Temporal dynamics of early somatosensory processing in goal-directed actions

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Abstract

Tactile sensitivity is temporally modulated on a moving limb. Yet the functional significance of this modulation remains unclear. Using electroencephalography (EEG), we examined the temporal dynamics of early somatosensory processing in goal-directed reaching. Participants reached for their unseen left hand while brief vibrations were applied to the moving right index finger in different movement phases. Psychophysical results revealed tactile suppression during reaching, with sensitivity improving around maximum speed relative to the late movement phase. The EEG results were consistent with the psychophysical data. Specifically, the short-latency P45 component, originating in primary somatosensory cortex, was suppressed during reaching, but recovered around maximum speed. Reduced P45 amplitudes were associated with stronger tactile suppression measured behaviorally, linking early cortical activity to perceptual changes. This temporally specific modulation of tactile perception associated with early cortical processing suggests that the somatosensory system dynamically adapts its sensitivity to facilitate sensorimotor control in goal-directed actions.

Introduction

Goal-directed movements depend on somatosensory signals. For example, tactile sensations directly arise from physical interactions involving the moving limb, and proprioceptive and tactile feedback inform about limb position and movement dynamics. Some of these somatosensory signals provide essential information for sensorimotor control[1], whereas others may be irrelevant. Because feedback-based mechanisms alone are inherently noisy[2] and temporally delayed[3], the sensorimotor system likely integrates sensory feedback with predictive feedforward models using efference copies of motor commands to estimate future sensory states of the moving limb[4].

A well-documented perceptual phenomenon related to movement is a reduction in tactile sensitivity on the moving limb, known as tactile suppression[5]. Similar suppression effects have also been observed in the auditory[6] and visual[7] system. Tactile suppression has been demonstrated across a wide range of actions, including finger movements[5], isometric force production[8], reaching[9,10] and balance control[11]. Thus, it has been proposed that this is a fundamental principle of sensorimotor control. Electroencephalographic (EEG) studies complement behavioral findings, showing reduced amplitudes of somatosensory evoked potential (SEP) during movement, particularly at short-latency postcentral components like the P45[12–14], associated with early-stage processing in primary somatosensory cortex (S1)[15]. Such attenuation of SEP amplitudes, termed SEP gating, has also been reported at even shorter-latency components, like the N20, reflecting earlier cortical processing[13,16].

Crucially, tactile suppression is not an all-or-nothing phenomenon: When sensory input from the moving limb is relevant to successfully perform the movement, tactile sensitivity is preserved to some extent, although partial suppression may persist. For example, tactile suppression is less pronounced on the index finger when grasping an object with a precision grip, as opposed to other parts of the hand and arm[10,17,18], or when grasping a slippery versus a high friction object without seeing it[19]. Tactile perception also recovers on the lower leg shortly before dealing with

perturbations during a balancing task[20]. Likewise, tactile suppression is evident during targeted reaching movements without visual feedback, which rely exclusively on somatosensory inputs[21]. Importantly, tactile suppression during such reaching actions follows distinct temporal dynamics, as it is transiently released around the time of maximum speed[21], i.e., the onset of the deceleration phase when sensory guidance of the movement becomes particularly important[22]. Together, these findings demonstrate a flexible modulation of tactile suppression according to the functional relevance of somatosensory feedback. However, because these observations are based on behavioral measures only, it remains unclear whether changes in tactile perception reflect corresponding neural modulations in early somatosensory processing.

Electrophysiological studies provide some evidence for relevance-based modulation of SEPs from the moving limb: short-latency SEP amplitudes increase during exploratory hand movements compared to rest[23], and during lower limb movements in a balancing task when an upcoming perturbation challenges posture[24]. In a magnetoencephalographic study, the somatosensory evoked field component M38 was enhanced during grasping movements relative to rest and further increased during a more complex ball rotation task[25]. Collectively, these findings demonstrate that early somatosensory processing is facilitated when somatosensory signals are important for the task at hand. While these electrophysiological studies did not measure psychophysical aspects of tactile perception, they show that early somatosensory processing may underlie the modulation of tactile suppression during movement as sensory relevance changes.

To jointly examine the behavioral and neural correlates of this dynamic modulation, we combined psychophysics with EEG recordings of SEPs during a reaching task previously shown to modulate tactile perception[21]. First, we asked whether tactile sensitivity is modulated across different movement phases; second, whether early cortical processing indexed by the P45 component shows a corresponding temporal modulation; and third, whether temporal changes in tactile sensitivity are linked to changes in cortical processing. Based on prior evidence[13,14], we

predicted that movement-related tactile suppression would correspond to reductions in the early P45 component and that the time course of P45 amplitudes would mirror the temporal dynamics of suppression across movement phases.

To address these questions, we presented fixed-intensity vibrotactile probe stimulations to the right hand while participants reached to their left hand, without visual feedback. Because maximum speed denotes the onset of the period when sensory guidance of the hand toward the target gains importance[22], stimulations were categorized into four movement phases defined relative to maximum speed (pre-max speed, around max speed, early post-max speed, and late post-max speed). After the end of each movement, a comparison vibration of various intensities was delivered to the participants' sternum. Participants then judged which of the two stimuli felt stronger. The same task was performed in a rest condition without reaching movements to account for idiosyncrasies in tactile sensitivity. In both movement and rest conditions, tactile sensitivity was quantified as the point of subjective equality (PSE) derived from the behavioral responses, while cortical processes were assessed by extracting SEPs to the finger stimulation recorded with EEG (see Methods).

Results

To account for individual differences in tactile processing, movement-related changes were expressed as difference scores, obtained by subtracting PSEs and P45 amplitudes obtained during movement from those measured at rest (PSE_{diff} and $P45_{diff}$; see Methods). Positive PSE_{diff} and $P45_{diff}$ values reflect reduced tactile sensitivity and smaller SEP amplitudes, respectively, during movement relative to rest.

