Perception of Facial Expressions of Emotion in Parkinson’s Disease

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ABSTRACT

Perceiving facial expressions of emotion to infer the emotional states of others is central to the regulation of individual and social behavior. Results from individual studies on whether those with Parkinson’s disease (PD) are impaired in perceiving facial expressions of emotion are mixed. Despite these mixed results, a recent meta-analysis (Gray & Tickle-Degnen, 2010) showed an overall averaged medium-sized impairment for all of the so-called “basic emotions”. Most of the individual studies that were included in this meta-analysis required participants to identify facial expressions of emotion by name and sampled only expressions of emotion that were of full-blown intensity. The ability to discriminate emotional expressions of graded intensity from neutral expressions and the ability to discriminate graded intensities of the same emotional expressions is unexplored in PD.

The determinants of impaired perception of facial expressions of emotion in PD are unknown. The results from the Gray and Tickle-Degnen meta-analysis (2010) indicated that perception of emotional expressions in PD was unrelated to depressive symptoms, medication status, disease progression, and perception of visual form. The studies reviewed by Gray and Tickle-Degnen (2010) measured perception of visual form with the Benton Face Recognition Test (BFRT; Benton, Sivan, Hamsher, Varney, & Spreen, 1994) or a similar test, all of which are traditionally regarded as measures of face recognition. Although the BFRT might tap the ability to perceive visual form, it has been shown to be sensitive only to large impairments in facial identity recognition (Duchaine & Weidenfeld, 2003). The studies in the meta-analysis measured disease severity with the Hoehn and Yahr
Staging Scale (Hoehn & Yahr, 1967), which has also been criticized as insensitive (Goetz et al., 2004).

A novel explanation for impaired perception of facial expressions of emotion in PD originates from the theory of embodied simulation (Goldman & Sripada, 2005, for a review) which states that we understand the emotional states of others by simulating the observed facial expressions of emotion. Based on this theory, systems with mirror-like properties that are engaged by voluntary control of facial musculature and simulation of observed emotional expressions might be compromised by the neurodegenerative processes of PD, thereby impairing both voluntary control of facial musculature and perception of emotional expressions. Support for this hypothesis will be shown by a positive link between voluntary facial musculature control and perception of emotional expressions in PD, which requires measurement of voluntary facial musculature control in PD. Previous studies measured voluntary control of facial musculature in PD mainly in the context of production of emotional expressions, and showed impairments. Only a few studies measured non-emotional facial movements in PD and found impairments, and there is only one item that measures facial expressivity on the gold-standard measure of disease severity, the Movement Disorders-sponsored revision of the Unified Parkinson’s Disease Rating Scales (MDS-UPDRS; Goetz et al., 2008). The previous work on voluntary control of facial musculature in PD only gives limited understanding to interchangeable terms such as hypomimia, facial masking, and facial bradykinesia that have been ascribed for decades as a symptom of PD (Bologna et al., 2013; Jancovic, 2008; Rinn, 1984).

The first aim of the research was to measure perception of facial expressions of emotion in PD, with particular emphasis on psychophysical measures of
discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions. These measures were first tested in a sample of healthy young adults (Marneweck, Loftus, & Hammond, 2013) and then in samples of healthy older adults and adults with PD. Measures of discriminating discrepant from two similar full-blown emotional expressions and recognizing full-blown emotional expressions were also taken. Results from three experiments showed a graded impairment on all measures of perception of emotional expression in PD. Performance by those with PD varied considerably on measures of perception of emotional expression, where some performed as well as some of the best-performing controls. The impairment in discriminating graded intensities of emotional from neutral expressions in PD was present at both brief and longer stimulus durations on emotion discrimination measures with both two-interval forced-choice and yes-no psychophysical procedures.

The second aim of the research was to measure some factors that might contribute to perception of facial expressions of emotion in PD, by examining its links with disease progression, perception of visual form (from faces, and from visual form more broadly), and voluntary control of facial musculature. The impaired ability to perceive facial expressions of emotion in PD was found to be a function of disease severity (as measured by MDS-UPDRS motor score; Goetz et al., 2008). After removing the variance shared with disease severity, perceiving emotional expressions correlated positively with perceiving visual form from faces, measured as discriminating graded changes in facial distinctiveness. This link is in line with recent propositions that perception of facial identity and emotional expressions share perceptual encoding mechanisms that facilitate recognition (Vuilleumier & Pourtois, 2007, for a review). Those with PD were also impaired in
perceiving visual forms more broadly, measured as the ability to discriminate radial frequency patterns with variations in amplitude modulation from perfect circles. Furthermore, there was a positive correlation between perceiving visual form and perceiving emotional expressions in PD, even after controlling for the variance shared with disease severity. From these findings, one contribution to impaired perception of emotional expressions in PD is a more general impairment in perception of visual form.

Voluntary control of facial musculature, measured by an adaptation of the Upper and Lower Face Apraxia Test (Bizzozero et al., 2000), was also shown to be impaired in PD. A revised error classification system showed that errors by those with PD were predominantly due to an impoverishment of facial movement with negligible differences between PD and control groups in errors due to loss of movement individuation, content errors (likened to ideational apraxia errors), and pauses before movement initiation. In line with embodied simulation accounts of perception of emotion, there was a positive correlation between voluntarily controlling facial musculature and perceiving facial expressions of emotion in PD, even after controlling for the variance shared with disease severity. Both voluntary facial musculature control and perception of emotional expressions might be impaired in PD by disruption of systems with mirror-like properties that are engaged by facial musculature control and simulation of observed expressions.

This research provides the first report of impaired discrimination of graded intensities of emotional expressions in PD, and replicates previous findings of impaired discrimination and recognition of full-blown emotional expressions. In addition, the findings from this research suggest a multifactorial contribution to impaired perception of facial expressions of emotion in PD. After removing disease
severity, the link between perception of emotional expression and visual form suggests that systems that are engaged by intermediate stages of visual form processing either receive poor input from peripheral visual systems due to impaired basic visual functions in PD (Archibald, Clarke, Mosimann, & Burn, 2009), or are themselves disrupted, thereby affecting perception of visual form that is not restricted to faces that convey emotion. In addition, and in line with embodied simulationist accounts of emotion perception, the link between perception of emotional expressions and voluntary control of facial musculature, after removing disease severity, suggests that systems with mirror-like properties that are engaged by voluntary control of facial musculature and simulation of observed expressions are also disrupted in PD, thereby affecting facial musculature control and perception of facial expression of emotion.
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STATEMENT OF CANDIDATE CONTRIBUTION

This thesis is submitted as a series of papers that have been prepared and submitted for publication. The four papers that were prepared and submitted for publication have been co-authored, and appear in Chapters 2, 3, 4, and 5. I declare that I was the primary author on all four papers.


Chapter 5: Marneweck, & Hammond (a revised version of this chapter was accepted for publication). Discriminating facial expressions of emotion and its link with perceiving visual form in Parkinson’s disease. Journal of the Neurological Sciences.

For each of these papers, I undertook the background research, stimuli and measures development, participant recruitment, data collection, data analyses, and writing of manuscript drafts, with guidance from Professor Geoff Hammond (Chapters 2, 3, 4, and 5), Associate Professor Andrea Loftus (Chapter 2), and Associate Professor
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The co-authors have given permission for these articles to be included as chapters in this thesis.

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CHAPTER 1: General Introduction

Facial expressions of emotion are an observable way in which we communicate our unobservable emotional states. To perceive facial expressions of emotion in order to infer the emotional states of others plays an important part in our daily lives. Perceiving accurately the facial expressions of others’ emotions contributes to social integration (Addington, Saeedi, & Addington, 2006), which in turn is essential for mental (Michelsen & Bildt, 2003) and physical well-being (Cohen, 2004). An impaired ability to perceive facial expressions of emotion can be detrimental to the regulation of individual and social behavior.

The Ability to Perceive Facial Expressions of Emotion in Parkinson’s Disease

The ability to perceive facial expressions of emotion is thought to draw on a number of neural structures, from occipital and posterior temporal visual cortices, amygdala, orbitofrontal cortex, somatosensory cortices, to the basal ganglia among others (see Adolphs, 2002a, for review). Given the involvement of the basal ganglia in emotional expression perception, many questioned whether Parkinson’s disease (PD), which is classically viewed as a disorder of movement caused by dopaminergic denervation in the basal ganglia, might also affect emotional expression perception. Findings from individual studies on whether persons with PD are impaired in their ability to perceive facial expressions of emotion are mixed. Some studies have reported impaired perception of facial expressions of emotion in PD (Ariatti, Benuzzi, & Nichelli, 2008; Assogna et al., 2010; Baggio et al., 2012; Beatty et al., 1989; Bediou et al., 2012; Blonder, Gur & Gur, 1989; Borod et al.,

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1 Although the class of abilities labeled emotion perception has sometimes been considered distinct from those labeled emotion recognition, I will use the term perception (or perceive) as it is often nowadays used (Adolphs, 2002b) to cover both domains.
1990; Buxton, MacDonald, & Tippett, 2013; Clark, Neargarder, & Cronin-Golomb, 2008; Clark, Neargarder, & Cronin-Golomb, 2010; Dujardin et al., 2004; Herrera, Cuetos, & Rodriguez-Ferreiro, 2011; Ibarretxe-Bilbao et al., 2009; Jacobs, Shuren, Bowers, & Heilman, 1995; Kan, Kawamura, Hasegawa, Mochizuki, & Nakamura, 2002; Lawrence, Goerendt, & Brooks, 2007; Narme, Bonnet, Dubois, & Chaby, 2011; Narme et al., 2013; Sprengelmeyer et al., 2003; Suzuki, Hoshino, Shigemasu, & Kawamura, 2006; Yip, Lee, Ho, Tsang, & Li, 2003) whereas others have found no such impairment (Adolphs, Schul, & Tranel, 1998; Cohen, Gagne, Hess, & Pourcher, 2010; Dewick, Hanley, Davies, Playfer, & Turnbull, 1991; Haeske-Dewick, 1996; Madeley, Ellis, & Mindham, 1995; Pell & Leonard, 2005; Tessitore et al., 2002; Ventura et al., 2012; Wieser et al., 2012). Despite mixed findings from individual studies, a recent meta-analysis by Gray and Tickle-Degnen (2010) showed an overall medium-sized impairment in PD in perceiving facial expressions of emotion for all of the so-called ‘basic’ emotions (anger, disgust, fear, happiness, sadness, and surprise). A similarly-sized impairment was shown by Gray and Tickle-Degnen (2010) in perception of emotion from voice in PD; however, this thesis will focus only on perception of facial expression of emotion. Gray and Tickle-Degnen (2010) also showed considerable variability between persons with PD in perception of emotional expressions, with some performing no different from healthy controls, reflected by wide confidence intervals for the medium-sized effect. This variability was unrelated to depression or medication status. Other factors that might contribute to variability in perception of facial expressions of emotion in PD are discussed in a later section.

Much like research on the ability to perceive emotional facial expressions in healthy and in clinical populations other than PD, studies in PD have focused on the
ability to recognize facial expressions of emotion, usually assessed as the ability to label or identify an emotion by name (retrieved either from memory or a list of emotions). The ability to recognize facial expressions of emotion has been measured with tests such as the Ekman 60 Faces (Young, Perrett, Calder, Sprengelmeyer, & Ekman, 2002), which requires selection from a list of six basic emotions the emotion that best fits the facial expression shown. The stimuli used in emotion recognition studies in PD generally depict emotions that are of full-blown intensity selected from validated stimulus sets (Pictures of Facial Affect, Ekman & Friesen, 1976; NimStim Face Stimulus Set, Tottenham et al., 2009). However, in everyday life, we express emotions at varying intensities, and only rarely do we express emotions at full-blown intensity (Motley & Camden, 1988). Perception of facial expressions of emotion that are less intense than full-blown expressions has been much less studied in PD. Three studies have investigated the ability to recognize graded intensities of facial expression of emotion in PD, and found patients to be impaired (Bediou et al., 2012; Buxton et al., 2013, Dujardin et al., 2004).

Despite the interest in recognizing facial expressions of emotion, the basic perceptual processes of discriminating emotional from neutral expressions and discriminating different intensities of emotional expressions remain unexplored in PD. Impaired abilities to discriminate emotional from neutral expressions and to discriminate subtle differences in the intensity at which emotions are expressed are likely to be as detrimental to social communication as an impaired ability to recognize emotional expressions by name. These basic perceptual abilities are fundamental for understanding others’ emotional states as they may assist or work in concert with more complex abilities of recognizing emotional expressions by name. Some studies have measured the basic perceptual ability to match visually presented
emotional expressions by selecting from a series of emotional expressions the one that best matches a target emotional expression (Ariatti et al., 2008; Blonder et al., 1989; Borod et al., 1990; Dewick et al., 1991; Haeske-Dewick, 1996; Jacobs et al., 1995; Pell & Leonard, 2005; Ventura et al., 2012). While accurately matching test with target expressions might require the ability to distinguish between emotions, it does not necessarily require the abilities to discriminate emotion from neutral or to discriminate differences in intensity; similar to studies on emotion recognition in PD, the stimuli used depict emotions that are of full-blown intensity. There are currently no sensitive psychophysical measures in PD of the basic perceptual abilities of discriminating graded intensities of emotional from neutral expressions and discriminating graded intensities of the same expressions of emotion from each other. Psychophysical methods with forced-choice procedures can offer objective, sensitive, and efficient measures of these basic perceptual abilities that are relatively free from response criterion effects (Pelli & Farrell, 1995).

Factors that might Contribute to Impaired Ability to Perceive Facial Expressions of Emotion in PD

Disease severity. The Gray and Tickle-Degnen (2010) meta-analysis showed that the variability in the ability to perceive facial expressions of emotion in PD was not related to the progression of the disease, as measured by the Hoehn and Yahr Staging Scale (Hoehn & Yahr, 1967). Although easy to administer and widely used, the Hoehn and Yahr Scale has been criticized for its lack of sensitivity. With only five scale options, a large range of impairment severity is collapsed together. The scale is also heavily weighted towards postural instability (Goetz et al., 2004) leaving other important components of PD unassessed (e.g. tremor, rigidity, bradykinesia). The Movement Disorders Society-sponsored revision of the Unified
Parkinson’s Disease Rating Scale (MDS-UPDRS) has been recommended as a more sensitive measure of the severity of the disease (Goetz et al., 2008). Since the meta-analysis by Gray and Tickle-Degnen (2010), there have been three studies (Baggio et al., 2012; Buxton et al., 2013; Wieser et al., 2012) on emotional expression recognition in PD that assessed disease severity with the UPDRS. Findings are mixed. Two of the studies reported moderate negative correlations between disease severity and recognition of graded (Buxton et al., 2013) and full-blown emotional expressions (Wieser et al., 2012), while one study reported little or no correlation between disease severity and recognition of full-blown emotional expressions (Baggio et al., 2012). The correlation coefficient reported by Baggio et al. (2012) needs to be interpreted with caution because most of the patients with PD were at an early stage of the disease progression with little range between patients in disease severity (UPDRS motor score $M = 16$, $SD = 8$). The Buxton group sampled a slightly greater range in disease severity ($M = 29$, $SD = 11$), which better estimates the population correlation coefficient, and found a moderate negative correlation between disease severity and emotional expression recognition in PD. Further study is needed of the possible contribution of disease severity to recognition and discrimination of emotional expressions in PD, by using the MDS-UPDRS with a range between patients in disease severity.

**The ability to perceive visual form.** The evidence for impairment in PD in a range of visual functions (visual acuity, contrast sensitivity, color and motion perception among others) is well-documented and summarized by Archibald, Clarke, Mosimann, and Burn (2009). The idea that impaired visual abilities in PD are likely to contribute to impaired perception of facial expressions of emotion is not novel (Adolphs et al., 1998; Sprengelmeyer et al., 2003). Nevertheless, a systematic
investigation is lacking of the potential link between the ability to perceive visual form and the ability to perceive facial expressions of emotion in PD. Gray and Tickle-Degnen (2010) concluded that an impaired ability to perceive emotional expressions was unrelated to the ability to perceive visual form, based on finding no difference between PD and control groups on the Benton Facial Recognition Test (BFRT; Benton, Sivan, Hamsher, Varney, & Spreen, 1994) and on other similar tests. The BFRT, which requires matching one of six test faces with a simultaneously presented target face, is traditionally viewed as a measure of face identity recognition. Although the BFRT might tap the ability to perceive visual form, it has been shown to be sensitive only to large impairments in facial identity recognition (Duchaine & Weidenfeld, 2003). Since the meta-analysis (Gray & Tickle-Degnen, 2010), one study found a positive correlation between the ability to perceive non-emotional information (discriminating spatial distance differences between facial features) and emotional information from faces (recognizing emotional expressions) in PD (Narme et al., 2011), suggesting that the ability to extract visual form from faces contributes to impaired recognition of emotional expressions in PD. Another possibility is that perceiving emotional and non-emotional information from faces are both impaired in PD by an even broader impairment in perceiving visual form that is not restricted to faces.

**Voluntary control of facial musculature.** The theory of embodied simulation states that we understand the facial expressions of emotion of others by simulating or enacting within ourselves the observed facial expression of emotion (Goldman & Sripada, 2005, for a review). This theory predicts that disruption of neural substrates that are engaged by production of emotional expressions will impair both production and perception of emotional expressions. Consistent with the
embodied simulation theory, there is evidence from lesion studies that impairments co-occur in the production and perception of facial expressions of emotion (Adolphs, Tranel, Damasio, & Damasio, 1994; Adolphs, Tranel, & Damasio, 2003; Calder, Keane, Manes, Antoun, & Young, 2000; Sprengelmeyer et al., 1999), suggesting that these processes might be subserved by the same neural substrates. This supposition is supported by the demonstration of neural systems with mirror-like properties that are activated during both production and observation of facial expressions of emotion in healthy adults (Carr, Iacobini, Dubeau, Mazziotta, & Lenzi, 2003; Hennenlotter et al., 2005; Jabbi, Swart, & Keysers, 2007; Leslie, Johnson-Frey, & Grafton, 2004; van der Gaag, Minderaa, & Keysers, 2007; Wicker et al., 2003). In PD, these neural systems might be compromised by the disease process, thereby impairing perception and production of emotional expressions. There is preliminary support for this hypothesis from studies that documented moderate positive correlations between producing and recognizing emotional expressions in PD, both of which were shown to be impaired (Benke, Bosch, & Andrew, 1998; Borod et al., 1990; Jacobs et al., 1995). In addition, there have been several reports of impaired voluntary and spontaneous production of facial expressions of emotion in PD (Bowers et al., 2006; Buck & Duffy, 1980; Jacobs et al., 1995; Katsikitis & Pilowsky, 1988; Marsili et al., in press; Piteain, Clemie, Gray, & Pentland, 1990; Simons, Ellgring, & Smith Pasqualini, 2003; Simons, Smith Pasqualini, Reddy, & Wood, 2004; Smith, Smith, & Ellgring, 1996), which might contribute to the “masked” expressionless face, an important clinical feature in PD (Bologna et al., 2013; Jancovic, 2008; Rinn, 1984). The co-occurrence of impaired abilities to produce and perceive emotional expressions in PD might be in part due to disruption of neural systems that are engaged by simulation of observed expressions.
Although the case for embodied simulation in contributing to emotion perception is a compelling one, the process by which this simulation occurs is less clear-cut. The favored models of embodied simulation for emotion perception that are overviewed by Goldman and Sripada (2005) can be categorized broadly into overt and covert accounts. Overt accounts specify that imitation of the observed expression by facial muscle activation produces the experience of that emotion within the observer, thereby facilitating perception of emotion. Inconsistent with this model, Hess and Blairy (2001) found that while facial mimicry occurred during emotional expression recognition, its occurrence was unrelated to accuracy in emotional expression recognition. Also inconsistent with this model, persons with congenital facial musculature paralysis were either intact in recognizing others’ emotional expressions (Bogart & Matsumoto, 2010; Keillor, Barrett, Crucian, Kortenkamp, & Heilman, 2002) or were not completely unable to do so (Bate, Cook, Mole, & Cole, 2013; Calder, Keane, Cole, Campbell, & Young, 2000). Goldman and Sripada (2005) suggest interpreting these findings with caution, as these patients, given their long-standing nature of facial paralysis, might have developed compensatory strategies that allow normal to near-normal performance on emotion recognition measures. Nevertheless, together these findings suggest that overt facial mimicry is not critical for perceiving facial expressions of emotion.

Covert accounts of embodied simulation propose that observing others’ emotional expressions directly activates the neural substrates that enable perception of emotion, and bypasses overt facial mimicry. Covert accounts are viewed as superior to overt accounts (Goldman & Sripada, 2005) in that they do not assume a causal role for facial musculature control in the perception process, and are therefore unthreatened by results from Bogart and Matsumoto (2010), Keillor et al. (2002),
Bate et al. (2013), and Calder et al. (2000). Therefore, in line with a covert account of embodied simulation, systems that are engaged by simulation of observed expressions might be compromised in PD, thereby contributing to impaired perception of facial expressions of emotion.

The studies that have investigated systems with mirror-like properties during observation and production of emotional expressions (Carr et al., 2003; Hennenlotter et al., 2005; Jabbi et al., 2007; Leslie et al., 2004; van der Gaag et al., 2007; Wicker et al., 2003) found this mirroring activity in multiple regions, from the ventrolateral premotor cortex and pars opercularis of the inferior frontal gyrus, to the anterior cingulate, insula, amygdala, and somatosensory cortical areas. It has been suggested that some of these regions are engaged by experience and expression of emotion (e.g. insula for disgust: Jabbi, Bastiaansen, & Keysers, 2008; Phillips et al., 1997; Small et al., 1999, 2003; insula for other emotions: Carr et al., 2003; Hennenlotter et al., 2005; Jabbi et al., 2007; van der Gaag et al., 2007), while others are known to be involved in voluntary facial movements more generally, regardless of its intended meaning (e.g. premotor cortex: Morecraft, Stilwell-Morecraft, & Rossing, 2004). In PD, it might be that these systems with mirror-like properties that are involved in voluntary control of facial musculature (regardless of its intended meaning) are disrupted, thereby contributing to both impaired voluntary control of facial musculature and perception of emotional expressions. One way to assess this hypothesis is by behavioral examination of the link between voluntary facial musculature control and perception of emotional expressions in PD; both of these abilities might be impaired in PD partly as a consequence of disruption to systems with mirror-like properties that are engaged by simulation of observed expressions and motor control.
To assess an embodied simulationist account of the ability to perceive emotional expressions in PD requires measurement of facial musculature control. Facial musculature control in PD has predominantly been studied in the context of facial expression of emotion. Spontaneous facial expressions of emotion were shown to be impaired in PD during conversations and in response to emotionally evocative stimuli (Buck & Duffy, 1980; Katsikitis & Pilowsky, 1988; Pitcairn et al., 1990; Simons et al., 2003; Simons et al., 2004; Smith et al., 1996). These studies supported an earlier influential proposal by Rinn (1984) that the “masked” expressionless face in PD originates from reduced spontaneity in facial expressions of emotion, while leaving voluntary facial expressions of emotion intact. However, inconsistent with Rinn’s proposal, voluntary facial expressions of emotion in response to verbal command (e.g. “look happy”) were also shown to be impaired in PD (Borod et al., 1990; Bowers et al., 2006; Jacobs et al., 1995; Marsili et al., in press; Simons et al., 2003; Simons et al., 2004); these studies scored the quality and intensity of reproduced emotional expressions either by blind raters using Likert Scales, the Facial Action Coding System (FACS; Ekman & Friesen, 1978) which codes observable facial movement as “action units” or action unit combinations, or by sophisticated digital imaging analyses (Bowers et al., 2006; Marsili et al., in press.) That voluntary emotional expressions appear to also be impaired in PD gave rise to a more recent proposition (Bowers et al., 2006) that the impression of a mask-like face is not only a result of impaired spontaneous emotional expression, but also of impaired voluntary emotional expression.

