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Target-Speed-Dependent Effects of Water Immersion on Wrist Speed Modulation and Somatosensory Processing

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Abstract

Water immersion alters both movement-related mechanical demands and somatosensory inputs, but whether its effects on motor control depend on target speed and long-term motor experience in water remains unclear. Eighteen competitive swimmers and 15 non-swimmers performed a wrist flexion speed-modulation task under land and water conditions. After adapting to 50% of maximal wrist flexion speed, participants modulated movement speed to 10%, 30%, 50%, 70%, and 90% of maximum. Single-pulse somatosensory evoked potentials and paired-pulse inhibition at interstimulus intervals of 30 and 100 ms (PPI30 and PPI100) were recorded before and after motor adaptation. Movement error was greater in water at the 10% and 30% targets, and the Water–Land difference decreased linearly as target speed increased. N20–P25 amplitude was reduced in water, indicating attenuation of the initial somatosensory-evoked response in the primary somatosensory cortex (S1). After motor adaptation in water, swimmers showed higher PPI30 and lower PPI100 values than non-swimmers, indicating that group differences in paired-pulse processing depended on interstimulus interval. Lower PPI100 values were associated with greater stabilization of the practiced 50% reference speed, whereas no significant associations were found at other target speeds. These findings indicate that water immersion affects wrist speed modulation in a target-speed-dependent manner and attenuates resting S1 responsiveness. In addition, paired-pulse somatosensory processing after motor adaptation in water differed according to long-term competitive swimming experience, whereas its behavioral association was specific to immediate stabilization of the practiced reference speed.

Introduction

Human motor control arises from interactions between the body and the external environment. Motor commands are generated and updated by predicting movement outcomes based on internal models and estimating body state and motor errors from sensory information. Somatosensory information is essential for perceiving body position and movement, controlling ongoing movements, and acquiring and retaining motor skills (Riemann and Lephart 2002; Proske and Gandevia 2012). Motor adaptation has mainly been studied by manipulating force fields or visuomotor transformations within a fixed environment. These studies have examined how predicted and actual sensory feedback are used to correct movement errors (Wolpert et al. 1995; Kawato 1999; Wolpert and Ghahramani 2000; Franklin and Wolpert 2011). However, when the external environment changes, both the mechanical conditions acting on the body and the sensory information available for motor control also change. Therefore, understanding changes in motor output across environments requires examining the accompanying changes in sensory processing.

The water environment simultaneously alters multiple sensory and mechanical conditions. In water, buoyancy reduces the effective load on the body, while hydrostatic pressure and contact with water continuously provide somatosensory input that differs from that on land (Sato et al. 2012; Filingeri et al. 2017). During movement in water, hydrodynamic resistance creates mechanical demands that differ from those on land, while water flow and skin deformation provide additional somatosensory input

(Takagi et al. 2014; Kusanagi et al. 2017). Thus, both sustained and movement-dependent somatosensory inputs differ between water and land environments. Previous studies have examined the effects of the water environment on gait, postural control, and muscle activity (Kaneda et al. 2009; Kaneda 2020). Water immersion has also been shown to alter short-latency somatosensory evoked potentials (SEPs) and afferent inhibition (Sato et al. 2012, 2013), although both studies examined sensory processing at rest. Few studies have examined sensorimotor skill and somatosensory processing together in the same participants in a water environment.

Such a water environment may also affect the ability to accurately match motor output to a target position or speed. Indeed, water immersion increases variability when reproducing a passively presented elbow position (Bock 1994) and increases visuomotor tracking error (Weber et al. 2020). Speed modulation may be particularly affected because hydrodynamic resistance increases with movement speed (Kudo et al. 2013; Koga et al. 2022), and somatosensory input also varies with target speed. Thus, the effect of the water environment on speed modulation accuracy may not be uniform but may differ across speed ranges. To capture this speed-dependent environmental effect, it is necessary to assess not only maximal motor output but also how accurately motor output can be matched to different target speeds.

