JNeurosci
THE JOURNAL OF NEUROSCIENCE

**Research Articles | Behavioral/Cognitive**

# The balance N1 attenuates but persists during self-initiated and repeated perturbations, even without active balance control

https://doi.org/10.1523/JNEUROSCI.1028-26.2026

Received: 17 June 2026
Revised: 26 August 2026
Accepted: 27 September 2026

Copyright © 2026 the authors

*This Early Release article has been peer reviewed and accepted, but has not been through the composition and copyediting processes.The final version may differ slightly in style or formatting and will contain links to any extended data.*

**Alerts:** Sign up at www.jneurosci.org/alerts to receive customized email alerts when the fully formatted version of this article is published.

# The balance N1 attenuates but persists during self-initiated and repeated perturbations, even without active balance control

Running title: N1 tracks uncertainty yet persists without control

Key words: balance N1, sensorimotor uncertainty, EEG, habituation, self-initiation

Lucas Mensink[1], Daphne N. R. Jansen[1], Monika Moraczewska[1], Matto Leeuwis[1], Johan Pel[1], Patrick A. Forbes[1]

[1]Department of Neuroscience, Erasmus MC, University Medical Center Rotterdam, 3015 GD Rotterdam, The Netherlands

Number of pages: 32
Number of figures: 4
Number of words abstract: 226
Number of words introduction: 650
Number of words discussion: 1498

Conflict of interest: the authors have no relevant interests to disclose

Funding: This study was supported by the Dutch Research Council (NWO) Exacte en Natuurwetenschappen, VI.Vidi.203.066 (to Patrick A. Forbes).

Corresponding author:

Patrick A. Forbes
Department of Neuroscience
Erasmus Medical Centre
PO Box 2040
3000 CA, Rotterdam
The Netherlands
Phone: +31631917511
E-mail: p.forbes@erasmusmc.nl
https://orcid.org/0000-0002-0230-9971

JNeurosci Accepted Manuscript

# Abstract

Standing balance requires rapid responses to postural disturbances, and the balance N1 has been proposed to reflect cortical involvement in corrective motor output. Our recent work shows, however, that the N1 persists even when active balance control is removed and corrective muscle activity is largely absent, suggesting it reflects sensory rather than motor processing. Because the N1 is also modulated by contextual factors such as postural threat, stability, and perturbation predictability, it may reflect uncertainty associated with perturbation-evoked sensory signals. We tested this in 11 participants (3 males and 8 females) using toes-up perturbations across three manipulations expected to alter sensorimotor uncertainty: perturbation timing through 15 unexpected perturbations before and after a block of self-initiated trials; repeated exposure through 60 self-initiated trials; and body-motion variability by comparing active balance with passive support and by imposing prerecorded whole-body motion during 60 additional self-initiated trials with passive support. Self-initiation reduced N1 amplitude by 21.3 ± 13.9% relative to unexpected perturbations, and this attenuation was equivalent across active and passive conditions. Habituation through repeated self-initiated perturbations produced a further 24.0 ± 17.7% decline, again independent of active balance. When perturbations were subsequently delivered unexpectedly, N1 amplitudes increased, confirming that attenuation was specific to the self-initiated context. Finally, imposing prerecorded whole-body motion during passive support did not change perturbation-evoked N1 amplitude, suggesting that body-motion variability does not drive the cortical response when active balance control is absent. These results indicate that the balance N1 is shaped by predictability and repetition, consistent with prediction error or surprise-based accounts of cortical responses to balance perturbations.


# Significance statement

The balance N1, a cortical response to postural disturbances, has long been thought to reflect corrective motor output. We recently showed it instead reflects sensory processing, as it persists even without active balance control. Here we test whether this processing scales with uncertainty associated with perturbation-evoked sensory signals by manipulating perturbation timing through self-initiation, habituation through repeated exposure, and variability of ongoing whole-body motion. Self-initiation and repetition both attenuated N1, regardless of whether participants actively balanced or were passively supported. However, increasing whole-body motion variability did not modify the response, indicating that not all sources of uncertainty modulate the N1. These findings recast the balance N1 as tracking the sensory predictability of the perturbation, not balance-correcting motor output.

# Introduction

Human balance control is largely mediated by subcortical structures, yet cortical activity is thought to contribute under challenging postural demands such as sudden perturbations (Jacobs and Horak, 2007). A prominent candidate marker of this cortical contribution is the balance N1, a negative electroencephalography (EEG) potential occurring 100–200 ms after a postural perturbation (Bolton, 2015; Payne et al., 2019a). The N1 has often been interpreted as motor-related based on several observations. It has been localized to the supplementary motor area (Marlin et al., 2014; Protzak et al., 2026), a region involved in movement preparation and action selection (Tanji, 1994); its amplitude has been related to subsequent balance-correcting responses (Payne and Ting, 2020; Solis-Escalante et al., 2021; Parr et al., 2024); and N1 activity has been used to predict longer-latency muscle

JNeurosci Accepted Manuscript

responses (Boebinger et al., 2024). However, our recent findings challenge this direct motor-output interpretation: when participants' whole-body movements were externally restricted during support-surface rotations, corrective muscle responses dropped markedly (~60%), yet the N1 remained comparable to that during active balance (Jansen et al., 2026). Its persistence under these conditions also suggests that ankle-related somatosensory input is a primary sensory contributor to the N1. Thus, the N1 may reflect cortical processing of perturbation-evoked somatosensory signals rather than directly supporting the execution of balance-correcting motor responses. Importantly, the N1 does not simply encode this sensory input, as its amplitude dynamically scales with postural threat, stability and perturbation predictability (Adkin et al., 2006; Adkin et al., 2008; Mochizuki et al., 2008; Solis-Escalante et al., 2021). In this view, the N1 may instead reflect how the brain evaluates perturbation-evoked sensory information, with N1 magnitude scaling with uncertainty associated with these signals.

This uncertainty-based interpretation aligns with theoretical accounts in which cortical responses reflect deviations from expected sensory input, often framed as sensory prediction error or surprise. Sensory prediction error refers to the discrepancy between expected and actual sensory feedback (Wolpert et al., 1995; Friston, 2005), whereas surprise quantifies how strongly an event violates expectations given the current context (Itti and Baldi, 2009; Friston, 2010). As perturbation timing and characteristics become more predictable, perturbation-evoked sensory signals should be more consistent with expectations. Thus, if the N1 reflects prediction error or surprise, its amplitude should decrease as perturbations become more predictable. An empirical test of an uncertainty-based account of the N1, however, have produced mixed results. Early work reported a complete absence of the N1 when participants were informed of perturbation timing, suggesting that removing uncertainty about perturbation onset can abolish the N1 response (Adkin et al., 2006). Others found only partial reductions following repeated or self-initiated perturbations (Mochizuki et al., 2009b; Mierau et al., 2015; Payne et al., 2019b). One possibility is that uncertainty associated with the perturbation-evoked sensory input remained because direction or acceleration varied (Payne et al., 2019b), or the number of repetitions was limited (~10, Mierau et al., 2015). Moreover, the natural variability during standing (Mensink et al., 2025) produces ongoing whole-body motion that adds sensorimotor uncertainty surrounding the perturbation. Thus, it remains unclear how uncertainty arising from either perturbation timing, perturbation repetition, or whole-body motion variability determines the scaling of the N1 response.

Here, we directly tested how N1 amplitude scales with these three sources of uncertainty. Using a robotic balance simulator that reproduced the mechanics and sensory feedback of upright stance, we manipulated these three factors. We hypothesized that N1 amplitude would decrease when participants self-initiated the perturbation and attenuate further with repetition, thereby reducing uncertainty about perturbation timing and characteristics, respectively. Because ongoing body motion introduces additional variability in body-state sensory information, we further expected the N1 to be smallest when participants were externally supported during repeated self-initiated perturbations, where this balance-related sensory variability was minimized. We then passively moved participants through their own prerecorded balance motion to determine whether reinstating body-state variability increased the perturbation-evoked N1. Together, these experiments evaluate how uncertainty from distinct sources shapes cortical responses to balance perturbations.

Neurosci Accepted Manuscript

# Methods and materials

## Participants

Eleven healthy adults [3 males, 8 females; age = 22.2 ± 3.9 yr (mean ± SD), range 19-33 yr] were recruited from Erasmus University Medical Center and surrounding regions to participate in the study. All participants had no self-reported history of neurological disorders or significant musculoskeletal injuries that may affect balance. The study used a within-subject repeated-measures design, with all experimental comparisons performed within participants and each condition comprising 15–60 trials. The balance N1 has excellent within-participant reliability when averaged across approximately six trials (Mirdamadi et al., 2025), and our condition averages therefore included sufficient trials to obtain a reliable estimate. The sample size was also comparable to that of related balance N1 studies (Solis-Escalante et al., 2021, n = 11) and our previous work using a similar paradigm (Jansen et al., 2026, n = 10 and 16). The experimental protocol conformed to the Declaration of Helsinki and was approved by the Medical Research Ethics Committee Erasmus Medical Center (NL76700.078.21, 14-04-2021). The protocol was explained before the experiment and all participants provided written informed consent before participating.

## Experimental setup

Participants stood in a custom-built robotic balance simulator designed to replicate the mechanics of natural upright stance modelled as an inverted pendulum system in both anteroposterior (AP) and mediolateral (ML) directions (Forbes et al., 2016; Qiao et al., 2023; Rasman et al., 2024; Jansen et al., 2026). The simulator consisted of four independently actuated components: a backboard frame, hip and shoulder harnesses, and an ankle-tilt platform (footplate), each driven by its own servo-motor-powered linear actuator (Figure 1A). Mechanical specifications for the actuators, motors, and linkages are detailed in Rasman et al. (2024).

