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Impaired Perception of Biological Motion in Parkinson's Disease

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Abstract

Objective—We examined biological motion perception in Parkinson's disease (PD). Biological motion perception is related to one's own motor function and depends on the integrity of brain areas affected in PD, including posterior superior temporal sulcus. If deficits in biological motion perception exist, they may be specific to perceiving natural/fast walking patterns that individuals with PD can no longer perform, and may correlate with disease-related motor dysfunction.

Method—26 non-demented individuals with PD and 24 control participants viewed videos of point-light walkers and scrambled versions that served as foils, and indicated whether each video depicted a human walking. Point-light walkers varied by gait type (natural, parkinsonian) and speed (0.5, 1.0, 1.5 m/s). Participants also completed control tasks (object motion, coherent motion perception), a contrast sensitivity assessment, and a walking assessment.

Results—The PD group demonstrated significantly less sensitivity to biological motion than the control group ($p<.001$, Cohen's $d=1.22$), regardless of stimulus gait type or speed, with a less substantial deficit in object motion perception ($p=.02$, Cohen's $d=.68$). There was no group difference in coherent motion perception. Although individuals with PD had slower walking speed and shorter stride length than control participants, gait parameters did not correlate with biological motion perception. Contrast sensitivity and coherent motion perception also did not correlate with biological motion perception.

Conclusion—PD leads to a deficit in perceiving biological motion, which is independent of gait dysfunction and low-level vision changes, and may therefore arise from difficulty perceptually integrating form and motion cues in posterior superior temporal sulcus.

Keywords

biological motion; visual perception; perception-action coupling; gait analysis; neurodegenerative diseases

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Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disease that affects visual perception and the ability to carry out vision-based activities of daily living (Davidsdottir, Cronin-Golomb, & Lee, 2005; Seichepine et al., 2011). With respect to basic motion perception, which depends on regions such as the middle temporal area (MT) and the temporo-parieto-occipital junction (Andersen, 1997; Grossman et al., 2000; Sunaert, Van Hecke, Marchal, & Orban, 1999), some studies have found deficits in PD relative to control participants (Trick, Kaskie, & Steinman, 1994), whereas others have found no difference (Amick, Cronin-Golomb, & Gilmore, 2003). A more consistent observation is that PD impairs higher-level motion perception, such as the perception of motion-defined objects including squares and spheres (Uc et al., 2005), motion-defined surfaces (Castelo-Branco et al., 2009), and second-order motion (Ezzati, Khadjevand, Zandvakili, & Abbassian, 2010). These findings suggest PD-related alterations in extrastriate cortex that mediates visual motion perception (Putcha et al., 2014).

One form of motion perception that has not been studied in PD to date is the perception of human movement, or biological motion. Biological motion perception is an important cue to others' actions, identity, personality, communicative intent, and emotional state (Atkinson, Dittrich, Gemmell, & Young, 2004; Clarke, Bradshaw, Field, Hampson, & Rose, 2005; Cutting & Kozlowski, 1977; Dittrich, 1993; Heberlein, Adolphs, Tranel, & Damasio, 2004). Even when visual motion cues are isolated to the motion of a set of point-lights on the major joints of the body (known as point-light walkers or PLWs), observers readily perceive a human. Observers robustly perceive biological motion even when PLW stimuli are presented for very brief durations, and when obscured by a mask (Blake & Shiffrar, 2007; Chang & Troje, 2009; Cutting, Moore, & Morrison, 1988; Johansson, 1973).

Sensitivity to biological motion may be reduced in PD because of disease-related motor deficits. There is strong evidence that action perception and action production are intricately connected ("perception-action coupling") and that the motor system partially mediates action perception by simulating or embodying observed actions (Gallese, Gernsbacher, Heyes, Hickok, & Iacoboni, 2011; Iacoboni et al., 2005; Shiffrar, 2011). Activity in the ventral premotor cortex, part of the mirror neuron network that activates both when perceiving and producing actions, predicts individual performance on biological motion tasks (Gilaie-Dotan, Kanai, Bahrami, Rees, & Saygin, 2013). Clinical disorders that affect motor function including Asperger's syndrome, paraplegia, and hemiplegia have all been associated with reduced sensitivity to biological motion (Arrighi, Cartocci, & Burr, 2011; Price, Shiffrar, & Kerns, 2012; Serino et al., 2009). These findings suggest that in PD, motor dysfunction may affect visual perception (e.g., Dayan, Inzelberg, & Flash, 2012). There may be specific difficulty in perceiving biological motion that individuals with the disease can no longer perform (e.g., fast, healthy walking patterns) with relatively spared perception of motion that is similar to their own motor ability (e.g., slow, shuffling walking patterns). While previous studies have found associations between motor function and biological motion perception in other clinical populations (e.g., between unsteadiness and perception of PLW stimuli in Asperger's syndrome; Price et al., 2012), the present study is the first to

specifically examine the association between gait characteristics and perception of walking from biological motion.

A second reason that biological motion perception may be impaired in PD is because of alterations in basic, low-level visual abilities such as contrast sensitivity and coherent motion perception. Reduced contrast sensitivity, which is a common finding in PD (Bodis-Wollner et al., 1987), would presumably make biological motion cues less salient and more difficult to perceive. Alternatively or additionally, a deficit in biological motion perception could be explained by reduced sensitivity to low-level coherent motion, or by reduced sensitivity to motion-defined forms more generally. The contribution of coherent motion seems less likely given mixed findings in PD, whereas difficulty perceiving motion-defined forms is more consistently observed and may therefore play a role in biological motion perception.

A third possibility is that deficient biological motion perception in PD may result directly from changes specifically to the posterior superior temporal sulcus (STS), which is a polysensory region that integrates motion and form cues and is consistently associated with biological motion perception in healthy observers (Carter, Hodgins, & Rakison, 2011; Grossman, Battelli, & Pascual-Leone, 2005). This possibility was raised by a study that found that reduced perception of emotional gestures in PD was associated with decreased functional activity in the STS (Lotze et al., 2008). There is also evidence for a visual corticostriatal loop connecting the tail of the caudate to extrastriate visual cortex, including posterior STS (Seger, 2013), which may be dysfunctional in PD. If biological motion perception is impaired in PD, but the deficit is independent of changes in gait and in basic vision/motion perception, then extrastriate regions such as the STS may be the more likely source of the dysfunction.

