Impaired effort perception, not reward sensitivity, impacts the effort-reward trade-off in Parkinson's disease

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Abstract

Deciding whether and how to act depends on a trade-off between the effort required to execute a given action and the potential reward for completing it. Impairments in this effort-reward trade-off have been proposed to underlie reduced movement vigor, or bradykinesia, in Parkinson's disease, but the underlying mechanism remains unclear. Indeed, several mechanisms could alter the effort-reward trade-off, each with unique implications for understanding bradykinesia. Here, we examined three potential sources of impairment in the effort-reward trade-off in people with Parkinson's disease (both on and off dopamine medication) by separately interrogating reward sensitivity, perceptions of effort, and the mapping between effort and reward compared to age- and sex-matched neurotypical controls. We found that people with PD exhibited behavior consistent with increased perception of effort during isometric force production, compared to controls. In contrast, using a well-established psychometric test, we found no difference in reward sensitivity between groups. People with Parkinson's disease also showed no difference from control participants in the slope of the effort-reward trade-off function, suggesting a similar effort-reward mapping between groups. Within the Parkinson's disease group, none of the factors tested were modulated by dopaminergic medication status. Taken together, our results suggest that changes in perceived effort, rather than reduced reward sensitivity, may be an important contributor to reduced vigor in people with Parkinson's disease, and this impairment may not be mitigated by dopaminergic medication.

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Introduction

Deciding whether and how to act depends, in part, on a trade-off between the effort required for a given task and the potential reward for completing it. This is exemplified by the stark contrast between a child eagerly leaping out of bed on Christmas morning and their reluctance to rise before a big test at school. Indeed, the effort-reward trade-off is critical for motivating our actions: we are willing to make more vigorous movements – i.e., move faster[1–5] or more forcefully,[6,7] make quicker decisions,[8–10] and perform more difficult cognitive tasks[11] – given the prospect of greater reward.

Changes to the effort-reward trade-off are thought to underlie the reduced movement vigor, termed bradykinesia, characteristic of Parkinson's disease (PD).[12–14] This is based on findings that people with PD retain the capacity to produce greater forces and move faster when sufficiently motivated.[14,15] People with PD are also less willing to produce the same magnitude of force, in exchange for a given reward, than neurotypical controls.[16–22] However, it remains unclear which of several potential mechanisms, each with unique implications for understanding bradykinesia, might underlie an impaired effort-reward trade-off in PD.

An impaired effort-reward trade-off could stem from reduced sensitivity to the rewards offered in exchange for effort production.[18,19] From this standpoint, bradykinesia could be remedied simply by increasing movement incentives. PD has long been thought to reduce reward sensitivity due to the overlap between its pathophysiology – degeneration of dopamine-producing cells in the substantia nigra[23–25] – and the role of midbrain dopamine circuits in reward processing.[26–28] In support of this view, people with PD have difficulty learning from reward feedback.[29–31] Yet, an effort-reward trade-off hinges on established (i.e., baseline) sensitivities to the value of potential movement outcomes rather than on learning novel reward values.[16–22] Many studies have used a decision-making task[32,33] to assess sensitivity to different reward values in people with PD.[34–37] Yet these studies have typically provided individuals with a continuous tally of their "earnings," allowing them to learn from past decisions. Thus, any differences in reward sensitivity between PD and controls may be confounded by differences in the ability to learn from reward outcomes. Whether PD specifically alters baseline reward sensitivity and how this may impact the effort-reward trade-off in this population remains unclear.

Aside from a reduction in reward sensitivity, PD could also cause movements to be misperceived as more effortful than they actually are, leading to erroneously reduced force

production (e.g., bradykinesia) and requiring greater rewards to compensate. Effort perception is typically tested in one of two ways. The first has participants match a prescribed force level, treating the sense of effort as a product of peripheral somatosensation and the central processing of ongoing motor commands.[38–42] The other method has participants retrospectively rate perceived effort following force production,[43–46] effectively assessing cognitive beliefs about one's effort. Interestingly, peripheral deafferentation does not impair force-matching performance despite altering cognitive beliefs about perceived exertion,[47] suggesting that these two approaches might assay distinct perceptual phenomena. While PD is known to impair somatosensation,[48–50] the impact on sensorimotor and cognitive perceptions of effort remains unknown.

Finally, rather than directly changing reward sensitivity or effort perception, PD could selectively distort the mapping between effort and reward. This would appear as a systematic difference in the rate at which people with PD are willing to increase effort production in response to rewards of increasing magnitude (i.e., a bias in the slope of the effort-reward relationship) compared to neurotypical controls. Prior work examining the effort-reward mapping has yielded mixed results,[16,17,22] perhaps because participants were required to produce forces throughout the experimental task and may have experienced muscular fatigue. Fatigue can induce a paradoxical increase in perceived effort despite decreases in actual force production,[39] creating the appearance of a distorted effort-reward mapping. Hence, it remains unclear exactly how PD biases the mapping between effort and reward.

Here, we systematically investigated three factors that could underlie an altered effort-reward trade-off in PD by comparing reward sensitivity, effort perception (assessed using both sensorimotor- and cognition-based measures), and the slope of the effort-reward relationship between individuals with PD and neurotypical control participants. Because dopamine has been implicated in reward sensitivity, we additionally examined the PD group on and off their dopaminergic medication. Our results showed that PD impairs effort perception, such that exerted forces were perceived as greater during both force matching and retrospective ratings of perceived exertion. In contrast, reward sensitivities and effort-reward relationship slopes were similar in the PD and control groups, regardless of medication status. These results add important context to prior findings of distorted effort-reward trade-offs in people with PD, and have critical implications for understanding and treating bradykinesia in this population.

Materials and methods

Participants

We recruited 44 participants with a diagnosis of idiopathic mild to moderate PD (Hoehn and Yahr[51] <=3) and 40 neurotypical controls matched for age (± 3 years) and sex. Participants were excluded if they had a deep brain stimulator implant, any neurologic diagnoses aside from PD, any psychiatric diagnoses unrelated to PD, or any significant orthopedic or cognitive impairments (<20 on the Montreal Cognitive Assessment[52,53]) that could interfere with testing. Participants were excluded due to an inability to complete all tasks (n=4), impaired cognition as measured by the MOCA (n=2), or were subsequently diagnosed with a Parkinson's plus syndrome after testing (n=2). This left a final sample of 37 participants with PD (11 female) and 39 controls (13 female). There were no differences in age between the final samples of PD and control participants (mean difference [95% HDI] = 0.07 [-3.3 3.4], $p_{difference}$ = 51.8%). Participant demographics are summarized in Table 1. All participants were paid $20/hour for study participation plus additional bonuses based on the rewards earned during the experiment. All participants provided written informed consent prior to the study, and all experimental procedures were approved by the Thomas Jefferson University Institutional Review Board.

Study procedure

Using a Kinarm Exoskeleton Robot (Kinarm, Canada), participants completed three tasks in the following order over a single experimental session: the force-matching, reward sensitivity, and effort-reward mapping tasks (detailed below). This fixed task order was chosen to mitigate muscular fatigue. Direct vision of the hand and arm were occluded in all tasks. Tasks were custom programmed in Simulink R2015a (Mathworks, United States) (see Data Availability Statement). Analyses were performed in MATLAB (v2022a) and Python (v3.13.5). Because motor symptoms are typically lateralized in PD, tasks were performed with the self-reported, more-affected arm, with the same arm used for the ON and OFF sessions. Control participants performed the tasks using the arm that matched the dominance/non-dominance of the tested arm of the PD participant to whom they were matched. In terms of dopamine-affecting medications, 34 participants with PD were taking a carbidopa/levodopa medication, of whom 5 were also taking a dopamine agonist. Of the

remaining 3, one participant was only taking MOA-B and COMT inhibitors, one was only taking a dopamine agonist, and the other was not taking any dopamine medication. Thirty-two participants with PD agreed to be tested in both the ON and OFF medicated states in separate sessions (testing order counterbalanced), with testing at least 18 days apart (mean ± SD = 50.1 ± 24.8 days). Two participants with PD completed only the OFF test: one was not on any medication, and the other was unable to return for the ON test. For the ON test, participants were tested 2.7 ± 2.9 hours after their most recent dose of dopaminergic medication. For the OFF test, participants were tested 14.8 ± 3.9 hours after their last dose, having skipped a single morning dose. Motor symptom severity as measured by Part III of the Movement Disorders Society Unified Parkinson's Disease Rating (UPDRS)[54] was greater for people with PD during the OFF phase (8.3 [5.4 11.5], $p_{difference}$ = 100.0%).

Table 1 Demographics

	Group	
	PD	**Control**
Age (years)	71.0 ± 7.0	70.9 ± 7.4
Race	W: 31 B: 3 A: 3	W: 34 B: 5 A: 0
Montreal Cognitive Assessment	26.1 ± 2.9	26.0 ± 2.1
UPDRS section III (motor section)	ON: 31.9 ± 13.5 OFF: 40.5 ± 13.3	
Hours since last dopamine medication dose	ON: 2.7 ± 2.9 OFF: 14.8 ± 3.9	
Levodopa equivalent dose	175.7 ± 82.4	
Months since diagnosis	68.2 ± 65.2	

Mean ± 1 SD. UPDRS: Unified Parkinson's disease rating scale

Reward sensitivity task

Procedure

To assess reward sensitivity, we adapted a utility task from the prior literature[32,33,55,56]. Participants were given the choice between a guaranteed monetary reward and a 50/50 chance of winning a different monetary reward (i.e., the certain and gamble options, respectively), displayed within 8 cm square targets 15 cm from a start circle (2 cm radius) on the right and left sides of the screen (Fig. 1A). On each trial, the certain and gamble options could appear in either target (side randomized). Visual feedback of the index fingertip was provided with a cursor (0.5 cm radius).

