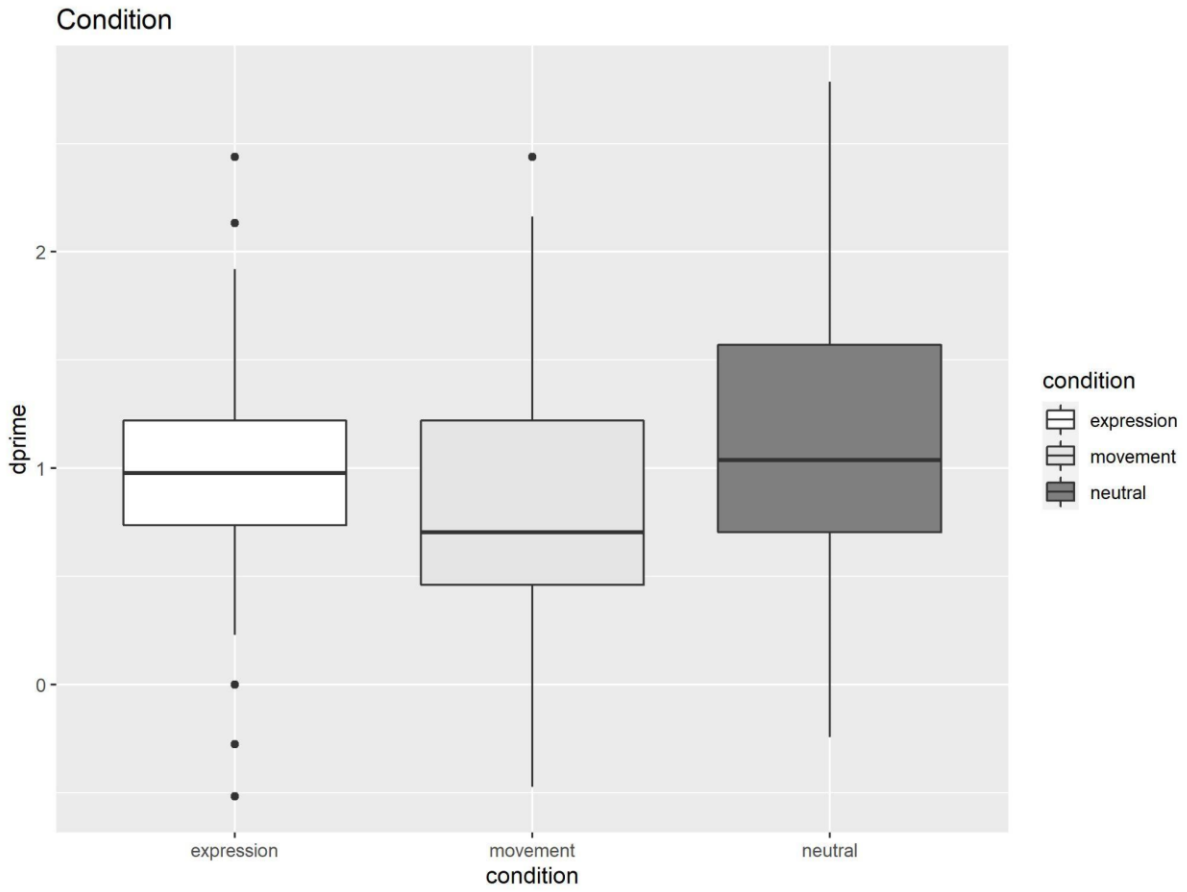

Figure 5.6 Graphical representation of dprime scores in the three experimental conditions.

5.4 Discussion

Parkinson's Disease is a degenerative pathology with unknown aetiology and is the second most frequent neurodegenerative disorder for frequency after Alzheimer disease (Tysnes & Storstein, 2017). It is characterized by motor and non-motor symptoms, among which a difficulty in recognizing facial expressions (Argaud et al., 2018). An explanation that has been proposed for such deficits postulates that PD affects neural substrates connected to basal ganglia, which in turn prevent patients from correctly recognizing emotional expressions (Wagenbreth et al., 2016; Péron et al., 2012).

Another explanation is the one given by the embodied cognition theory, which postulates that in order to understand an emotional expression, we simulate it (Gallese, 2005).

However, PD patients might not be as good at simulating it due to the presence of hypomimia and facial bradykinesia (Bowers et al., 2006), or to altered functioning of the mirror neuron system (Pohl et al., 2017). In order to further shed light on this matter, in study 1, we tested non-emotional facial expression recognition in a sample of PD participants compared to healthy controls. By using non-emotional expressions, we aimed to disentangle the contribution of emotion recognition impairment and the contribution of facial simulation of expressions in facial expression recognition. Moreover, to make sure that eventual expression recognition deficits were not due to general cognitive decline or perceptual malfunctioning, we administered a similar test to our participants investigating facial identity recognition.

Deficits in recognizing face identity have been reported in a minority of studies on PD patients (Sprengelmeyer et al., 2003; Marneweck et al., 2014). We know from face recognition models that we process faces using two separate yet interacting systems: the ventral one, involved in processing structural information, and the dorsal one, involved in processing information related to facial expressions and biological motion (O'Toole et al., 2002; Duchaine & Yovel, 2015). Those systems interact in the recognition of face identity. Thus, as PD patients were

reported to have deficits in the recognition of facial expressions, we wanted to understand whether they also have difficulties in recognizing identities of faces encoded through an expression. In Study 2, we tested a group of PD patients and an HC group in the recognition of the identity of faces encoded through non-emotional facial expressions, with a rigid movement or as neutral and static.

Results of study 1 reveal that our patients do not show difficulties in recognizing facial expressions or identities compared to the control group.

These results seem to be in line with the study from Derya and colleagues (2019), who did not find difficulties with non-emotional expressions in PD compared to HC. It is also in line with a recent study from Kuehne, Polotzek, Haghikia, Zaehle & Lobmaier (2023) finding no effect of facial feedback in emotion recognition in PD patients. This might indicate that PD patients have a deficit in general emotion recognition and not in the expressions themselves (Gray & Tickle-Degnen, 2010). This would also be sustained by studies finding emotion processing difficulties also in auditory stimuli, such as deficits in the recognition of emotion from prosody (Ariatti, Benuzzi & Nichelli, 2008; Kan, Kawamura, Hasegawa, Mochizuki & Nakamura, 2002). It must be noted that the two explanations for the expression recognition deficit in PD are not mutually exclusive. It might be that a deficit in emotion recognition and problems in expression mimicry interact in causing the deficit of patients with emotional expressions, and the deficit of mimicry itself is not as big to emerge for non-emotional facial expression recognition.

However, it might also be that the stimuli used in the present experiment were not suitable for measuring facial expression recognition. This is because we used static and not dynamic facial stimuli, which have been demonstrated to be more effective (Lotze, Reimold, Heymans, Laihinen, Patt & Halsband, 2009). In addition, it would be interesting to enlarge our sample

and verify the laterality of the insurgence of the deficit. Indeed, some authors separated their patients based on the laterality of the insurgence of symptoms. They found difficulties with expressions only in the patients with LPD whose insurgence is in the right hemisphere (Garrido-Vásquez et al., 2016). The right hemisphere is linked to expression processing and biological motion (Grossman et al., 2000), thus it might be that only considering these patients would reveal some interesting results.

Results from study 2 revealed that faces presented through a rigid head movement were more difficult to recognize: however, no group differences were found in this.

These results are coherent with Study 1 as our participants did not show difficulties in recognizing non-emotional expressions (study 1), and this did not reflect on identity recognition of faces encoded with non-emotional expressions (study 2), even if those stimuli were dynamic. Once again, this result seems to point in the direction of impairment in general emotion processing rather than in the expression processing in PD. Once again, it might be that there is also an influence of simulation of expressions for their understanding, but not as strong for non-emotional expressions. When looking at PD data from study 2 alone, a worse recognition of faces encoded through a rigid head movement was found coherently with the group analysis. However, this result was not found when HC data was analyzed alone. This might indicate that with a bigger sample, an effect of the group would emerge in the total analyses. The result obtained here for PD patients, even if it does not emerge from group analyses, might reflect a difficulty in processing movement in PD. This might be due to an impairment in circuits implicated in biological motion processing such as STS (Jaywant, Shiffrar, Roy & Cronin-Golomb, 2016).

However, it cannot be attributed to embodied account of expression simulation as no effect is found for expressions. It is interesting to note that in the PD group analyses, scores relative to

hypomimia significantly affected dprime scores relative to all conditions. The higher the hypomimia scores, the lower the dprime scores. It might be that motor disturbances are an effect of disease severity, and they might be linked to cognitive decline (Reijnders, Ehrt, Lousberg, Aarsland & Leentjens, 2009; Gasca-Salas & Urso,2020). Thus, it might be that the effect of hypomimia scores that we observe is due to general cognitive impairment rather than specifically linked to hypomimia. In fact, we observe a general effect rather than an effect specific to the expression condition. This would also be coherent with the results obtained by authors finding perceptual impairment in PD (Marneweck & Hammond, 2014) as it could be associated with disease severity too.

This study, although relevant, is not without limitations. Firstly, we did use static expressions in study 1. Many other studies used static expressions: however, to be sure that an effect cannot be observed, we could have also used dynamic expressions. Moreover, in both studies 1 and 2, we did not compare our non-emotional expressions with emotional expressions. This would make our results more solid. Finally, as a face recognition test, we used the BFRT in its original version. A new version was published in 2022 (Murray, Bennetts, Tree & Bate,2022): however, it was published only after data collection began.

In conclusion, from these two studies, it emerged that Parkinson's Disease patients do not show difficulties in recognizing static non-emotional facial expressions compared to Healthy Controls. Moreover, they do not show deficits in the recognition of identites of faces encoded through a dynamic non-emotional expression. These results point into an explanation of the deficit reported for PD in recognizing emotional expressions which is predominantly tied to emotion processing rather than to expression simulation.

6. Study 5[5]: face recognition in patients suffering from Posterior Cerebral Artery stroke

6.1 General introduction

The present study concerns data from the Back of the Brain (BoB) project, a project aimed at contributing to our understanding of the architecture of visual recognition of complex stimuli (Rice et al., 2021). To do so, 64 patients were selected for the project based on their lesion location (i.e., areas supplied by the Posterior Cerebral Artery) rather than their symptomatology. This methodology represents a novelty with respect to the current literature on recognition of complex objects, which is primarily focused on single case studies of patients selected based on their symptomatology (e.g., Barton, 2008). 64 patients with Posterior Cerebral Artery (PCA) stroke and 46 healthy controls underwent a large test battery assessing low-level, intermediate, and high-level perceptual processing. One of the advantages of the used battery is that it contains comparable tests of face, object and word perception. The present study focuses specifically on face processing. A full description of the tests composing the battery can be found elsewhere (Robotham, Kerry, Rice, Leff, Ralph & Starrfelt, 2021), while a brief description of the tests considered for the present study can be found in *Table 6.1*.

