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A Meta-Analysis of Performance on Emotion Recognition Tasks in Parkinson's Disease

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Individuals with Parkinson's disease (PD) have shown deficits in the ability to recognize emotion. However, these results have been inconsistent. In addition, questions remain about whether any deficit in PD is secondary to depression and broader cognitive impairments, and the effects of stimulus modality, task type, and specific emotion remain unclear. A meta-analysis of 34 comparisons, using data from 1,295 individual participants, was conducted to (a) provide a reliable estimate of the magnitude of the purported deficit in emotion recognition and (b) examine the influence of several potential moderators of emotion recognition abilities in PD. Results show a robust link between PD and specific deficits in recognizing emotion, from both the face and the voice (overall effect size $g = 0.52$). The deficit extends across stimulus modalities and task types and is particularly acute with respect to negative emotions. Although this deficit does not appear to be secondary to comorbid depression or visuospatial impairments, the potential role of working memory constraints warrants further investigation. We highlight the potential implications of these findings for communication abilities in PD.

Keywords: Parkinson's disease, emotion recognition, prosody, facial displays, meta-analysis

The ability to infer other people's emotional states is crucial for normal social interaction. Emotional states, broadcast through relatively uncontrollable and often subtle changes in facial and prosodic configurations, preview an individual's future course of action. As a result, being able to discern meaning from these subtle changes confers the ability to plan appropriate social behaviors and to maintain interpersonal harmony. Indeed, throughout the life span, those who are more successful in deciphering others' emotional states are more successful, both socially and vocationally (Boyatzis & Satyaprasad, 1994; Carton, Kessler, & Pape, 1999; Elfenbein, Foo, White, Tan, & Aik, 2007; Kornreich et al., 2002).

There is reason to believe that individuals with Parkinson's disease (PD) might be impaired in the recognition of emotion from facial cues. A defining feature of PD is the loss of dopaminergic innervation to the ventral striatum, subthalamic nucleus (STN), and other basal ganglia structures. The ventral striatum and STN have known connections with other brain regions important for facial emotion recognition, including the orbitofrontal cortex and the amygdala (Adolphs, 2002; Le Jeune et al., 2008). The basal ganglia more generally appear to play a role in recognizing emotions from facial cues, as part of a distributed network of cortical and subcortical structures (Adolphs, 2002; Cancelliere & Kertesz, 1990). There is also reason to suspect deficits in the recognition of emotion from prosody in PD, as prosodic cues are processed by the basal ganglia, the right frontoparietal cortex and potentially the amygdala (Adolphs, Damasio, & Tranel, 2002; Breitenstein, Daum, & Ackermann, 1998; Kotz et al., 2003; Paulmann, Pell, & Kotz, 2008; Pell & Leonard, 2003; Starkstein, Federoff, Price, & Leiguarda, 1994).

Indeed, several studies (e.g., Ariatti, Benuzzi, & Nichelli, 2008; Beatty, Goodkin, Weir, & Staton, 1989; Blonder, Gur, & Gur, 1989) have documented emotion recognition deficits in PD relative to matched controls. However, other studies (e.g., Adolphs, Schul, & Tranel, 1998; Caekebeke, Jennekens-Schinkel, Van der Linden, & Buruma, 1991; Madeley, Ellis, & Mindham, 1995; Pell & Leonard, 2005) have failed to document a deficit. Still others (e.g., Lawrence, Goerendt, & Brooks, 2007; Suzuki, Hoshino, Shigemasu, & Kawamura, 2006) have documented deficits in recognizing some of the so-called "basic" emotions (anger, fear, disgust, happiness, sadness, and surprise) but not others. Finally, although some studies (e.g., Ariatti et al., 2008; Dara, Monetta, & Pell, 2008) have documented deficits in recognizing emotion from both facial displays and prosody, others (Clark, Neargarder, & Cronin-Golomb, 2008; Kan, Mimura, Kamijima, & Kawamura, 2004) have documented deficits in recognizing emotion only in one stimulus modality.

These inconsistencies may result from the absence of a robust emotion recognition deficit in PD. Alternatively, they may result from substantial cross-study variations in methodology. For instance, there has been substantial heterogeneity in patient samples across studies, in terms of disease severity and duration, mental status, age, medication status, and other criteria. This is important given that individual differences in intelligence, age, attention, verbal ability, and task-specific motivation are known to influence emotion recognition abilities (Herba & Phillips, 2004). On a related note, studies have varied in the extent to which PD patients have been matched with control group members on important individual different characteristics. For instance, some studies

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have included among their PD groups individuals with significantly higher self-reported depression symptomology than controls. As depression itself is linked with deficits in identifying the emotional tone of faces (Feinberg, Rifkin, Schaffer, & Walker, 1986) and voices (Kan et al., 2004), it is possible that significant group differences in some past studies resulted from inadequate control of such individual difference factors. Finally, there has been substantial variation across studies in the assessment of emotion recognition abilities. Different studies have administered emotion recognition tasks that vary considerably in terms of their difficulty and the extent to which they place demands on nonemotional processes. Recent studies (e.g., Clark et al., 2008; Suzuki et al., 2006) have carefully controlled for task difficulty or task requirements that could otherwise inflate group differences. Earlier studies, however, tended to disregard the potential impact of these task factors.

We conducted a meta-analysis to assess the existing literature comparing emotional recognition abilities in individuals with PD and healthy controls. Our first aim was to provide a reliable estimate of the magnitude of the purported deficit in emotion recognition. Our second aim was to examine the influence of several potential moderators of emotion recognition abilities in PD. We identified, *a priori*, six potential moderators. Three potential moderators concerned the emotion recognition tasks. They were stimulus modality, task type, and emotion displayed. The other three potential moderators concerned the participants. They were medication status, depression status, and performance on executive function and visuospatial ability tasks. In the following sections, we review evidence for the potential moderating role of these six factors.

Stimulus Modality

An early study of emotion recognition abilities in PD asked participants to match the emotional tone conveyed in brief spoken passages to appropriate facial expressions (Scott, Caird, & Williams, 1984). The PD patients were impaired on this task, though the source of the deficit—difficulty discerning meaning from prosodic cues, difficulty discerning meaning from facial cues, or both—was undetermined. More recent studies have examined the processes of facial and prosodic emotion recognition separately. Some studies (e.g., Ariatti et al., 2008; Dara et al., 2008; Yip, Lee, Ho, Tsang, & Li, 2003) have documented deficits in emotion recognition from both facial displays and prosody, suggesting the existence of a cross-modal deficit. This would imply that in everyday conversations, individuals with PD are unable to compensate for difficulties inferring meaning from faces by focusing on voices (or vice versa). However, other studies have documented modality-specific deficits, though not consistently (facial only: Clark et al., 2008; prosody only: Kan et al., 2004). Within the broader neuropsychological literature, it is unclear whether a single system underlies the recognition of a given emotion from different sensory modalities. For instance, one study (Phillips et al., 1997) revealed that both facial and prosodic expressions of fear activate the amygdala; however, the same study revealed that facial expressions of disgust, but not vocal expressions of disgust, activate the anterior insula. Thus, the extent to which the neural substrates involved in emotion recognition are modality-specific may vary as a function of the specific emotion being portrayed. To our knowledge, there has never been a meta-analytic comparison of PD-related deficits in recognizing emotion from facial versus prosodic displays. The current paper addresses this question by deriving and statistically comparing estimates of deficit effect sizes based on the interpretation of facial and prosodic emotional expressions.