A trial began with the participants moving the cursor into the start circle. After a random delay (500-700ms), the two target options appeared on the screen. After another 1800ms delay, an auditory "go" signal cued participants to reach to their preferred option as quickly and accurately as possible. If a reach was initiated before the go signal, the targets were extinguished, and the trial was removed from analysis (4.5% of trials). Keeping these trials in the analysis did not change the results. The trial ended once the cursor remained inside a target for 500ms, or if no target was intersected within 6 seconds after the go signal (less than 0.1% of trials).

Participants completed 5 blocks, each containing 27 combinations of certain (10-50 cents) and gamble (10-100 cents) reward magnitudes in randomized order (135 trials total). To reduce fatigue, a rest break was provided after the second and fourth blocks. The result of the gamble, the running total of their earnings, or the number of remaining trials in the task were not displayed to participants, as this may have encouraged people to change their behavior based on previously observed gamble outcomes. Prior to the task, participants were informed of the 50/50 probability of the gamble options and that a portion of their earnings would be added to their study payment. To ensure they understood the task instructions, participants completed 5 practice trials before performing the task.

Data analysis

The expected values (EV) of the certain and gamble options were calculated for each trial:

$$EV = \sum p(R) * R \qquad (1)$$

where R represents the reward magnitude, and p represents the probability. The EV difference was calculated as the EV of the gamble option minus the certain option, and we recorded the number of times the gamble option was selected for each EV difference. We fit a logistic function with two free parameters to each participant's choice data using maximum likelihood estimation:

$$p_{choose\ gamble} = {}^{1}/_{(1 + e^{-(EV\ difference - PSE)/beta})} \qquad (2)$$

The point of subjective equality (PSE) represented the EV difference at which the participant was equally likely to select the certain and gamble options. The beta parameter reflects noise in the decisions. To ensure stable parameter fits, we used 60 initializations sampled from normal distributions centered on empirical estimates of the PSE and beta values. Fitting a

more traditional utility function to the data[32,33] resulted in less stable fits, but had no impact on the overall results.

PSE was taken as a measure of participants' general reward sensitivity. More positive PSE values indicated that participants required the gamble option to have greater expected values to overcome the uncertainty associated with choosing it. We also quantified the just noticeable difference (JND) as the change in expected value difference between the 75th and 25th percentiles of the logistic fit. The JND reflected the minimum required change in reward value for a participant to reliably alter their choice behavior. Greater JNDs indicated a lower sensitivity to changes in the EV difference between the certain and gamble options.

Participants were removed from this analysis if their choice data could not be reliably fit with a logistic function, i.e., their probability of choosing the gamble option was never above $p=0.75$ or below $p=0.25$ for all EV differences. We removed 4 control participants, 2 PD participants in both ON and OFF conditions, 3 PD participants in the ON condition only, and 3 PD participants in the OFF condition only. To confirm that removing these individuals did not bias our results, we also calculated the probability of choosing the gamble option on trials where the expected values of the two options were equal as a secondary measure of reward sensitivity.

Asking participants to move as quickly and accurately as possible after the go signal also allowed us to assess movement vigor during this task. Specifically, we calculated the peak velocity of the reach to the target. Because peak velocity is modulated by movement amplitude and the large targets afforded a range of reach amplitudes, we normalized peak velocity by reach amplitude. To determine whether reward magnitude affected the vigor of these reaching movements[3,57] we estimated the slope of a linear regression for each individual, with the total reward on offer in each trial as the predictor and normalized reach velocity as the outcome (see Supplementary Material).

Force matching task

Procedure

To assess sensorimotor and cognitive perceptions of effort, participants performed a force-matching task with retrospective effort ratings. This task involved an isometric elbow extension force, as people with PD have greater difficulty performing extension versus flexion movements.[58] First, we assessed participants' maximum voluntary contraction (MVC)

by asking them to extend their elbow and flex their triceps as hard as possible, keeping their shoulder and wrist relaxed. The average of 3 measurements was taken as the MVC. However, to avoid exceeding the maximum torque of the Kinarm robot's motors, the maximum force was set to 20 N for anyone whose actual MVC exceeded 20 N (28 PD ON, 26 PD OFF, and 38 control participants).

For each trial, participants first produced an initial force with the aid of visual feedback consisting of a rectangular bar of fixed height (Fig. 2a). On each trial, the target force level was mapped to the top of the bar, while its fill increased in proportion to the magnitude of force produced. Participants were instructed to "fill the bar" without exceeding the bar's height. The initial force was considered successful if participants maintained an isometric force magnitude within ±1 N of the target force for 1 s, or 5 s passed after exceeding a minimum force level (the target magnitude minus 5% of the maximum force). Participants rested for 2 s, then had 5 s to produce the match force, i.e., reproducing and maintaining the perceived initial force magnitude without visual feedback until the trial ended. Finally, participants rated the perceived effort of their exertion on a scale of 0 to 10, with 10 representing the greatest possible effort and 0 representing no effort. Participants completed 4 trials at each of 20%, 40%, 60%, and 80% of the maximum force (order randomized) for a total of 16 trials. One PD participant performed an earlier version of this task in which they were asked to match 50% of their max force, with all five force levels repeated three times (15 total trials). Participants completed two runs of the force-matching task. In the first run, participants performed the initial and match forces using the same arm (i.e., the more affected arm for the PD groups). In the second run, match forces were produced with the arm opposite the one used to produce the initial forces (see Supplementary Material).

To reduce fatigue, a brief rest break was provided halfway through each run of the force matching task. To ensure that participants understood the procedure, prior to the start of the task they performed one practice trial each at 25%, 50%, and 75% of the maximum force in order from smallest to largest, during which they received visual feedback during both the initial and match force production phases.

Data analysis

We calculated the initial and match forces as the mean elbow extension force produced when the force level reached a plateau (i.e., the longest stable period of force production). These plateaus were identified by visual inspection, as movement impairments (e.g., tremor) in

some participants prevented the use of a purely automated method. Trials were removed when the initial force never reached the goal threshold, defined as the goal force minus 5% of the max force (0.69% of all trials). Retaining these trials in the analysis did not change the results.

We used simple linear regressions to analyze these data. Goal forces, initial forces, match forces, and subjective ratings data were all mean-centered, allowing us to interpret regression intercepts as the outcome relative to the mean force level. We quantified initial force performance by regressing the initial forces against the goal forces for each participant. We quantified match force performance by regressing the match forces against the initial forces for each participant. We quantified subjective ratings by regressing the ratings against the initial forces for each participant. In all cases, a slope and intercept term from each regression was extracted for statistical analysis. The intercepts and slopes for the match forces were used to quantify the sensorimotor perception of effort, while the intercepts and slopes for the subjective ratings were used to quantify the cognitive perception of effort.

We also tested whether physical fatigue, as reflected in decreases in initial and/or match forces across trials, could have affected our results. We calculated the difference between either the initial or match force and the respective goal force, then regressed this difference against trial number for each participant. In both cases, more negative slopes would indicate a progressive decrease in force output across trials, suggesting greater fatigue.

Effort-reward mapping task

Procedure

To assess the mapping between effort and reward, participants performed a decision-making task adapted from the prior literature[17,18] in which they indicated their willingness to produce varying magnitudes of isometric elbow extension forces in exchange for varying monetary rewards. Forces were represented as a rectangular bar with a red line inside it (Fig. 3a). The top of the bar corresponded to 100% of the maximum force determined in the force-matching task, with the red line indicating the offered force magnitude in each trial (20%, 40%, 60%, 80%, and 100% of the max force). Five reward levels were presented (10¢, 20¢, 30¢, 40¢, and 50¢). Each combination of force and reward was presented randomly 5 times (125 trials total).

Participants pressed buttons to accept or reject each offer. To mitigate the chances of muscular fatigue impacting choice behavior,[59,60] participants did not immediately produce the offered force upon accepting. Instead, five trials were randomly selected from the pool of accepted offers to be performed at the end of the task; successful force production on each trial meant the offered monetary reward was added to their study payment. If participants accepted fewer than five offers, trials would be randomly selected from the pool of all possible offers. Prior to the task, participants were informed of the minimum and maximum reward and force levels.

To familiarize participants with each force level offered, participants produced each force level (except for the 100% level) before beginning the decision-making task. Additionally, participants completed 2 rounds of practice with the decision trials to familiarize them with the prospective nature of the task.

Data analysis

Using participants' choice behavior, we calculated the probability of accepting each combination of effort and reward values. Using these probabilities, we calculated the expected force (EF) that people were likely to accept at each reward level:

$$EF_R = \sum p(F_R) * F_R \qquad (3)$$

Where F = force magnitude, R = reward, and p = probability. We next regressed expected force against reward for each participant. The slope of this regression quantified the mapping between effort and reward, since it reflects the change in expected force that someone is willing to accept for a unit change in reward. Higher slopes indicated a greater willingness to exert additional effort for a unit change in reward.

