BRIEF REPORT

Bodily Maps of Symptoms and Emotions in Parkinson's Disease

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ABSTRACT: Background: Emotions are reflected in bodily sensations, and these reflections are abnormal in psychiatric conditions. However, emotion-related bodily sensations have not been studied in neurological disorders.

Objective: The aim of this study was to investigate whether Parkinson's disease (PD) is associated with altered bodily representations of emotions.

Methods: Symptoms and emotion-related sensations were investigated in 380 patients with PD and 79 control subjects, using a topographical self-report method, termed body sensation mapping. The bodily mapping data were analyzed with pixelwise generalized linear models and principal component analyses.

Results: Bodily maps of symptoms showed characteristic patterns of PD motor symptom distributions. Compared with control subjects, PD patients showed decreased parasternal sensation of anger, and longer PD symptom duration was associated with increased abdominal sensation of anger ($P_{FWE} < 0.05$). The PD-related sensation patterns were abnormal across all basic emotions ($P < 0.05$).

Conclusions: The results demonstrate altered bodily maps of emotions in PD, providing novel insight into the nonmotor effects of PD. © 2024 The Authors. *Movement Disorders* published by Wiley Periodicals LLC on behalf of International Parkinson and Movement Disorder Society.

Key Words: bodily sensation mapping; emotion; Parkinson's disease; nonmotor symptoms

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Introduction

Emotions tune our bodies for different situations. They modulate attention, decision-making, and cognitive functions to ensure survival and during everyday life. Emotions are associated with physical responses, mediated by the sensorimotor and autonomic nervous systems. For example, when a worker hears about layoffs with simultaneous bonuses to the CEO, a hiker faces a grizzly bear, or a mother sees her newborn, emotions prepare them broadly for action.[1,2]

Body sensation mapping (BSM) is a relatively new technique for quantitative, topographical mapping of bodily sensations associated with emotions.[2] The method has been shown to yield consistent results across cultures and developmental stages, independent from sex.[3,4] (Neuro)psychiatric disorders, such as schizophrenia and autism, affect emotion processing, reflected in the bodily maps of emotions.[5,6] However, it is unclear whether neurodegenerative disorders alter the bodily representations of emotions.

Parkinson's disease (PD) is a common, diffuse neurodegenerative disorder defined and diagnosed by the motor symptoms.[7] Yet, PD is also associated with substantial nonmotor symptom burden.[7] The symptoms of PD, especially in the advanced disease stage, are not

stable but show considerable fluctuations, primarily linked with brain dopamine levels, but are also modulated by emotional states.[8,9] Moreover, basal ganglia, the brain structures primarily affected in PD, play a crucial role in emotional processing.[10-12] Unsurprisingly, previous studies have suggested abnormalities in the recognition of emotional prosody and facial expressions in PD.[13,14]

In this study, we investigated bodily sensations of motor and sensory symptoms and emotions in individuals with PD and control subjects. We hypothesized that PD is associated with abnormal bodily maps of emotions corresponding to the known autonomic deficits caused by the disease.

Subjects and Methods

Study Sample

A total of 380 individuals with PD and 79 control subjects participated in the study. The study protocol was approved by Turku University Hospital Clinical Research Services Board. The need for separate ethics board review was waived. Written informed consent was obtained, and the study was conducted according to the principles of the Declaration of Helsinki. Participants were recruited through the Finnish Movement Disorders Association via e-mail. The inclusion criterion for the PD group was an established diagnosis of PD by a neurologist, and for the control group, no evidence of PD or other neurodegenerative parkinsonism syndrome. Data collection was conducted online on a platform hosting the emBODY-tool and a questionnaire about demographic and clinical information, including age, sex, education, alcohol consumption, and smoking, and PD disease duration (from diagnosis), duration of motor symptoms (self-reported), dopaminergic medications, and device-aided treatments.[15]