Task Type

Studies in this area have also varied in terms of the type of task participants are asked to complete. Some (e.g., Beatty et al., 1989; Clark et al., 2008; Lachenal-Chevallet et al., 2006) only presented identification tasks, which require participants to select the appropriate label for a given emotional expression. Others (Jacobs, Shuren, Bowers, & Heilman, 1995; Madeley et al., 1995; Yoshimura, Kawamura, Masaoka, & Homma, 2005) only presented discrimination tasks. Discrimination tasks typically involve judging whether two stimuli express the same or a different emotion. Alternatively, they may require the participant to match a target emotional face (or voice) with one of several alternatives. Both identification and discrimination require participants to categorize the intended meaning of the stimulus, but only identification presents the added demand of producing a semantic label. The majority of published reports have presented participants with both identification and discrimination tasks. In addition, some studies have asked participants to rate the extent to which a series of facial displays or prosodic samples express a given emotion, either alone (Adolphs et al., 1998) or in conjunction with identification and discrimination tasks (Pell & Leonard, 2005; Suzuki et al., 2006). It is believed that because rating tasks do not require the act of categorization, they make fewer demands on working memory (Pell & Leonard, 2003). In any case, the task-type variation in past studies has made it difficult to integrate divergent results. In this meta-analysis we statistically compared deficit effect sizes derived from identification, discrimination, and rating tasks.

Emotion

It is currently unclear whether PD selectively (or even disproportionately) impairs the recognition of specific emotions. In the broader neuropsychological literature, there is substantial evidence for neuroanatomical specificity in the recognition of emotions, particularly for disgust and fear (e.g., Adolphs, Tranel, Damasio, & Damasio, 1994; Sprengelmeyer et al., 1997). Indeed, there is some evidence for selective or disproportionate deficits in PD. However, this evidence is inconsistent, even within single studies. For instance, Suzuki and colleagues (2006), using a refined assessment method that controlled for task difficulty, found a deficit in emotion recognition that was specific to disgust and did not emerge when conventional assessment methods were used. However, conventional assessment methods allowed Kan and colleagues (2004) to document a disproportionate deficit in the recognition of disgust and fear. Unmedicated PD patients in a study by Sprengelmeyer et al. (2003) were impaired in the recognition of facial expressions of anger and disgust, relative to a group of medicated PD patients. However, patients in a study by Lawrence et al. (2007) who were acutely withdrawn from dopamine replacement therapy (DRT) showed normal recognition of disgust, heightened recognition of fear, and diminished recognition of anger.

Clearly, it is difficult to draw conclusions about the specificity of emotion recognition deficits in PD on the basis of past studies. This may be in part because individual studies have based their conclusions on the presence or absence of statistically significant differences, which are biased by sample size. Here, when possible, we present a straightforward comparison of effect sizes derived from investigations of the recognition of specific emotions.

Medication Status

As mentioned, another source of variation among past studies has been the medication status of PD patients. The majority of studies have selected patients who are receiving some form of DRT to combat the pathology of the domaminergic system that characterizes PD. Some studies, however, have intentionally selected participants who are not on DRT, either because they were in the early stages of the disease and not yet taking medication (Dujardin, Blairy, Defebvre, Duhem, et al., 2004; Sprengelmeyer et al., 2003) or were withdrawn from their medication for purposes of the study (Caekebeke et al., 1991; Lawrence et al., 2007). Even among the studies using participants who are generally receiving DRT, there may be substantial "on–off" variation depending on the time of day and when participants last took their medication, with some participants in a state of optimal medication and some participants not. Clarifying the role of medication status may have implications beyond the study of PD, in that it will evaluate the contribution of dopamine-modulated brain regions in the normal recognition of emotion. Therefore, in this paper, we calculated and statistically compared two effect sizes: one derived from a comparison of patients known to be in an optimally medicated state versus healthy controls, and one derived from a comparison of patients known to be off medication versus healthy controls. To the extent that the dopaminergic system makes a powerful contribution to emotion recognition abilities, the latter effect size should be greater than the former.

Depression Status

Depression status is another potential patient-related moderator of the emotion recognition deficit in PD. As mentioned, depression itself is associated with emotion recognition deficits (e.g., Feinberg et al., 1986), and there is a high incidence of depression among individuals with PD (Cummings, 1992). Therefore, when individuals with PD show a deficit in emotion recognition abilities, it is possible that this deficit is not specific to PD but, rather, secondary to depression. Past studies have varied considerably in the ways in which they have dealt with the issue of comorbid depression among their PD participants. The majority of studies have screened out potential participants who have any psychiatric illness. However, only some have taken the additional step of administering a self-report inventory of depression symptomology among the remaining individuals in the PD group, and only some of these have also administered the self-report inventory to the control group to test for significant group differences. Without demonstrating that individuals in the PD group score no higher on self-reports of depression symptomology than their control group counterparts, it is difficult to conclude that a deficit in emotion recognition in PD is not secondary to depression. We took two approaches to clarifying the role of comorbid depression in the emotion recognition deficit in PD. First, restricting our analysis to studies that administered self-report inventories of depression symptomology to both participant groups, we derived and compared two effect sizes: one from studies that essentially controlled for depression by showing no significant group difference in depression symptomology, and one from studies showing greater depression symptomology among the PD group. (No studies showed greater depression symptomology among the control group.) To the extent that any emotion recognition deficit is secondary to depression in PD, the second effect size should be greater than the first. Our second approach was to compute, when possible, the average within-study correlation between emotion recognition accuracy and depression symptomology. To the extent that any emotion recognition deficit is secondary to depression in PD, this correlation should be more negative.

Performance on Executive Function and Visuospatial Ability Tasks

Accurately describing the emotion portrayed in another person's face or voice requires a variety of lower level cognitive abilities. Both facial and prosodic emotion recognition require categorization and working memory skills, which are part of the broader class of executive functions—a set of cognitive processes that allow one "to plan, manipulate information, initiate and terminate activities, and recognize errors"(Goverover, 2004, p. 738). Executive function impairments are commonly noted even in PD patients who do not have dementia (e.g., Zgaljardic, Borod, Foldi, & Mattis, 2003). In addition, facial emotion recognition requires the ability to discriminate facial features, and visuospatial declines have also been reported in PD (Levin, Llabre, Reisman, & Weiner, 1991). Therefore, it is plausible that emotion recognition deficits in PD are at least partially dependent on deficits in executive function (for both facial and prosodic emotion recognition) and visuospatial ability (for facial emotion recognition only). A number of studies of emotion recognition in PD have dealt with this possibility by assessing participants' performance on standardized tasks tapping executive function and visuospatial ability. In this work, to the extent possible, we explored the possibility that deficits in emotion recognition among individuals with PD are part of a more general pattern of cognitive impairment.

Method

Literature Search

To locate relevant studies, we conducted database searches of PsycINFO and PubMed. We began using the keyword *Parkinson* in conjunction with each of the following keywords: *facial expression, decoding*, and *prosody.* We examined the reference list of these articles to search for more potentially relevant studies. This resulted in 257 potentially eligible papers. The abstracts of these papers were then reviewed. After this review, 203 were excluded for a variety of reasons (e.g., they were review papers rather than original studies; they focused on the production rather than the recognition of emotion in PD; they focused on neuroanatomy rather than behavioral performance). Fifty-four potentially eligible papers were read. Twenty of these were excluded, generally because they did not present emotional material to participants (n =

18) or did not include a non-PD control group ($n = 2$, both comparing emotion recognition among PD patients pre- and post-deep brain stimulation). This resulted in the inclusion of 34 papers. One (Haeske-Dewick, 1996) contributed two independent comparisons to the analysis (i.e., there were two different PD groups, each with its own control group), but two papers (Pell & Leonard, 2003, 2005) reported data provided by the same participants. Therefore, the overall effect size estimate—pooled across stimulus modality, task type, and emotion, and participant characteristics—was based on a total of 34 independent comparisons. We conducted the first search in the summer of 2007 and updated it periodically, with the final update occurring in August 2009.