Although cases of pure acquired prosopagnosia are rare, milder impairments in face processing have been estimated to be present in 20 to 80% of acquired brain-injured patients, depending on the test administered (Valentine, Powell, Davidoff, Letson & Greenwood, 2006). Investigations on acquired prosopagnosics have been used in the literature to shed light on mechanisms subserving face processing: however, they are usually recruited based on specific pattern of impairments and not lesions. The present study is the first to our knowledge to assess

[5] This study is in preparation with Ro Julia Robotham and Randi Starrfelt

face recognition abilities formally with a wide variety of tests in a sample of patients recruited based on having lesions in areas related to this ability.

By using the data collected for the BoB project, in the present study we wanted to deepen our understanding of the mechanisms behind different aspects of face processing. We were interested in answering five main research questions pre-registered in the Open Science Framework prior to data analysis, which can be found at the following link: https://osf.io/m7vkq?mode=&revisionId=&view_only=. Firstly, we were interested in understanding whether face perception and face memory can be dissociated to some extent (RQ1) as those two processes were reported to have different brain substrates (Dalrymple et al., 2014) and deficits in the processes have been reported to occur both in conjunction and separately (e.g., Dalrymple et al., 2011). Moreover, we were interested in deepening our understanding of the degree of separation between deficits in mid-level and face perception (RQ2). Indeed, some mid-level deficits can be present together with face processing deficits in acquired prosopagnosics (Monti et al., 2019). However, although low level visual deficit such as field deficits can limit processing efficiency, they do not account for face processing deficit. This has been demonstrated by Barton (2008) who described a subject without field deficits but impaired in processing faces.

In addition, RQ3 was aimed at investigating whether the ability to match two identical faces can be dissociated from that of discriminating that two faces are different as those functions are reported to rely on separate mechanisms (White et al., 2017). Furthermore, we wanted to extend our knowledge on the degree of association or dissociation between face and object processing, which is widely debated in the literature (RQ4) (e.g., Barton et al., 2019). Eventually, we tried to sum up the criteria for diagnosing acquired prosopagnosia (Albonico & Barton, 2019) and see how many of our patients can be diagnosed as prosopagnosics according

to those criteria (RQ5). Each research question will be described separately in the following paragraphs.

By answering the five research questions, the present study has three main objectives. The first objective is to understand the degree of separation between face processing and other functions, such as mid-level visual perception or recognition of categories other than faces. In fact, heterogeneous results have been documented in the literature concerning the separation between mid-level perception and face processing on one side and between object and face processing on the other (Monti et al., 2019; Barton et al., 2019). Thus, the debate of a specificity of face processing versus other functions is still open.

The second objective is to deepen our understanding of mechanisms specific to face processing, such as the relation between unfamiliar face perception and face memory. Within the face processing domain, several theoretical models exist on its functioning (e.g., Bruce & Young, 1986; Haxby et al., 2000; Haxby & Gobbini, 2011; Duchaine & Yovel, 2015). Having data on so many patients give us the opportunity to assess different aspects of face processing in detail, such as perception and memory, and the level of segregation between them.

Thirdly, we wanted to investigate the prevalence of severe face recognition impairments that may qualify as acquired prosopagnosia in our sample. Acquired prosopagnosia is considered to be a rather rare deficit. However, a worsening in face recognition, even if not severe enough to fulfil criteria for prosopagnosia, was observed in a large proportion of patients with acquired lesions (Valentine et al., 2006). This makes it relevant to deepen our understanding of the mechanisms behind face processing.

To do so, data were analysed using the Single Case methodology to detect both deficits in single tests and dissociations between the different tests considered. Dissociations can be divided into strong and classical. A strong dissociation refers to cases where the patients' scores

significantly differ from the control sample on both task X and task Y, and, in addition, the difference between the two tasks is greater than that of the control sample. A classical dissociation occurs when the patient is impaired in task X but not task Y, and the difference between task X and Y is greater than that observed in the control sample (Crawford & Garthwaite, 2005). In the present study, only classical dissociations were considered as they are more informative on the distinction between the considered cognitive processes (Gerlach, Lissau & Hildebrandt, 2018). Moreover, age was included as a covariate in all analyses. This choice was made in light of what was done in the first published paper on the BoB data where the authors found age to correlate with performance in all domains in the control group (Rice et al., 2021).

Name of the test	About the test
Cambridge Face Memory Test (Duchaine & Nakayama, 2006)	During the test, participants learn a set of 6 new male faces, and then are asked to recognise them amongst two distractors, either in the presence of visual noise or without. The dependent measure for this test is accuracy.
Delayed Matching and Surprise Recognition test of Words, Objects and Faces (WOF test) (Robotham, 2019)	The Delayed Matching Test assesses the ability to build a short-term representation of a stimulus and then match it with the same or a novel stimulus. The Surprise Recognition test that is administered directly after is an old/new recognition paradigm that assesses whether participants can recognise stimuli that were used in the Delayed Matching part of the test. Processing of words, objects and faces are assessed independently in each part.
Leuven Perceptual Organisation Screening Test (Torfs et al., 2014), modified	This test includes 15 subtests assessing a wide range of midlevel processes. The following sub-tests were included in the BoB-protocol: Fine shape discrimination; Shape ratio discrimination (Efron); RFP contour integration; Figure–ground segmentation; Embedded figure detection; RFP texture segmentation; Kinetic object segmentation; Dot counting; Global motion detection. Participants were presented with a target image at the top of the screen and three test images below. Participants must determine as fast as possible which of the test images are most similar to the target image. For the BoB-project, a modified version of the test was created in OpenSesame to enable both accuracy and reaction time measurements. Also, the video subtests were different due to technical reasons.
Cambridge House Memory Test (Martinaud et al., 2012)	The test has the same experimental set-up as the CFMT but involves learning a set of 6 new houses and then recognising them amongst distractors, either in the presence of visual noise or without. The dependent measure for this test is accuracy.
Face recognition questionnaire (Freeman et al., 2015)	The following 10 questions were selected from the Face Identity Recognition part of the questionnaire by Freeman and colleagues (2015): □ I can usually remember what someone's face looks like, even if I've only met them once. □ I find it difficult to decide whether I know a face or not. □ I have trouble finding my friends in a crowded room. □ I occasionally fail to recognise myself in old photos. □ I often have conversations with people who appear to know me, but (at least initially) I have no idea who they were. □ I often rely on distinctive bodily features, hair, or clothing to help identify people. □ I rarely confuse characters in TV programs. □ I usually recognise my friends in old photographs. □ If I saw my neighbour at the shops, I would recognise them. □ If a friend changed their hairstyle I would most likely be able to identify them. To ensure that potential problems were indeed related to brain injury, two additional questions were added: □ My ability to recognise faces has got worse since my stroke/head injury (Same scale as questions above) □ Is there anything else you want to tell me about your ability to recognise faces before your stroke/head injury? (Open question) Answer was on a scale from 1 (Definitely agree) to 4 (Definitely disagree)
Famous Face Naming (Roberts et al., 2015)	The test contains 40 items, pictures of famous faces are presented one at a time centrally on a screen and participants are asked to name the person out loud as quickly and as accurately as possible. The dependent measure for this test was accuracy.

Table 6.1. A description of face and object tests used for the present study.

6.2 RQ1 = Can face perception and face memory be dissociated in PCA stroke patients?

6.2.1 Introduction

According to the literature, face perception and face memory are separate processes. Impaired face memory and preserved face perception has been demonstrated in both acquired (Barton et al., 2004; Dalrymple et al., 2011; Tippett et al., 2000) and developmental prosopagnosics (Dalrymple et al., 2014; Ulrich et al., 2017; Tian et al., 2020). Different brain structures seem to be involved in those processes: acquired prosopagnosics with primarily perceptual deficits show lesions to occipitotemporal regions, while acquired prosopagnosics with prevalent memory deficits show more anterior lesions, even though the latter has also been shown to lead to face perception deficits (Dalrymple et al., 2014). However, there are also cases where face perception and memory deficits are associated, both in acquired (Barton et al., 2004; Busigny et al., 2014; Dalrymple et al., 2011) and developmental (Liu et al., 2021) prosopagnosia.

By running this analysis, we want to investigate the incidence of perceptual and memory deficits in our patients and if there are cases where those two deficits are dissociated.

6.2.2 Method

To investigate the present question, we analysed performance on the Cambridge Face Memory Test (CFMT-Duchaine & Nakayama, 2006) and the new tests designed specifically for the BoB project, namely the Delayed Face Matching (DFM) test and the Face Surprise Recognition (FSR) test (for a detailed description of the test battery and new tests administered see Robotham et al., 2021). We regarded the matching subpart of the CFMT and the Delayed Face Matching as face perception tests, while the recognition subpart of the CFMT and the Face Surprise Recognition were accounted as face memory tests. The noise part of the CFMT recognition was excluded from the analyses (Corrow, Albonico & Barton, 2018).

6.2.3 Analyses

We compared each patient to the control group on each subtest by using a Bayesian Test for Deficit allowing for covariates (Crawford et al., 2011) implemented in R Studio (R Core Team, 2017) through the package "*Singcar*" (Rittmo & McIntosh, 2021). Subsequently, the performance of each patient was compared to the control group for both subparts of CFMT to assess the presence of classical dissociations. The same was done to compare the performance of the Delayed Matching Test and the Surprise Recognition Test. To do so, we conducted a Bayesian standardized difference test allowing for covariates (Crawford et al., 2011) implemented in R Studio (R Core Team, 2017) through the package "*Singcar*". In both analyses, Age was entered as a covariate.

6.2.4 Results

CFMT scores were obtained for all 64 patients and 45 controls. Results concerning the CFMT subparts are summarised in *Table 6.2*. As is visible in the upper part of the table, patients are generally less accurate at both CFMT matching and recognition subparts compared to controls. 17 patients resulted as impaired in CFMT matching, 9 of which showed a classical dissociation with CFMT recognition (impaired matching and preserved recognition). 5 patients were impaired only in CFMT recognition, none of which showed a classical dissociation with the CFMT matching subtest. Eventually, 10 patients showed impairment in both subparts of CFMT.

	Matching	**Recognition**
Patients	Mean(Sd) = 83,77(19,02)	Mean(Sd) = 55,73(17,24)
Controls	Mean(Sd) = 97,16(5,63)	Mean(Sd) = 72,89(19,75)

	Only matching	**Dissociation**	**Only recognition**	**Dissociation**	**Both tests**
N impaired	17/64	9/17	5/64	0/5	10/64

Table 6.2. Results relative to the CFMT matching and recognition subtests.

A graphical representation of the results is presented in *Figure 6.1*.