It is reasonable to suppose that non-emotional facial movement is also impaired in PD, thereby contributing with diminished emotional expressions to facial masking. Nevertheless, non-emotional facial movement has been less systematically
explored than emotional facial movement. Studies on non-emotional facial movement in PD have been limited to specific facial areas, with impairment evidenced in voluntary, spontaneous, and reflex blinking rate and amplitude (Agostino et al., 2008; Korosec, Zidar, Reits, Evinger, & Vanderwerf, 2006), and jaw and upper lip movement amplitude during speech (Connor, Abbs, Cole, & Gracco, 1989). Simons et al. (2003; 2004) is the only group that has measured, using the FACS (Ekman & Friesen, 1978), the ability to voluntarily reproduce a limited set of non-emotional facial movements and found these movements to be impaired in PD. Although the FACS is sensitive to compare reproduced action unit patterns with requested action unit patterns, it is not always a practical tool available to clinicians and researchers. FACS certification requires extensive and costly training. The only other measure of voluntary control of facial musculature without emotional content in PD is one item on the MDS-UPDRS, on which patients are rated for their facial expressivity when sitting in front of the examiner for 10s without moving or speaking. Although this item can pick up some loss of voluntary control of facial musculature that is present when not moving or speaking (e.g. reduced control in keeping mouth closed and reduced blinking), the item does not measure the ability to control a range of facial muscles. In sum, there is no simple and systematic measure of voluntary control of facial musculature in PD, which leaves our understanding of this ability (and facial masking) limited to impaired emotional expression, and assessed generally by subjective judgment in clinical settings.

**Key Aims**

This project had two key aims. The first aim was to measure the ability to perceive facial expressions of emotion in PD, with an emphasis on discriminating emotional expressions of graded intensity from neutral expressions and
discriminating different intensities of the same emotional expressions. These basic perceptual processes are unexplored in PD, and the use of graded intensities of emotional expressions is also lacking. A systematic investigation of discrimination of emotional expressions of graded emotional intensity is required for a more complete understanding of perception of emotional expressions in PD. In addition, the determinants of impaired perception of emotional expressions in PD are currently not well understood, justifying the second aim: to explore some of the factors that might contribute to impaired perception of facial expressions of emotion in PD, by examining its links with disease severity, voluntary control of facial musculature (testing an embodied simulationist account), and the ability to perceive visual form (from faces, and from visual form more broadly). Four experiments were conducted to investigate these aims. Experiment 1 was conducted with healthy young adults, and Experiments 2, 3, and 4 were conducted with samples of patients with PD and healthy controls. Each aim, and how it is addressed in each experiment, will be discussed in turn.

**Aim 1: To measure the ability to perceive facial expressions of emotion in PD.** Psychophysical measures with two-interval forced-choice (2IFC) procedures of discriminating graded intensities of emotional from neutral expressions and of discriminating graded intensities of the same emotional expressions were used for four of the “basic” emotions, anger, disgust, happiness, and sadness. The measures were first tested in a sample of healthy young adults in Experiment 1. These measures were then used on samples of PD patients and healthy controls over the course of three experiments (Experiment 2, Experiment 3, and Experiment 4), which gave the opportunity to test the reliability of findings. With the aim of replicating previous findings, Experiment 3 also investigated recognition of full-blown
emotional expressions and discrimination of discrepant from similar full-blown emotional expressions. Experiment 4 compared PD and control groups on the psychophysical measure of discriminating graded intensities of anger from neutral expressions with longer stimulus durations (to reduce any effects of slower visual processing times in PD) and on an anger discrimination measure with a single-stimulus yes-no procedure (to reduce the working memory demands imposed by sequential stimuli). Measures of cognitive functioning and depressive symptoms were included in all three experiments on PD and control groups to assess their potential contribution to perception of facial expressions of emotion.

**Aim 2: To explore some factors that might contribute to the ability to perceive facial expression of emotion in PD.** Links were investigated between perceiving facial expressions of emotion and disease severity, the ability to voluntarily control facial musculature (to test an embodied simulationist account), and the ability to perceive visual form (from faces, and from visual form more broadly).

**Disease severity.** The gold-standard measure for quantification of disease severity, the MDS-UPDRS (Goetz et al., 2008), was administered to patients in all three experiments (Experiments 2, 3, and 4) to investigate the contribution of disease severity to the ability to perceive emotional expressions in PD.

**Voluntary control of facial musculature.** An embodied simulationist account of emotion perception in PD was tested by examining the link between voluntary facial musculature control and the ability to perceive emotional expressions in PD in Experiment 2 and Experiment 3. Voluntary facial musculature control was measured with an adaptation of the Upper and Lower Face Apraxia Test (Bizzozero et al., 2000), a test of the ability to control a range of upper and lower
non-emotional facial movements. Although the original test was devised to measure apraxia, it serves also as a measure of voluntary control of facial musculature.

**The ability to perceive visual form.** To investigate perception of visual form that is specific to faces and its link with perception of emotional expressions in PD, Experiment 3 measured the ability to discriminate graded changes in facial distinctiveness and the ability to recognize facial identity in PD patients and controls. Facial distinctiveness has been defined by the extent to which a face stands out in a crowd (Bruce & Young, 2012, p. 274). Distinctiveness is known to predict identity recognition (Bartlett, Hurry, & Thorley, 1984; Light, Kayra-Stuart, & Hollander, 1979; Vokey & Read, 1992), and has been included as an important dimension in “face-space” models of identity recognition (Busey, 1998; Valentine, 1991).

To investigate perception of visual form that is not restricted to faces and its link with perception of emotional expressions in PD, Experiment 4 measured the ability to discriminate radial frequency (RF) patterns with variations in amplitude modulation from perfect circles in PD patients and controls. RF patterns are a family of smooth closed shapes that vary from one another and each from a perfect circle in a clearly defined way: different patterns can be created by modulating the radial frequency (the number of lobes), the amplitude (sharpness or depth of the lobe), and the orientation (the direction of the lobe). The use of RF patterns is considered a powerful tool to examine the ability to perceive visual forms (Loffler, 2008, for a review).

**General Methods**

**Participants**

Healthy young adults were recruited for Experiment 1 on the development of psychophysical measures of emotion discrimination. Patients with PD were
diagnosed by local neurologists and recruited from the Edith Cowan University Parkinson’s Centre and from Parkinson’s Western Australia newsletters. Healthy older adult controls were recruited from the wider community. All participants gave written informed consent to the procedures of the experiments.

**Demographic and clinical characteristics.** For the experiments with patients and controls, age, sex, and reported number of years of formal education were recorded. General cognitive functioning was measured using the Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005) and depressive symptoms were measured using the Geriatric Depression Scale (GDS; Sheikh & Yesavage, 1986). For patients with PD, the severity of the disease was measured with the MDS-UPDRS motor score (Goetz et al., 2008) and the number of years since diagnosis was recorded. Dopamine replacement medication type and dose were converted and reported as the levodopa dose equivalent (Tomlinson et al., 2010).

**Psychophysical Measures**

**Discriminating emotional expressions of graded intensities.** Measures were taken of the ability to discriminate graded intensities of emotional from neutral expressions and the ability to discriminate graded intensities of the same emotional expressions, of four of the “basic” emotions, anger, disgust, happiness, and sadness. Graded intensities of emotional expressions were created by morphing photographs of models expressing full-blown emotions (Tottenham et al., 2009) with their neutral expressions. For Experiments 1, 2, and 3, the psychophysical measures used a 2IFC procedure with the Method of Constant stimuli with 200-ms stimulus durations and a 200-ms inter-stimulus interval. For Experiment 4, the ability to discriminate anger from neutral expressions was measured with 2IFC and yes-no procedures (to reduce the working memory demands imposed by sequential stimuli) with stimulus
Discriminating graded intensities of facial distinctiveness. Faces that varied in distinctiveness were created by morphing photographs of models (showing neutral expressions), which were rated by graduate students as highly distinctive, each with an average, typical-looking face of the same sex (created by morphing 26 males and 26 females respectively; Rhodes et al., 2011). The ability to discriminate graded intensities of facial distinctiveness was measured using a 2IFC procedure with the Method of Constant Stimuli. On each trial, participants indicated which of the two sequentially presented faces was more distinctive. The stimulus size, viewing distance, stimulus duration, and inter-stimulus interval were matched to the measures of discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions.

Discriminating RF patterns of varying amplitude modulations. The ability to discriminate RF patterns with varying amplitude modulations from perfect circles was measured using a 2IFC procedure with the Method of Constant Stimuli with 200-ms stimulus durations. The stimulus size, viewing distance, and inter-stimulus interval were matched to the measures of discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions.

Measures of Perceiving Full-Blown Emotional Expressions and Facial Identity

The ability to discriminate the discrepant emotional facial expression from two expressions of the same emotion, all of full-blown intensity (Karolinska Directed Emotional Faces database; Lundqvist, Flykt, & Öhman, 1998), was measured with a three-alternative forced-choice procedure (Palermo, O’Connor,
Davis, Irons, & McKone, 2013). On each trial, three faces, two expressing the same emotion and third expressing a different emotion, were presented simultaneously and side-by-side. Participants signalled which of the three faces expressed the discrepant emotion.

The ability to recognize full-blown emotional expressions (Lundqvist et al., 1998) was measured with a six-alternative forced-choice procedure that required selection of one of six emotion labels that best suited the facial expression of emotion shown (Palermo et al., 2013).

The ability to recognize identity from faces was measured with the established measure, the Cambridge Face Memory Test (CFMT; Duchaine & Nakayama, 2006).

Measure of Voluntary Control of Facial Musculature

Voluntary control of facial musculature was measured with an adaptation of the Upper and Lower Face Apraxia Test (Bizzozero et al., 2000), a test of the ability to make a range of upper and lower non-emotional facial movements. The adapted version included standardized instructions and demonstrations in video format and a refined classification system for errors that was based on observations of typical errors made by patients and controls. Data were collected from three enrolment phases (each of which formed part of the experiments on patients with PD and controls).

Data Analysis

As recommended by Cumming, Fidler, Kalinowsky, and Lai (2012) in their paper on statistical recommendations for the American Psychological Association, measures of effect size (Hedges’ g) are reported to quantify overall differences between control and patient groups, and Pearson correlation coefficients and 95%
confidence intervals are reported for correlational analyses, to provide a more informative analysis of empirical results than statistical significance testing. However, statistical significance testing have been added to manuscripts that form part of the experimental chapters of this thesis in the event that previous reviewers deemed it necessary for the purposes of publication.

**Chapter Outlines**

The next chapter, Chapter 2, describes the use of the psychophysical measures of discriminating graded intensities of emotional from neutral expressions and discriminating graded intensities of the same emotional expressions in healthy young adults (Experiment 1). Chapter 3 describes the adaptation of the Upper and Lower Face Apraxia Test (Bizzozero et al., 2000) that also specifies the changes in voluntary control of facial musculature that occur in PD. Chapter 4 describes Experiment 2 and Experiment 3 that investigated in patients with PD and healthy controls the ability to perceive emotional expressions, measured as discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions, discriminating discrepant from similar full-blown emotional expressions, and recognizing full-blown emotional expressions. The links between perception of emotional expressions and voluntary facial musculature control (Experiments 2 and 3) and perception of visual form that is specific to faces (measured as discriminating differences in facial distinctiveness and as recognizing facial identity on the CFMT; Experiment 3) in PD were also investigated. Chapter 5 describes Experiment 4 on patients with PD and healthy controls, which investigated the link between the abilities to perceive visual form (measured as discriminating RF patterns with varying amplitude modulations from perfect circles) and to perceive emotional expressions of graded intensities from neutral expressions in PD.
Experiment 4 also tested any potential effects of brief and sequential stimuli presentations on measures of discriminating graded intensities of emotional from neutral expressions. Chapter 6 gives an overview and interpretation of the findings from this research, and considerations for future research.


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Small, D. M., Zald, D. H., Jones-Gotman, M., Zatorre, R. J., Pardo, J. V., Frey, S.,


CHAPTER 2: Psychophysical Measures of Sensitivity to Facial Expression of Emotion

Abstract

We report the development of two simple, objective, psychophysical measures of the ability to discriminate facial expressions of emotion that vary in intensity from a neutral facial expression and to discriminate between varying intensities of emotional facial expression. The stimuli were created by morphing photographs of models expressing four basic emotions, anger, disgust, happiness and sadness with neutral expressions. Psychometric functions were obtained for 15 healthy young adults using the Method of Constant Stimuli with a two-interval forced-choice procedure. Individual data points were fitted by Quick functions for each task and each emotion, allowing estimates of absolute thresholds and slopes. The tasks give objective and sensitive measures of the basic perceptual abilities required for perceiving and interpreting emotional facial expressions.
Introduction

The ability to perceive the facial expressions of emotion of others is central to the regulation of social behavior. Emotion perception has been studied in different populations including children (Gao & Maurer, 2009), young adult (Elfenbein & Ambady, 2002), and the aging (Sullivan & Ruffman, 2004), and in both healthy and clinical populations (Assogna, Pontieri, Caltagirone, & Spalletta, 2008; Harms, Martin, & Wallace, 2010; Hippolyte, Barisnikov, & Van der Linden, 2008; Hofer et al., 2009, Montagne, Kessels, De Haan, & Perrett, 2007). Much of this research has focused on the ability to recognize facial expressions of emotion, usually assessed as the ability to identify specific emotions by name (retrieved either from memory or a list of names) or to distinguish different expressions of emotion (Bell et al., 2011; Calder, Keane, Cole, Campbell, & Young, 2000; Clark, Neargarder, & Cronin-Golomb, 2008; Young & Hugenberg, 2010). The Ekman 60 Faces Test (Young, Perrett, Calder, Sprengelmeyer, & Ekman, 2002) exemplifies a recognition test that requires participants to select from a list of six basic emotions the emotion that best describes the facial expression shown. The stimuli used in identification studies generally depict full-blown emotional facial expressions selected from validated stimulus sets (Matsumoto & Ekman, 1988; Tottenham et al., 2009). In everyday life, however, emotions are generally expressed with graded intensity. There has been some interest in the ability to identify graded intensities of facial expressions of emotion with rating scales (Dujardin et al., 2004). There has also been interest in using dynamic morphed stimuli (from an emotional face to a neutral face, and from one emotion to another) to measure the point at which an emotion becomes apparent from a neutral expression and at which a change in emotion is detected (Fiorentini & Viviani, 2011; Montagne et al., 2007; Niedenthal, Halberstadt, Margolin, & Innes-
The ability to distinguish between confusable expressions has been assessed with tests such as the Emotion Hexagon Test (Young et al., 2002), which requires participants to name the emotional term that best describes images composed of graded blends of two confusable emotional expressions (such as happiness and surprise and disgust and anger).

Despite the interest in the ability to identify and to distinguish facial expressions of emotion, the basic perceptual abilities that may assist, or work in concert, with the more complex processes of identifying a specific emotion by name and distinguishing between emotions remain less explored. Measurements of the perceptual processes target the ability to discriminate speedily the visual properties of facial expressions that indicate the emotion and its intensity (Adolphs, 2002). In contrast, the more complex processes place demands on verbal processes, including vocabulary (Adolphs, 2002), and on working memory (Phillips, Channon, Tunstall, Hedenstrom, & Lyons, 2008). There are currently no sensitive, psychophysical measures of the fundamental perceptual abilities of discriminating emotional from neutral facial expressions and discriminating varying intensities of facial expressions of emotion. Psychophysical methods with forced-choice procedures offer objective, sensitive, and efficient measures of perceptual processes that are relatively free from response criterion effects. The aim of this study was to determine the usefulness of psychophysical measures of the ability to discriminate emotional from neutral expressions and to discriminate between graded intensities of emotional expression for four commonly expressed emotions, anger, disgust, happiness, and sadness. The emphasis of the paper is on the demonstration of the method and the usefulness of the general approach.
Methods

Participants

Fifteen healthy young adult volunteers (nine females) with no reported neurological impairments were tested. Their ages ranged from 22 to 27 years. Two other volunteers participated in a preliminary phase to select the stimuli. The procedures were approved by the Institutional Ethics Committee and all participants gave written informed consent.

Materials and Procedures

Development of the stimulus set. We selected colored photographs of models expressing emotions from a validated set (the NimStim Face Stimulus Set; Tottenham et al., 2009) for each of four basic emotions, anger, disgust, happiness, and sadness. The six Caucasian models (three male, three female) that produced the highest agreement of their intended expressions in a validation study (Tottenham et al., 2009) were used. Neutral expressions of the models (rated as an expressive intensity of zero) were morphed with their full-blown emotional expressions (rated as an expressive intensity of 100%) in steps of 5% with Norrkross MorphX software (Wennerberg, 1997) to create graded intensities of expression for each emotion. The Norrkross software is a freeware, open-source program that allows morphing of two photographic images creating a prototypical facial image from exemplars using a sophisticated morphing algorithm that implements the principles described by Benson and Perrett (1993, as cited by Pearson & Adamson, 2004). The software is widely used in research (Akrami, Liu, Treves, & Jagadeesh, 2009; Ishikawa & Mogi, 2011; Liu & Jagadeesh, 2008; Pearson & Adamson, 2004; Vida & Mondloch, 2009). Similar to the work of Pearson and Adamson (2004), an average of 75 key points were allocated to identify points of similarity between the faces, with more points
assigned around areas of greater change with increasing emotional intensity, such as around the pupils, eyelids, eyebrows, lips, and nose. The software also allows for the points to be connected with Bezier curves to define the warping region for further precision (Pearson & Adamson, 2004). Expressions of anger and happiness, which are typically expressed with an open mouth, were morphed with open-mouth neutral expressions, and expressions of disgust and sadness, which are typically expressed with a closed mouth, were morphed with closed-mouth neutral expressions. Two models were selected for each emotion to ensure that judgments were not made only of the specific features of a single model. Figure 1 shows an example of the morphed stimuli from 10% to 80% expressivity of disgust.

Figure 1. Morphed stimuli of a neutral expression (defined as 0% expressivity) and a full-blown expression of disgust (defined as 100% expressivity). The eight images vary in 10% equally spaced increments starting from 10% to 80% expressivity of disgust.

**Experimental procedure.** The ability to discriminate an emotional from a neutral expression and to discriminate between different intensities of expression
of the same emotion was measured with two tasks (with two variants of the second) using a two-interval forced-choice layout with the Method of Constant Stimuli. On each trial, two faces of the same model were presented successively on a computer screen for 200 ms with a 200-ms blank inter-stimulus interval. The 200-ms inter-stimulus interval was sufficiently long to prevent transformational apparent motion from the first to the second image (Kawahara, Yokosawa, Nishida, & Sato, 1996). The face stimuli were 6.8 cm high and 5.4 cm wide subtending visual angles of 6.6° and 5.2° at a viewing distance of 59 cm. Response time was unlimited and followed by a 1-s blank window (no feedback given) before the next trial commenced.

In the task that required discrimination of a neutral from an emotional expression, the face with the neutral expression appeared randomly in either the first or the second interval and a face expressing one of seven levels of intensity of the tested emotion appeared in the other interval. The seven intensity levels ranged from 5% to 35% of the full-blown expression in equally spaced increments. The stimulus levels and range were chosen using pilot data from two participants to obtain unbiased and precise absolute thresholds (Swanson & Birch, 1992). On each trial, participants were required to signal which interval contained the face expressing the emotion by clicking either the left or right button on a mouse for the first or second interval respectively. The stimulus pairs were presented in randomized blocks of 14 trials (seven intensities X two models). There were 20 blocks resulting in a total of 280 trials, giving 40 trials for each intensity increment. The task was repeated for each of the four emotions with a 2-min break between each.
In the task that required discrimination between different intensities of the same emotion, two faces expressing different intensities were randomly assigned to the two intervals. The two facial expressions varied in five intensity steps from 5% to 25% in equally spaced increments, again chosen using pilot data (Swanson & Birch, 1992). Participants were required to signal which interval contained the face expressing the higher intensity by clicking either the left or right mouse button for the first or second observation interval respectively. Two variants of this task were run in the same session: one sampled expression intensities from a low intensity range (from 10% to 50% of the full-blown expression) and the other from a high intensity range (from 50% to 90% of the full-blown emotion). The stimuli used to define each intensity difference for each of these two sub-tasks are shown in Table 1; each intensity difference was defined by four different intensity pairs to establish generality of discriminating intensity differences across the range of intensities. The stimulus pairs were presented in randomized blocks of 40 trials, with one presentation of each of the four definitions of the five intensity differences (see Table 1) for each of two models in each block. There were five blocks for a total of 200 trials, giving 40 trials for each intensity difference.
Table 1
Pairings of the different emotional intensities used to define each intensity difference for the task requiring discrimination of graded intensities for the low intensity and high intensity ranges.

<table>
<thead>
<tr>
<th>Intensity range</th>
<th>5%</th>
<th>10%</th>
<th>15%</th>
<th>20%</th>
<th>25%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Low range</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pair 1</td>
<td>10,15</td>
<td>10,20</td>
<td>10,25</td>
<td>10,30</td>
<td>10,35</td>
</tr>
<tr>
<td>Pair 2</td>
<td>15,20</td>
<td>15,25</td>
<td>15,30</td>
<td>15,35</td>
<td>15,40</td>
</tr>
<tr>
<td>Pair 3</td>
<td>20,25</td>
<td>20,30</td>
<td>20,35</td>
<td>20,40</td>
<td>20,45</td>
</tr>
<tr>
<td>Pair 4</td>
<td>25,30</td>
<td>25,35</td>
<td>25,40</td>
<td>25,45</td>
<td>25,50</td>
</tr>
<tr>
<td><strong>High range</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pair 1</td>
<td>50,55</td>
<td>50,60</td>
<td>50,65</td>
<td>50,70</td>
<td>50,75</td>
</tr>
<tr>
<td>Pair 2</td>
<td>55,60</td>
<td>55,65</td>
<td>55,70</td>
<td>55,75</td>
<td>55,80</td>
</tr>
<tr>
<td>Pair 3</td>
<td>60,65</td>
<td>60,70</td>
<td>60,75</td>
<td>60,80</td>
<td>60,85</td>
</tr>
<tr>
<td>Pair 4</td>
<td>65,70</td>
<td>65,75</td>
<td>65,80</td>
<td>65,85</td>
<td>65,90</td>
</tr>
</tbody>
</table>

The two tasks were done in counterbalanced order in two separate testing sessions separated by at least 24 h. The presentation order of emotions within each task followed a Latin Square sequence and the order of the two sub-tasks was counterbalanced. Participants read standardized instructions before each task and were given five practice trials immediately before each task began.