To test tactile suppression during movement, we compared PSE_{diff} from each movement phase to zero using paired t-tests. Tactile sensitivity was suppressed relative to rest, with PSE_{diff} significantly greater than zero in all phases of the movement: pre-max speed ($t(33) = 3.47$, $p =$

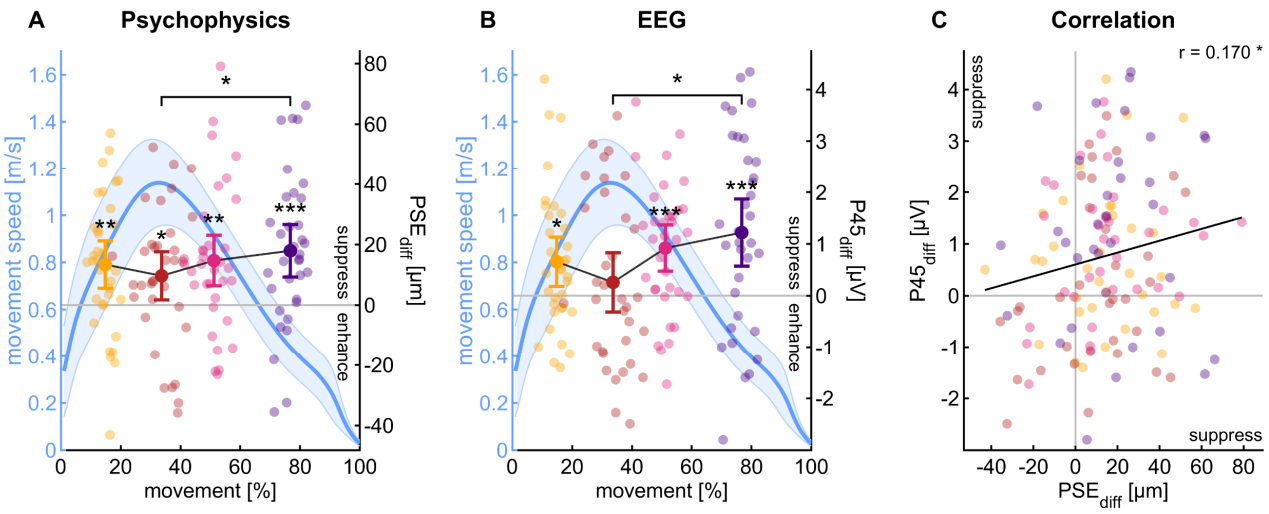

Fig. 1: Movement-related suppression in tactile perception and SEP amplitude.

Average time-normalized movement speed across participants (n = 34; blue line, left y-axis). Shaded area indicates standard error. The right y-axis shows mean PSE_{diff} (A) and $P45_{diff}$ (B) for the four movement phases: pre-max speed (yellow), around max speed (red), early post-max speed (pink), and late post-max speed (purple). Error bars represent 95% confidence intervals. Dots illustrate individual participants. **A**. Tactile perception was suppressed in all four movement phases (paired t-tests against zero). Tactile suppression was weaker around max speed relative to the late post-max speed phase (univariate ANOVA, paired t-tests). **B**. P45 amplitude was comparable to rest around max speed and suppressed in all other movement phases (paired t-test against zero). $P45_{diff}$ was smaller around max speed than in the late post-max speed phase, indicating less SEP gating mid-reach relative to later in the movement (univariate ANOVA, paired t-tests). **C**. Scatterplot of $P45_{diff}$ versus PSE_{diff}. Each point represents one participant in one movement phase. The black line shows the pooled ordinary least squares regression of $P45_{diff}$ on PSE_{diff} across all observations. Stronger tactile suppression during movement was significantly associated with lower P45 amplitude (Pearson correlation). * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.

0.002, $d_z = 0.59$), around max speed ($t(33) = 2.48$, $p = 0.019$, $d_z = 0.42$), early post-max speed ($t(33) = 3.59$, $p = 0.002$, $d_z = 0.62$), and late post-max speed ($t(33) = 4.20$, $p < 0.001$, $d_z = 0.72$). A repeated-measures ANOVA used to assess the temporal dynamics of suppression revealed a main effect of movement phase ($F(3, 99) = 4.66$, $p = 0.004$, $\eta^2_p = 0.12$). Post-hoc comparisons showed weaker suppression (i.e., improved sensitivity) around max speed than in the late post-max phase ($t(33) = -3.3$, $p = 0.014$, $d_z = -0.57$), in line with previous findings[21]. Suppression did not differ significantly between the other movement phases (all $t(33) \geq -2.5$, $p \geq 0.053$, $d_z \geq -0.43$; see Fig. 1A).

We focused the EEG analysis on electrode CP3 located over S1 contralateral to the stimulated right hand, since P45 amplitude is maximal at this site [26]. Figure 2A shows the waveforms, and Figure 2B the respective scalp maps. To assess SEP gating during movement, we tested $P45_{diff}$ against zero with paired t-tests. $P45_{diff}$ was greater than zero for most movement phases, reflecting gating of the P45: pre-max speed ($t(33) = 2.78$, $p = 0.010$, $d_z = 0.48$), early post-max speed ($t(33) = 4.18$, $p < 0.001$, $d_z = 0.72$), and late post-max speed ($t(33) = 3.83$, $p < 0.001$, $d_z = 0.66$). Importantly, $P45_{diff}$ was not significantly different from zero around maximum speed ($t(33) = 0.91$, $p = 0.367$, $d_z = 0.16$), indicating a transient recovery of early cortical processing around the onset of reach deceleration. These findings demonstrate gating of somatosensory signals during movement, except during the time when sensory guidance of the hand gains importance.

A repeated-measures ANOVA revealed a temporal modulation of $P45_{diff}$ across movement phases ($F(3, 99) = 3.35$, $p = 0.044$, $\eta^2_p = 0.092$). Post-hoc t-tests showed significantly smaller $P45_{diff}$ around max speed than in the late post-max phase ($t(33) = -2.53$, $p = 0.049$, $d_z = -0.43$), supporting the recovery of processing around the time of maximum speed, consistent with the psychophysical results. No significant differences were observed in $P45_{diff}$ between the other movement phases (all $t(33) \geq -2.41$, $p \geq 0.066$, $d_z \geq -0.41$; see Fig. 1B). Lastly, a significant Pearson correlation between PSE_{diff} and $P45_{diff}$ ($t(134) = 2.00$, $p = 0.048$, $r = 0.170$) revealed that stronger tactile suppression in the PSEs was associated with reduced P45 amplitudes (see Fig. 1C).

Taken together, these findings suggest that early cortical processing contributes to the temporal modulation observed in tactile suppression during reaching. Perceptual reports, however, are shaped not only by early cortical processes, but also by higher-order cognitive mechanisms (e.g., stimulus evaluation and decision-making processes[27].