Statistical analysis

All statistical analyses were performed in Python using the PyMC[61] (v5.23.0) and Bayes Toolbox.[62] We performed Bayesian t-tests for between-subject (PD ON vs controls, PD OFF vs controls) and within-subject (PD ON vs. PD OFF) comparisons for each outcome of interest.[63] Between-subjects comparisons assumed the data from an individual participant was generated from one of two t-distributions, depending on the group. Distributions for each group had a mu and sigma parameter, while a single nu parameter was shared between the groups. Within-subjects comparisons assumed the data were generated from a single t-

distribution with a single mu, sigma, and nu parameter. For all comparisons, we were most interested in the posterior distribution of the mu parameters. Using wide and uninformative priors for each model parameter to avoid undue influence on the posterior[64], we used Bayes' rule to estimate the posterior distributions. We used Markov Chain Monte Carlo sampling with 4 chains, 10,000 samples, and 2,000 tuning steps. We visually inspected the trace and the posterior predictive checks of the model against the data to ensure a reasonable model fit.[64]

For the between-subject comparisons, we subtracted the posterior distributions of the mu parameters. This distribution reflected the difference in means that most likely generated the data. The same interpretation can be applied to the posterior distribution of differences for the within-subject comparisons. We report the magnitude of the effect as the posterior mean and 95% high-density interval (HDI), i.e., the narrowest span of credible values that contains 95% of the distribution (i.e., the true difference falls within this range with 95% certainty). We also calculated the probability (reliability) of a difference as the percentage of the posterior distribution of differences that fall on one side of 0 ($p_{difference}$); higher values indicate a greater probability of a true difference. We considered both the magnitude and reliability of the effect together when interpreting our results, with $p_{difference}$ values >95% being considered sufficiently reliable. For each analysis, we were primarily interested in the effect of disease (PD ON versus controls) and the effect of medication state (PD ON versus PD OFF). The difference between PD OFF and controls is reported in the Supplementary Material. To make inferences about the similarity between groups, we also calculated a Bayes factor.[65–67]

Bayesian regressions were used to make inferences regarding the relationship between outcomes. Here, we assumed that each outcome (y) for each participant (i) was generated from a t-distribution with a mu, sigma, and nu parameter, where the mu parameter depends on the size of a predictor variable (x) and two beta parameters (β_0 and β_1) as follows:

$$y_i \sim StudentsT(\mu_i, \delta, \nu) \qquad (4)$$

$$\mu_i = \beta_0 + \beta_1 x \qquad (5)$$

For this analysis, we report standardized beta coefficients by performing the analysis on z-scored data (zbeta). We calculated the posterior in the same manner as the Bayesian t-tests and estimated the magnitude (95% HDI) and reliability ($p_{zbeta>0}$) of the effect.

Results

The goal of this study was to examine three potential factors contributing to effort-reward trade-off impairments in people with PD: 1) a reduced sensitivity to rewards, 2) an increased perception of effort, or 3) a biased effort-reward mapping. Our sample of people with PD was mildly bradykinetic according to the speed of their reaching movements relative to controls and moved even more slowly when OFF their dopaminergic replacement medication (see Supplementary Material).

Reward sensitivity was similar between people with PD and neurotypical controls

We tested the hypothesis that PD decreases reward sensitivity using a conventional decision-making task from the behavioral economics literature (Fig. 1a).[32,33,55,56] Reward sensitivity was quantified as the point of subjective equality (PSE) on a psychometric function fit to participants' choice data. PSE values were similar between the groups (Fig. 1c). The PSE difference between PD ON and controls (mean difference [95% HDI] = -0.1 cents [-3.6 3.7], $p_{difference}$ = 51.2%) and between PD ON and PD OFF (0.6 cents [-0.9 2.3], $p_{difference}$ = 82.4%) were both extremely small and unreliable (as was the difference between PD OFF and controls, see Supplementary Material). Using a Bayes factor (BF) analysis,[65,66] we found moderate evidence for equal PSE values between PD ON and controls (BF_{10} = 0.26) and between PD ON and PD OFF (BF_{10} = 0.28). We also observed no group differences in the degree to which movement vigor (i.e., reaching speed) changed across different magnitudes of reward on offer (Supplementary Fig. 1), consistent with the idea that people with PD did not respond differently to reward. Therefore, contrary to theories about PD and reward processing, we found that PD and dopamine availability did not reduce reward sensitivity.

Since a reduction in reward sensitivity could also manifest as greater noise in decision making between the certain and gamble options, we calculated the just noticeable difference (JND) (Fig. 1c). The difference in JND values between PD ON and controls (-2.7 cents [-7.3 1.7], $p_{difference}$ = 87.9%) and between PD ON and PD OFF (0.1 cents [-2.5 2.9], $p_{difference}$ = 55.3%) were both small and unreliable. Thus, we did not observe an impact of PD or dopamine availability on the sensitivity to changes in reward value.

Since psychometric functions could not be fit to 12 participants (see Methods), as a simpler measure of reward sensitivity, we also examined the probability of choosing the gamble

option when the gamble and fixed options had equal expected value. As with our primary analysis, differences in the probability of choosing the gamble option were small and not reliable (PD ON vs control = -0.05 [-0.21 0.11], $p_{difference}$ = 73.1%; PD ON vs PD OFF = 0.02 [-0.06 0.09], $p_{difference}$ = 65.7%). Likewise, Bayes Factor analysis revealed moderate evidence for equality between PD ON and controls (BF_{10} = 0.28) and between PD ON and PD OFF (BF_{10} = 0.23). Taken together, these analyses do not support the hypothesis that reward sensitivity is impaired in people with PD or that it is influenced by dopamine availability.

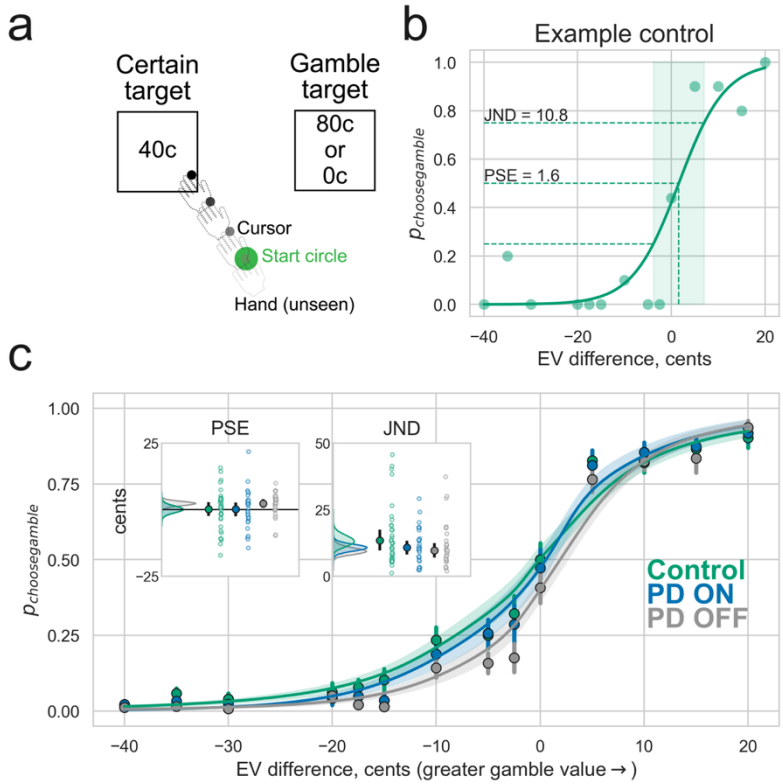

Fig. 1: Reward sensitivity. (a) Participants made decisions by reaching to either a certain target (guaranteed win) or a gamble target (50/50 chance of winning a monetary reward or nothing). We calculated expected value (EV) differences for each choice (Gamble – Certain). **(b)** Example control participant that was closest to the median of all PSEs. Empirical probability of choosing the gamble at each EV level (dots) along with the fitted logistic function (solid line). **(c)** The empirical data averaged across the participants and groups (circles) against the mean logistic functions created from individual parameter values. Error bars and shading represent 1 SEM. For this analysis, we removed 12 participants (4 controls, 8 PD: 3 ON only, 3 OFF only and 2 both ON and OFF) because their data could not be fit with a logistic function (see Methods). **(insets)** Kernel density estimates of the posterior means for each group's PSE and JND, plotted alongside the empirical group means (filled circles) and individual datapoints (open circles). Error bars are the 95% HDIs from each posterior distribution.

The sensorimotor perception of effort was greater in people with PD

We used a force matching task to assess a sensorimotor-based perception of effort (Fig. 2a). Initial forces were similar between groups (Fig. 2b & d). Although one of the intercept differences was reliable, we did not interpret it as meaningful given its small size (PD ON vs controls = -0.5% max force [-0.9 -0.1], $p_{difference}$ = 98.9%; PD ON vs PD OFF = 0.1% max force [-0.3 0.4], $p_{difference}$ = 68.6%). Slope differences were not large or reliable (PD ON vs controls = -0.001 [-0.01 0.01], $p_{difference}$ = 57.8%; PD ON vs PD OFF = 0.01 [-0.005 0.01], $p_{difference}$ = 84.7%).

Our primary measure of the sensorimotor-based perception of effort was the match force produced following each initial force. Our regression analysis showed the PD ON group exhibited much lower intercepts than the control group (Fig. 2e; -7.2% max force [-12.6 -1.8], $p_{difference}$ = 99.5%). While the PD ON condition had numerically greater intercepts compared to PD OFF, this difference was unreliable (2.5% max force [-0.9 5.9], $p_{difference}$ = 92.8%). To assess whether this increased effort perception was consistent across force levels, we examined the difference in regression slopes. Slope differences were unreliable for both comparisons (PD ON vs controls = -0.03 [-0.13 0.08], $p_{difference}$ = 68.8%; PD ON vs PD OFF = 0.05 [-0.01 0.10], $p_{difference}$ = 93.7%). Together, these results suggested that people with PD showed an increase in the sensorimotor-based perception of effort, which was not significantly impacted by dopamine availability.