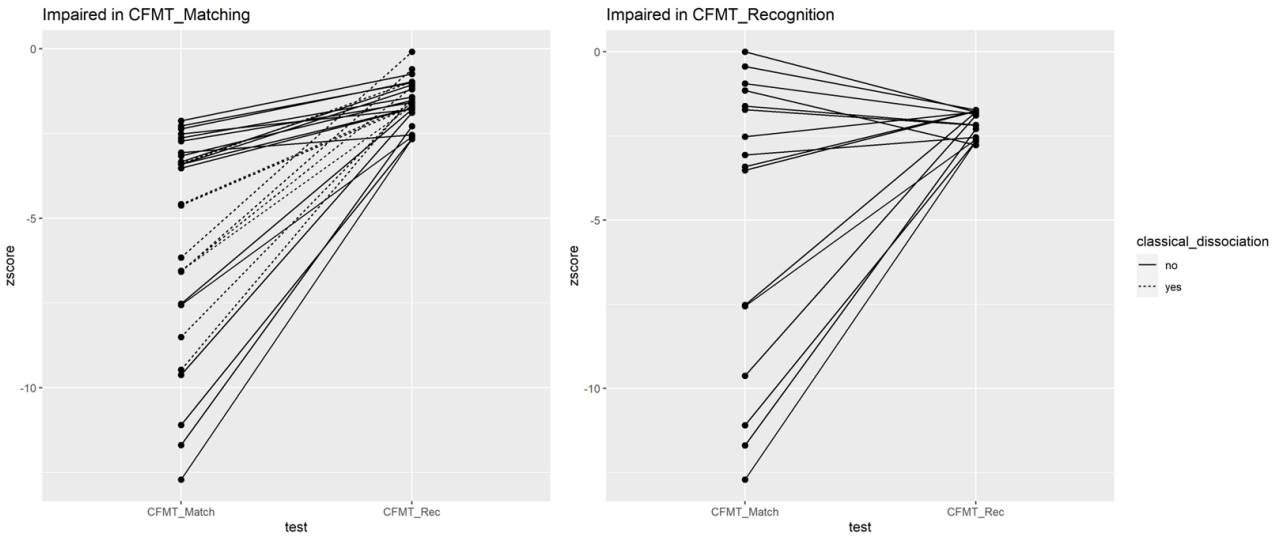

Figure 6.1. In the plots, z-scores on the two CFMT subtests are represented for participants impaired in CFMT matching and recognition subparts. Moreover, a dotted line represents those patients that also show a significant classical dissociation between the two z-scores.

Delayed Face Matching scores were obtained for 63 patients and 43 controls. Face Surprise Recognition scores were obtained for 62 patients and 43 controls. In both the Delayed Face Matching and Face Surprise Recognition tests, both accuracy and reaction times were recorded. In order to consider both data, a composite score was calculated for each subject to reduce the two scores to one factor. Factors were calculated through a factor reduction performed through the program SPSS 27 following the method used by Rice and colleagues (2021). Single case analyses were then performed on the composite scores. Results concerning the Delayed Face Matching and Face Surprise Recognition tests are summarised in *Table 6.4*.

	Matching	Recognition
Patients	Mean(sd) = -0.377(1.104)	Mean(sd) = -0.286(1.185)
Controls	Mean(sd) = 0.514(0.483)	Mean(sd) = 0.413(0.375)

	Only DFM	Dissociation	Only FSR	Dissociation	Both
N impaired	12/63	4/12	5/62	2/5	16/62

Table 6.4. Results relative to the Delayed Face Matching and Face Surprise Recognition tests.

As is visible in the upper part of the table, patients perform worse than controls at both Delayed Face Matching and Face Surprise Recognition tests. 12 patients resulted as impaired in the Delayed Face Matching test based on single case statistics, 4 of which showed a classical dissociation with the Face Surprise Recognition test (impaired Delayed Face Matching and normal Face Surprise Recognition). 5 patients were impaired only in the Face Surprise Recognition test, 2 of which showed a significant classical dissociation with Delayed Face Matching test (impaired Face Surprise Recognition and normal Delayed Face Matching). Finally, 16 patients showed impairment in both subparts of CFMT.

A graphical representation of the results is presented in *Figure 6.2*.

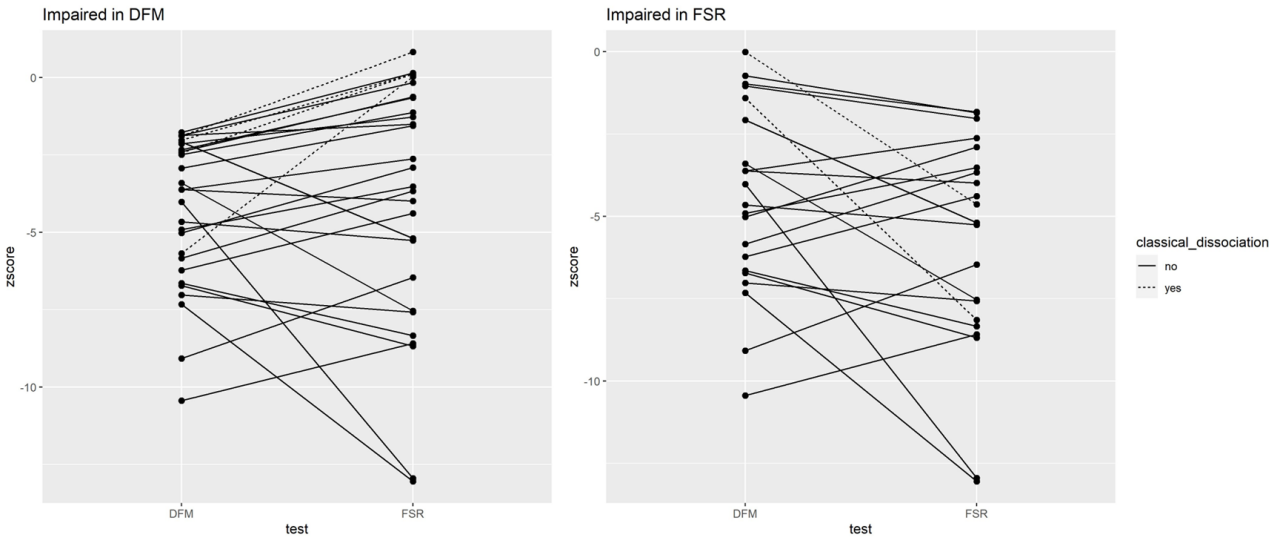

Figure 6.2. In the two plots, scores on the two Delayed Face Matching and Face Surprise Recognition tests are represented, respectively, for participants impaired in Delayed Face Matching and Face Surprise Recognition tests. Moreover, a dotted line represents those patients that also show a significant dissociation between the two scores.

Contrary to CFMT results, we can see that dissociations occur in both directions. This is likely because the Delayed Face Matching and Face Surprise Recognition are independent tests and not subparts of the same test as with CFMT. This, in turn, might lead to more reliable psychometric properties.

Methodological box

Delayed Face Matching and Face Surprise Recognition tests measure both reaction times (RT) and the percentage of correct responses (PC). A measure that includes both RT and PC is the Inverse Efficiency Score (IES, Townsend & Ashby, 1978; Townsend & Ashby, 1983). This score is obtained by dividing RT by the proportion of correct responses (PC). However, many controversies exist regarding its use. Bruyer & Brysbaert (2011) state that IES is a good measure only when there is a small percentage of errors and when RT and PC are highly correlated. These controversies brought us to use composite scores instead of IES in the present analysis. To ensure that composite scores were suitable to represent the performance of participants in Delayed Face Matching and Face Surprise Recognition subtests, we correlated the created composite scores with both response times and accuracy separately. Results for Delayed Face Matching test revealed a negative correlation between reaction times and composite scores (cor = -0.87, $p < 2.2e-16$) and a positive correlation between accuracy and composite scores (cor = 0.87, $p < 2.2e-16$). Results for Face Surprise Recognition test revealed a negative correlation between reaction times and composite scores (cor = -0.89, $p < 2.2e-16$) and a positive correlation between accuracy and composite scores

(cor = 0.89, $p < 2.2e-16$). Thus, we can conclude that composite scores accurately account for both RT and PC. As an exploratory analysis, we also computed IES for our data and correlated this score with our composite scores. Results indicate that the correlation between the two measures is high for both Delayed Face Matching (cor = -0.96, $p < 2.2e-16$) and Face Surprise Recognition (cor = -0.94, $p < 2.2e-16$) tests. In conclusion, it seems that composite scores and IES scores are equally good in measuring reaction times and accuracy, at least for the data that was considered.

6.2.5 Discussion

Scores in tests of face perception and face memory were analysed separately to explore the possibility of selective deficits. Results indicate that there are selective deficits in face perception and face memory in the sample. This double dissociation was found in the CFMT subtests and the new tests created for the present study. The fact that there are cases where face perception is impaired but recognition is preserved might seem counterintuitive. In fact, if face perception is necessary for its subsequent retrieval, we would expect face perception deficits to lead to face memory deficits. A possible explanation of such dissociation might be that those participants, in the recognition stage, rely on familiarity mechanisms which have been shown to be preserved in participants with difficulties with faces (Burns, Tree & Weidemann, 2014; Stumps, Saad, Rothlein, Verfaellie & DeGutis, 2020). Moreover, a selective deficit for face memory was observed in the new tests but not for the CFMT subtests. This might be due to the fact that CFMT subtests were not created to be considered separately and show different variances. Thus it might be that they are not adequate to explore this dissociation. In conclusion, these results indicate that face perception and face memory can be selectively impaired. Thus, the dissociation found in the present data should be investigated further by using tests that were created to be administered separately and not two subparts of the same test as we did with the

CFMT. Moreover, tests using more trials would be beneficial in further understanding the relation between face perception and memory.

6.3 RQ2 = To what extent is face perception dissociated from low- and mid-level perceptual processes?

6.3.1 Introduction

Impairments in one or more subtests of L-Post (Torfs, Vancleef, Lafosse, Wagemans & de-Wit, 2014), a test of mid-level perceptual processing, is often used as an exclusion criterion in studies on congenital prosopagnosics, as face perception deficits are commonly dissociated from general perceptual deficits in developmental cases (e.g., Macaskill et al., 2021; Fry et al., 2020; Stumps et al., 2020). However, in patients with an acquired impairment, the distinction between the two deficits is not as predictable. Indeed, some mid-level deficits can be present together with face processing deficits in acquired prosopagnosics (Monti et al., 2019).

By running this analysis, we want to investigate whether there are cases of impaired face perception with preserved mid-level processing in our sample of patients. On the other hand, we also want to explore the possibility that patients with face perception deficits show mid-level perceptual deficits.

6.3.2 Method

To investigate the present question, we considered the matching part of the CFMT and the Delayed Face Matching test as face perception tests and the L-Post as a measure of mid-level perceptual processing.

6.3.3 Analyses

We compared each patient to the control group on each subtest by using a Bayesian Test for Deficit allowing for covariates (Crawford et al., 2011) implemented in R Studio (R Core Team,

2017) through the package "*Singcar*" (Rittmo & McIntosh, 2021). Subsequently, the performance of each patient was compared to the control group for CFMT matching and L-Post to assess the presence of dissociations. The same was done to compare the performance of the Delayed Face Matching Test and the L-Post. To do so, we conducted a Bayesian standardized difference test allowing for covariates (Crawford et al., 2011) implemented in R Studio (R Core Team, 2017) through the package "*Singcar*". In both analyses, Age was entered as a covariate.