**Data Analysis**

Individual data obtained in each of the three determinations (discriminating emotional from neutral expressions, and discriminating different intensities of emotion in both the low and high intensity range) for each of the four emotions (anger, disgust, happiness, and sadness) were fitted with Quick functions (Gilchrist, Jerwood, & Ismaiel, 2005; Quick, 1974) constrained to begin at 50%. The functions generally fitted the individual data well; the median $R^2$ values for the four emotions ranged from .89 to .96 when discriminating emotional from neutral expressions, from .89 to .95 when discriminating different low-range intensities of emotion, and from .83 to .92 when discriminating different high-range intensities of emotion. The
functions fitted to the individual and mean data for each of the three psychophysical tasks for each of the four emotions are shown in the figures. The absolute thresholds were taken as the intensity increment from neutral or the intensity differentiation that produced 75% correct performance. Summary descriptive statistics of thresholds and slopes are shown in the tables. Thresholds and slopes could not be obtained in 11 of the 180 individual determinations because the range of constant stimuli used did not capture a complete psychometric function or because the fit to the individual data points was poor. Participants were excluded from the statistical analyses of between-emotion effects on threshold and slope if one or more determinations were missing. Although the purpose of this report is to show the practical applicability of the method, and not the implications of the results themselves, we report one-way repeated-measures analyses of variance on thresholds and slopes for those participants with complete data sets. The Greenhouse Geisser correction was applied to the data where the sphericity assumption was violated. The statistical analyses serve to show the sensitivity of the measures to the emotional expression tested and are not used to make any claims about the nature of processing expressions of the different emotions.

Results

**Discriminating Emotional from Neutral Expressions**

Figure 2 shows the functions fitted to individual and mean data points. All functions increased monotonically with increasing differentiation of the expressive from the neutral face for each of the four emotions, with individual variation in level and slope. Mean absolute thresholds and slopes with the sample sizes for each are shown in Table 2. Thresholds ranged from about 8% to about 17% and were lowest for expressions of happiness and highest for expressions of sadness. Analysis of 11
thresholds of complete data sets showed a significant main effect of Emotion \((F (3, 30) = 16.10, p < .001, \eta^2_p = .62)\). There was no effect of emotion for slopes of 11 data sets, \((F (1.49, 14.86) = 0.90, p > .05, \eta^2_p = .08)\).

Figure 2. Quick functions fitted to individual (gray lines) and mean accuracy (black symbols and lines) when discriminating emotion from neutral expressions for each of the four emotions.

Table 2

<table>
<thead>
<tr>
<th></th>
<th>Anger</th>
<th>Disgust</th>
<th>Happiness</th>
<th>Sadness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Threshold</td>
<td>10.5</td>
<td>13.6</td>
<td>7.8</td>
<td>16.9</td>
</tr>
<tr>
<td></td>
<td>(3.6)</td>
<td>(3.0)</td>
<td>(1.9)</td>
<td>(5.0)</td>
</tr>
<tr>
<td>Slope</td>
<td>2.2</td>
<td>2.4</td>
<td>1.5</td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td>(2.0)</td>
<td>(1.0)</td>
<td>(0.6)</td>
<td>(0.8)</td>
</tr>
<tr>
<td>N</td>
<td>15</td>
<td>14</td>
<td>15</td>
<td>12</td>
</tr>
</tbody>
</table>

Note. Satisfactory curve fits were not obtained for four participants, one in the Disgust condition and three in the Sadness condition.
Discriminating Different Intensities of Emotional Expression

Figure 3 shows the functions fitted to individual and mean data points. The mean functions increased with increasing intensity differentiation of the emotional expressions in both intensity ranges, with individual variation again evident. Mean thresholds and slopes derived from the fitted functions with the sample sizes for each are shown in Table 3. Absolute thresholds for each emotional expression were similar in both intensity ranges, and, consistent with the previous measure, were lowest for discriminating different intensities of happiness and highest for discriminating different intensities of sadness. The main effect of Emotion was significant from analyses of 12 data sets for the low intensity range ($F(3, 33) = 22.70, p < .001, \eta^2_p = .67$) and 11 data sets for high intensity range ($F(1.39, 13.87) = 6.88, p = .01, \eta^2_p = .41$). The slopes for each emotional expression were similar in the two intensity ranges, and were similar for the emotional expressions in each of the intensity ranges (low intensity range, $F(3, 33) = 0.29, p > .05, \eta^2_p = .02$; high intensity range, $F(1.35, 13.50) = 2.37, p = .14, \eta^2_p = .19$).
Figure 3. Quick functions fitted to individual (gray lines) and mean accuracy (black symbols and lines) when discriminating different intensities of emotional expression for each intensity range (left panels: low intensity range; right panels: high intensity range) for each of the four emotions.
Table 3

*Mean absolute thresholds and slopes for discriminating between varying intensities of the four emotions for the low- and high intensity ranges. The N for each emotion in each range is shown and the standard deviations are in parentheses.*

<table>
<thead>
<tr>
<th></th>
<th>Anger</th>
<th>Disgust</th>
<th>Happiness</th>
<th>Sadness</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Low Range</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Threshold</td>
<td>7.4 (2.8)</td>
<td>7.3 (2.3)</td>
<td>6.3 (2.5)</td>
<td>12.7 (4.1)</td>
</tr>
<tr>
<td>Slope</td>
<td>1.5 (0.5)</td>
<td>1.5 (1.0)</td>
<td>1.5 (0.6)</td>
<td>1.7 (0.4)</td>
</tr>
<tr>
<td>N</td>
<td>13</td>
<td>15</td>
<td>15</td>
<td>14</td>
</tr>
<tr>
<td><strong>High Range</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Threshold</td>
<td>7.1 (1.9)</td>
<td>8.9 (5.5)</td>
<td>6.1 (2.3)</td>
<td>11.4 (4.3)</td>
</tr>
<tr>
<td>Slope</td>
<td>1.0 (0.4)</td>
<td>1.2 (0.5)</td>
<td>1.3 (0.7)</td>
<td>1.9 (1.5)</td>
</tr>
<tr>
<td>N</td>
<td>14</td>
<td>13</td>
<td>15</td>
<td>14</td>
</tr>
</tbody>
</table>

*Note.* Satisfactory curve fits were not obtained for three participants in the low-range variant of the task, two in the Anger condition and one in the Sadness condition and for four participants in the high-range variant of the task, one in the Anger condition, two in the Disgust condition, and one in the Sadness condition.

**Discussion**

The results show that the psychophysical method described here is suitable for measuring sensitivity to facial expressions of four basic emotions in healthy young adults. The mean data in each of the measures were well fitted by the Quick functions, giving estimates of threshold and slope for each expression in each of the three tasks. The data also reveal individual differences in discrimination performance in the sample of healthy young adults, presumably reflecting in part individual differences in sensitivity to gradations in intensity of facial expressions of emotion. Psychometric functions could not be fitted in some cases in which performance was at or above 75% correct at the smallest constant stimulus value. This issue is easily addressed in future work by increasing the range of constant stimulus values by
selecting them to form a multiplicative scale, with each value a multiple of the
previous value, rather than the additive scale used here. Selecting the constant
stimuli in this way will allow the full range of psychophysical performance to be
captured. Future work could also merge the low- and high-range variants for further
efficiency, given similar threshold and slope values found for both variants. We
suggest selecting intensity pairs across the entire intensity range to define each
intensity difference.

The method offers four major advantages over commonly used methods of
sensitivity to emotional expression such as identification and rating the perceived
intensity of an expressed facial emotion. First, the forced-choice methodology is
relatively free from response biases and subjective criterion, and therefore gives an
objective measure of sensitivity that is not matched by subjective measures. Second,
forced-choice methodology gives sensitive measures of the ability to discriminate
facial expressions of emotion. The sensitivity to small variations in the intensity with
which an emotion is expressed is shown by the small absolute thresholds, which
ranged from about 7% to about 17% (with a median of 9%) in the different
measures. This sensitivity makes the method capable of detecting small changes in
sensitivity to emotional expression that might result from an experimental
manipulation or that might emerge with healthy aging or the progression of a
neurological disorder. Third, the method is broadly applicable to the expression of
different emotional states. Although broadly applicable, the method’s sensitivity
revealed differences in the ability to detect changes in different emotional
expressions, with consistently smaller absolute thresholds for detecting changes in
happiness than sadness, and intermediate thresholds for disgust and anger. Emotional
expressions vary in the visual range of expressivity, so the magnitude of a 5%
change will vary depending on the emotion. Changes in an emotion such as happiness, which is typically expressed with an open mouth and with extensive changes in facial features, will be discriminated with a smaller percentage change than an emotion that is more subtly expressed, such as sadness, which is typically expressed with a closed mouth and less extensive changes in facial features. The variations in ability to detect changes for different emotional expressions are consistent with previous research on more complex processes showing that expressions of positive emotions are easier to identify than negative emotions (Elfenbein & Ambady, 2002). Fourth, the method is efficient, which is an important factor when testing aged or clinical populations. A psychometric function with 40 observations at each of seven points can be obtained in about 12 minutes. The method could be made even more efficient by estimating threshold level with an adaptive procedure, in which the stimuli are changed contingent on the observer’s response, in place of the constant stimulus method used here. Fifth, the method is simple to administer and easily understood by participants. Although the results reported here are from a select sample of young, healthy, educated adults, our research in progress shows that the method is equally applicable to samples of patients with Parkinson’s disease and their age-matched controls, and so encourages its use in other atypical and healthy aging populations. It has been shown that emotion recognition is modulated by the mood of the perceiver (Niedenthal et al., 2000; 2001). It remains to be determined if the processes of emotion detection and discrimination measured by the methods described here are also susceptible to mood.

The basic perceptual abilities measured by the methods reported here, the ability to discriminate an emotional from a neutral expression, and the ability to differentiate between two different levels of expression of the same emotion, may
assist or work in concert with more complex social decision making and behavior. These measures, therefore, allow the contribution of lower-order perceptual determinants of higher-order disorders of emotional judgment to be detected.
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422.


CHAPTER 3: Voluntary Control of Facial Musculature in Parkinson’s Disease

Foreword

Having established the reliability of the psychophysical measures of discriminating emotional from neutral expressions and different intensities of the same emotional expressions in a healthy adult sample prior to their use in PD and healthy older control samples, the next step in examining an embodied simulationist hypothesis of impaired emotional expression perception in PD was to find a reliable measure of voluntary control of facial musculature. A literature search revealed that aside from its measurement in the context of the ability to produce emotional expressions, voluntary control facial musculature in PD had not been systematically measured. There is only one item that assesses facial expressivity on the Movement Disorders Society-sponsored revision of the Unified Parkinson’s Disease Rating Scale (MDS-UPDRS; Goetz et al., 2008). The next chapter describes the development of an adaptation of the Upper and Lower Face Apraxia Test (Bizzozero et al., 2000) as a measure of voluntary control of facial musculature in PD. The chapter reports on specific changes that occur in voluntary facial musculature control in PD. This measure of voluntary facial musculature control is then used again in Chapter 4 to examine its link with perception of emotional expressions in PD (testing an embodied simulationist hypothesis of impaired perception in emotional expressions in PD).
Abstract

Aside from being measured in the context of producing facial expressions of emotion, the ability to voluntarily control a range of facial muscles in Parkinson’s disease (PD) has not been systematically measured. We used in three enrolment phases an adaptation of the Upper and Lower Face Apraxia test, a measure of the ability to make voluntary movements of the upper and lower face in PD patients and healthy controls. Errors were scored due to (1) pauses prior to movement initiation, (2) loss of individuation, (3) impoverished movement, (4) no movement at all, or (5) content errors (likened to ideational apraxia errors). The results show impaired voluntary control of facial musculature in most but not all with PD (with large effect sizes) which correlated positively and highly with disease severity. Errors by PD patients were predominantly due to impoverished movement and individuation loss whereas those made by controls were predominantly due to individuation loss. Patients committed more errors than controls due to impoverishment and no movement, with negligible differences between groups in other errors. In sum, similarly to spontaneous and voluntary emotional expressions, voluntary non-emotional facial movements are impoverished in PD; impoverishment of all movement types will likely contribute to the mask-like facial appearance that is seen with disease progression. These findings also illustrate the utility of an adapted Face Apraxia test as a practical and sensitive measure of voluntary facial musculature control in PD. The test can be used to supplement clinical observations and as a research tool.
Introduction

A “masked” expressionless face is an important clinical sign of Parkinson’s disease (PD; Bologna et al., 2013; Jancovic, 2008; Rinn, 1984). In a now classic and influential review on facial expression, Rinn (1984) proposed that the impression of a mask-like face in PD originates from reduced spontaneous emotional expression, with voluntary emotional expression remaining intact. Following Rinn’s proposal, studies found that PD patients were impaired in spontaneous emotional expressions during conversation or in response to emotionally evocative stimuli (Katsikitis & Pilowsky, 1988; Pitcairn, Clemie, Gray, & Pentland, 1990; Simons, Ellgring, & Smith Pasqualini, 2003; Simons, Smith Pasqualini, Reddy, & Wood, 2004; Smith, Smith, & Ellgring, 1996). These findings have informed our understanding of facial masking in PD as a reduced spontaneity in facial expression of emotion. However, in contrast to Rinn’s proposal, PD patients have also been shown to be impaired in voluntarily expressing emotions in response to verbal command (e.g. “look happy”; Borod et al., 1990; Bowers et al., 2006; Jacobs, Shuren, Bowers, & Heilman, 1995; Marsili et al., in press; Simons et al., 2003; Simons et al., 2004). In these studies, responses have either been scored by blind raters using Likert scales, the Facial Action Coding System (FACS; Ekman & Friesen, 1978) which codes observable facial movements as “action units” or action unit combinations, or by digital imaging analyses (Bowers et al., 2006; Marsili et al., in press). These findings that were in contrast to earlier propositions (Rinn, 1984) spurred the more recent conclusion (Bowers et al., 2006) that facial masking in PD is not limited to spontaneous facial expressions of emotion, but also involves voluntary facial expressions of emotion.

It is reasonable to suspect that non-emotional facial movement is also impaired in PD, thereby contributing with diminished emotional expressions to facial
masking. Nevertheless, voluntary non-emotional facial movements remain less systematically explored than voluntary emotional facial movements in PD. Studies on non-emotional facial movement in PD have been limited to specific facial areas, with impairment evidenced in voluntary, spontaneous, and reflex blinking rate and amplitude (Agostino et al., 2008; Korosec, Zidar, Reitz, Evinger, & Vanderwerf, 2006) and amplitude of jaw and upper lip movement during speech (Connor, Abbs, Cole, & Gracco, 1989). Simons et al. (2003; 2004) is the only group that has measured, using the FACS, voluntarily imitating a limited set of non-emotional facial movements and found these movements to be impaired in PD. Although the FACS procedure is sensitive to compare action unit patterns with requested patterns, it is not always a practical tool available to clinicians and researchers; FACS certification requires extensive and costly training. There is only one item that assesses facial expressivity on the Movement Disorders Society-sponsored revision of the Unified Parkinson’s Disease Rating Scale (MDS-UPDRS; Goetz et al., 2008). On this item, PD patients are rated for their facial expressivity for 10s without moving or speaking. Although this item may pick up some loss of facial musculature control (e.g. reduced control in keeping mouth closed, reduced blinking), the item does not measure the ability to control a range of facial muscles. Therefore, not only is there limited understanding of voluntary facial musculature control in PD, there is no sensitive and practical measure of this ability in PD. As a result, the symptom of facial masking and its understanding remains restricted to impaired emotional expression, and mainly assessed by subjective judgment in clinical contexts.

We measured voluntary facial musculature control in PD in three enrolment phases with an adaptation of the Upper and Lower Face Apraxia Test (Bizzozero et al., 2000), a 38-item test of the ability to make upper and lower facial movements in
response to verbal command and demonstration. Each reproduction is scored as a pass or fail, with set criteria that determine an item as failed. This measure has been shown to be a sensitive measure of voluntary facial musculature control in stroke patients (Bizzozero et al., 2000). Initial observations revealed that errors by participants did not necessarily fit the criteria set for errors in the original test. Facial movements by patients were often impoverished, with reproductions that were of lower amplitude than the demonstrated movement. Observations of impoverished facial movement are also commonly reported in clinical contexts (Tickle-Degnen & Lyons, 2004; Tickle-Degnen, Zebrowitz, & Ma, 2011). We also observed loss of individuation of movement in PD and control groups, where the requested movement was temporally coupled with uninstructed movement. Individuation loss has also been reported with manual movements in healthy aging (Hortobagyi & DiVita, 2006; Mattay et al., 2002; Shim, Lay, Zatsiorsky, & Latash, 2004) and in PD (Vaillancourt, Slifkin, & Newell, 2002). Guided by these typical errors, we scored errors due to impoverished movement and individuation loss separately, allowing for a refined error analysis. We also introduced a category for content errors, which might indicate the presence of ideational apraxia. We observed reproductions by participants at times resembled the demonstrated movement but were incorrect in their content e.g. placing the tongue in the cheek when asked to puff out the cheek. The overarching aim of this study was to explore a range of lower and upper non-emotional facial movements in persons with PD and healthy controls; in doing so, we also aimed to determine the utility of an adaptation of the Face Apraxia Test (Bizzozero et al., 2000) as a sensitive and practical measure of voluntary facial musculature control in PD.
Methods

Participants

Sixty-six participants (Control \( n = 32 \), PD \( n = 34 \)), 49 participants (Control \( n = 24 \), PD \( n = 25 \)), and 40 participants (Control \( n = 17 \), PD \( n = 23 \)) took part in Enrolment Phases 1, 2, and 3 respectively. There were 22 participants unique to Phase 1 (Control \( n = 12 \), PD \( n = 10 \)), four unique to Phase 2 (Control \( n = 3 \), PD \( n = 1 \)), five unique to Phase 3 (Control \( n = 2 \), PD \( n = 3 \)), and 23 and 26 participants who took part in two or three phases, respectively (two: Control \( n = 13 \), PD \( n = 10 \); three: Control \( n = 10 \), PD \( n = 16 \)). The enrolment phases were approximately one year apart. PD and control groups were well matched in each phase on demographic and clinical characteristics (Table 1). Patients were diagnosed by a neurologist. The local Institutional Ethics Committee approved the study procedures and all participants gave written informed consent.
Table 1
Demographic and clinical characteristics of participant groups in Enrolment Phase 1, 2, and 3.

<table>
<thead>
<tr>
<th></th>
<th>PHASE 1</th>
<th>PHASE 2</th>
<th>PHASE 3</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>CONTROL</td>
<td>PD</td>
<td>CONTROL</td>
</tr>
<tr>
<td>Age (years)</td>
<td>66 (51-79)</td>
<td>66 (46-80)</td>
<td>67 (55-80)</td>
</tr>
<tr>
<td>Males (Females)</td>
<td>11 (21)</td>
<td>20 (14)</td>
<td>12 (12)</td>
</tr>
<tr>
<td>Education (years)</td>
<td>15 (7-22)</td>
<td>12 (7-20)</td>
<td>16 (8-22)</td>
</tr>
<tr>
<td>MoCA</td>
<td>27 (21-30)</td>
<td>28 (22-30)</td>
<td>28 (25-30)</td>
</tr>
<tr>
<td>GDS</td>
<td>1 (0-5)</td>
<td>2 (0-13)</td>
<td>1 (0-5)</td>
</tr>
<tr>
<td>Years diagnosed</td>
<td>-</td>
<td>5 (1-19)</td>
<td>-</td>
</tr>
<tr>
<td>MDS-UPDRS-III</td>
<td>-</td>
<td>38 (10-56)</td>
<td>-</td>
</tr>
<tr>
<td>LED</td>
<td>-</td>
<td>788 (0-2046)</td>
<td>-</td>
</tr>
</tbody>
</table>

Note. Values (except male and female numbers) are expressed as median (range); MoCA scores range from 0 to 30, a score ≥ 26 reflects normal cognitive functioning; GDS scores range from 0 to 15, a score of ≥ 6 suggests depression warranting assessment; MDS-UPDRS-III motor scores range from 0 to 132 (most advanced); LED = Daily Levodopa Dose Equivalent (Tomlinson et al., 2010).
Materials and Procedures

For PD patients, each of the enrolment phases were conducted approximately 1.5 hours before their next scheduled dopamine replacement medication intake; we did not have ethical approval to compare patients that were on dopamine replacement therapy with those that were depleted from dopamine replacement therapy. At the start of each phase, patients were assessed for disease severity, using the motor subscale (III) of the MDS-UPDRS (Goetz et al., 2008), depressive symptoms (Geriatric Depression Scale; GDS; Sheikh & Yesavage, 1986), and general cognitive functioning (Montreal Cognitive Assessment; MoCA; Nasreddine et al., 2005). The ability to voluntarily control facial musculature was measured using an adaptation of the Upper and Lower Face Apraxia Test (Bizzozero et al., 2000) in which participants were required to make 9 upper and 29 lower facial movements. In the original test, the examiner demonstrates each item, and the participant’s response follows immediately. For standardization purposes in our study, the instructions and demonstrations of each item were recorded in a video-clip lasting 7 minutes and watched by all participants on an 11-inch MacBook Air. For scoring purposes, participants’ faces were filmed with each face in full frontal view. Participants were instructed to reproduce the intensity and duration of each demonstration as accurately as possible.

Two independent raters, one blind to the study, scored the accuracy of each reproduction as correct or incorrect. For scoring purposes, the videorecording of each participant’s set of reproductions was viewed alongside the video-clip of instructions and demonstrations. Initial observations of typical errors committed by both groups guided the process of modification of the scoring structure for a more refined classification of errors committed by PD and control participants. Incorrect
items were assigned to one of five error categories: (1) the reproduction was preceded by a pause during which unsolicited movements might have been present; (2) there was a loss of individuation: the instructed movement was executed, but was unfocused, with the instructed movement accompanied by uninstructed movement. The instructed movement was either intermittently or continuously present. This error was also scored if participants reproduced an instructed movement sequence (for example, moving the jaw from left to right, three times), but with an increase in the number of elements in the sequence (for example, moving their jaw from left to right six times, instead of three times); (3) the reproduction was executed but impoverished, either by reduction of amplitude of movement or by reduction in the number of elements of a sequential movement; (4) there was no movement at all; or (5) there was a content error, where the reproduction resembled the demonstrated movement but was incorrect in its content.

