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Research Report
**Vocal emotion processing in Parkinson's disease:
Reduced sensitivity to negative emotions**
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ABSTRACT

To document the impact of Parkinson's disease (PD) on communication and to further clarify the role of the basal ganglia in the processing of emotional speech prosody, this investigation compared how PD patients identify basic emotions from prosody and judge specific affective properties of the same vocal stimuli, such as valence or intensity. Sixteen non-demented adults with PD and 17 healthy control (HC) participants listened to semantically-anomalous pseudo-utterances spoken in seven emotional intonations (anger, disgust, fear, sadness, happiness, pleasant surprise, neutral) and two distinct levels of perceived emotional intensity (high, low). On three separate occasions, participants classified the emotional meaning of the prosody for each utterance (identification task), rated how positive or negative the stimulus sounded (valence rating task), or rated how intense the emotion was expressed by the speaker (intensity rating task). Results indicated that the PD group was significantly impaired relative to the HC group for categorizing emotional prosody and showed a reduced sensitivity to valence, but not intensity, attributes of emotional expressions conveying anger, disgust, and fear. The findings are discussed in light of the possible role of the basal ganglia in the processing of discrete emotions, particularly those associated with negative vigilance, and of how PD may impact on the sequential processing of prosodic expressions.

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1. Introduction

In speech communication, listeners attend to relative changes in pitch, duration, and loudness, or speech *prosody*, to infer the emotions or affective state of a speaker (Banse and Scherer, 1996; Scherer, 1986). Recent interest in the neurocognitive processing of emotions from a speaker's voice indicates that these abilities are governed by a distributed

neural network involving cortical and subcortical structures (Pell, 2006; Schirmer and Kotz, 2006). For example, many reports have sought to elaborate the role of cortical regions, such as right temporal and bilateral prefrontal areas, at different stages of processing emotional prosody (Beaucousin et al., 2007; Wildgruber et al., 2005a,b). Recent studies have also drawn attention to the involvement of subcortical structures in vocal emotion processing, such as the amygdala (Sander et al., 2005; Scott et al., 1997) and especially the basal

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ganglia (Adolphs et al., 2002; Anderson and Phelps, 1998; Van Lancker Sidtis et al., 2006), which was the primary focus of the present investigation.

1.1. Basal ganglia contributions to prosody

In the last two decades, compelling evidence that the basal ganglia are engaged in the processing of speech prosody has accumulated from clinical and neuroimaging sources. In a comparative lesion study, Cancelliere and Kertesz (1990) concluded that deficits for recognizing emotions from vocal expressions are highly prevalent in patients with focal lesions that affect the basal ganglia when compared to other lesion sites (see also Starkstein et al., 1994; Weddell, 1994). Neuroimaging investigations have arrived at similar claims about the importance of the basal ganglia in processing emotional prosody (Kotz et al., 2003; Wildgruber et al., 2005a,b). These data fit coherently with observations that emotional prosody is typically impaired in patients with basal ganglia degeneration due to idiopathic Parkinson's disease as well (Blonder et al., 1989; Breitenstein et al., 2001; Pell and Leonard, 2003).

Parkinson's disease (PD) is marked by the interruption of dopaminergic input to the striatum which progressively influences the transmission of information from the basal ganglia to the neocortex via thalamocortical pathways, although damage in the early stages of the disease is relatively confined to the basal ganglia (Alexander et al., 1986). Consequently, non-demented adults with PD are frequently studied to derive insights about functional properties of the basal ganglia (Lieberman, 2000). To date, studies have consistently found that adults with mild to moderate PD are impaired for recognizing the emotional meaning of prosodic cues in speech when compared to a matched control group (Blonder et al., 1989; Breitenstein et al., 1998, 2001; Pell, 1996; Pell and Leonard, 2003; Schröder et al., 2006; Scott et al., 1984; Yip et al., 2003). Similar difficulties have been observed in patients with Huntington's disease as well (Speedie et al., 1990; Sprengelmeyer et al., 1996), reinforcing the view that the basal ganglia play an essential part in systems devoted to prosodic communication.

In a recent set of studies, Pell and Leonard (2003, 2005) compared how 21 aging adults with or without PD recognize basic emotions from prosody and from cues in other communication channels such as facial expressions or verbal cues. The participants' comprehension was assessed with both identification and emotional rating tasks which varied in underlying task demands. The results demonstrated that the PD patients were significantly impaired to recognize emotions strictly from prosodic cues irrespective of task demands, and that their ability to decode emotions from other channels was relatively spared. The PD group was particularly impaired for recognizing the emotion "disgust" in the vocal channel. These prosodic deficits could not be explained by obvious cognitive impairments in the PD group, such as working memory limitations or executive dysfunction. The authors interpreted these data as further evidence that the basal ganglia play a critical and potentially direct role in prosodic processing by promoting efficient decoding of emotional information from vocal cue sequences in speech (Pell and Leonard, 2003). A similar account of how the basal ganglia contribute to tasks which rely on temporal sequencing in speech has been described by Meyer et al. (2004).

Consistent with Pell and Leonard's (2003) observation that PD patients were poor at recognizing vocal expressions of disgust, the possible involvement of the basal ganglia in neural systems for processing discrete emotions has also been described (Anderson and Phelps, 1998; Calder et al., 2001). Much of this support is gathered from studies of emotional face processing, which underscore that PD patients often fail to recognize expressions of disgust (Dujardin et al., 2004; Sprengelmeyer et al., 1996; Suzuki et al., 2006). The ventral striatum has also been implicated in the processing of anger (Calder et al., 2004), and it has been noted that PD patients often display selective difficulties to express anger and disgust through prosody when compared to other emotions (Caekebeke et al., 1991; Pell et al., 2006). Thus, the idea that the basal ganglia participate in dedicated networks for processing specific emotions is firmly entrenched, especially for disgust, although data imply that these impairments are not always easy to detect in PD patients even when facial stimuli are presented (Adolphs et al., 1998; Dewick et al., 1991; Pell and Leonard, 2005). Nonetheless, the impact of different emotion categories on the comprehension of vocal stimuli by PD patients should continue to be monitored.

1.2. Sensitivity to affective dimensions of emotional prosody in PD

While it is increasingly clear that basal ganglia disturbance is associated with problems to assign emotional meanings to prosody in a variety of contexts, a precise understanding of whether PD patients can evaluate the affective properties of vocal stimuli in light of their brain damage has not been reached. As discussed below, the possibility that some of the difficulties experienced by PD patients on prosody tasks stem from an inability to evaluate certain affective properties of paralinguistic events, such as emotional arousal and/or valence, cannot be dismissed. The main aim of this investigation was to look further at how different emotionally-relevant details of vocal stimuli are processed in the context of PD, permitting additional insights about the role of the basal ganglia in vocal emotion processing.

Perspectives on the structure of emotions vary, and yet it is commonly accepted that systematic differences in valence and arousal form central properties of an emotional stimulus (Russell, 1980; Scherer, 1986). Valence attributes of an emotional event require an evaluation of the positive–negative (pleasant–unpleasant) quality of the stimulus, whereas cues to arousal require evaluation of the emotional intensity of the stimulus (here, intensity is defined as the perceived strength of the emotion expressed). With respect to valence, in addition to the emotion-specific deficits described above, several studies have reported greater difficulty for PD patients to recognize negative rather than positive emotions from vocal cues (Breitenstein et al., 1998; Pell and Leonard, 2003) and from facial expressions (Cheung et al., 2006; Dujardin et al., 2004; Suzuki et al., 2006). It is unclear whether these impairments reflect an inability to process discrete emotions as previously assumed or whether the patients fail to fully appreciate the broader, negative characteristics of vocal stimuli in certain contexts (Anderson and Phelps, 1998). Similarly, it has been suggested that the processing of acoustic cues which signal

the arousal component of emotional prosody is disturbed in PD patients (Breitenstein et al., 1998), and related studies have reported impairments for processing the arousal features of emotional pictures in adults with PD (Bowers et al., 2006; Wieser et al., 2006). These collective findings raise the possibility that PD patients are less sensitive to basic affective features of vocal emotion expressions, contributing in part to their difficulties on prosody tasks and possibly in broader aspects of their social lives.