The primary somatosensory cortex (S1) plays an important role in environment-dependent sensorimotor control. S1 is involved in the early cortical processing of somatosensory input, and this processing changes during movement and motor learning (Cohen and Starr 1987; Ostry et al. 2010; Vahdat et al. 2011). Somatosensory processing in S1 can be assessed noninvasively using scalp-recorded somatosensory evoked potentials (SEPs). The N20 and P25 components of short-latency SEPs elicited by peripheral nerve stimulation reflect early cortical responses in S1, and their peak-to-peak amplitude is widely used as an index of the magnitude of the initial response to input (Allison et al. 1989; Allison et al. 1991; Cruccu et al. 2008). In paired-pulse SEPs, two stimuli are delivered at a defined interval, and the SEP response to the second stimulus is suppressed by the first stimulus. This paired-pulse inhibition (PPI) is used as an index of inhibitory function in S1 (Höffken et al. 2013). Because the magnitude of inhibition varies with the interstimulus interval (ISI), different ISIs can be used to assess S1 inhibitory function with different temporal characteristics. Combining single-pulse SEPs with paired-pulse SEPs at multiple ISIs therefore enables separate assessment of S1 excitability and inhibitory function in the water environment. Previous studies have examined the effects of water immersion to the axillary level on single-pulse SEPs (Sato et al. 2012) and paired-pulse inhibition after whole-hand water-flow stimulation (Le Cong et al. 2022). To our knowledge, no previous study has measured paired-pulse SEPs during water immersion to the axillary level to assess inhibitory processing following input to S1.

Sensorimotor responses to an environment may also depend on long-term motor experience in that environment. Long-term sports experience is associated with specific patterns of perceptual judgment and somatosensory processing (Di Russo et al. 2006; Aglioti et al. 2008; Fourkas et al. 2008; Yarrow et al. 2009; Yamashiro et al. 2013). Competitive swimmers train for many years in an environment characterized by hydrostatic pressure, water flow, and skin deformation. Compared with non-swimmers,

swimmers show greater sensitivity to passive displacement in water (Kusanagi et al. 2017) and distinct characteristics in joint-angle modulation skill and M1 intracortical inhibition in water (Sato et al. 2020). Long-term competitive swimming experience may therefore also be associated with differences in S1 excitability and inhibitory function in the water environment. However, it remains unclear whether early input responses and subsequent inhibitory processing in S1 differ between swimmers and non-swimmers in the water environment.

In this study, speed modulation skill was defined as the ability to match actual movement speed to a target speed. We aimed to determine whether the effect of the water environment on speed modulation skill differed across target speeds and to examine differences in S1 excitability and inhibitory function between land and water environments. Furthermore, by comparing competitive swimmers and non-swimmers, we examined whether behavioral and neurophysiological responses to the water environment differed according to long-term competitive swimming experience. In addition, we examined the relationship between speed modulation skill and S1 excitability and inhibitory function in the water environment.

The hypotheses were as follows. First, we predicted that the effect of the water environment on speed modulation skill would depend on target speed. At low target speeds, hydrodynamic resistance and water flow associated with movement would be small, providing limited sensory and mechanical information for speed modulation. We therefore predicted lower modulation accuracy in water than on land. In contrast, as target speed increased, hydrodynamic resistance, water flow, and the resulting somatosensory input would increase, reducing the negative effect of the water environment. Second, we predicted that N20–P25 amplitude would be lower in water and that S1 inhibitory function assessed using paired-pulse SEPs would vary with environment and motor adaptation, with different response patterns in swimmers and non-swimmers.

Methods

Participants

Thirty-three healthy right-handed adults aged 18–24 years participated in this study, including 18 competitive swimmers and 15 non-swimmers. Detailed profiles of the swimmers are presented in Table 1. All competitive swimmers had at least 10 years of swimming experience, trained at least six times per week, and had competed at the national level. Non-swimmers were university athletes who had received specialized competitive training for more than 10 years but had no history of continuous swimming training during the previous 10 years, except for compulsory swimming classes at school. None of the participants had a history of neurological or psychiatric disorders or used medications that could affect neural activity.

Table 1
Characteristics of elite competitive swimmers

No.	Sex	Age (y.o.)	Event	Age when started swimming (y.o.)	Performance level (WA point)
1	M	22	400 m FR	8	795
2	M	21	100 m FLY	7	786
3	M	21	200 m BR	9	828
4	M	20	100 m FR	10	705
5	M	22	200 m BR	5	732
6	M	22	200 m FR	6	674
7	M	19	100 m FR	5	687
8	M	21	200 m FLY	8	786
9	M	20	400 m FR	3	800
10	M	20	100 m FR	6	762
11	M	19	400 m IM	3	792
12	M	20	100 m BR	8	681
13	M	21	200 m FR	4	786
14	F	20	200 m FLY	10	808
15	F	19	100 m FLY	6	755
16	F	21	200 m FR	11	714
17	F	20	100 m FR	4	824
18	F	22	100 m FLY	5	764

WA points indicate swimming performance level normalized to the world-record value for each event. Abbreviations: F, female; M, male; FLY, butterfly; BR, breaststroke; FR, freestyle; IM, individual medley.