Bodily Sensation Maps

The BSM maps (resolution 171 × 522 pixels) were collected with the emBODY-tool.[2] The participants were asked to color (1) the bodily locations of the currently experienced motor and sensory symptoms (clumsiness, stiffness, tremor, numbness, and pain) and (2) bodily sensations associated with basic emotions (anger, disgust, fear, happiness, sadness, surprise, and neutral) on an outline of the human body (Supporting Information Fig. S1). With emotions, the participants were instructed to think carefully what they feel in their body while feeling the emotion in question and to separately color the areas of the body in which they feel the activity increasing or decreasing. Coloring was performed by pressing the mouse button and simultaneously dragging the cursor on the body template. As in the previous studies using this method, emotions were mapped to a body outline without separating front and back side, because emotions are typically localized inside the body rather than superficially.[2,5,6] The raw data matrices were converted to NIfTI (Neuroimaging Informatics Technology Initiative) format to facilitate the preprocessing, and the data were smoothed with Gaussian kernel smoothing ($\sigma = 20$) respecting the body outline boundaries, to control for possible minor anatomical imprecision in the drawings. The preprocessing of the BSM data is described in detail in the Supporting Information Methods (Data S1).

Statistics

Statistical analyses were performed with R 4.3.2 (https://www.r-project.org) and pixelwise generalized linear model analyses with Statistical Parametric Mapping software (SPM12; https://www.fil.ion.ucl.ac.uk/spm/software/spm12/). Group differences in demographic and clinical data were investigated using Mann–Whitney *U* test or Fisher's exact test, as appropriate.

First, to evaluate focal between-group differences and clinical associations of the bodily maps of symptoms and sensations, we analyzed the BSM data with pixelwise generalized linear models applying family-wise error (FWE) correction to correct for multiple comparisons across the whole-body template. The bodily maps of symptoms and emotions were visualized by calculating pixelwise Cohen's D maps for each group separately.

Second, to characterize patterns of symptoms and emotion-associated sensations in PD more generally, we used principal component analysis to identify the two components best explaining the overall variance. In this analysis, more positive/negative expression of a component indicates more/less prevalent presence of the component loading pattern in a group compared with the other. Statistical significance of these components was evaluated using permutation tests. The between-group comparisons of the pattern expression values were conducted using Mann–Whitney *U* test and binary logistic regression. The *P* values less than 0.05 were considered significant across all analyses. For the covariance pattern expression values, median and 95% bootstrapped confidence interval of median (10,000 iterations) were chosen as descriptive statistics.

Finally, to evaluate the robustness of the observed covariance patterns, we created random forest classification models for the reclassification of data to control and PD groups. Differences between classification model performance scores were tested using Mann–Whitney *U* test. More details are available in the Supporting Information Methods (Data S1).

Results

There were no significant differences in age, alcohol use, or smoking status. However, the PD group had

TABLE 1 *Demographic and clinical characteristics*

Characteristics	Groups		*P* Value
	Controls (n = 79)	PD (n = 380)	
Age (y)	67 [64, 73]	67 [62, 72]	0.522
Sex, n (male/female) [% male]	31/48 [39.2%]	217/163 [57.1%]	0.005*
Education, n [%]			0.012*
Primary school education or equivalent	1 [1.3%]	36 [9.5%]	
Second degree education	37 [46.8%]	184 [48.4%]	
Higher education	37 [46.8%]	134 [35.3%]	
Not reported	4 [5.1%]	26 [6.8%]	
Smoking, n [%]			0.154
Yes	6 [7.6%]	16 [4.2%]	
Ex-smoker	6 [7.6%]	50 [13.2%]	
No	65 [82.3%]	269 [70.8%]	
Not reported	2 [2.5%]	45 [11.8%]	
Alcohol consumption (doses per week) ($n_C = 71$, $n_{PD} = 281$)	1 [0, 2.5]	1 [0, 4]	0.160
Motor symptom duration (y) (n = 366)	–	6 [3, 10]	–
Disease duration (from the diagnosis, y) (n = 379)	–	4 [2, 7]	–
Levodopa equivalent daily dose[a] (n = 330)	–	455 [300, 700]	–
Pump treatment for PD (yes/no [%]) (n = 339)	–	5/334 [1.4%]	–
Deep brain stimulation treatment for PD (yes/no [%]) (n = 331)	–	16/315 [4.8%]	–