The STS is part of a distributed network of brain regions associated with social perception. Deficits in social perception are well documented in PD, including difficulties in the perception of facial emotions, theory of mind, and processing of verbal and nonverbal social cues (Buxton, MacDonald, & Tippett, 2013; Clark, Neargarder, & Cronin-Golomb, 2008, 2010; Freedman & Stuss, 2011; Pell et al., 2014). There is evidence that sensitivity to biological motion is associated with social cognition (Miller & Saygin, 2013), suggesting that a possible biological motion perception impairment may be part of a broader impairment in social perception and cognition in PD.

The goals and hypotheses of the present study are as follows. The first goal was to determine whether non-demented individuals with PD are impaired in perceiving biological motion. We predicted that the PD group would perform more poorly on a biological motion recognition task than would control participants, but that performance would be relatively intact on tasks of object motion and coherent motion perception. The second goal was to determine correlates of biological motion perception in PD. In line with evidence of perception-action coupling, we hypothesized that individuals with PD would be specifically impaired at perceiving biological motion outside their motor repertoire (i.e., fast, natural walking patterns), and that objectively measured gait characteristics in PD would correlate with performance on the biological motion perception task. We predicted that the

hypothesized biological motion perception deficit in PD would be independent of contrast sensitivity and coherent motion perception.

Method

Participants

The study included 26 non-demented individuals with PD and 24 healthy control participants. The sample size was determined by a power analysis with power = 80%, a medium effect size (0.30), and alpha = .05. Participants (PD and control) were recruited through the Parkinson's Disease and Movement Disorders Clinic at Boston Medical Center, Boston University's Sargent College of Health and Rehabilitation Sciences, the Michael J. Fox Foundation Trial Finder, and other community sources. PD and control participants were matched for age, education, and male:female ratio. All were native speakers of English or completed high school in English, had at least 12 years of education, and lived at home rather than in an institution. Individuals with PD met clinical criteria for mild to moderate idiopathic PD (Hoehn & Yahr Stage 1-3). Motor disability was quantified using the Unified Parkinson's Disease Rating Scale motor subscale. All but one individual with PD were taking medications for their motor symptoms and were in the "on" state during testing. Levodopa equivalent dosage (LED; mg/day) was calculated for each individual with PD using a standardized formula (Tomlinson et al., 2010). Table 1 displays demographic and clinical characteristics of the PD group and control group.

Exclusion criteria included serious chronic medical, neurological (other than PD), or psychiatric illness; mental retardation; history of intracranial surgery; history of traumatic brain injury with loss of consciousness greater than a few seconds; current or previous substance or alcohol abuse; and diagnosis of eye disease such as significant current macular degeneration, cataract, or glaucoma. Use of antidepressants and anxiolytics was permitted in the PD group only, because of the frequent use of these medications in this population. Use of such medications in the control group, or other psychoactive medications in either group, was grounds for exclusion.

Procedure

All procedures were approved by the Boston University Institutional Review Board in accordance with the Declaration of Helsinki. Participants were explained the study procedures prior to participating and signed an informed consent form.

Screening Measures and Questionnaires—Dementia was screened by using the Columbia-Modified Mini Mental State Examination and all participants scored above 27/30. Individuals with PD and control participants were administered the Geriatric Depression Scale and the Beck Anxiety Inventory to assess symptoms of depression and anxiety, respectively.

Basic Vision and Motion Perception Assessment—Visual acuity was assessed using the Lighthouse Near Visual Acuity Test at a distance of 16 inches (40 cm). Participants had corrected binocular visual acuity equal to or greater than 20/40 (logarithm mean angle

of resolution, log mean angle of resolution ≤ 0.3). Contrast sensitivity was determined using the Functional Acuity Contrast Test at a distance of 16 inches (40 cm). This test provides contrast sensitivity values for five spatial frequencies (1.5, 3, 6, 12, and 18 cycles per degree). These values were used in correlation analyses to determine the relation between contrast sensitivity and biological motion perception.

To assess basic, low-level coherent motion perception, a random-dot kinematogram task was used. Stimuli were created in which a subset of dots moved either leftward or rightward (signal), while additional dots moved in random directions across the screen (noise). The following signal to noise ratios were used: 3, 6, 12, 24, 48 and 96%. For each signal-to-noise ratio, eight trials were presented in which the dots moved directly left and eight in which they moved directly right, for a total of 96 trials. Each stimulus was shown for 500 msec. Participants reported verbally whether the subset of coherent dots was moving leftward or rightward. A perception threshold was calculated by fitting each participant's data to a Weibull function $y=1-0.5e^{-(\frac{x}{a})^b}$, where y is the proportion of correct responses, x is the proportion signal to noise, and a and b are two curve-fitting parameters. The Weibull function is a psychometric function used to model data from two-alternative forced-choice paradigms and estimate the signal to noise ratio (coherence level) that corresponds to participant performance (i.e., estimate the perception threshold). We computed the percent coherence that corresponded to 80% correct performance.

Biological Motion Perception Assessment—Biological motion perception was tested using PLW stimuli; the procedures for stimulus creation are described in detail elsewhere (Kaiser, Delmolino, Tanaka, & Shiffrar, 2010; Kaiser, Shiffrar, & Pelphrey, 2012; Thomas & Shiffrar, 2010). One male and one female adult actor each walked on a treadmill in a ReActor motion capture system (Ascension Technology Corporation, Milton, VT). Each actor wore 13 sensors attached to the head, shoulders, elbows, and wrists, hips, knees, and feet.