Briefly, AP body motion was controlled by moving the backboard, ML motion was driven by movement of the hip and shoulder harnesses, and foot rotation was generated by motion of the ankle-tilt platform. Participants stood barefoot on a force plate (AMTI BP400 × 600; Watertown, MA, USA) fixed to the ankle-tilt platform, with feet hip-width apart and the ankle joint axis (Sado et al., 2021) aligned to the axis of platform rotation. Seatbelts attached to the hip and shoulder harnesses secured participants at the level of the greater trochanter and sternal notch, respectively. The harnesses were padded with medium-density foam and additional foam was placed between participants and the belts to improve comfort. The hip and shoulder harnesses prevented forward falls and were supported with gas springs to offset the weight of the motors and sliders, preventing additional load for the participant when balancing upright.

A real-time controller (PXI-8880; National Instruments, TX, USA) operating at 500 Hz simulated body dynamics and relayed motor commands via an FPGA module (PXI-7846R; National Instruments, Austin, TX, USA), which interfaced with the motor drives. The inverted pendulum simulation incorporated individual physical parameters, including mass (63.7 ± 8.0 kg, height (172.4 ± 7.4 cm), center of mass (CoM) height (86.1 ± 3.1 cm), ankle height (7.6 ± 0.3 cm), and ankle width (i.e., stance width, which was equal to hip width, 16.0 ± 0.8 cm). CoM height was estimated by having participants lie supine on a balanced board that was rolled over a transverse rod until equilibrium was achieved. The CoM height was measured from the ankle joint (Sado et al., 2021) to the board's tipping point.

J Neurosci Accepted Manuscript

While standing in the simulator, participants were part of the control loop and generated ankle torques to control upright body motion (see Figure 1A). In the AP direction, motion was governed about an axis aligned with the ankle joints. When participants balanced with their center of mass anterior from their ankle joints, as in natural standing, they generated stabilizing torque from plantarflexor muscles to maintain balance. Applying greater plantarflexor torque than the toppling torque due to gravity rotated the body posteriorly. Balance control in the mediolateral axis followed a similar inverted pendulum model, with the center of rotation located between the ankles. ML angular position was translated to lateral displacement at the hip and shoulders, simulating postural mechanics for a given stance width while assuming that the pelvis stays perpendicular to the torso (Day et al., 1993; Goodworth and Peterka, 2012; Qiao et al., 2023).

Motion limits were imposed to act as safety boundaries when participants lost balance. These limits were chosen such that participants could autonomously return to the balance range from the end position by applying corrective torque in the opposite direction. In the AP direction, angular positions were limited to 6° anterior and 3° posterior; ML motion was limited to ±3°. When participants exceeded these limits, supportive torques were gradually introduced over a 1° buffer zone to halt further motion, aided by damping to ensure smooth deceleration (Rasman et al., 2024).

## Familiarization and baseline

Prior to experimental testing, participants familiarized themselves with balancing in the robot by exploring the full range of motion. They were then exposed to practice perturbations until they could maintain balance within the motion limits for at least two consecutive trials. All participants met this criterion during their first two practice perturbations and therefore completed only two practice trials. Participants were also informed that the perturbations would be identical in direction and magnitude throughout the experiment. Each participant then performed two baseline trials: a preferred posture trial and a target trial. The preferred posture trial was a one-minute quiet standing period in which participants stood at their preferred whole-body orientation. Data from this trial were used to calculate participant-specific standard deviations of angular position, angular velocity and ankle torque, which dictated the position, velocity and torque ranges that were tolerated in subsequent trials. In the target trial, participants balanced at a 2.5° forward angle for two minutes while receiving real-time feedback of their position and velocity (see Protocol). Data from these target trials were used to generate motion profiles used in trials that simulated natural balance uncertainty (see Protocol).

## Perturbations and general procedures

Controlled ankle perturbations were delivered by rotating the ankle-tilt platform (footplate) toes-up using a smooth ramp trajectory of 0.15 radians over 0.3 seconds (see Figure 1C; Jansen et al., 2026). These rotations caused the whole-body to move posteriorly, necessitating participants to generate dorsiflexion torque for stability. The perturbations followed a minimum-jerk profile as described by (Vlutters et al., 2015), with zero acceleration at both the start and end of the perturbation to ensure smooth rotations.

To minimize recording artifacts in the EEG data, participants were asked to refrain from blinking or contracting facial/throat muscles once they were at the correct whole-body orientation until ~2-3 s after receiving the perturbation. Perturbations were delivered when participants were stable and remained for ~1-2 s within range of the target angle (2.5 degrees anterior). Unexpected perturbations were delivered by the experimenter unpredictably by randomizing their timing, with a minimum time of 16 s between each

JNeurosci Accepted Manuscript

perturbation. Self-initiated perturbations were triggered when participants pressed a handheld button. The typical delay between button presses and measured perturbation onset was estimated at 20 ms. This delay reflected registration of the button press and initiation of the footplate movement profile by the real-time system (4 ms), a 7-8 ms delay between commanded and actual footplate position, and noise in identifying the precise onset of footplate movement within the motor encoder signal. In nine participants, a small number of trials occurred in which participants were just outside the required position/velocity (Balance Control trials) or torque (No-Control trials) ranges when the button was pressed but returned within range shortly thereafter, resulting in longer button-to-perturbation delays of approximately 50–200 ms. These events were infrequent, occurring in no more than five trials per affected participant and no more than two trials per condition. Because these longer and less predictable delays could alter the temporal predictability of self-initiation, these trials were excluded from the analysis. After a perturbation, participants typically required about 5-15 s to recover their balance in either condition and return their whole-body angle to the target, resulting in an average interval of 26 ± 5 s between perturbation onsets.

## Protocol

After familiarisation, participants completed the main experimental tasks in two separate conditions: Balance Control and No-Control as described below. The order of these conditions was counterbalanced across participants to account for order effects.

### Balance Control

In the Balance Control condition, participants actively maintained their upright posture by actively generating ankle torques that controlled the motion of their whole body through the robotic system. This condition included three blocks of trials. In the first block, referred to as Unexpected-pre, participants were exposed to a total of 15 toes-up footplate rotations that were delivered with unexpected timing. Participants received real-time visual feedback of their whole-body movement on a screen placed ~1.5 m in front of them. Whole-body movement was presented as a stationary red circle on a black background that turned green when their center of mass angle was within 1.5 × baseline position SD of the target angle (2.5° anterior) and their whole-body velocity was below 2 × baseline velocity SD. Perturbations were delivered only when the target was green; once the target turned green, the experimenter triggered the footplate rotation after a random delay of 1–8 s, without informing the participant. At the onset of the footplate rotation, the visual feedback disappeared, leaving a black screen. The second block, Habituation, allowed participants to self-initiate the perturbations by pressing a handheld button and tested the effects of removing timing uncertainty about when a perturbation would occur. A total of 60 self-initiated Habituation trials were completed. The final block, Unexpected-post, repeated the structure of the first block with 15 toes-up rotations delivered with the same unexpected timing. Because perturbations were only delivered (either unexpected or self-initiated) provided participants were within the prescribed range of body position and velocity, any preparatory postural adjustment intended to counter the upcoming perturbation, such as leaning forward, would have moved participants outside the window and prevented the perturbation from being delivered. This constraint therefore precluded large anticipatory adjustments to the perturbation.

### No-Control

In the No-Control condition, participants had no control over their posture and the backboard was fixed at an anterior lean angle of 2.5°. Participants were informed before these trials that they would have no control over their movement. This condition tested whether removing

J Neurosci Accepted Manuscript

sensory uncertainty about whole-body motion (present during active balance control) enables greater attenuation in the N1 across habituation trials. Participants were instructed to generate ankle plantarflexor torque at a level equivalent to the torque needed to maintain a whole-body angle of 2.5 degrees forward during the Balance Control trials (i.e., T = $0.0436 \cdot mgl$). To do so, participants received feedback on ankle torque magnitude, conveyed using the same visual cue (i.e., a red circle that turned green when torque was within 1.5 × baseline torque SD of the target torque). Perturbations could only be delivered when the participant's ankle torque was within this range. The first two blocks of the No-Control condition (i.e., Unexpected-pre and Habituation), were identical in structure to those in the Balance Control condition.

Following these two blocks, participants completed an additional block of Motion-variability trials in which they had no influence over their whole-body motion and pressed a button to initiate perturbations. However, unlike during the earlier Habituation trials in the No-Control condition, the backboard was not fixed at the target angle. Instead, the system moved in the whole-body in the anteroposterior plane according to motion profiles recorded during the target baseline trial when participants actively balanced at a 2.5° anterior lean using approaches similar to (Leeuwis et al., 2024; Jansen et al., 2026). These computer-controlled motion profiles were presented at three scaled magnitudes of the same signal (1×, 2×, and 4×), and the backboard continued moving according to the selected profile when the footplate rotation was applied. The Motion-variability trials were designed to gradually reintroduce unpredictable whole-body motion, and thus sensory uncertainty, while keeping participants out of the control loop. Since we expected N1 amplitudes to be most attenuated at the end of the Habituation block in the No-Control condition, the Motion-variability trials were designed to test whether this expected attenuation would be driven by ongoing motion variability rather than active balance control per se. Specifically, if motion variability on its own were the determining factor, reintroducing whole-body motion should increase N1 amplitudes. Each scaling level was tested in a separate 20-trial block, for a total of 60 Motion-variability trials. As in the Balance Control condition, the No-Control condition ended with a second block of Unexpected-post trials. Participants were given two scheduled 5–10 min breaks off the robot: one after the baseline trials and another between the Balance Control and No-Control conditions.