For each numeric variable, median with 25th and 75th percentiles [in brackets] are reported, unless stated otherwise.
Abbreviations: PD, Parkinson's disease; C, Control.
[a]Levodopa equivalent daily dose was calculated as described previously.[3]
*$P < 0.05$.

more males and lower education levels compared with control subjects (Table 1). There were significant differences between the groups in both the pixelwise linear models ($P_{FWE} < 0.05$) and principal component analysis identifying the two main covariance patterns ($P < 0.001$) of which the first component showed more positive expression in individuals with PD ($P < 0.05$) and the second in control subjects ($P < 0.05$) (Supporting Information Tables S1 and S2). Adding sex or education as a covariate did not change the significance of any of the findings (Supporting Information Tables S1 and S2).

Maps of the Motor and Sensory Symptoms

The bodily maps of the control subjects were used as reference for motor and sensory symptoms associated with PD (Fig. 1A). In PD, clumsiness mapped primarily to distal limbs, stiffness to torso and limbs, and tremor to hands, aligning with clinically expected localization of bradykinesia, rigidity, and tremor, respectively (Fig. 1B). In the linear models, there were significant differences in bodily maps of symptoms corresponding to the motor symptoms of PD (clumsiness, stiffness, tremor), but not with sensory symptoms (numbness, pain) in individuals with PD compared with control subjects ($P_{FWE} < 0.05$; Fig. 1C). The principal component analysis indicated more extensive PD-related patterns of all studied motor and sensory symptoms (Fig. 1D, Supporting Information Fig. S3).

Maps of the Emotion-Related Sensations

Visually, emotion-associated maps of the groups showed a tendency toward overall weaker sensations in PD compared with control subjects; that is, the bodily sensations associated with emotions in control subjects were absent or less intensive in the PD group (Fig. 2A,B). In the linear models, individuals with PD showed reduced anger-related parasternal sensations compared with control subjects ($P_{FWE} < 0.05$; Fig. 2C). Adding sex or education as a covariate (Supporting Information Fig. S4) or balancing the group analysis with random oversampling (Supporting Information Fig. S5) did not change the significance of these findings. Motor symptom duration was significantly associated with anger-related sensation in the abdomen ($P_{FWE} < 0.05$), but not with other emotion-related