Sample size was evaluated in R version 4.3.1 (R Core Team 2023) using a simulation-based power analysis targeting the Environment × Target interaction in trial-level APE, the primary behavioral outcome, based on pilot data from seven participants. Simulations with a significance level of alpha = .05 and power of 1 - beta = .80 indicated that approximately 30–33 participants were required. A total of 33 participants were included in the final analysis.

The study was approved by the Research Ethics Committee of the Faculty of Health and Sport Sciences, University of Tsukuba (approval no. Tai 025–91) and was conducted in accordance with the Declaration of Helsinki. All participants received an explanation of the study and provided written informed consent before the experiment.

Experimental Design

Two environmental conditions were used: land and water. All participants completed both conditions in a counterbalanced crossover design, with the two sessions separated by 5 days.

In each environmental condition, participants completed the following sequence: measurement of maximal wrist flexion speed, resting SEP/PPI recording before adaptation (T0), a speed adaptation task targeting 50% of maximal flexion speed, resting SEP/PPI recording after adaptation (T1), and a 10–90% speed modulation task (Fig. 1).

Environmental Conditions

All experiments were conducted in an experimental water tank. In the water condition, participants wore swimsuits and sat on a chair immersed to the axillary level (Fig. 2). Water temperature was maintained at 34.5 ± 0.5°C. Measurements began after participants had sufficient time to acclimate following immersion.

The land condition was conducted in the same tank without water. Participants sat on the same chair, and posture and upper-limb position were matched to those in the water condition. In both conditions, participants were instructed to remain as relaxed as possible and avoid unnecessary muscle activity during measurement.

Behavioral Assessment: Wrist Flexion Speed Modulation Task Setting

A wrist flexion task was used to assess speed modulation skill. The forearm was secured on an armrest parallel to the floor, and participants performed wrist flexion from the maximally dorsiflexed position in response to a start cue displayed on a monitor in front of them. Flexion speed was measured using two infrared sensors and calculated by dividing the angular distance between the sensors by the transit-time difference (Figs. 3 and 4).

Participants first performed three maximal-effort flexion movements, and the mean of the three trials was defined as maximal flexion speed (Max). They then performed a speed adaptation task targeting 50% of Max using numerical feedback presented on the monitor. Adaptation was considered complete when movement speed fell within 45–55% of Max for five consecutive trials. Trial-level error was quantified as the absolute percentage error (APE): APE (%) = |actual speed - target speed| / maximal speed × 100. A smaller APE indicated greater accuracy in modulating movement speed to the target. The number of trials from task onset until the adaptation criterion was reached was defined as Trials to Adapt. APE for each trial was plotted against trial number, and the area under the curve (AUC) from task

onset to attainment of the adaptation criterion was calculated using the trapezoidal rule. A smaller AUC indicated less cumulative error during adaptation, and fewer Trials to Adapt indicated attainment of the adaptation criterion in fewer trials. Participants subsequently performed a speed modulation task without feedback at targets of 10%, 30%, 50%, 70%, and 90% of Max. The five conditions were presented in random order, with 10 trials performed at each target speed.

Stimulation paradigm

Electrical stimulation was delivered through a pair of disposable surface electrodes (BlueSensor BRS-50-K/C3, Ambu A/S, Ballerup, Denmark) placed in a bipolar configuration over the median nerve at the right wrist, with the cathode positioned proximally. Stimulation intensity was set to 1.1 times the motor threshold, defined as the intensity that elicited a slight thumb movement. In the water condition, the stimulation electrodes and surrounding area were covered with waterproof film (3M Tegaderm 16006JPS), following procedures used in previous water-immersion electrophysiological studies (Nakazawa et al. 2004; Sato et al. 2012; Le Cong et al. 2022). The same covering was applied in the land condition to ensure comparable electrode conditions.

Three stimulation conditions were used: single-pulse stimulation and paired-pulse stimulation with ISIs of 30 and 100 ms. The three conditions were pseudorandomly interleaved within the same recording session, with 500 trials per condition and 1,500 trials in total. Stimulation was controlled using a pulse control system (Pulse Timer II; Medical Try System, Tokyo, Japan) and synchronized with EEG recording.