The goal of our study was to specify the communication profile of adults living with PD and to elaborate the role of the basal ganglia in the processing of vocal cues to emotion. This investigation was built on our previous study (Pell and Leonard, 2003) by evaluating the comprehension of both emotions and affective properties of prosodic expressions by a new PD sample. Each participant completed tasks of categorizing the emotional meaning of utterances, with and without congruent semantic features, and tasks which required them to rate specific affective (valence or intensity) characteristics of the same stimuli. Our materials were selected carefully to control for the perceived emotion and intensity level of the prosodic expressions to achieve a sensitive test of how these factors may affect PD patients when engaged in each form of stimulus processing. Based on the literature reviewed, we predicted that PD patients would be impaired overall to categorize emotions from prosodic cues and that selective deficits for certain negative emotional expressions could emerge when categorizing emotions and/or when independently rating the valence of prosodic expressions. Predictions about the impact of emotional intensity cues on the performance of the PD group could not be made with certainty in light of the limited data available.

2. Results

2.1. Identifying emotions from prosody and verbal cues

As a control condition, the ability of each participant group to recognize emotions from utterances containing congruent prosodic and verbal semantic cues was assessed by the prosody semantic identification task. In general, the proportion of correct responses obtained for the HC group ($M=0.83$, $S.D.=0.16$) and for the PD group ($M=0.81$, $S.D.=0.15$) across emotions was relatively comparable. A 2×7 ANOVA with factors of Group (PD, HC) and Emotion (anger, disgust, fear, sadness, happiness, pleasant surprise, neutral) revealed a significant main effect of Emotion, $F(6, 186)=6.60$, $p<0.001$, but no significant main or interactive effect involving Group (both F 's <1.69 , p 's >0.13). Post-hoc Tukey's (HSD) inspection of the emotion effect revealed that happiness ($M=0.71$) and pleasant surprise ($M=0.75$) were associated with greater errors in this task than all other emotions (range of 0.80 for neutral to 0.88 for fear).

2.2. Identifying emotions from prosody alone

The ability of PD and HC participants to judge emotional prosody from pseudo-utterances in our three main tasks—pure prosody emotion identification, valence rating, and intensity

rating—is displayed together in Table 1 according to the emotion and intensity level of the stimuli presented across tasks.

a) Pure-prosody identification — A 2×7 (Group \times Emotion) ANOVA first tested how Group (PD, HC) influenced the proportion of correct identification responses by Emotion (anger, disgust, fear, sadness, happiness, pleasant surprise, neutral) when only prosody conveyed these meanings. There was a significant main effect of Group on emotion identification, $F(1, 31)=6.04$, $p=0.02$, $r=0.37$. Post-hoc comparisons indicated that the PD group made more errors overall than the HC group in the pure prosody task.¹ There was also a significant main effect of Emotion, $F(6, 186)=7.29$, $p<0.001$, which was explained by the fact that neutral utterances ($M=0.81$) were identified more accurately by both groups than all other emotions (range of 0.52 for fear to 0.59 for anger). The interaction of Group and Emotion was not significant for this analysis, $F(6, 186)=1.32$, $p=0.25$. A comparison of overall group performance in the pure prosody versus the prosody semantic task is illustrated in Fig. 1 which shows that the PD patients experienced selective difficulties identifying emotions on the pure prosody task.

A second $2 \times 2 \times 2$ ANOVA involving Group (PD, HC), Valence (positive, negative) and Intensity (high, low) was then run. The results yielded a main effect of Group, $F(1, 31)=3.91$, $p=0.057$, $r=0.33$, which reaffirmed that the PD group performed less accurately than the HC group overall. A significant main effect of Intensity was also observed, $F(1, 31)=19.43$, $p=0.0001$, $r=0.62$, and an interaction of Valence and Intensity, $F(1, 31)=21.68$, $p<0.001$, $r=0.64$. The interaction was explained post-hoc by the fact that all participants were more accurate to identify negative emotions when they were high ($M=0.67$) versus low ($M=0.44$) in intensity, whereas accuracy rates did not differ for positive emotions of high ($M=0.57$) and low ($M=0.55$) intensity. However, there was no influence of either Valence or Intensity on the identification scores of the two groups (all two- and three-way interactions with Group, F 's <1.0 , p 's >0.40).

b) Valence rating — The participants' ability to rate the positive/negative valence of the same auditory stimuli was first examined in a 2×7 ANOVA with factors of Group and Emotion. There was a significant main effect of Group, $F(1, 31)=8.80$, $p=0.006$, $r=0.47$, and Emotion, $F(6, 186)=22.32$, $p<0.001$, as well as an interaction of Group by Emotion, $F(6, 186)=5.00$, $p<0.001$. Post-hoc examination of the interaction indicated that for three of the emotions—anger, disgust, and fear—the PD group differed from the HC group by assigning significantly higher valence ratings to these emotions (in fact, the mean ratings for the PD group fell on the positive side of the rating scale, in contrast to what was observed

¹ For the pure prosody task, a second 2×7 (Group \times Emotion) ANOVA was run excluding the three patients with depression. Again, the results did not show any difference from the main analysis revealing a main effect of Group, $F(1, 28)=12.97$, $p<0.001$, Emotion, $F(6, 168)=5.35$, $p<0.01$, and Emotion \times Group interaction, $F(1, 168)=4.76$, $p<0.01$. Post-hoc analysis of the interaction confirmed that the PD group assigned significantly higher (i.e., positive) ratings to expressions of anger, disgust, and fear when compared to the control group.

Table 1 – Judgements of emotional prosody by individuals with Parkinson's disease (PD) and healthy control (HC) participants when presented the same set of pseudo-utterances in three distinct tasks

Emotion	Intensity	Type of judgement					
		Pure prosody identification (proportion correct)		Valence rating (–3 to +3)		Intensity rating (1 to 5)	
		PD	HC	PD	HC	PD	HC
Anger	High	0.58 (0.27)	0.72 (0.29)	+0.44 (1.83)	–1.01 (1.58)	3.98 (0.53)	4.06 (0.53)
	Low	0.42 (0.18)	0.62 (0.16)	+0.34 (1.03)	–0.88 (0.79)	3.67 (0.55)	3.69 (0.58)
Disgust	High	0.78 (0.18)	0.74 (0.31)	+0.28 (1.43)	–1.16 (1.20)	3.59 (0.63)	3.59 (0.83)
	Low	0.39 (0.22)	0.40 (0.28)	+0.20 (0.82)	–0.63 (0.85)	3.11 (0.33)	3.22 (0.59)
Fear	High	0.45 (0.21)	0.63 (0.32)	+0.78 (1.23)	–0.68 (1.65)	4.23 (0.42)	4.41 (0.50)
	Low	0.61 (0.27)	0.38 (0.33)	+0.05 (1.19)	–1.07 (1.24)	3.52 (0.59)	3.82 (0.72)
Sadness	High	0.67 (0.28)	0.79 (0.24)	–1.31 (0.93)	–1.65 (1.01)	3.03 (0.55)	3.21 (0.65)
	Low	0.31 (0.21)	0.40 (0.27)	–0.48 (0.78)	–0.82 (0.55)	2.64 (0.60)	2.90 (0.64)
Happiness	High	0.47 (0.31)	0.65 (0.28)	+1.42 (1.16)	+1.28 (1.16)	3.66 (0.33)	3.50 (0.72)
	Low	0.53 (0.33)	0.47 (0.32)	+0.61 (0.95)	+0.78 (0.62)	2.33 (0.67)	2.44 (0.78)
Surprise	High	0.56 (0.21)	0.59 (0.23)	+1.25 (1.07)	+0.91 (1.53)	3.86 (0.57)	4.00 (0.70)
	Low	0.47 (0.18)	0.72 (0.30)	+1.08 (0.84)	+0.85 (1.00)	3.30 (0.57)	3.31 (0.81)
Neutral	Neutral	0.72 (0.35)	0.90 (0.14)	–0.35 (0.65)	+0.18 (1.06)	1.83 (0.67)	1.88 (0.82)

for the HC group).² The groups did not differ in how they rated sadness, happiness, pleasant surprise, or neutral utterances. These patterns are illustrated in Fig. 2.