# Data recording and processing

## EEG

We recorded EEG using a 64-channel cap (Infinity Headcap, TMSi, Oldenzaal, Netherlands) following the 10-20 system, connected to a 128-channel REFA amplifier which sampled at 2048 Hz. Conductive electrode gel (Electro-Gel, Electro-Cap International, Ohio, USA) was applied to each electrode site using a syringe. Impedance was maintained below 10 kΩ for Cz and surrounding electrodes (FCz, FC1, FC2, C1, C2, CP1, CPz, CP2) and below 20 kΩ for the remaining electrodes. A reference electrode was placed on the head of the right ulna. Signal quality was visually inspected before data collection, with flat or noisy channels corrected or excluded during further analysis. Actively shielded electrode cables were used to reduce motion-related artifacts. To further limit cable movement, the EEG cable bundle was secured to the robotic simulator at the shoulder and hip supports, and the amplifier was positioned close behind the participant at approximately knee height. During Unexpected-pre and -post trials, real-time EEG monitoring ensured perturbations were delivered during low-noise periods. Data were recorded using a fiberoptic-to-USB interface (Fusbi) and stored via MATLAB (Version 23.2.0.2485118, R2023b) using custom EEG scripts (Delorme and Makeig, 2004).

J Neurosci Accepted Manuscript

EEG data were processed using MATLAB 2023b (The MathWorks, Natick, MA, USA). Raw EEG data were filtered with a 1 Hz high-pass and 40 Hz low-pass cutoff, using a zero-phase finite impulse response (FIR) filter. The high-pass filter attenuated the slow pre-perturbation cortical activity previously observed during self-triggered balance perturbations and attributed to anticipation or changes in postural set (Mochizuki et al., 2008). It would also attenuate slow movement-related potentials, such as the classical readiness potential that can precede voluntary movements (Shibasaki and Hallett, 2006). Notably, previous work using comparable self-triggered balance perturbations also found little discernible readiness-potential activity associated with the small triggering movement itself (Mochizuki et al., 2008). Finally, because the button-to-perturbation interval was short and highly consistent (~20 ms), and trials with longer delays were excluded, any residual button-related activity occurred at a similar latency relative to perturbation onset across retained self-initiated trials.

Channels exhibiting flatline periods exceeding 5 s or a correlation below 0.77 with neighbouring channels were excluded. The remaining data were segmented into 2.5 s epochs, time-locked to perturbation onset (time = 0), with each epoch beginning 1 s before the perturbation. Single-trial epochs were baseline-corrected by subtracting the mean voltage from a pre-perturbation window (-150 to -50 ms). Artifactual components (e.g., eye blinks and muscle activity) were removed using independent component analysis (ICA; runica algorithm; Delorme and Makeig, 2004). Components were additionally inspected visually and removed when they reflected clear non-neural activity, including residual motion-related artifacts or line noise. Since the balance N1 response is typically maximal at Cz, N1 amplitude was quantified at this electrode as the minimum EEG magnitude between 100–200 ms post-perturbation on a trial-by-trial basis.

## EMG

We recorded EMG from the right tibialis anterior (TA) muscle using pairs of Ag-AgCl surface electrodes (Blue Sensor M, Ambu A/S, Ballerup, Denmark). This muscle was selected because of its contribution to balance recovery during toes-up perturbations (Di Giulio et al., 2009; Jansen et al., 2026). An additional ground electrode was placed on the right wrist. The skin was cleaned with isopropyl alcohol and prepared with Nu-Prep gel. Electrodes were placed with a 2 cm inter-electrode distance and secured with tape. EMG signal quality was verified by inspecting the signal during periods of voluntary dorsiflexion and plantarflexion contractions and periods of maintaining relaxed muscles.

Raw EMG data were high-pass filtered at 10 Hz (zero-phase FIR filter, 2 Hz transition bandwidth), rectified, and low-pass filtered at 30 Hz (zero-phase FIR filter, 7.5 Hz transition bandwidth). Single-trial EMG amplitudes were normalized to the maximum value observed across all trials for each participant. EMG data were segmented into 2.5 s epochs, time-locked to perturbation onset (time = 0), with each epoch beginning 1 s before the perturbation. To focus on balance-corrective responses rather than short-latency reflexes, response amplitudes were quantified by extracting the peak EMG response over a 200–800 ms window following perturbation onset on a trial-by-trial basis (Nashner, 1976; Henry et al., 1998; Carpenter et al., 1999; Jansen et al., 2026).

## Kinematics

We recorded angular position of the whole-body center of mass (CoM) using encoders embedded in the hip, shoulder, and backboard motors, sampled at 500 Hz. From these position data, we derived whole-body angular velocity and acceleration through first- and second-order numerical differentiation, respectively. Kinematic data were segmented into the same 2.5 s epoch as EEG and EMG data. From these trial-by-trial responses, we extracted

Neuroscience Accepted Manuscript

the peak whole-body velocity following perturbation onset for Balance Control trials to provide a qualitative comparison to the muscle responses underlying the balance motion. From the baseline trials, we used the position and velocity signal to establish the target movement range that participants needed to remain under before perturbations could be delivered (see Protocol).

## Statistics

All statistical analyses were performed in JASP (JASP Team, 2024) with a significance threshold of $\alpha$=0.05. To test the influence of participants controlling their balance, we first compared the N1 amplitudes between the Balance Control and No-Control conditions during the first block of externally triggered perturbations (Unexpected-pre) using a paired-samples *t*-test on the mean N1 amplitude averaged across all 15 Unexpected-pre trials in each condition. To test our hypothesis that self-initiation of perturbations would attenuate balance N1 responses, we then performed a 2×2 repeated-measures ANOVA with perturbation type (Self-initiated vs. Unexpected) and balance condition (Balance Control vs No-Control). To ensure an equal contribution of trials within each condition, the N1 amplitudes were averaged over 15 trials for both Unexpected-pre (i.e., all trials) and Self-initiated (i.e., the first 15 Habituation trials) perturbations.

To test our hypothesis that self-initiating the perturbations over an extended series of perturbations (60 trials) in the different balancing conditions decreased N1 amplitudes, we performed a 2×2 repeated-measures ANOVA comparing average N1 amplitudes across the first and last five Habituation trials and across the balance conditions (Balance Control vs. No-Control). We restricted the early and late windows to five trials each so that the early estimate reflected the initial, largely unhabituated response. Including additional trials in the early window may incorporate the effect of habituation which during normal balance can emerge during the initial phase of repeated perturbation exposure (Mierau et al., 2015) and thereby reduce the contrast between the early and late phases. Five trials are sufficient to obtain a stable estimate of the N1: its amplitude reaches excellent internal reliability, which plateaus after approximately six trials (Mirdamadi et al., 2025), and the N1 can also be quantified reliably at the single-trial level (Payne et al., 2019b). Similarly, to test whether the attenuation observed across the Habituation trials would generalize to when perturbations were then delivered unexpectedly, we ran a 2×2 repeated-measures ANOVA with the last 15 Self-initiated and the 15 Unexpected-post trials, and condition (Balance Control vs. No-Control). The same 2×2 repeated-measures ANOVAs were performed on mean TA EMG amplitude, testing the effects of self-initiation, habituation, and generalization on muscle activation using the same trials as for the N1 analysis. In all analyses where significant condition × perturbation type or condition × timing interactions were observed, post-hoc pairwise comparisons were conducted with Holm correction for multiple comparisons.

Finally, to test our hypothesis that reinstating whole-body movement variability in the No-Control condition would increase N1 amplitudes, we performed a one-way repeated-measures ANOVA with four levels: the last 20 self-initiated trials of the Habituation block without imposed motion, chosen to match the 20-trial Motion-variability blocks, and the three Motion-variability blocks (1×, 2×, and 4×). To directly test the predicted increase, we used planned simple contrasts comparing each imposed-motion level (1×, 2×, and 4×) against the no-motion baseline.

JNeurosci Accepted Manuscript

# Results

## Self-initiated perturbations reduce N1 amplitudes

In both the Balance Control and No-Control conditions, participants first experienced 15 perturbations triggered externally by the experimenter with unpredictable timing (Unexpected-pre). Trial-by-trial group-average responses (Figure 2A) showed that N1 amplitude decreased slightly across this initial block in both conditions, and that average responses during No-Control (-27.40 ± 1.99 μV) decreased by only ~2% relative to Balance Control (-28.76 ± 2.07 μV), a non-significant difference ($t(10) = -0.696$, $d = -0.210$, $p = 0.503$). This is consistent with our previous finding that removing balance control produces modest changes in N1 amplitude (Jansen et al., 2026).

We next examined how self-initiation affects balance-related cortical responses by comparing N1 amplitudes evoked by unexpected versus self-initiated perturbations in both the Balance Control and No-Control conditions. Although participants performed 60 self-initiated trials in the Habituation block (Figure 2A), we analyzed only the first 15 self-initiated trials for this comparison to match the trials counts with the Unexpected-pre block. N1 responses were markedly smaller when perturbations were self-initiated compared to when they were unexpected (see Figure 2). In the Balance Control condition, N1 amplitudes decreased from -28.76 ± 2.07 μV during externally triggered perturbations to -22.14 ± 1.48 μV during self-initiated perturbations, corresponding to an attenuation of 20.3 ± 21.2%. A similar attenuation was observed in the No-Control condition, where amplitudes decreased from -27.40 ± 1.99 μV to -21.17 ± 1.82 μV, a reduction of 22.5 ± 13.8%. Across both conditions, this attenuation (21.3 ± 14.0%) produced a significant main effect of perturbation type ($F(1,10) = 21.40$, $p = 9.415 \times 10^{-4}$, $\eta^2 = 0.39$), suggesting that uncertainty about perturbation onset influences the N1. In contrast, balance condition had no significant effect on N1 amplitude ($F(1,10) = 0.36$, $p = .561$, $\eta^2 = 0.013$), and the effect of self-initiation was similar in both conditions (i.e., no interaction: $F(1,10) = 0.05$, $p = .822$, $\eta^2 = 3.535 \times 10^{-4}$).