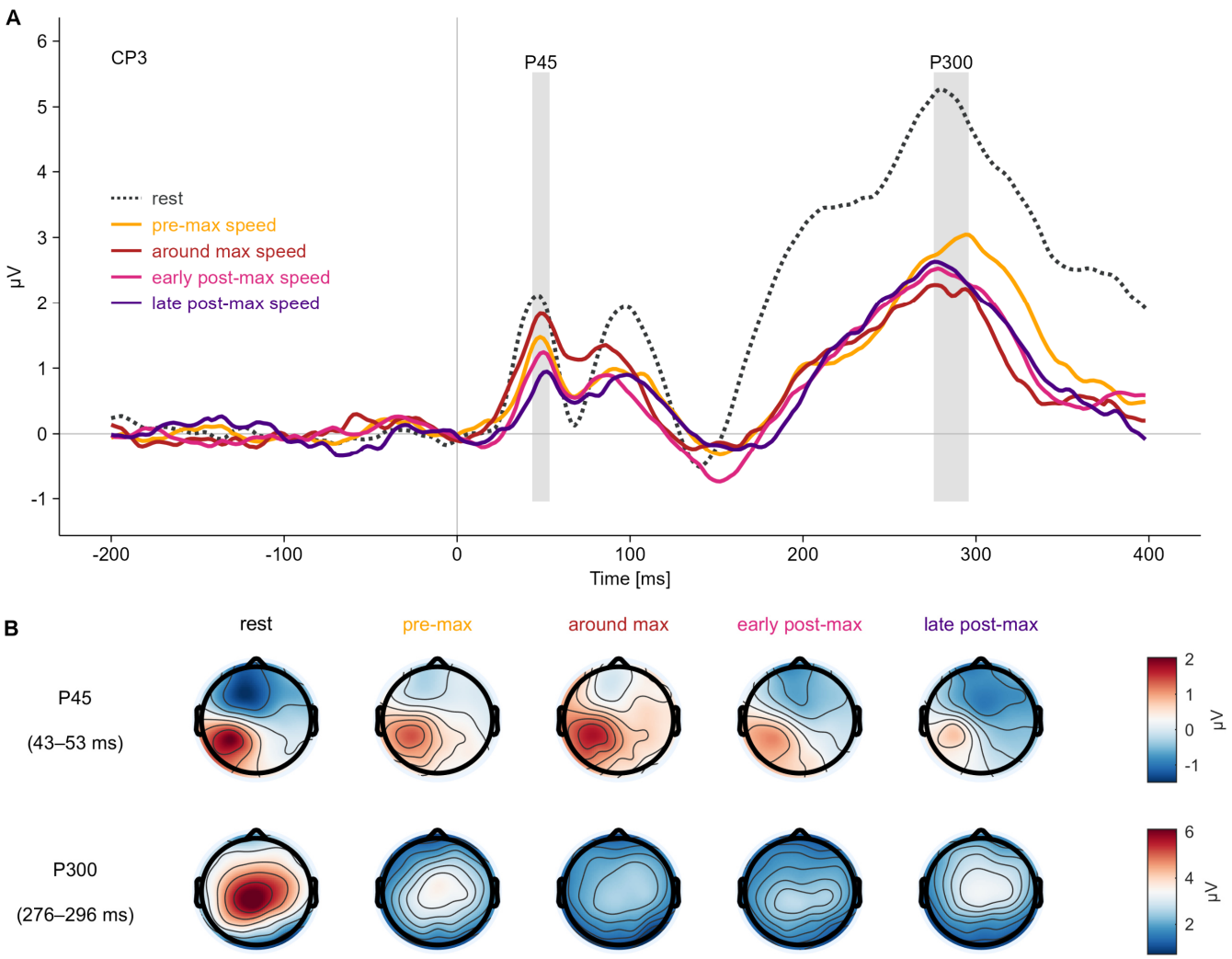

Fig. 2: Grand-averaged SEPs during rest and movement.

A. Grand-averaged SEPs at CP3 (n = 34), time-locked to standard vibration onset, with P45 and P300 highlighted. **B**. Scalp topographies for the P45 and P300 across the rest condition and movement phases.

To test whether later cortical mechanisms likewise exhibited dynamic changes across the movement, we also examined SEP gating in the P300 component (see Fig. 2). $P300_{diff}$ was significantly greater than zero in all phases, reflecting consistent gating of the P300 throughout the movement: pre-max ($t(33) = 3.97$, $p < 0.001$, $d_z = 0.68$), around max ($t(33) = 3.95$, $p < 0.001$, $d_z = 0.68$), early post-max ($t(33) = 4.81$, $p < 0.001$, $d_z = 0.82$), and late post-max ($t(33) = 2.85$, $p = 0.010$, $d_z = 0.49$). Additionally, in contrast to the PSE_{diff} and $P45_{diff}$, the $P300_{diff}$ did not significantly differ between the four movement phases ($F(2.08, 68.59) = 0.43$, $p = 0.658$, $\eta^2_p = 0.013$), i.e., we found no temporal modulation at this later stage of somatosensory processing.

Discussion

We report temporal dynamics in tactile suppression across a goal-directed reaching movement, reflected in weaker suppression around maximum speed. This temporal modulation of perception was paralleled in early somatosensory processing. As expected, P45 amplitudes were reduced during movement relative to rest, consistent with previous EEG evidence of gating at early stages of cortical processing[12–14]. Crucially, however, there was one exception: P45 responses near maximum speed were comparable to rest and significantly larger than later in the movement. Moreover, reductions in P45 amplitude correlated with stronger tactile suppression, suggesting a link between changes in tactile sensitivity during movement and early somatosensory processes. In contrast, P300 amplitudes were consistently reduced during movement and did not vary across movement phases. This suggests that the reported temporal modulation is unlikely to be caused by cognitive processes, but reflects a genuine regulation of sensorimotor processes.

Our findings are in line with previous reports of temporal modulation in sensory processing during reaching. For instance, feedback signals from a moving arm are temporally regulated during reaching movements, with distinct phases of strong upregulation[28,29]. In the context of tactile perception, increased sensitivity has been reported in the swing phase of a kicking leg[30]. Similarly, a previous study using the same task reported a complete release from suppression in this movement phase, with tactile sensitivity around maximum speed matching sensitivity at rest[21]. In contrast, we observed tactile suppression throughout all phases of the movement, including during maximum speed. This discrepancy may reflect differences in psychophysical task demands: earlier work used a binary detection response immediately after movement offset[21], whereas our delayed comparison task involved memorizing the standard stimulus and evaluating it against a second vibration several seconds after movement completion. The P300 is known to be sensitive to cognitive demands related to memory and stimulus maintenance, particularly under dual-task conditions[31]. In our study, it was consistently gated across all movement phases, suggesting the cognitive load of retaining the

stimulus in memory during the movement may have sustained perceptual tactile suppression, even at phases of preserved early sensory processing. Critically, however, the P45 component provides a temporally precise index of sensory encoding tied to low-level stimulus features such as intensity[32,33] and is unaffected by memory demands or decision processes. Here, we observed a clear preservation of P45 amplitudes around maximum speed, comparable to rest. Thus, while overall perceptual suppression was stronger in our task, perhaps due to higher-order cognitive mechanisms, both studies converge on the same temporal pattern of a transient release from suppression around maximum speed. Our findings extend earlier observations[21] by linking perceptual modulation during movement to changes in early sensory processing.