We report the Cronbach’s alpha and the percent agreement between raters (Stemler, 2004) by adding the number of cases that received the same rating by raters and dividing that number by the total number of cases rated. We report the mean of both raters’ scores in each of the enrolment phases and measures of effect size (Hedges’ g) to quantify group differences. Unless otherwise stated, we report pooled percent error scores on the Face Apraxia Test across enrolment phases with percent error scores for those unique to each of the enrolment phases and averaged percent error scores for those who did two or three enrolment phases. Pearson correlation coefficients with 95% confidence intervals (CI) are reported for correlational analyses.
Results

The adapted Face Apraxia Test showed good inter-rater reliability (Nunnally, 1978) with Cronbach’s alphas of .98, .97, and .92 for Enrolment Phases 1, 2, and 3 respectively for total scores. Supplemental Table 1 shows that Cronbach’s alphas for each of the five error categories for all phases were also acceptable. The proportion correct consensus estimate was .91 for Phase 1, and .90 for Phases 2 and 3. The adapted test also demonstrated good test-retest reliability with large positive correlations (Control $r = .85$, 95% CI: .67, .93; PD $r = .85$, 95% CI: .69, .93) between first- (Phase 1 Control $n = 20$, PD $n = 24$; Phase 2 Control $n = 3$, PD $n = 2$) and second-time performance (Phase 2 Control $n = 18$, PD $n = 22$; Phase 3 Control $n = 5$, PD $n = 4$).

The box-and-whisker plots in Figure 1 show that the PD group on average made more errors than the control group on the adapted Face Apraxia Test in all enrolment phases (Interquartile Range: Phase 1 Control: 15.8, PD: 20.3; Phase 2 Control: 12.5, PD: 18.0; Phase 3 Control: 17.5, PD: 20.0). The mean percent error rate was higher in the PD ($M = 38.8$, $SD = 14.7$) than control group ($M = 26.6$, $SD = 12.2$), and the effect size ($g = .89$) is considered large by conventional standards. Mean percent of errors were higher in the PD than control group for upper (Control $M = 27.1$, $SD = 14.4$; PD $M = 40.3$, $SD = 15.1$; $g = .89$) and lower facial movements (Control $M = 25.1$, $SD = 12.4$; PD $M = 33.9$, $SD = 20.7$; $g = .51$).
Figure 1. Box-and-whisker plots showing percent error scores for control (filled boxes) and PD (open boxes) groups on the adapted Upper and Lower Face Apraxia Test in Enrolment Phase 1 (left), Enrolment Phase 2 (middle), and Enrolment Phase 3 (right).

Table 2 shows that the majority of errors committed by both groups was due to loss of individuation and impoverished facial movements. The percent of individuation loss errors was higher in the control than PD group whereas percent of impoverishment errors was higher in the PD than control group, indicating a shift towards impoverished facial movements with the additional burden of PD. Table 2 also shows that patients committed more errors due to impoverishment and ‘no movement at all’ than controls, as demonstrated by large and moderate effect sizes respectively. Negligible effect size for errors due to other categories indicate that patients made no more errors than controls due to individuation loss, pauses, and content errors.
Table 2
Mean percent errors and mean number of errors for participant groups for each error category with pooled data from those unique to one phase and mean scores for those who completed two or three phases. Hedges’ g quantifies group differences.

<table>
<thead>
<tr>
<th>Error Category</th>
<th>MEAN PERCENT OF ERRORS</th>
<th>MEAN NUMBER OF ERRORS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CONTROLS</td>
<td>PD</td>
</tr>
<tr>
<td>Pauses</td>
<td>3.5 (4.4)</td>
<td>2.4 (3.3)</td>
</tr>
<tr>
<td>Loss of individuation</td>
<td>59.2 (16.9)</td>
<td>45.1 (15.6)</td>
</tr>
<tr>
<td>Impoverishment</td>
<td>29.2 (17.7)</td>
<td>44.3 (17.3)</td>
</tr>
<tr>
<td>No movement at all</td>
<td>2.2 (4.0)</td>
<td>3.3 (4.5)</td>
</tr>
<tr>
<td>Content errors</td>
<td>6.0 (7.1)</td>
<td>4.9 (4.8)</td>
</tr>
</tbody>
</table>
The error profile in Figure 2 of patients at varying stages of the disease (as indicated by the MDS-UPDRS-motor score) shows an increase in the total error number with disease progression ($r = .71$, 95% CI: .51, .84). The figure also shows that the number of errors due to impoverishment increased with disease progression (slope = .19) more so than the number of errors due to individuation loss (slope = .08), no movement (slope = .08), pauses (slope = .03), and content errors (slope = .02). We also calculated correlations between first- and second-time performance for the most predominant error types in PD, impoverishment and loss of individuation, for those who participated in at least two phases ($n = 26$). Correlations between first- and second-time performance was positive and moderate for the numbers of errors made due to impoverishment ($r = .61$, 95% CI: .29, .81) and individuation loss ($r = .60$, 95% CI: .28, .80). After removing the common variance shared with MDS-UPDRS-motor scores, there was little to no correlations between percent errors on the Face Apraxia Test, MoCA scores ($r = -.25$, 95% CI: -.52, .07) and depressive symptoms (measured by the GDS, $r = .18$, 95% CI: -.14, .47).
Figure 2. Mean errors due to impoverishment (clear), individuation loss (lightest gray), no movement (medium gray), content (dark gray), and pauses (darkest gray) for patients assigned to each of five severity stages (10 - 20: \( n = 5 \); 21 - 30: \( n = 5 \); 31 - 40: \( n = 12 \); 41 - 50: \( n = 13 \); 51 - 60: \( n = 5 \)).

**Discussion**

Our results show the utility of the adapted Face Apraxia Test (Bizzozero et al., 2000) in PD. The adapted test includes standardized video-recorded instructions and demonstrations and three new categories for error classification (individuation loss; impoverished movements; content errors) for an improved resolution of voluntary facial musculature control in PD. With good inter-rater and test-retest reliability, this measured showed impaired upper and lower facial movements without emotional content in PD. Most errors by patients were due to individuation loss and impoverishment, with a marked increase in percent of impoverishment errors and a marked decrease in percent of individuation loss errors relative to controls. The mean number of errors due to impoverishment and
‘no movement at all’ was higher in the patient than control group, whereas group differences were negligible in mean number of errors due to individuation loss, pauses, and content errors. There was a strong positive correlation between total number of errors and disease severity, but not with cognitive functioning or depressive symptoms, where impoverishment errors increased more so with disease progression than other error types.

The mask-like facial appearance in PD has historically been conceptualized as diminished spontaneous emotional facial expressions (Rinn, 1984). Later work supported claims that diminished voluntary emotional expressions in PD add to a mask-like facial appearance (Borod et al., 1990; Bowers et al., 2006; Jacobs et al., 1995; Marsili et al., in press; Simons et al., 2003; Simons et al., 2004). Our data, with others (Agostino et al., 2008; Connor et al., 1989; Korosec et al., 2006; Marsili et al., in press; Simons et al., 2003; Simons et al., 2004) show that PD patients are also impaired in a range of upper and lower non-emotional facial movements, which likely contribute, with blunted emotional expressions, to facial masking in PD. That impaired voluntary non-emotional facial movement was predominantly due to impoverished movement in PD further strengthens the claim that it too contributes to facial masking. The largest difference in number of errors between groups was those due to impoverished facial movements and ‘no movement at all’; the latter might be considered the most pronounced case of impoverishment, which might give rise to the most pronounced case of facial masking. Clinical observations of facial masking, which have been shown to affect practitioners’ impressions of patient personality, mood, and cognitive competency (Tickle-Degnen & Lyons, 2004; Tickle-Degnen et al., 2011), are likely a product of impoverished spontaneous and
voluntary emotional expressions, and as we show here, of voluntary non-emotional facial movements. Simply put, less movement of any kind on the face might give rise to the impression of a mask-like face in PD.

The total number of errors correlated positively and highly with disease severity, where impoverished voluntary facial movement increased more than other error types with disease progression. We showed this shift towards impoverishment with a sample of patients in early to moderate stages of disease progression. We expect that impoverishment of facial movement to become even more pronounced in the more advanced stages of the disease. We interpret with caution the negligible difference between groups in the number of errors due to pauses, given that this error type might have been underestimated. It was relatively difficult to capture errors due to pauses because the video-recorded data did not include a time-marker of windows in which participants were instructed to respond. Bowers et al. (2006) have shown with sophisticated computer imaging methodology that voluntary emotional movements of the face are slower to initiate in PD than controls, which suggests at least some role of bradykinesia to impaired voluntary control of facial musculature, and facial masking. There was little difference between groups in content errors, which suggests ideational apraxia of the face is no more common in early to moderate stages of PD than that in healthy age-matched controls. These findings are also congruent with the evidence summarized by Zadikoff and Lang (2005) of no ideational apraxia of the upper limbs in PD. The low incidence of content errors and the positive correlation with motor severity but not with general cognitive functioning suggests that impaired voluntary facial musculature control in PD is more a part of the motor symptoms than the non-motor symptoms of the disease.
Our results demonstrate that the adapted Face Apraxia Test (Bizzozero et al., 2000) is a reliable measure of voluntary facial musculature control in PD, which can be used in research and clinical settings to further understand the determinants of facial masking. There are several advantages to the use of this test in PD. The test is practical, free, and easy to administer and score. Unlike the FACS, it does not require intensive and costly training. The test is also comprehensive, whereas previous PD research on non-emotional facial movements has been limited to specific facial areas (Agostino et al., 2008; Connor et al., 1989; Korosec et al., 2006) or to a small set of facial movements (Simons et al., 2003; Simons et al., 2004). The revised error categories allow identifying a range of errors that are not otherwise captured by other measures of facial musculature control in PD. Finally, the test is sensitive to detect between- and within-group differences in voluntary facial musculature control in PD and controls. A limitation of the present study is that we did not compare voluntary facial musculature control in patients that were on dopamine replacement therapy with those that were depleted from dopamine replacement therapy. Therefore, the role of dopamine replacement therapy in voluntary facial movement cannot be determined. However, others have found little to no improvement in upper (Agostino et al., 2008) and lower facial movements (Marsili et al., in press) in patients that were on- than off dopamine replacement therapy. In addition, similar to all measures of voluntary facial musculature control that provide a demonstration for each item, the Face Apraxia Test requires imitating demonstrated movements. It is unknown whether movement imitation inflates or deflates impairment in voluntary facial musculature control in PD. In sum, the work described here adds to the current literature on facial musculature control in
PD by showing that a range of upper and lower voluntary non-emotional movements of the face are impaired. Furthermore, that these voluntary facial movements were impaired mainly due to impoverishment makes it likely to give rise, with impoverished emotional expressions, to the impression of the mask-like facial appearance that is seen with the progression of the disease.
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**Supplementary Material**

Table 1  
*Cronbach’s alpha for error assignment by two raters to each of the error categories on the adapted version of the Upper and Lower Face Apraxia Test for Enrolment Phases 1, 2, and 3.*

<table>
<thead>
<tr>
<th></th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pauses</td>
<td>.85</td>
<td>.64</td>
<td>.90</td>
</tr>
<tr>
<td>Individuation loss</td>
<td>.96</td>
<td>.94</td>
<td>.93</td>
</tr>
<tr>
<td>Impoverished movement</td>
<td>.95</td>
<td>.94</td>
<td>.93</td>
</tr>
<tr>
<td>No movement at all</td>
<td>.95</td>
<td>.94</td>
<td>.95</td>
</tr>
<tr>
<td>Content errors</td>
<td>.93</td>
<td>.94</td>
<td>.98</td>
</tr>
</tbody>
</table>
CHAPTER 4: Perception of Facial Expressions of Emotion and its Link with Voluntary Control of Facial Musculature in Parkinson’s Disease

Foreword

The previous two chapters described the use of psychophysical measures of discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions in a healthy adult sample, and the use of the adapted Face Apraxia Test (Bizzozero et al., 2000) as a measure of voluntary facial musculature control in PD. Having established the reliability of these measures, the two key aims of the thesis could now be investigated, (1) to measure perception of facial expressions of emotion in PD, with particular emphasis on discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions, and (2) to measure some factors that might contribute to impaired perception of facial expressions of emotion in PD. The next chapter describes two experiments (Experiments 2 and 3) on persons with PD and healthy controls. Towards addressing the first aim, Experiment 2 measures in both patients and controls discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions. Experiment 3 again measures discriminating graded intensities of the same emotional expressions, but with an increased range of stimulus values as to capture a fuller range of psychophysical performance than in Experiment 2, and measures of discriminating discrepant from similar full-blown emotion expressions and recognizing full-blown emotional expressions. Towards addressing the second aim, the links are measured between perceiving emotional expressions, disease severity, and voluntary control of facial musculature (to test an embodied simulationist
account of emotion perception) in Experiments 2 and 3. In addition, Experiment 3 also explores the link between perceiving emotional expressions and perceiving visual form from faces, measured as the ability to discriminate facial distinctiveness and the ability to recognize facial identity. As the following chapter is a standalone paper that has been submitted for publication, Experiments 2 and 3 of the thesis are referred to in this chapter as Experiments 1 and 2.
Abstract

We investigated in two sets of experiments in PD patients and healthy controls the perceptual ability to discriminate (1) graded intensities of emotional from neutral expressions (2) graded intensities of the same emotional expressions (3) full-blown discrepant emotional expressions from two similar expressions, and the more complex recognition ability to label full-blown emotional expressions. We tested an embodied simulationist account of emotion perception in PD, which predicts a link between the ability to perceive emotional expressions and facial musculature control. We also explored the contribution of the ability to extract facial information (besides emotion) to emotional expression perception in PD. Those with PD were, as a group, impaired relative to controls (with large effect sizes) in all measures of discrimination and recognition of emotional expressions, although some patients performed as well as the best-performing controls. In support of embodied simulation, perception of emotional expressions correlated positively with voluntary control facial musculature (after partialling out disease severity and age). Patients were also impaired at extracting information other than emotion from faces, specifically discriminating and recognizing identity from faces (with large effect sizes); identity discrimination correlated positively with emotional expression perception but not with voluntary facial musculature control (after partialling out disease severity and age). The results indicate that impaired sensory and sensorimotor processes, which are a function of disease severity, affect emotional expression perception in PD.
Introduction

The ability to perceive the facial expressions of emotion of others is central to the regulation of social behavior. Findings from studies that have investigated whether those with Parkinson’s disease (PD) have impaired perception of facial expressions of emotion have been mixed. Some studies have reported impaired perception of facial expressions of emotion in PD (Ariatti, Benuzzi, & Nichelli, 2008; Assogna et al., 2010; Baggio et al., 2012; Bediou et al., 2012; Buxton, MacDonald, & Tippett, 2013; Clark, Neargarder, & Cronin-Golomb, 2008; Clark, Neargarder, & Cronin-Golomb, 2010; Dujardin et al., 2004; Herrera, Cuetos, & Rodriguez-Ferreiro, 2011; Ibarretxe-Bilbao et al., 2009; Jacobs, Shuren, Bowers, & Heilman, 1995; Kan, Kawamura, Hasegawa, Mochizuki, & Nakamura, 2002; Lawrence, Goerendt, & Brooks, 2007; Narne, Bonnet, Dubois, & Chaby, 2011; Narne et al., 2013; Sprengelmeyer et al., 2003; Suzuki, Hoshino, Shigemasu, & Kawamura, 2006; Yip, Lee, Ho, Tsang, & Li, 2003), while others have found no impairment (Adolphs, Schul, & Tranel, 1998; Cohen, Gagne, Hess, & Pourcher, 2010; Haeske-Dewick, 1996; Pell & Leonard, 2005; Ventura et al., 2012; Wieser et al., 2012). Although the results from individual studies are mixed, a recent meta-analysis found that, overall, PD groups were impaired in perceiving emotions from facial cues, documented by an averaged moderate effect size (Hedges’ $g = .48$) for all of the “basic emotions” (Gray & Tickle-Degnen, 2010).

Gray and Tickle-Degnen (2010) documented substantial variability in emotion perception impairment in PD, reflected in wide confidence intervals of effect sizes, which was not related to medication, depression, or disease severity. The studies in the meta-analysis measured disease severity with the Hoehn and Yahr Staging Scale (Hoehn & Yahr, 1967), which, although widely used and simple to
administer, has been criticized as insensitive. Goetz and colleagues (2004) reported that the scale is heavily weighted towards postural instability, leaving other important components of PD unassessed; furthermore, with only five scale options, a large range of impairment severity is collapsed together. The Movement Disorders Society-sponsored revision of the Unified Parkinson’s Disease Rating Scale (MDS-UPDRS) has been recommended as a more sensitive measure (Goetz et al., 2008). The Gray and Tickle-Degnen meta-analysis also indicated that the variability in emotion perception in PD was not due to a broader inability to extract other information than emotion from faces, as perception of non-emotional information from faces was intact. Most studies in the meta-analysis measured this ability with the Benton Facial Recognition Test (BFRT; Benton, Sivan, Hamsher, Varney, & Spreen, 1994). However, the BFRT has been shown to be an insensitive measure of face identity processing skill, and sensitive only to large deficits (Duchaine & Weidenfeld, 2003). The link between emotion perception and the broader ability to extract information other than emotion from faces, using sensitive measures, has been relatively unexplored in PD.

The determinants of impaired perception of facial expressions of emotion in a disorder of movement are not well understood. The theory of embodied simulation of emotion perception, which states that perceiving emotions of others is facilitated by overtly or covertly simulating the observed behavior (see Goldman & Sripada, 2005, for a review), suggests one potential determinant of the impairment seen in PD. In line with covert embodied simulation theories, systems with mirror-like properties that are engaged by control of facial musculature and by simulation of observed expressions might be compromised by the neurodegenerative processes of PD, thereby contributing to impaired facial musculature control (i.e. facial masking).
and impaired perception of emotional expressions. A link between voluntary control of facial musculature and perception of emotional expressions in PD will provide support for this simulationist account of emotion perception in PD.

Studies on perception of facial expressions of emotion have typically investigated the ability to discriminate emotional expressions (by judging whether two or more stimuli express the same or different emotion) or the ability to recognize emotional expressions (by selecting from a list the emotion word that best fits the presented expression). Most previous studies on perception of facial expressions of emotion in PD have measured the ability to recognize emotional expressions. Some studies (Ariatti et al., 2008; Jacobs et al., 1995; Pell & Leonard, 2005; Ventura et al., 2012) have measured the ability to discriminate whether a series of stimuli express the same or different emotion. Most previous studies also only assess the ability to perceive emotions that are expressed at full-blown intensity, when in everyday life emotions are expressed with varying intensities. In the current experiments, we studied basic perceptual abilities of discriminating graded expressions of emotion with psychophysical methods. Forced-choice psychophysical procedures offer objective and sensitive measures of perceptual processes that are relatively free from response criterion effects. In Experiment 1, we used previously developed psychophysical measures of discriminating graded intensities of facial expressions of emotion from neutral expressions and discriminating different intensities of the same facial expressions of emotion (Marneweck, Loftus, & Hammond, 2013). In Experiment 2, we again used measures of discriminating different intensities of the same emotional expressions, and measures of labelling full-blown emotional expressions, discriminating discrepant from similar full-blown expressions, and sensitive measures of face identity perception. To test an embodied simulation
account of emotion perception, we measured in both experiments the relationship between continuous measures of the ability to voluntarily control facial musculature and the ability to perceive emotional facial expressions in PD.

Methods

Participants

Sixty-six participants (Control \textit{n} = 32, Patient \textit{n} = 34) were tested in Experiment 1 and 49 (Control \textit{n} = 24, Patient \textit{n} = 25) were tested in Experiment 2, approximately one year after Experiment 1. Forty participants participated in both experiments (Control \textit{n} = 18, Patient \textit{n} = 22) whereas 26 participants (Control \textit{n} = 14, Patient \textit{n} = 12) were unique to Experiment 1 and nine participants (Control \textit{n} = 6, Patient \textit{n} = 3) were unique to Experiment 2. Table 1 shows demographic and clinical characteristics of both groups in each experiment; groups were on aggregate well matched for age and scores on measures of general cognitive functioning and depressive symptoms. Patients were diagnosed by a neurologist and recruited from the Edith Cowan University Parkinson’s Centre research database and through Parkinson’s Western Australia newsletter advertisements. The Institutional Ethics Committee approved the procedures and all participants gave written informed consent.
Table 1
Demographic and clinical characteristics of participant groups in Experiment 1 (Control n = 32, Patient n = 34) and Experiment 2 (Control n = 24, Patient n = 25) with Hedges’ g quantifying group differences.

<table>
<thead>
<tr>
<th></th>
<th>EXPERIMENT 1</th>
<th>EXPERIMENT 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CONTROL</td>
<td>PATIENT</td>
</tr>
<tr>
<td>Age (years)</td>
<td>66 (51-79)</td>
<td>66 (46-80)</td>
</tr>
<tr>
<td>Males (Females)</td>
<td>11 (21)</td>
<td>20 (14)</td>
</tr>
<tr>
<td>Education (years)</td>
<td>15 (7-22)</td>
<td>12 (7-20)</td>
</tr>
<tr>
<td>MoCA</td>
<td>27 (21-30)</td>
<td>28 (22-30)</td>
</tr>
<tr>
<td>GDS</td>
<td>1 (0-5)</td>
<td>2 (0-13)</td>
</tr>
<tr>
<td>Years diagnosed</td>
<td>-</td>
<td>5 (1-19)</td>
</tr>
<tr>
<td>MDS-UPDRS-III</td>
<td>-</td>
<td>38 (10-56)</td>
</tr>
<tr>
<td>LED</td>
<td>-</td>
<td>788 (2046)</td>
</tr>
</tbody>
</table>

Note. All values (except number of males and females) are expressed as median (minimum-maximum range); MoCA = Montreal Cognitive Assessment, score ranges from 0 (most severe) to 30, a score ≥ 26 reflects normal cognitive functioning; GDS = Geriatric Depression Scale, score ranges from 0 to 15 (most severe), a score of 6 or more is suggestive of depression that should warrant thorough assessment; MDS-UPDRS-III = motor scale of Unified Parkinson’s Disease Rating Scale, score ranges from 0 to 132 (most advanced disease state); LED = Daily Levodopa Dose Equivalent (Tomlinson et al., 2010). Statistical significance (p < .05) is denoted with an asterisk.
Design

There were three testing sessions in Experiment 1 and two testing sessions in Experiment 2, with all sessions separated by at least 24 hours. In each of the two experiments, PD patients were tested at the same time of the day in each of the testing sessions (~1 to 1.5 hours prior to their next medication intake) with the aim that performance in each session would receive the same effect from dopamine replacement medication (however, Gray and Tickle-Degnen, 2010, reported no such effects). Control participants completed each of the sessions at the same time of the day. In each experiment, patients were initially assessed for disease severity using the MDS-UPDRS motor severity subscale (Goetz et al., 2008). We took measures of the ability to voluntarily control facial musculature (the Upper and Lower Face Apraxia Test, Bizzozero et al., 2000), general cognitive functioning (Montreal Cognitive Assessment, MoCA; Nasreddine et al., 2005), and depressive symptoms (Geriatric Depression Scale, GDS; Sheikh & Yesavage, 1986). The MoCA has been shown to be a sensitive measure of general cognitive functioning in PD, discriminating very well between no cognitive impairment, mild cognitive impairment and dementia in PD (Dalrymple-Alford et al., 2010; Nazem et al., 2009; Zadikoff et al., 2008). The GDS also has been shown to be a sensitive measure of depressive symptoms in PD (Weintraub, Oehlberg, Katz, & Stern, 2006). In Experiment 1, we used psychophysical measures of the ability to discriminate graded intensities of emotional from neutral expressions and of the ability to discriminate graded intensities of the same emotional expressions. In Experiment 2, we again used the measure of discriminating graded intensities of the same emotional expressions (with an increased range in stimulus levels to capture a wider range of sensitivities than in Experiment 1), measures of discriminating and recognizing
expressions at full-blown intensity, and a measure of facial identity recognition, the Cambridge Face Memory Test (CFMT; Duchaine & Nakayama, 2006). We also included a psychophysical measure of the ability to extract information besides emotion from faces, the ability to discriminate differences in facial distinctiveness (defined as how much a face stands out in a crowd, Bruce & Young, 2012, p. 274). This measure was matched to the psychophysical measure of discriminating differences in emotional intensities. Distinctiveness predicts identity recognition (Bartlett, Hurry, & Thorley, 1984; Light, Kayra-Stuart, & Hollander, 1979; Vokey & Read, 1992) and has been included as an important dimension in “face-space” models of identity recognition (Busey, 1998; Valentine, 1991). There were standardized instructions for all measures.