## Habituation of N1 during self-initiated perturbations is not affected by balance control

Having established that self-initiation reduces N1 amplitudes within the first 15 trials, we next tested whether N1 responses further habituate across the 60 repeated self-initiation trials, and whether this habituation depends on active balance control. Group average N1 responses showed a progressive decrease in the N1 magnitude throughout the 60 trials in both Balance Control and No-Control conditions (see Figure 2A). In the Balance Control condition, N1 amplitudes decreased from -23.73 ± 2.03 μV in the first five trials to -20.20 ± 1.40 μV in the last five trials, corresponding to an attenuation of 11.1 ± 21.7%. In the No-Control condition, amplitudes declined from -23.36 ± 1.98 μV to -15.35 ± 2.28 μV, reflecting a decrease of 30.9 ± 32.1%. This overall reduction with repetition (24.1 ± 18.0%) was confirmed by a significant main effect of timing ($F(1,10) = 19.30$, $p = 1.349 \times 10^{-3}$, $\eta^2 = 0.27$). In contrast, balance condition had no significant main effect on mean N1 amplitude ($F(1,10) = 2.24$, $p = .165$, $\eta^2 = 0.06$), and the level of habituation did not reliably differ between the Balance Control and No-Control conditions (i.e., no timing × condition interaction $F(1,10) = 1.59$, $p = .236$, $\eta^2 = 0.04$). Notably, the mean responses at the end of the 60 trials also did not reach zero for either condition. This was with the exception of one participant who showed no discernible N1 at the end of habituation in the No-control condition.

JNeurosci Accepted Manuscript

The attenuation observed during repeated self-initiation raises a further question: does the attenuation represent context-specific changes that occur with self-initiation, or a generalized reduction in responses to the repeated perturbation? To test this, we compared N1 amplitudes in the last 15 Self-initiated trials with those in the Unexpected-post trials. The last 15 perturbations of the self-initiated block elicited a mean N1 amplitude of -16.86 ± 1.08 μV across both balance condition, while the Unexpected-post trials yielded -23.99 ± 1.67 μV, an increase of 46.3 ± 40.8%. Statistical analysis confirmed a significant difference ($F(1,10) = 20.61$, $p = 1.074 \times 10^{-3}$, $\eta^2 = 0.490$), indicating that the attenuation observed during repeated self-initiated perturbations did not carry over to externally triggered ones. These responses were also unaffected by balance condition ($F(1,10) = 2.442$, $p = .149$, $\eta^2 = 0.017$), and no interaction between timing and condition was observed ($F(1,10) = 2.560$, $p = .141$, $\eta^2 = 0.037$). Together, these results show that the N1 habituates reliably with repeated self-initiation but rarely disappears, regardless of whether participants are actively balancing or externally supported. Furthermore, this habituation is specific to participants self-initiating the perturbations rather than a general reduction in responses to the repeated perturbation.

## Variability of whole-body state does not reinstate N1 amplitude

We next examined whether reintroducing (and exaggerating) variability about the body's kinematic state, without restoring balance control, would increase the N1 response following the period of habituation. In the No-Control condition only, participants completed three additional blocks of trials of self-initiated perturbations while a pre-recorded whole-body motion trajectory (captured during the baseline trial) was imposed in the anterior-posterior direction at three velocity levels: 1× (original), 2× and 4×. If increased whole-body state variability enhances the perturbation's unpredictability during self-initiated trials, increasing the magnitude of imposed motion should increase the N1 relative to the self-initiated trials in which no motion was imposed. In contradiction to this hypothesis, mean N1 amplitudes were similar and did not differ across velocity levels (see Figure 3): -15.43 ± 1.52 μV with no imposed motion, -16.49 ± 1.53 μV at 1×, -16. 36 ± 1.38 μV at 2×, and -16. 16 ± 1.37 μV at 4× ($F(3,30) = 0.38$, $p = .771$, $\eta^2 = 0.036$). Planned contrasts of the motion trials against the no-motion baseline confirmed this, with no level of imposed motion significantly increasing the N1 (all $p > .35$, all $d < 0.23$). Thus, variability in whole-body state prior to the perturbation during the No-Control condition does not appear to drive the amplitude of the balance N1.

## Muscle activity is prominent only during active balance and is modulated by self-initiation and repetition

So far, our results show that N1 amplitude was modulated by predictability and repetition and was not influenced by whether participants were actively controlling their balance motion or being externally supported. To test whether this pattern mirrors changes in motor output or instead reflects further dissociation between cortical and motor responses, we next examined TA EMG and peak whole-body velocity responses across the same trial blocks and conditions. In the average responses during the Balance Control condition (Figure 4A), perturbations elicited strong bursts of stabilizing dorsiflexor activity that progressively decreased across trials within each phase of the experiment. Peak whole-body velocity followed a similar trend (see inset Figure 4A), mirroring the muscle activity driving the balance motion. In contrast, TA activity in the No-Control condition remained nearly absent throughout the experiment, despite robust N1 responses, reinforcing the dissociation between this cortical response and balance-correcting motor output (Jansen et al., 2026).

We compared TA amplitudes between unexpected and self-initiated perturbations. During the Unexpected-pre phase, TA responses in the Balance Control condition were largest at

JNeurosci Accepted Manuscript

the start of the block and declined across the subsequent 15 trials. This is consistent with prior work showing that adaptation to repeated perturbations diminishes responses within a similar number of trials (Horak and Nashner, 1986; Keshner et al., 1987; Oude Nijhuis et al., 2010; Welch and Ting, 2014). These responses then showed a brief increase on the first Self-initiated trial before continuing to decrease across the 60 trials. This isolated increase may represent a first-trial response to the novel self-initiation context, consistent with the exaggerated initial muscles responses observed when participants first encounter a new perturbation condition (Keshner et al., 1987; Oude Nijhuis et al., 2010). However, no corresponding increase was evident in the N1, and peak angular velocity showed at most a much smaller first-trial elevation (see inset Figure 4A), indicating that the pronounced TA response was not accompanied by a similar increase in cortical activity or whole-body movement.

Comparison of these two phases (Unexpected-pre vs. Habituation block) showed that TA responses were substantially larger for unexpected perturbations (0.547 ± 0.038) than for self-initiated perturbations (0.355 ± 0.045), indicating that the corrective TA response was reduced when perturbation onset was self-controlled and temporally predictable. In contrast, TA responses in the No-Control condition were markedly smaller compared to the Balance Control condition in both Unexpected-pre (0.031 ± 0.006, Figure 4C) and Habituation blocks (0.019 ± 0.003), consistent with the nearly absent responses throughout all trials. These effects were confirmed by a strong main effect of balance condition ($F(1,10) = 136.01$, $p = 3.891 \times 10^{-7}$, $\eta^2 = 0.826$) and perturbation type ($F(1,10) = 37.89$, $p = 1.075 \times 10^{-4}$, $\eta^2 = 0.048$), as well as a significant condition × type interaction ($F(1,10) = 22.82$, $p = 7.493 \times 10^{-4}$, $\eta^2 = 0.037$). These results indicate that self-initiation produced a larger attenuation of TA activity in Balance Control ($t(10) = 5.472$, $d = 1.950$, $p = 5.442 \times 10^{-4}$) than in No-Control ($t(10) = 2.262$, $d = 0.122$, $p = .047$).

To quantify habituation during self-initiated perturbations, we next compared mean TA amplitudes between the first and last five trials. In the Balance Control condition, TA amplitudes declined substantially across the 60 trials from 0.405 ± 0.047 to 0.227 ± 0.040. On the other hand, in the No-Control condition, responses were absent throughout and did not change (first five: 0.023 ± 0.005; last five: 0.020 ± 0.005). These effects yielded a clear main effect of balance condition ($F(1,10) = 53.19$, $p = 2.623 \times 10^{-5}$, $\eta^2 = 0.691$), a significant decline over the 60 trials ($F(1,10) = 23.03$, $p = 7.246 \times 10^{-4}$, $\eta^2 = 0.065$), and a strong interaction effect between condition and timing ($F(1,10) = 23.45$, $p = 6.788 \times 10^{-4}$, $\eta^2 = 0.061$), reflecting that TA amplitudes declined across the 60 trials in Balance Control ($t(10) = 4.829$, $d = 1.707$, $p = 2.047 \times 10^{-3}$) but not in No-Control ($t(10) = 1.218$, $d = 0.027$, $p = .251$). These results indicate that habituation of muscle responses during self-initiated perturbations occurred only when participants actively controlled their balance.

Finally, when perturbations returned to being unexpected in the Balance Control condition, TA responses rebounded on the first trial and then settled at a consistent level that remained elevated relative to that reached during self-initiation (Figure 4A). Consistent with the observed rebound, TA amplitude increased by 60.2 ± 47.7% from the last 15 Self-initiated trials (0.229 ± 0.036) to the Unexpected-post trials (0.342 ± 0.052), indicating that attenuation of the muscle response did not persist when perturbations became unexpected again. In contrast, in the No-Control condition TA responses were similarly low across both blocks (Self: 0.018 ± 0.003; Unexpected-post: 0.023 ± 0.003). This produced a strong main effect of condition ($F(1,10) = 37.50$, $p = 1.122 \times 10^{-4}$, $\eta^2 = 0.704$) and a significant effect of timing ($F(1,10) = 15.48$, $p = 2.558 \times 10^{-3}$, $\eta^2 = 0.033$). A condition × timing interaction ($F(1,10) = 13.64$, $p = 4.155 \times 10^{-3}$, $\eta^2 = 0.029$) was also observed, reflecting the rebound in

Neuroscience Accepted Manuscript

TA activity in Balance Control ($t(10) = -3.823$, $d = -1.072$, $p = 6.715 \times 10^{-3}$) and absence of a muscle response throughout No-Control ($t(10) = -3.072$, $d = -0.053$, $p = .012$).