Our results align with the theory of optimal feedback control, which proposes that feedback gains are flexibly modulated based on state-dependent task demands during movement[34,35]. Consistent with this, visuomotor gains peak mid-reach and decrease upon approaching the target, scaling with distance to the target rather than distance traveled[29]. Tactile signals play a critical role in the transport phase of reaching[36], possibly by providing proprioceptive cues to guide the movement[37,38]. Thus, the reduction in tactile suppression around maximum speed may reflect a transient upregulation of somatosensory feedback to support precise control during the shift from movement initiation to feedback-driven movement deceleration towards the target[22]. Alternatively, rather than reflecting a genuine release from suppression mid-reach, the temporal modulation we observed may primarily result from stronger suppression later in the movement, as the motor system shifts from position control to movement termination[34] or reduces sensitivity in anticipation of contact with the target[39]. However, our SEP results argue against a purely late-suppression account, since only the mid-reach phase showed P45 amplitudes comparable to rest, whereas earlier and later phases were clearly gated. This pattern highlights mid-reach as a distinct processing window, rather than simply an early point before late-phase suppression emerges. While anticipatory suppression toward approaching the target may still contribute, our data are consistent with the idea that

heightened somatosensory demands during the transition to feedback-driven motor control selectively preserve early somatosensory processing mid-reach.

Importantly, the reduction in tactile suppression we observed around maximum speed is counter to previous findings showing that higher movement speed increases suppression[40,41] and reduces cortical somatosensory responses[14,42], perhaps due to masking by stronger reafferent signals[8,43]. These effects were observed in studies involving isolated flexion or extension about a single joint, which lack precision demands. In contrast, suppression during complex movements requiring precision appears unaffected by increased reafferent input from stronger forces[44]. In our paradigm, the goal-directed nature of the movement toward a somatosensory target without visual feedback likely increased the relevance of tactile information just before the onset of the deceleration phase. Our findings indicate that suppression is not merely a function of movement parameters like speed, and support the idea that it is modulated by the task-dependent relevance of somatosensory input[10,18,45].

This also aligns with previous evidence that short-latency SEPs on a moving limb are modulated by movement demands. For example, facilitated short-latency responses have been reported during balance control, exploratory movements, and precision tasks[23–25,46]. Further evidence that somatosensory processing is modulated according to dynamic task demands across movement phases comes from gait studies, which report phase-dependent suppression and facilitation of neural responses across the step cycle[47–49], and corresponding variations in tactile sensitivity[50]. Our results extend this literature by showing that SEP amplitudes also vary across the time course of a somatosensory-guided reaching movement, and by relating these neural modulations to concurrent changes in perception.

Unlike traditional SEP paradigms that use transcutaneous nerve stimulation to activate a diverse population of mixed fibers[51], our approach employed mechanical vibrations in the preferred range of Pacinian mechanoreceptors, which selectively engage RAII-type Aβ fibers[52,53]. This

stimulation enabled us to examine the modulation of early cortical responses associated with naturalistic, low-threshold mechanoreceptor signals, rather than the broad, temporally synchronized activation of motor and other sensory fibers[54]. While our stimulation selectively targeted Pacinian mechanoreceptors, the reaching movement likely generated input from a range of cutaneous and proprioceptive receptors. The P45 component, where we observed gating effects, arises primarily from areas 1 and 3b of S1[15,55,56], with possible contributions from secondary somatosensory cortex (S2)[57]. Although areas 1 and 3b are often considered modality-specific, widespread cutaneous-proprioceptive integration has been reported across S1, with linear multimodal effects emerging as early as 20 ms post-stimulation[58]. Thus, the P45 may reflect not only RAII-driven responses but also integration with other afferent sources. Given this convergence, the modulation in early cortical gating we observed may reflect broader regulation of somatosensory input, in addition to selective tuning of RAII activity.

Since movement-related gating of somatosensory responses has been documented at multiple earlier stages of the ascending pathway, from first-order synapses in the spinal dorsal column to thalamic nuclei[42,59,60], the reductions in P45 amplitude observed here may reflect decreased afferent inflow into S1. At the spinal level, gating of somatosensory input is mediated by presynaptic inhibition exerted by local interneurons[61]. In primates, this presynaptic inhibition is at least partly driven by descending pathways from the brainstem and cortex, and emerges before EMG onset of voluntary movement[62]. Consistent with this, in the rodent whisker system, movement-related gating at the first synaptic stage is abolished when S1, S2, and posterior parietal cortex are lesioned, indicating top-down control of sensory gating already at the first synaptic stage[63]. Converging evidence from humans likewise implicates S1, S2, posterior parietal cortex, and supplementary motor area in down-regulating somatosensory perception during movement[64,65]. While these cortical areas may be involved in top-down control of presynaptic inhibition, the observation that gating can be stronger at cortical than spinal levels in primates supports the idea that additional cortico-cortical

235 mechanisms modulate sensory gain[60]. For example, in rodents, primary motor cortex activates
236 inhibitory neurons in primary auditory cortex to selectively attenuate expected, self-generated
237 sounds[66], illustrating how motor-related signals can recruit local inhibitory circuits in sensory cortex.
238 Together, these findings are consistent with cortical control of tactile feedback gains across the
239 course of a goal-directed movement, with descending pathways regulating incoming afferent signals
240 throughout the dorsal column-medial lemniscal pathway and intracortical circuits further shaping
241 early cortical responses. Consequently, the transient release from P45 gating we observed around
242 maximum speed may reflect a temporary relaxation of presynaptic gating along the pathway, a
243 reduction of intracortical inhibition, or both. To answer this, future studies combining cortical
244 measures with subcortical recordings will be needed to determine whether this modulation extends
245 throughout the somatosensory pathway, and to compare its strength across spinal and supraspinal
246 levels.