In one condition ("Natural"), the actors were instructed to walk as they normally would at three different speeds (0.5, 1.0, 1.5 m/s). In a second condition ("Parkinsonian"), the actors were instructed to walk like someone with PD: i.e., with short, shuffling steps, decreased arm swing, and a forward postural lean. One actor had extensive experience working with adults with PD, and the other actor was coached on how to approximate a parkinsonian gait pattern. With the parkinsonian gait, both actors walked at the same three speeds (0.5, 1.0, 1.5 m/s). These PLW stimuli were shown to eight observers (lab members) who had extensive experience conducting cognitive and motor assessments with individuals with PD in the lab and regularly observed parkinsonian gait. Observers were asked to rate how parkinsonian the gait appeared in each stimulus on a 1—5 Likert scale (1 = least parkinsonian; 5 = most parkinsonian). The observers' perceptual ratings confirmed that slower speeds with parkinsonian gait were perceived as more parkinsonian, while faster speeds with normal gait were perceived as least parkinsonian (mean ratings provided in Table 2). Inter-observer reliability was high (intraclass correlation coefficient = .912).

The purpose of these manipulations of gait and speed was to create two sets of walking patterns, one normal and one abnormal (abnormal being similar to parkinsonian walking), to test the hypothesis that the PD group would be better at perceiving abnormal, slow walking than perceiving fast, healthy walking patterns presumably outside their motor repertoire. Each actor walked in one direction only during motion capture. While rendering the movies, these PLW stimuli were flipped to create an equal number of walking trials in the opposite direction. All PLW stimuli were five seconds in duration to ensure sufficient time for participants to perceive and process full strides and inter-limb coordination patterns.

This procedure created a set of 24 "coherent" PLW stimuli (2 actors × 2 directions × 3 speeds × 2 gait types). For each coherent stimulus, a "scrambled" PLW stimulus was created by randomizing the starting location of individual point-lights on the body; this procedure ensured the same local dot motions in the scrambled stimuli, but destroyed the spatio-temporal coherence required to perceive a human walking. The scrambled stimuli served as foils in a two-alternative forced choice detection task. The 48 stimuli (24 coherent and 24 scrambled) were each presented twice for a total of 96 trials. Participants verbally reported whether or not they perceived a human walking ("yes" or "no") and the examiner recorded the response. Eight practice trials were conducted using additional stimuli, prior to the experimental trials. The dependent variable was an unbiased measure of sensitivity, *d'*, calculated using the standardized hit rate minus the standardized false alarm rate. Hit rates and false alarm rates were collapsed across actors and directions.

In a second biological motion perception task, coherent and scrambled PLW stimuli were presented in visual noise masks. Following Thomas and Shiffrar (2010), masks were created by duplicating each point-light and randomizing its starting location within a one to five point radius of one of the points defining the original walker (e.g., the "head dot" could be duplicated and positioned near the "right wrist dot"). This procedure was repeated for all 48 PLW stimuli, creating a corpus in which half of the stimuli were coherent+masked and half were scrambled+masked. This masking procedure creates a more challenging psychophysical task, which renders the local motions of individual dots uninformative and therefore requires the participant to detect the global spatiotemporal structure of the human form. Examples of stimuli are provided in Figure 1. Participants completed a two-alternative forced choice detection task using coherent-masked and scrambled-masked stimuli that were each presented twice (total of 96 trials). They were instructed to decide whether or not they perceived a human walking "within a cloud of extra dots." Participants responded verbally ("yes" or "no") and responses were recorded by the examiner. Eight practice trials were conducted using additional stimuli, prior to the experimental trials. The main outcome measure was d'. Hit rates and false alarm rates were collapsed across actors and directions.

Object Motion Perception Assessment—This task served as a control condition in order to stringently test whether potential perceptual deficits were specific to aspects of *biological* motion because object motion mirrors biological motion in its complexity (i.e., animate, non-rigid, jointed), but is non-biological. Object motion perception invokes neural substrates that differ from those invoked by biological motion perception (Beauchamp, Lee, Haxby, & Martin, 2003). Following Kaiser et al. (2010), the object was a "John Deere Loader" toy tractor (Peg Perego, 124.5 × 63.5 cm) filmed as it moved on a treadmill. 13

sensors were attached to the tractor (4 on each wheel, 3 on the bucket and bucket pivot joint, 1 on the top, and 1 at the back). The tractor moved on the treadmill at 1.0 m/s in one direction only and the motion capture system was used to create a point-light stimulus of the moving tractor. Stimulus construction was analogous to that for the biological motion perception task in several respects. While rendering the movie, the point-light object stimulus was flipped to create a stimulus moving in the opposite direction. The stimuli were five seconds in duration. For each coherent point-light object stimulus, a scrambled point-light object stimulus was created by randomizing the starting location of each dot, analogous to the procedure described above. Participants viewed 20 stimuli (2 moving directions × 2 stimuli type [coherent/scrambled] × 5 repetitions of each) and had to decide whether or not they perceived a moving tractor. Answers were given verbally (“yes” or “no”) and d’ was the dependent variable, collapsed across the two moving directions. Participants completed four practice trials prior to the experimental trials.

In a second object motion perception task, coherent and scrambled stimuli were presented in visual noise masks. The masking procedure was analogous to that for the biological motion perception task (i.e., 13 additional dots, duplicated from the original stimulus with a randomized starting position). Participants again viewed 20 stimuli (2 moving directions × 2 stimuli type (coherent/scrambled) × 5 repetitions of each) and reported verbally (“yes” or “no”) whether or not they perceived a moving tractor. The outcome measure was d’. Participants completed four practice trials prior to the experimental trials.

Administration of screening measures, questionnaires, and vision tests was completed in a quiet testing room. The motion perception experiments used a CRT monitor (Mitsubishi Diamond Pro 21 inch monitor with 160 Hz max refresh rate) at a distance of 60 cm. All stimuli were presented with MatLab 2009a (MathWorks, Natick, MA) and Psychophysics Toolbox version 3.0 (Brainard, 1997; Kleiner, Brainard, & Pelli, 2007; Pelli, 1997). Participants sat in a comfortable, adjustable seat and were given frequent breaks as needed.