A 2×2×2 ANOVA involving Group (PD, HC), Valence (positive, negative) and emotional Intensity (high, low) was subsequently run on the data for the six emotions without neutral. Results indicated a significant main effect of Group, $F(1, 31)=6.60, p=0.01, r=0.42$, a main effect of Valence, $F(1, 31)=67.92, p<0.001, r=0.83$, and an interaction of Group by Valence, $F(1, 31)=5.98, p=0.02, r=0.40$. Post-hoc tests of the interaction showed that the PD group assigned higher (i.e., more positive) valence ratings to the class of negative emotions than the HC group, whereas there was no difference in how the two groups assigned ratings to the class of positive emotions. There was also a significant Valence×Intensity interaction, $F(1, 31)=10.62, p=.003, r=0.51$; the valence of high ($M=-0.54$) and low ($M=-0.41$) intensity exemplars of negative emotions were rated as similar in valence, whereas for positive emotions high intensity exemplars were rated as more positive ($M=1.22$) than low intensity exemplars ($M=0.83$). The intensity of utterances did not influence valence ratings in the form of a main effect or as an interaction with Group (all F 's<1.62, p 's>0.21).

c) Intensity rating — To understand factors which may have influenced judgements of the intensity of emotional prosody in our experiment, a 2×7 ANOVA first examined how the groups assigned intensity ratings as a function of the seven emotions. Results yielded a main effect of Emotion, $F(6, 186)=95.03, p<0.001$. In general, the participants rated anger, fear, and surprise as most intense, greater than disgust, which in turn was more intense than happiness and sadness, which in turn exceeded neutral. There was no main or interactive

effect of Group on these findings (both F 's<1.0, p 's>0.54). A 2×2×2 ANOVA then considered how group intensity ratings may have been influenced by valence or intensity dimensions of prosodic expressions omitting the data for neutral. There was a significant main effect on the intensity ratings for Valence, $F(1, 31)=14.05, p=0.001, r=0.56$, and Intensity, $F(1, 31)=117.27, p<0.001, r=0.89$, and a significant interaction of Valence and Intensity, $F(1, 31)=19.95, p<0.001, r=0.63$. Post-hoc comparisons confirmed that “high” intensity utterances were always rated as more intense than “low” intensity utterances irrespective of valence; however, whereas similar ratings were assigned to high intensity exemplars of negative ($M=3.77$) and positive ($M=3.75$) emotions, low intensity exemplars of negative emotions ($M=3.32$) were always perceived as more intense than for positive emotions ($M=2.85$). There was no evidence that these patterns were influenced by PD status for this analysis (all main and interactive effects with Group, F 's<1.0, p 's>0.40).

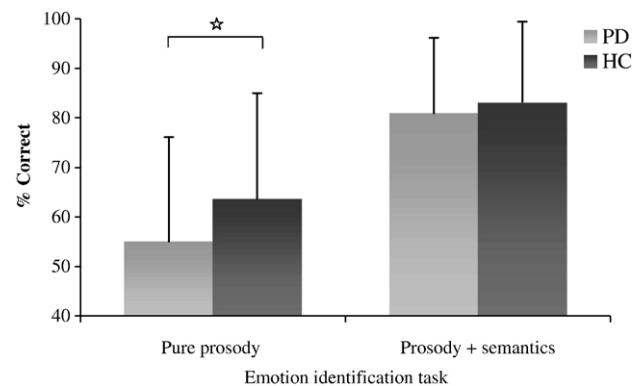


Fig. 1 – Mean recognition of seven distinct emotions from pseudo-utterances (pure prosody task) and utterances with emotionally-biasing prosody and semantic cues (prosody + semantic task) by adults with Parkinson's disease (PD) and healthy controls (HC).

² In the event that depression contributed to prosody identification, a second 2×7 (Group×Emotion) ANOVA was run for the valence rating task excluding the three subjects who were identified as depressed. The outcome of this ANOVA was the same as the main analysis revealing a main effect of Group, $F(1, 28)=5.12, p=0.03$, and Emotion, $F(6, 168)=7.57, p<0.001$.

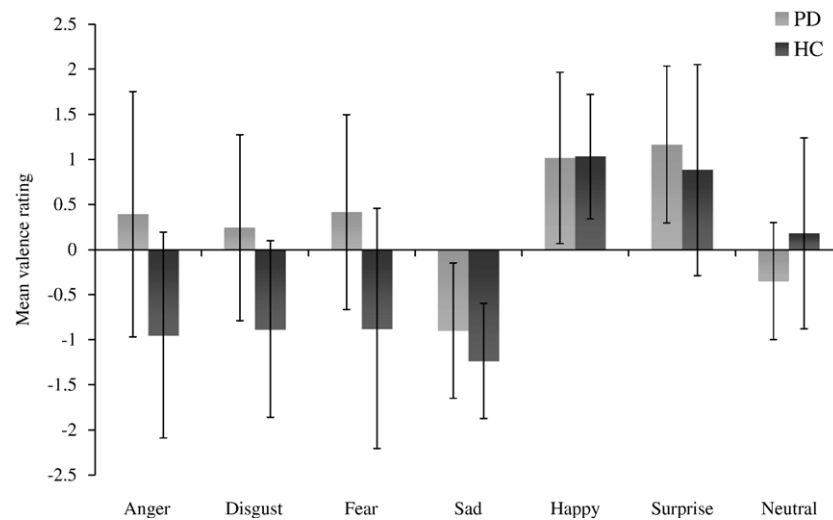


Fig. 2 – Mean group valence ratings of vocal stimuli on a scale from strongly positive (+3) to strongly negative (–3) according to the emotion category of the utterance.

2.3. Relationship between prosody tasks and neuropsychological and clinical variables

To contextualize performance in the four prosody tasks and in reference to some of our background measures, Pearson correlation analyses ($p < 0.05$) were briefly undertaken for the PD group. Based on previously reported relationships in the prosody literature, the background tests of interest here were: emotional (static) face identification; two purported measures of “frontal lobe” functioning, verbal working memory/listening span and Tower of London (measures reported in Table 2); depression (HDI scores); and duration of PD in years (as an estimate of overall disease severity). Prior to the analysis, a z-score transformation was applied to the distribution of measures within the PD group to permit direct comparison of prosody tasks which inferred performance based on accuracy scores (pure prosody, prosody semantic identification tasks) or continuous ratings (valence rating, intensity rating); the mean and standard deviation of the HC group’s performance on the same task always served as the reference point for the transformation. The performance of the PD group on the pure prosody identification task showed a significant, positive correlation with scores in the prosody semantic task ($r = 0.58$) and a marginally significant correlation with the emotional face identification task ($r = 0.49$). Performance on the identification (especially pure prosody) tasks was not related to the ability to rate valence or intensity features of the same stimuli, nor with either “frontal lobe” measure (there was a trend whereby pure prosody identification was associated with working memory capacity ($r = 0.45$) but this was not significant). The ability to rate valence versus intensity characteristics of prosody from emotional utterances did not demonstrate a significant relationship.

We then looked for possible correlations between key clinical variables, such as depression and disease duration, and the performance of PD patients in the two prosody tasks in which they were impaired (valence rating and pure prosody identification). There was no significant evidence of a

relationship between scores on either prosody task and the two clinical variables, suggesting that neither depression nor disease duration contributed in an important manner to the performance of the PD group in these tasks. To further clarify the role of disease severity on our data, we then examined the distribution of individual PD patients according to disease duration to construct two sub-groups around the PD group mean ($M = 8.2$ years): “early” PD (disease duration = 3.0 to 7.0 years, $n = 11$) and “advanced” PD (disease duration = 9.0 to 27.0 years, $n = 5$). Performance of “early” and “advanced” PD patients appeared quite similar in the prosody tasks (for example, “early” patients scored 55.8% and “advanced” patients scored 53.7% correct in the pure prosody identification task). Separate t-tests comparing “early” and “advanced” PD patients revealed no significant differences for pure prosody identification, $t(14) = 0.33$, $p = 0.74$, nor for valence rating, $t(14) = 0.08$; $p = 0.94$.

3. Discussion

This report delves into recent debate about the cognitive sequelae of Parkinson’s disease and the role of the basal ganglia in processing emotional prosody. A novel array of tasks was designed to tap how PD patients process different emotional and affective meanings of prosody when listening to a controlled set of vocal stimuli. As anticipated, our PD patients were impaired in the ability to identify emotions from utterances that did not provide any verbal–semantic cues, and they also displayed problems to rate the valence of prosodic cues for certain emotions, especially for anger, disgust, and fear. In contrast, there was no evidence in our data that PD patients were differentially sensitive to the intensity of emotional stimuli in the vocal channel as discussed below.