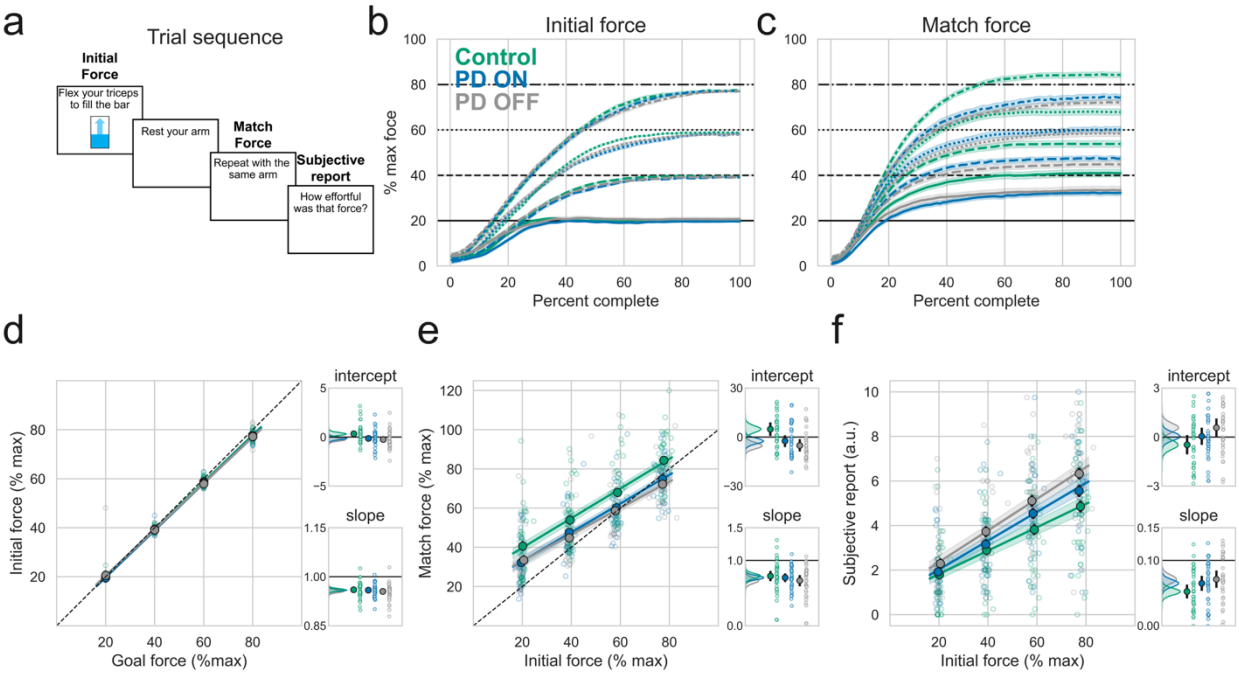

Fig. 2: Perception of effort. (a) Task sequence**.** Participants first produced an initial force guided by visual feedback to one of 4 different force levels (20, 40, 60, or 80% max force). After a brief rest, they were asked to produce the same force with the same arm, this time without any visual feedback. After resting, they were asked to rate how effortful the movement was on a scale of 0 to 10 (10 being their max effort and 0 being no effort). Mean initial **(b)** and match **(c)** force traces for each group (colors) and each goal force, denoted with different line styles. For the purposes of these plots, each participant's force trace data was averaged at each percentile so everyone had 100 data points. Shading represents the standard error of the mean (SEM), and horizontal black lines represent each goal force, with a different style line for each goal. **(d-f, main panels)** Each outcome of interest (y-axis) is plotted against the goal or initial forces. Solid circles with black outlines represent the overall group means, error bars (not visible for all datapoints) represent 1 SEM, and open circles represent individual participant means at each goal force level. The solid lines are the mean regression lines for each group, and shading represents 1 SEM. **(d-f, inset panels)** Kernel density estimates of the posterior distribution of group means for the intercepts (top) and slopes (bottom), calculated using Bayesian t-tests. The empirical group means (filled circles) and individual datapoints (open circles) are plotted to the right. Error bars for these plots (not visible for all datapoints) reflect the 95% HDIs for each respective posterior distribution. Note that the intercepts are calculated from the mean-centered data. **(d)** Performance on the initial forces, when the feedback was visible, was similar across groups and conditions. **(e)** Match forces were lower in the PD ON group than in controls, indicating an increase in the sensorimotor-based perception of effort. **(f)** Subjective effort ratings were higher and increased faster for the PD OFF group compared to controls (Supplementary Material).

It was important to test whether physical fatigue may have affected performance in this task. When examining progressive changes in produced forces over trials, we did not observe any differences in the pattern of initial forces produced between the PD ON and control groups (0.001 [-0.04 0.04], $p_{difference}$ = 52.9%) or between the PD ON and PD OFF conditions (0.02 [-0.02 0.06], $p_{difference}$ = 82.0%). The same was true of the match forces (PD ON vs controls = 0.16 [-0.31 0.61], $p_{difference}$ = 75.7%; PD ON vs PD OFF = -0.10 [-0.52 0.30], $p_{difference}$ = 69.5%). Therefore, we did not find evidence of group differences in or differences across the two PD medication states, suggesting that fatigue could not account for the differences in sensorimotor effort observed above.

The cognitive perception of effort was increased in people with PD

Cognitive perception of effort was measured by asking participants to retrospectively rate the level of effort they perceived to have exerted after each trial in the force-matching task (Fig. 2f). Our regression analysis showed greater intercepts and slopes for the PD ON group compared to controls, although these differences were unreliable (intercept difference = 0.5 [-0.2 1.3], $p_{difference}$ = 90.4%; slope difference = 0.01 [-0.003 0.03], $p_{difference}$ = 94.5%). Likewise, the intercepts and slopes were lower for the PD ON condition compared to PD OFF, but not reliably so (intercept difference = -0.5 [-1.0 0.2], $p_{difference}$ = 93.6%; slope difference = -0.01 [-0.02 0.01], $p_{difference}$ = 87.2%). Note that both the intercept and slope were reliably greater for the PD OFF group relative to controls (see Supplementary Material). Combined, these results suggested that having PD and being off dopamine medication together increased the cognitive perception of effort.

The mapping between effort and reward was similar between people with PD and neurotypical controls

Lastly, we tested the hypothesis that PD selectively biases the mapping between effort and reward (Fig. 3a). Across all combinations of force and reward levels, the PD ON group was slightly less likely to accept trials than controls, but this difference was unreliable (-0.05 [-0.11 0.02], $p_{difference}$ = 93.2%). There were no differences in acceptance rate between PD ON and PD OFF conditions (0.01 [-0.05 0.07], $p_{difference}$ = 62.5%). Thus, there was little difference in overall willingness across groups to accept effort in exchange for reward in this task.

In estimating the effort-reward mapping as the change in the expected force a participant was willing to exert per unit change in reward, we found no differences in slopes between the PD ON and control groups (0.05 [-0.25 0.34], $p_{difference}$ = 62.7%), or between PD ON and PD OFF (-0.02 [-0.23 0.19], $p_{difference}$ = 56.5%). Hence, people with PD, regardless of medication status, exhibited a similar willingness to increase effort per unit change in reward as control participants.

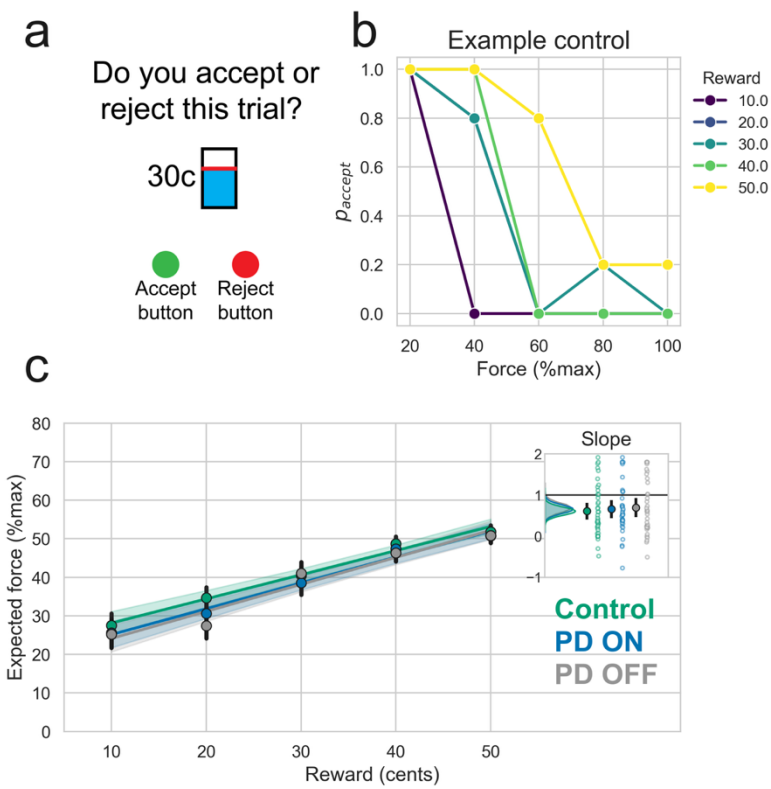

Fig. 3. The mapping between effort and reward. (a) Participants decided if they were willing to produce a prospective isometric elbow extension force for a monetary reward by pressing either the accept or reject button. Each combination of 5 different force and 5 different reward levels were presented 5 times each. We calculated the expected force a participant was willing to perform at each reward level. Then, we regressed the expected force against reward for each participant. **(b)** Example control subject that was closest to median slope of all participants. Dots represent the probability of accepting the offer at each force and reward level. **(c)** Average empirical expected forces at each reward level (circles) plotted against the mean regressions. Error bars and shading represent 1 SEM. **(Inset)** The slope for the regression was our primary measure of the effort-reward mapping. Kernel density estimates of the posterior mean distributions are plotted alongside the empirical group means (filled circles) and individual datapoints (open circles). Error bars for these plots reflect the 95% HDIs for each posterior mean distribution.

Cognitive perception of effort predicted the willingness to produce effort in exchange for reward

A key question was how performance across each of our experimental tasks was related. Thus, as a post hoc analysis, we examined whether reward sensitivity or the perception of effort influenced participants' overall willingness to exert effort in exchange for reward (i.e., the acceptance rate during the effort-reward mapping task). Reward sensitivity (PSE) was not related to acceptance rates in the effort-reward mapping task for any group (PD ON z-scored beta = 0.10 [-0.28 0.48], $p_{zbeta>0}$ = 70.9%; control zbeta = -0.06 [-0.41 0.29], $p_{zbeta>0}$ = 62.7%; PD OFF zbeta = -0.03 [-0.43 0.38], $p_{zbeta>0}$ = 55.5%).