Gait Assessment—All participants completed a gait assessment in the laboratory to determine whether gait characteristics were associated with biological motion perception. Spatiotemporal gait parameters were derived using tri-axial Geneactiv accelerometers (ActivInsights Ltd., Cambridgeshire, UK) at a sampling frequency of 100 Hz (dynamic range: +/− 8g; resolution: 12 bit [3.9 mg]). Participants wore the accelerometer on the right ankle while walking in a straight line down a hallway. One participant wore the accelerometer on the left ankle because the signal was not discernable from the right ankle. Participants completed four walking trials: two trials walking for 10m each, and two trials walking for 20m each, with different distances to provide diversity in walking to better approximate naturalistic settings. All participants were instructed to walk at their natural, comfortable walking pace.

Acceleration data was extracted using EMG Works software (Delsys Inc., Natick, MA) and temporal gait parameters were based on automatic peak detection functions of the software which identified maximal (heel-strike) and secondary (toe-off) peaks from the x-axis of the accelerometer, which was oriented in the sagittal plane of the lower limb. This procedure is similar to previous methods that have validated accelerometer-measured heel-strikes and

toe-offs using footswitches and force plates (Boutaayamou et al., 2012; Heiden & Burnett, 2004; Lee, Cho, Lee, Yang, & Lee, 2010; Willemsen, Bloemhof, & Boom, 1990).

At the end of each trial, once participants had reached a yellow tape marker that specified the end of the walking trial, they often took extra steps past the marker to maintain their standing posture and to turn and face the examiner for the subsequent trial. To ensure that we did not include these additional steps past the specified endpoint, we first inspected the heel-strike amplitudes of five consecutive strides from the middle of the trial. We then excluded any strides at the end of the trial where the amplitude of the heel-strike was less than 33% of the amplitude of the strides in the middle of the trial.

One stride was defined as the time between successive heel strikes of the same leg. The spatiotemporal walking variables that were identified included: stride length (distance of walking trial / number of strides); stride frequency (number of strides / time to complete the walking trial); and walking speed (distance of the walking trial / time to complete the trial). For each participant, all spatiotemporal walking variables were calculated separately for the four walking trials and then averaged together to determine the mean and standard deviation (variability) across the four trials.

Statistical Analyses

Group differences on the motion perception tasks were analyzed using mixed design ANOVA with Group (PD, control) as the between-subjects variable and the relevant within-subjects variables (e.g., Mask Condition [Mask, No Mask]; PLW Speed [0.5, 1.0, 1.5 m/s]; PLW Gait Type [parkinsonian, natural]). Statistical tests were two-tailed with alpha of .05. Post-hoc comparisons for main and interaction effects were conducted using t-tests. We report 95% confidence intervals (in square brackets) whenever appropriate.

For simple main effects and interaction effects in ANOVA, we report effect size using eta-squared (η^2), the proportion of the total variability in the data that is accounted for by that effect. For post-hoc comparisons of two means, we report effect size using Cohen's *d*, where .2 is a small effect, .5 is a medium effect, and .8 is a large effect. The pooled standard deviation was used as the standardizer (denominator) for calculating Cohen's *d*.

To determine whether biological motion perception was associated with gait parameters or visual function, Pearson correlations (two-tailed) were conducted. To account for the large number of correlation analyses, we used a conservative alpha level of .01.

Results

Demographics and Clinical Measures

A series of one-way ANOVAs was conducted to examine group differences on demographic and clinical measures. As shown in Table 1, there were no significant group differences in age, education, general cognitive status, depression, or anxiety (all F's < 3.68, p's > .05). There was no difference in male:female ratio between groups ($\chi^2 = .35$, $p = .56$) and there were no gender × group interactions on any perception tests (all F's < 1.96, p's > .05).

Assessment of Basic Vision and Coherent Motion Perception

The PD and control groups did not differ in visual acuity ($F(1,48) = 2.43$, $p = .13$, $\eta^2 = .05$). Group differences in contrast sensitivity (FACT chart) were determined using a 2×5 mixed design ANOVA with a between subjects factor of Group (PD, control) and within subjects factor of spatial frequency (1.5, 3, 6, 12, and 18 cycles/degree). There was a main effect of spatial frequency ($F(4,180) = 237.83$, $p < .001$, $\eta^2 = .84$) and a main effect of Group ($F(1,45) = 5.8$, $p = .02$, $\eta^2 = .11$). Individuals with PD had poorer contrast sensitivity than control participants regardless of spatial frequency (Mean difference = .11 [.02, .20], $p = .02$, $d = .7$). There was no Group × spatial frequency interaction ($F(4,180) = 1.27$, $p = .28$, $\eta^2 = .004$).

On the coherent motion perception task, one individual with PD and one control participant were excluded from the analysis because it appeared that they did not understand the task and performed at chance level at all coherence levels. An additional three individuals with PD were excluded because their threshold values were greater than two standard deviations from the group mean. There was no group difference in coherent motion perception threshold (mean difference = .34 [−3.37, 4.04], $t(43) = .18$, $p = .86$, $d = .06$).

Biological Motion Perception Task—Overall Performance by Mask Condition

The outcome measure, d', was calculated using the hit rate and false alarm rate across all trials (i.e., collapsed across all stimulus speeds and gaits). As shown in Figure 2, results demonstrated a significant main effect of Group ($F(1,48) = 19.4$, $p < .001$, $\eta^2 = .29$), a significant main effect of Mask Condition ($F(1,48) = 247.56$, $p < .001$, $\eta^2 = .82$), and a significant Group × Mask Condition interaction ($F(1,48) = 6.03$, $p = .02$, $\eta^2 = .02$). Post-hoc *t*-tests revealed that individuals with PD had poorer sensitivity to biological motion than control participants in both the No Mask (mean difference = .35 [.08, .62], $t(34) = 2.59$, $p = .01$, $d = .72$) and Mask conditions (mean difference = .85 [.45, 1.25], $t(48) = 4.32$, $p < .001$, $d = 1.22$), though the effect size was much larger in the Mask condition. For both groups, masked stimuli rendered the task more difficult as sensitivity to biological motion was lower in the Mask than the No Mask condition (mean difference in the PD group = 1.86 [1.54, 2.18], $t(25) = 12.04$, $p < .001$, $d = 3.09$; mean difference in the control group = 1.36 [1.09, 1.63], $t(23) = 10.33$, $p < .001$, $d = 2.61$).