# Discussion

In this study, we examined how the balance N1 and corrective muscle responses to standing balance perturbations change when uncertainty associated with the perturbation-evoked sensory signals is altered through self-initiation, repetition and whole-body motion variability. Self-initiation and repetition both attenuated the N1 in the Balance Control condition, but this effect was similar in the No-Control condition, indicating that the body-state variability inherent in active balancing does not drive the N1. Furthermore, reintroducing and exaggerating whole-body motion during No-Control did not increase N1 amplitude, suggesting that increasing body-state uncertainty through isolated body-motion is insufficient to increase the N1 when active balance control is removed. In contrast, N1 amplitudes increased again when perturbations returned to being unexpected, indicating that attenuation was specific to the self-initiated context. Muscle responses mirrored the N1 during active balance, decreasing with self-initiation and repetition and rebounding when perturbations became unexpected, providing a plausible explanation for the common assumption that cortical and muscle responses are tightly coupled. However, the two responses diverged when balance engagement was removed: TA activity was virtually absent throughout No-Control, whereas N1 modulation remained comparable across Balance Control and No-Control. Together, these results indicate that the attenuation of the balance N1 is shaped by the predictability of perturbation timing and repetition, whereas increasing body-state variability alone does not enhance the response, and further show that this modulation does not simply reflect or directly drive balance-correcting motor output.

## Self-initiation of perturbations reduces N1 amplitude

In line with previous work (Adkin et al., 2006; Adkin et al., 2008; Mochizuki et al., 2009a), we found that the balance N1 was attenuated when perturbations were predictable in timing compared to being unexpectedly triggered. Specifically, N1 amplitudes during Balance Control decreased by ~20% when comparing the 15 unexpected trials with the first 15 self-initiated trials. This attenuation was accompanied by parallel decreases in perturbation-evoked TA activity, consistent with the commonly assumed coupling between cortical and muscular responses to balance perturbations during active balance. Such coupling has been taken to support the interpretation that N1 attenuation reflects a reduced need for cortical engagement in coordinating postural responses. Indeed, Adkin et al. (2006) used externally delivered perturbations preceded by an auditory cue and interpreted the resulting N1 attenuation in this way. However, our No-Control results argue against this interpretation. Participants were fully supported and generated virtually no corrective muscle activity, yet N1 amplitudes were comparable to those in Balance Control, and the reduction in N1 amplitude from unexpected to self-initiated perturbations was similar between conditions. Thus, predictability of the perturbation can modulate the N1 independently of the need to produce a corrective balance response.

These findings are more consistent with the view that the N1 reflects cortical processing of perturbation-evoked sensory signals. Within predictive coding frameworks (Wolpert et al., 1995; Rao and Ballard, 1999; Friston, 2005), the brain generates expectations about upcoming sensory input, and cortical responses can reflect differences between expected and received signals. Self-initiation reduces uncertainty about perturbation onset, making the resulting perturbation-evoked sensory signals more predictable. The accompanying

J Neurosci Accepted Manuscript

attenuation of the N1 is therefore consistent with reduced sensory prediction error or surprise, rather than a diminished requirement for cortically-driven motor coordination. This interpretation is also consistent with isolated sensory attenuation paradigms across modalities (e.g. the auditory and somatosensory N1), where self-generated or predictable stimuli evoke smaller cortical responses than unexpectedly delivered stimuli, also in scenarios where no corrective motor response is required (Hughes et al., 2013; Kiepe et al., 2021).

## N1 habituation from repeated self-initiated perturbations is independent of balance control but does not generalize

We also found that repeated exposure to identical self-initiated perturbations resulted in an additional ~25% reduction in N1 amplitude across the 60 trials, indicating that self-controlled timing and repetition have cumulative effects. This extends previous reports of N1 habituation during repeated unexpectedly triggered balance perturbations (Mierau et al., 2015; Payne et al., 2019b), a pattern partially observed during our Unexpected-pre block. Importantly, similar repetition-related habituation was present in No-Control, where participants were externally supported and not actively balancing. This finding is difficult to reconcile with the threat-based interpretation of Payne et al. (2019b), who linked N1 habituation to reduced startle reflexes, because participants in No-Control faced no threat of instability. Instead, our findings align more closely with the interpretation that habituation reflects a progressive reduction of sensory prediction error or surprise (Mierau et al., 2015): with repeated exposure, participants gain knowledge about the characteristics of the perturbation, making the evoked sensory signals increasingly predictable. Crucially, because this attenuation occurred even when balance-correcting muscle responses were largely absent, our findings also do not support the explanation that N1 habituation is driven by the perturbation's implications for stability or the control actions it evokes (Stokkermans et al., 2022; Boebinger et al., 2024).

When perturbations returned to being unexpected, N1 amplitudes increased for all participants and in both conditions, indicating that attenuation during self-initiation did not generalize across contexts. This was particularly striking in the No-Control condition, where participants completed the 60-trial Habituation block and an additional 60 Motion-variability trials before the Unexpected-post block. Even after this extended exposure to self-initiated trials, N1 amplitudes rebounded substantially when perturbations became externally triggered again. This context specificity suggests that N1 attenuation was tied to the predictive context established during self-initiation, rather than reflecting a generalized reduction in responsiveness to the perturbation.

## State variability without active balance does not increase the N1

We also expected that removing variability in ongoing motion in the No-Control condition would enhance N1 attenuation. Contrary to this hypothesis, the degree of habituation was similar between the Balance Control and No-Control conditions. To test our prediction further, we then reintroduced and exaggerated variability within the No-Control condition by imposing whole-body motion with the perturbation. This manipulation was intended to disrupt the internal estimate of body state, based on models showing that noisy or ambiguous sensory cues reduce the reliability of these estimates (van Beers et al., 1998; Peterka, 2002), yet it did not increase N1 amplitude. Thus increasing body-state uncertainty alone was insufficient to enhance the N1 when active balance demands were absent. Importantly, this conclusion is limited to the No-Control context and does not establish that state

JNeurosci Accepted Manuscript

variability is unimportant during active balance, where fluctuations in body state are directly relevant to task performance and stereotyped cortical activity is observed during naturally occurring balance variations in quiet standing (Varghese et al., 2015; Zaback et al., 2023). One possibility is that participants discounted the imposed motion as irrelevant because it had no consequence for task performance, consistent with the idea that sensorimotor uncertainty influence internal estimates and corrective responses primarily when they are relevant to task goals (Körding and Wolpert, 2004; Todorov, 2004). Testing comparable manipulations during active balance will therefore be important to determine whether state variability plays a stronger role when it directly affects postural control.

We hypothesized that the N1 would reach its lowest values by the end of the habituation block in the No-Control condition, where timing uncertainty, perturbation novelty and body-state variability were all simultaneously minimized. Based on prior reports that timing predictability of the perturbation alone can abolish the N1 (Adkin et al., 2006; Adkin et al., 2008), this combination of factors could have been expected to eliminate the response entirely. Yet only one participant showed no discernible N1 in the final 5 trials of the habituation block in the No-Control condition. This persistence suggests that individuals differ substantially in how far predictability and repetition attenuate the N1; again, within a sensory prediction error or surprise framework, this may reflect individual differences in how strongly expectations modulate the cortical response to perturbation-evoked sensory signals.

## Dissociation between cortical and muscular responses

The TA EMG results help clarify both the coupling and dissociation between cortical and muscular responses during balance perturbations. During active balance control, muscle responses closely tracked the N1 across all phases of the experiment: declining with temporal predictability and repetition, and rebounding when perturbations returned to being unexpected. This parallel modulation provides a plausible explanation for why the N1 has often been interpreted as closely linked to the generation of corrective motor output (Peterson and Ferris, 2019; Payne and Ting, 2020; Stokkermans et al., 2023; Boebinger et al., 2024). In conventional perturbation paradigms, where participants are always actively controlling balance, cortical and muscular responses naturally covary, making it difficult to determine whether the N1 reflects motor drive itself or upstream processes that coincidentally scale alongside corrective responses. The No-Control condition breaks this coupling. As expected from our previous findings (Jansen et al. 2026), TA activity was virtually absent when participants were externally supported and no longer controlling balance, yet a robust N1 remained. This dissociation cautions against treating the N1 as a readout of corrective motor output or that cortical activity can be inferred from muscle responses (Boebinger et al., 2024). Crucially, the present findings extend this dissociation by showing that the N1 remains sensitive to the uncertainty of perturbation-evoked sensory signals even when corrective motor responses are largely absent, supporting the view that it reflects how these signals are evaluated in relation to their expectedness rather than the motor response used to counteract the disturbance.

JNeurosci Accepted Manuscript

# References


Adkin AL, Quant S, Maki BE, McIlroy WE (2006) Cortical responses associated with predictable and unpredictable compensatory balance reactions. Exp Brain Res 172:85-93.

Adkin AL, Campbell AD, Chua R, Carpenter MG (2008) The influence of postural threat on the cortical response to unpredictable and predictable postural perturbations. Neurosci Lett 435:120-125.

Boebinger S, Payne A, Martino G, Kerr K, Mirdamadi J, McKay JL, Borich M, Ting L (2024) Precise cortical contributions to sensorimotor feedback control during reactive balance. PLoS Comput Biol 20:e1011562.

Bolton DA (2015) The role of the cerebral cortex in postural responses to externally induced perturbations. Neurosci Biobehav Rev 57:142-155.

Carpenter MG, Allum JH, Honegger F (1999) Directional sensitivity of stretch reflexes and balance corrections for normal subjects in the roll and pitch planes. Exp Brain Res 129:93-113.

Day BL, Steiger MJ, Thompson PD, Marsden CD (1993) Effect of vision and stance width on human body motion when standing: implications for afferent control of lateral sway. J Physiol 469:479-499.