6.3.4 Results

CFMT match scores were obtained for all 64 patients and 45 controls. L-Post scores were obtained for 63 patients and 45 controls. Results concerning the CFMT match and L-Post are summarised in *Table 6.5.* As is visible in the upper part of the table, patients are generally less accurate at both CFMT matching and L-Post compared to controls. 11 patients were impaired in L-Post based on single case statistics, 6 of whom showed a classical dissociation with CFMT matching (impaired in L-Post but preserved in CFMT matching). 10 patients were impaired only in CFMT matching, 4 of whom showed a classical dissociation with the L-Post test (impaired in CFMT matching but preserved in L-Post). Finally, 16 patients showed impairment in both tests.

	CFMT_matching	**L-Post**
Patients	Mean(sd) = 83,77(19,02)	Mean(sd) = 70,58(18,09)
Controls	Mean(sd) = 97,16(5,63)	Mean(sd) = 82.62(8.14)

	Only L-Post	**Dissociation**	**Only CFMT_mat**	**Dissociation**	**Both**
N impaired	11/63	6/11	10/64	4/10	16/63

Table 6.5. Results relative to the CFMT match and L-Post tests.

A graphical representation of the results can be seen in *Figure 6.6.*

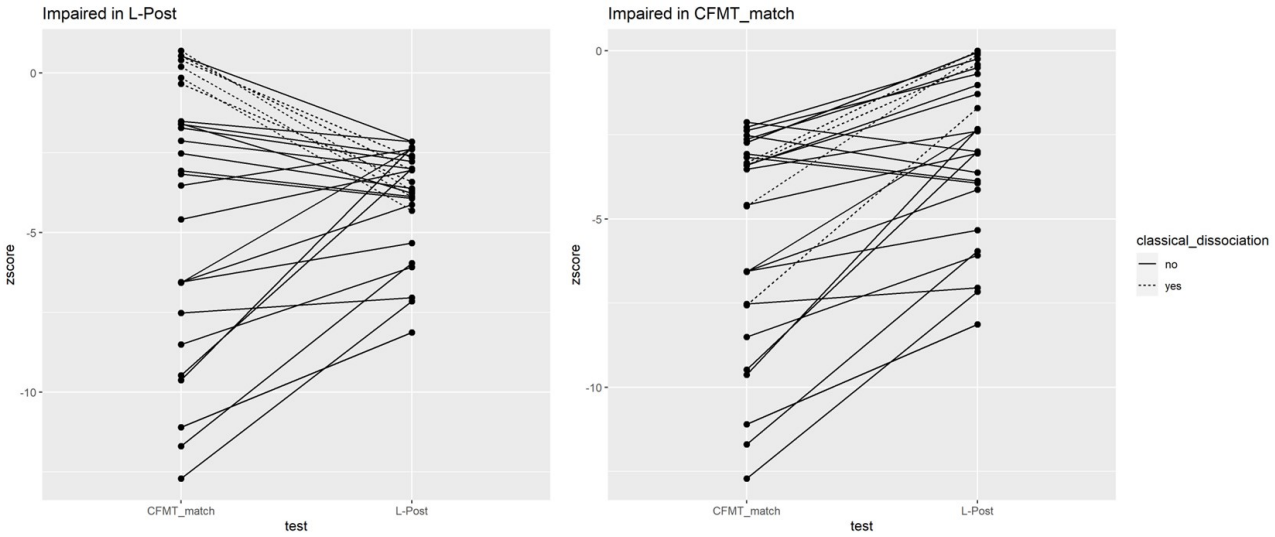

Figure 6.6. In the two plots, z-scores of L-Post and CFMT matching are represented, respectively, for participants impaired in L-Post and CFMT_match tests. Moreover, a dotted line represents those patients that also show a classical dissociation between the two scores.

Delayed Face Matching scores were obtained for 63 patients and 43 controls. L-Post scores were obtained for 63 patients and 45 controls. Results concerning the Delayed Face Matching and L-Post tests are summarised in *Table 6.6.* As is visible in the upper part of the table, patients are generally less accurate at both Delayed Face Matching and L-Post. 9 patients were significantly impaired only in Delayed Face Matching, 3 of whom showed a classical dissociation with L-Post (impaired in Delayed Face Matching and preserved in L-Post). 7 patients were impaired only in L-Post, 2 of whom showed a significant dissociation with the Delayed Face Matching test (impaired in L-Post and preserved in Delayed Face Matching). 19 patients showed impairment in both tests.

	Matching	L-Post
Patients	Mean(sd) = -0.38(1.10)	Mean(sd) = 70,58(18,09)
Controls	Mean(sd) = 0.53(0.48)	Mean(sd) = 82,62(8,14)

	Only matching	Dissociation	Only L-Post	Dissociation	Both
N impaired	9/63	3/9	7/63	2/9	19/63

Table 6.6. Results relative to the Delayed Face Matching and L-Post tests.

A graphical representation of the results is shown in *Figure 6.7*.

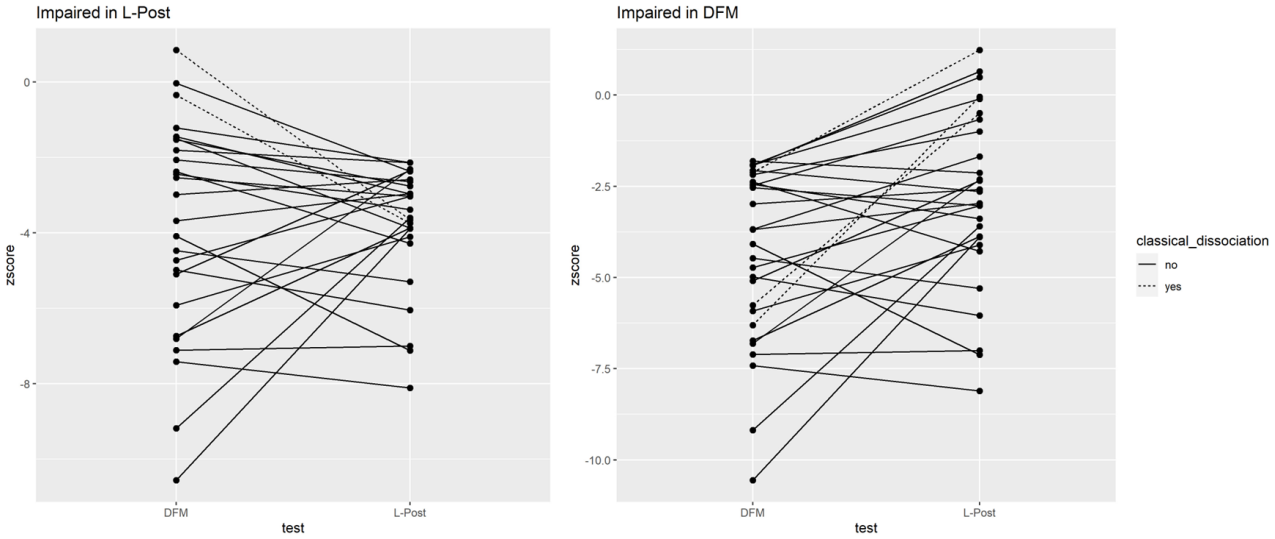

Figure 6.7. In the two plots, scores of L-Post and Delayed Face Matching are represented, respectively, for participants impaired in L-Post and Delayed Face Matching tests. Moreover, a dotted line represents those patients that also show a classical dissociation between the two scores.

6.3.5 Discussion

Scores in mid-level visual perception and face perception tests were analysed to explore the possibility of selective deficits. Results indicate that there can be selective deficits in both mid-level visual perception and in face perception. This pattern was present when comparing the CFMT matching subtest to the L-Post and the new test for face matching (DFM) to the L-Post. A few cases showing a selective impairment in mid-level visual perception but

preserved face perception were identified. This result is interesting as we would expect patients having a deficit in mid-level visual perception also to show difficulties in face perception. This is also a result in favour of the existence of a system specific for faces. Moreover, the opposite classical dissociation is also observed, where some patients show normal mid visual perception but impaired face perception. Again, this might reflect the use of at least partially specific mechanisms for face processing. It must also be taken into consideration that to investigate a dissociation between mid-level visual processing and face perception we considered the matching subpart of the CFMT. Again, this is a subpart of a test and not a test validated to be administered alone and the matching part shows a wide variability in patients. Thus, the results concerning the CFMT should be interpreted cautiously.

6.4 **RQ3 = Are there specific patterns for face matching?**

6.4.1 Introduction

Matching faces and discriminating whether two faces are different may rely on separate mechanisms (Fysh et al., 2018; Berger et al., 2021). Developmental prosopagnosics have been reported to differ in face and object matching compared to the normal population: in a study by White and colleagues (2017), DP's did not show differences in match/non-match trials when matching objects while they were more impaired in match trials for faces compared to non-match trials. In addition, Berger and colleagues (2021) found a significant worse matching ability in DPs than in controls. However, they did not find significant differences between match and non-match trials. Exploring these two processes in patients with an acquired lesion might inform us about which processes might be affected in cases of acquired lesions. To do so, we analyzed match and nonmatch trials in a face and an object recognition task.

By running this analysis, we aim to determine whether there is a difference in the match- and non-match trials in the face and object matching tests.

6.4.2 Method

To investigate the present question, we considered the match and non-match trials of the Delayed Face Matching test and Delayed Object Matching test.

6.4.3 Analyses

We compared each patient to the control group on match and non-match trials separately for both tests by using a Bayesian Test for Deficit allowing for covariates (Crawford et al., 2011) implemented in R Studio (R Core Team, 2017) through the package "*Singcar*" (Rittmo & McIntosh, 2021). Subsequently, the performance of each patient was compared to the control group for match and non-match trials for both tests to assess the presence of dissociations. To do so, we conducted a Bayesian standardized difference test allowing for covariates (Crawford

et al., 2011) implemented in R Studio (R Core Team, 2017) through the package "*Singcar*". In both analyses, age was entered as a covariate.

6.4.4 Results

Delayed Face Matching scores were obtained for 63 patients and 43 controls. Results concerning the match and non-match trials comparison for Delayed Face Matching test are summarised in *Table 6.7.* As is visible in the upper part of the table, patients are generally less accurate than controls in both match and non-match trials. 10 patients were significantly impaired in match trials, 5 of whom showed a classical dissociation with non-match trials (impaired in match but preserved in non-match trials). 10 patients were impaired only in non-match trials, 3 of whom showed a classical dissociation with the match trials (impaired in non-match and preserved in match trials). 16 patients showed impairment in both tests.

	Match	**Non-match**
Patients	Mean(sd) = -0,179(1,094)	Mean(sd) = -0,606(1,096)
Controls	Mean(sd) = 0,637(0,465)	Mean(sd) = 0,267(0,643)

	Only Match	**Dissociation**	**Only Non-match**	**Dissociation**	**Both**
N impaired	10/63	5/10	10/63	3/10	16/63

Table 6.7. Results on the match and non-match trials of the Delayed Face Matching test.

A graphical representation of the results can be found in *Figure 6.8*.