Biological Motion Perception Task—Effect of PLW Gait Type

We conducted a three-way (Group × Mask Condition × PLW Gait Type) ANOVA. The outcome measure, d', was calculated using the hit rate and false alarm rate separately for natural gait and parkinsonian gait (i.e., collapsed across PLW Speeds). The three-way Group × Mask Condition × PLW Gait Type interaction was not significant ($F(1,48) = 1.39$, $p > .05$, $\eta^2 = 001$). The analysis revealed a significant Group × Mask Condition interaction ($F(1,48) = 10.26$, $p < .01$, $\eta^2 = .03$) and a significant Mask Condition × PLW Gait Type interaction ($F(1,48) = 65.92$, $p < .001$, $\eta^2 = 04$). Regardless of PLW Gait Type, the PD group had lower sensitivity to biological motion than the control group in the No Mask condition (mean difference = .26 [.06, .47], $t(31) = 2.68$, $p = .01$, $d = .75$) and in the Mask condition (Figure 3; mean difference = .82 [.46, 1.18], $t(48) = 4.6$, $p < .001$, $d = 1.3$). Sensitivity to biological motion was lower in the Mask condition than in the No Mask condition for the PD group

(mean difference = 1.5 [1.21, 1.78], $t(25) = 10.91$, $p < .001$, $d = 2.46$) and for the control group (mean difference = .94 [.73, 1.15], $t(23) = 9.21$, $p < .001$, $d = 2.50$). Across all participants, sensitivity to biological motion was greater when viewing PLWs with a Parkinsonian gait than with a Natural gait in the Mask condition (mean difference = .71 [.54, .88], $t(49) = 8.42$, $p < .001$, $d = 1.02$), but there was no difference in the No Mask condition (mean difference = .06 [−.03, .16], $t(49) = 1.33$, $p > .05$, $d = .15$).

Biological Motion Perception Task—Effect of PLW Speed

We conducted a three-way (Group × Mask Condition × PLW Speed) ANOVA. The outcome measure, d', was calculated using the hit rate and false alarm rate separately for the three PLW speeds (i.e., collapsed across PLW Gait Types). The three-way Group × Mask Condition × PLW Speed interaction was not significant ($F(2,96) = .08$, $p > .05$, $\eta^2 < .001$). The analysis revealed a significant Group × Mask Condition interaction ($F(1,48) = 10.86$, $p < .01$, $\eta^2 = .03$) and a significant Mask Condition × PLW Speed interaction ($F(2,96) = 59.75$, $p < .001$, $\eta^2 = .11$). Regardless of PLW Speed, the PD group had lower sensitivity to biological motion than the control group in the No Mask condition (mean difference = .21 [.04, .37], $t(30) = 2.72$, $p = .01$, $d = .76$) and in the Mask condition (Figure 4; mean difference = .72 [.38, 1.06], $t(48) = 4.31$, $p < .001$, $d = 1.23$). Sensitivity to biological motion was lower in the Mask condition than in the No Mask condition for the PD group (mean difference = 1.26 [1.00, 1.53], $t(25) = 9.91$, $p < .001$, $d = 2.22$) and for the control group (mean difference = .75 [.58, .93], $t(23) = 8.88$, $p < .001$, $d = 2.42$).

Across all participants, in the No Mask condition, sensitivity to PLWs at 1.5 m/s was greater than at 0.5 m/s (mean difference = .15 [.04, .26], $t(49) = 2.65$, $p = .01$, $d = .39$), and greater for 1 m/s than 0.5 m/s (mean difference = .12 [.00, .25], $t(49) = 2.02$, $p <..05$, $d = .3$). There was no difference in performance for PLWs moving at 1.5 vs. 1.0 m/s (mean difference = .03 [−.06, .11], $t(49) = .60$, $p > .05$, $d = .09$). Across all participants, in the Mask condition, sensitivity to biological motion was greater for PLWs moving at 1.5 m/s than at 1.0 m/s (mean difference = 1.24 [1.05, 1.42], $t(49) = 13.38$, $p < .001$, $d = 1.88$) and 0.5 m/s (mean difference = .62 [.44, .81], $t(49) = 6.75$ $p < .001$, $d = .83$). Sensitivity to biological motion was poorer at 1 m/s than at 0.5 m/s (mean difference = .62 [.38, .86], $t(49) = 5.16$, $p < .001$, $d = .80$).

Object Motion Perception Task

As shown in Figure 5, there was a significant main effect of Group ($F(1,48) = 4.34$, $p = .04$, $\eta^2 = .08$), a significant main effect of Mask Condition ($F(1,48) = 15.6$, $p < .001$, $\eta^2 = .23$), and a significant Group × Mask Condition interaction ($F(1,48) = 4.42$, $p = .04$, $\eta^2 = .07$). Post-hoc *t*-tests revealed that individuals with PD did not differ from control participants in the No Mask condition (mean difference = .09 [−.11, 28], $t(48) = .90$, $p = .37$, $d = .26$), whereas in the Mask condition, individuals with PD had significantly lower sensitivity to object motion than control participants (mean difference = .38 [.06, .71], $t(47) = 2.39$, $p = .02$, $d = .68$). Within-group comparisons of performance on the No Mask vs. Mask condition showed no difference for control participants (mean difference = .13 [−.1, .36], $t(23) = 1.17$, $p = .26$, $d = .28$), but significantly poorer performance in the PD group in the Mask than the No Mask condition (mean difference = .43 [.25, .61], $t(25) = 4.86$, $p < .001$, $d = .91$).