Materials and Procedures

Psychophysical measures of discriminating facial expressions of emotion of graded intensity. The ability to discriminate graded intensities of an emotional from a neutral expression and to discriminate between different intensities of expression of the same emotion was measured in two tasks using a two-interval forced-choice procedure with the Method of Constant Stimuli, for each of four emotions, anger, disgust, happiness, and sadness (Marneweck et al., 2013). On each trial, two faces of the same model were presented successively on a computer screen for 200 ms with a 200-ms blank inter-stimulus interval. Each of the four emotions (NimStim Face Stimulus Set, Tottenham et al., 2009), expressed by two models in Experiment 1 and four models in Experiment 2, were graded in emotional intensity by morphing full-blown expressions of each model with their neutral expression (see Marneweck et al., 2013 for details).

In the task that required discrimination of a neutral from an emotional
expression (Experiment 1), the face with the neutral expression appeared randomly in either the first or the second interval and a face expressing one of seven levels of intensity of the tested emotion appeared in the other interval. The seven intensity levels ranged from 5% to 35% of the full-blown expression in equally spaced increments. On each trial participants signalled the interval containing the emotional face by clicking either the left or right button on a mouse for the first or second interval respectively. The stimuli pairs were presented in randomized blocks of 14 trials, with one presentation of each of the seven intensity increments for each of two models in each block. There were 20 blocks for a total of 280 trials, giving 40 trials for each intensity increment.

In the task that required discrimination between different intensities of the same emotion (Experiment 1 and 2), each of two faces expressing different intensities was randomly assigned to the two intervals and participants signalled the interval containing the face expressing the higher intensity by clicking either the left or right mouse button for the first or second interval respectively. In Experiment 1, the two facial expressions varied in five intensity steps from 5% to 25% in equally spaced increments and two variants of this task were run: one sampled emotional intensities from a low intensity range (from 10% to 50% of the full-blown expression) and the other from a high intensity range (from 50% to 90% of the full-blown expression). In Experiment 2, the five intensity steps varied in a multiplicative of 2 from 3% to 48% to capture a wider range of sensitivity to expression of emotion than in Experiment 1. In Experiment 2, intensities were sampled from a mid-intensity range (from 22% to 76% of the full-blown expression). The intensity pairs ($n = 4$) used to define each intensity difference for each of the experiments are shown in Supplementary Table 1. For
Experiment 1, intensity pairs were presented in five randomized blocks of 40 trials, with one presentation of each of the four definitions of the five intensity differences for each of two models in each block, which resulted in a total of 200 trials, giving 40 trials for each intensity difference. For Experiment 2, with an increase to four models expressing the varying intensities of emotional expressions, there were three randomized blocks of 80 trials, with each block containing each of the four definitions of the five intensity differences for each of four models, giving a total of 240 trials, with 48 trials for each intensity difference.

All measures were repeated for each of four emotions, anger, disgust, happiness, and sadness with breaks between each. Presentation of the two psychophysical measures in Experiment 1 was counterbalanced and the presentation of the four emotions followed a Latin Square sequence in both experiments.

**Psychophysical measures of discriminating graded differences in facial distinctiveness.** The ability to discriminate graded differences in facial distinctiveness was measured using a two-interval forced-choice task with the Method of Constant Stimuli. The stimulus duration, inter-stimulus interval, stimulus size, and viewing distance were matched to the psychophysical measures of discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions. The measure was also piloted to be of equal difficulty to psychophysical measures of emotional expression discrimination for control participants. To create a set of faces that varied in distinctiveness, we had 62 healthy young adults rate the distinctiveness of each of ten Caucasian faces (with neutral expressions; Tottenham et al., 2009). Participants were asked to rate the
distinctiveness of each face on a 9-point Likert scale (1 = average, typical looking, would not stand out in a crowd to 9 = very distinctive, would stand out very much in a crowd). Two male and two female models with the highest mean rating of distinctiveness were selected and morphed, using standard morphing procedures, with an average face of the same sex (created by morphing 26 faces of males and females, respectively; Rhodes et al., 2011), creating a set of faces that vary in distinctiveness from an average, typical face to a very distinctive face.

On each trial of the measure of discriminating facial distinctiveness differences, two faces that varied in distinctiveness were randomly assigned to one of two intervals. The two faces varied in five distinctiveness steps, 30, 40, 60, 90, and 120%. Participants were asked to indicate which interval contained the more distinctive face by clicking the left or right mouse button for the first or second observation interval, respectively. Supplementary Table 2 shows the stimulus pairs that defined each distinctiveness difference. The pairs were presented in randomized blocks of 60 trials: three definitions for each of five differences in distinctiveness expressed by four models. There were four blocks for a total of 240 trials, giving 48 trials for each distinctiveness difference.

**Discriminating discrepant from similar expressions of full-blown intensity.** The ability to discriminate the discrepant emotional facial expression from two expressions of the same emotion was measured in a three-alternative forced-choice measure (Palermo, O’Connor, Davis, Irons, & McKone, 2013). On each trial, three faces, two expressing the same emotion and third expressing a discrepant emotion, were presented simultaneously and side by side for 4500 ms. Participants signalled which of the three faces expressed the discrepant emotion by pressing the 1, 2, or 3 key to identify its location. Keypad responses were recorded during the
A 4500-ms presentation of the faces and up to 7000 ms after the faces were erased. A total of 100 target faces (Karolinska Directed Emotional Faces database; Lundqvist, Flykt, & Öhman, 1998) expressed anger, disgust, sadness, happiness, fear, and surprise at full-blown emotional intensity.

**Verbally labelling emotional expressions at full-blown intensity.** The ability to verbally label a facial expression from a list of basic emotions, anger, disgust, fear, happiness, sadness, and surprise, was measured in a six-alternative forced-choice measure (Palermo et al., 2013). On each trial, a target face expressing one of six full-blown emotional expressions was presented for 600 ms. A total of 144 target faces (Lundqvist et al., 1998) were selected, with 24 from each emotion category. On each trial, participants were required to say out loud the appropriate label from the six options provided. Verbal responses were recorded during the 600 ms presentation period and for a further 10 000 ms during which the six emotion labels remained across the bottom of the screen.

**Learning and recognizing unfamiliar faces.** For the CFMT (Duchaine & Nakayama, 2006), participants recognized images of six target faces in three stages. In the first stage, each of the six target faces was presented as separate items, each appearing for 3000 ms at three viewing angles, after which participants were required to identify the target face from two distractor faces. Participants were asked to identify the target face with novel lighting and viewing angles in stage two and with colored noise added to the images in stage three. A total possible score of 72 is obtained by summing the number of correct items across the three stages.

**Voluntarily controlling facial musculature.** An adaptation of the Upper and Lower Face Apraxia Test (Bizzozero et al., 2000) required participants to make movements of the upper (nine items) and lower (29 items) face that do not
necessarily convey emotion. Upper facial movements comprised forehead, eye, and nose movements and lower facial movements comprised cheek and mouth movements. Some items required sequential movements. All items required visual input while some items required auditory and visual input to reproduce the movement (e.g. whistling). In the original test, the examiner demonstrates each item, and the participant’s response follows immediately after. For the purposes of standardization in our experiments, the instructions and demonstrations of each item were recorded in a short video-clip and watched by all participants, whose responses were filmed for scoring. Participants attempted to reproduce the intensity and duration of each demonstration movement as accurately as possible. Two independent raters, one blind to the experiment, scored the accuracy of each reproduction as a pass (given a score of 1) or fail (given a score of 0). Initial observations of typical errors committed by both groups led to a reclassification of errors. An item was scored as an error in voluntary control of facial musculature if (1) the reproduction was preceded by a pause during which unsolicited movements might have been present; (2) there was a loss of individuation; the instructed movement was executed, but was unfocused and accompanied by uninstructed movement that was intermittently or continuously present, or by an increase in the number of elements in a sequential movement; (3) the reproduction was executed but impoverished, either by reduction of amplitude of movement or by reduction in the number of elements of a sequential movement; (4) there was no movement at all; or (5) there was a content error, where reproductions resembled the demonstrated movement but were incorrect in their content e.g. placing tongue in cheek when asked to puff out the cheek.
Data Analysis

For the psychophysical measures of discrimination of emotional expressions and facial distinctiveness, accuracy was defined as the percent correct at each stimulus level. For Experiment 1, functions were not fitted to calculate absolute thresholds and slope because few participants reached asymptote due to the range limitation in the constant stimuli. Although absolute thresholds and slopes were derived from functions fitted to data in Experiment 2, we chose to not report these for consistency between experiments. We used the mean percent scores across stimulus levels to quantify group difference on the psychophysical measures of emotion discrimination and distinctiveness discrimination. To limit underestimating group differences we excluded performance at the lowest constant-stimulus level which was usually around chance performance. Accuracy was defined as mean percent correct scores for measures of discriminating discrepant from similar expressions, verbally labelling expressions, recognizing unfamiliar face identities, and voluntarily controlling facial musculature (means of two raters’ percent error scores; Cronbach’s alpha was deemed acceptable (Nunnally, 1978) at .98 and .97 for Experiment 1 and Experiment 2).

As suggested by Cumming, Fidler, Kalinowsky, and Lai (2012) in their paper on statistical recommendations for the American Psychological Association, we report measures of effect size (Hedges’ g) to quantify overall differences between control and patient groups. In addition to calculating overall differences between control and patient groups, we also compared control performance with patient performance who, based on ranking their MDS-UPDRS-III motor scores that indicate overall motor disease severity, were allocated to one of two bands of disease severity: low (with scores ranging from
0 to 30; Experiment 1 \( n = 10 \); Experiment 2 \( n = 8 \) and high (with scores greater than 30; Experiment 1 \( n = 24 \); Experiment 2 \( n = 17 \)). Also as recommended by Cumming et al. (2012), we report Pearson correlation coefficients with 95% confidence intervals (CI) to quantify correlations between measures; CI’s that do not include zero indicate a statistically significant correlation, where CI’s that include zero indicate that the correlation is not statistically significant. Partial correlations are reported, removing the variance that is shared with disease severity (UPDRS-III motor score) and age (in years) when these variables correlated with each of the variables entered into the correlation. In Experiment 2 where more than one independent variable (e.g. voluntary control of facial musculature, distinctiveness discrimination, CFMT accuracy) was correlated with the dependent variable, we have adjusted for Type 1 error by dividing the \( p \) value of .05 by the number of independent variables that were correlated with the dependent variable. After removing disease severity, there were no significant correlations between general cognitive functioning, depressive symptoms, sex, or years of education and (1) measures of emotional expression perception, (2) measures of discriminating and identifying facial identity, and (3) measures of voluntary control of facial musculature (Supplementary Table 3).

**Results**

**Experiment 1**

**Impairment in PD in discriminating graded intensities of emotional expressions.** Figure 1 (Panel A) shows the mean accuracy for each group increased with increasing differentiation of the expression from the neutral face for each of the four emotions and with increasing intensity differentiation of the emotional expressions for both intensity ranges. The figure also shows that the patient group
performed more poorly than controls on all measures and that the group difference increased with stimulus separation. Table 2 shows that effect sizes of the group differences were larger than .8, and thus large by conventional standards. Despite the overall large group differences, the box-and-whisker plots (Panel B, Figure 1) show that some patients performed as well as some of the best-performing controls. As Table 3 shows, we found that the effect sizes were substantially greater when comparing control performance with those patients in the higher severity band than those in the lower severity band.
Figure 1. Panel A showing mean percent correct scores (+/- 1 standard error) for patient (open circles) and control (closed circles) groups when discriminating emotion from neutral (left column) and when discriminating different emotional intensities in a low- (middle column) and high intensity range (right column) in Experiment 1. Panel B showing box-and-whisker plots of mean percent correct scores for patient (clear) and control (dark) groups when discriminating emotions (of 10% to 35% emotional intensity) from neutral expressions (left) and when discriminating 10% to 25% differences in intensity of the same emotional expressions within a low- (middle) and high intensity range (right). Results are shown for each of the four emotions presented, anger, disgust, happiness, and sadness.
Table 2
Effect size measures (Hedges’ g) of group differences between patient and control groups on mean percent scores when discriminating emotional expressions (10% to 35% emotional intensity) from neutral expressions and when discriminating 10% to 25% differences in intensity of the same emotional expressions in a low- and high intensity range in Experiment 1 (Control n = 32, Patient n = 34).

<table>
<thead>
<tr>
<th>Emotions</th>
<th>Anger</th>
<th>Disgust</th>
<th>Happiness</th>
<th>Sadness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emotion from Neutral</td>
<td>0.88</td>
<td>0.96</td>
<td>1.35</td>
<td>1.05</td>
</tr>
<tr>
<td>Different intensities (low)</td>
<td>1.22</td>
<td>1.14</td>
<td>1.43</td>
<td>1.24</td>
</tr>
<tr>
<td>Different intensities (high)</td>
<td>1.65</td>
<td>1.48</td>
<td>1.32</td>
<td>1.02</td>
</tr>
</tbody>
</table>

Table 3
Effect size measures (Hedges’ g) of group differences between control groups and PD severity bands (lower and higher) on mean percent scores when discriminating emotional expressions (10% to 35% emotional intensity) from neutral expressions and when discriminating 10% to 25% differences in intensity of the same emotional expressions in a low- and high intensity range in Experiment 1. The lower severity band included patients (n = 10) with MDS-UPDRS motor scores ranging from 0 to 30. The higher severity band included patients (n = 24) with scores greater than 30.

<table>
<thead>
<tr>
<th>Severity band</th>
<th>Low</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emotion from Neutral</td>
<td>.50</td>
<td>2.01</td>
</tr>
<tr>
<td>Different intensities (low)</td>
<td>.65</td>
<td>2.04</td>
</tr>
<tr>
<td>Different intensities (high)</td>
<td>.76</td>
<td>2.14</td>
</tr>
</tbody>
</table>

The link between discriminating emotional expressions and voluntarily controlling facial musculature in PD. The box-and-whisker plots in Figure 2 show that the patient group performed more poorly in the Face Apraxia Test than the control group ($g = .83$), with individual variation in performance in both groups (but more so in the patient group). Errors by PD patients were mostly due to impoverishment of facial movement (49%) followed by individuation loss (40%), whereas errors by controls were mostly due to individuation loss (63%) followed by impoverishment (24%), indicating a shift towards impoverishment with the additional burden of PD. There was a strong, positive, and significant correlation in patient performance on psychophysical measures of emotion discrimination (mean $r$
Aggregate accuracy from all emotion discrimination measures correlated negatively, highly, and significantly with the total percent of errors on the Face Apraxia Test ($r = .73$, 95% CI: -.86, -.52, $p < .05$). After partialling out common variance of disease severity (UPDRS-motor score) and age (in years) with voluntary control of facial musculature and emotion discrimination accuracy, the correlation coefficient was negative, moderate, and significant (partial $r = -.52$, 95% CI: -.73, -.22, $p < .05$).

Figure 2. Box-and-whisker plots showing percent error scores for patient (clear) and control (dark) groups when voluntarily controlling facial musculature in the Upper and Lower Face Apraxia Test (Bizzozero et al., 2000) in Experiment 1.

Experiment 2

Impairment in PD in discriminating graded intensities of emotional facial expressions. Figure 3 shows that, as in Experiment 1, accuracy increased with increasing intensity differentiation for each group. The patient group again performed more poorly than controls, with the group difference increasing with stimulus separation, and with some patients performing as well as some of the best-performing controls. Effect sizes of group differences in accuracy were large for all emotions (anger, $g = 1.74$; disgust, $g = 1.64$; happiness, $g = 1.35$; sadness, $g = 1.35$).
We again found substantially greater effect sizes in the higher severity sub-group \((n = 17, g = 2.24)\) than the lower severity sub-group \((n = 8, g = 1.35)\).

![Figure 3](image)

*Figure 3.* Panel A showing mean percent correct scores (+/- standard error) for patient (open circles) and control (closed circles) groups when discriminating different intensities of the same expressions in a mid-intensity range in Experiment 2. Box-and-whisker plots in Panel B showing mean percent correct scores for patient (clear) and control (dark) groups when discriminating intensity differences of 6% to 48%. Results are shown for each of the four emotions presented, anger, disgust, happiness, and sadness.
For the 40 participants who completed both experiments (Control $n = 18$, Patient $n = 22$), there was a positive, strong, and significant correlation between psychophysical measures of discriminating emotional expressions from the two experiments ($r = .87$, 95% CI: .77, .93, $p < .05$), illustrating the reliability of the measures. The effect sizes that quantified group differences on emotion discrimination measures for those participants who completed both experiments were identical in each experiment (both $g$’s = 1.79).

**Impairment in PD in discriminating discrepant from similar full-blown expressions and in verbally labelling full-blown expressions.** Figure 4 shows two features of interest that are similar to the results from psychophysical measures of emotion discrimination from each experiment. First, patients performed more poorly than controls when discriminating discrepant from similar full-blown expressions ($g = .92$) and when verbally labelling full-blown expressions ($g = .96$). Second, both groups showed variability in each of the measures, with generally more variability in the patient group, and with some patients performing as well, or almost as well, as the best-performing controls. Effect sizes were again substantially greater when comparing control performance with performance in the higher- than lower severity sub-group when discriminating discrepant from similar expressions (lower severity $g = .13$; higher severity $g = 1.71$) and when verbally labelling expressions (lower severity $g = .29$; higher severity $g = 1.51$).
Figure 4. Box-and-whisker plots showing mean percent correct scores for patient (clear) and control (dark) groups when discriminating discrepant from two similar emotional expressions (panel A) and when verbally labelling six basic expressions of emotion (panel B) in Experiment 2.

The link between measures of perception of emotional expressions in PD.

There were positive, moderate to strong, and significant correlations in patient performance between measures of discriminating differences in intensity of the same emotional expressions, discriminating discrepant from similar expressions, and verbally labelling emotional expressions (mean $r = .67$, 95% CI: .37, .84, $p < .05$). To limit the number of comparisons made, we use the aggregate accuracy from all measures of emotion discrimination and recognition in the correlational analyses with measures of discriminating facial distinctiveness, recognizing facial identity, and voluntary control of facial musculature.

The links between perception of emotional expressions, facial distinctiveness discrimination, and facial identity recognition in PD. Figure 5 shows that on average patients performed more poorly than controls when discriminating differences in facial distinctiveness; similar to the group differences in perceiving emotional expressions, the effect size of group differences in accuracy was large ($g = 1.44$). The same pattern, poorer performance in the patient than
A control group was seen in accuracy on the CFMT (Controls $M = 67, SD = 13$, $g = .98$; Patient $M = 55, SD = 10$). After removing the common variance shared with disease severity and age, there was a positive, moderate to strong, and significant correlation between aggregate accuracy from measures of emotional expression perception and the measure of facial distinctiveness discrimination (partial $r = .67$, 95% CI: .37, .84, $p < .016$), but there was no significant correlation with facial identity recognition in the CFMT (partial $r = .11$, 95% CI: -.30, .48, $p > .016$).

![Figure 5. Mean percent correct scores (+/- 1 standard error) and a box-and-whisker insert for patient (open circles/clear) and control (closed circles/dark) groups when discriminating differences in facial distinctiveness in Experiment 2.]

The link between perception of emotional expressions and voluntary control of facial musculature in PD. As in Experiment 1, the patient group ($M = 39, SD = 14$) showed higher mean percent errors than the control group ($M = 25, SD = 12$) on the adapted Face Apraxia Test ($g = 1.05$). There was again a shift in PD towards impoverishment of facial movement, where errors by patients were mostly due to impoverishment (48%) followed by individuation loss (42%), whereas errors by controls were mostly due to individuation loss (54%) followed by impoverishment (31%). After partialling out disease severity and age, there was a
negative, moderate, and significant correlation between voluntary control of facial musculature and aggregate accuracy from emotional expression perception measures (partial $r = -.59$, 95% CI: -.80, -.26, $p < .016$). After removing disease severity and age, there was no significant partial correlation between voluntary control of facial musculature and facial distinctiveness discrimination (partial $r = -.26$, 95% CI: -.59, .15, $p > .025$) and facial identity recognition (partial $r = -.20$, 95% CI: -.55, .33, $p > .025$).

**The Link between Perception of Emotional Expressions and Disease Severity in PD after Partialling out Potential Demographic and Clinical Confounds**

In both experiments, we found that impaired perception of emotional expressions in PD was a function of disease severity. We considered the possibility that larger impairment in the higher- than lower severity bands on measures of emotional expression perception are due to an increase in general cognitive functioning impairment, age, or depressive symptoms in the higher severity band. To do this, we correlated disease severity with measures of emotional expression perception in PD, after removing the variance shared with general cognitive functioning, depressive symptoms, and age, in each of the two experiments. Disease severity correlated negatively, moderately, and significantly with psychophysical measures of emotional expression discrimination in Experiment 1 (partial $r = -.49$, 95% CI: -71, -18, $p < .05$) and with measures of emotional expression perception in Experiment 2 (partial $r = -.50$, 95% CI: -.75, -.13, $p < .05$), even after removing the variance shared with age, general cognitive functioning, and depressive symptoms.

**Discussion**

The two experiments show impaired discrimination of emotional expressions of graded intensity and impaired discrimination and recognition of full-blown
emotional expressions in those with PD. There were large group differences between patient and control groups on all measures, with the relative emotion perception impairment shown to be a function of disease severity that had little to no correlation with general cognitive functioning, depressive symptoms, sex, and number of years of education. The second experiment also showed impaired discrimination of facial distinctiveness and impaired recognition of facial identity in PD where distinctiveness discrimination, but not identity recognition, correlated with measures of emotion perception after removing the effect of disease severity and age. Both experiments showed that, after removing disease severity and age, the ability to voluntarily control facial musculature correlated with the ability to perceive emotional information from faces, but not with the ability to perceive non-emotional information from faces.