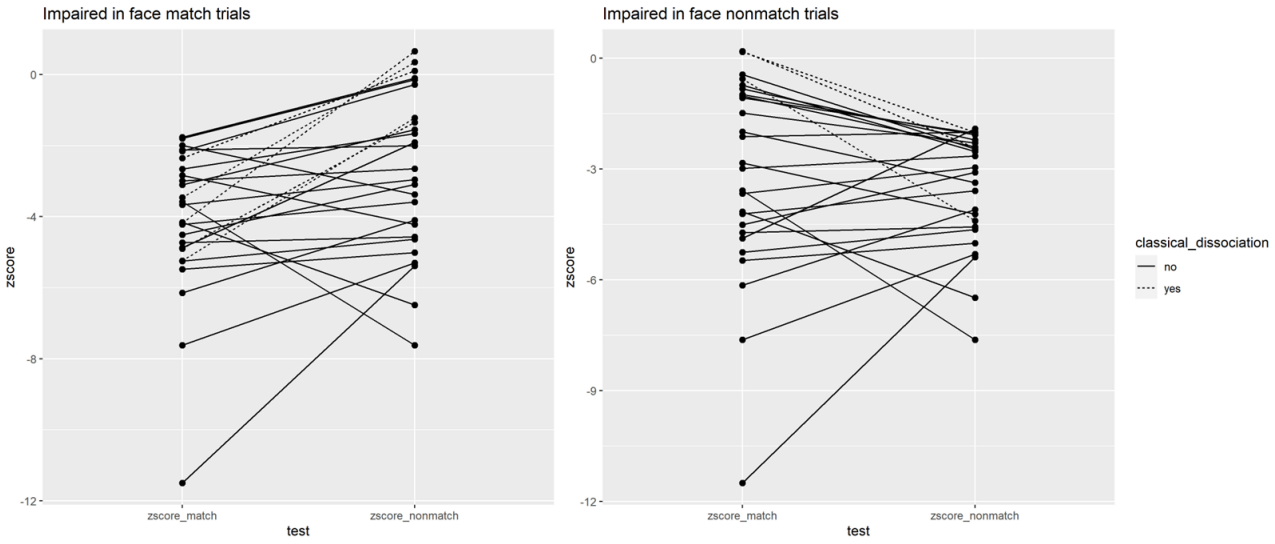

Figure 6.8 In the two plots, scores of Delayed Face Matching match and non-match trials are represented, respectively, for participants impaired in match and non-match trials. Moreover, a dotted line represents those patients that also show a significant dissociation between the two scores.

Delayed Object Matching scores were obtained for 63 patients and 43 controls. Results concerning the match and non-match trials comparison for Delayed Object Matching test are summarised in *Table 6.8.* As is visible in the upper part of the table, patients are generally less accurate at both match and non-match trials. 11 patients showed significant impairment on the match trials, 10 of whom showed a classical dissociation with non-match trials (impaired match and preserved non-match trials). 6 patients were impaired only in non-match trials, 3 of whom showed a classical dissociation with the match trials (impaired non-match and preserved match trials). 12 patients showed impairment in both tests.

	Match	Non-match
Patients	Mean(sd) =-0,255(1,014)	Mean(sd) = -0,679(0,890)
Controls	Mean(sd) = 0,410(0,462)	Mean(sd) = -0,083(0,732)

	Only Match	Dissociation	Only Non-match	Dissociation	Both
N impaired	11/63	10/11	6/63	3/6	12/63

Table 6.8. Results relative to the match and non-match trials comparison for Delayed Object Matching test.

A graphical representation of the results can be found in *Figure 6.9*

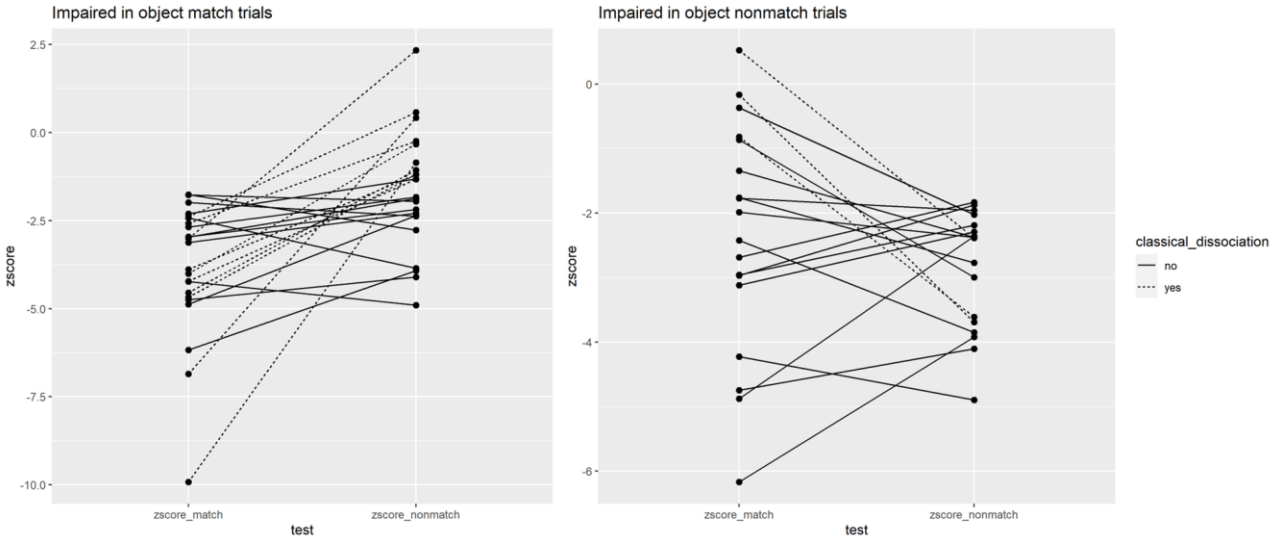

Figure 6.9. In the two plots, scores of delayed object match and non-match trials are represented, respectively, for participants impaired in match and non-match trials. Moreover, a dotted line represents those patients that also show a significant dissociation between the two scores.

In the literature, different patterns have been shown in match and nonmatch trials for faces with respect to objects. Thus, we further investigated the performance of those patients showing a classical dissociation between match and nonmatch trials with faces to explore their performance with objects. When looking at the five patients selectively impaired in face match trials, three of them show the same pattern for objects (i.e., impaired object matching

and classical dissociation with nonmatch trials). The remaining two patients showed no impairment in match or nonmatch object trials.

6.4.5 Discussion

Scores in match and nonmatch trials were analyzed to investigate whether there are differences between match and nonmatch trials for faces and objects, respectively, in posterior stroke patients. Results indicate the presence of selective deficits in both match and nonmatch trials compared to controls in our group of patients. There were more patients with selective deficits in match trials than patients with deficits in nonmatch trials. This result corresponds to the results obtained by White and colleagues (2017), who find developmental prosopagnosics (DPs) to be impaired in match but not nonmatch trials compared to controls. The authors interpret this result as a possible effect of a conservative criterion used by their group of DPs in that these participants are biased in responding that the two faces are different. In contrast, controls are biased in responding that the two faces are the same. This might apply to our group of patients too. In addition, the authors state that this result might be due to the fact that DPs use a part-based strategy for face processing to a larger extent compared to controls. According to the literature, processing faces usually requires configural processing, while object processing relies more on a part-based strategy (Richler & Gauthier, 2014). This explanation could fit our results; if true, we would expect different results for faces and objects. However, the patients of the present study are impaired in match more than in nonmatch trials both for faces and for objects. Thus, this interpretation cannot explain the present results. Moreover, in our sample of patients, match trials are more frequently selectively impaired for objects than faces. This result might mean that mechanisms impaired in our sample of patients concern general perceptual functioning rather than a processing mechanism specific to faces. Eventually, we wanted to deepen our understanding of the patients showing a selective deficit

for face-match trials. In particular, we wanted to understand whether they showed the same pattern in object match trials or not. Thus, we looked at their performance in object match trials. However, results are mixed in this regard and do not show a specific pattern. Three of them showed the exact same pattern of dissociation in object match trials, while the other two were preserved both in object match and nonmatch trials.

This prevents us from drawing any conclusion on which mechanisms might have been disrupted. These results might reflect the fact that there is a large individual variability in the match/nonmatch performance already in healthy individuals (Berger et al., 2021), which is, in turn, represented and even exaggerated in our sample of patients with an acquired lesion. Alternatively, these results might be due to the specific deficit caused in each patient by the specific lesions, which can involve low or higher-level perceptual mechanisms such as part-based or configural processing.

6.5 RQ4 = Is memory for faces dissociated from memory for other within-object categories?

6.5.1 Introduction

A longstanding question in the literature regards the level of association between deficits of face recognition and object recognition deficits. In particular, as faces are exemplars belonging to the same category, the debate concerns whether patients having difficulties in recognizing faces also have difficulties in recognizing objects belonging to the same category (within-class recognition) (Damasio, Damasio, & van Hoessen, 1982). The literature regarding acquired prosopagnosia (AP) is mixed. On the one hand, many studies report AP patients to be spared in within-class recognition e.g., of cars (Busigny, Graf, Mayer & Rossion, 2010; Schiltz & Rossion, 2006; Busigny & Rossion, 2010; Henke, Schweinberger, Grigo, Klos & Sommer, 1998; Rezlescu, Pitcher & Duchaine, 2012), birds, boats, chairs (Schiltz & Rossion, 2006) or vegetables (Henke et al., 1998). On the other hand, other studies report AP patients to have deficits in within-class recognition of the same type of objects (Barton, Cherkasova, Press, Intriligator & O'Connor, 2004; Barton, 2008; Barton & Corrow, 2016; De Haan & Campbell, 1991; Toftness, 2019). However, it might be that potential differences between face and within-category object processing are caused by a failure to benefit from experience with these types of objects and not a domain-general impairment in their recognition (Fry, Wilmer, Xie, Verfaellie & DeGutis, 2020). That is why some authors tested acquired prosopagnosics on novel objects for which neither patients nor control have previous experience. Also in this case, evidence is mixed with studies finding dissociation with face recognition (Rezlescu, Barton, Pitcher & Duchaine, 2014; Riddoch, Johnston, Bracewell, Boutsen & Humphreys, 2008) and others finding an association of deficits (Gauthier, Behrmann & Tarr, 1999). In conclusion, the literature includes heterogeneous results (Barton, Albonico, Susilo, Duchaine & Corrow,

2019). That is why we decided to compare face and within-category object recognition in our sample of patients with an acquired lesion compared to the control group.

6.5.2 Method

To investigate the present question, we investigated performance on the Cambridge Face Memory Test (CFMT) and the Cambridge House Memory Test (CHMT).