Direct Comparison of Perception Tasks

To compare performance of the PD group across perception tasks, we computed *z* scores for each individual with PD using the mean and standard deviation of the control group, separately for the biological motion perception task (Mask condition), object motion perception task (Mask condition), and coherent motion perception task. Individuals with PD had significantly lower *z* scores (more impairment) on the biological motion perception task (mean *z* score = −1.23, *SD* = 1.08) compared to the object motion perception task (mean *z* score = −0.75, *SD* = 1.21; $t(25) = 2.29$, $p = .031$) and the coherent motion perception task (mean *z* score = .05, *SD* = .95; $t(21) = 4.83$, $p < .001$).

Gait and Visual Correlates of Biological Motion Perception

Table 3 displays values for gait parameters in PD and control participants. Compared to the control group, the PD group had significantly slower walking speed ($F(1,48) = 25.45$, $p < .001$, $\eta^2 = .35$) and shorter stride length ($F(1,48) = 17.05$, $p < .001$, $\eta^2 = .26$). There were no group differences in stride frequency, or in the variability (standard deviation) of walking speed, stride length, and stride frequency (all F's < 2.76, all p's > .05, all $\eta^2 < .05$). In neither group were there any significant correlations between mean or standard deviation of gait parameters and sensitivity to biological motion perception, in either the No Mask or the Mask condition (all r's < .45, all p's > .01).

In determining whether contrast sensitivity was associated with biological motion perception, one participant with PD was excluded whose contrast sensitivity at 1.5 cpd was 4.5 standard deviations below the group mean. In the control group, No Mask condition, the only significant correlations appeared between contrast sensitivity at 1.5 cycles/degree and d' for PLW stimuli moving at 1.0 m/s ($r = .52$, $p = .01$) and between contrast sensitivity at 12 cycles/degree and d' for PLW stimuli moving at 1.0 m/s ($r = .54$, $p = .01$). There were no significant correlations in the Mask condition. In the PD group, no significant correlations emerged between contrast sensitivity and sensitivity to biological motion in the No Mask and Mask conditions (all r's < .38, all p's > .01). We also found no significant correlations between coherent motion perception and biological motion perception in either the No Mask or Mask conditions, in either group (all r's < .3, all p's > .05). Together, these results suggest that contrast sensitivity and coherent motion perception did not account for the PD group's reduced sensitivity to biological motion.

Discussion

The main goal of the present study was to determine whether biological motion perception is impaired in PD without dementia. In accord with our hypothesis, the results showed less sensitivity to biological motion in the PD group than in the matched healthy control group. This group difference emerged regardless of whether the PLW stimuli were presented with or without a visual noise mask but, as expected, the deficit was more extreme under the visual mask condition (large effect) than under the no-mask condition (medium effect size). These results demonstrate that PD is associated with a deficit in extracting the global spatiotemporal features of human motion under suboptimal viewing conditions, as may occur in the natural environment.

The PD group also showed less sensitivity to object (non-biological) motion than control participants, though only when the object was presented in a noise mask. This finding suggests that those with PD may have a generalized deficit in perceiving motion-defined forms, which is consistent with prior reports of PD-related alterations in perception of motion-defined objects (Uc et al., 2005), motion-defined surfaces (Castelo-Branco et al., 2009), and second-order motion (Ezzati et al., 2010). Individuals with PD, however, performed significantly worse on the biological motion task than the object motion task. Moreover, the standardized group difference in biological motion perception (large effect size) was almost twice that of the group difference in object motion perception (medium effect size), indicating that in PD, the magnitude of the deficit is larger for human motion than object motion.

In healthy adults, perception of biological motion and object motion activates overlapping regions in occipital, parietal, and temporal cortices (Virji-Babul, Cheung, Weeks, Kerns, & Shiffrar, 2007), as well as regions that are unique to each type of motion perception. Within the lateral temporal cortex, biological motion consistently activates the STS and superior temporal gyrus, whereas object motion is associated with activity in middle temporal gyrus and inferior temporal sulcus (Beauchamp, Lee, Haxby, & Martin, 2002; Beauchamp et al., 2003). Our results suggest that in PD, activity in superior temporal regions may be more compromised than activity in middle and inferior temporal regions, possibly arising from dysfunctional connections between the caudate and extrastriate cortex (Seger, 2013), or structural and functional changes to these regions (Lotze et al., 2008; Pereira et al., 2009).

The second goal of this study was to examine gait and visual correlates of biological motion perception in PD. We predicted that the PD group would have particular difficulty in perceiving biological motion outside their motor repertoire. Contrary to our hypothesis, individuals with PD did not have poorer sensitivity to PLW stimuli moving at fast speeds compared to slow speeds, or to PLW stimuli with a natural gait compared to a parkinsonian gait. These findings were surprising given evidence of specific impairments in perceiving motion that patients can no longer perform themselves in other motor disorders (Arrighi et al., 2011; Serino et al., 2009), and previous findings that have demonstrated impaired perception-action coupling in PD (Poliakoff, Galpin, Dick, Moore, & Tipper, 2007; Tremblay, Léonard, & Tremblay, 2008).

One limitation of our approach of using PLW stimuli with parkinsonian gait was that the stimuli were actors imitating a parkinsonian gait, which may not have effectively activated the mirror neuron system of PD participants. A second limitation is that the noise mask may not have adequately masked the parkinsonian PLW stimuli (e.g., due to the lack of movement of the dots), given that all participants had increased sensitivity to parkinsonian PLW stimuli compared to natural PLW stimuli. To further investigate the perception-action association, we calculated spatiotemporal gait parameters (mean and standard deviation walking speed, stride length, and stride frequency). Although the PD group had significantly slower walking speed and shorter stride length than the control group, there was no association between these gait characteristics and biological motion perception in either group, providing further evidence against an association between gait deficits in PD and biological motion perception.