Delorme A, Makeig S (2004) EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. J Neurosci Methods 134:9-21.

Di Giulio I, Maganaris CN, Baltzopoulos V, Loram ID (2009) The proprioceptive and agonist roles of gastrocnemius, soleus and tibialis anterior muscles in maintaining human upright posture. J Physiol 587:2399-2416.

Forbes PA, Luu BL, Van der Loos HF, Croft EA, Inglis JT, Blouin JS (2016) Transformation of Vestibular Signals for the Control of Standing in Humans. J Neurosci 36:11510-11520.

Friston K (2005) A theory of cortical responses. Philos Trans R Soc Lond B Biol Sci 360:815-836.

Friston K (2010) The free-energy principle: a unified brain theory? Nature Reviews Neuroscience 11:127-138.

Goodworth AD, Peterka RJ (2012) Sensorimotor integration for multisegmental frontal plane balance control in humans. J Neurophysiol 107:12-28.

Henry SM, Fung J, Horak FB (1998) EMG responses to maintain stance during multidirectional surface translations. J Neurophysiol 80:1939-1950.

Horak FB, Nashner LM (1986) Central programming of postural movements: adaptation to altered support-surface configurations. J Neurophysiol 55:1369-1381.

Hughes G, Desantis A, Waszak F (2013) Mechanisms of intentional binding and sensory attenuation: the role of temporal prediction, temporal control, identity prediction, and motor prediction. Psychol Bull 139:133-151.

Itti L, Baldi P (2009) Bayesian surprise attracts human attention. Vision Research 49:1295-1306.

Jacobs JV, Horak FB (2007) Cortical control of postural responses. J Neural Transm (Vienna) 114:1339-1348.

Jansen DNR, Mensink L, Leeuwis M, Forbes PA (2026) Cortical responses to balance perturbations persist without active postural control. J Physiol.

Keshner EA, Allum JH, Pfaltz CR (1987) Postural coactivation and adaptation in the sway stabilizing responses of normals and patients with bilateral vestibular deficit. Exp Brain Res 69:77-92.

Kiepe F, Kraus N, Hesselmann G (2021) Sensory Attenuation in the Auditory Modality as a Window Into Predictive Processing. Frontiers in Human Neuroscience Volume 15 - 2021.


JNeurosci Accepted Manuscript

Körding KP, Wolpert DM (2004) Bayesian integration in sensorimotor learning. Nature 427:244-247.
Leeuwis M, Asar Y, White JJ, Rasman BG, Forbes PA (2024) Different mechanisms of contextual inference govern associatively learned and sensory-evoked postural responses. Proceedings of the National Academy of Sciences 121:e2404909121.
Marlin A, Mochizuki G, Staines WR, McIlroy WE (2014) Localizing evoked cortical activity associated with balance reactions: does the anterior cingulate play a role? J Neurophysiol 111:2634-2643.
Mensink L, Nasrabadi AM, Rasman BG, Blouin JS, Forbes PA (2025) The sense and control of standing balance in the presence of motor noise. J Appl Physiol (1985) 139:918-933.
Mierau A, Hülsdünker T, Strüder HK (2015) Changes in cortical activity associated with adaptive behavior during repeated balance perturbation of unpredictable timing. Front Behav Neurosci 9:272.
Mirdamadi JL, Poorman A, Munter G, Jones K, Ting LH, Borich MR, Payne AM (2025) Excellent test-retest reliability of perturbation-evoked cortical responses supports feasibility of the balance N1 as a clinical biomarker. J Neurophysiol 133:987-1001.
Mochizuki G, Sibley KM, Cheung HJ, McIlroy WE (2009a) Cortical activity prior to predictable postural instability: is there a difference between self-initiated and externally-initiated perturbations? Brain Res 1279:29-36.
Mochizuki G, Sibley KM, Esposito JG, Camilleri JM, McIlroy WE (2008) Cortical responses associated with the preparation and reaction to full-body perturbations to upright stability. Clin Neurophysiol 119:1626-1637.
Mochizuki G, Sibley KM, Cheung HJ, Camilleri JM, McIlroy WE (2009b) Generalizability of perturbation-evoked cortical potentials: Independence from sensory, motor and overall postural state. Neurosci Lett 451:40-44.
Nashner LM (1976) Adapting reflexes controlling the human posture. Exp Brain Res 26:59-72.
Oude Nijhuis LB, Allum JH, Valls-Solé J, Overeem S, Bloem BR (2010) First trial postural reactions to unexpected balance disturbances: a comparison with the acoustic startle reaction. J Neurophysiol 104:2704-2712.
Parr JVV, Mills R, Kal E, Bronstein AM, Ellmers TJ (2024) A “Conscious” Loss of Balance: Directing Attention to Movement Can Impair the Cortical Response to Postural Perturbations. The Journal of Neuroscience 44:e0810242024.
Payne AM, Ting LH (2020) Balance perturbation-evoked cortical N1 responses are larger when stepping and not influenced by motor planning. J Neurophysiol 124:1875-1884.
Payne AM, Ting LH, Hajcak G (2019a) Do sensorimotor perturbations to standing balance elicit an error-related negativity? Psychophysiology 56:e13359.
Payne AM, Hajcak G, Ting LH (2019b) Dissociation of muscle and cortical response scaling to balance perturbation acceleration. J Neurophysiol 121:867-880.
Peterka RJ (2002) Sensorimotor integration in human postural control. J Neurophysiol 88:1097-1118.
Peterson SM, Ferris DP (2019) Group-level cortical and muscular connectivity during perturbations to walking and standing balance. NeuroImage 198:93-103.
Protzak J, Mirdamadi JL, Borich MR, Ting LH (2026) A primary central source determines perturbation-evoked N1 amplitudes in younger adults. Journal of Neurophysiology 135:1315-1323.
Qiao CZ, Nasrabadi AM, Partovi R, Belzner P, Kuo C, Wu LC, Blouin J-S (2023) Multidirectional Human-in-the-Loop Balance Robotic System. IEEE Robotics and Automation Letters.
Rao RP, Ballard DH (1999) Predictive coding in the visual cortex: a functional interpretation of some extra-classical receptive-field effects. Nat Neurosci 2:79-87.
Rasman BG, Blouin JS, Nasrabadi AM, van Woerkom R, Frens MA, Forbes PA (2024) Learning to stand with sensorimotor delays generalizes across directions and from hand to leg effectors. Commun Biol 7:384.

JNeurosci Accepted Manuscript

778 Sado N, Shiotani H, Saeki J, Kawakami Y (2021) Positional difference of malleoli-midpoint
779 from three-dimensional geometric centre of rotation of ankle and its effect on ankle
780 joint kinetics. Gait Posture 83:223-229.
781 Shibasaki H, Hallett M (2006) What is the Bereitschaftspotential? Clinical Neurophysiology
782 117:2341-2356.
783 Solis-Escalante T, Stokkermans M, Cohen MX, Weerdesteyn V (2021) Cortical responses to
784 whole-body balance perturbations index perturbation magnitude and predict reactive
785 stepping behavior. Eur J Neurosci 54:8120-8138.
786 Stokkermans M, Solis-Escalante T, Cohen MX, Weerdesteyn V (2022) Midfrontal theta
787 dynamics index the monitoring of postural stability. Cereb Cortex.
788 Stokkermans M, Solis-Escalante T, Cohen MX, Weerdesteyn V (2023) Distinct cortico-
789 muscular coupling between step and stance leg during reactive stepping responses.
790 Frontiers in Neurology 14.
791 Tanji J (1994) The supplementary motor area in the cerebral cortex. Neuroscience Research
792 19:251-268.
793 Todorov E (2004) Optimality principles in sensorimotor control. Nat Neurosci 7:907-915.
794 van Beers RJ, Sittig AC, Denier van der Gon JJ (1998) The precision of proprioceptive
795 position sense. Exp Brain Res 122:367-377.
796 Varghese JP, Beyer KB, Williams L, Miyasike-daSilva V, McIlroy WE (2015) Standing still: is
797 there a role for the cortex? Neurosci Lett 590:18-23.
798 Vlutters M, Boonstra TA, Schouten AC, van der Kooij H (2015) Direct measurement of the
799 intrinsic ankle stiffness during standing. J Biomech 48:1258-1263.
800 Welch TDJ, Ting LH (2014) Mechanisms of Motor Adaptation in Reactive Balance Control.
801 PLOS ONE 9:e96440.
802 Wolpert DM, Ghahramani Z, Jordan MI (1995) An internal model for sensorimotor
803 integration. Science 269:1880-1882.
804 Zaback M, Missen KJ, Adkin AL, Chua R, Inglis JT, Carpenter MG (2023) Cortical potentials
805 time-locked to discrete postural events during quiet standing are facilitated during
806 postural threat exposure. The Journal of Physiology 601:2473-2492.

807

808

JNeurosci Accepted Manuscript

**Figure 1. Experimental setup, protocol, and representative data. A**, Participants stood barefoot on a force plate mounted within a robotic balance simulator that reproduced the mechanics of upright stance as an inverted pendulum. Ankle torque exerted on the force plate was fed into a body mechanics simulation, which computed the resulting whole-body angle and prescribed the backboard (anteroposterior) motion and hip/shoulder harness (mediolateral) motion. The participant and robot are integrated together to replicate the natural sensorimotor control loop whereby the CNS integrates sensory signals to generate balance-correcting motor commands. Toes-up footplate perturbations were delivered in one of two ways: self-initiated by the participant pressing a handheld button (button icon), or unpredictably by the experimenter (question mark icon). In the No-Control condition (orange), the backboard was immobilized at -2.5° anterior whole-body angle with participants supported (lock icon), and in the Motion-variability condition the robot's movement was computer-controlled according to a pre-recorded motion profile (computer icon). **B**, Experimental protocol. Before the main experimental trials, participants first performed two baseline balancing sessions (not shown). Participants then completed either all No-Control (orange) or all Balance Control (blue) trials, with condition order counterbalanced across participants (6 and 5 participants, respectively). Both balance conditions included an Unexpected-pre block (15 trials), a Habituation block (60 self-initiated trials), and a final Unexpected-post block (15 trials). The No-Control condition also included an additional Motion-variability block (60 self-initiated trials) in which whole-body motion was externally imposed by the robot while participants had no control over their motion. The imposed motion was 1×, 2×, and 4× of balance variability (in that order) recorded during the Baseline trial. **C**, Representative single-participant data averaged across trials within each block. For the Habituation block, data are shown separately for the early (first 5 trials) and late (last 5 trials) phases. Rows show, from top to bottom: Cz EEG, tibialis anterior (TA) EMG, whole-body angular velocity, and footplate perturbation angle. The solid vertical line indicates perturbation onset; dashed vertical lines delimit the 100–200 ms window used to quantify the N1 response. For the Motion-variability column, three shades of orange indicate the 1×, 2×, and 4× motion levels.