6.5.3 Analyses

We compared each patient to the control group on CFMT and CHMT by using a Bayesian Test for Deficit allowing for covariates (Crawford et al., 2011) implemented in R Studio (R Core Team, 2017) through the package "*Singcar*" (Rittmo & McIntosh, 2021). Subsequently, the performance of each patient was compared to the control group for CFMT and CHMT to assess the presence of dissociations. To do so, we conducted a Bayesian standardized difference test allowing for covariates (Crawford et al., 2011) implemented in R Studio (R Core Team, 2017) through the package "*Singcar*". In both analyses, Age was entered as a covariate.

6.5.4 Results

Data for the CFMT were obtained for 64 patients and 45 controls. Data for the CHMT were obtained for 53 patients and 36 controls. Results are summarised in *Table 6.9.* 18 patients were significantly impaired on CFMT, none of whom showed a dissociation with CHMT. 9 patients were impaired only in CHMT, 2 of whom showed a significant dissociation with CFMT (preserved face and impaired house recognition). 7 patients showed significant impairment in both tests.

	Faces	Houses
Patients	Mean(sd) = 65,73(16,47)	Mean(sd) = 81,64(13,75)
Controls	Mean(sd) = 82,7(13,1)	Mean(sd) = 89,7(5,63)

	Only faces	Dissociation	Only houses	Dissociation	Both
N impaired	18/64	0/9	9/53	2/9	7/53

Table 6.9. Results relative to CFMT and CHMT score.

A graphical representation of the results can be found in *Figure 6.10.*

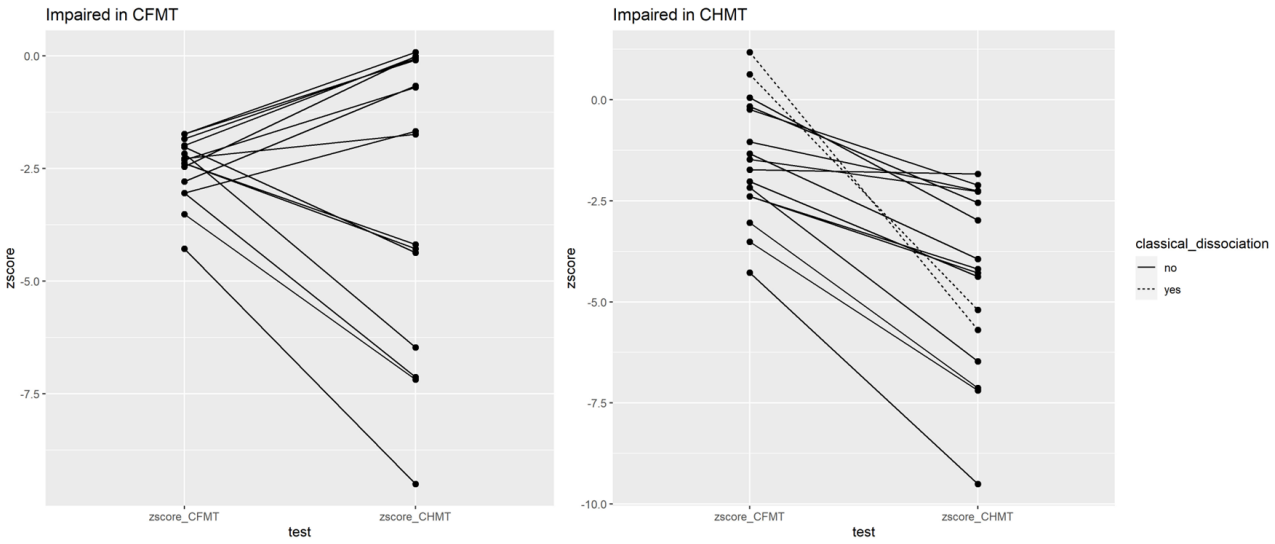

Figure 6.10. In the two plots, scores of CFMT and CHMT are represented, respectively, for participants impaired in CFMT and CHMT. Moreover, a dotted line represents those patients that also show a significant dissociation between the two scores.

It must be underlined that 9 of the patients who were impaired in the CFMT were not tested with the CHMT. This is because it was the last test of the battery and was left out when patients were tired or severely impaired in the other tests. Thus, the results described above might be misleading. That is why we decided to compare face processing and within-class object processing for the new tests that were created for the present study: the Delayed Face Matching test was compared to the Delayed Object Matching Test and the Face Surprise recognition test was compared to the Object Surprise Recognition test. These tests are structured exactly in the same way, only the stimulus category differs, and the choices in the object tests are always within category (both targets and foils are from the same object category).

Results of the Delayed Matching Face and object comparison were obtained for 63 patients and 45 controls. They are summarised in *Table 6.10.* 8 patients were selectively impaired in DFM, 4 of whomh showed a significant classical dissociation with Delayed Object Matching (impaired Delayed Face Matching and preserved Delayed Object Matching). 4 patients were only impaired in Delayed Object Matching, 1 of whom showed a classical dissociation with Delayed Face Matching (impaired Delayed Object Matching and preserved Delayed Face Matching). 22 patients were impaired in both tests.

	DFM	**DOM**
Patients	Mean(sd) = -0.377(1.104)	Mean(sd) = -0.351(1.072)
Controls	Mean(sd) = 0.514(0.483)	Mean(sd) = 0.536(0.666)

	Only faces	**Dissociation**	**Only objects**	**Dissociation**	**Both**
N impaired	8/63	4/8	4/63	1/4	22/63

Table 6.10. Results relative to Delayed Face Matching and Delayed Object Matching comparison. A graphical representation of these results can be found in *Figure 6.11*.

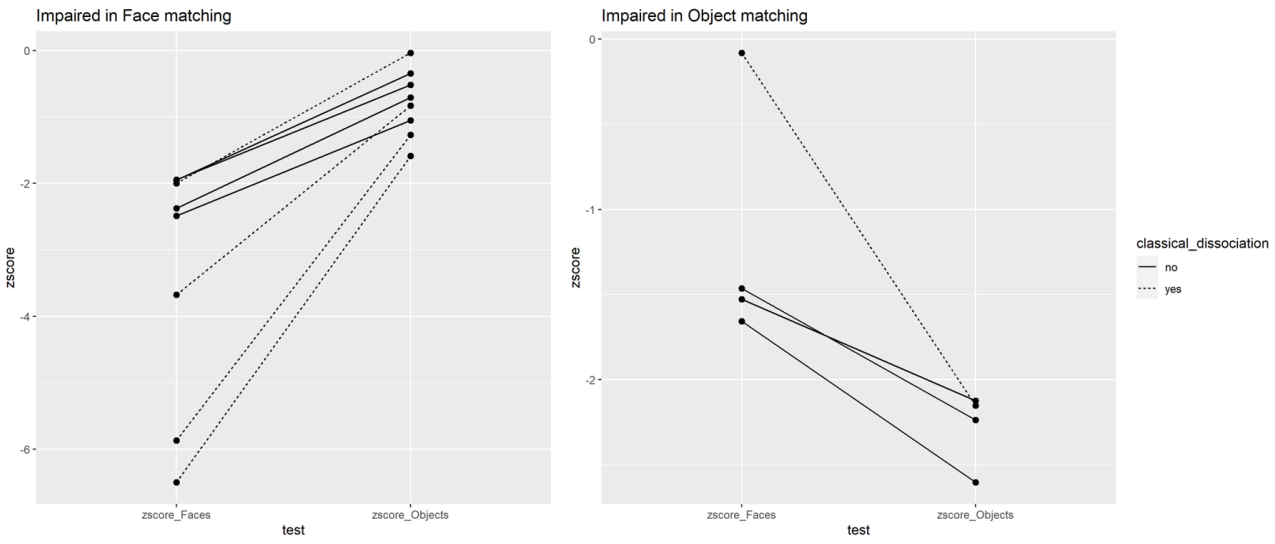

Figure 6.11 In the two plots, scores of Delayed Face Matching and Delayed Object Matching are represented, respectively, for participants impaired in Delayed Face Matching and DOM. Moreover, a dotted line represents those patients that also show a classical dissociation between the two tests.

Results of the Surprise recognition test for faces and objects were obtained for 62 patients and 43 controls. They are summarised in *Table 6.11.* 7 patients were selectively impaired in Face Surprise Recognition, 5 of which showed a classical dissociation with Object Surprise Recognition (impaired Face Surprise Recognition and preserved Object Surprise Recognition). 11 patients were only impaired in Object Surprise Recognition, 5 of which showed a classical dissociation with Face Surprise Recognition (impaired Object Surprise Recognition and preserved Face Surprise Recognition). 14 patients were impaired in both tests.

	FSR	**OSR**
Patients	Mean(sd) = -0.286(1.185)	Mean(sd) = -0.331(1.121)
Controls	Mean(sd) = 0.413(0.375)	Mean(sd) = 0.427(0.527)

	Only faces	**Dissociation**	**Only objects**	**Dissociation**	**Both**
N impaired	7/62	5/7	11/62	5/10	14/62

Table 6.11. Results relative to Face Surprise Recognition (FSR) and Object Surprise Recognition (OSR) comparison. A graphical representation of the results can be found in *Figure 6.12.*

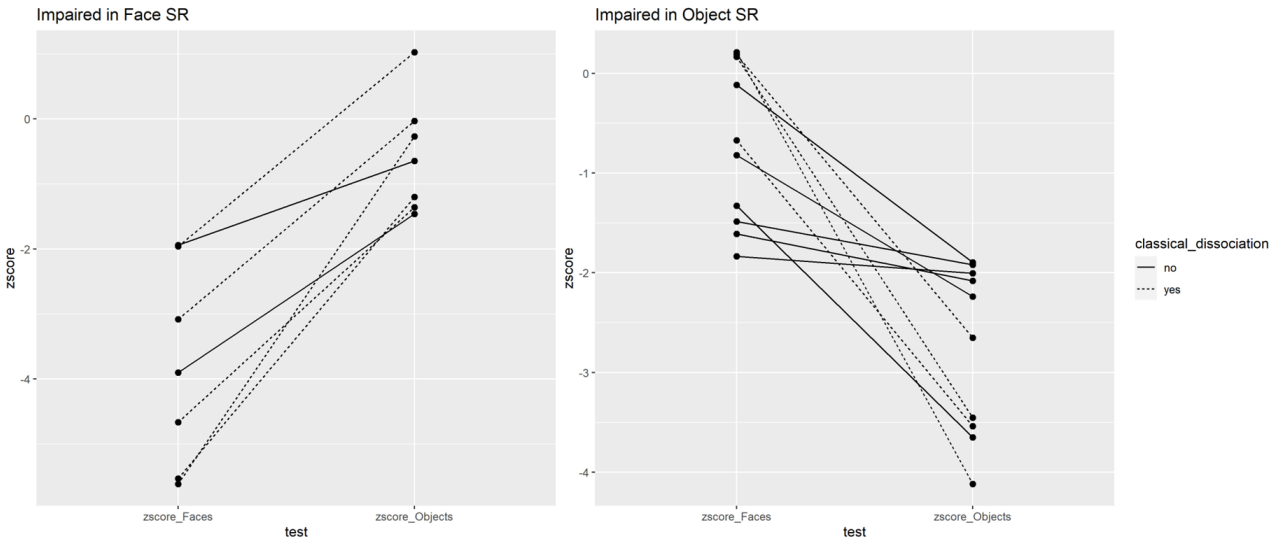

Figure 6.12 In the two plots, scores of Face Surprise Recognition (Face SR) and Object Surprise Recognition (Object SR) are represented, respectively, for participants impaired in Face Surprise Recognition and Object Surprise Recognition. Moreover, a dotted line represents those patients that also show a classical dissociation between the two tests.