The prosodic impairments we observed did not appear to be a consequence of reduced cognitive capacities which are commonly associated with PD, as our patients were not demented and performed well in most tests of executive

functioning. Although there were marked differences in the verbal working memory performance of individuals within the PD group as compared to the HC group—and these deficits are known to contribute to certain communication deficits in PD patients (Hochstadt et al., 2006; Monetta and Pell, 2007)—measures from our three main prosody tasks did not show a meaningful correlation with measures of working memory capacity. Prosody deficits also did not seem to be a consequence of increasing disease severity, as patients in the early years of PD performed in a manner comparable to patients at a more advanced stage of the disease. Finally, it is unlikely that task-related response demands of associating emotional meanings from one channel to another, i.e., from prosodic expressions to verbal labels which specified the response alternatives, was a factor in the performance of our PD group (Breitenstein et al., 1998; Pell and Leonard, 2003). This assertion is substantiated by the fact that the patients could successfully associate emotional meaning from facial expressions or verbal scenarios with the verbal labels in background tasks which imposed relatively similar demands on the participants.

Another critical factor to consider in studies of affective processing in PD is the role of depression. Three patients in our PD group fit the criteria for depression (and were receiving anti-depressants) and this fact led to significant group differences on our depression measure overall. However, our analyses for the pure prosody identification and valence rating task established that the PD group was significantly impaired relative to the control group even when these three patients were removed from the analyses and there was no evidence of a correlation between depression and our prosody measures for the PD group overall. Therefore, depression does not seem to be a major factor in explaining why our PD patients exhibited difficulties in prosodic processing, an observation which is consistent with our previous studies (Pell, 1996; Pell and Leonard, 2003) and the bulk of the literature on this topic for comprehension (Benke et al., 1998; Breitenstein et al., 2001) and expression (Darkins et al., 1988) of emotional prosody. Thus, while it is likely that clinically significant signs of depression, with or without PD, can contribute to difficulties on tasks of emotional processing or prosody (Kan et al., 2004) this feature does not constitute the source of prosodic deficits in most PD patients.

It can therefore be argued that the prosodic difficulties of our PD group reflect one of the primary consequences of interruptions within the basal ganglia/fronto-striatal system on communication processes, as claimed previously in studies of PD patients (Breitenstein et al., 1998; Pell and Leonard, 2003), patients with focal basal ganglia lesions (Cancelliere and Kertesz, 1990; Starkstein et al., 1994), and functional neuroimaging studies of healthy adults (Kotz et al., 2003; Wildgruber et al., 2005a,b). Our findings underscore that the intact basal ganglia may be essential for deriving certain emotional meanings from the prosodic element of speech, although the source of these deficits cannot be accredited to a more generalized impairment in the comprehension of emotions. Our background tests established that the current PD patients could identify emotions from utterances with emotional verbal semantics (Kan et al., 2002; Pell, 1996) and from static facial expressions (Adolphs et al., 1998; Dewick et al., 1991; Pell

and Leonard, 2005) in a manner resembling healthy adults. This means that, despite evidence here of a significant, positive correlation in the ability of the PD patients to identify emotions in the pure prosody, prosody semantic, and static face tasks, they were only impaired when judging “pure prosody” stimuli, pointing to the selectivity of their impairments to emotional meanings encoded through prosody.

It is commonly held, from different theoretical viewpoints, that humans evaluate vocal expressions of emotion in reference to the intrinsic valence (positive–negative) and intensity of these events (Russell, 1980; Scherer, 1986). When PD patients were required to rate emotional utterances along each of these continua, the results of the valence rating task indicated that the patients were abnormally sensitive to the underlying valence of certain negative emotions, although not all. Both the PD and HC participants rated sad utterances as strongly negative and happy and pleasant surprise utterances as strongly positive, whereas the PD patients rated utterances conveying anger, disgust, and fear as having a positive connotation unlike healthy listeners. A review of Fig. 2 demonstrates that the HC group had little difficulty in associating the seven emotion expressions with the appropriate positive or negative connotation in the expected manner, which suggests that the task was suitable for tapping evaluations of valence properties for the vocal stimuli presented. The ability of the PD patients to normally register the valence of “sad”, “happy” and “pleasant surprise” utterances also implies that the patients could successfully use the rating scale in both directions when attending to valence characteristics of the stimuli and that they could evaluate the negative implications of prosodic cues in certain contexts. Thus, it is reasonable to conclude that the PD patients were selectively aberrant in their sensitivity to specific emotions in the valence rating task, namely anger, disgust, and fear.

On the other hand, the performance of the PD group did not deviate from that of the HC group when rating the relative intensity of vocal stimuli (i.e., intensity rating task). In addition, although we found that measures of pure prosody identification and valence rating were each systematically influenced by the pre-determined (high/low) intensity level of emotional stimuli, the influence of these features on the performance of the PD and HC groups did not vary in any instance. Therefore, there were strong indications in the data that PD patients could decode the intensity/arousal dimension of vocal expressions in a comparable manner to healthy listeners. Previously, Breitenstein et al. (1998) suggested that prosodic comprehension difficulties in PD could be due to a failure to process certain acoustic cues, such as elevated mean pitch which tends to correlate with arousal; however, these researchers presented a small number of emotions for comprehension and did not directly manipulate the intensity dimension of their stimuli. Our direct test of this factor provides no evidence that PD patients are insensitive to the intensity attributes of prosody for a wide range of emotion expressions.

One possible reason that our PD patients were impaired to judge affective valence but not intensity is that the basal ganglia are preferentially involved in the processing of certain negative emotions irrespective of their intensity of expression. The literature on emotional face recognition suggests that

specific neural structures such as the basal ganglia support the processing of anger (Calder et al., 2004; Lawrence et al., 2007) and disgust (Calder et al., 2000; Phillips et al., 1997; Sprengelmeyer et al., 1998) in communication. Impaired recognition of disgust has also been observed in PD patients (Pell and Leonard, 2003; Yip et al., 2004) and patients with basal ganglia dysfunction due to Huntington's disease (Gray et al., 1997; Sprengelmeyer et al., 1997). These findings appear to fit with the current observation that PD patients were insensitive to the valence attributes of anger and disgust. We additionally found that PD patients were abnormal in how they processed fear, an emotion that is frequently linked to properties of the amygdala (Adolphs et al., 1994; Calder et al., 1996; Phillips et al., 1998) and which is often impaired in patients with PD as well (Breitenstein et al., 1998; Yip et al., 2004). There is growing evidence that the neuropathological development of PD involves changes in the amygdala (Braak et al., 1996; Braak et al., 1994; Harding et al., 2002) and that the amygdala, in conjunction with the basal ganglia, may be vital to process vocal attributes of fear (Anderson and Phelps, 1998). These data might explain our patients' impairment for rating fear in certain conditions. If so, future research may find that impaired processing of certain negative emotions in PD is due to the multiple effects of basal ganglia damage on emotion-specific brain networks for processing these expressions. However, this explanation is somewhat problematic for our data because PD patients were only selectively impaired for negative emotions in the valence rating and not the corresponding pure prosody identification task, and they were not impaired at all to recognize emotional faces in our background task.

An alternative possibility is that deficits for processing anger, disgust, and fear in PD patients reflect a central inability to generate an appropriate affective response to certain aversive emotions, which hampers their ability to assess the valence of these expressions. According to appraisal theorists (e.g., Scherer, 1986), a critical distinction shared by expressions of anger, disgust, and fear is that these emotions involve greater negative vigilance and urgency to withdraw from a situation than the other emotions tested in this study — sadness, happiness, pleasant surprise, or neutral. In the context of PD, it can be speculated that the transfer of information from a cognitive appraisal mechanism which initiates or plans appropriate somato-motor changes in response to aversive vocal stimuli is somehow disrupted (Anderson and Phelps, 1998; Bowers et al., 2006). The involvement of the basal ganglia in an appraisal process which retrieves somato-motor information to inform the nature of potentially aversive emotional stimuli (and to trigger subsequent actions) is reasonable, given the related role of the basal ganglia for transmitting information in the planning of overt motor responses in behaviour. The specificity of a basal ganglia mechanism for appraising aversive vocal stimuli has been suggested previously (Anderson and Phelps, 1998). This explanation, if proven true, is in line with the view that understanding a speaker's emotion is achieved by *simulating* somato-motor elements of an observed emotion through the activation of associated brain regions (Adolphs, 2002).