Studies that were included satisfied the following criteria:

1. The patient group had to consist entirely of adults with PD. Most studies (approximately 75%) reported that patients were either formally diagnosed (by neurologists or by attending physicians on the basis of neurological assessment) or recruited from outpatient PD clinics. The remaining papers did not report the method of confirming PD diagnosis.
2. The study had to include a healthy control group. Thirteen studies (37.5%) matched the participant groups on age, education/intellectual functioning, and gender composition. An additional 12 matched only on age and education/intellectual functioning. Four studies matched on age alone, two matched on age and gender composition, and three did not report matching criteria.
3. If data regarding percentage accuracy (or means that could be converted to percentage accuracy) and variance (e.g., standard deviation) were not included in original paper, we contacted individual authors and solicited this information.[1]

Participant Characteristics

A total of 1,295 individual participants contributed data to this meta-analysis (701 healthy controls, 594 individuals with PD). Control and PD participants were both, on average, 63 years old ($SD = 5.39$ and 5.34, respectively). PD participants' mean Hoehn and Yahr score was 2.32 ($SD = 0.62$), indicating bilateral symptoms with some balance deficit but physical independence (Hoehn & Yahr, 1967). Patients averaged 6.84 years ($SD = 2.92$) since diagnosis.

The majority (roughly 80%) of studies reported that patients were being treated with antiparkinsonian medication; however, many of these did not explicitly indicate whether patients were at their optimally mediated "on" stage during assessment. A different group of four studies (Breitenstein, Van Lancker, Daum, & Waters, 2001; Dujardin, Blairy, Defebvre, Duhem, et al., 2004; Lawrence et al., 2007; Sprengelmeyer et al., 2003) included at least one comparison involving patients who were not receiving medication, either because they were early in the course of the disease or because the study was investigating the effects of withdrawal from DRT on emotion recognition. The remaining three studies did not indicate the medication status of patients.

Roughly 80% of studies reported screening out patients who had signs of dementia. One study (Beatty et al., 1989) did not screen for dementia and included patients who scored significantly lower than controls on the Mini-Mental State Exam (Folstein, Folstein, & McHugh, 1975). The remaining studies did not report exclusion criteria. Other common exclusion criteria, for patients and control groups, were presence of neurological or psychiatric diseases (other than PD), hearing or vision disturbance, and history of substance abuse. Twenty studies administered common self-report inventories of depression to members of both groups (control and PD). Of these, seven reported no significant group differences, 10 reported significantly higher scores among the PD group, and the remaining three did not report results. An additional six studies administered depression inventories only to members of the PD group, and five of these reported that all or most patients scored within normal ranges. Finally, nine studies provided no information regarding an assessment of depression severity.

Task Characteristics

The 34 papers included in this meta-analysis contributed a total of 74 nonindependent comparisons. Forty-four comparisons compared the performance of individuals with PD and healthy controls on tasks assessing facial emotion recognition. Of these, roughly half (24) presented participants with the Pictures of Facial Affect stimuli (Ekman & Friesen, 1976), perhaps the most commonly used standard set of facial affect. Other common stimulus sets were the Japanese and Caucasian Facial Expressions of Emotion (JACFEE) series (Matsumoto & Ekman, 1988), used in six comparisons; and a subtest of the Florida Affect Battery (FAB; Bowers, Blonder, & Heilman, 1991), used in five comparisons. Of the 44 comparisons of facial emotion recognition, 15 involved tests of discrimination (e.g., deciding which of two photographs matches a named expression; deciding whether two posers were expressing the same emotion), 26 involved the identification or labeling of an emotion (generally forced choice rather than open ended); and the remaining three were rating tasks (i.e., rating the extent of each emotion portrayed in a given face).

There were 28 comparisons of the ability to infer emotion from prosody; of these, six used a subtest of the FAB and the remaining used original stimulus sets constructed by the authors (generally nonsense utterances or emotionally neutral content, all spoken in emotional vocal tones). Nine involved tests of discrimination, 18 involved tests of identification, and one involved a rating task.

Finally, two comparisons asked participants to match facial to prosodic expressions (i.e., selecting the prosodic material that best matches a given facial expression of emotion). In most of the 74 total comparisons, participants were presented with portrayals of anger, disgust, fear, happiness, sadness, surprise, or some combination thereof.

Data Extraction

All data were extracted by the first author and checked by a research assistant. Data were extracted from text when possible. If means and variance data were not included in the article, we contacted the study authors requesting this information. This left three studies for which data had to be extracted from figures. Two

[1] One study did not provide data sufficient to calculate effect size (Madeley et al., 1995). Because the authors reported no significant effect of PD on emotion recognition accuracy, we conservatively assigned a value of 0.00 as the effect size g.

research assistants, working independently, extracted data; their data were averaged. The first author performed all calculations. The second author checked all calculations.

Statistical Analyses

To draw meaningful comparisons of means and variance across comparisons, we computed several effect size statistics. The effect size statistic used to measure the strength of the effect was Hedge's *g*, a variation on Cohen's *d* that corrects for biases due to small sample sizes. Our analysis involved multiplying the raw effect size *g* by the inverse of its variance so that the more reliably estimated effect sizes had more weight in the aggregated analysis. The resulting weighted average *g* describes the magnitude and direction of difference in accuracy scores between PD groups and control groups. Positive *g*s indicate a deficit among the Parkinson's disease group. The associated 95% confidence intervals indicate the range within which the effect size *g* is expected to fall 95% of the time. The *Z* statistic is calculated as the weighted average *g* divided by the square root of the variance. If *Z* exceeds 1.96, we can reject the null hypothesis that the population effect size is zero. The Q_W statistic tests the degree of homogeneity within each aggregated effect size. In tests of potential moderator variables, the Q_B statistic tests the degree of homogeneity between moderator divisions (e.g., facial expression recognition versus prosodic expression recognition; Hedges & Olkin, 1985). Q_B identifies significant differences between moderator divisions.

In computing these effect size statistics, we used both fixed and random effects models. In comparison with fixed effects models, random effects models are more conservative and have the advantage of allowing for the generalization of findings to studies beyond those included in the analysis (Shadish & Haddock, 1994). However, due to the relatively small number of comparisons used in many of the analyses, which calls into question the reliability of the random effects model statistics (Hedges & Vevea, 1998), we focus on results derived from the fixed effects model. We present results from both models in the associated tables.

In many studies used in this meta-analysis, participants contributed data to more than one comparison. We considered it important to ensure that each estimate of effect size was based on independent data. Therefore, if more than one PD group (e.g., right- vs. left-hemiparkinsonism; early- vs. late-stage PD) was compared against a single control group, we generally collapsed across the two PD groups before comparing performance data against the control group.[2]

Results

Aggregated Effect Size

Figure 1 is a forest plot of the individual study effect size estimates (standardized mean differences). With fixed effects analysis, the 34 independent comparisons from 1,295 independent participants yield a mean effect size of 0.52, 95% CI [0.40, 0.63], $Z = 8.69$, $p < .001$. The random effects analysis produced a similar result, effect size $g = 0.46$, 95% CI [0.26, 0.65], $Z = 4.52$, $p < .001$ (see Table 1).

The magnitude of this difference, on the standardized mean difference scale, was about half of a standard deviation. This effect would be considered medium in magnitude (Cohen, 1988). A scatter plot of the effect sizes by sample size indicates that the effects center on a median *g* of approximately 0.39 (see Figure 2). A majority (approximately 75%) of the comparisons reveal a deficit among the Parkinson's disease sample relative to the control group. In sum, the initial answer to our query is that on the average individuals with Parkinson's disease are impaired in their ability to recognize emotion from facial and prosodic displays. At the same time, the mean effect size derived from the fixed effects model is highly heterogeneous across samples, $Q_W(33) = 99.62$, $p < .001$. The diversity of the effect sizes suggests a need to explore potential moderating variables. We begin with stimulus modality.

Stimulus Modality

Table 2 presents the effect of Parkinson's disease on emotion recognition as a function of stimulus modality: facial versus prosodic expressions. Participants with PD appear to be more impaired in judging emotion from prosodic expressions ($g = 0.70$) than from facial expressions ($g = 0.48$), $Q_B(1) = 3.87$, $p < .05$. Again, the mean effect sizes are highly heterogeneous within both stimulus modalities, facial, $Q_W(27) = 79.57$, $p < .001$, and prosodic, $Q_W(14) = 77.22$, $p < .001$. Accordingly, we next explored the moderating potential of task type.