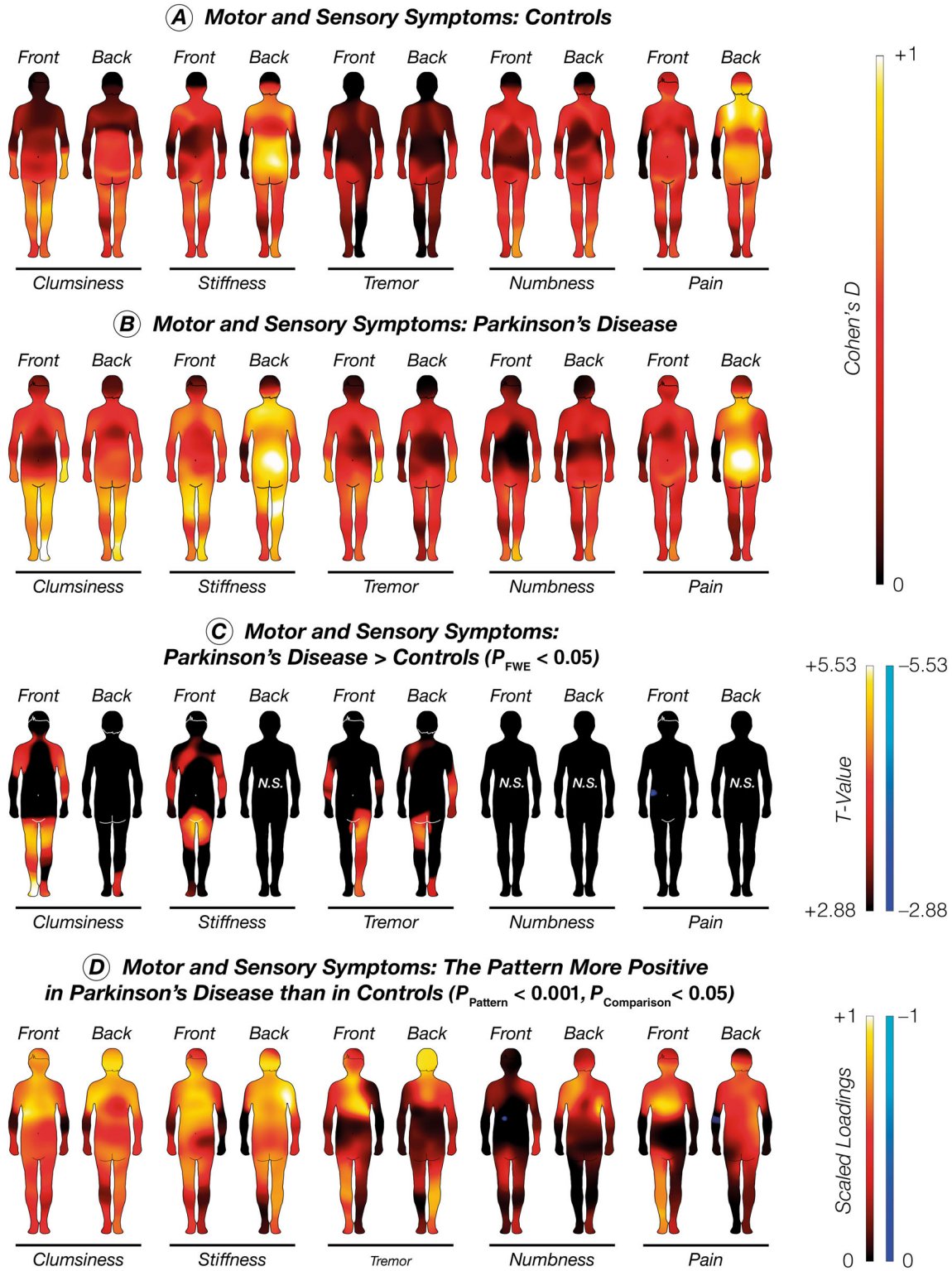

FIG. 1. Symptom-related bodily maps. The pixelwise effect sizes in control subjects (**A**) and individuals with Parkinson's disease (**B**); there are significant differences between groups in the linear model ($P_{FWE} < 0.05$) (**C**) and PD-related pattern ($P < 0.001$ for the pattern; $P < 0.05$ for expression compared with control subjects) (**D**). In Parkinson's disease, clumsiness, stiffness, and tremor map to the body regions commonly affected by bradykinesia, rigidity, and tremor, respectively (**B**). These motor, but not sensory, symptoms also showed significant regional differences compared with the controls (**C**). The principal component analysis showed similar but clearly more widespread patterns associated with Parkinson's disease (**D**). The red to yellow scale indicates positive values; the blue to light blue scale indicates negative values. N.S., nonsignificant. [Color figure can be viewed at wileyonlinelibrary.com]