**Figure 2.** EEG responses to balance perturbations. (**a**) Trial-by-trial N1 amplitude at Cz (mean ± SEM across participants) for Balance Control (blue) and No-Control (orange) conditions. Participants (n = 11) first received 15 unexpected perturbations (Unexpected-pre), followed by 60 self-initiated perturbations (shaded region), and finally 15 additional unexpected perturbations (Unexpected-post). (**b**) Group-averaged ERPs at Cz (± SEM) for three comparisons: *Self-initiation* (unexpected-pre versus the first 15 self-initiated trials);

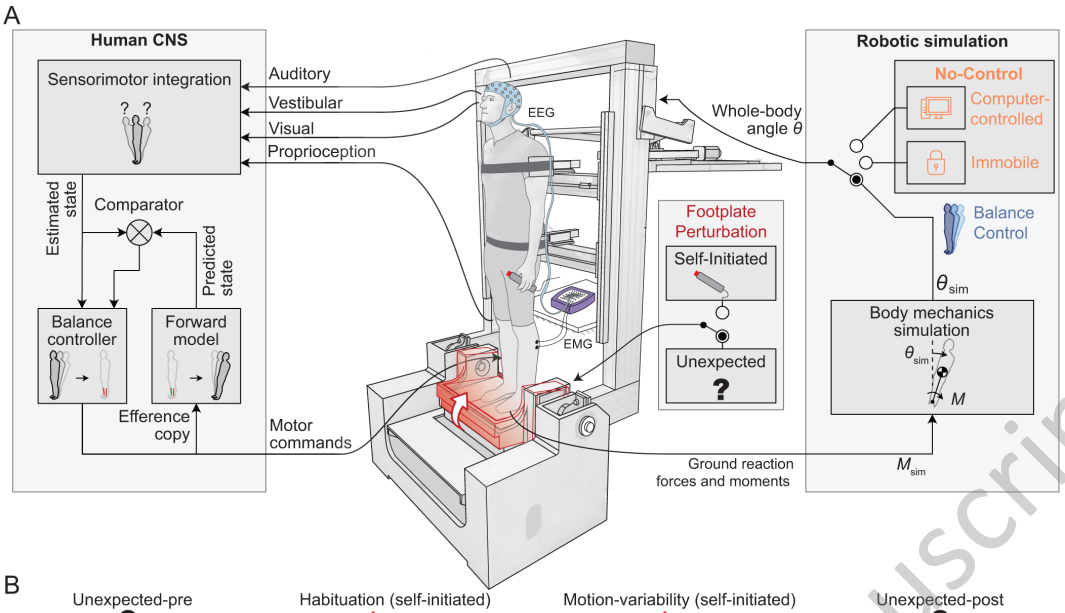
JNeurosci Accepted Manuscript

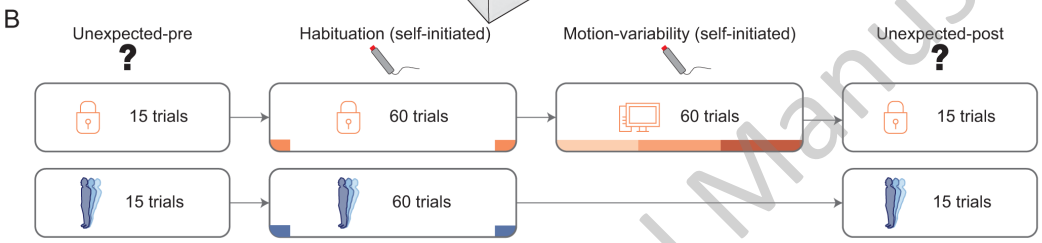
*Habituation* (first 5 versus last 5 self-initiated trials); *Generalization* (last 15 self-initiated trials versus unexpected-post trials). Left and right columns show Balance Control and No-Control conditions, respectively. (**c**) Individual participant N1 amplitudes with group mean (± SEM, grey symbol), for conditions shown in (b). Double asterisks (**) indicate a significant main effect ($p < .01$) for each comparison.

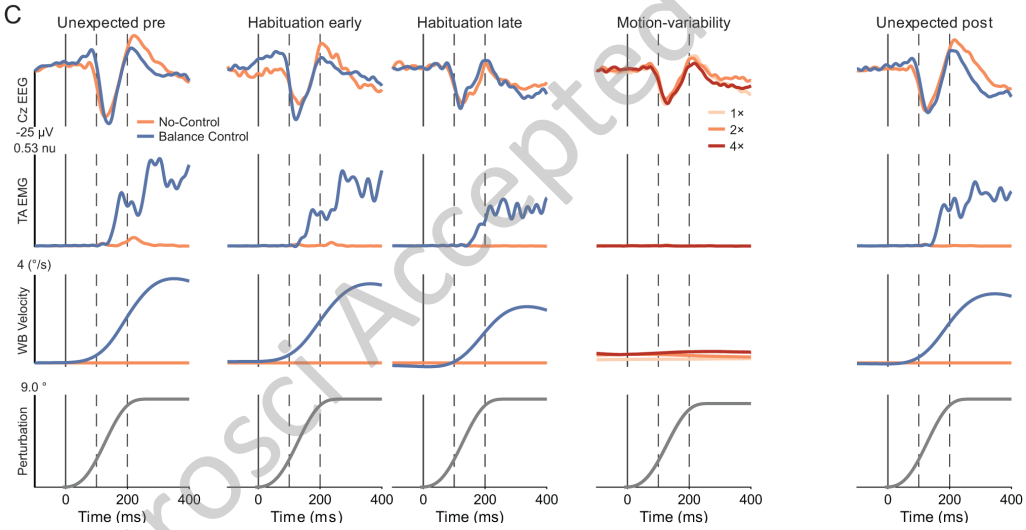
**Figure 3.** EEG responses at Cz across levels of imposed whole-body motion during self-initiated perturbations in the No-Control condition. (**a**) Mean N1 amplitude across participants (n = 11) over trials, for the no-motion baseline (last 20 self-initiated habituation trials) and the three imposed whole-body motion levels: no motion, 1×, 2×, and 4×. Shaded areas represent ±1 SEM. (**b**) Grand average ERPs for each level, with shaded areas indicating ±1 SEM. All conditions evoke a clear N1 response with highly similar characteristics across Motion-variability levels. (**c**) Individual participant means for each level. Grey symbols represent the group mean ± SEM. N1 amplitude did not increase when motion was imposed and was similar between motion levels.

**Figure 4.** Muscle responses to balance perturbations. (A) Trial-by-trial maximum tibialis anterior (TA) EMG amplitude (mean ± SEM across participants) for the Balance Control (blue) and No-Control (orange) conditions across the full experiment (n = 11). Participants first received 15 unexpected perturbations (Unexpected-pre), followed by 60 self-initiated perturbations (Habituation; shaded region), and finally 15 unexpected perturbations (Unexpected-post). The inset shows the peak whole-body posterior angular velocity per trial (mean ± SEM) for the Balance Control condition over the same 90 trials. (B) Group-averaged TA EMG time courses (± SEM) for three comparisons: *Self-initiation* (unexpected-pre versus the first 15 self-initiated trials); *Habituation* (first 5 versus last 5 self-initiated trials); *Generalization* (last 15 self-initiated trials versus Unexpected-post trials). Left and right columns show Balance Control and No-Control conditions, respectively. (C) Individual participant maximum TA EMG amplitudes with group mean ± SEM (grey), for each contrast in (B). Asterisks reflect pairwise comparisons from the significant interactions (* $p < .05$, ** $p < .01$, *** $p < .001$)