247 These gating mechanisms are part of a broader bidirectional sensorimotor control loop in
248 which motor commands shape sensory inflow, while somatosensory feedback, in turn, shapes motor
249 output[67]. At the spinal level, somatosensory afferents interface with interneuron circuits that regulate
250 motoneuron excitability via presynaptic inhibition[61], and these inhibitory interneurons receive
251 corticospinal projections from motor and sensory cortices, placing the modulating effect of afferent
252 input under top-down cortical control[68]. When this presynaptic inhibition is disrupted by genetic
253 ablation of the corresponding interneurons, goal-directed reaching in rodents deteriorates, consistent
254 with a failure to appropriately down-regulate proprioceptive feedback gains[69]. Conversely, when
255 cutaneous input is blocked by local anesthesia, the reaching component of grasping is impaired and
256 hand paths become more variable[36], indicating that both excessive and absent somatosensory
257 feedback can disrupt accurate motor control. Together, these findings suggest that sensory gain is
258 dynamically tuned to current task demands, and that the modulation in gating of early cortical
259 responses observed here reflects such task-dependent adjustments of reafferent gain, suppressing

redundant somatosensory input while allowing behaviorally relevant feedback to influence motor circuits[67]. Consequently, the changes in perceived tactile intensity during movement may represent the behavioral expression of this distributed sensorimotor gating[67,70].

Limitations

One limitation concerns the potential overlap of SEPs with other task-related events such as movement offset and target contact. This overlap would primarily affect the later P300 component, whereas the earlier P45 likely occurred mostly during ongoing movement across all phases. Even so, because these overlapping events are not systematically aligned with stimulation timing, any influence they exert should be random across the SEP waveform and thus contribute as noise rather than as a systematic bias. To further mitigate these effects, we subtracted the EEG signal recorded during reaching alone from that recorded during reach with tactile stimulation. The subtracted signal was time-locked to the corresponding stimulation timing post movement onset and therefore included the same unsystematic movement-related activity present during the stimulation epochs. We acknowledge that this approach assumes an additive relationship between movement- and stimulation-related signals, although prior work has questioned this assumption[71]. Nevertheless, the resulting SEP waveforms closely corresponded to canonical patterns reported for vibrations, including a fronto-parietal dipole for the P45, and a centro-parietal positivity for the P300[32,72,73], supporting the validity of the method.

Conclusion

In sum, our findings provide clear evidence that tactile suppression is not uniform throughout movement but is temporally tuned, with a distinct reduction at the moment of maximum speed. Crucially, this temporal modulation of tactile sensitivity was accompanied by changes in early somatosensory processing, suggesting a selectively preserved engagement of S1 when somatosensory input from the moving limb is highly relevant for the sensory guidance of motor output. This supports the view that suppression is flexibly tuned to task demands, with reduced

gating mid-reach likely reflecting an upregulation of tactile feedback to control a somatosensory-guided movement. Further, our findings suggest that higher-order processes contribute to a general dampening of tactile perception but not to its fine-grained temporal tuning. This novel relation of psychophysical modulation to early neural markers underscores that tactile suppression is a flexible, context-sensitive mechanism tightly coupled to sensorimotor demands.

Materials and Methods

Experimental Design

Vibrations (20 ms, 280 Hz) were delivered using a vibrotactile stimulation device (PiezoTac tactor; Engineering Acoustics Inc., Casselberry, FL, USA). The tactor comprised a skin contactor (6.4 mm diameter) mounted on a shielded cantilever. Cantilever vibrations produced sinusoidal oscillations perpendicular to the skin. One contactor was placed on the palmar side of the proximal phalanx of the right index finger, leaving the fingertip uncovered. A second contactor was positioned on the sternum, approximately 3 cm below the clavicle. The contactors were fixed to the participant's skin using medical tape. The experiment was controlled by custom MATLAB (The MathWorks Inc., Natick, MA, USA) software using the Psychtoolbox extension[74]. The position of an infrared marker on the nail of the right index finger was recorded at 250 Hz with an Optotrak Certus motion tracking system (Northern Digital Inc., Waterloo, ON, Canada).

Procedure

Participants placed both hands on a table in front of them. The left hand rested palm-down with the thumb and index finger on specific markers positioned 10 cm to the left of the body midline, to ensure consistent placement. The right hand rested on a keypad located 18 cm to the right of the midline and 20 cm in front of the participant. Throughout the experiment, participants' view of their hands was occluded using a tilted cardboard screen, which allowed them to see the top of the computer monitor for instructions. At the beginning of each trial, participants held down a key with their right index finger to initiate the trial. Text on the computer screen then indicated the target

finger on the left hand (thumb or index), and participants had 2500 ms to initiate the movement. Upon releasing the key, participants performed a reaching movement with the right hand to touch the nail of the specified target finger. A standard vibration (peak-to-peak displacement: 119 µm) was delivered to the moving right index finger at one of five possible latencies relative to movement onset (50, 150, 250, 350, or 450 ms), which was determined by the key release. After reaching the target, participants rested their right hand next to the left hand on the table. A comparison vibration was delivered to the sternum 3000 ms after the standard vibration, well after movement completion, while both hands were still. The comparison stimulus had one of seven possible intensities (peak-to-peak displacement: 47, 71, 95, 119, 143, 167, or 192 µm). Participants indicated which of the two vibrations felt more intense by pressing foot pedals: the left pedal for the first vibration (on the hand) and the right pedal for the second vibration (on the sternum; see Fig. 3 for the trial sequence). Each stimulation time was presented 70 times for a total of 350 trials. Additionally, 70 trials (16.67% of all trials) were included to record movement-related EEG without vibrotactile stimulation, matching the number of trials for each stimulation time. In these omission trials, participants were informed that no vibration was given 3000 ms after movement onset and no tactile discrimination response was required. Participants completed 10 blocks of 42 trials each. Within each block, all combinations of stimulation time and comparison intensity were presented once, along with seven omission trials in random order. Trials were repeated at the end of the block if participants initiated the movement too early, less than 180 ms after target presentation, suggesting the cue had not been processed, or if they failed to initiate movement within 2500 ms after the cue. Self-paced breaks were provided between blocks. Before starting the experiment, participants familiarized themselves with the reaching movement and the discrimination task in 20–40 practice trials.