6.5.5 Discussion

A classical model of face processing deficits is that of Farah (2004). The author states that faces are "special" as double dissociations between faces and objects have been reported. According to this model, cases where there is preserved face, but impaired object recognition should not exist. Although Farah's model is still a reference to the literature, advances have been made on the debate on how special faces actually are. Acquired deficits in recognizing faces and objects belonging to the same category have been reported to be associated (e.g., Toftness, 2019) but also dissociated (e.g., Rezlescu et al., 2012). In the present study, we wanted to investigate their pattern of association/dissociation in a sample of patients with an acquired lesion to the territory supplied by the posterior cerebral artery. To our knowledge, the present study is the first to investigate face and object processing in a big sample of patients with an acquired lesion. Results of the comparison between the CFMT and the CHMT show that a classical dissociation with impaired face but preserved object processing did not occur in our sample. In contrast, there were two cases of the opposite dissociation (impaired objects but preserved face processing). These results must be interpreted cautiously as many patients impaired in CFMT were not tested on the CHMT. That is why we explored this matter by analysing results from the new tests created for the present study (i.e., Delayed Face Matching, Face Surprise Recognition, Delayed Object Matching, and Object Surprise Recognition). Results indicate that a classical dissociation between face and object processing is possible in both directions (i.e., both impaired face with preserved object processing and impaired object with spared face processing). Our results are heterogeneous and are in line with those obtained by Barton and colleagues (2019), who find results going in both directions. A possible interpretation the authors propose is that patients having a deficit both in face and object processing have a general perceptual deficit. In contrast, those showing a dissociation have a deficit specific to faces or objects. This interpretation is coherent with our results as our patients show

heterogeneous lesions and, consequently, are likely to have different types of deficits. On the same line, it might also be that the association of deficits arises from damage to domain-general stages (i.e., low-level sensory processing and high-level cognitive functions), and deficits specific for faces arise from damage to intermediate stages that are face-specific (Eimer, 2018). A final remark concerns general object processing: in the present analyses, faces and within-category tasks with objects were analysed. However, a measure of general object processing is missing. This type of measure could help us further clarify the nature of the observed deficit and dissociations.

6.6 RQ5 = How many acquired prosopagnosic can we diagnose in this group of 64 PCA patients based on published criteria?

6.6.1 Introduction

According to Albonico & Barton (2019), acquired prosopagnosia can be diagnosed if the following criteria are fulfilled: a coherent lesion on imaging, self-reported difficulty in face recognition compared to pre-accident abilities, and poor performance on a test of face recognition. Together with confirmation by MRI scan and self-reported difficulties in recognition, Corrow and colleagues (2016) suggest diagnosing prosopagnosia on the basis of impairment in at least two face familiarity tests instead of just one, as in Albonico & Barton (2019). Based on this, we used the following criteria to diagnose prosopagnosia:

- Self-reported difficulty in face recognition. To fulfil this criteria patients had to report a worsening of their face recognition abilities following their stroke and had to score minimum 2 sd below the control mean on the general questionnaire on face recognition abilities

- Impairment (i.e., at least 2 sd below the control mean) in at least 2 face familiarity tests within CFMT, surprise recognition test, and Famous Faces Recognition Test
- Coherent lesion according to imaging. As patients were recruited based on their lesion site in the areas supplied by the Posterior Cerebral Artery, they were all considered potential candidates for prosopagnosia.

By diagnosing patients with prosopagnosia, we aim to determine whether there are patients with acquired prosopagnosia in the sample, the proportion of patients fulfilling the diagnostic criteria, and what their scores are in the Cambridge Face Memory Test, the Surprise Recognition Test and the Famous Faces Recognition Test Are the patterns generally similar to those of other patients, or do acquired prosopagnosics behave differently?

6.6.2 Methods

Only patients reporting a worsening of difficulties in face recognition following their stroke are included in the analysis. The score of each patient on the CFMT, surprise recognition test, and Famous Faces Recognition Test is compared to the control group to identify patients scoring 2 sd below the control mean in at least 2 of the 3 tests.

6.6.3 Results and discussion

Within the present research question, we aimed at diagnosing potential acquired prosopagnosic patients in our sample. We adopted criteria present in the literature and diagnosed patients reporting worsening performance with faces after the injury which in addition showed impaired performance in at least two tests of face recognition between CFMT, the Surprise Recognition Test and the Famous Faces Test. It must be noted that the chosen criteria for diagnosing prosopagnosia are strict compared to the literature: when considering the self-reported worsening with faces, we considered both the information given by a specific question relative to the self-reported worsening since the accident and the information given by a questionnaire

on general difficulties with faces. Usually, in the literature just one measure of self-reported worsening with faces is considered (e.g., Barton et al., 2019): nevertheless, we considered both the available measures to have a complete picture. This might have reduced the potential number of diagnosed prosopagnosic patients. This number might also have been reduced due to the fact that the question about self-reported worsening with faces was missing in four cases. Two of these four patients would have met the other diagnostic criteria but could not be classified as having prosopagnosia. Despite the strict criteria, three patients in the sample can be diagnosed as having acquired prosopagnosia.

If we look at the performance of the diagnosed patients on tests other than the diagnostic ones, two aspects are interesting. First, all of them have total scores in L-Post test that are 2 sd below the control mean (all of them are also impaired when using Crawford test). Thus, all of the diagnosed prosopagnosic patients do not show dissociation between mid-level perceptual processing and face processing. In addition, if we look at their performance with objects, one of them scores 2 sd below the controls mean on the object surprise recognition test (same result when considering results from the Crawford test), while the second has a normal score in that test (confirmed by results obtained using the Crawford test). The third acquired prosopagnosic does not have a score on that test. In conclusion, when looking at the performance of our diagnosed prosopagnosics in domains other than face processing, we see that all of them are impaired in mid-level visual processing but only one of them is impaired in Object recognition. In addition, it is also interesting to note that 33 patients reported a subjective worsening of face recognition abilities compared to premorbid abilities, even if they did not reach our criteria for diagnosis. Moreover, 18 patients were impaired in at least one of the diagnostic tests (i.e., Cambridge Face Memory Test; Face Surprise Recognition, and Famous Faces Test. These

results are in line with those reported by Valentine and colleagues (2006) about high prevalence of mild face recognition impairments in brain-injured patients.

Although cases of pure acquired prosopagnosia are rare, milder impairments in face processing have been estimated to be present in 20 to 80% of acquired brain-injured patients, depending on the test administered (Valentine, Powell, Davidoff, Letson & Greenwood, 2006).

6.7 General Discussion

In the present study, data from the Back of the Brain (BoB) project were analysed. This project aimed at contributing to our understanding of the neural substrates behind the recognition of complex stimuli (Rice et al., 2021). 64 patients with lesion to the areas supplied by the Posterior Cerebral Artery were tested together with 46 healthy controls. The choice of choosing patients based on the lesion location represents a novelty as the majority of studies select patients only based on their symptomatology (e.g., Barton et al., 2008). All patients underwent a large battery assessing low-level, intermediate and high-level perceptual processing. Infarction to Posterior Cerebral Artery can include the occipital, inferomedial temporal, and posterior parietal lobes (Cals, Devuyst, Asfar, Karapanayiotides & Bogousslavsky, 2002; Park, Yoon & Rhee, 2011; Busigny et al., 2014). Those territories are claimed to be deputed to different levels of visual perception, including territories generally associated with face perception (Van Belle et al., 2011). Thus, in the present study, we specifically aimed at focusing on face processing abilities. The present paper was specifically focused on analysing data related to questions about to face processing performance. In particular, we tried to answer five preregistered research questions: the first one (RQ1) regarded the degree of independence between face perception and face memory. Results indicated that face perception and face memory can be selectively impaired but this happens only in a minority of cases while most patients show these functions to be associated. RQ2 aimed to understand the degree of independence between general mid-level visual perception and face perception. Once again, selective deficits were found in a minority of patients. RQ3 aimed to understand whether there are differences between performance on match and non-match trials for faces and objects in our patients compared to controls. Results indicate selective deficits both for match and non-match trials, with more patients showing significant deficits for match than nonmatch trials and no differences between faces and objects. RQ4 was aimed at exploring whether deficits were specific for faces or if they extended

to object processing. Results indicate that a classical dissociation between face and object processing is possible in both directions. Eventually, RQ5 was aimed at diagnosing our patients with acquired prosopagnosia. Only three patients conformed to (admittedly strict) criteria for acquired prosopagnosia. However, 33 out of 64 patients reported difficulties with faces arising after the brain injury. Moreover, 18 patients were impaired in at least one of the face tests that was used to diagnose prosopagnosia in our study.

The first main aim of the present study was that of understanding the level of independence of face processing from other visual perceptual functions (i.e. mid-level visual perceptual processing and object processing). Results of RQ2 and RQ4 revealed that in our sample of patients, face processing can be dissociated from other, related functions. We know from the literature that face processing has a core neural substrate composed of the fusiform face area, the occipital face area, and the superior temporal sulcus and an extended system widespread throughout the brain (Haxby et al., 2000; Haxby & Gobbini, 2011; Elbich & Sherf, 2017). As suggested by Eimer (2018), association of deficits might arise from impairment at the level of domain-general processes while specific deficits might be the result of impairment to face-specific stages. If this is true, it might be that the majority of our patients showing a deficit for faces associated with other visual functions have damage to domain-general processes. This is in line with recent domain-general views of agnostic deficit such as that suggested by Strappini, Pelli, Di Pace & Martelli (2017) who compare agnosic vision with crowded vision. This result is further supported by the fact that all of our three diagnosed prosopagnosic patients showed abnormal performance in L-Post. Thus, we can conclude that face processing systems and general visual perceptual systems can be segregated, but that selective deficits are possible, perhaps reflecting that visual recognition is subserved by a network composed by regions with a distributed and graded functional specialization (Behrmann & Plaut, 2020).

As far as the second aim of the study is concerned, RQ1 and RQ3 helped us in understanding whether face memory and face perception are dissociable and whether face matching and discrimination rely on separate mechanisms. Face perception and memory could be selectively impaired. Moreover, face matching and discrimination seem to rely on at least partially separate mechanisms even though this distinction does not seem specific for faces but for perceptual processing in general.

Eventually, the third aim of the present paper was that of understanding the prevalence of prosopagnosia in our sample of patients with an acquired lesion to the posterior cerebral artery. Despite using strict diagnostic criteria, three patients reached criteria for diagnosis of acquired prosopagnosia. What is even more interesting is that 33 of our patients reported subjective worsening with faces after the accident and that 18 patients were impaired in at least one of our diagnostic tests. Thus, it seems that acquired difficulties with faces are not so rare after all. Consequently, developing new rehabilitative procedures becomes even more urgent (Gobbo, Calati, Silveri & Daini, 2022).