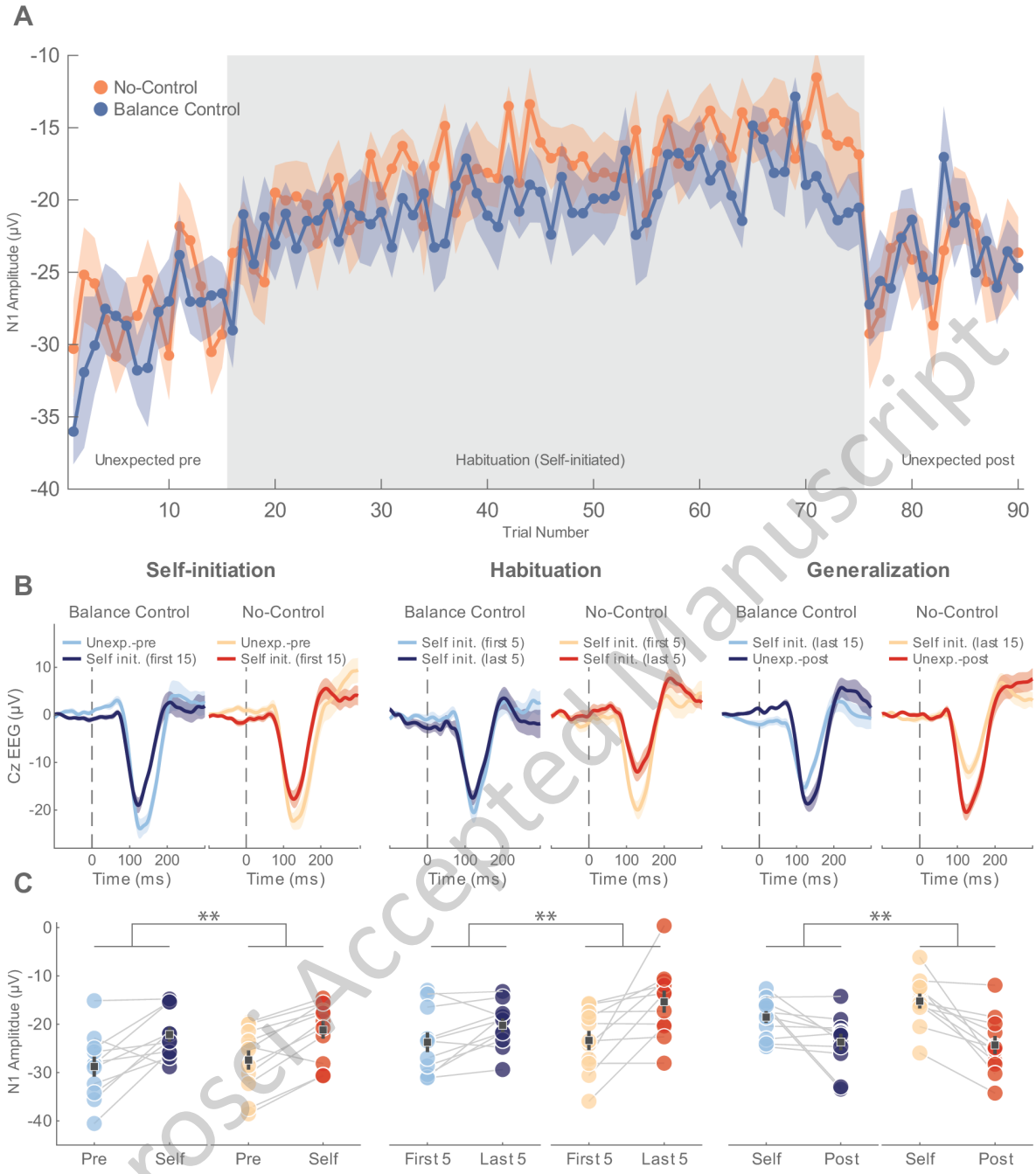
JNeurosci Accepted Manuscript



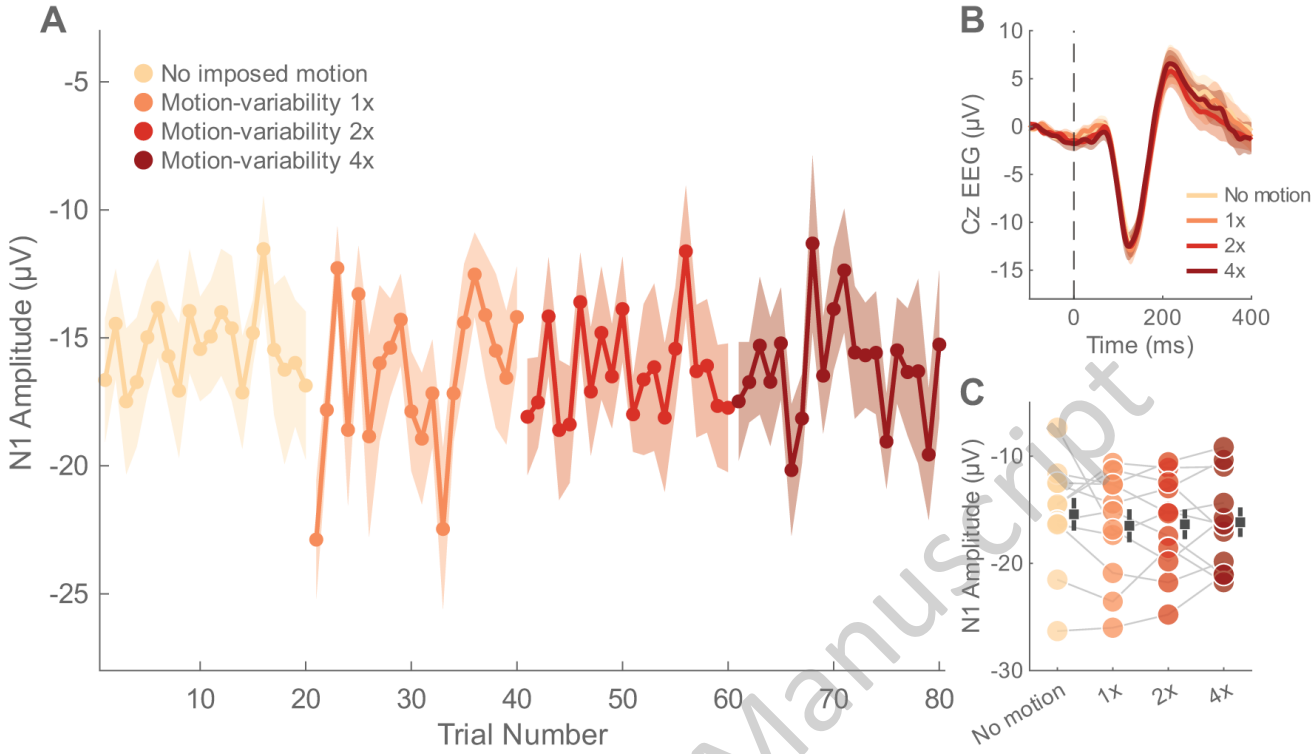



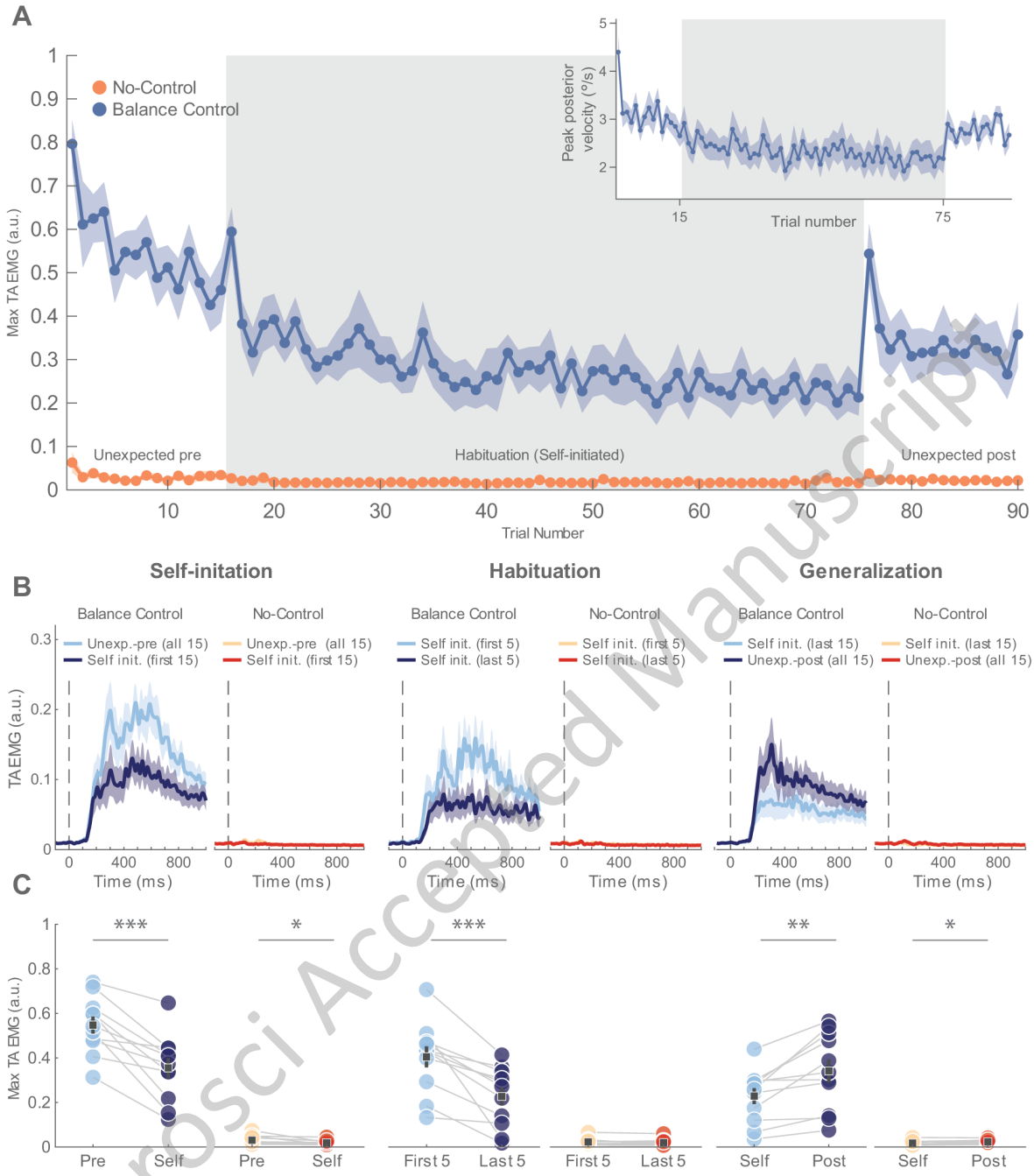

JNeurosci Accepted Manuscript




JNeurosci Accepted Manuscript




JNeurosci Accepted Manuscript




JNeurosci Accepted Manuscript