Task Type

We further partitioned effect size variance into groups according to whether participants were asked to complete identification, discrimination, or rating tasks, separately for facial versus prosodic expressions. We first examined the moderating role of task type with regard to facial emotion recognition. As indicated in Table 3, the effect size estimates derived from identification tasks ($g = 0.50$) and discrimination tasks ($g = 0.62$) were higher than that derived from rating tasks ($g = 0.05$). However, the effect size estimate derived from rating tasks was based on only three comparisons and may have been skewed by the very low effect sizes reported by two of these (Adolphs et al., 1998; Pell & Leonard, 2005). Therefore, the stability of this effect size estimate is questionable, and further studies are needed to provide a more reliable estimate of the degree of deficit PD participants may experience with rating tasks. The effect size estimate derived from discrimination tasks is significantly greater than that derived from identification tasks, $Q_B(1) = 9.32$, $p < .01$.

With regard to prosodic expressions, only one study (Pell & Leonard, 2003) produced an effect size estimate for rating tasks ($g = 0.31$). Effect size estimates derived from identification tasks ($g = 0.88$) and discrimination tasks ($g = 0.61$) were substantially higher. Disregarding data from the one estimate of effect size derived from rating tasks, the deficit appears greatest when based on identification tasks, $Q_B(1) = 3.70$, $p < .10$ (see Table 4). In sum, for both facial expressions and prosodic expressions, the

[2] Three studies used a group of PD patients who had undergone deep-brain stimulation (DBS) for medically intractable motor symptoms (Biseul et al., 2005; Dujardin, Blairy, Defebvre, Krystkowiak, et al., 2004; Le Jeune et al., 2008). In these cases, we disregarded the data from the post-DBS groups and used only data from the more representative pre-DBS groups.

deficit appears to be greater for identification and discrimination tasks than for rating tasks, although this conclusion is qualified by the fact that very few studies presented participants with rating tasks.

Specific Emotion

We next examined whether the extent of recognition deficit is moderated by the particular emotion being expressed. Although many studies pooled their results across emotions, some reported results separately for the emotions of anger, disgust, fear, happiness, sadness, and surprise. To maximize the number of comparisons used in this analysis, we chose not to separate results according to stimulus modality (facial, prosodic) and task type (identification, discrimination, rating) and instead collapsed across these factors. The number of comparisons in this analysis ranged from 11 (recognition of fear and surprise) to 17 (recognition of anger and sadness). As indicated in Table 5, the level of deficit ranged from 0.12 (surprise) to 0.32 (anger) and tended to be greater for negative emotions than for the relatively positive emotions of happiness and surprise. The omnibus test for differences in effect size was not significant, $Q_B(5) = 0.36$. However, this test is likely too unfocused to address the specific question of whether negative emotions produce greater deficits than positive emotions. Therefore, to supplement this analysis we also conducted a more focused contrast analysis using the contrast weight (λ) +1 for effect sizes associated with negative emotions (anger, disgust, fear, sadness) and the contrast weight −2 for effect sizes associated with relatively positive emotions (happiness, surprise; Rosenthal & Rubin, 1982). This analysis compared negative and positive emotion recognition within each study and then collapsed those findings across all studies. This more focused analysis suggests that the effect sizes associated with negative emotions were somewhat greater, $Z = 1.94$, $p = .05$ (see Figure 3).

Medication Status

We next questioned whether medication status at the time of assessment moderated the level of emotion recognition deficit. We selected the comparison groups conservatively, including only studies that explicitly reported whether participants were in a relatively hypodopaminergic or dopamine-replete state during the assessment of emotion recognition ability. (We did not use studies that only reported that participants were generally receiving dopaminergic therapy.) This resulted in 22 comparisons, 16 including participants in a relatively dopamine-replete state and six including participants in a relatively hypodopaminergic state. In this and the following analysis, we collapsed across the three stimulus factors (stimulus modality, task type, emotion expressed) to maximize the number of comparisons. As indicated in Table 6, the effect size estimate derived from participants in a relatively hypodopaminergic state ($g = 0.50$) was somewhat higher than that derived from participants in a dopamine-replete state ($g = 0.27$), though this difference was not significant, $Q_B(1) = 1.73$, *ns*.

Depression

Our next analysis examined the possibility that the increased incidence of depression in PD explains the emotion recognition deficit. Again, we chose comparisons conservatively, using only studies that explicitly reported whether participants in the PD group were relatively more (or less) depressed than their control group counterparts in terms of self-reported depression status. This selection process resulted in 17 comparisons. As previously mentioned, in seven of these comparisons the two groups were identified as equally depressed; in the remaining 10, the Parkinson's disease group self-reported as relatively more depressed than the control group. (In no studies did the control group members produce significantly higher scores on depression inventories.) As indicated in Table 7, the effect size estimates derived from the two types of comparisons were identical ($g = 0.49$). This suggests that depression status does not moderate the emotion recognition deficit.

Another way to examine this question involves the within-study correlations between emotion recognition accuracy and scores on depression inventories. Thirteen studies reported that there was no significant correlation between accuracy and scores on depression inventories. Of these, eight reported the actual correlation, the mean of which was $r = .07$ ($SD = 0.14$). In sum, results derived from these two statistical approaches indicate that patients' depression level did not moderate the extent of their emotion recognition deficit.

Role of Executive Function and Visuospatial Deficits

We next investigated the potential that the deficits in emotion recognition are secondary to executive dysfunction. This effort was hampered by the relatively small number of studies that included executive function tasks and included data on the presence of between-groups differences (i.e., whether participants in the PD group performed significantly worse on executive function tasks). In addition, among the subset of studies that included executive function tasks and presented data on group differences ($n = 8$), there was substantial methodological variance because different studies focused on different aspects of executive function. For example, Breitenstein and colleagues (2001) were primarily interested in working memory and selective attention aspects of executive function and therefore included verbal fluency tasks, the Wisconsin Card Sorting Test (Nelson, 1976), and a modified version of the Listening Span Test (Daneman & Carpenter, 1980). By contrast, Clark et al. (2008) were mainly interested in categorization aspects of executive functioning and therefore included a nonemotional categorization test. Inspection of the papers revealed that six datasets included PD participants who performed significantly worse than control group participants on a task tapping at least one aspect of executive function, and two papers reported a lack of significant group differences. Because the sample size for this comparison would be relatively small and unstable, we did not compute and compare separate effect sizes. Instead, we present a qualitative review of this issue in the Discussion section.

Next, we turned to an exploration of visuospatial deficits among PD participants. The most commonly used test of visuospatial ability was the Benton Facial Recognition Task (Benton, Hamsher, Varney, & Spreen, 1983), which requires participants to discriminate between the faces of different unfamiliar people, all with neutral expressions. Nineteen separate studies (reporting on 20 separate comparisons) administered this or a similar task to both participant groups and reported the results. Of these, 15 comparisons included no significant group differences and the remaining