We also assessed tactile sensitivity at rest by asking participants to perform the discrimination task without movement, while both hands remained on the table in the same position

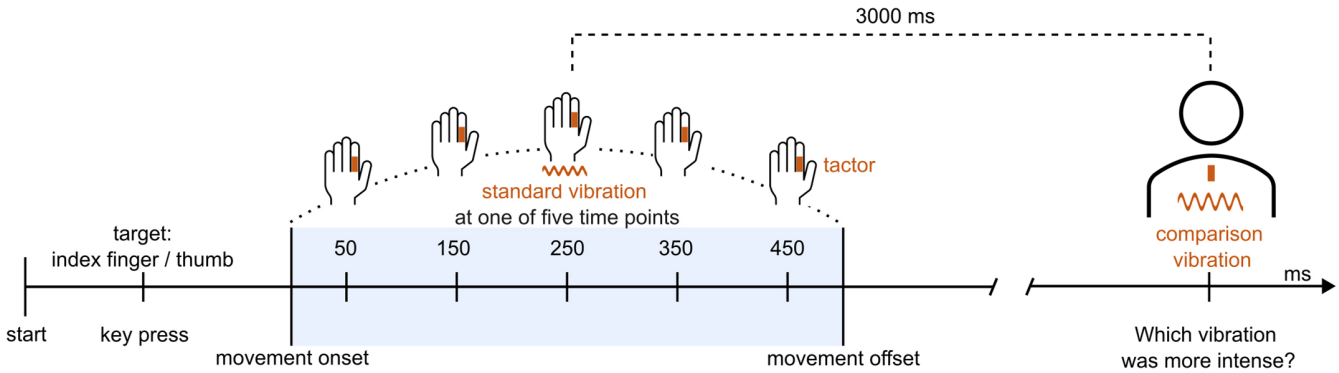

Fig. 3: Trial Sequence.

Participants initiated each trial by pressing and holding a key with the right index finger. Text on the monitor indicated the target finger of the left hand. Participants released the key and reached toward the target finger of the left hand. A standard vibration of fixed amplitude was delivered to the right index finger at one of five time points during the movement (250 ms shown here). After an ISI of 3000 ms, a comparison vibration of varying amplitude was presented on the sternum. Participants judged which vibration felt stronger using foot pedals. Participants also performed the same psychophysical task with both hands at rest.

adopted after the reaching movements. This enabled us to assess relative changes between rest and movement phases and determine the effect of movement on tactile processing. The standard vibration was presented to the right index finger at one of five delays (50, 150, 250, 350, or 450 ms) after a fixed interval of 700 ms from trial onset, replicating the relative timing used during movement. A comparison vibration followed on the sternum 3000 ms later. Participants judged vibration intensity using foot pedals. Each of the seven comparison intensities was presented twice per stimulation time (70 trials total). Trials ended only after participants had responded and at least 6000 ms had elapsed from trial onset, ensuring a trial duration consistent with the reaching block. Trials started automatically after a foot pedal was pressed. The rest condition was divided into two blocks, separated by a short break, and both blocks were administered either before or after the movement blocks, counterbalanced across participants.

Participants

Thirty-eight participants (self-reported gender: 22 female, 16 male; mean age: 25.7 years, SD = 4.1) provided written informed consent and received either course credit or 10 €/hour as

compensation. The study was approved by the ethics committee of the Justus Liebig University Giessen and conducted in accordance with the Declaration of Helsinki[75], with the exception of pre-registration. All participants were right-handed, as assessed by the Edinburgh Handedness Inventory[76] (M = 90.8, range = 28.6–100). Two participants were excluded for slow movements, leaving too few stimulus presentations in the late post-max speed phase to fit psychometric functions (see Kinematic Analysis); one participant was excluded because 86% of trials were rejected (see Kinematic Analysis); one participant was excluded as a statistical outlier (see Statistical Analysis).

Kinematic Analysis

The kinematic data were analyzed in MATLAB (R2021a; The MathWorks Inc., Natick, MA). Movement speed was computed by numerical differentiation of the three-dimensional data divided by the sampling interval of 4 ms. Movement onset was defined as the key release. Mean reaction time was 547 ± 240 ms (standard deviation across participants). Movement offset, i.e., when participants touched the target finger, was determined using the multiple sources of information method to form a composite score[77]: Coarse spatial constraints excluded frames with lateral positions near the start zone (all positions within 20 cm to the left and 10 cm to the right of the start position were excluded). To lower the likelihood of frames after touching the target finger, frames were down-weighted when the moving index finger was within 3 cm of the location on the table where participants rested their hand after the touch. A timing gate increased the likelihood of a frame being the end of the movement once speed fell below 0.10 m/s after the speed maximum (identified within 400 ms following movement onset) and maintained this elevated score for 80 ms. Lower instantaneous speed, smaller x (toward the target hand), and larger y (further from the body) also increased the score. The movement end was defined as the frame with the highest composite score likelihood.

Movement duration (M = 562 ms, SD = 100 ms) was defined as the time between movement onset and offset. Time of maximum movement speed was determined for each trial. To account for

inter-individual differences in movement duration, the timing of maximum speed was normalized to the total movement duration in each trial. Across participants, the average maximum speed was 1.22 ± 0.19 m/s, occurred after 202 ± 62 ms and at 33 ± 6% of the movement. Stimulation trials were then assigned to four bins defined relative to the time of maximum speed: (1) pre-max speed (2) window of ± 5% around max speed (3) early post-max speed, the first 40% of the remaining post-max duration; and (4) late post-max speed, the final 60% of the remaining post-max duration. These time bins will be referred to as the four movement phases. We used a 40/60 rather than 50/50 proportion to divide the post-max time to balance trial counts: The average movement duration was 562 ms, while the latest stimulation occurred 450 ms after movement onset, which resulted in too few late-phase trials for stable psychometric fits under a 50/50 post max speed split in eight participants. The 40/60 proportion allowed for stable psychometric fits in seven of these eight participants. Critically, the 40/60 split also yielded more balanced trial counts per movement phase, with phases 1–4 containing on average 32.9%, 13.7%, 27.1%, and 26.3% of trials, respectively. We allowed fewer trials in the small window around maximum speed to maintain temporal specificity in this movement phase.

Trials were excluded from all further analyses if the movement end could not be determined (empty composite score; < 1% of trials), if >25% of motion-tracking samples were missing in the last third of the movement due to marker occlusion (1% of trials), if the mean speed during the first 20% of the movement was below 10 m/s (1% of trials), indicating delayed movement initiation after start key release and incorrectly timed tactile stimulations, if the movement duration was >3 SD above the participant's within-block mean (1% of trials), or if the stimulation occurred after the movement had ended (4% of trials).