Our findings argue against deficient perception-action coupling as the underlying mechanism of altered human motion perception in PD. The biological motion deficit was not associated with walking dysfunction, and the deficit did not appear to be tied to difficulty simulating or embodying actions (mediated by premotor cortex and the mirror neuron system) outside the PD group's motor repertoire. Rather, impaired biological motion perception, and to a lesser extent object motion perception, may arise from perceptual difficulty in the spatiotemporal integration of form and motion cues, an ability largely dependent on posterior STS (biological motion) and middle temporal cortex (object motion). Deficient perception-action coupling as it relates to human motion perception may become more apparent at later disease stages in PD that are characterized by greater motor impairment.

Our results extend previous findings on impaired social perception in PD (Clark et al., 2008, 2010; Pell et al., 2014), and suggest that the disease results in a broad impairment in social processing. Given the relation between biological motion perception and "higher order" social cognition such as theory of mind (Miller & Saygin, 2013), reduced sensitivity to biological motion may contribute to other observed social difficulties in the disease.

In the present study, biological motion perception was not associated with contrast sensitivity, despite the PD group having poorer contrast sensitivity than healthy control participants. We assessed contrast sensitivity using the Functional Acuity Contrast Test, which is coarser than a threshold measure, and it is possible that contrast sensitivity measured in a more sensitive psychophysical test may demonstrate an association with biological motion perception in PD. Biological motion perception was also not associated with low-level coherent motion perception mediated by area MT, as there were no group differences in perception threshold on a coherent motion perception task, nor did these thresholds correlate with performance on the biological motion task. These findings provide additional support that insensitivity to biological motion in PD is independent of motor and basic, low-level perceptual changes.

The possibility exists that the observed deficit in biological motion perception can be explained by poor visual attention or reduced processing speed, which occur commonly in PD (Jokinen et al., 2013; Zhou et al., 2012). Attention has been shown to play a role in action recognition from biological motion, particularly in noisy conditions (Thompson & Parasuraman, 2012). It is unlikely that attention was a major contributor to our findings, however. First, the biological motion and object motion tasks had an equal number of signal and noise dots (i.e., equal attentional demands), but PD participants performed worse on the biological motion task. Second, point-light stimuli were presented for 5 seconds to ensure participants had sufficient time to attend to and process each stimulus. Finally, PD participants were not impaired on the coherent motion task, which was presumably the most attentionally demanding as stimuli were presented for only 0.5 seconds with many more noise dots than the biological motion and object motion tasks.

In summary, the present study demonstrated that non-demented individuals with PD are impaired relative to control participants in perceiving biological motion (large effect), and also in perceiving object motion (medium effect). The impairment is significantly worse for

biological motion than for object motion. Reduced sensitivity to biological motion in PD may arise from changes in STS and lateral middle temporal cortex, rather than from deficiencies in perception-action coupling or in lower-level visual perception. Of clinical relevance, difficulties in perceiving the movements and actions of others, though independent of the severity of individuals' own gait dysfunction, may lead to difficulty in interpersonal communication and social functioning for those living with the disease.

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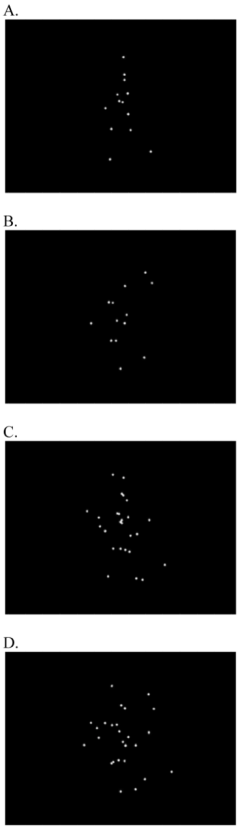

Figure 1. Static images of point-light walker videos by condition

A. human walker with no mask; B. scrambled walker with no mask; C. human walker in a noise mask; and D. scrambled walker in a noise mask.

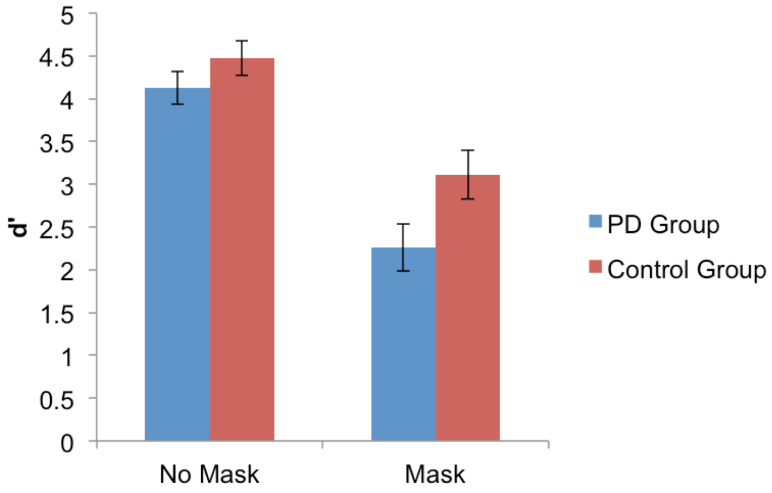

Figure 2. Sensitivity to biological motion by Mask Condition and Group
Error bars represent 95% confidence intervals.

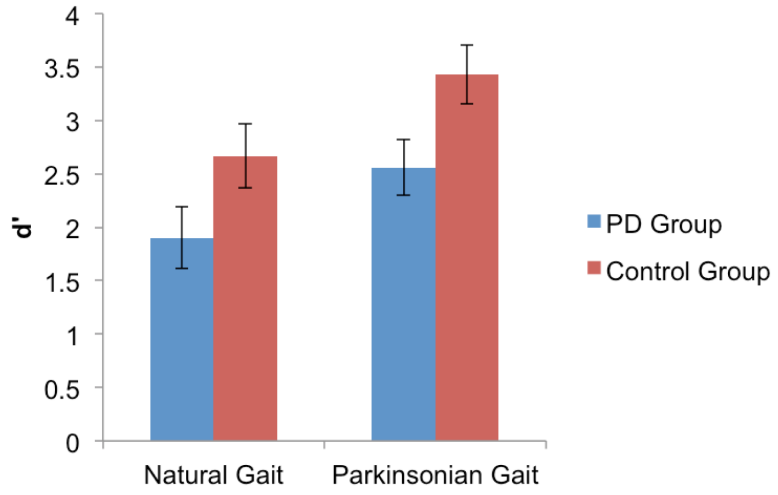

Figure 3. Sensitivity to biological motion by PLW Gait Type (Natural, Parkinsonian) and Group in the Mask condition

Error bars represent 95% confidence intervals.