The impairment in PD in extracting emotional content from faces is not exclusive to full-blown expressions of emotion as shown in the majority of previous research (Gray & Tickle-Degnen, 2010) and replicated in the current findings but is also evident in discriminating facial expressions of emotion expressed at sub-maximal intensities. Impaired verbal labelling of sub-maximal intensities of emotional expressions in PD was recently shown by Buxton et al. (2013) and previously by Dujardin et al. (2004). The impairment in both discrimination and recognition of emotional expressions shown, where psychophysical discrimination measures do not place an added demand on verbal processes, suggests a cascade of lower- to higher-order impairment in perception of facial expression of emotion in PD. Unlike the meta-analysis (Gray & Tickle-Degnen, 2010), which showed no relationship between emotion perception and disease severity measured by the Hoehn and Yahr Staging Scale (Hoehn & Yahr, 1967), we found performance in the
patient group to be a function of disease severity measured by the MDS-UPDRS; effect sizes were substantially greater when comparing controls with patients in a higher severity than in a lower severity sub-group. Furthermore, the link between disease severity and perception of emotional expression in PD was maintained even after removing the variance shared with general cognitive functioning, age, and depressive symptoms. It is likely that the insensitivity of the Hoehn and Yahr Scale (Goetz et al., 2004) obscured the link between disease severity and emotion perception in previous research. Similarly, unlike findings in the meta-analysis that indicated patients were not impaired in identity recognition, our data show impaired discrimination and recognition of facial identity in PD. The null findings by Gray and Tickle-Degnen (2010) are likely due to insensitive measures of identity recognition (Duchaine & Weidenfeld, 2003).

The moderately-sized link between voluntary control of facial musculature and emotional expression perception in PD, and the smaller link with identity perception, is consistent with the embodied simulationist account that simulation of an observed expression of emotion contributes to perception of that expression of emotion. According to embodied simulationist accounts of emotion perception, the link between voluntary control of facial musculature and perception of emotional expressions in PD is mediated by simulation of observed emotional expressions, which is disrupted, thereby contributing to impaired perception of emotional expressions. The process by which simulation occurs might be overt, covert, or both (see Goldman & Sripada, 2005). Interpretations of the link between voluntary control of facial musculature and perception of emotional expressions in PD from an overt simulationist perspective will differ from that of a covert simulationist perspective. According to the overt account of embodied simulation in emotion
perception, PD patients with impaired voluntary control of facial musculature will be unable to physical simulate or mimic the expressions of others, thereby contributing to impaired perception of emotional expressions in PD. According to the covert account of embodied simulation, neural systems with mirror-like properties that are engaged by voluntary control of facial musculature and simulation of observed expressions are disrupted by the neurodegenerative processes in PD, thereby contributing to both impaired voluntary control of facial musculature and perception of emotional expressions. The present data do not rule out either overt or covert account of embodied simulation, as we did not measure the process of simulation directly. However, data from others (Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000; Anders et al., 2012; Calder, Keane, Cole, Campbell, & Young, 2000; Carr, Iacobini, Dubeau, Mazziotta, & Lenzi, 2003; Damasio, 1994; Hess & Blairy, 2001; Keillor, Barrett, Crucian, Kortenkamp, & Heilman, 2002; Wicker et al., 2003) suggest this simulation is likely a covert process. In line with covert embodied simulationist accounts of emotion perception, evidence for a human mirror neuron system in healthy individuals in ventrolateral premotor cortex and pars opercularis of the inferior frontal gyrus among others, which is activated during facial musculature control in producing facial expressions of emotion and observation of facial expressions of emotion, has been reported (Carr et al., 2003; Wicker et al., 2003). Support for a covert embodied simulationist account comes from Anders et al. (2012) who found that Parkin mutation carriers (who show mild reduction of dopamine metabolism in the basal ganglia in the absence of clinical motor signs) were as a group slightly impaired relative to controls in emotional expression recognition; carriers who were least impaired showed the strongest ventrolateral premotor cortex activity, consistent with an embodied simulationist
hypothesis that compensatory activity in premotor cortex reduced the impairment. In addition, overt facial mimicry seems unlikely when discriminating facial expressions of emotions at the brief presentation durations (200 ms) in the psychophysical measures. Furthermore, there is accumulating evidence that physical mimicry of the expressions of others is not critical for intact emotional expression recognition in those with congenital facial muscular paralysis (Calder et al., 2000; Keiller et al., 2002) and in healthy observers (Hess & Blairy, 2001). Therefore, in line with the covert embodied simulation account, disruption of systems with mirror-like properties that are engaged by voluntary control of facial musculature and simulation of observed expressions might explain in part impaired voluntary control of facial musculature and impaired perception of emotional expressions in PD. As a limitation, the correlations between voluntary facial musculature control and emotion perception we report is an indirect examination of the covert embodied simulation account. Given that we did not manipulate covert simulation as a more direct approach, these findings do not provide strong definitive evidence for the covert embodied simulationist account. That this simulation is a covert process is speculative given our present data. However, with evidence against overt simulation (Calder et al., 2000; Hess & Blairy, 2001; Keillor et al., 2002) and evidence that supports covert simulation (Anders et al., 2012; Carr et al., 2003; Wicker et al., 2003), we interpret that the link between voluntary facial musculature control and emotion perception is mediated by covert simulation, a system that might be disrupted by the neurodegenerative processes in PD, thereby contributing to impaired voluntary facial musculature control and emotional expression perception. The association of emotion production and recognition in PD reported by others (Benke, Bosch, & Andrew, 1998; Borod et al., 1990; Jacobs et al., 1995) might be
mediated at least to some extent by the same sensorimotor neural circuitry that allows inference of emotion from facial expressions. That facial distinctiveness discrimination was unrelated to voluntary control of facial musculature in PD suggests that the link between emotional expression discrimination and voluntary control of facial musculature was not mediated by a more general impairment in visually extracting any information from faces.

The link between measures of emotional expression perception and facial distinctiveness discrimination suggests that visual sensory processes also contribute to impaired emotional expression perception in PD. The view that a broader underlying inability to extract visual information from faces impairs perception of emotional expressions in PD has also been supported by the correlation between recognition of emotional expressions and discrimination of spatial differences in facial features (Narme et al., 2011) and by reports of abnormal visual scanning patterns during recognition of emotional expressions (Clark et al., 2010) in PD. Links between perception of emotional and non-emotional information from faces are inconsistent with the dominant theory of face perception (Bruce & Young, 1986) that makes a division between perception of emotional and non-emotional information on faces; a neuroanatomical division of these processes has also been proposed (Haxby, Hoffman, & Gobbini, 2000). However, findings from others (see Vuilleumier & Pourtois, 2007, for a review) have led to the proposition for an interaction of identity and emotion perception where both share perceptual encoding mechanisms that enable perception. Our data suggest an interaction of identity and emotional expression perception processes, as evidenced by the link between distinctiveness discrimination and emotional expression perception; both might be impaired by a broader underlying visual impairment in PD (Archibald, Clarke,
Mosimann, & Burn, 2009, for a review). Our data also suggest bifurcation of these processes, as evidenced by the link between voluntary facial musculature control and emotional expression perception, but not with identity, and by little or no correlation between identity recognition (CFMT performance) and emotional expression perception. Therefore, it appears that sensory and sensorimotor processes are impaired in PD and that both contribute to deficits in visually deciphering emotional content from faces.

Together, the results of these experiments show impairment in PD in discriminating varying intensities of emotional from neutral expressions and discriminating varying intensities of the same emotional expressions, and replicate previous reports of impairment in discriminating and recognizing full-blown emotional expressions, all of which is a function of disease severity. While we show that this impairment is unrelated to general cognitive functioning, we cannot determine the role of decline in specific cognitive functions in impaired emotional expression perception in PD. Our results support the likelihood of a multifactorial contribution to impaired perception of emotional expressions in PD, which includes, but is not necessarily limited to, disrupted sensory and sensorimotor processes.
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to Mini-Mental State examination score. *Journal of the American Geriatric Society, 57*(2), 304-308.


event-related brain potentials. *Cortex, 48*(9), 1207-1217.


**Supplementary Materials**

Table 1  
*Pairings of the different emotional intensities used to define each intensity difference for the task requiring discrimination of graded intensities for the low intensity and high intensity ranges in Experiment 1, and for the mid intensity range in Experiment 2.*

<table>
<thead>
<tr>
<th>EXPERIMENT 1</th>
<th>Intensity range</th>
<th>5%</th>
<th>10%</th>
<th>15%</th>
<th>20%</th>
<th>25%</th>
</tr>
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<tbody>
<tr>
<td>Low range</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>Pair 1</td>
<td>10,15</td>
<td>10,20</td>
<td>10,25</td>
<td>10,30</td>
<td>10,35</td>
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<td>Pair 2</td>
<td>15,20</td>
<td>15,25</td>
<td>15,30</td>
<td>15,35</td>
<td>15,40</td>
<td></td>
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<tr>
<td>Pair 3</td>
<td>20,25</td>
<td>20,30</td>
<td>20,35</td>
<td>20,40</td>
<td>20,45</td>
<td></td>
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<tr>
<td>Pair 4</td>
<td>25,30</td>
<td>25,35</td>
<td>25,40</td>
<td>25,45</td>
<td>25,50</td>
<td></td>
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<td>High range</td>
<td></td>
<td></td>
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<tr>
<td>Pair 1</td>
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<td>55,70</td>
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<td>60,75</td>
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<td>Pair 4</td>
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<td>65,75</td>
<td>65,80</td>
<td>65,85</td>
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<table>
<thead>
<tr>
<th>EXPERIMENT 2</th>
<th>Intensity range</th>
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<th>6%</th>
<th>12%</th>
<th>24%</th>
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<td>Mid-range</td>
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<td></td>
</tr>
<tr>
<td>Pair 1</td>
<td>22,25</td>
<td>22,28</td>
<td>22,34</td>
<td>22,46</td>
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<td>Pair 2</td>
<td>39,42</td>
<td>38,44</td>
<td>36,48</td>
<td>32,56</td>
<td>24,72</td>
<td></td>
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<td>Pair 3</td>
<td>56,59</td>
<td>54,60</td>
<td>50,62</td>
<td>42,66</td>
<td>26,74</td>
<td></td>
</tr>
<tr>
<td>Pair 4</td>
<td>73,76</td>
<td>70,76</td>
<td>64,76</td>
<td>52,76</td>
<td>28,76</td>
<td></td>
</tr>
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</table>

Table 2  
*Pairings of the different distinctiveness intensities used to define each distinctiveness difference for the task requiring discrimination of differences in distinctiveness in Experiment 2.*

<table>
<thead>
<tr>
<th>Intensity range</th>
<th>30%</th>
<th>40%</th>
<th>60%</th>
<th>90%</th>
<th>120%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pair 1</td>
<td>40, 70</td>
<td>40, 80</td>
<td>40, 100</td>
<td>40, 130</td>
<td>40, 160</td>
</tr>
<tr>
<td>Pair 2</td>
<td>90, 120</td>
<td>90, 130</td>
<td>80, 140</td>
<td>60, 150</td>
<td>50, 170</td>
</tr>
<tr>
<td>Pair 3</td>
<td>150, 180</td>
<td>140, 180</td>
<td>120, 180</td>
<td>90, 180</td>
<td>60, 180</td>
</tr>
</tbody>
</table>
Table 3
Partial correlation coefficients (with 95% CI’s in parentheses), after removing disease severity (MDS-UPDRS motor score), between general cognitive functioning (MoCA score), depressive symptoms (GDS score), sex, years of education and (1) mean percent correct scores of measures of emotion discrimination in Experiment 1 (n = 34), (2) mean percent correct scores on measures of emotion discrimination and recognition in Experiment 2 (n = 25), (3) mean percent correct scores of discriminating facial distinctiveness and mean percent correct scores of recognizing facial identity (CFMT) in Experiment 2 (n = 25), and (4) mean percent correct scores across both experiments of psychophysical measures of emotion discrimination and voluntary control of facial musculature (n = 37).

<table>
<thead>
<tr>
<th></th>
<th>MoCA</th>
<th>GDS</th>
<th>SEX</th>
<th>EDUCATION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EXPERIMENT 1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychophysical emotion discrimination</td>
<td>.17 (.17, .48)</td>
<td>.17 (.17, .48)</td>
<td>-.08 (-.41, .27)</td>
<td>.17 (-.18, .48)</td>
</tr>
<tr>
<td><strong>EXPERIMENT 2</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotion discrimination and recognition</td>
<td>.32 (-.09, .63)</td>
<td>.19 (-.22, .55)</td>
<td>.23 (-.18, .57)</td>
<td>.27 (-.14, .60)</td>
</tr>
<tr>
<td>Discriminating facial distinctiveness</td>
<td>.31 (-.10, .63)</td>
<td>.39 (-.01, .68)</td>
<td>-.06 (-.44, .34)</td>
<td>-.03 (-.42, .37)</td>
</tr>
<tr>
<td>Recognizing facial identity</td>
<td>.08 (-.33, .46)</td>
<td>-.28 (-.61, .13)</td>
<td>.32 (-.09, .63)</td>
<td>-.28 (-.61, .13)</td>
</tr>
<tr>
<td><strong>POOLED DATA ACROSS EXPERIMENTS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychophysical emotion discrimination</td>
<td>.17 (.16, .47)</td>
<td>.08 (-.25, .39)</td>
<td>.17 (-.16, .47)</td>
<td>.25 (-.08, .53)</td>
</tr>
<tr>
<td>Voluntary control of facial musculature</td>
<td>-.22 (-.51, .11)</td>
<td>-.15 (-.45, .28)</td>
<td>-.15 (-.45, .18)</td>
<td>-.31 (-.58, .02)</td>
</tr>
</tbody>
</table>

Note. *p < .05; **adjusted p < .0125 (adjusting for multiple correlations by dividing the significance level of .05 by the number of independent variables that is correlated with each dependent variable); for the pooled data across experiments, we pooled the accuracy on each of these measures for patients unique to each of the experiments (Experiment 1 n = 12, Experiment 2 n = 3) with the averaged accuracy on measures across experiments of those who did both experiments (n = 22).
CHAPTER 5: Discriminating Facial Expressions of Emotion and its Link with Perceiving Visual Form in Parkinson’s Disease

Foreword

The previous chapter showed large impairments in PD in discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions in Experiments 2 and 3. In these two experiments, brief stimulus durations (200 ms) and the two-interval forced-choice (2IFC) format of the psychophysical measures of emotion discrimination might have inflated the impairment in PD due to slower visual processing times and working memory demands of the 2IFC procedure. The next chapter describes a follow-up experiment on patients and controls in which the psychophysical measure of discriminating graded intensities of anger from neutral expressions was administered with two stimulus durations (200 ms and 1000 ms) and with a 2IFC and yes-no procedure. A similar level of impairment on the yes-no measure and with prolonged stimulus durations will suggest that slower visual processing times and working memory demands of the 2IFC procedure did not exaggerate the impairment in PD in discriminating graded intensities of emotional from neutral expressions. The previous chapter also showed that the ability to extract distinctiveness information from faces contributed to impaired perception of facial expressions of emotion in PD; both of these abilities might be affected by a more general impaired ability to perceive visual form. Deficits in PD in a range of basic visual functions are extensively documented (Archibald, Clarke, Mosimann, & Burn, 2009, for a review). The follow-up experiment that is described in the next chapter investigated the contribution of perception of visual form, measured as discriminating radial
frequency (RF) patterns of varying amplitude modulations from perfect circles, to impaired perception of emotional expressions in PD.
Abstract

We investigated the link between the ability to perceive facial expressions of emotion and the ability to perceive visual form in Parkinson’s disease (PD). We assessed in persons with PD and healthy controls the ability to discriminate graded intensities of facial expressions of anger from neutral expressions and the ability to discriminate radial frequency (RF) patterns with modulations in amplitude from a perfect circle. Those with PD were, as a group, impaired relative to controls in discriminating graded intensities of anger from neutral expressions and discriminating modulated amplitudes of RF patterns from perfect circles; these two abilities correlated positively and moderately to highly, even after removing the variance that was shared with disease severity. The results indicate that the impaired ability to perceive visual form is likely to contribute to the impaired ability to perceive facial expressions of emotion in PD, and that both are related to the progression of the disease.
Introduction

A recent meta-analytic review of perception of facial expressions of emotion in Parkinson’s disease (PD) concluded that those with PD were impaired in perceiving facial expressions of emotion and that this impairment was unrelated to the ability to perceive visual form (Gray & Tickle-Degnen, 2010). The studies reviewed measured this latter ability with the Benton Facial Recognition Test (BFRT; Benton, Sivan, Hamsher, Varney, & Spreen, 1994) or a similar test, all of which are traditionally viewed as measures of face recognition. The BFRT has been shown to be sensitive only to large impairments in facial identity recognition (Duchaine & Weidenfeld, 2003). There have been two reports that those with PD are impaired in perceiving facial information other than emotion (Narme, Bonnet, Dubois, & Chaby, 2011; Marneweck, Palermo, & Hammond, 2014, June 16); these studies used measures of extracting non-emotional facial information that might have been more sensitive to differences between PD and control groups. Furthermore, both studies showed positive correlations between the abilities to perceive emotional and non-emotional information from faces (Narme et al., 2011; Marneweck et al., 2014, June 16). Both the ability to perceive emotional and non-emotional information from faces might be affected by a more general impairment of the ability to perceive visual form. A range of basic visual functions are known to be impaired in PD (Archibald, Clarke, Mosimann, & Burn, 2009, for a review). Whether there is a link between the ability to perceive facial expressions of emotion and the ability to perceive visual form in PD is yet to be tested.

In previous work we found large impairments in those with PD in discriminating emotional expressions of graded intensity from neutral expressions and discriminating variations in intensity of the same emotional expressions of four
commonly expressed emotions, anger, disgust, happiness, and sadness (Marneweck et al., 2014, June 16). These psychophysical measures used a two-interval forced-choice (2IFC) procedure, with, on each trial, successive presentation of stimuli with brief stimulus durations and a brief inter-stimulus interval. Given previous reports of impairment in PD in working memory (Lee et al., 2010) and in the processing time of visually presented information (Johnson et al., 2004), it is possible that the sequential stimuli in the 2IFC procedure and the brief stimulus durations exaggerated the impairment in PD.

The aims of the current experiment were two-fold. First, we investigated the link between perception of visual form and perception of facial expression of emotion in PD. To investigate perception of visual form, we measured the ability to discriminate radial frequency (RF) patterns of varying modulations in amplitude from a perfect circle. RF patterns, first created by Wilkinson, Wilson, and Habak (1998), are a family of smooth closed shapes that differ from each other and each from a perfect circle in a clearly defined way: different patterns can be created by modulating the radial frequency (the number of lobes), amplitude (affecting the sharpness or depth of the lobe), and the orientation (the direction of the lobe). To investigate the link between the abilities to perceive visual form and facial expressions of emotion in PD, we correlated measures of discriminating RF3 patterns from perfect circles with measures of discriminating graded intensities of anger from neutral expressions. As a second aim, we investigated whether impairment in PD in discriminating anger from neutral facial expressions was present when measured with longer stimulus durations in a 2IFC procedure (to reduce any effects of slower visual processing times) and with a single-stimulus yes-
no procedure (to reduce the working memory demands imposed by sequential stimuli).

**Methods**

**Participants**

Forty-two participants (Control $n = 18$, PD $n = 24$) were tested. Table 1 shows the demographic and clinical characteristics of each group; groups were on aggregate well matched for age, sex, and scores on measures of general cognitive functioning (Montreal Cognitive Assessment, MoCA; Nasreddine et al., 2005) and depressive symptoms (Geriatric Depression Scale, GDS; Sheikh & Yesavage, 1986). Patients were diagnosed by a neurologist and recruited from the Edith Cowan University Parkinson’s Centre research database and from previous research participation. Control participants were recruited from the local community and from previous research participation. The Institutional Ethics Committee approved the experimental procedures and all participants gave written informed consent.

**Table 1**

*Demographic and clinical characteristics of participant groups.*

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>CONTROL</th>
<th>PD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>70 (53-80)</td>
<td>68 (58-82)</td>
</tr>
<tr>
<td>Males (Females)</td>
<td>12 (6)</td>
<td>16 (8)</td>
</tr>
<tr>
<td>Education years</td>
<td>16 (11-20)</td>
<td>14 (9-19)</td>
</tr>
<tr>
<td>MoCA</td>
<td>27 (22-30)</td>
<td>27 (19-29)</td>
</tr>
<tr>
<td>GDS</td>
<td>1 (0-4)</td>
<td>1 (0-10)</td>
</tr>
<tr>
<td>Years diagnosed</td>
<td>-</td>
<td>8 (2-22)</td>
</tr>
<tr>
<td>MDS-UPDRS-III</td>
<td>-</td>
<td>40 (19-57)</td>
</tr>
<tr>
<td>LED</td>
<td>-</td>
<td>1057 (0-2662)</td>
</tr>
</tbody>
</table>

*Note.* All values (except number of males and females) are expressed as median (minimum-maximum range); MoCA = Montreal Cognitive Assessment, score ranges from 0 (most severe) to 30, a score ≥ 26 reflects normal cognitive functioning; GDS = Geriatric Depression Scale, score ranges from 0 to 15 (most severe), a score of 6 or more is suggestive of depression that should warrant thorough assessment; MDS-UPDRS-III = motor scale of Unified Parkinson’s Disease Rating Scale, score ranges from 0 to 132 (most advanced disease state); LED = Daily Levodopa Dose Equivalent (Tomlinson et al., 2010).
**Materials and Procedures**

Participants completed in one session that lasted between 1.5 to 2 hours psychophysical measures of (1) discriminating graded intensities of anger from neutral expressions in a 2IFC procedure and in a yes-no procedure with the Method of Constant Stimuli, and (2) psychophysical measures of discriminating RF patterns with varying amplitude modulations from perfect circles using a 2IFC procedure with the Method of Constant Stimuli. Patients were initially assessed for severity of motor symptoms using the Movement Disorders Society-sponsored revision of the Unified Parkinson’s Disease Rating Scale (MDS-UPRDS; Goetz et al., 2008). Patients completed the testing session approximately 2 hours before their next dopamine replacement medication.