Psychophysics Analysis

Logistic psychometric functions were fitted to participants' responses in the psychophysical task using the maximum likelihood estimation implemented in the Psignifit 4 toolbox[78] in

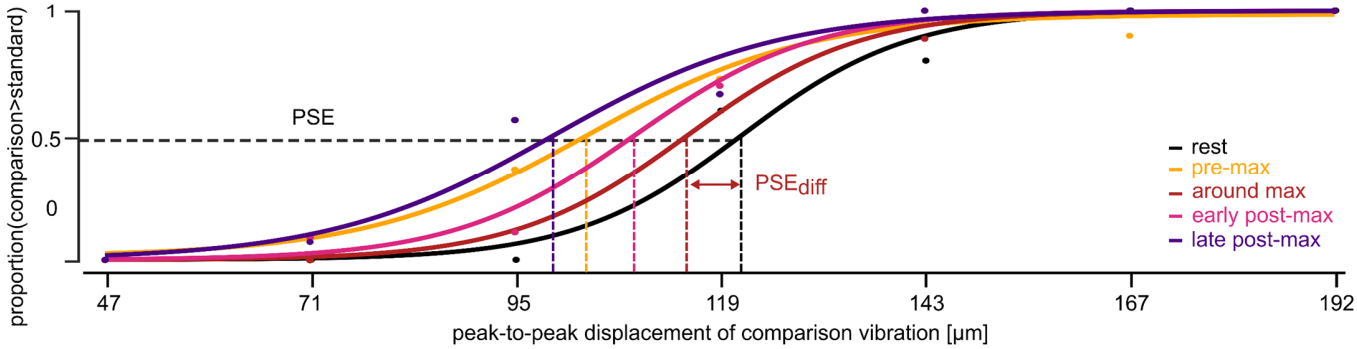

Fig. 4: Psychometric curves of one example participant.

Psychometric curves were fitted to the participant's responses in the comparison task, both for the rest condition and for each movement phase. The PSE was determined as the 50% point of each curve. To control for individual differences in tactile sensitivity, the PSE from each movement phase was subtracted from the PSE in the rest condition, yielding one difference score per movement phase (PSE_{diff}).

MATLAB. Separate functions were fitted for each participant, both for the four movement phases and for the resting condition. Across participants, the mean number of trials included in the rest condition, pre-max, around max, early post-max, and late post-max phases was 70, 108, 45, 88, and 83, respectively. PSEs were derived from the 50% point of each function. To control for inter-individual differences in tactile intensity discrimination, the PSE in each of the four movement phases was subtracted from the corresponding values in the rest condition to compute four normalized difference scores (PSE_{diff}) per participant (see Fig. 4).

EEG Analysis

EEG data were recorded from 37 channels (Fp1, C3, P3, P4, C4, AF7, AF3, AFz, F1, F5, FT7, FC3, C1, C5, TP7, CP3, P1, P5, PO7, PO3, POz, PO4, PO8, P6, P2, CPz, CP4, TP8, C6, C2, FC4, FT8, F6, AF8, AF4, F2, FCz) placed according to the international 10–20 system, using a BrainProducts actiCap with a 64-channel LiveAmp system and BrainVision Recorder software (Brain Products GmbH, Gilching, Germany), sampled at 500 Hz. In addition, electrodes were placed at the outer canthi of both eyes and below the left eye to record horizontal and vertical eye movements. Data were processed using eeglab 2024.2[79] in MATLAB. Preprocessing involved high-pass filtering (non-causal Butterworth impulse response, half-amplitude cut-off at 0.1 Hz,

415 12 dB/octave roll-off), removal of DC offsets, and attenuation of 50 Hz line noise using Zapline-
416 plus[80]. Data were re-referenced offline to the average of the linked earlobes. Noisy channels were
417 identified through visual inspection and interpolated. Independent component analysis (ICA) was
418 performed using the Infomax algorithm, with PCA rank reduction used when channels had been
419 interpolated. Ocular artifacts were removed by ICA and ICLabel classification[81]. Remaining
420 artifacts were rejected via artifact subspace reconstruction. Stimulation triggers were latency-
421 corrected by 4 ms as determined in a dedicated test run of the experiment using a StimTrak system
422 (BrainProducts, Germany) with an audio adapter and a contact microphone attached to the
423 stimulator's skin contactor. Data were low-pass filtered using a non-causal Butterworth filter (40 Hz
424 half-amplitude cut-off, 12 dB/octave roll-off). EEG data were segmented into epochs from -460 to
425 400 ms relative to the vibration onset at the possible five stimulation time points (50, 150, 250, 350,
426 and 450 ms post movement onset) and baseline-corrected using the 200 ms pre-stimulus interval.
427 For omission trials, epochs were extracted in the same manner around the five time points (50, 150,
428 250, 350, and 450 ms post movement onset), to obtain the EEG activity associated with movement
429 at the corresponding vibration delivery times.

430 These omission epochs were averaged separately for each time point and subsequently
431 subtracted from the respective stimulation epochs, isolating the SEPs (see Fig. 5A). The resulting
432 difference waves were grouped into four movement phases by stimulation timing relative to each
433 trial's maximum speed (pre-max, around max, early post-max, late post-max; see Kinematic
434 Analysis) and averaged within each phase (see Fig. 5B). Mean trials per participant were 64, 96, 41,
435 80, and 78 in the rest condition, pre-max speed, around max speed, early post-max speed, and late
436 post-max speed phases, respectively. On average, 61 omission trials were analyzed per person and
437 time point. The analysis focused on electrode CP3 over S1 contralateral to the stimulated right hand.
438 Components were identified individually from each participant's within-subject average trace across
439 the rest condition and the four movement phases (see Fig. S1). Within predefined ranges, positive