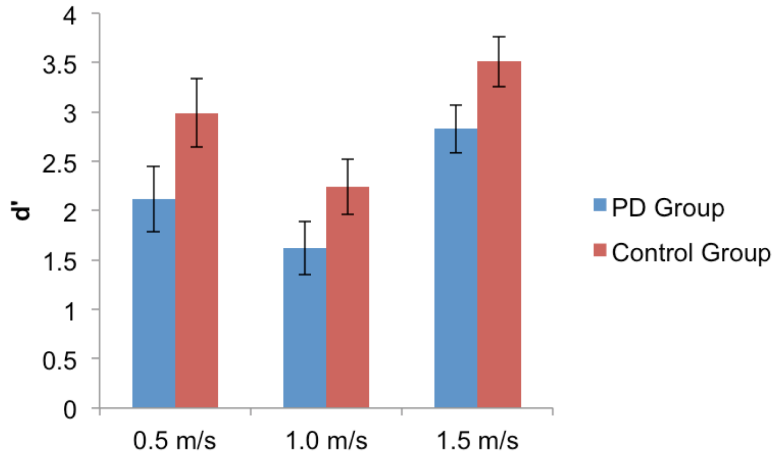

Figure 4. Sensitivity to biological motion by PLW Speed (0.5, 1.0, 1.5 m/s) and Group in the Mask condition

Error bars represent 95% confidence intervals.

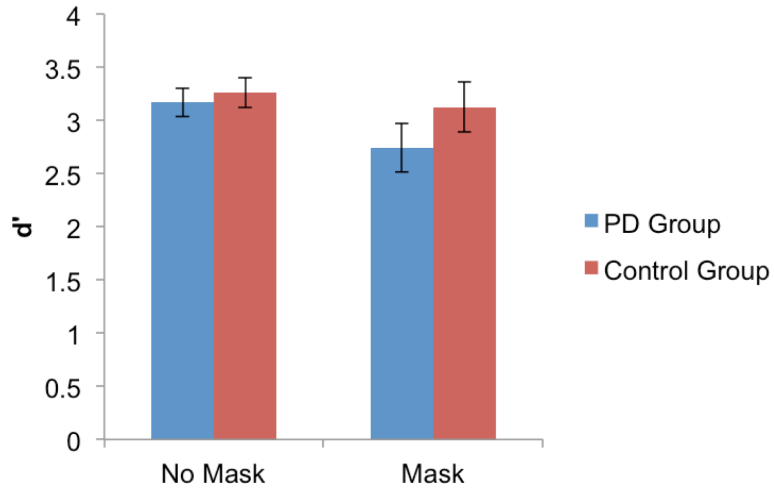

Figure 5. Sensitivity to object motion by Mask Condition and Group
Error bars represent 95% confidence intervals.

Table 1

Participant Characteristics by Group

Measure	Parkinson's disease (N=26)	Control (N=24)	Significance
Age (years)	65.1 (7.9)	62.5 (8.6)	*NS*
Education (years)	16.5 (2.1)	17.3 (1.8)	*NS*
Men:Women	13:13	10:14	*NS*
Unified Parkinson's Disease Rating Scale	18.6 (8.0)	--	--
Hoehn & Yahr Stage (median)	2.0	--	--
Levodopa Equivalent Dosage (mg/day)	469 (268)	--	--
Acuity (log mean angle of resolution)	0.08 (0.1)	0.03 (0.12)	*NS*
Mini Mental State Examination	28.5 (0.9)	28.9 (0.8)	*NS*
Geriatric Depression Scale	6.1 (4.1)	4.1 (5.0)	*NS*
Beck Anxiety Inventory	5.2 (5.0)	3.1 (3.8)	*NS*

Note. Values presented are means (standard deviations), unless otherwise indicated.

Table 2

Mean Perceptual Ratings of Point-Light Walker Stimuli

Stimulus Gait	Stimulus Speed	Mean Rating (1-5)
Natural	0.5 m/s	3.06
Natural	1.0 m/s	1.31
Natural	1.5 m/s	1.31
Parkinsonian	0.5 m/s	3.69
Parkinsonian	1.0 m/s	2.75
Parkinsonian	1.5 m/s	2.38

Note. Eight lab members who had extensive experience working with adults with Parkinson's disease were asked to rate the point-light walker stimuli on a scale of 1—5, with 1 being "least parkinsonian" and 5 being "most parkinsonian."

Table 3

Mean [95% Confidence Intervals] of Gait Parameters by Group

Walking Parameter	Parkinson's disease (N=26)	Control (N=24)	Significance
Mean Walking Speed (m/s)	1.16 [1.10, 1.21]	1.35 [1.29, 1.41]	$p < .001$
SD Walking Speed (m/s)	0.07 [0.06, 0.09]	0.09 [0.08, 0.10]	*NS*
Mean Stride Length (m)	1.33 [1.27, 1.39]	1.52 [1.45, 1.60]	$p < .001$
SD Stride Length (m)	0.06 [0.04, 0.08]	0.06 [0.04, 0.08]	*NS*
Mean Stride Frequency (stride/s)	0.87 [0.85, 0.90]	0.89 [0.86, 0.92]	*NS*
SD Stride Frequency (strides/s)	0.03 [0.03, 0.04]	0.04 [0.04, 0.04]	*NS*

Note. SD = standard deviation. Walking parameters were calculated for each participant by taking the mean and standard deviation (as an index of variability) across four straight-line walking trials.