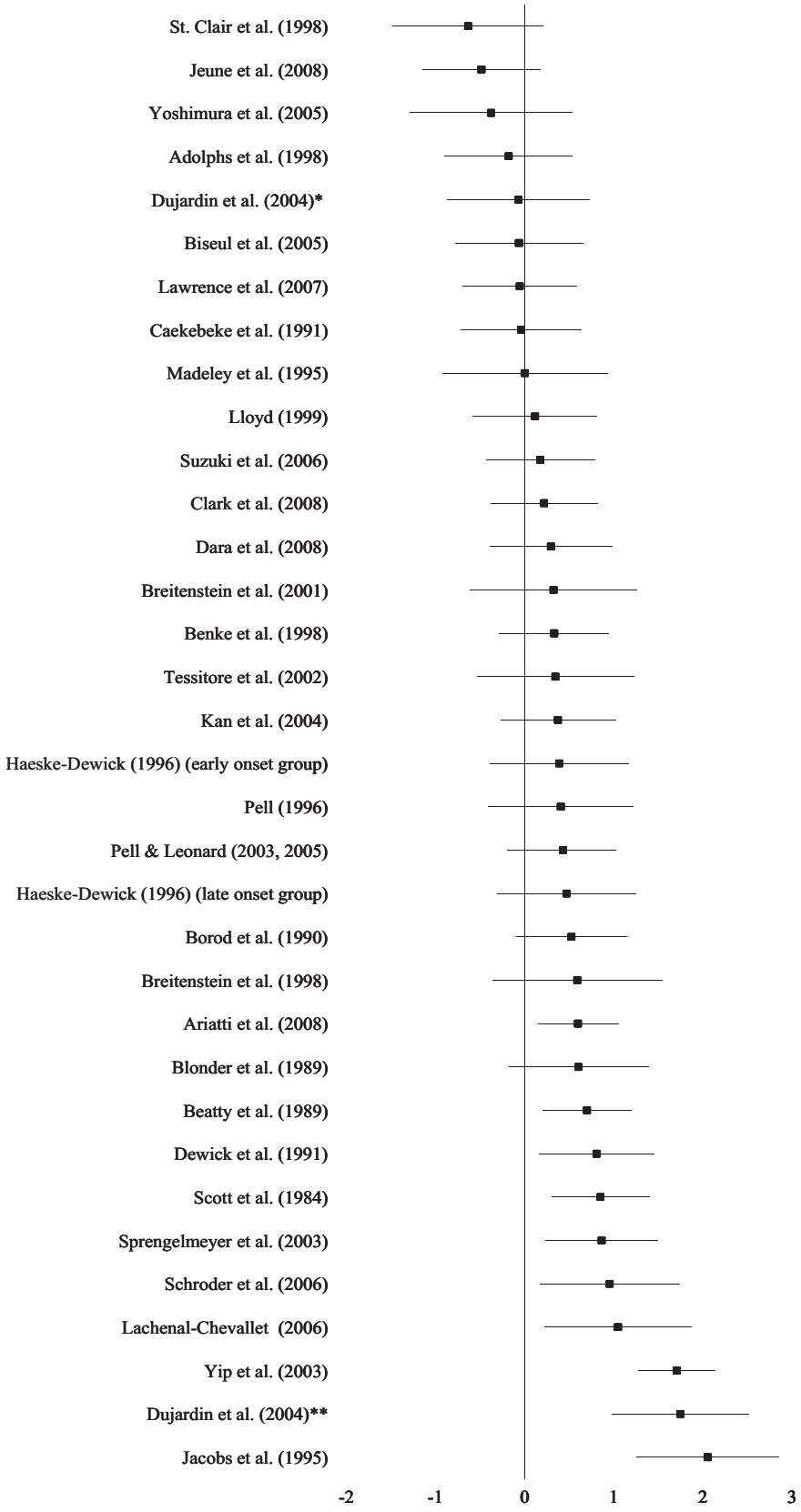

Figure 1 (opposite).

Table 1
Summary of Effect Sizes for Emotion Recognition Across Stimulus Modalities

Test	*g*	95% CI	*Z*	Q_W	*k*	*N*
Fixed effects	.52	.40, .63	8.69***	99.62***	34	1,295
Random effects	.46	.26, .65	4.52***	33.33	34	1,295

Note. g = weighted mean effect size g; Z = test of the null hypothesis that the mean effect size is zero; Q_W = within-class test of homogeneity; k = number of independent comparisons.
*** $p < .001$.

five reported a deficit among the PD group. We computed and compared separate emotion recognition effect size estimates in these subgroups. As indicated in Table 8, the aggregated effect size g from studies showing normal performance on a control task ($g = 0.39$) was not significantly different from that derived from studies showing abnormal performance ($g = 0.58$), $Q_B(1) = 1.22$, *ns*. This suggests that excluding data provided by participants who show abnormal control task performance would not impact the overall results in a meaningful way.

File Drawer Analysis

We conducted a file drawer analysis to determine how many unpublished studies with effect sizes of zero would be required to render the obtained effects nonsignificant. This analysis is based on effect sizes for each of the 34 independent comparisons used in the primary analysis (i.e., pooled across stimulus modality, task type, emotion, and participant characteristics). Using the formula provided by Rosenthal (1979), one finds that 1,073 studies averaging null results are required to render the results of the meta-analysis nonsignificant. In other words, to make the present results nonsignificant would require roughly 32 times more null comparisons than the number used in the current analysis. We can compare this with a conservative tolerance level ($5k + 10$), the value of which indicates the minimum acceptable number of "file drawer studies," with k equal to the number of comparisons used in the meta-analysis. Because the file drawer statistic (1,073) exceeds the tolerance level (195), we conclude that it is unlikely that there are enough unretrieved or unpublished studies in existence to render the results of the meta-analysis nonsignificant.

Discussion

We found a robust link between PD and impaired recognition of emotion from faces and voices. Relative to matched control groups, individuals with PD showed significant deficits in the ability to recognize the emotion portrayed in facial and prosodic stimuli. The overall impairment effect size g of 0.52 corresponds to an r of 0.26. This means that theoretically, if half the population had PD and half did not, those who did not would have a 37% chance of having impaired emotion recognition abilities, compared with a 63% chance for those with PD (Rosenthal & Rosnow, 1991).

Across studies, the level of emotion recognition deficit does not appear to be related to the level of motor disability, as the correlation across studies between participants' average reported Hoehn and Yahr (1967) staging and deficit effect size g was $r(32) = -0.07$, *ns*. This suggests that motor disability and the deficit in emotion recognition may result from different forms of brain pathology. However, the average PD patient included in this meta-analysis exhibited mild to moderate bilateral motor disability, and a different pattern may have emerged if more severely affected patients had been the focus of investigation. Although not specifically investigated here, there is no reason to expect that the level of emotion recognition deficit is related to the predominant side of motor signs and symptoms (St. Clair, Borod, Sliwinski, Cote, & Stern, 1998).

Although beyond the scope of this meta-analysis, the likely cause of this deficit is pathology in neural circuits involved in emotion recognition, particularly within basal ganglia structures including the ventral striatum and STN. Three reports included in this meta-analysis are particularly relevant to this question because they include data from patients following STN deep-brain stimulation (DBS). Inactivation of the STN through DBS helps reverse parkinsonian symptoms but produces emotional and cognitive deficits (for a review, see Biseul et al., 2005). In each study of emotion recognition accuracy following STN DBS, there were larger deficits postsurgery (when the STN was inactivated) than presurgery (Biseul et al., 2005; Dujardin, Blairy, Defebvre, Krystkowiak, et al., 2004; Le Jeune et al., 2008). These results support the conclusion that basal ganglia circuits, particularly the STN, play a role in emotion recognition. Some (Biseul et al., 2005; Le Jeune et al., 2008) have suggested that impairments in emotion recognition following STN DBS result from altered projections to cortical areas, particularly the orbitofrontal cortex (OFC), which has already been implicated in emotion recognition (Adolphs, 2002). Indeed, Le Jeune et al. (2008) observed a positive correla-

Figure 1 (opposite). Forest plot of standardized mean difference in emotion recognition accuracy scores for participants in the control group minus participants with Parkinson's disease (PD). Horizontal lines are 95% confidence intervals for individual studies and boxes are centered on individual study estimates. A positive score means participants with PD performed worse. Scores have been pooled across participant groups when appropriate and across multiple-dependent measures (defined by stimulus modality, task type, and emotion expressed). * Dujardin, Blairy, Defebvre, Krystkowiak, et al. (2004). ** Dujardin, Blairy, Defebvre, Duhem, et al. (2004).