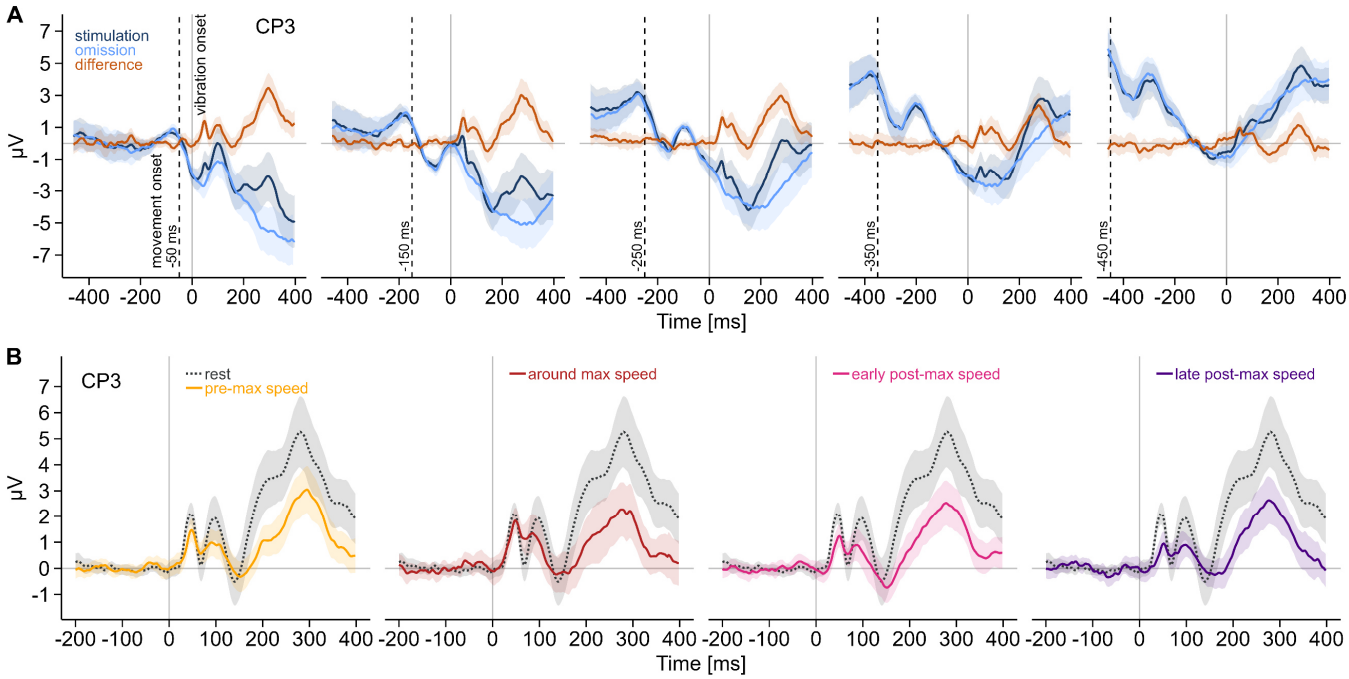

Fig. 5: Grand-averaged EEG activity and difference waves across movement phases.

A. Grand-averaged EEG responses (n = 34) for stimulation and omission trials. Stimulation trials with movement and vibrations delivered at five time points (50, 150, 250, 350, 450 ms) post movement onset. EEG was epoched to vibration onset and baseline corrected to the 200 ms pre-stimulation interval. Omission trials with movement only, epoched at the same five time points (50, 150, 250, 350, 450 ms) post movement onset and baseline corrected to the preceding 200 ms. Difference waves computed as stimulation minus omission to isolate SEPs. The dotted vertical line shows movement onset. Omission traces in all subpanels are the same data re-epoched; stimulation traces differ by stimulation time. Shaded bands show 95% CIs. **B**. Grand-averaged difference-wave SEPs (n = 34) grouped into four phases relative to maximum movement speed: pre-max speed (yellow), around max speed (red), early post-max speed (pink), late post-max speed (purple). Rest condition SEPs, shown for reference (black dotted), are the same in each subpanel. Shaded bands show 95% CIs.

peaks were located for the P45 (35–55 ms) and P300 (250–350 ms). Measurement windows were set at ± 5 ms of peak latency for P45 and ± 10 ms for P300. Mean amplitudes were then extracted per participant, rest condition and movement phase within these windows. Next, we computed differences scores by subtracting the mean component amplitude of each of the four movement phases from the mean component amplitude of the rest condition, resulting in one $P45_{diff}$ and $P300_{diff}$ score per movement phase. This rest-movement subtraction was chosen to maintain consistency with the computation of the psychophysical measure.

447 Statistical Analysis

448 To assess whether movement suppressed vibration perception, we compared the PSE_{diff} from
449 each of the four movement phases with zero using paired, two-sided *t*-tests. Modulation of tactile
450 perception across the movement time course was tested with a univariate repeated-measures
451 ANOVA on the PSE_{diff} from the four movement phases. Post hoc comparisons used paired-samples
452 t-tests between movement phases.

453 To examine modulations in the SEPs associated with movement, we tested $P45_{diff}$ and
454 $P300_{diff}$ from each movement phase against zero using paired, two-sided t-tests. To assess a
455 modulation of component amplitudes across the four movement phases, univariate repeated-
456 measures ANOVAs were performed on $P45_{diff}$ and $P300_{diff}$ separately, followed by paired post-hoc
457 t-tests between movement phases. Planned contrasts that were motivated by significant differences
458 in PSE_{diff} were one-sided; all other comparisons were two-sided. The covariation of perception with
459 changes in SEPs was tested using Pearson correlation. To assess whether SEP amplitudes varied
460 with changes in tactile perception, Pearson correlations were computed between SEP differences
461 and corresponding PSE_{diff} values across phases.

462 The false discovery rate was controlled within each test family using the Benjamini-
463 Hochberg method[82]. Effect sizes are reported as Cohen's d_z and partial η^2 [83]. Data were analyzed in
464 R, v.4.3.3[84] with a significance level of $\alpha = 0.05$. Greenhouse-Geisser correction was applied when
465 sphericity was violated (Mauchly's test). Univariate outliers were defined as $|z| > 3$ based on means
466 and standard deviation within movement phases. Bivariate outliers were defined by Mahalanobis
467 distance (D^2) with a cutoff of $\chi^2(2, 0.999) = 13.82$[85,86]. One participant was classified as both a
468 univariate outlier ($z = -3.63$) within $P45_{diff}$ in the around max speed phase and as a bivariate outlier
469 for $P45_{diff}$ and PSE_{diff} ($D^2 = 23.09$). Data from this participant were excluded from the primary
470 analyses (see Supplement and Fig. S2 for the analysis without this outlier exclusion).

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Acknowledgments

We thank Malaika Grace Alphonsus, Fabian Krause, Athina Nestoropoulou and Rebecca Oliveri for their valuable help in data acquisition.

Funding

This work was supported by the Deutsche Forschungsgemeinschaft (German Research Foundation, DFG) as part of the Collaborative Research Centre SFB/TRR 135, Project A4, Grant 222641018, and under Germany's Excellence Strategy (EXC 3066/1 "The Adaptive Mind", Project No. 533717223).

Author contributions

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Methodology: EF, DV, LKM, KF

Investigation: EF

Visualization: EF

Supervision: DV, LKM, KF

Writing—original draft: EF

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Competing interests

Authors declare that they have no competing interests.