For the perception of effort, lower force-matching intercepts (i.e., greater sensorimotor perceptions of effort) were associated with reduced acceptance rates during the effort-reward mapping task in the PD ON group (zbeta = 0.28 [-0.06 0.62], $p_{zbeta>0}$ = 94.9%) but this relationship was just under our reliability threshold. The relationship was unreliable for controls (zbeta = 0.19 [-0.13 0.54], $p_{zbeta>0}$ = 87.6%) and for PD OFF (zbeta = -0.03 [-0.37 0.34], $p_{zbeta>0}$ = 56.8%). In contrast, steeper slopes of the subjective effort ratings (i.e., greater cognitive perception of effort) were reliably related to lower acceptance rates during the effort-reward mapping task for both PD ON (zbeta = -0.47 [-0.78 -0.15], $p_{zbeta>0}$ = 99.7%) and controls (zbeta = -0.31 [-0.62 0.01] $p_{zbeta>0}$ = 97.2%). For the PD OFF group, this relationship was unreliable but remained in the same direction (zbeta = -0.20 [-0.57 0.15], $p_{zbeta>0}$ = 86.8%). Together, these results suggest that perceptions of effort (particularly as reported through subjective ratings) influence how people decide to trade off effort for reward.

Discussion

The effort-reward trade-off is critical for motivating our actions, and impairments in this trade-off have been proposed to underlie low movement vigor (i.e., bradykinesia) in people with PD. We assessed three potential sources of an effort-reward trade-off impairment by individually interrogating reward sensitivity, perceptions of effort, and the mapping between effort and reward. We found that effort perception was disrupted in people with PD compared to neurotypical controls. In contrast, we found no difference between people with PD and controls in either reward sensitivity or the effort-reward mapping, nor was dopaminergic medication status associated with these factors. Overall, our results suggest that changes to the perception of effort, but surprisingly not reduced reward sensitivity, may be an important contributor to reduced vigor in people with PD.

Effort perception has largely been measured using one of two methods: matching a previously experienced force magnitude[38,41,47,68–71] or rating perceived effort following force production.[43–46] The former method tests sensorimotor-based perceptions,[38–42] while the latter likely hinges on more cognitive processes. We observed that people with PD exhibited behavior consistent with perceptions of increased effort, relative to control participants, on both measures.

Disrupted sensorimotor-based perceptions of effort could arise from a primary sensory impairment or impaired central processing of an ongoing motor command.[38–42] Several

studies have reported reduced sensitivity to limb position changes in people with PD, consistent with a primary somatosensory deficit (for review, see[48]). However, such an impairment would make it difficult to sense changes in force magnitude, contradicting the reliable scaling of match force magnitudes with increasing initial force levels we observed here. Moreover, we observed no impairment when people with PD produced initial forces with the more impaired arm but matched them with the less impaired arm (Supplementary Fig. 2). Prior literature has also shown that people with PD can accurately scale isometric elbow flexion forces to produce instructed percentages of their voluntary maximum force in a task that was not constrained by the Kinarm robot's maximum force output.[72,73] A seeming discrepancy between the sensory and force production literature may stem from the fact that these sensations arise from distinct pathways: while the perception of limb position changes likely relies on signaling from muscle spindles (whose sensitivity is known to be disrupted in PD[74,75]), isometric force perception is mediated by Golgi tendon organs (GTOs).[40] It is currently unknown whether PD alters the processing of GTO signals, although the fact that people with PD can reliably scale their forces here and in prior studies suggests this is not the case.

Changes in sensorimotor effort perception could alternatively arise from disruptions in the central processing of the ongoing motor command. People with PD produced match forces that were consistently lower than those of control participants, suggesting a systematic bias in the perceived effort associated with them. Studies of analogous force-matching paradigms have shown that when asked to reproduce externally applied[42,76,77] or visually calibrated[38,78] forces using only reafferent somatosensation, neurologically healthy individuals systematically overshoot the target force. This is thought to reflect compensation for a centrally mediated perceptual attenuation of the predicted component of the feedback signal derived from motor commands. Notably, control participants showed this overproduction of matching forces in our study, whereas people with PD did not. PD-related disruptions of reafferent attenuation has been shown to increase with ratings of symptom severity and current dopaminergic medication dose.[79,80] Hence, disrupted effort perception in PD could stem from impaired reafference processing.

When asked to retrospectively rate their perceived effort following match force production, people with PD, particularly when OFF their dopaminergic medication, reported greater exertion than control participants. This aligns with our interpretation that lower match forces reflected an increase in perceived effort and is consistent with prior work showing that people

with PD rate the effort associated with everyday activities as greater than that of neurotypical controls.[46] We also found that effort perception ratings correlated with the overall willingness to exert effort in exchange for reward particularly for the PD ON and control groups, such that higher effort ratings were associated with reduced acceptance of effort-reward pairings in the trade-off task. These results suggest that while sensorimotor-based perceptions of effort are also impaired in PD, the effort-reward trade-off may depend more on cognitively mediated perceptions of effort.

While the effort-reward mapping was similar between people with PD and control participants, prior literature on the effort-reward trade-off in PD reports mixed results.[17–19,22,81]. A distinguishing feature of our study is that we did not require participants to immediately exert the forces they accepted after each decision, thereby reducing muscular fatigue throughout the task. Physical fatigue increases the perception of effort[60] and reduces the willingness to exert effort in exchange for reward,[59] and could account for the observed impairments in the effort-reward trade-off noted in prior studies. Moreover, while prior studies quantified the effort-reward mapping as the overall willingness to exert effort for reward, we instead quantified it as the change in expected force a participant was willing to exert per unit change in reward. These methodological differences could contribute to the mixed findings across studies.

We were surprised to find no evidence of reduced reward sensitivity in people with PD ON or OFF their dopaminergic medication relative to controls. We assessed reward sensitivity using a utility task commonly employed in the behavioral economics literature to quantify the perceived (subjective) value of a given monetary reward.[32,33,55,56] Our findings conflict with prior studies that have observed changes in reward sensitivity in people with PD using the same utility task,[34,35,37] and with physiological studies linking dopamine neuron activity to subjective reward value.[82–85] However, prior studies using utility tasks typically provided reward feedback immediately after each decision, which biases decisions and precludes acquiring a stable measure of reward sensitivity.[86,87] To avoid this confound, we did not provide decision feedback in our study. Notably, one prior study that similarly withheld decision feedback also found no reward sensitivity differences between people with PD and control participants.[21]

How can we reconcile our results with the well-established connection between dopamine signaling and reward during reinforcement learning,[88–91] and findings of dopamine-dependent impairments in reinforcement learning in people with PD?[29–31,92–94] First, we note that our

reward sensitivity task did not involve learning: each decision was independent, and no feedback was available as a teaching signal to update the values of subsequently presented choice options. Instead, our reward-sensitivity task focused on evaluating the perceived current value of a familiar reward. Dopamine signaling has been predominantly linked to reward in the context of learning, with recent work suggesting that dopamine can even serve as a value-free teaching signal.[95] In contrast, dopamine signaling might not be as critical for recalling the value assigned to already learned rewards.[96–98] Thus, dopamine-dependent reinforcement learning impairments in people with PD may reflect a problem with learning rather than baseline reward sensitivity. Additionally, a significant proportion of studies relating dopamine and reward focus on the ventral tegmental area and its connections to the prefrontal cortex,[83–85,88–90] whereas PD primarily affects the substantia nigra and regions of the basal ganglia that communicate with motor cortex, especially early in disease progression.[24,99–105] Hence, it is an oversimplification to conclude that, because dopamine signaling has been linked to reward and dopamine-producing neurons degenerate in PD, people with PD must have impaired reward sensitivity. Critically, people in earlier stages of PD still exhibit decreased movement vigor that is thought to reflect an underlying imbalance in the effort-reward trade-off. Our findings suggest that, for these individuals, bradykinesia may be driven by increased effort perception rather than decreased reward sensitivity.

In summary, we observed that people with PD show a selective increase in perceived effort relative to neurotypical controls, without a concomitant change in reward sensitivity or the mapping between effort and reward. We posit that biased effort perception underlies previous findings of altered effort-reward trade-offs in people with PD and contributes to the reduction in movement vigor (i.e., bradykinesia) commonly observed in this population. Indeed, people with PD frequently produce slower movements than neurotypical individuals unless sufficiently motivated,[14,15] and even then, their movement vigor does not respond to reward as effectively.[3] Our results imply that these effects on movement vigor stem from an underlying disruption of the perception of effort. Notably, this aligns with conventional rehabilitation approaches for PD, which aim to recalibrate people's perception of their own movements rather than providing greater incentives (e.g.,[106]). Our results suggest that further exploration of this approach may be beneficial when designing future treatments for people with PD.

Data availability

Simulink code to run the tasks on the Kinarm is available on the lab GitHub page (force-matching task: https://github.com/CML-lab/KINARM_Iso_Force_Matching; reward sensitivity task: https://github.com/CML-lab/KINARM_Utility_Test; effort-reward mapping task: https://github.com/CML-lab/KINARM_Effort_Reward_Mapping). All data and analysis code used for generating figures and conducting statistical analyses is available on https://osf.io/krw49.

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Competing interests

The authors report no competing interests.

Supplementary material

Supplementary material is available at *Brain* online.