The idea that the basal ganglia facilitate meaningful processing of sequential information in speech such as

prosodic expressions has also been advanced (Meyer et al., 2004; Pell and Leonard, 2003). This hypothesis, while not the focus of this study, could partially explain the pattern of findings observed across our three pure prosody tasks. Acoustic investigations show that the perceived intensity of prosodic expressions correlates strongly with a single prosodic cue, the relative amplitude (loudness) of the vocal stimulus (Banse and Scherer, 1996; Juslin and Laukka, 2003). On the other hand, the identification of basic emotion categories or rating valence properties of these stimuli cannot be predicted from a single acoustic-perceptual parameter in speech, but rather, require attention to multiple cue changes over time (e.g., changes in relative pitch height and variation, voice quality, speech rate, and amplitude, Banse and Scherer, 1996; Scherer, 2003). Acoustic analyses of the stimuli presented here confirm that the emotion categories had distinct acoustic profiles even when a small number of common parameters are examined. It is therefore possible that the PD patients evaluated in this study were able to execute prosodic judgements when this processing involved a single, highly salient acoustic parameter such as in the intensity rating task, but that meanings were increasingly lost when judgements relied on more complex processing of multiple prosodic cues over time (Pell and Leonard, 2003).

Interestingly, we observed a corresponding pattern of results when the same PD patients were asked to rate attitudinal meanings of prosody from a similar set of utterances; we found that the PD patients could successfully harness a single cue, pitch direction, to rate the intended politeness of speakers, but that they failed to appropriately judge the confidence of speakers which is signaled by a more complex acoustic representation (Monetta et al., *in press*). This comparison highlights that it is the sequential organization of the cues and not the type of cue that is likely affected in PD and perhaps other conditions which compromise the basal ganglia. Without doubt, the role of the basal ganglia in sequencing for motor (Marsden and Obeso, 1994) as well as cognitive (Harrington et al., 2004; Hochstadt et al., 2006) functions is firmly established. This specialization may well extend to elaborating the various meanings encoded by prosodic cue sequences in speech, whether they refer to emotions, attitudes, or other vocal intentions.

In summary, recurring evidence that PD patients exhibit deficits for emotional prosody and that they tend to be less sensitive to negative displays of emotion suggest different ways that the basal ganglia and fronto-striatal circuitry are critically involved in vocal emotion processing. In light of evidence that understanding emotional prosody activates a wide cortical and sub-cortical network in the brain (e.g., Kotz et al., 2003), it appears that this form of information processing is critically modulated by the basal ganglia in at least two ways: the basal ganglia serve a general role in sequencing behaviours, including perceptual sequencing; and possibly, these structures are crucial to retrieve somato-motor information necessary to understand certain types of negative emotional stimuli. Additional research which manipulates the affective dimensions of emotional stimuli from different dynamic sources will prove highly useful for corroborating these claims.

4. Experimental procedures

4.1. Participants

Sixteen adults (7 female, 9 male) diagnosed with idiopathic Parkinson's disease (PD) participated in the study. The patients averaged 66.0 years in age (S.D.=9.0), had 14.8 years of formal education (S.D.=2.9) and were native speakers of English. Diagnosis of idiopathic PD was confirmed on the basis of motor criteria by a residing neurologist; the average duration of PD (post-diagnosis) was 8.2 years (S.D.=3.6, Range=3.0–27.3). The severity of motor signs for these individuals was characterized as mild to moderate (stages I to IV according to [Hoehn and Yahr, 1967](#) criteria, Mean=2.7). All patients were receiving medication for PD during testing as follows: Levodopa-carbidopa ($n=10$), Dopamine agonists/Mirapex ($n=5$), MAO-B inhibitor/Selegiline ($n=3$), COMT inhibitor ($n=3$), amantadine ($n=6$) and Permax ($n=4$). Three PD patients were receiving an antidepressant.

A group of 17 healthy control (HC) participants (7 female, 10 male) matched for age (Mean=66.9 years, S.D.=9.3), and education (Mean=15.5 years, S.D.=2.5) were tested for comparison purposes. With the exception of PD, individuals with serious medical or neurological conditions (e.g., stroke) or a history of alcohol abuse were excluded from both groups. As well, individuals in each group were first screened using the Mattis Dementia Rating Scale ([Mattis, 1988](#)) to establish evidence of intact intellectual status before entry into the study.³ A *t*-test confirmed that there were no differences between the groups in overall mental status, $t(31)=-0.76$, $p=0.40$. Depression was estimated using the Hamilton Depression Inventory-Short form; one PD participant satisfied the criteria for severe depression (score=21.5), one PD patient was moderately depressed (score=14) and one individual with PD was considered mild (score=10.5). The two groups differed significantly on the depression measure, $t(31)=2.56$, $p=0.01$, although this difference was no longer evident when the three PD patients who fit the criteria for depression were removed from the analysis, $t(28)=1.77$, $p=0.09$. A pure-tone audiometric screening was performed at the onset of testing to ensure that each participant had acceptable hearing to engage in an auditory experiment, with minimal thresholds of 30 db HL in each ear at the frequencies most important for speech intelligibility (0.5, 1, 2 kHz).

4.2. Neuropsychological testing

All PD and HC participants were assessed using standardized tests of neuropsychological performance, including traditional measures of "frontal lobe" executive functions, and tasks of emotion processing which did not involve prosody. The following neuropsychological tests were administered to each participant: Forward Digit Span; a listening span/verbal working memory task adapted for use with brain-damaged populations ([Tompkins et al., 1994](#)); Color Trail-Making test; verbal fluency; Tower of London; Warrington Recognition

Memory test for faces and words ([Warrington, 1984](#)); and the Benton Phoneme Discrimination and Face Recognition Subtests. In addition, we administered two emotional processing tasks from our previous work which probed how well participants recognize emotions from communicative stimuli which do not contain prosodic cues. In one task (Emotion identification to verbal descriptions), participants were required to name what emotion they would feel in response to a verbal scenario that was described by the examiner (e.g., *You learn that a close friend is moving to another city*). In a second task, participants identified the emotion conveyed by static facial expressions presented on a computer screen (stimuli taken from [Pell and Leonard, 2005](#)). The performance of each group on these tests and the outcome of significance tests are reported fully in [Table 2](#).

4.3. Prosody stimuli

All participants completed four experimental tasks which tapped the comprehension of emotional prosody in various ways, with or without congruent semantic cues. In the three main tasks, language-like "pseudo-utterances" (e.g., *Someone mugged the pazing*) were presented which were semantically-anomalous but which communicated specific emotions unambiguously through the prosody; this type of stimulus has been used successfully in previous studies aimed at tapping prosodic processes independent of concurrent language content (e.g., [Pell and Leonard, 2003](#); [Scherer et al., 1991](#)). In the remaining task (prosody-semantic emotion identification), utterances of similar length and complexity were presented which contained both semantic and prosodic cues which communicated the intended emotion (e.g., for sad: *I didn't make the team*). Utterances in all tasks were approximately 6–10 syllables in length and, once recorded, ranged between 1.2 and 2.5 s in duration when spoken naturally to communicate different target emotions.

Perceptual data from a validation study were used to select stimuli which controlled carefully for the perceived category membership of the emotion expression as well as the relative intensity of expressions included within each emotion category. (There has been little attempt in the existing literature to control for the potential impact of intensity on vocal emotion processing and recognition.) All stimuli were taken from a database of emotionally-inflected utterances in English which is described in detail elsewhere ([Pell, 2002](#); [Pell et al., in review](#)). Stimuli were digitally recorded in a sound-attenuated chamber by six male and six female actors to express seven distinct emotions: anger, disgust, fear, sadness, neutral, happiness, and pleasant surprise. The same set of speakers portrayed all emotions in all conditions. All items were rated by a pilot group of young listeners to establish: a) the level of consensus within the pilot group (in %) for recognizing the intended emotion of the utterance; and b) the strength of the emotion expressed. In the validation study, the raters first judged the target emotion category from among the seven alternatives and subsequently rated the perceived intensity of the selected emotion along a five-point scale (where "5" signifies that the emotion is very strong). All stimuli identified in reference to the target emotion at a level exceeding four times chance by raters in the validation study (i.e.,

³ One additional PD patient was initially entered into the study but did not qualify based on this criterion.