For the measure of emotional expression discrimination, we selected four models expressing full-blown anger from the NimStim Face Stimulus Set (Tottenham et al., 2009); we graded these expressions in emotional intensity by morphing full-blown expressions of each model with their neutral expression (see Marneweck, Loftus, & Hammond, 2013, for details). Given that our previous findings showed a similar level of impairment in PD for each of the four emotions (anger, disgust, happiness, and sadness), we only tested anger. Each face showing an angry or neutral expression was set in a rectangular white background that was 6.8 cm high and 5.4 cm wide subtending visual angles of 6.6° and 5.2° at a viewing distance of 59 cm. The order of the 2IFC procedure and the yes-no procedure of the emotional expression discrimination measure was counterbalanced. On each trial of the 2IFC procedure, two faces of the same model appeared successively on a computer screen at a duration of 200 ms or 1000 ms with a 200-ms blank inter-stimulus interval. The face with the neutral
expression appeared randomly in either the first or the second interval and the face expressing one of five levels of intensity of anger (set at 5, 9, 16, 29, and 52% of the full-blown expression) appeared in the other interval. On each trial participants signalled the interval containing the angry face by clicking either the left or right button on a mouse to indicate the first or second interval respectively. There were ten randomized blocks of 40 trials (with a break after the fifth block), with each block containing five intensities of anger each expressed by each of the four models at two stimulus durations, giving 400 trials in total. For the emotion discrimination measure with the yes-no procedure, each trial showed one face either with a neutral expression or with an expression of anger at one of three emotional intensities (9%, 16%, 29%); each face was presented at a duration of 200 ms or 1000 ms. Participants indicated with mouse-click whether the face was emotional by clicking the left mouse button or neutral by clicking the right mouse button. There were ten randomized blocks of 48 trials (with a break given after the fifth block) with each block containing three neutral expressions and three intensities of anger by each of four models at two stimulus durations, giving 480 trials in total.

For the measure of discriminating RF patterns with varying amplitude modulations from perfect circles (with a 2IFC procedure with the Method of Constant Stimuli), RF patterns were created by application of a sinusoidal modulation to the radius of a perfect circle, with three cycles of modulation around the full $2\pi$ radians, producing an RF3 pattern (see Figure 1). The distance from the center to a specific point in the modulated pattern, $r'$, is given by: $R0 * (1 + A * \sin (w * \angle + \phi))$, where $R0$ is the unmodulated radius of the pattern, $A$ is the maximum modulation amplitude expressed as the proportion of the radius of the unmodulated
pattern, $\angle$ is the polar angle that defines a direction in the fronto-parallel plane relative to the center of the pattern, $\phi$ is a polar angle that allows the pattern to be rotated in the fronto-parallel plane, and $w$ is the frequency of modulation. Five depths of amplitude modulation were created .002, .004, .008, .016, and .032 in proportion to the radius of the circle. There were four variations of each amplitude modulation such that the apex of the RF3 pattern appeared either at the top, left, right, or bottom of the pattern. The Weber contrast was set to -.7 to avoid visible persistence of the first stimulus which was present at higher contrast. The stimulus size, viewing distance, and inter-stimulus interval were identical to those in the 2IFC measure of emotional expression discrimination; the stimulus duration was set at 200 ms. One each trial, the perfect circle appeared in either the first or second interval and the RF3 pattern with one of five amplitude modulations appeared in the other interval. Participants indicated with a mouse-click whether the RF3 pattern appeared in the first or the second interval by clicking the left or right button, respectively. There were ten randomized blocks of 20 trials, with each block containing the five variations of amplitude modulation each with four apex orientations, giving 200 trials in total.

*Figure 1.* RF3 patterns with specifications of variation in amplitude modulation (in proportion to the radius of the circle) and apex orientation.
Data Analysis

Individual data obtained with the 2IFC psychophysical measures of discrimination of angry expressions and RF3 patterns were fitted with Cumulative Gaussian functions which generally fitted the individual data well; the median $R^2$ value was .94 for both discriminating anger from neutral expressions and for discriminating RF3 patterns from perfect circles. Absolute thresholds were taken as the stimulus level at which 75% correct performance was reached. For the emotion discrimination measure, there were two determinations for each participant, one for each of the two stimulus durations, giving 84 determinations in total. For the RF3 pattern discrimination measures, there was one determination for each participant giving 42 determinations in total. Thresholds could not be obtained for six of the 84 determinations for the emotion discrimination measure (two of which came from the same participant) and for one of the 42 determinations for the RF3 pattern discrimination measure, because the range of constant stimuli did not capture the complete psychometric function or because the fit to the individual data points was poor. One of the determinations in the emotion discrimination and RF3 pattern discrimination measures respectively for which thresholds could not be obtained came from one of the PD patients. For the emotion discrimination measure, seven thresholds (200 ms duration $n = 3$; 1000 ms duration $n = 4$) that were obtained from patients exceeded 100; these thresholds were set arbitrarily to 100, which is conceptually equivalent to a full-blown emotional expression. For the emotion discrimination measure with a yes-no procedure, we calculated d-prime as a measure of sensitivity for each of the three intensities of angry expressions. We report measures of effect size (Hedges’ $g$) to quantify group differences for emotion and
RF3 pattern discrimination measures. We also report a mixed analyses of variance to examine the effect of stimulus duration and the interaction between stimulus duration and group for the emotion discrimination measure with the yes-no procedure. To quantify the relationship between emotion and RF3 pattern discrimination in PD, we report Pearson correlation coefficients with 95% confidence intervals (CI). Instead of using absolute threshold scores in the correlations between 2IFC measures of RF3 pattern and emotion discrimination, which could not be obtained for all patients, we use mean percent correct scores across stimulus durations and stimulus levels; we exclude the lowest stimulus level at which most participants performed at chance. We similarly use the mean d-prime values across stimulus durations and stimulus levels (excluding the lowest stimulus level) of the emotion discrimination measure with the yes-no procedure for its correlation with RF3 pattern discrimination. There was little or no correlation between measures of emotion discrimination and general cognitive functioning and depressive symptoms, with 95% CI’s overlapping zero; for brevity, these are reported in Supplementary Material (Table 1).

**Results**

Figure 2 shows functions fitted to mean data points for PD and control groups for the 2IFC measure of discriminating anger from neutral expressions with 200 ms and 1000 ms stimulus durations. All functions increased monotonically with increasing differentiation of the expressive from the neutral face. The figure also shows that the PD group performed more poorly than the control group at both stimulus durations, and that an increase in stimulus duration made little or no difference to performance in either group. Table 2 shows mean absolute thresholds were higher for the PD than control group, with very large effect sizes for the group
differences at both stimulus durations. The table also shows that there was greater variability in thresholds in the PD than control group, and that some in the PD group had thresholds similar to some of the best-performing controls.

![Figure 2](image)

*Figure 2.* Cumulative Gaussian functions fitted to mean percent correct scores for PD (gray) and control (black) groups when discriminating graded intensities of anger from neutral expressions with a 2IFC procedure. Dashed lines and unfilled circles show data for stimulus durations of 200 ms and solid lines and filled circles show data for stimulus durations of 1000 ms.

Table 2

Mean estimates of absolute threshold (with standard deviation followed by range in parentheses), taken as the emotional intensity level at which 75% correct performance was reached, for the 2IFC measure of discriminating graded intensities of anger from neutral expressions for PD (200 ms, n = 21; 1000 ms, n = 22) and control groups (200 ms, n = 18; 1000 ms, n = 17), with Hedges’ g quantifying the difference between groups.

<table>
<thead>
<tr>
<th></th>
<th>CONTROL</th>
<th>PD</th>
<th>g</th>
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<tbody>
<tr>
<td>200 ms duration</td>
<td>17 (6; 7-28)</td>
<td>38 (23; 10-100)</td>
<td>1.13</td>
</tr>
<tr>
<td>1000 ms duration</td>
<td>16 (4; 9-24)</td>
<td>32 (21; 11-100)</td>
<td>1.00</td>
</tr>
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</table>

Figure 3 shows mean d-prime values for both groups for each of the three intensity increments at each stimulus duration for emotion discrimination with the yes-no procedure. Sensitivity to expressions of anger increased with increasing
intensity of the expression ($F (2, 78) = 195.89, p < .05, \eta_p^2 = .83$). The PD group showed lower sensitivity than the controls at both stimulus durations; the effect size of the mean group differences across intensity increments was large for both stimulus durations (200 ms $g = 1.35$; 1000 ms $g = 1.00$). Although there was a significant effect of stimulus duration ($F (1, 39) = 22.88, p < .05, \eta_p^2 = .37$), indicating a greater sensitivity to angry expressions at the longer stimulus duration (which is shown in Figure 3), there was no interaction between stimulus duration and group ($F (1, 39) = .007, p > .05, \eta_p^2 = .00$), indicating that the longer duration was no more beneficial for the PD than control group.

![Figure 3](image.png)

*Figure 3.* Mean d-prime values (+/- 1 standard error) for PD (gray) and control (black) groups when discriminating graded intensities of anger from neutral expressions with a yes-no procedure. Dashed lines show data for stimulus durations of 200 ms, and solid lines show data for stimulus durations of 1000 ms.

Figure 4 shows functions fitted to mean data points for PD and control groups for the measure of discriminating RF3 patterns of varying amplitude modulations from perfect circles. Functions of both groups increased monotonically with increasing amplitude modulation, with the PD group performing more poorly than controls. As was the case in the measure of emotion discrimination, absolute
thresholds were higher and more variable in the PD ($M = .031$, $SD = .025$) than control group ($M = .009$, $SD = .003$), with a large effect size for the group difference ($g = 1.03$). Again, some with PD performed as well as some of the best-performing controls (PD Range: .005, .075; Control Range: .004, .017).

Figure 4. Cumulative Gaussian functions fitted to mean percent correct scores for PD (gray) and control (black) groups when discriminating RF3 patterns of varying amplitude modulations from perfect circles.

The correlations of discriminating RF3 patterns from circles with discriminating anger from neutral expressions with the 2IFC and yes-no procedures were positive and moderate to large (2IFC $r = .86$, 95% CI: .70, .94 and yes-no $r = .61$, 95% CI: .28, .81). Table 3 shows that the severity of the disease, as measured by the motor score on the MDS-UPDRS, correlated moderately and negatively with the 2IFC measures of emotion discrimination and RF3 pattern discrimination; the correlation coefficient was smaller, with CI’s just overlapping zero, between the MDS-UPDRS motor scores and the emotion discrimination measure with the yes-no procedure. The correlations between discriminating RF3 patterns from perfect circles and discriminating anger expressions from neutral expressions remained positive and moderate to large after removing the common variance shared with disease severity.
(2IFC $r = .79$, 95% CI: .57, .90; yes-no $r = .51$, 95% CI: .14, .76). Therefore, after removing the common variance shared with disease severity, RF3 pattern discrimination shared 62% and 26% of variance with emotional expression discrimination as measured by the 2IFC and yes-no procedures respectively.

Table 3

<table>
<thead>
<tr>
<th>Emotion discrimination measure</th>
<th>MDS-UPDRS motor score</th>
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<tbody>
<tr>
<td>2IFC procedure</td>
<td>-.59</td>
</tr>
<tr>
<td>Yes-no procedure</td>
<td>-.38</td>
</tr>
<tr>
<td>RF3 pattern discrimination measure</td>
<td>-.59</td>
</tr>
</tbody>
</table>

Discussion

The results show large impairments in PD than control groups in discriminating graded intensities of anger from neutral expressions at brief and long stimulus durations on the 2IFC and yes-no procedures and in discriminating RF3 patterns of varying amplitude modulations from perfect circles, with positive and moderate to large correlations between these abilities, even after controlling for disease severity.

The positive and moderate to large correlations between the abilities to perceive facial expressions of emotion and visual form are incongruent with the conclusion from the Gray and Tickle-Degnen meta-analysis (2010) that these abilities are unrelated. This conclusion was based on evidence from individual studies that the ability to perceive visual form was intact in PD. The measures of visual form perception in these studies, however, were insensitive to differences
between PD and control groups. Here we show impaired visual form perception in PD on a measure of discriminating RF3 patterns of varying amplitude modulations from perfect circles. RF patterns are considered a powerful tool to investigate the intermediate stages of visual form processing that underlie the transformation from low-level features, such as edge orientation, to high-level object representations such as faces (Loffler, 2008, for a review). The impairment in RF3 pattern discrimination in PD also adds to the current body of literature that shows impairment in a range of basic visual functions (Archibald et al., 2009).

The strength of the correlation between the ability to perceive emotional expression and the ability to perceive visual form remained relatively unchanged after removing the common variance shared with disease severity. After removing this variance, there remained 62% shared variance between 2IFC measures of RF3 pattern discrimination and emotional expression discrimination, and 26% shared variance between measures of RF3 pattern discrimination and emotional expression discrimination with the yes-no procedure. The greater shared variance between the 2IFC procedures is presumably due to their common procedural elements. The 26% of shared variance between RF3 pattern discrimination and emotional expression discrimination with the yes-no procedure after removing the variance associated with disease severity indicates the extent of common variance between these measures that is not attributable to the similarity of the procedures. It is possible that neural systems that are engaged by intermediate stages of visual form processing receive poor input from more peripheral visual systems due to well-documented impairments in basic visual functions (Archibald et al., 2009), thereby affecting processing of visual forms that include but are not restricted to human faces that convey emotion. Alternatively, it might be that systems that are engaged by
intermediate stages of visual form processing, like peripheral systems, are compromised in PD, thereby affecting processing of visual forms that include but are not restricted to faces that convey emotion. After taking into account the variance that is shared with the impaired visual form perception, there still remains variance that is unaccounted for in emotion discrimination with the 2IFC and yes-no procedures respectively. Thus the impaired ability to perceive emotional expressions in PD is not entirely explained by the impaired ability to perceive visual form. Our previous work (Marneweck et al., 2014, June 16) showed a link between voluntary control of facial musculature and the ability to perceive emotional expressions in PD. This link is consistent with an embodied simulationist account of emotion perception (see Goldman & Sripada, 2005, for a review) that systems that are engaged by voluntary control of facial musculature and simulation of observed facial expressions are disrupted in PD, thereby impairing both voluntary control of facial musculature and perception of facial expression of emotion. Together these findings support the likelihood of a multifactorial contribution to impaired perception of facial expressions of emotion in PD.

The impairment in PD in discriminating graded intensities of anger from neutral expressions replicates previous findings by our group (Marneweck et al., 2014, June 16). Previously, we showed that those with PD were impaired in discriminating emotional from neutral expressions with emotional intensity increments sampled from a narrower range (5 to 35% of the full-blown expression) than we used here (5 to 52% of the full-blown expression). We show that the relative impairment in PD is present with higher intensities of the expression. There was also a similar level of impairment in PD on a measure of emotion discrimination with a single-stimulus yes-no procedure, which unlike the 2IFC procedure, does not require
the ability to hold information from the first stimulus while processing that of the second stimulus; this suggests that the impairment in PD in emotional expression discrimination as reported here and previously on the measure with the 2IFC procedure was not exaggerated by working memory demands. The impairment in PD was also present with longer stimulus durations on both emotional expression discrimination measures, which indicates that brief stimulus durations did not exaggerate the impairment in emotional expression discrimination in PD. Although longer stimulus durations were no more beneficial for PD than control groups, performance across groups generally improved with increasing stimulus duration on the yes-no procedure but not on the 2IFC procedure; sensitivity was greater in both groups at the longer stimulus duration in the yes-no procedure but not in the 2IFC procedure. One of the key differences between the 2IFC and yes-no procedures is that the 2IFC procedure allows a visual comparison of the emotional and neutral expression. The yes-no procedure requires recollection of an emotional and neutral expression without having the familiarity that is given by visual comparison. Nevertheless, the level of impairment in PD is similar at brief and longer durations in both 2IFC and yes-no procedures. This indicates that the impairment in PD in discrimination of facial expressions of emotion as measured here was not exaggerated by slower visual processing times or working memory demands required by sequential stimuli.
References


Supplementary Material

Table 1
Correlation coefficients (with 95% CI’s in parentheses) of measures of emotion discrimination with 2IFC and yes-no procedures with measures of depressive symptoms, as measured by the GDS, and general cognitive functioning, as measured by the MoCA, in PD (n = 24).

<table>
<thead>
<tr>
<th>Emotion discrimination measure</th>
<th>GDS</th>
<th>MOCA</th>
</tr>
</thead>
<tbody>
<tr>
<td>2IFC procedure</td>
<td>-.05 (-.44, .36)</td>
<td>.28 (-.14, .61)</td>
</tr>
<tr>
<td>Yes-no procedure</td>
<td>.06 (-.35, .45)</td>
<td>.29 (-.13, .62)</td>
</tr>
</tbody>
</table>
CHAPTER 6: General Discussion

The ability to perceive facial expressions of emotion in Parkinson’s disease (PD) has received considerable interest in the last few decades. Despite this interest, which includes a meta-analysis showing impaired perception of emotion in PD (Gray & Tickle-Degnen, 2010), a systematic investigation was lacking of the basic perceptual processes involved in perceiving facial expressions of emotion, particularly discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions. In addition, the determinants of impaired perception of emotional expression in PD remained poorly understood. Insofar as these questions are left unanswered, the potential to ameliorate impaired perception of emotion in PD remains distant. The aims of this research were to explore perception of facial expressions of emotion in PD, with an emphasis on psychophysical measures of discriminating graded intensities of emotional expressions from neutral expressions and graded intensities of the same emotional expressions, and to investigate some factors that might contribute to impaired perception of emotional expressions in PD. Overall, persons with PD were as a group impaired on psychophysical measures of discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions; discriminating discrepant from similar full-blown expressions and recognizing full-blown expressions were also impaired in the PD group. However, some persons with PD performed as well as some of the best-performing controls. There were moderate to large correlations between perceiving facial expression of emotion and (1) discriminating changes in facial distinctiveness, (2) discriminating radial frequency (RF) patterns of varying amplitude modulations from perfect
circles, and (3) voluntary control of facial musculature, all of which were correlated with the progression of the disease.

The reliability and sensitivity of psychophysical measures of discriminating graded intensities of emotional from neutral expressions and discriminating graded intensities of the same emotional expressions was first demonstrated in a healthy young adult sample in Experiment 1 (Marneweck, Loftus, & Hammond, 2013). The measures were shown to be broadly applicable to facial expressions of different emotional states, but they were also shown to detect differences in sensitivity to different emotions. Emotional expressions vary in the visual range of expressivity, so the magnitude of a 5% change will vary depending on the emotion. For example, change in an emotion such as happiness, which is typically expressed with an open mouth and with extensive facial change, will be discriminated with smaller percentage change than emotions that are expressed more subtly, such as sadness, which is typically expressed with a closed mouth and with less extensive facial change. Furthermore, the measures were shown to be sensitive to small variations in intensity at which an emotion is expressed (as shown by small thresholds in young adults, ranging from 7% to 17% with a median of 9%), making it capable of detecting small changes in sensitivity to facial expressions of emotion that might emerge from PD.

Data from three experiments (Experiments 2, 3, and 4) confirmed that persons with PD were as a group impaired compared to controls on psychophysical measures of discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions, for all emotions tested, anger, disgust, happiness, and sadness. The group impairment in PD in discriminating graded intensities of emotional from neutral expressions was present
with both brief and prolonged stimuli durations and on psychophysical measures with both two-interval forced-choice (2IFC) and yes-no procedures. The PD group was also impaired in discriminating discrepant from similar full-blown emotional expressions and in recognizing full-blown emotional expressions. However, performance by persons with PD varied considerably on all of these measures, with some performing as well as some of the best-performing controls; some of the contributing factors to this variability are discussed later. In summary, this research gives the first report of impairment in PD in discriminating varying intensities of emotional expressions from neutral expressions and varying intensities of the same emotional expressions, and replicates previous reports of impairment in PD in discriminating and recognizing full-blown emotional expressions.

Large effect sizes on psychophysical emotion discrimination measures with both 2IFC and yes-no procedures suggest that the impairment in PD was not inflated by the requirement to process sequential stimuli in the 2IFC procedure, which might have placed additional working memory demands. From a limited number of studies on the effect of working memory on perceiving emotion in PD, Gray and Tickle-Degnen (2010) suggested that there was preliminary evidence for an effect of working memory in perceiving emotion from voices in PD. Findings here suggest that perceiving emotion from faces was not differentially affected by working memory in patients and controls, at least as is measured by the psychophysical task with the 2IFC procedure of discriminating emotional from neutral expression. In addition, that discriminating graded intensities of emotional from neutral expressions was similarly impaired with brief and longer stimulus durations suggests that impaired discrimination of emotional expressions in PD, as is measured here, is not secondary to slowing of visual processing times. Although different stimulus
durations have been used by individual studies on perceiving emotional expressions in PD, this research gives the first direct manipulation of stimulus durations on discriminating emotional expressions in PD. In sum, these findings indicate that the impaired ability to discriminate facial expressions of emotion in PD, as measured here, was not inflated by slower visual processing times or working memory demands required by sequential stimuli.

The effect sizes of the impairment in PD on psychophysical measures of discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions were larger in all three experiments (Experiment 2 mean $g = 1.22$; Experiment 3 mean $g = 1.52$; Experiment 4 mean $g = 1.17$) than the averaged medium-sized effect ($g = .48$) for all of the basic emotions reported by Gray and Tickle-Degnen (2010). This indicates that psychophysical measures of discriminating graded intensities of emotional from neutral expressions and graded intensities of the same emotional expressions were more sensitive to the impairment in PD than those sampled on average by Gray and Tickle-Degnen (2010). The majority of studies included in the meta-analysis focused on recognizing emotional expressions by name, with only some looking at discriminating emotional expressions. Although it is possible that discriminating facial expressions of emotion is more impaired than recognizing facial expressions of emotion in PD, giving larger effect sizes, evidence for this view is mixed. Gray and Tickle-Degnen (2010) found slightly larger effect sizes for discriminating ($g = .62$) than recognizing ($g = .50$) emotions from faces and voices. Results from Experiment 3 showed larger effect sizes for discriminating graded intensities of the same emotional expressions ($g = 1.52$) than recognizing full-blown emotional expressions ($g = .96$). However, in contrast to the view that discrimination is more impaired than recognition of
emotional expressions in PD, Experiment 3 also showed that there was little
difference between effect sizes for discriminating discrepant from similar
expressions ($g = .92$) and recognizing emotional expressions ($g = .96$). Given these
mixed findings, there is not strong support for the view that discriminating emotional
expressions is more impaired in PD than recognizing emotional expressions.
Psychophysical measures of emotion discrimination might be more sensitive to the
impairment in PD due to other factors, which are discussed next, giving larger effect
sizes than other discrimination and recognition measures used in Experiment 3 and
most measures sampled by the meta-analysis (Gray & Tickle-Degnen, 2010).

Three studies (Bediou et al., 2012; Buxton, MacDonald, & Tippett, 2013;
Dujardin et al., 2004) found similarly large effect sizes to those reported here. All
these studies measured recognition of graded intensities of emotional expressions,
whereas the majority of studies surveyed by Gray and Tickle-Degnen (2010), giving
an averaged medium-sized effect, measured recognition of full-blown emotional
expressions. This suggests that larger effect sizes reported here and previously
(Bediou et al., 2012; Buxton et al., 2013; Dujardin et al., 2004) might be due to the
use of graded than full-blown intensities of emotional expressions in measures of
perception of facial expressions of emotion. In line with this suggestion, the
impairment in PD from the present findings was slightly larger for discriminating
graded intensities of emotional expressions than discriminating and recognizing full-
blown emotional expressions. Together these findings suggest that the ability to
perceive subtler expressions of emotion is more difficult for those with PD than the
ability to perceive full-blown expressions of emotion. That full-blown expressions
are easier to perceive than subtle expressions is certainly the case in healthy adults
(Hess, Blairy, & Kleck, 1997). Given that subtler expressions are more commonly
observed than full-blown expressions, the level of impairment in PD shown here probably matches that which is evident in everyday life. In sum, it appears that measures of perceiving graded intensities of facial expressions of emotion are more sensitive to the impairment in PD, giving larger effect sizes, than measures of perceiving full-blown intensities of facial expressions of emotion.