References

1. Summerside EM, Shadmehr R, Ahmed AA. Vigor of reaching movements: reward discounts the cost of effort. *Journal of Neurophysiology*. 2018;119(6):2347-2357. doi:10.1152/jn.00872.2017

2. Shadmehr R, Reppert TR, Summerside EM, Yoon T, Ahmed AA. Movement Vigor as a Reflection of Subjective Economic Utility. *Trends in Neurosciences*. 2019;42(5):323-336. doi:10.1016/j.tins.2019.02.003

3. Manohar SG, Chong TTJ, Apps MAJ, et al. Reward Pays the Cost of Noise Reduction in Motor and Cognitive Control. *Current Biology*. 2015;25(13):1707-1716. doi:10.1016/j.cub.2015.05.038

4. Takikawa Y, Kawagoe R, Itoh H, Nakahara H, Hikosaka O. Modulation of saccadic eye movements by predicted reward outcome. *Exp Brain Res*. 2002;142(2):284-291. doi:10.1007/s00221-001-0928-1

5. Manohar SG, Finzi RD, Drew D, Husain M. Distinct Motivational Effects of Contingent and Noncontingent Rewards. *Psychol Sci*. 2017;28(7):1016-1026. doi:10.1177/0956797617693326

6. Marbaker RM, Schmad RC, Al-Ghamdi RA, Sukumar S, Ahmed AA. Reward invigorates isometric gripping actions. *Journal of Neurophysiology*. 2025;133(4):1282-1294. doi:10.1152/jn.00384.2024

7. Pessiglione M, Schmidt L, Draganski B, et al. How the Brain Translates Money into Force: A Neuroimaging Study of Subliminal Motivation. *Science*. 2007;316(5826):904-906. doi:10.1126/science.1140459

8. Hübner R, Schlösser J. Monetary reward increases attentional effort in the flanker task. *Psychon Bull Rev*. 2010;17(6):821-826. doi:10.3758/PBR.17.6.821

9. Krebs RM, Boehler CN, Egner T, Woldorff MG. The neural underpinnings of how reward associations can both guide and misguide attention. *J Neurosci*. 2011;31(26):9752-9759. doi:10.1523/JNEUROSCI.0732-11.2011

10. Viviani R, Messina I, Bosch JE, et al. Effects of genetic variability of CYP2D6 on neural substrates of sustained attention during on-task activity. *Transl Psychiatry*. 2020;10(1):338. doi:10.1038/s41398-020-01020-z

11. McGuigan S, Zhou SH, Brosnan MB, Thyagarajan D, Bellgrove MA, Chong TTJ. Dopamine restores cognitive motivation in Parkinson's disease. *Brain*. 2019;142(3):719-732. doi:10.1093/brain/awy341

12. England AC, Schwab RS. Parkinson's syndrome. *N Engl J Med*. 1961;265:837-844 concl. doi:10.1056/NEJM196110262651706

13. Herz DM, Brown P. Moving, fast and slow: behavioural insights into bradykinesia in Parkinson's disease. *Brain*. 2023;146(9):3576-3586. doi:10.1093/brain/awad069

14. Mazzoni P, Hristova A, Krakauer JW. Why Don't We Move Faster? Parkinson's Disease, Movement Vigor, and Implicit Motivation. *J Neurosci*. 2007;27(27):7105-7116. doi:10.1523/JNEUROSCI.0264-07.2007

15. Schmidt L, d'Arc BF, Lafargue G, et al. Disconnecting force from money: effects of basal ganglia damage on incentive motivation. *Brain*. 2008;131(5):1303-1310. doi:10.1093/brain/awn045

16. Banwinkler M, Dzialas V, Rigoux L, et al. Putaminal dopamine modulates movement motivation in Parkinson's disease. *Brain*. 2024;147(10):3352-3357. doi:10.1093/brain/awae214

17. Chong TTJ, Bonnelle V, Manohar S, et al. Dopamine enhances willingness to exert effort for reward in Parkinson's disease. *Cortex*. 2015;69:40-46. doi:10.1016/j.cortex.2015.04.003

18. Costello H, Yamamori Y, Kieslich K, et al. Impaired reward sensitivity in Parkinson's depression is unresponsive to dopamine treatment. *Brain*. 2025;148(6):2122-2134. doi:10.1093/brain/awaf098

19. Le Heron C, Plant O, Manohar S, et al. Distinct effects of apathy and dopamine on effort-based decision-making in Parkinson's disease. *Brain*. 2018;141(5):1455-1469. doi:10.1093/brain/awy110

20. Morris LA, Suzuki H, Lee S, et al. Apathy, effort-based decisions and brain integrity in Alzheimer's and Parkinson's diseases. *Brain*. Published online October 17, 2025:awaf393. doi:10.1093/brain/awaf393

21. Nobis L, Maio MR, Saleh Y, et al. Role of serotonin in modulation of decision-making in Parkinson's disease. *J Psychopharmacol*. 2023;37(4):420-431. doi:10.1177/02698811221144636

22. Zénon A, Duclos Y, Carron R, et al. The human subthalamic nucleus encodes the subjective value of reward and the cost of effort during decision-making. *Brain*. 2016;139(6):1830-1843. doi:10.1093/brain/aww075

23. Ehringer H, Hornykiewicz O. [Distribution of noradrenaline and dopamine (3-hydroxytyramine) in the human brain and their behavior in diseases of the extrapyramidal system]. *Klin Wochenschr*. 1960;38:1236-1239. doi:10.1007/BF01485901

24. Fearnley JM, Lees AJ. AGEING AND PARKINSON'S DISEASE: SUBSTANTIA NIGRA REGIONAL SELECTIVITY. *Brain*. 1991;114(5):2283-2301. doi:10.1093/brain/114.5.2283

25. Marsden CD. Parkinson's disease. *The Lancet*. 1990;335(8695):948-949. doi:10.1016/0140-6736(90)91006-V

26. Schultz W. Behavioral theories and the neurophysiology of reward. *Annu Rev Psychol*. 2006;57:87-115. doi:10.1146/annurev.psych.56.091103.070229

27. Daw ND, O'Doherty JP, Dayan P, Seymour B, Dolan RJ. Cortical substrates for exploratory decisions in humans. *Nature*. 2006;441(7095):876-879. doi:10.1038/nature04766

28. Steinberg EE, Keiflin R, Boivin JR, Witten IB, Deisseroth K, Janak PH. A causal link between prediction errors, dopamine neurons and learning. *Nat Neurosci*. 2013;16(7):966-973. doi:10.1038/nn.3413

29. Frank MJ, Seeberger LC, O'Reilly RC. By Carrot or by Stick: Cognitive Reinforcement Learning in Parkinsonism. *Science*. Published online December 10, 2004. doi:10.1126/science.1102941

30. Pessiglione M, Seymour B, Flandin G, Dolan RJ, Frith CD. Dopamine-dependent prediction errors underpin reward-seeking behaviour in humans. *Nature*. 2006;442(7106):1042-1045. doi:10.1038/nature05051

31. Shiner T, Seymour B, Wunderlich K, et al. Dopamine and performance in a reinforcement learning task: evidence from Parkinson's disease. *Brain*. 2012;135(6):1871-1883. doi:10.1093/brain/aws083

32. Bernoulli D. Exposition of a New Theory on the Measurement of Risk. *Econometrica*. 1954;22(1):23-36. doi:10.2307/1909829

33. Von Neumann J, Morgenstern O. *Theory of Games and Economic Behavior*. Princeton University Press; 1944:xviii, 625.

34. Kobayashi S, Asano K, Matsuda N, Ugawa Y. Dopaminergic influences on risk preferences of Parkinson's disease patients. *Cogn Affect Behav Neurosci*. 2019;19(1):88-97. doi:10.3758/s13415-018-00646-3

35. Djamshidian A, Jha A, O'Sullivan SS, et al. Risk and learning in impulsive and nonimpulsive patients with Parkinson's disease. *Movement Disorders*. 2010;25(13):2203-2210. doi:10.1002/mds.23247

36. Sharp ME, Viswanathan J, McKeown MJ, Appel-Cresswell S, Stoessl AJ, Barton JJS. Decisions under risk in Parkinson's disease: Preserved evaluation of probability and magnitude. *Neuropsychologia*. 2013;51(13):2679-2689. doi:10.1016/j.neuropsychologia.2013.08.008

37. Cherkasova MV, Corrow JC, Taylor A, et al. Dopamine replacement remediates risk aversion in Parkinson's disease in a value-independent manner. *Parkinsonism & Related Disorders*. 2019;66:189-194. doi:10.1016/j.parkreldis.2019.08.014

38. Logan LM, Semrau JA, Cluff T, Scott SH, Dukelow SP. Effort matching between arms depends on relative limb geometry and personal control. *Journal of Neurophysiology*. 2019;121(2):459-470. doi:10.1152/jn.00346.2018

39. Proske U, Allen T. The neural basis of the senses of effort, force and heaviness. *Exp Brain Res*. 2019;237(3):589-599. doi:10.1007/s00221-018-5460-7

40. Proske U, Gandevia SC. The Proprioceptive Senses: Their Roles in Signaling Body Shape, Body Position and Movement, and Muscle Force. *Physiological Reviews*. 2012;92(4):1651-1697. doi:10.1152/physrev.00048.2011

41. Scotland S, Adamo DE, Martin BJ. Sense of effort revisited: Relative contributions of sensory feedback and efferent copy. *Neuroscience Letters*. 2014;561:208-212. doi:10.1016/j.neulet.2013.12.041

42. Shergill SS, Bays PM, Frith CD, Wolpert DM. Two Eyes for an Eye: The Neuroscience of Force Escalation. *Science*. 2003;301(5630):187-187. doi:10.1126/science.1085327

43. Borg G a. V. Psychophysical bases of perceived exertion. *Medicine & Science in Sports & Exercise*. 1982;14(5):377.

44. D. Egan A, B. Winchester J, Foster C, R. McGuigan M. Using Session RPE to Monitor Different Methods of Resistance Exercise. *J Sports Sci Med*. 2006;5(2):289-295.