Table 2 – Neuropsychological characteristics of individuals with Parkinson's disease (PD) and healthy control (HC) participants

Task	PD (n=16)		HC (n=17)		t-test
	M	S.D.	M	S.D.	
<i>Perception, memory and executive functions</i>					
Mattis Dementia Rating Scale (/144)	137.8	5.0	139.1	4.4	NS
Hamilton Depression Inventory-SF (/33)	6.3	5.3	2.5	2.8	p=0.01
Benton Phoneme Discrimination (/30)	27.3	1.9	27.7	1.5	NS
Benton Face Recognition (/54)	41.9	5.4	47.6	3.9	p<0.05
Warrington Recognition Memory Face (/50)	39.2	5.3	40.8	5.5	NS
Warrington Recognition Memory Word (/50)	45.6	5.6	48.4	1.8	NS
Auditory digit span, forward (/9)	6.8	0.9	7.2	1.6	NS
Working memory/listening span (correct words recalled, /42)	28.2	5.1	35.5	5.2	p<0.001
Tower of London (initiation time in seconds)	109.8	17.9	117.5	17.6	NS
Verbal fluency task: animals	15.5	4.7	21.2	10.0	NS
Trail-making test B (seconds to complete)	115.7	52.4	93.4	23.8	NS
<i>Emotion processing abilities</i>					
Identify emotions from verbal scenarios (/10)	7.4	1.6	7.9	1.6	NS
Identify emotions from static faces (%)	81.3	21.3	87.3	15.3	NS

NS: Non significant, p>0.05. Group differences were always PD<HC.

57% or greater) were considered candidates for the experiment, although most items were identified by the pilot group at levels greater than 80% correct target identification (see Table 3). To categorize the stimuli as “high” and “low” intensity, the distribution of intensity ratings for items within each category was examined since this distribution is known to vary naturally among emotion categories (e.g., surprise or anger tend to be rated as more intense in general when compared to disgust; see Russell, 1980). On the basis of the distribution of responses obtained from the validation study, stimuli were categorized as having “high” intensity for a given emotion when the mean pilot ratings for an item fell in the upper end of the rating distribution, whereas stimuli were categorized as having “low” intensity when ratings fell in the lower end of the distribution for that emotion. A summary of the perceptual characteristics of stimuli presented in our main tasks and major acoustic features of the selected items are provided in Table 3.

4.4. Prosody tasks

Three central tasks evaluated emotional processing for prosodic features alone and a control task evaluated emotional processing when semantic cues further biased an emotional response. The control task (*prosody-semantic emotion identification*) presented the semantically well-formed sentences and required participants to identify the emotion of the speaker following each utterance from one of seven alternatives (anger, disgust, fear, sadness, neutral, happiness, pleasant surprise). This task was included to isolate possible effects due to prosody versus semantic cues in speech and to facilitate comparisons with previous research (Pell, 1996; Pell and Leonard, 2003).

The three central tasks were constructed from a unitary set of emotionally-inflected pseudo-utterances which were presented in each task but judged differently in each case. A total of eight utterances representing each of the seven emotions

were presented in each task; half of the items representing each emotion had obtained relatively “low” intensity ratings for that category and half of the items had obtained relatively “high” intensity ratings for stimuli within that category. Half of the items representing each emotion/intensity level had been produced by a male versus a female speaker to regulate possible gender effects at the stage of encoding specific emotions. As well, individual speakers were always represented within each emotion category with approximately the same frequency. In the *pure prosody* emotion identification task, participants listened to each utterance and then identified the emotion of the speaker based on their prosodic features in a seven forced-choice response format (alternatives=anger, disgust, fear, sadness, neutral, happiness, pleasant surprise). In the *affective valence rating* task, participants listened to the utterances on a different occasion and rated how positive or negative the speaker sounds in reference to a continuous scale (where -3=very negative and +3=very positive). In the *affective intensity rating* task, participants rated the *strength* of the emotion produced by the speaker following each of the utterances (where 1=not at all strong and 5=very strong). When the valence and intensity rating tasks were administered, participants were never instructed to rate utterances in reference to specific emotion qualities of the stimuli and no mention of specific emotions was made by the examiner in these contexts.

4.5. Procedure

The neuropsychological background testing was always completed first during two 1-h sessions, followed by a 30-min testing session to administer the prosody comprehension tasks (each testing session was separated by a 1-week interval). PD patients were tested at their home at a time of a day at which their motor symptoms were typically least severe, while HC participants were tested in a laboratory at McGill University. For the prosody comprehension tasks,

stimuli were presented by a portable computer controlled by SuperLab 2.0 software (Cedrus corporation, USA). Affective valence and intensity rating task were always presented in the first session and the two identification tasks (pure prosody and prosody-semantic) were presented in the second session. All tasks were fully counter balanced for presentation order between participants within each session. Stimuli within each prosody task were separately randomized and then played over high-quality, volume-adjustable headphones at a comfortable listening level. Participants were seated approximately 60 cm from the computer screen; they were instructed to listen closely to each utterance and then make a decision about the emotion of the speaker (i.e., category or rating judgement) by pressing a button on a Cedrus 730 response box. The response alternatives (emotion labels or numerical scale) were clearly marked both on the response box and on the computer screen. Testing always began with a verbal explanation of the task requirements and a brief practice block (8 trials) followed by two experimental blocks of 28 trials each (7 emotions×8 tokens=56 total trials). No time limitations were imposed and there was no predecided ISI; the next trial was presented only once the participant had provided a response. Participants were paid a nominal fee at the end of testing to cover travel-related expenses and the inconvenience of testing.

4.6. Statistical analyses

Data analysis compared how participants with and without PD judged emotional utterances in each prosody task using analysis of variance (ANOVA). In each of the four tasks, a 2×7 mixed ANOVA was performed to establish how the indepen-

dent groups (PD, HC) judged utterances as a repeated measure of the seven emotion categories investigated (anger, disgust, fear, sadness, happiness, pleasant surprise, neutral). For the three main prosody tasks (i.e., those involving pseudo-utterances), a secondary analysis tested the general effects of prosody valence and intensity on group performance by focusing on data for the six basic emotion categories, excluding neutral, which could be further differentiated by the perceived intensity of the emotion target (high, low). For these analyses, negative emotions (anger, disgust, fear, sadness) and positive emotions (happiness, pleasant surprise) were each averaged according to the level of emotional intensity (high, low) and then submitted to a 2×2×2 ANOVA involving group, valence, and intensity. A final analysis directly compared performance across prosody tasks with the neuropsychological variables. Significant main or interactive effects, whenever warranted, were examined post-hoc using Tukey's HSD procedure ($p<0.05$). A correlation (r -type) estimation of effect size was computed as prescribed by Rosnow and Rosenthal (2003).

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Table 3 – Perceptual and acoustic features of pseudo-stimuli presented in the three main prosody tasks, by emotion and intensity level

Emotion target	Intensity level	Target recognition (%) ^a	Emotion intensity (1 to 5) ^a	Mean f0 (Hz) ^b	f0 variation (S.D.) (Hz) ^c	Mean amplitude (dB) ^d	Speech rate (seconds/syllable)
Anger	High	86.8	3.78	1.67	0.31	1.09	0.22
	Low	80.0	2.77	2.58	0.16	1.07	0.23
Disgust	High	90.0	3.45	2.57	0.21	1.04	0.39
	Low	68.3	2.43	1.47	0.22	1.02	0.27
Fear	High	83.3	3.62	7.25	0.14	1.06	0.21
	Low	84.3	3.19	5.06	0.10	1.07	0.19
Sadness	High	96.8	3.89	2.40	0.27	0.97	0.27
	Low	82.5	2.35	1.39	0.13	0.95	0.27
Happiness	High	86.8	3.62	4.89	0.33	1.06	0.22
	Low	78.5	2.29	2.58	0.19	1.04	0.22
Surprise	High	69.8	4.18	6.68	0.33	1.11	0.23
	Low	81.3	3.20	4.36	0.32	1.11	0.22
Neutral	Neutral	80.4	1.95	-0.12	0.11	1.00	0.19

^a Reflects the consensus of a pilot group of young listeners who first performed a seven choice forced-response emotion identification task, where chance=14.3%, followed by an emotional intensity judgement of the identified target (Pell, 2002; Pell et al., in review).