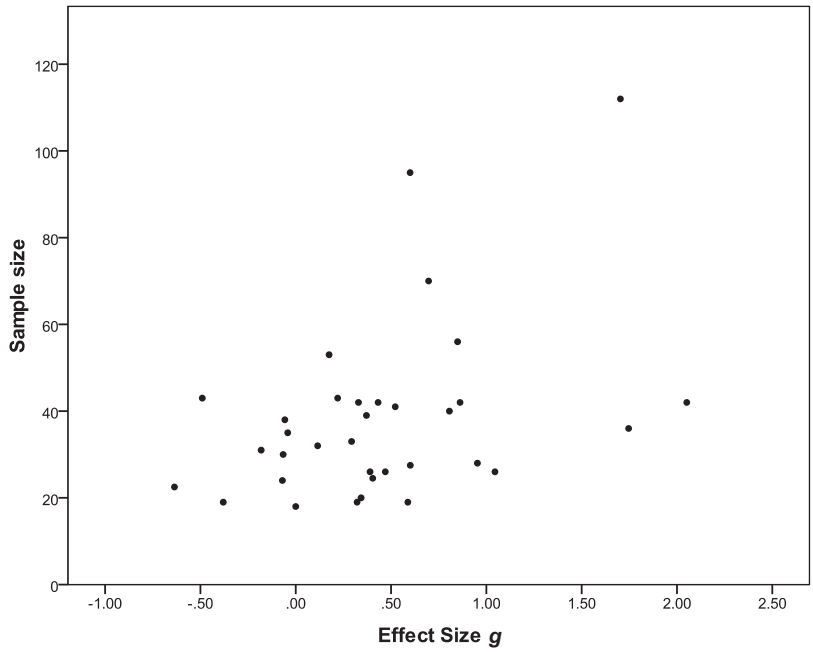

Figure 2. Scatter plot of the mean weighted effect sizes (*g*) in relation to total sample size (control plus PD).

tion between decreased glucose metabolism, mainly in the right OFC, and impaired recognition of fearful faces among PD patients who had undergone STN DBS 3 months earlier. More broadly, this result adds to a growing literature on negative cognitive and emotional changes following STN DBS (Temel et al., 2006).

It is possible that the emotion recognition deficit results from pathology in other brain areas. For instance, Lewy bodies are present in well over 90% of patients diagnosed with PD (Hughes, Daniel, Ben-Shlomo, & Lees, 2002), and Lewy body disease is the main neuropathological substrate of dementia in PD (Aarsland, Perry, Brown, Larsen, & Ballard, 2005). Lewy body disease is associated with abnormal function of visual cortical areas (Mosimann et al., 2004). Could Lewy body disease therefore partially account for the observed deficits in facial emotion recognition? No research, to our knowledge, has linked Lewy body disease specifically with face processing. It is true that Capgras delusions, the belief that imposters have replaced family members or friends, are characteristic of Lewy body dementia (Harciarek & Kertesz, 2008), and Capgras delusions appear to result from an impaired ability to draw affective meaning from perceived faces (Baldwin, Snowden, & Mann, 1995; Ellis, Lewis, Moselhy, & Young, 2000). However, as mentioned previously, about 80% of the studies included in this meta-analysis excluded patients with dementia. It therefore seems likely that the majority of patients in our sample did not have substantial Lewy body disease at the time of assessment, although those who later progressed to dementia associated with Parkinson's disease (PD–D) might have experienced this form of neuropathology.

There may be another, less direct, pathway between PD and facial emotion recognition deficits. The "reverse simulation" account of emotion recognition (Goldman & Sripada, 2005) begins with the observation that in everyday life, a perceiver will spontaneously

Table 2
Summary of Effect Sizes for Emotion Recognition as a Function of Stimulus Modality

Test	*g*	95% CI	*Z*	Q_W	*k*	*N*	Q_B
Fixed-effects model							
Facial displays	.48	.35, .60	7.47***	79.57***	28	1,110	
Prosodic displays	.70	.54, .87	8.21***	77.22***	15	635	
Between-classes effect							3.87*
Random-effects model							
Facial displays	.41	.19, .64	3.58***	26.36	28	1,110	
Prosodic displays	.63	.38, .88	4.85***	25.06	15	635	
Between-classes effect							1.46

Note. g = weighted mean effect size g; Z = test of the null hypothesis that the mean effect size is zero; Q_W = within-class test of homogeneity; k = number of independent comparisons; Q_B = between-class test of homogeneity.
* $p < .05$. *** $p < .001$.

Table 3
Summary of Effect Sizes for Emotion Recognition as a Function of Task Type, Facial Displays Only

Test	*g*	95% CI	*Z*	Q_W	*k*	*N*	Q_B
Fixed-effects model							
Identification tasks	.50	.37, .63	7.50***	67.86***	23	1,044	
Discrimination tasks	.62	.44, .80	6.69***	32.91**	14	528	
Rating tasks	.05	–.32, .42	0.27	1.34	3	126	
Between-classes effect							9.32**
Random-effects model							
Identification tasks	.43	.20, .67	3.64***	21.64	23	1,044	
Discrimination tasks	.54	.26, .83	3.72**	15.39	14	528	
Rating tasks	.05	–.32, .42	0.27	1.34	3	126	
Between-classes effect							9.38**

Note. *g* = weighted mean effect size *g*; *Z* = test of the null hypothesis that the mean effect size is zero; Q_W = within-class test of homogeneity; *k* = number of independent comparisons; Q_B = between-class test of homogeneity.
** $p < .01$. *** $p < .001$.

mimic his interaction partner's emotional expressions, albeit in an attenuated and largely covert manner (Dimberg, Thunberg, & Elmehed, 2000). Through a process of "facial feedback," the muscular activity induced by mimicry produces a corresponding emotional state in the perceiver (Adelmann & Zajonc, 1989), so that both perceiver and partner experience the same emotional state (Hatfield, Cacioppo, & Rapson, 1993). Finally, the induction of an emotional state in the perceiver serves as input in the classification of the partner's emotional state (Stel & van Knippenberg, 2008). However, anything that prevents the production of muscular activity in the perceiver inhibits emotional contagion and emotion recognition (Stel, Van Baaren, & Vonk, 2008; Stel & van Knippenberg, 2008). Therefore, individuals with PD may experience deficits in emotion recognition, at least in part, because they have a reduced ability to spontaneously mimic displays of emotion (Smith, Smith, & Ellgring, 1996). Preliminary support for this interpretation comes from correlations, among individuals with PD, between the ability to recognize emotions and the ability to produce emotional expressions (Benke, Bosch, & Andree, 1998; Borod, Welkowitz, Alpert, & Brozgold, 1990; Jacobs, Shuren, Bowers, & Heilman, 1995). Across these three studies, the average correlation *r* between emotion recognition and emotion production was 0.47. Lawrence and colleagues (2007) suggested that evidence of intact emotion recognition abilities among people who have congenital facial paralysis (Calder, Keane, Cole, Campbell, & Young, 2000) is inconsistent with this explanation. However, it is possible that people who have never been able to produce facial displays of emotion may develop the ability to recognize them using compensatory strategies that draw on different neural networks (Bolte et al., 2006). In any case, further work is necessary to determine whether the ability to produce emotional expressions is a necessary prerequisite to the ability to recognize emotions, or if both processes simply share a common neural substrate that is damaged in PD (Jacobs et al., 1995).

Stimulus Modality

Our data indicate that the deficit in emotion recognition in PD is cross modal, in that it is apparent in the recognition of emotion from both faces and voices. The cross-modal nature of the impairment provides support for the notion that in PD, basal ganglia pathology produces a decline in emotion recognition independent of stimulus

Table 4
Summary of Effect Sizes for Emotion Recognition as a Function of Task Type, Prosodic Displays Only

Test	*g*	95% CI	*Z*	Q_W	*k*	*N*	Q_B
Fixed-effects model							
Identification tasks	.88	0.70, 1.05	9.71***	105.83***	14	600	
Discrimination tasks	.61	0.40, 0.82	5.71***	30.95***	8	397	
Rating tasks	.31				1	42	
Between-classes effect							3.70†
Random-effects model							
Identification tasks	.74	0.40, 1.08	4.26***	19.66	14	600	
Discrimination tasks	.58	0.25, 0.92	3.44**	9.18	8	397	
Rating tasks	.31				1	42	
Between-classes effect							0.01

Note. *g* = weighted mean effect size *g*; *Z* = test of the null hypothesis that the mean effect size is zero; Q_W = within-class test of homogeneity; *k* = number of independent comparisons; Q_B = between-class test of homogeneity (comparing identification and discrimination tasks only).
† $p < .10$. ** $p < .01$. *** $p < .001$.