45. Padmanabhan P, Casamento-Moran A, Kim A, et al. Dopamine facilitates the translation of physical exertion into assessments of effort. *npj Parkinsons Dis*. 2023;9(1):51. doi:10.1038/s41531-023-00490-4

46. Solomon NP, Robin DA. Perceptions of effort during handgrip and tongue elevation in Parkinson's disease. *Parkinsonism & Related Disorders*. 2005;11(6):353-361. doi:10.1016/j.parkreldis.2005.06.004

47. Lafargue G, Paillard J, Lamarre Y, Sirigu A. Production and perception of grip force without proprioception: is there a sense of effort in deafferented subjects? *Eur J of Neuroscience*. 2003;17(12):2741-2749. doi:10.1046/j.1460-9568.2003.02700.x

48. Conte A, Khan N, Defazio G, Rothwell JC, Berardelli A. Pathophysiology of somatosensory abnormalities in Parkinson disease. *Nat Rev Neurol*. 2013;9(12):687-697. doi:10.1038/nrneurol.2013.224

49. Avanzino L, Fiorio M, Conte A. Actual and Illusory Perception in Parkinson's Disease and Dystonia: A Narrative Review. *Front Neurol*. 2018;9:584. doi:10.3389/fneur.2018.00584

50. Demirci M, Grill S, McShane L, Hallett M. A mismatch between kinesthetic and visual perception in Parkinson's disease. *Annals of Neurology*. 1997;41(6):781-788. doi:10.1002/ana.410410614

51. Hoehn MM, Yahr MD. Parkinsonism: onset, progression and mortality. *Neurology*. 1967;17(5):427-442. doi:10.1212/wnl.17.5.427

52. Nasreddine ZS, Phillips NA, Bédirian V, et al. The Montreal Cognitive Assessment, MoCA: A Brief Screening Tool For Mild Cognitive Impairment. *Journal of the American Geriatrics Society*. 2005;53(4):695-699. doi:10.1111/j.1532-5415.2005.53221.x

53. Hoops S, Nazem S, Siderowf AD, et al. Validity of the MoCA and MMSE in the detection of MCI and dementia in Parkinson disease. *Neurology*. 2009;73(21):1738-1745. doi:10.1212/WNL.0b013e3181c34b47

54. Goetz CG, Tilley BC, Shaftman SR, et al. Movement Disorder Society-sponsored revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS): Scale presentation and clinimetric testing results. *Movement Disorders*. 2008;23(15):2129-2170. doi:10.1002/mds.22340

55. Mosteller F, Nogee P. An Experimental Measurement of Utility. *Journal of Political Economy*. 1951;59(5):371-404.

56. Coombs CH, Komorita SS. Measuring Utility of Money through Decisions. *The American Journal of Psychology*. 1958;71(2):383-389. doi:10.2307/1420083

57. Sukumar S, Shadmehr R, Ahmed AA. Effects of reward and effort history on decision making and movement vigor during foraging. *Journal of Neurophysiology*. 2024;131(4):638-651. doi:10.1152/jn.00092.2023

58. Robichaud JA, Pfann KD, Comella CL, Brandabur M, Corcos DM. Greater impairment of extension movements as compared to flexion movements in Parkinson's disease. *Exp Brain Res*. 2004;156(2):240-254. doi:10.1007/s00221-003-1782-0

59. Müller T, Klein-Flügge MC, Manohar SG, Husain M, Apps MAJ. Neural and computational mechanisms of momentary fatigue and persistence in effort-based choice. *Nat Commun*. 2021;12(1):4593. doi:10.1038/s41467-021-24927-7

60. Hogan PS, Chen SX, Teh WW, Chib VS. Neural mechanisms underlying the effects of physical fatigue on effort-based choice. *Nat Commun*. 2020;11(1):4026. doi:10.1038/s41467-020-17855-5

61. Salvatier J, Wiecki TV, Fonnesbeck C. Probabilistic programming in Python using PyMC3. *PeerJ Comput Sci*. 2016;2:e55. doi:10.7717/peerj-cs.55

62. Kim HE. bayes-toolbox: A Python package for Bayesian statistics. *Journal of Open Source Software*. 2023;8(90):5526. doi:10.21105/joss.05526

63. Kruschke JK. Bayesian estimation supersedes the t test. *Journal of Experimental Psychology: General*. 2013;142:573-603. doi:10.1037/a0029146

64. Kruschke J. *Doing Bayesian Data Analysis: A Tutorial with R, JAGS, and Stan*. Elsevier Science; 2014. https://books.google.com/books?id=FzvLAwAAQBAJ

65. Jeffreys the late H. *Theory of Probability*. Third Edition, Third Edition. Oxford University Press; 1998.

66. Rouder JN, Speckman PL, Sun D, Morey RD, Iverson G. Bayesian t tests for accepting and rejecting the null hypothesis. *Psychonomic Bulletin & Review*. 2009;16(2):225-237. doi:10.3758/PBR.16.2.225

67. Linde M, Tendeiro JN, Selker R, Wagenmakers EJ, van Ravenzwaaij D. Decisions about equivalence: A comparison of TOST, HDI-ROPE, and the Bayes factor. *Psychol Methods*. 2023;28(3):740-755. doi:10.1037/met0000402

68. Brooks J, Allen TJ, Proske U. The senses of force and heaviness at the human elbow joint. *Exp Brain Res*. 2013;226(4):617-629. doi:10.1007/s00221-013-3476-6

69. Carson RG, Riek S, Shahbazpour N. Central and peripheral mediation of human force sensation following eccentric or concentric contractions. *The Journal of Physiology*. 2002;539(3):913-925. doi:10.1113/jphysiol.2001.013385

70. Jones LA, Hunter IW. Force sensation in isometric contractions: a relative force effect. *Brain Research*. 1982;244(1):186-189. doi:10.1016/0006-8993(82)90919-2

71. McCloskey DI, Ebeling P, Goodwin GM. Estimation of weights and tensions and apparent involvement of a "sense of effort." *Experimental Neurology*. 1974;42(1):220-232. doi:10.1016/0014-4886(74)90019-3

72. Stelmach GE, Teasdale N, Phillips J, Worringham CJ. Force production characteristics in Parkinson's disease. *Exp Brain Res*. 1989;76(1):165-172. doi:10.1007/BF00253633

73. Stelmach GE, Worringham CJ. The preparation and production of isometric force in Parkinson's disease. *Neuropsychologia*. 1988;26(1):93-103. doi:10.1016/0028-3932(88)90033-4

74. Bartels EM, Harrison A. Muscle spindle receptors and their impact on Parkinson´s disease and Cerebral Palsy subjects. *J Muscle Res Cell Motil*. 2025;46(1):1-8. doi:10.1007/s10974-024-09682-8

75. Dietrichson P. The Role of the Fusimotor System in Spasticity and Parkinsonian Rigidity. In: Desmedt JE, ed. *Human Reflexes, Pathophysiology of Motor Systems, Methodology of Human Reflexes*. Vol 3. S.Karger AG; 1973:0. doi:10.1159/000394163

76. Bays PM, Wolpert DM. Computational principles of sensorimotor control that minimize uncertainty and variability. *The Journal of Physiology*. 2007;578(2):387-396. doi:10.1113/jphysiol.2006.120121

77. Blakemore SJ, Wolpert DM, Frith CD. Central cancellation of self-produced tickle sensation. *Nat Neurosci*. 1998;1(7):635-640. doi:10.1038/2870

78. Therrien AS, Richardson BA, Balasubramaniam R. Continuous theta-burst stimulation to primary motor cortex reduces the overproduction of forces following removal of visual feedback. *Neuropsychologia*. 2011;49(10):2941-2946. doi:10.1016/j.neuropsychologia.2011.06.023

79. Macerollo A, Chen JC, Korlipara P, et al. Dopaminergic treatment modulates sensory attenuation at the onset of the movement in Parkinson's disease: A test of a new framework for bradykinesia. *Movement Disorders*. 2016;31(1):143-146. doi:10.1002/mds.26493

80. Wolpe N, Zhang J, Nombela C, Ingram JN, Wolpert DM, Rowe JB. Sensory attenuation in Parkinson's disease is related to disease severity and dopamine dose. *Sci Rep*. 2018;8(1):15643. doi:10.1038/s41598-018-33678-3

81. Le Bouc R, Rigoux L, Schmidt L, et al. Computational Dissection of Dopamine Motor and Motivational Functions in Humans. *J Neurosci*. 2016;36(25):6623-6633. doi:10.1523/JNEUROSCI.3078-15.2016

82. Schultz W, Stauffer WR, Lak A. The phasic dopamine signal maturing: from reward via behavioural activation to formal economic utility. *Current Opinion in Neurobiology*. 2017;43:139-148. doi:10.1016/j.conb.2017.03.013

83. Stauffer WR, Lak A, Schultz W. Dopamine Reward Prediction Error Responses Reflect Marginal Utility. *Current Biology*. 2014;24(21):2491-2500. doi:10.1016/j.cub.2014.08.064

84. Lak A, Stauffer WR, Schultz W. Dopamine prediction error responses integrate subjective value from different reward dimensions. *Proceedings of the National Academy of Sciences*. 2014;111(6):2343-2348. doi:10.1073/pnas.1321596111

85. Hill DF, Hickman RW, Al-Mohammad A, Stasiak A, Schultz W. Dopamine neurons encode trial-by-trial subjective reward value in an auction-like task. *Nat Commun*. 2024;15(1):8138. doi:10.1038/s41467-024-52311-8

86. Nasioulas A, Potier E, Cerrotti F, Lebreton M, Palminteri S. Feedback-induced attitudinal changes in risk preferences. *Nat Commun*. 2026;17(1):992. doi:10.1038/s41467-025-67729-x

87. Tymula A, Wang X, Imaizumi Y, et al. Dynamic prospect theory: Two core decision theories coexist in the gambling behavior of monkeys and humans. *Science Advances*. 2023;9(20):eade7972. doi:10.1126/sciadv.ade7972

88. Ramaswamy A, Steele D, Roiser JP, et al. Electrophysiological Correlates of Reinforcement Learning in the Human Ventral Tegmental Area. *bioRxiv*. Preprint posted online January 27, 2026:2026.01.27.701696. doi:10.64898/2026.01.27.701696