There might be another factor, in addition to the use of graded intensities of emotional expressions, which gave larger effect sizes here than the averaged effect found by Gray and Tickle-Degnen (2010). Effect sizes for perceiving full-blown expressions from Experiment 3 were also larger than the averaged effect from the meta-analysis (Gray & Tickle-Degnen, 2010). Larger effect sizes might also be a result of including in the present experiments more persons with PD at the more advanced stages of disease progression than those reviewed by the meta-analysis. Yip, Lee, Ho, Tsang, and Li (2003) showed similarly large impairments to those reported here in emotional expression recognition in PD with the inclusion of persons with PD at later stages of disease progression. Furthermore, the findings from the present research showed moderate positive correlations between disease severity and perception of emotional expressions, even after controlling for age, general cognitive functioning, and depressive symptoms. Those who were assigned to a higher severity band demonstrated much larger impairments in discriminating and recognizing emotional expressions than those assigned to a lower severity band. That disease severity adversely affects perception of emotional expression in PD is in line with positive correlations shown by Buxton et al. (2013) and Wieser et al. (2012), but is incongruent with results from Gray and Tickle-Degnen (2010) and Baggio et al. (2012) that these variables are unrelated. The meta-analysis finding that disease severity was not related to emotion perception in PD is likely due to the
insensitivity of the Hoehn and Yahr Staging Scale (Goetz et al., 2004). The lack of correlation between disease severity and perception of emotional expressions shown by the Baggio group (2012) might be due to sampling only a limited range in disease severity. With the use of the gold-standard measure of disease severity, the Movement Disorders sponsored-revision of the Unified Parkinson’s Disease Rating Scale (MDS-UPDRS; Goetz et al., 2008), on samples of individuals with PD at early and mild-to-moderate stages in disease progression in all three experiments, this research showed that the progression of the disease adversely affects perception of facial expressions of emotion in PD. From the considerations above, it appears that two factors, the intensity of emotional expressions examined and the disease status of patients, most likely contributed to larger effect sizes in the present research than the averaged medium effect size found by Gray and Tickle-Degnen (2010) of the impairment in perceiving facial expressions of emotion in PD.

Other variables were found to also contribute to impaired perception of facial expressions of emotion in PD, even after removing the variance that was shared with disease severity. Results showed moderate to large partial correlations between the ability to perceive facial expressions of emotion and (1) the ability to discriminate graded changes in facial distinctiveness (2) the ability to discriminate RF3 patterns of varying amplitude modulations from perfect circles, and (3) the ability to voluntarily control facial musculature in PD. Together these findings suggest a multifactorial contribution to impaired perception of facial expressions of emotion. Each of the examined variables will be discussed in turn.

Those with PD were not exclusively impaired in perceiving emotional information from faces but were also impaired in perceiving non-emotional information from faces. The ability to discriminate graded changes in facial
distinctiveness and the ability to recognize facial identity (as measured by the Cambridge Face Memory Test, CFMT; Duchaine & Nakayama, 2006) were also impaired in PD. That Gray and Tickle-Degnen (2010) found negligible differences between PD and control groups in face recognition is likely due to insensitive measures of face identity recognition used in most of the studies they reviewed (Duchaine & Weidenfeld, 2003). After removing the variance shared with disease severity, perceiving emotional expressions correlated positively and moderately with discriminating graded changes in facial distinctiveness, but not with recognizing facial identity. The link between perceiving emotional expressions and facial distinctiveness differences remained relatively unchanged even after removing variance shared with age, and is in line with the positive correlation reported by Narme, Bonnet, Dubois, and Chaby (2011) between perceiving emotional information from faces, measured as recognizing emotional expressions, and non-emotional information from faces, measured as discriminating spatial differences in facial features. The links reported here and by Narme et al. (2011) are inconsistent with the dominant theory of face perception (Bruce & Young, 1986) that separates perception of emotional and non-emotional information from faces, but are consistent with recent propositions (see Calder & Young, 2005, and Vuilleumier & Pourtois, 2007, for reviews) that perception of identity and emotional expressions share perceptual encoding mechanisms that facilitate perception. Both abilities to perceive emotional and non-emotional information from faces might have been impaired in PD by a more general impaired ability to perceive visual form. Indeed, the findings from Experiment 4 showed that those with PD were impaired in perceiving visual form, measured as discriminating RF3 patterns of varying amplitude modulations from perfect circles with a 2IFC procedure. RF patterns have
been considered to be a powerful tool to study intermediate stages of visual form processing, which underlie the transformation from low-level features, such as edge orientation, to high-level object representations such as faces (Loffler, 2008). Impairment in perception of visual form in PD is consistent with extensively documented impairments in a range of basic visual functions in PD (see Archibald, Clarke, Mosimann, & Burn, 2009, for a review). Furthermore, the present results showed a link between the ability to perceive visual form and the ability to discriminate graded intensities of anger from neutral expressions in PD. This link, which remained relatively unchanged after removing the common variance shared with disease severity, was shown between the 2IFC measure of discriminating RF3 patterns from perfect circles and both 2IFC and yes-no measures of discriminating intensities of anger from neutral expressions. The shared variance between the visual form perception with the 2IFC procedure and the emotion discrimination measure with the yes-no procedure indicates the extent of variance that is not attributable to the similarity of the procedures. From these findings, one contribution to impairment in perception of emotional expressions in PD is a more general impairment in perception of visual form. A broader impairment in perceiving visual form might have contributed to both impairments in perceiving emotional and non-emotional information from faces. It might be that neural systems that are engaged by intermediate stages of visual form processing are relatively intact in PD, but receive poor input from more peripheral parts of the visual system due to well-documented impairments in basic visual functions (Archibald et al., 2009), thereby impairing perception of visual forms that include but are not restricted to faces that convey emotional and non-emotional information. Alternatively, it might be that neural systems that are engaged by intermediate stages of visual form processing, like
peripheral parts of the visual system, are disrupted in PD, thereby impairing the ability to perceive visual forms that include but are not restricted to faces that convey emotional and non-emotional information. Neither of these possibilities can be ruled out from the present findings, but both offer a plausible explanation for the broader impaired ability to perceive visual form in PD, which contributes to impaired facial expressions of emotion.

After taking into account the variance shared with disease severity and perception of visual form, there remained variance unaccounted for in perception of facial expressions of emotion in PD. This indicates that impairment in perception of emotional expressions in PD is not entirely explained by disease severity and by impairment in perception of visual form. Experiments 2 and 3 showed a moderate link between voluntary facial musculature control and perception of emotional expressions in PD, after removing the shared variance with disease severity and age. This link is in line with embodied simulationist accounts of emotion perception, which state that we understand the emotions of others by simulating or enacting within ourselves the observed facial expression of emotion (Goldman & Sripada, 2005, for a review). According to the embodied simulation view, disruption of the process of simulation will be accompanied not only by disruption of the processes relating to producing expressions of emotion, but also the process of perceiving expressions of emotion. As was predicted by this embodied simulationist account, both voluntary control of facial musculature and perception of facial expressions of emotion were shown to be disrupted and related to each other in PD. Similarly, the association between emotion production and recognition in PD (Benke, Bosch, & Andrew, 1998; Borod et al., 1990; Jacobs, Shuren, Bowers, & Heilman, 1995) might be mediated at least to some extent by neural circuitry that is engaged by simulation
of observed expressions, and which has been disrupted, thereby contributing to the co-occurrence of impairments in perception and production of emotional expression (Borod et al., 1990; Bowers et al., 2006; Buck & Duffy, 1980; Jacobs et al., 1995; Katsikitis & Pilowsky, 1988; Marsili et al., in press; Pitcairn, Clemie, Gray, & Pentland, 1988; Simons, Ellgring, & Smith Pasqualini, 2003; Simons, Smith Pasqualini, Reddy, & Wood, 2004; Smith, Smith, & Ellgring, 1996). Together, these findings are in line with an embodied simulationist account, where the processes of simulation of observed facial expressions of emotion might be disrupted in PD, thereby contributing to impaired perception of facial expression of emotion. The processes (e.g. overt, covert) by which simulation might occur cannot be determined from this project, as they were not directly measured. As a start, in light of accumulating evidence against overt perspectives of embodied simulation in emotion perception (Bate, Cook, Mole, & Cole, 2013; Bogart & Matsumoto, 2010; Calder, Keane, Cole, Campbell, & Young, 2000; Hess & Blairy, 2001; Keillor, Barrett, Crucian, Kortenkamp, & Heilman, 2002), the link between voluntary facial musculature control and emotional expression perception will be further interpreted from a covert perspective of embodied simulation. From a covert perspective, systems with mirror-like properties that are engaged by simulation of observed expressions and voluntary control of facial musculature might be disrupted by the neurodegenerative processes in PD, thereby contributing to impaired voluntary control of facial musculature and impaired perception of emotional expressions.

If systems with mirror-like properties that are engaged by simulation are indeed disrupted in PD then the following question might be considered: which of the systems with mirror-like properties that are engaged by production and perception of emotion have been disrupted in PD? The studies that have investigated
the systems with mirror-like properties during observation and production of emotional expressions have found mirroring activity in multiple regions (Carr, Iacobini, Dubeau, Mazziotta, & Lenzi 2003; Hennenlotter et al., 2005; Jabbi, Swart, & Keysers, 2007; Leslie, Johnson-Frey, & Grafton, 2004; van der Gaag, Minderaa, & Keysers, 2007; Wicker et al., 2003). It has been suggested that some of these regions are engaged by experience and expression of emotion (e.g. insula for disgust: Jabbi, Bastiaansen, & Keysers, 2008; Phillips et al., 1997; Small et al., 1999, 2003; insula for other emotions: Carr et al., 2003; Hennenlotter et al., 2005; Jabbi et al., 2007; van der Gaag et al., 2007), while others are known to be involved in voluntary facial movement more generally, regardless of its intended meaning (e.g. premotor cortex: Morecraft, Stilwell-Morecraft, & Rossing, 2004). It cannot be ascertained from the present data whether any or all of these regions are disrupted by the neurodegenerative processes in PD and contribute to impaired perception of facial expressions of emotion. Current opinions on the neurophysiology of the basal ganglia emphasize the involvement of at least three distinct cortico-striato-pallido-thalamo ‘loops’ that connect the putamen, caudate nucleus, and the nucleus accumbens with different cortical and subcortical regions, including motor and limbic regions (Alexander, DeLong, & Strick, 1986). The motor and limbic loops each contain systems with mirror-like properties that have been found to activate during production and perception of facial expressions of emotion (Carr et al., 2003; Hennenlotter et al., 2005; Jabbi et al., 2007; Leslie et al., 2004; van der Gaag et al., 2007; Wicker et al., 2003). The motor loop includes the premotor cortex and the limbic loop includes the anterior cingulate, insula, and amygdala. Given their links with the basal ganglia, these systems with mirror-like properties within motor and limbic loops might be compromised in PD (at least in the later stages of the disease;
see Morrish, Sawle, & Brooks, 1996), and in turn contribute to impaired perception of facial expressions of emotion. Anders et al. (2012) have found that disruption of systems with mirror-like properties within the motor loop affected perception of emotional expressions in a group of Parkin mutation carriers, who show mild reduction of dopamine metabolism in the basal ganglia in the absence of clinical motor signs. The impairment among Parkin mutation carriers was graded, whereby those who were least impaired in perceiving emotional expressions showed the strongest ventrolateral premotor cortex activity. Consistent with a covert simulationist account, Anders et al. (2012) argued that compensatory activity in systems with mirror-like properties that are engaged by motor control reduced the impairment in perception of emotional expression. The link shown here between perception of emotional expressions and voluntary control of facial musculature does suggest at least some involvement of systems that are engaged by voluntary control of facial musculature in perception of emotional expressions in PD. Together the findings from Anders et al. (2012) and from here support the supposition that systems with mirror-like properties that are engaged by simulation of observed expressions within the motor loop are disrupted in PD, thereby contributing to impaired perception of facial expressions of emotion. Nevertheless, systems in the limbic loop, which have also been shown to be engaged by production of emotional expressions and simulation of observed expressions (Carr et al., 2003; Hennenlotter et al., 2005; Jabbi et al., 2007; van der Gaag et al., 2007; Wicker et al., 2003), might be compromised by the disease process, thereby also contributing to impaired perception of emotional expression in PD. In summary, there are systems in the limbic and motor loops that are thought to have mirror-like properties that simulate the observed expressions of others, thereby facilitating perception of emotional
expressions. These systems might be disrupted by the neurodegenerative processes in PD, thereby contributing to impaired perception of facial expressions of emotion. The link found here between voluntary control of facial musculature and perception of emotional expressions supports a covert embodied simulationist account of impaired perception of emotional expressions in PD, whereby disruption of systems that are engaged by voluntary control of facial musculature and observed expressions of emotion contribute to impaired perception of emotional expressions.

In the investigation of the link between voluntary control of facial musculature and perception of facial expressions of emotion in PD, the present research also gave insight into the ability of patients to voluntarily control facial musculature. Given the influential and historical conceptualization (Rinn, 1984) of the “masked” expressionless face in PD as reduced spontaneity in expression of emotion, previous studies of facial musculature control in PD have predominantly focused on facial movements that convey emotion, and consistently showed impairments, whereas facial movements that do not convey emotion have been less systematically investigated. The present findings of impairment in a range of upper and lower non-emotional facial movements in PD supports and extends previous reports of impairment in specific facial areas (Agostino et al., 2008; Connor, Abbs, Cole, & Gracco, 1989; Korosec, Zidar, Reits, Evinger, & Vanderwerf, 2006; Marsili et al., in press) and in a limited set of non-emotional facial movements (Simon et al., 2003; Simons et al., 2004). Together, these findings confirm that impaired facial musculature control is not restricted to production of facial expressions of emotion, and likely gives rise, with diminished expressions of emotion, to the impression of a mask-like face in PD. With an adapted version of the Face Apraxia Test (Bizzozero et al., 2000) with a refined error analysis, those with PD showed an increase in
impoverishment of non-emotional facial movement but little to no change in loss of movement individuation, content errors, or pauses prior to movement initiation, compared to healthy controls. That impaired voluntary non-emotional facial movement was predominantly due to impoverished movement in PD strengthens the claim that it too contributes with diminished expressions to facial masking. The low incidence of content errors (which can be likened to ideational apraxic errors; Zadikoff & Lang, 2005) and the positive correlation with motor severity but not with general cognitive functioning suggests that impaired voluntary control of facial musculature is more a part of the motor than the non-motor symptoms of the disease. Finally, these findings demonstrate the use of an adaptation of the Face Apraxia Test (Bizzozero et al., 2000) as a practical and sensitive measure of voluntary control of facial musculature in PD that can be used in research and clinical settings to further understand the determinants of facial masking.

**Limitations and Considerations for Future Research**

A consequence of the use of psychophysical measures of discriminating emotional expressions with the Method of Constant Stimuli in participants with considerable performance variation was that thresholds were unobtainable for some of those participants. This was also the case in Experiment 3 and Experiment 4 when the emotion discrimination measures included an increased range in stimulus levels, in an attempt to capture a wider range of sensitivities than those captured by Experiment 2; even so, some participants remained at floor performance across stimulus levels. A solution that can enable thresholds to be obtained for individuals with a range of abilities is to use an adaptive staircase method that tailors the stimulus levels to the ability of each participant.
To infer the emotional states of others accurately, we often concurrently process information from facial expression, prosody, gesture, and posture, rather than relying solely on information from a single modality. Gray and Tickle-Degnen (2010) showed an impaired ability to perceive emotion from prosody in PD, which suggests the existence of a multimodal impairment in perceiving emotion. However, no study in those with PD has measured the ability to simultaneously monitor multiple channels to infer emotional states of others. In addition, there is accumulating evidence that perception of facial expressions of emotion is routinely influenced by the context in which it occurs, and is not driven only by the structural features of a face (see Feldman Barrett, Mesquita, & Gendron, 2011, for a review). Whether perception of simultaneously presented multimodal information including contextual information for inference of others’ emotional states will help or hinder those with PD remains to be shown.

Even though persons with PD that were recruited for the present research were at a range of stages in their disease progression, those at the more advanced stages of the disease were not adequately represented. It is likely that the ability to perceive facial expressions of emotion will become progressively more impaired in the more advanced stages of the disease. It might be that factors that do not contribute to perception of emotional expression in early to moderate stages of PD (e.g. general cognitive functioning, depressive symptoms, working memory, and slower visual processing times), contribute to progressively larger impairments in perception of emotional expression in later stages of the disease.

Given that ethical approval did not allow for patients to be withdrawn from dopamine replacement therapy, patients were always tested approximately 1.5 hours prior to their next dopamine replacement medication intake, with the aim that
performance would receive the same effect from medication across testing sessions. Nevertheless, the effect of dopamine replacement therapy on perception of facial expressions of emotion cannot be ascertained from this project. Although Gray and Tickle-Degnen (2010) found that persons off dopamine replacement therapy (by withdrawal of therapy or by never having started therapy) were more impaired ($g = .50$) in emotion perception than persons on dopamine replacement therapy ($g = .27$), the difference in impairment between these groups was not significant. Gray and Tickle-Degnen (2010) raised the possibility that the true group difference between dopaminergic and hypodopaminergic states in emotion perception might have been skewed by two factors. First, dopamine levels in an optimally medicated state of a patient at a more advanced stage of PD might not be dissimilar to that of a patient that is withdrawn from or not yet started dopamine replacement therapy at an earlier stage of PD. Second, grouping patients that were not yet receiving therapy (because they were recently diagnosed) with patients that were withdrawn from therapy (presumably at later stages of the disease) could have skewed the size of the impairment in those off dopamine replacement therapy. Furthermore, a lack of reports from individual studies regarding medication status allowed for a total of 22 comparisons, between 16 in a relatively dopamine-replete state and only six in a hypodopaminergic state. An important future step is to investigate the function of the dopaminergic system for emotion perception in PD in a set of patients at various stages of disease progression when they are each off and on dopamine replacement therapy. One study (Tessitore et al., 2002) has compared controls ($n = 10$) with patients at an early stage of the disease ($n = 10$) that were each in a hypodopaminergic and dopaminergic state in discriminating discrepant from similar expressions. Results showed lower accuracy, but not significantly lower, in emotion
discrimination in patients in a hypodopaminergic state compared to controls and when patients were in a dopaminergic state; these differences between dopaminergic and hypodopaminergic states might become more apparent at later stages of the disease, and if studied with larger sample sizes.

The exploration of some of the potential factors that might contribute to impaired perception of facial expressions of emotion in PD was done by correlational analyses between behavioral measures. The interpretation of findings, that disruption of neural systems engaged by intermediate visual form processing, facial musculature control, and simulation of observed expressions, contributes to impaired perception of emotional expression in PD, is based only on correlations between behavioral measures. Therefore, the work described here gives only an indirect examination, and does not provide strong definitive evidence for any of the proposed determinants of impaired perception of facial expressions of emotion. In addition, the examined determinants were themselves intercorrelated, hence there is the problem of multicollinearity. This multicollinearity problem was dealt with, as best possible, by partialling out the variance shared by disease severity in all correlational analyses where disease severity intercorrelated with independent and dependent variables.

There might be an alternative explanation for the link between voluntarily controlling facial musculature and perceiving facial expressions of emotion in PD. The Face Apraxia Test (Bizzozero et al., 2000) and the measures of perceiving emotional expression all require visual input i.e. viewing the demonstration on the Face Apraxia Test to be reproduced and viewing the emotional or neutral expressions to be discriminated and recognized. The variance shared by perceiving emotional expressions and voluntarily controlling facial musculature might have
been due to a broader impairment in visual function, which affected performance on all measures. However, after removing disease severity, the ability to discriminate changes in facial distinctiveness correlated with the ability to discriminate graded intensities of emotional expressions in PD, but not with voluntary control of facial musculature. That facial distinctiveness discrimination, which required the same visual input as the measure of emotion discrimination, was unrelated to voluntary control of facial musculature after partialling out disease severity suggests that the link between emotion discrimination and voluntary control of facial musculature was not mediated solely by impaired visual function. Nevertheless, the potential effect of imitating demonstrated facial movements on the Face Apraxia Test in PD (and other voluntary facial musculature control measures in PD that are currently in use) needs further investigation.

One of the key criticisms of embodied simulationist accounts for action understanding is that it remains unclear empirically and conceptually how motor systems with mirror-like properties in humans form the basis of “understanding”. This criticism is based on findings that other mechanisms are also involved in understanding the actions of others (see Hickok, 2009, for a review). The link found here between perceiving visual form and perceiving emotional expressions in PD suggests that an embodied simulationist account does not entirely explain impaired perception of expression of emotion in PD. A revised view put forth by Mahon and Caramazza (2008) on the role of the motor system in action understanding is that the motor system can influence or augment understanding to some degree, but without sticking to the empirically untenable stance that action understanding is dependent on the motor system. To illustrate this view, Hickok (2009) uses an example of a person that understands the concept of playing a saxophone even when that person
has never played such an instrument; however, if a person has had the motor knowledge of having played the saxophone before, it will enrich their understanding of observing the saxophone being played e.g. knowing that the mouth is in the right place on the mouthpiece, that the fingers are placed on the correct tone holes etc. This revised view fits well with present results, which suggests that disruption of systems with mirror-like properties that simulate observed expressions are not solely responsible, but instead contributes alongside disruption of visual systems to impaired perception of facial expressions of emotion in PD.

Conclusions

In summary, the research from this thesis showed for the first time that most but not all persons with PD are impaired in discriminating graded intensities of facial expressions of emotional from neutral expressions and graded intensities of the same facial expressions of emotion. The research from this thesis also replicated previous reports of impaired discrimination and recognition of full-blown facial expressions of emotion. Together, this work gives a more complete understanding of impaired perception of emotional facial expression in PD, particularly by repeated demonstrations of impaired discrimination of varying intensities of emotional from neutral expressions and impaired discrimination of varying intensities of the same emotional expressions at brief and longer durations and on 2IFC and yes-on measures. To work towards identifying the contributing factors to impaired perception of emotional expression in PD, the research from this thesis showed that the impaired perception of facial expressions of emotion is an increasing function of the severity of the disease, which is uninflated by impairment in processing of sequential stimuli and visual processing times, and unrelated to general cognitive functioning, depressive symptoms, sex, and years of education. Furthermore, the
research from this thesis showed links between perception of emotional expression, perception of visual form, and voluntary control of facial musculature in PD (even after removing the variance that is shared with disease severity). These correlations provide preliminary evidence for a multifactorial contribution to impaired perception of facial expressions of emotion in PD, which includes, but is not necessarily limited to, disrupted sensory and sensorimotor processes.
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