^b Data for each speaker were normalized to adjust for interspeaker differences in voice pitch using a z score transformation, $f0i = (f0i - f0mean)/s$, where $f0i$ is the observed $f0$ mean of the utterance, $f0mean$ is the mean $f0$ of all neutral utterances recorded by that speaker, and s is the standard deviation (Pell, 2001).

^c The impact of interspeaker differences in pitch height on $f0$ variation due to emotion was normalized by dividing the observed $f0$ S.D. of each utterance by the corresponding mean $f0$ for that item, per speaker.

^d Potential differences in recording levels and microphone distance for multiple speakers were corrected by dividing the observed mean amplitude of the utterance by the mean amplitude of all neutral utterances recorded for that speaker.

REFERENCES

- Adolphs, R., 2002. Neural systems for recognizing emotion. *Curr. Opin. Neurobiol.* 12, 169–177.
- Adolphs, R., Damasio, Tranel, D., Damasio, H., 1994. Impaired recognition of emotion in facial expressions following bilateral damage to human amygdala. *Nature* 372, 669–672.
- Adolphs, R., Schul, R., Tranel, D., 1998. Intact recognition of facial emotion in Parkinson's disease. *Neuropsychologia* 12 (2), 253–258.
- Adolphs, R., Damasio, H., Tranel, D., 2002. Neural systems for recognition of emotional prosody: a 3-D lesion study. *Emotion* 2 (1), 23–51.
- Alexander, G.E., DeLong, M.R., Strick, P.L., 1986. Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annu. Rev. Neurosci.* 9, 357–381.
- Anderson, A.K., Phelps, E.A., 1998. Intact recognition of vocal expressions of fear following bilateral lesions of the human amygdala. *NeuroReport* 9 (16), 3607–3613.
- Banse, R., Scherer, K.R., 1996. Acoustic profiles in vocal emotion expression. *J. Pers. Soc. Psychol.* 70 (3), 614–636.
- Beaucousin, V., Lacheret, A., Turbelin, M.-R., Morel, M.I., Mazoyer, B., Tzourio-Mazoyer, N., 2007. fMRI study of emotional speech comprehension. *Cerebral Cortex* 17 (2), 339–352.
- Benke, T., Bösch, S., Andree, B., 1998. A study of emotional processing in Parkinson's disease. *Brain Cogn.* 38 (1), 36–52.
- Blonder, L.X., Gur, R.E., Gur, R.C., 1989. The effects of right and left hemiparkinsonism on prosody. *Brain Lang.* 36, 193–207.
- Bowers, D., Miller, K., Mikos, A., Kirsch-Darrow, L., Springer, U., Fernandez, H., et al., 2006. Startling facts about emotion in Parkinson's disease: blunted reactivity to aversive stimuli. *Brain* 129, 3356–3365.
- Braak, H., Braak, E., Yilmazer, D., De Vos, R.A.I., Jansen, E.N.H., Bohl, J., et al., 1994. Amygdala pathology in Parkinson's disease. *Acta Neuropathol.* 88 (6), 493–500.
- Braak, H., Braak, E., Yilmazer, D., De Vos, R.A.I., Jansen, E.N.H., Bohl, J., 1996. Pattern of brain destruction in Parkinson's and Alzheimer's disease. *J. Neural Transm.* 103 (4), 455–490.
- Breitenstein, C., Daum, I., Ackermann, H., 1998. Emotional processing following cortical and subcortical brain damage: contribution of the fronto-striatal circuitry. *Behav. Neurol.* 11, 29–42.
- Breitenstein, C., Van Lancker, D., Daum, I., Waters, C.H., 2001. Impaired perception of vocal emotions in Parkinson's disease: influence of speech time processing and executive functioning. *Brain Cogn.* 45 (2), 277–314.
- Caekebeke, J.F.V., Jennekens-Schinkel, A., van der Linden, M.E., Buruma, O.J.S., Roos, R.A.C., 1991. The interpretation of dysprosody in patients with Parkinson's disease. *J. Neurol. Neurosurg. Psychiatry* 54, 145–148.
- Calder, A.J., Young, A.W., Rowland, D., Perrett, D.I., Hodges, J.R., Etcoff, N.L., 1996. Facial emotion recognition after bilateral amygdala damage: differentially severe impairment of fear. *Cogn. Neuropsychol.* 13 (5), 699–745.
- Calder, A.J., Keane, J., Manes, F., Antoun, N., Young, A.W., 2000. Impaired recognition and experience of disgust following brain injury. *Nat. Neurosci.* 3 (11), 1077–1078.
- Calder, A.J., Lawrence, A.D., Young, A.W., 2001. Neuropsychology of fear and loathing. *Nat. Rev., Neurosci.* 2, 352–363.
- Calder, A.J., Keane, J., Lawrence, A.D., Manes, F., 2004. Impaired recognition of anger following damage to the ventral striatum. *Brain* 127 (9), 1958–1969.
- Cancelliere, A.E.B., Kertesz, A., 1990. Lesion localization in acquired deficits of emotional expression and comprehension. *Brain Cogn.* 13, 133–147.
- Cheung, C.C.Y., Lee, T.M.C., Yip, J.T.H., King, K.E., Li, L.S.W., 2006. The differential effects of thalamus and basal ganglia on facial emotion recognition. *Brain Cogn.* 61, 262–268.
- Darkins, A.W., Fromkin, V.A., Benson, F., 1988. A characterization of the prosodic loss in Parkinson's disease. *Brain Lang.* 34 (2), 315–327.
- Dewick, H.C., Hanley, J.R., Davies, A.D.M., Playfer, J., Turnbull, C., 1991. Perception and memory for faces in Parkinson's disease. *Neuropsychologia* 29 (8), 785–802.
- Dujardin, K., Blairy, S., Defebvre, L., Duhem, S., Noël, Y., Hess, U., et al., 2004. Deficits in decoding emotional facial expressions in Parkinson's disease. *Neuropsychologia* 42, 239–250.
- Gray, J.M., Young, A.W., Barker, W.A., Curtis, A., Gibson, D., 1997. Impaired recognition of disgust in Huntington's disease gene carriers. *Brain* 120 (11), 2029–2038.
- Harding, A.J., Stimson, E., Henderson, J.M., Halliday, G.M., 2002. Clinical correlates of selective pathology in the amygdala of patients with Parkinson's disease. *Brain* 125 (11), 2431–2445.
- Harrington, D., Boyd, L., Mayer, A., Shaltraw, D., Lee, R., Huang, M., et al., 2004. Neural representation of interval encoding and decision making. *Cogn. Brain Res.* 21, 193–205.
- Hochstadt, J., Nakano, H., Lieberman, P., Friedman, J., 2006. The roles of sequencing and verbal working memory in sentence comprehension deficits in Parkinson's disease. *Brain Lang.* 97, 243–257.
- Hoehn, M.M., Yahr, M.D., 1967. Parkinsonism: onset, progression and mortality. *Neurology* 17, 427–442.
- Juslin, P., Laukka, P., 2003. Communication of emotions in vocal expression and music performance: different channels, same code? *Psychol. Bull.* 129 (5), 770–814.
- Kan, Y., Kawamura, M., Hasegawa, Y., Mochizuki, S., Nakamura, K., 2002. Recognition of emotion from facial, prosodic and written verbal stimuli in Parkinson's disease. *Cortex* 38 (4), 623–630.
- Kan, Y., Mimura, M., Kamijimo, K., Kawamura, M., 2004. Recognition of emotion from moving facial and prosodic stimuli in depressed patients. *J. Neurol. Neurosurg. Psychiatry* 75 (12), 1667–1671.
- Kotz, S.A., Meyer, M., Alter, K., Besson, M., von Cramon, D.Y., Friederici, A.D., 2003. On the lateralization of emotional prosody: an event-related functional MR investigation. *Brain Lang.* 86, 366–376.
- Lawrence, A.D., Goerendt, I.K., Brooks, D.J., 2007. Impaired recognition of facial expressions of anger in Parkinson's disease patients acutely withdrawn from dopamine replacement therapy. *Neuropsychologia* 45, 65–74.
- Lieberman, M.D., 2000. Intuition: a social cognitive neuroscience approach. *Psychol. Bull.* 126 (1), 109–137.
- Marsden, C.D., Obeso, J.A., 1994. The functions of the basal ganglia and the paradox of stereotaxic surgery in Parkinson's disease. *Brain: J. Neurol.* 117 (Pt 4), 877–897.
- Mattis, S., 1988. *Dementia Rating Scale Professional Manual*. Psychological Assessment Resources, Odessa, FL.
- Monetta, L., Pell, M.D., 2007. Effects of verbal working memory deficits on metaphor comprehension in patients with Parkinson's disease. *Brain Lang.* 101, 80–89.
- Monetta, L., Cheang, H., Pell, M.D., in press. Understanding speaker attitudes from prosody by adults with Parkinson's disease. *J. Neuropsychol.*
- Meyer, M., Steinhauer, K., Alter, K., Friederici, A., von Cramon, D.Y., 2004. Brain activity varies with modulation of dynamic pitch variance in sentence melody. *Brain Lang.* 89, 277–289.
- Pell, M.D., 1996. On the receptive prosodic less in Parkinson's disease. *Cortex* 32 (4), 693–704.
- Pell, M.D., 2001. Influence of emotion and focus location on prosody in matched statements and questions. *J. Acoust. Soc. Am.* 109 (4), 1668–1680.
- Pell, M.D., 2002. Evaluation of nonverbal emotion in face and voice: some preliminary findings on a new battery of tests. *Brain Cogn.* 48, 499–504.
- Pell, M.D., 2006. Cerebral mechanisms for understanding emotional prosody in speech. *Brain Lang.* 96, 221–234.