Table 5
Summary of Effect Sizes for Emotion Recognition as a Function of Emotion Expressed

Test	*g*	95% CI	*Z*	Q_W	*k*	*N*	Q_B
Fixed-effects model							
Anger recognition	.32	.16, .47	3.96***	41.23	17	699	
Disgust recognition	.30	.14, .47	3.59***	52.59	15	633	
Fear recognition	.29	.10, .48	2.97**	46.20	11	489	
Happiness recognition	.20	.04, .35	2.41*	22.63	16	667	
Sadness recognition	.27	.12, .43	3.41***	33.15	17	701	
Surprise recognition	.12	–.07, .31	1.24	9.77	11	452	
Between-classes effect							0.36
Random-effects model							
Anger recognition	.33	.06, .60	2.37*	16.25	17	699	
Disgust recognition	.39	–.04, .82	1.80	11.38	15	633	
Fear recognition	.17	–.27, .61	0.77	7.77	11	489	
Happiness recognition	.16	–.04, .35	1.53	9.34	16	667	
Sadness recognition	.28	.04, .52	2.28*	13.37	17	701	
Surprise recognition	.12	–.08, .32	1.18	9.74	11	452	
Between-classes effect							0.53

Note. g = weighted mean effect size g; Z = test of the null hypothesis that the mean effect size is zero; Q_W = within-class test of homogeneity; k = number of independent comparisons; Q_B = between-class test of homogeneity.
* $p < .05$. ** $p < .01$. *** $p < .001$.

modality. Clinically, this finding indicates that individuals with PD may have difficultly compensating for their trouble discerning the emotional message in a partner's face by focusing on the tone of voice, or vice versa. Although the deficit appears to be cross model, it is larger for the recognition of emotion from prosody (effect size $g = 0.70$) than from facial displays (effect size $g = 0.49$). Indeed, there has been more consensus in the literature regarding the presence of a deficit in recognizing prosodic cues to emotion, as compared to facial cues to emotion (for a review, see Pell & Leonard, 2003). There are at least three potential explanations for this finding. First, as discussed in detail below, prosodic emotion recognition might be more susceptible to the limitations in working memory capacity often noted in PD. Second, the basal ganglia might play a more substantial role in prosodic emotion recognition, as has been suggested (Adolphs, Damasio, Tranel, & Damasio, 1996; Pell & Leonard, 2003). Finally, this finding might result from the fact it is typically more difficult to infer emotion from prosodic displays than from facial displays (Scherer, 2003), and thus tests of prosodic recognition may yield more variance for detecting group differences. Future research that more carefully controls for task difficultly would be in a position to rule out this alternative possibility.

Task Type

Our results revealed a novel pattern with regard to task type. For both facial displays and prosody, identification and discrimination

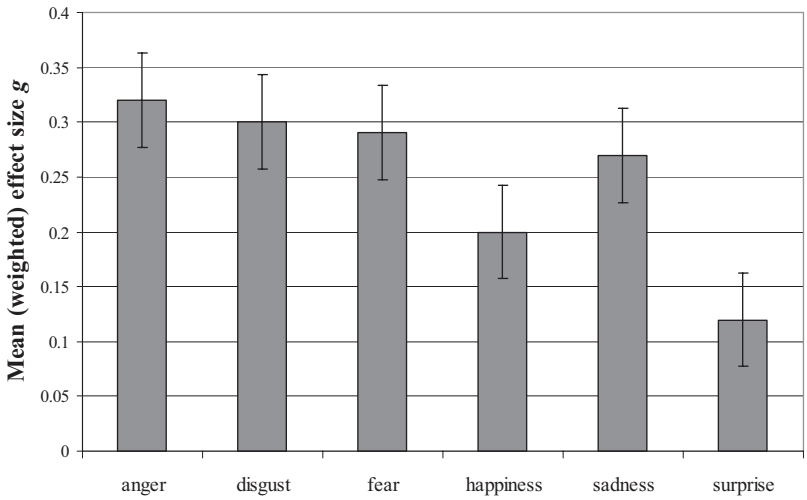

Figure 3. Mean deficit (weighted effect size g) as a function of emotion expressed, across both stimulus modalities (derived from fixed-effects model). Error bars show standard error of the estimate. More positive effect sizes indicate greater differences favoring controls over PD participants.

Table 6
Summary of Effect Sizes for Emotion Recognition as a Function of Participants' Medication Status

Test	*g*	95% CI	*Z*	Q_W	*k*	*N*	Q_B
Fixed-effects model							
Hypodopaminergic state	.50	0.21, 0.80	3.32***	20.98	6	207	
Dopamine-replete state	.27	0.10, 0.44	3.08**	11.62	16	546	
Between-classes effect							1.73
Random-effects model							
Hypodopaminergic state	.49	–0.14, 1.11	1.52	4.98	6	207	
Dopamine-replete state	.27	0.11, 0.43	3.28**	12.95	16	546	
Between-classes effect							0.44

Note. *g* = weighted mean effect size *g*; *Z* = test of the null hypothesis that the mean effect size is zero; Q_W = within-class test of homogeneity; *k* = number of independent comparisons; Q_B = between-class test of homogeneity.
** $p < .01$. *** $p < .001$.

tasks produced greater deficits than rating tasks. However, in making sense of these findings, it is important to keep in mind that the effect size estimates for rating tasks were based on very few studies and may have been skewed by the relatively small effect sizes reported in two studies that used the same procedures (Adolphs et al., 1998; Pell & Leonard, 2005). Suzuki and colleagues (2006) reported a substantially larger effect size using a refined rating procedure that may have been less susceptible to difficulty artifacts (i.e., being unable to detect a difference because of ceiling effects). We believe that the present data do not justify the conclusion that rating tasks, in general, reveal smaller deficits in emotion recognition than identification or discrimination tasks. Regarding identification and discrimination tasks only, we found opposite patterns for facial displays and prosody. Discrimination tasks yielded a significantly greater deficit in facial emotion recognition, but identification tasks yielded a significantly greater deficit in prosodic emotion recognition. We have, at present, no explanation for this pattern and suggest the need for future research to resolve it.

Emotion

Individuals with PD were more impaired in recognizing negative emotions (anger, disgust, fear, and sadness) than relatively positive emotions (happiness, surprise). For at least two reasons, it is unlikely that this finding results simply from artifacts reflecting different difficulty levels across emotions. First, although it is true that negative emotions (particularly fear) are generally more difficult to interpret than positive emotions (for a review, see Suzuki et al., 2006), this pattern only holds true for facial expressions. Positive emotions are very difficult to detect from the voice (Johnstone & Scherer, 2000; Scherer, 2003). Second, Suzuki and colleagues (2006) used item response theory to control for difficulty and still demonstrated a selective impairment in the recognition of one negative emotion (disgust) from facial displays. Instead, as many have suggested, individuals with PD may be particularly impaired in recognizing negative emotions (from both the face and the voice) because of dysfunction in specific neural circuits. Evidence in support of this explanation is strongest for three of the four negative emotions: anger, disgust, and fear. Lawrence et al. (2007) reviewed evidence suggesting a central role for ventral striatal dopamine systems in anger recognition and suggested that dysfunction in these systems explains their finding of a selective impairment in anger recognition during acute withdrawal from DRT (see also Dujardin, Blairy, Defebvre, Krystkowiak, et al., 2004). With regard to disgust recognition, both neuropsychology and neuroimaging support the notion that the basal ganglia and the

Table 7
Summary of Effect Sizes for Emotion Recognition as a Function of Group Differences in Depression Status

Test	*g*	95% CI	*Z*	Q_W	*k*	*N*	Q_B
Fixed-effects model							
Groups equally depressed	.49	.20, .78	3.28**	4.54	7	203	
PD group more depressed	.49	.28, .70	4.61***	19.5*	10	384	
Between-classes effect							0.00
Random-effects model							
Groups equally depressed	.48	.26, .70	4.37***	8.28	7	203	
PD group more depressed	.48	.12, .83	2.59**	8.10	10	384	
Between-classes effect							0.00