A final issue worth considering is the utility of dissociations and associations. Dissociations are often considered to be informative in neuropsychology as proof of independent mechanisms (Gray & Cook, 2018; Towler & Tree, 2018). Specifically, in the case of patients with an acquired lesion, an association might reflect either a lesion to adjacent networks or affection of common processes, while a dissociation reflects a more selective deficit and, at the same time, is thought to constitute proof of a specific substrate for one function (Garrido, Duchaine & DeGutis, 2018). In our study, dissociations informed us on the segregation between functions. In conclusion, from the present study it emerged that face processing can be dissociated from other visual perceptual functions. However, this seems to be the exception and not the rule as

it happens only in a minority of cases. Moreover, also within face processing it is possible to dissociate between different functions such as perception and memory or matching and discriminating. Eventually, we were able to diagnose three patients with acquired prosopagnosia and in addition to that, half of the patients reported subjective worsening with faces after the accident and one-third of them showed deficits in at least one of the diagnostic tests.

7. General discussion

In this dissertation, I investigated the mechanisms behind face recognition, its neural substrates and new potential rehabilitation for prosopagnosic patients, and I contributed to extending current models of face recognition. Overall, our results on non-emotional facial expressions indicated that they aid identity recognition, particularly for poor recognizers. This suggests their potential utility for rehabilitation of prosopagnosia. Surprisingly, motor mimicry of expressions due to the activity of subcortical structures does not have a role in identity recognition. On the contrary. the Superior Temporal Sulcus and pre-Supplementary Motor Area are implicated in this facilitation. Moreover, an analysis of data from patients with PCA stroke suggested that face recognition can be dissociated from low/mid-level perceptual functions and object processing: however, this seemed the exception and not the rule, as in most cases, those functions were associated.

Study 1 reviewed existing literature on the rehabilitation of object agnosia and prosopagnosia. Regarding prosopagnosia, treatments involving holistic perceptual processing (categorization or matching) of faces seem to be the most effective and can be generalized to new views and perspectives of faces (Davies-Thompson et al., 2017; Corrow et al., 2019; De Gutis et al., 2007; DeGutis et al., 2014). Therefore, it seems that a holistic analysis of face-like stimuli is effective for both acquired and developmental prosopagnosia. However, the literature on prosopagnosia rehabilitation is still limited and new studies addressing the topic are required. Moreover, it might be that the lack of studies on the rehabilitation of agnosias has its origins also in the scarce clarity about the mechanisms subserving face processing. It must also be noted that no official guidelines for diagnosing agnosias exist.

We know from models of face recognition that facial identity and expressions are processed by functionally and anatomically separate but interacting systems (Haxby et al., 2000; O'Toole, 2002; Duchaine & Yovel, 2015). However, it is not clear yet to what degree those systems interact in facial identity recognition nor the specific contribution of emotions and of motion. Study 2 was aimed at clarifying these aspects: we expected to find a motion advantage similar to that observed in the literature. To do so, in the first experiment, we presented faces encoded through dynamic non-emotional expressions, dynamic rigid head motion, or as neutral and static, thus providing only featural information. Participants were asked to recognize them among distractors. Results showed that faces encoded through dynamic non-emotional facial expressions were easier to recognize, but only for poor recognizers. No relation with face recognition individual abilities was observed in the other two conditions. To better disentangle the role of the non-emotional expressions from the one of motion, we presented the same stimuli of Experiment 1 to a new sample of participants as a succession of static images interleaved by a grey mask with the aim of removing motion (Experiment 2). Results showed no difference in the recognition of faces among conditions. Thus, seeing faces without motion cancelled the facilitatory role of expressions on identity recognition for poor recognizers. Taken together, these results tell us that when the system used to elaborate facial identity is deficient, facial expressions become more helpful for recognition. In addition, motion seems to be crucial in this facilitation. Our results are in line with the supplemental information hypothesis (O'Toole, 2002). This hypothesis suggests that moving faces are more informative as dynamic identity signatures are processed in addition to the invariant features of faces. Coherently, we demonstrated that expression processing is preserved despite individuals' ability in face recognition. Moreover, this ability allows poor recognizers to use information

conveyed from non-rigid motion in addition to structural information for identity recognition, and this is the reason why they show facilitation.

Study 3 aimed at deepening our understanding of the neural bases subserving the relation between facial expression and identity processing. A brain area implicated in perceptual processing of facial expressions is the Superior Temporal Sulcus (Bernstein et al., 2018; Sliwinska & Pitcher, 2018). In addition, STS has been shown to be more selective for dynamic than static faces (Fox et al., 2019; Pitcher et al., 2011). Aside from STS, the pre-Supplementary Motor Area (preSMA) is implicated not only in the motor system, but also in the perception of facial expression (Johnston et al., 2013; Hardwick et al., 2018). It has been hypothesized that preSMA is part of an extended mirror neuron system that is supposed to participate in facial expressions recognition (Van der Gaag et al., 2007).

The described studies show evidence in favor of an implication of STS and preSMA in facial expression and facial motion processing. However, it remains to be seen what their interaction is with areas deputed to the analysis of face identity. In the literature, used expressions are always emotional while we wanted to investigate the sole role of expressions in this relation independently from the emotional content. That is why we decided to stimulate them with TMS in a task of matching faces encoded through non-emotional facial expressions, rigid head movement, or as static. Results of Study 3 show an active role of both STS and preSMA in identity recognition in comparison to Sham stimulation. When looking specifically at the sham stimulation condition, we replicated results from Study 2. Particularly, faces encoded through facial expressions were recognized equally despite individual face recognition abilities, while faces encoded as neutral were recognized better as recognition abilities increased. This result confirms that expression aid recognition in poor recognizers. Results concerning preSMA stimulation showed an increase in the recognition of faces encoded as neutral. Moreover, it

cancelled the facilitatory role of expressions in identity recognition for poor recognizers observed during the Sham stimulation. This suggests an active role of preSMA in expression simulation. STS, when stimulated, caused a decrease in the recognition of faces encoded through a rigid head movement. This result adds to existing evidence STS role in biological motion processing (Grossman et al., 2000). STS stimulation also caused an increase in recognition of faces encoded through facial expressions with respect to the Sham stimulation. This did not match the expectations of a decrease in expression processing. However, this might be due to the temporal dynamics of the facial recognition circuit (Pitcher et al., 2014), and studies considering this aspect might help in a clearer understanding of STS's role in identity recognition.

Study 4 aimed at understanding the interplay between identity and expression recognition in Parkinson's Disease. As a matter of fact, Parkinson's Disease patients have been shown to have deficits in recognizing emotional expressions (Argaud et al., 2018). This deficit has been explained by different non-mutually exclusive accounts. Among those, it has been linked to neural substrates of emotion processing, which may be impaired due to the disease (e.g., Wagenbreth et al., 2016). Another possible interpretation of the emotion recognition deficit in PD is that it might be linked to impairments in emotion expressivity (Prenger & MacDonald, 2018). This interpretation relies on the embodied simulation theory, according to which emotion recognition is enhanced by internal simulations of the observed expressions. These simulations occur when viewing an emotional facial expression which in turn activates the corresponding emotion in the viewer (Argaud et al., 2018). If the first explanation is true and PD patients are mostly impaired in processing emotions, this deficit should not impact the processing of non-emotional facial expressions. In addition, it should not impact the recognition of the identity of faces encoded through an expression. On the contrary, if the

second explanation is the predominant cause of the expression recognition deficit, we should observe the same also in the processing of non-emotional facial expressions. In addition, this deficit should reflect in the recognition of identity. Thus, studying such patients also helped us understand the role of mimicry of expressions in the recognition of expressions themselves and in recognizing identities. Results reveal that Parkinson's Disease patients do not show difficulties in recognizing static non-emotional facial expressions compared to Healthy Controls. Moreover, they do not show deficits in the recognition of identities of faces encoded through a dynamic non-emotional expression. These results point to an explanation of the deficit reported for PD in recognizing emotional expressions predominantly tied to emotion processing rather than to expression simulation. In addition, these results might hint at a reduced role of subcortical facial mimicry of expressions in facial identity processing. Facial mimicry might be important, but mostly when mediated by cortical structures.

Eventually, Study 5 was aimed at deepening our understanding of face recognition processes in patients with an acquired lesion. In particular, we aimed to understand the degree of separation between face processing and other perceptual functions, such as low/mid-level visual perception or recognition of categories other than faces. In fact, heterogeneous results have been documented in the literature concerning the separation between low/mid-level perception and face processing on one side and between object and face processing on the other (Monti et al., 2019; Barton et al., 2019). Thus, the debate of the specificity of face processing versus other functions is still open. Furthermore, we wanted to study the specific mechanisms involved in face processing, such as the relation between unfamiliar face perception and memory and the relation between matching two identities that are identical and discriminating that two identities are different. Additionally, we wanted to investigate the prevalence of face recognition impairments in our sample. In fact, acquired prosopagnosia is considered to be a

rather rare deficit. However, a worsening in face recognition is observed in a large proportion of cases (Valentine et al., 2006). To do so, data from 64 patients with posterior cerebral artery stroke was analyzed using the Single Case methodology to detect both deficits in single tests and dissociations between the different tests considered. Results of Study 5 reveal that face processing can be dissociated from other visual perceptual functions. Besides, within face processing, it is possible to dissociate between different functions, such as perception and memory or matching and discriminating. Eventually, we were able to diagnose three patients with acquired prosopagnosia, and in addition to that, half of the patients reported subjective worsening with faces after the accident, and one-third of them showed deficits in at least one of the diagnostic tests.

This thesis project contributes to our knowledge of the state of the art of rehabilitation of prosopagnosia. Rehabilitative studies are scarce and highlight the need of updated cognitive models of face perception. With this thesis, we were able to extend current models on the mechanisms behind face processing. First, we confirmed the advantage of dynamic facial expressions in identity recognition, even without emotional content. Moreover, we provided evidence that the Superior Temporal Sulcus and pre- Supplementary Motor Area are involved in identity recognition. This adds to the literature which only investigates their involvement specific for expression recognition. Moreover, we provided evidence that motor simulation of expressions, impaired in PD patients, does not influence facial identity recognition. Eventually, by studying patients with an acquired lesion, we confirmed that face processing is a complex function subserved by multiple overlapping systems. Nevertheless, those systems are at least partially separated from one another.

Given the results obtained within this thesis, we can hypothesize that prosopagnosic patients might benefit from a treatment based on the potentiation of facial expression processing. This

could be beneficial for developmental prosopagnosics as they are often reported to have spared expression processing. Thus, potentiating it might also improve identity recognition. Moreover, acquired prosopagnosics are often reported to have spared Superior Temporal Sulcus, which we discovered to be implicated in the recognition of faces encoded through an expression. Thus, they might benefit from a treatment based on expressions too.

The obtained results are of theoretical and clinical interest suggesting potential developments both for cognitive models and rehabilitative studies.

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