- Pell, M.D., Leonard, C.L., 2003. Processing emotional tone from speech in Parkinson's disease: a role for the basal ganglia. *Cogn. Affect. Behav. Neurosci.* 3 (4), 275–288.
- Pell, M.D., Leonard, C.L., 2005. Facial expression decoding in early Parkinson's disease. *Cogn. Brain Res.* 23 (2–3), 327–340.
- Pell, M.D., Cheang, H.S., Leonard, C.L., 2006. The impact of Parkinson's disease on vocal-prosodic communication from the perspective of listeners. *Brain Lang.* 97 (2), 123–134.
- Pell, M.D., Alasseri, A., Paulmann, S., Kotz, S.A., in review. Factors in the recognition of emotional prosody: a comparison of three languages.
- Phillips, M.L., Young, A.W., Senior, C., Brammer, M., Andrew, C., Calder, A.J., et al., 1997. A specific neural substrate for perceiving facial expressions of disgust. *Nature* 389 (2), 495–498.
- Phillips, M.L., Young, A.W., Scott, S.K., Calder, A.J., Andrew, C., Giampietro, V., et al., 1998. Neural responses to facial and vocal expressions of fear and disgust. *Proc. R. Soc., B. Biol. Sci.* 265, 1809–1817.
- Rosnow, R., Rosenthal, R., 2003. Effect sizes for experimenting psychologists. *Can. J. Exp. Psychol.* 57 (3), 221–237.
- Russell, J.A., 1980. A circumplex model of affect. *J. Pers. Soc. Psychol.* 39 (6), 1161–1178.
- Sander, D., Grandjean, D., Pourtois, G., Schwartz, S., Seghier, M., Scherer, K.R., et al., 2005. Emotion and attention interactions in social cognition: brain regions involved in processing anger prosody. *NeuroImage* 28, 848–858.
- Scherer, K.R., 1986. Vocal affect expression: a review and a model for future research. *Psychol. Bull.* 99 (2), 143–165.
- Scherer, K.R., 2003. Vocal communication of emotion: a review of research paradigms. *Speech Commun.* 40, 227–256.
- Scherer, K.R., Banse, R., Wallbot, H.G., Goldbeck, T., 1991. Vocal cues in emotion encoding and decoding. *Motiv. Emot.* 15 (2), 123–148.
- Schirmer, A., Kotz, S.A., 2006. Beyond the right hemisphere: brain mechanisms mediating vocal emotional processing. *Trends Cogn. Sci.* 10 (1), 24–30.
- Schröder, C., Möbes, J., Schütze, M., Szymanowski, F., Nager, W., Bangert, M., et al., 2006. Perception of emotional speech in Parkinson's Disease. *Mov. Disord.* 21, 1774–1778.
- Scott, S., Caird, F., Williams, B., 1984. Evidence for an apparent sensory speech disorder in Parkinson's disease. *J. Neurol. Neurosurg. Psychiatry* 47, 840–843.
- Scott, S.K., Young, A.W., Calder, A.J., Hellowell, D.J., Aggleton, J.P., Johnson, M., 1997. Impaired auditory recognition of fear and anger following bilateral amygdala lesions. *Nature* 385, 254–257.
- Speedie, L.J., Brake, N., Folstein, S.E., Bowers, D., Heilman, K.E., 1990. Comprehension of prosody in Huntington's disease. *J. Neurol. Neurosurg. Psychiatry* 53, 607–610.
- Sprengelmeyer, R., Young, A.W., Calder, A.J., Karnat, A., Lange, H., Hömberg, V., et al., 1996. Loss of disgust: perception of faces and emotions in Huntington's disease. *Brain* 119, 1647–1665.
- Sprengelmeyer, R., Young, A.W., Sprengelmeyer, A., Calder, A.J., Rowland, D., Perrett, D., et al., 1997. Recognition of facial expressions: selective impairment of specific emotions in Huntington's disease. *Cogn. Neuropsychol.* 14 (6), 839–879.
- Sprengelmeyer, R., Rausch, M., Eysel, U.T., Przuntek, H., 1998. Neural structures associated with recognition of facial expressions of basic emotions. *Proc. R. Soc. Lond.* 265, 1927–1931.
- Starkstein, S.E., Federoff, J.P., Price, T.R., Leiguarda, R.C., Robinson, R.G., 1994. Neuropsychological and neuroradiologic correlates of emotional prosody comprehension. *Neurology* 44 (3), 515–522.
- Suzuki, A., Hoshino, T., Shigemasu, K., Kawamura, M., 2006. Disgust-specific impairment of facial expression recognition in Parkinson's Disease. *Brain* 129, 707–717.
- Tompkins, C.A., Bloise, C.G.R., Timko, M.L., Baumgaertner, A., 1994. Working memory and inference revision in brain-damaged and normally aging adults. *J. Speech Hear. Res.* 37, 896–912.
- Van Lancker Sidtis, D., Pachana, N., Cummings, J., Sidtis, J., 2006. Dysprosodic speech following basal ganglia insult: toward a conceptual framework for the study of the cerebral representation of prosody. *Brain Lang.* 97 (2), 135–153.
- Warrington, E., 1984. Recognition Memory Test. NFER-Nelson, Windsor.
- Weddell, R.A., 1994. Effects of subcortical lesion site on human emotional behavior. *Brain Cogn.* 25, 161–193.
- Wieser, M., Mühlberger, A., Alpers, G.W., Macht, M., Ellgring, H., Pauli, P., 2006. Emotion processing in Parkinson's disease: dissociation between early neuronal processing and explicit ratings. *Clin. Neurophysiol.* 117, 94–102.
- Wildgruber, D., Riecker, A., Hertrich, I., Erb, M., Grodd, W., Ethofer, T., et al., 2005a. Identification of emotional intonation evaluated by fMRI. *NeuroImage* 24, 1233–1241.
- Wildgruber, D., Riecker, A., Hertrich, I., Erb, M., Grodd, W., Ethofer, T., et al., 2005b. Identification of emotional intonation evaluated by fMRI. *NeuroImage* 24, 1233–1241.
- Yip, J.T.H., Lee, T.M.C., Ho, S.-L., Tsang, K.-L., Li, L.S.W., 2003. Emotion recognition in patients with idiopathic Parkinson's Disease. *Mov. Disord.* 18 (10), 1115–1122.
- Yip, J.T.H., Leung, K., Li, L.S.W., Lee, T.M.C., 2004. The role of sub-cortical brain structures in emotion recognition. *Brain Inj.* 18 (12), 1209–1217.