Note. *g* = weighted mean effect size *g*; *Z* = test of the null hypothesis that the mean effect size is zero; Q_W = within-class test of homogeneity; *k* = number of independent comparisons; Q_B = between-class test of homogeneity; PD = Parkinson disease.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 8
Summary of Effect Sizes for Emotion Recognition as a Function of Control Task Performance

Test	g	95% CI	Z	Q_W	k	N	Q_B
Fixed-effects model							
Normal control task performance	.39	.22, .56	4.48***	41.33***	15	606	
Abnormal control task performance	.58	.29, .87	3.89***	1.56	5	195	
Between-classes effect							1.22
Random-effects model							
Normal control task performance	.39	.09, .70	2.51*	13.91	15	606	
Abnormal control task performance	–.08	–.59, .43	–0.31	–3.46	5	195	
Between-classes effect							2.42

Note. g = weighted mean effect size g; Z = test of the null hypothesis that the mean effect size is zero; Q_W = within-class test of homogeneity; k = number of independent comparisons; Q_B = between-class test of homogeneity.
* $p < .05$. *** $p < .001$.

insula are involved in the cross-modal recognition of disgust, and both regions are dysfunctional in PD (for a review, see Suzuki et al., 2006). Supportive evidence comes from other studies demonstrating disproportionate impairments in disgust recognition (Kan et al., 2004; Lachenal-Chevallet et al., 2006; Sprengelmeyer et al., 2003). Disproportionate impairments in the recognition of fear have been reported in several papers (Ariatti et al., 2008; Kan et al., 2004; Lachenal-Chevallet et al., 2006; Sprengelmeyer et al., 2003). There is wide agreement that amygdala are involved in the cross-modal recognition of fear (e.g., Adolphs, Russell, & Tranel, 1999; see, e.g., Calder et al., 1996), and there is evidence that the amygdala is affected in PD (Bouchard et al., 2008; Tessitore et al., 2002). Finally, the left amygdala also appears to play a role in the recognition of sadness (Blair, Morris, Frith, Perrett, & Dolan, 1999), so pathology in that region in PD may help to explain abnormal processing of sad stimuli, when it has been found (Schroder et al., 2006). In sum, our review of past work provides support for making a broad valence-based distinction (negative vs. positive) with regard to the specificity of emotion recognition impairments in PD (Adolphs et al., 1999), likely because the areas that subserve the recognition of these emotions are most affected in PD. An important caveat, however, is that our analyses were based on the subset of studies that reported accuracy percentages for specific emotions.

Medication Status

The same caveat applies to our analysis of the role of medication status. We were able to identify relatively few studies that definitively noted whether patients were in a state of optimal medication during assessment, yielding a total of 22 comparisons (16 in a relatively dopamine-replete state and six in a hypodopaminergic state). Although we did find that the impairment effect size was larger among patients who were hypodopaminergic state at the time of testing—consistent with the purported role of dopamine in emotion recognition—the difference in effect sizes was not significant. This may be because some patients included in the relatively dopamine-replete group had such advanced disease that their dopamine levels even when optimally medicated were not dissimilar from patients who were in withdrawal from DRT or who had not yet begun DRT. On a related note, there was a good deal of heterogeneity among our group of patients in a hypodopaminergic state, with some not yet receiving medication because they were recently diagnosed grouped with others who were in withdrawal from normal DRT therapy. This heterogeneity may have obscured true group differences. In any case, it is interesting to note that we obtained a sizable emotion recognition deficit even though nearly all patients included in these studies were generally receiving medication.

Depression

Because of the high incidence of depression in PD, and because depression itself is associated with emotion recognition deficits, it is important to investigate whether any emotion recognition deficit in PD is secondary to depression. Two of our findings speak against this possibility. First, when we directly compared the extent of emotion recognition deficit among relatively depressed individuals with PD against those who were no more depressed than controls, we found no difference. This suggests that the emotion recognition deficit would remain unchanged even if we were to disregard all of the studies that included relatively more depressed PD patients. Second, the average within-study correlation between emotion recognition accuracy and scores on depression inventories was quite small ($r = .07$). These results suggest that the emotion recognition deficit in PD arises independently of depression. It may be that the co-occurrence of depression and PD results from a common neurochemical mechanism, as others have suggested (Frisina, Haroutunian, & Libow, 2009; Koerts, Leenders, Koning, Bouma, & van Beilen, 2008).

Role of Executive Function and Visuospatial Deficits

We examined the possibility that facial emotion recognition deficits in PD are secondary to more general deficits in visuospatial ability, which have been reported even in the early stages of the disease (Levin et al., 1991). Five datasets included in this meta-analysis included a PD group that performed significantly worse than the matched control group on a facial feature discrimination test (Beatty et al., 1989; Dara et al., 2008; Dewick, Hanley, Davies, Playfer, & Turnbull, 1991; two comparisons provided by Haeske-Dewick, 1996). By contrast, 15 datasets included a PD group that did not perform significantly worse than the matched control group. Moreover, the effect sizes derived from these two types of

comparisons were not significantly different from each other. These results suggest that the facial emotion recognition deficit in PD exists beyond a general deficit in face processing.

The situation is less clear with regard to executive function. A smaller subset of studies ($n = 8$) reported the results of at least one test of executive function. In this case, the bulk of the studies ($n = 6$) reported a significant group difference favoring those in the control group. For instance, PD participants in studies by Dujardin and colleagues (Dujardin, Blairy, Defebvre, Duhem, et al., 2004; Dujardin, Blairy, Defebvre, Krystkowiak, et al., 2004) showed a cognitive deficit pattern the authors labeled "moderate dysexecutive syndrome." More specifically, PD participants in studies reported by Breitenstein et al. (2001); Pell and Leonard (2003); and Dara et al. (2008) all performed significantly worse than controls on listening span tests, which tap verbal working memory. Working memory constraints appears to be impactful with regard to prosodic emotion recognition. Both Pell and Leonard and Dara et al. observed substantial correlations among PD participants between working memory capabilities and the ability to decode emotional prosody (rs = .51 and .45, respectively). Working memory deficits among individuals with PD have been linked with altered dopaminergic innervations to the dorsolateral prefrontal cortex and progressive compromise to the frontal–striatal pathways (Monetta & Pell, 2007). The observed relationship between prosodic emotion recognition and working memory makes sense in light of the fact that prosodic emotion recognition places relatively heavy demands on working memory capabilities. In typical facial emotion recognition tasks, faces remain on the screen while participants generate a response. By contrast, in typical prosodic emotion recognition tasks, participants must hold stimuli in working memory while attending to other task requirements (e.g., visually attending to verbal labels or other response prompts). Given these findings, it seems plausible that deficits in prosodic emotion recognition in PD are partially dependent on working memory constraints.

Clinical Significance

This robust deficit in emotion recognition has clinical significance. Interpersonal difficulties are common in PD and, in many cases, even more detrimental to quality of life than physical symptoms (Schreurs, De Ridder, & Bensing, 2000). There are several potential sources of interpersonal difficulty in PD, including the common tendency for perceivers, even those with advanced medical training, to mistake the symptoms of PD as indicators of negative personality traits (Pentland, Gray, Riddle, & Pitcairn, 1988; Tickle-Degnen & Lyons, 2004). The current results suggest that an additional source of interpersonal difficultly in PD may be the reduced ability to develop finely tuned appraisals of interaction partners' feelings and intentions. Indeed, the extent of emotion recognition deficit in PD appears to be correlated with a variety of interpersonal difficulties, such as complaints of frustration in social relations, feelings of social disconnection, and a desire to connect with others (Clark et al., 2008). Although the direction of this relationship has not been defined, it is likely that reduced emotion recognition abilities contribute to social stress. Social stress, in turn, accelerates the progression of age-related diseases (Hawkley & Cacioppo, 2004; Kiecolt-Glaser et al., 2005). One way to intervene in this potential vicious cycle is to provide individuals with PD feedback training in emotion recognition, which shows promise in elevating accuracy rates (Bolte et al., 2006; Elfenbein, 2006). Of course, the first step is to educate PD patients and their close associates about the potential for emotion recognition difficulties and associated consequences.

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