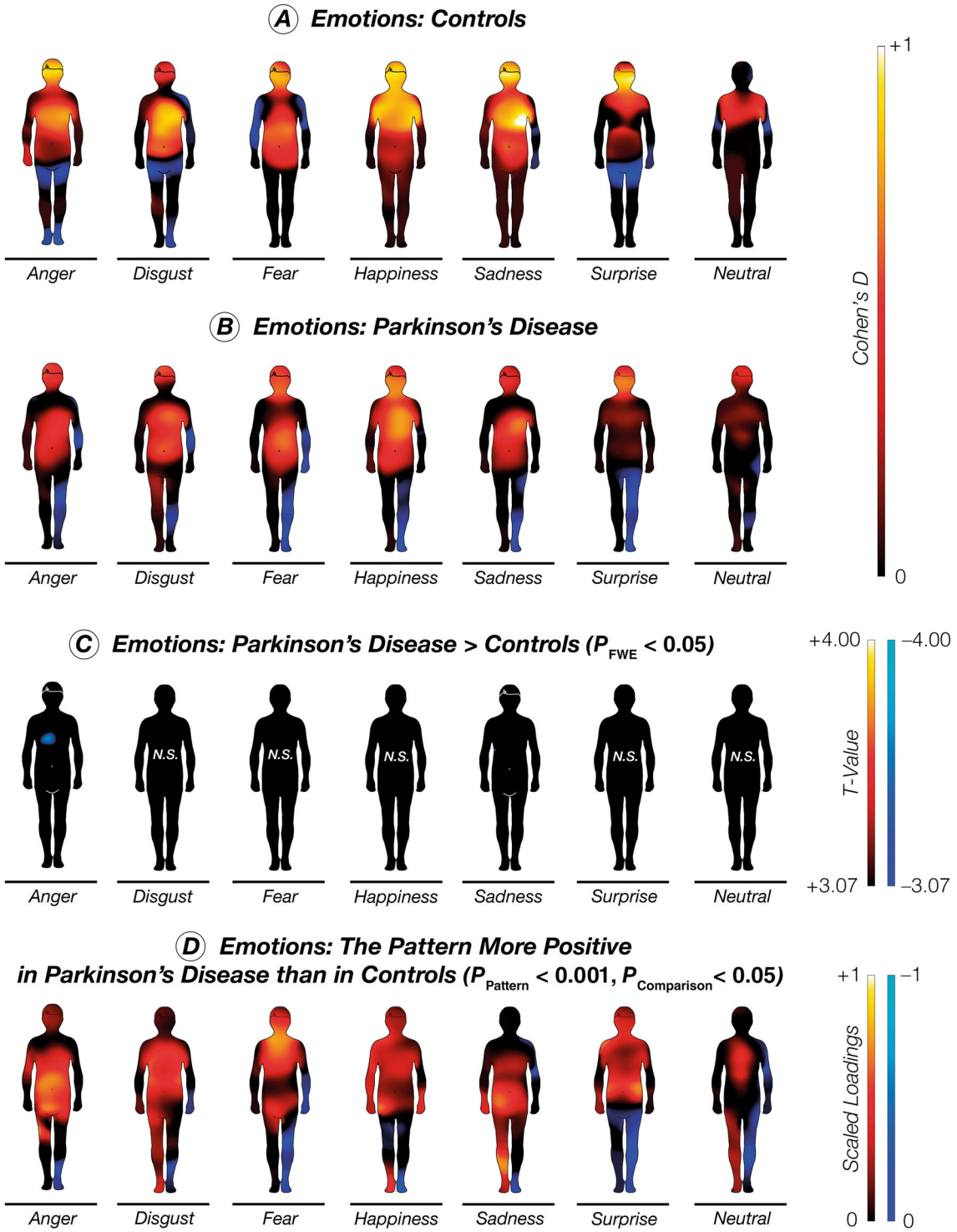

FIG. 2. Emotion-related bodily maps. The pixelwise effect sizes in control subjects (**A**) and individuals with Parkinson's disease (**B**); there were significant differences between groups in the linear model ($P_{FWE} < 0.05$) (**C**) and PD-related pattern ($P < 0.001$ for the pattern; $P < 0.05$ for expression compared with control subjects) (**D**). Visually, the bodily sensations associated with emotions show absent or less intensive expression in Parkinson's disease (**B**) compared with control subjects (**A**), with a significant difference between the groups in anger-related sensations (**C**). The principal component analysis indicated more widespread PD-related patterns across all emotions, localizing anger to the abdomen rather than chest in line with the linear model findings (**D**). The red to yellow scale indicates positive values; the blue to light blue scale indicates negative values. N.S., nonsignificant. [Color figure can be viewed at wileyonlinelibrary.com]

sensations, with or without the covariates (Supporting Information Fig. S6). None of the emotion-related sensations were associated with levodopa equivalent daily dose.