89. Schultz W. Getting Formal with Dopamine and Reward. *Neuron*. 2002;36(2):241-263. doi:10.1016/S0896-6273(02)00967-4

90. Schultz W, Dayan P, Montague PR. A Neural Substrate of Prediction and Reward. *Science*. 1997;275(5306):1593-1599. doi:10.1126/science.275.5306.1593

91. Zaghloul KA, Blanco JA, Weidemann CT, et al. Human substantia nigra neurons encode unexpected financial rewards. *Science*. 2009;323(5920):1496-1499. doi:10.1126/science.1167342

92. Cools R, Altamirano L, D'Esposito M. Reversal learning in Parkinson's disease depends on medication status and outcome valence. *Neuropsychologia*. 2006;44(10):1663-1673. doi:10.1016/j.neuropsychologia.2006.03.030

93. Costello H, Berry AJ, Reeves S, et al. Disrupted reward processing in Parkinson's disease and its relationship with dopamine state and neuropsychiatric syndromes: a systematic review and meta-analysis. *J Neurol Neurosurg Psychiatry*. 2022;93(5):555-562. doi:10.1136/jnnp-2021-327762

94. Smittenaar P, Chase HW, Aarts E, Nusselein B, Bloem BR, Cools R. Decomposing effects of dopaminergic medication in Parkinson's disease on probabilistic action selection – learning or performance? *European Journal of Neuroscience*. 2012;35(7):1144-1151. doi:10.1111/j.1460-9568.2012.08043.x

95. Greenstreet F, Vergara HM, Johansson Y, et al. Dopaminergic action prediction errors serve as a value-free teaching signal. *Nature*. 2025;643(8074):1333-1342. doi:10.1038/s41586-025-09008-9

96. Hollon NG, Arnold MM, Gan JO, Walton ME, Phillips PEM. Dopamine-associated cached values are not sufficient as the basis for action selection. *Proc Natl Acad Sci U S A*. 2014;111(51):18357-18362. doi:10.1073/pnas.1419770111

97. Sharpe MJ, Schoenbaum G. Evaluation of the hypothesis that phasic dopamine constitutes a cached-value signal. *Neurobiology of Learning and Memory*. 2018;153:131-136. doi:10.1016/j.nlm.2017.12.002

98. Sharpe MJ, Batchelor HM, Schoenbaum G. Preconditioned cues have no value. Behrens TE, ed. *eLife*. 2017;6:e28362. doi:10.7554/eLife.28362

99. Alexander GE, DeLong MR, Strick PL. Parallel Organization of Functionally Segregated Circuits Linking Basal Ganglia and Cortex. *Annual Review of Neuroscience*. 1986;9(1):357-381. doi:10.1146/annurev.ne.09.030186.002041

100. Morrish PK, Sawle GV, Brooks DJ. Clinical and [18F] dopa PET findings in early Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*. 1995;59(6):597-600. doi:10.1136/jnnp.59.6.597

101. Vaillancourt DE, Spraker MB, Prodoehl J, et al. High-resolution diffusion tensor imaging in the substantia nigra of de novo Parkinson disease. *Neurology*. 2009;72(16):1378-1384. doi:10.1212/01.wnl.0000340982.01727.6e

102. Du G, Lewis MM, Sen S, et al. Imaging nigral pathology and clinical progression in Parkinson's disease. *Movement Disorders*. 2012;27(13):1636-1643. doi:10.1002/mds.25182

103. Morrish PK, Sawle GV, Brooks DJ. An [18F]dopa-PET and clinical study of the rate of progression in Parkinson's disease. *Brain*. 1996;119 (Pt 2):585-591. doi:10.1093/brain/119.2.585

104. Strick PL, Dum RP, Picard N. Macro-organization of the Circuits Connecting the Basal Ganglia with the Cortical Motor Areas. In: Houk JC, Davis JL, Beiser DG, eds. *Models of Information Processing in the Basal Ganglia*. The MIT Press; 1994:117-130. doi:10.7551/mitpress/4708.003.0011

105. Leh SE, Ptito A, Chakravarty MM, Strafella AP. Fronto-striatal connections in the human brain: A probabilistic diffusion tractography study. *Neurosci Lett*. 2007;419(2):113-118. doi:10.1016/j.neulet.2007.04.049

106. Fox C, Ebersbach G, Ramig L, Sapir S. LSVT LOUD and LSVT BIG: Behavioral Treatment Programs for Speech and Body Movement in Parkinson Disease. *Parkinsons Dis*. 2012;2012:391946. doi:10.1155/2012/391946

Figure legends

Figure 1: Reward sensitivity. (a) Participants made decisions by reaching to either a certain target (guaranteed win) or a gamble target (50/50 chance of winning a monetary reward or nothing). We calculated expected value (EV) differences for each choice (Gamble – Certain). **(b)** Example control participant that was closest to the median of all PSEs. Empirical probability of choosing the gamble at each EV level (dots) along with the fitted logistic function (solid line). **(c)** The empirical data averaged across the participants and groups (circles) against the mean logistic functions created from individual parameter values. Error

bars and shading represent 1 SEM. For this analysis, we removed 12 participants (4 controls, 8 PD: 3 ON only, 3 OFF only and 2 both ON and OFF) because their data could not be fit with a logistic function (see Methods). **(insets)** Kernel density estimates of the posterior means for each group's PSE and JND, plotted alongside the empirical group means (filled circles) and individual datapoints (open circles). Error bars are the 95% HDIs from each posterior distribution.

Figure 2: Perception of effort. (a) Task sequence**.** Participants first produced an initial force guided by visual feedback to one of 4 different force levels (20, 40, 60, or 80% max force). After a brief rest, they were asked to produce the same force with the same arm, this time without any visual feedback. After resting, they were asked to rate how effortful the movement was on a scale of 0 to 10 (10 being their max effort and 0 being no effort). Mean initial **(b)** and match **(c)** force traces for each group (colors) and each goal force, denoted with different line styles. For the purposes of these plots, each participant's force trace data was averaged at each percentile so everyone had 100 data points. Shading represents the standard error of the mean (SEM), and horizontal black lines represent each goal force, with a different style line for each goal. **(d-f, main panels)** Each outcome of interest (y-axis) is plotted against the goal or initial forces. Solid circles with black outlines represent the overall group means, error bars (not visible for all datapoints) represent 1 SEM, and open circles represent individual participant means at each goal force level. The solid lines are the mean regression lines for each group, and shading represents 1 SEM. **(d-f, inset panels)** Kernel density estimates of the posterior distribution of group means for the intercepts (top) and slopes (bottom), calculated using Bayesian t-tests. The empirical group means (filled circles) and individual datapoints (open circles) are plotted to the right. Error bars for these plots (not visible for all datapoints) reflect the 95% HDIs for each respective posterior distribution. Note that the intercepts are calculated from the mean-centered data. **(d)** Performance on the initial forces, when the feedback was visible, was similar across groups and conditions. **(e)** Match forces were lower in the PD ON group than in controls, indicating an increase in the sensorimotor-based perception of effort. **(f)** Subjective effort ratings were higher and increased faster for the PD OFF group compared to controls (Supplementary Material).

Figure 3. The mapping between effort and reward. (a) Participants decided if they were willing to produce a prospective isometric elbow extension force for a monetary reward by pressing either the accept or reject button. Each combination of 5 different force and 5 different reward levels were presented 5 times each. We calculated the expected force a participant was willing to perform at each reward level. Then, we regressed the expected force against reward for each participant. **(b)** Example control subject that was closest to median slope of all participants. Dots represent the probability of accepting the offer at each force and reward level. **(c)** Average empirical expected forces at each reward level (circles) plotted against the mean regressions. Error bars and shading represent 1 SEM. **(Inset)** The slope for the regression was our primary measure of the effort-reward mapping. Kernel density estimates of the posterior mean distributions are plotted alongside the empirical group means (filled circles) and individual datapoints (open circles). Error bars for these plots reflect the 95% HDIs for each posterior mean distribution.

Supplementary Figure 1. Reward Vigor. The main plot depicts the group averaged normalized peak velocity plotted at each reward level (both z-scored). Each dot represents the mean of each group, and error bars are 1 standard error of the mean. Lines and shading represent the mean regression line and 1 standard error of the mean, respectively. Insets show the intercept and slopes of the participant level regressions. Kernel density estimates of the posterior mean distributions plotted alongside the empirical group means (filled circles) and individual datapoints (open circles). Error bars for these plots reflect the 95% HDIs for each posterior mean distribution.

Supplementary Figure 2. Perception of effort with the less impaired arm. Mean initial (a) and match (b) force traces for each group (colors) and each goal force, denoted with different line styles. For the purposes of these plots, each participant's force trace data was averaged at each percentile so everyone would have 100 data points. Shading represents standard error of the mean, horizontal black lines represent each goal force, with a different style line for each goal. (c-e, main panels) Each outcome of interest (y-axis) plotted against the goal or initial forces. Solid circles with black outlines represent the overall group means, error bars (not visible for all datapoints) represent 1 standard error of the mean (SEM), and open circles represent individual participant means at each goal force level. The solid lines are the mean

regression lines for each group and shading represents 1 SEM. (c-e, inset panels) Kernel density estimates of the posterior distribution of group means for the intercepts (top) and slopes (bottom), calculated using Bayesian t-tests. The empirical group means (filled circles) and individual datapoints (open circles) are plotted to the right. Error bars for these plots (not visible for all datapoints) reflect the 95% HDIs for each respective posterior distribution. Note that the intercepts are calculated from the mean-centered data. (c) Performance on the initial forces, when the feedback was visible, was similar across groups and conditions. (d) Match forces were similar for all groups and conditions. (e) Subjective effort ratings were higher for the PD OFF group compared to controls.