Similar to the motor and sensory symptoms, the principal component analysis showed more widespread PD-related covariance patterns of emotion-related

sensations, localizing anger to the abdomen rather than the chest (Fig. 2D). In addition to anger, PD-related patterns of disgust, fear, and surprise localized to different body regions, whereas the patterns of happiness and sadness were mostly more restricted compared with control-related patterns (Supporting Information Fig. S7).

Classification with Random Forest Models

The main classification model, including BSM pattern expression and demographic data, had a reasonable performance in the reclassification task. Across the 1000 repeats of first training a model and then testing it with a test dataset, this model had a median area under the curve of 0.745, 86.3% median sensitivity, and 100% median specificity, and performed better than the reference models without BSM pattern expression data ($P < 0.0001$) (Supporting Information Table S3).

Discussion

This study is the first to investigate the bodily sensations of motor and sensory symptoms and emotions in a neurological disorder (i.e., PD). The bodily maps of the motor symptoms corresponding to the parkinsonian cardinal motor symptoms matched well with clinical localization of these symptoms, demonstrating feasibility of this technique for topographical mapping of symptoms in PD. We further show that PD is associated with abnormal patterns of emotion-related bodily sensations. The bodily mapping data seem robust based on the reasonable performance in the reclassification task between individuals with PD and control subjects. Overall, our findings add novel information to the nonmotor symptoms of PD by showing that a neurodegenerative disorder can affect physical sensations of emotions.

The most prominent abnormality was decreased parasternal sensation related to anger with shift toward the abdomen along with disease progression. This finding may be related to cardiac sympathetic denervation, which is one of the major autonomic features in PD, contributing to the cardiovascular response defects in PD.[16] The cardiac sympathetic denervation process is typically present already early in the disease process and can be detected using [^{123}I]-meta-iodobenzylguanidine scintigraphy.[17] The results from previous studies of basic emotions and their autonomic counterparts in non-PD populations have also shown that anger has the strongest association with hemodynamic responses,[1] which may also be relevant for cardiovascular health.[18-20]

Our study is limited by the fact that the reported bodily sensations related to emotions are subjective, and it is not known how strongly they correlate with actual physiological responses or abilities experiencing or detecting emotions, which should be confirmed in an independent study with objective measures. In addition, as in the previous studies using BSM,[2,3,6] this study was conducted online, and therefore we were unable to clinically confirm the diagnoses. Although all individuals with PD reported to have an established diagnosis, some of the diagnoses are likely inaccurate because differential diagnostics of parkinsonism syndromes can be challenging even for neurologists.[21] However, this would only bias us against the present group differences. We also cannot exclude possible selection bias in our study population. For example, it is possible that patients with less cognitive impairment may be more likely to participate in this type of study. In addition, more severe motor symptoms and cognitive impairment could be reflected in the accuracy of the drawings. However, because the motor symptom maps aligned well with the known clinical symptom distribution, systematic bias in the observed group differences can be considered unlikely.

In conclusion, we show that PD is associated with altered emotion-related bodily sensations, demonstrating that these sensations are abnormal not only in psychiatric and neuropsychiatric disorders but also in a neurodegenerative disorder. Our study also demonstrates the feasibility of bodily sensation mapping technique in PD that could also be useful in other neurological disorders. However, the neurobiological and potential clinical relevance of these findings remain to be determined.

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Data Availability

The emBODY-tool is available online at https://version.aalto.fi/gitlab/eglerean/embody and group-level maps at https://github.com/kajuni/Bodily-maps-of-symptoms-and-emotions-in-PD. All other data are available from the corresponding author upon reasonable request, subject to the national and institutional regulations.

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Supporting Data

Additional Supporting Information may be found in the online version of this article at the publisher's web-site.

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A.H., E.J., E.G., L.N., and J.J. designed research; A.H., E.J., and E.G. collected data; K.J.N., L.N., and J.J. analyzed data; and K.J.N. and J.J. wrote the paper. All authors reviewed and critically revised the paper.

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