EEG Recording

SEPs were recorded at rest before (T0) and after (T1) the 50% speed adaptation task. Continuous EEG was recorded at a sampling rate of 2,500 Hz using a Brain Products actiCHamp 32-channel amplifier and actiCAP active electrodes. Scalp electrodes were positioned at C3, CP1, CP3, CP5, and P3 according to the international 10–20 system, and electrooculography (EOG) was recorded simultaneously. Fz served as the reference electrode, AFz as the ground electrode, and CP3 as the primary analysis electrode. Electrode impedance was maintained below 20 kΩ. Online recording filters were set at 0.1–120 Hz, with a 50-Hz notch filter.

SEP and PPI Analysis

A 3-Hz high-pass filter was applied offline to the EEG data. Data were epoched from − 20 to 200 ms relative to stimulus onset and baseline corrected using the mean signal from − 20 to 0 ms. Epochs exceeding ± 70 μV were rejected, and the remaining epochs were averaged separately for each stimulation condition.

For single-pulse SEPs, CP3 was selected as the primary analysis electrode because it reflects activity near the S1 contralateral to right median nerve stimulation. N20 at approximately 20 ms and P25 at approximately 25 ms after stimulation were identified in participant- and condition-specific averaged waveforms, and the N20–P25 peak-to-peak amplitude was calculated. When peak identification was

difficult, peaks were identified within a predefined search window based on the grand-average waveform. N20–P25 amplitude was used as an index of the initial somatosensory input response in S1. As supplementary analyses, signed amplitudes from the prestimulus baseline to the N20 and P25 peaks were calculated as the N20 and P25 amplitudes, respectively (Online Resource 1).

For paired-pulse stimulation, responses to the first and second stimuli overlapped temporally. Therefore, the single-pulse SEP waveform was subtracted from the paired-pulse waveform to obtain a corrected waveform representing the response to the second stimulus. For each corrected waveform at ISIs of 30 and 100 ms, the N20–P25 peak-to-peak amplitude was calculated relative to the second stimulus. PPI30 and PPI100 were calculated using the following equation:

PPI (%) = N20–P25 amplitude obtained from subtracted waveform / N20–P25 amplitude elicited by the single stimulus × 100.

Thus, a lower PPI value indicates a relatively stronger paired-pulse inhibition, whereas a higher PPI value indicates weaker inhibition.

Outcome Measurements and Statistical Analysis

Behavioral outcomes were trial-level APE as the primary outcome and AUC and Trials to Adapt from the speed adaptation task as secondary outcomes. Neurophysiological outcomes were N20–P25 amplitude, N20 amplitude, P25 amplitude, PPI30, and PPI100 at T0 and T1. Relationships between APE and neurophysiological measures were also examined.

All statistical analyses were performed in R version 4.3.1 (R Core Team 2023), with the significance level set at 5%. A linear mixed-effects model (LMM) was applied to trial-level APE, the primary behavioral outcome. Environment, Group, Target, and all interactions were included as fixed effects. To account for repeated measurements within participants and individual differences in the environmental effect, participant-specific random intercepts and random slopes for Environment were included. Model fit was compared between a random-intercept-only model and a model additionally including random slopes for Environment. The model including random slopes for Environment showed better fit than the random-intercept-only model (AIC = 23202.35 vs. 23265.99; BIC = 23347.04 vs. 23398.63; likelihood-ratio test: $\chi^2(2) = 67.64$, $p < .001$) and was therefore used for the primary analysis. Satterthwaite-approximated degrees of freedom were used for omnibus tests of fixed effects and comparisons of estimated marginal means. The nlme package version 3.1–162 was used for the LMM (Pinheiro et al. 2023), and the emmeans package version 1.11.1 was used to calculate estimated marginal means (Lenth 2025).

Post hoc comparisons following significant interactions were performed using estimated marginal means derived from the same model, with p values adjusted using the Holm method. Target-specific environmental differences were examined at each of the five target speeds following the Environment × Target interaction. The robustness of these comparisons was further evaluated using a Bonferroni-corrected sensitivity analysis. In addition, the 10%, 30%, 70%, and 90% targets were compared with the

50% target separately within the land and water conditions, with Holm adjustment applied to the four comparisons within each environment (Online Resource 1). Polynomial trend analysis was performed on the Water–Land differences at each target speed estimated from the same LMM. Linear through quartic components were tested, and their four p values were adjusted using the Holm method. Group-specific linear trends were also examined as supplementary analyses, with Holm adjustment applied across the two groups.

AUC and Trials to Adapt from the speed adaptation task were analyzed using two-way mixed analyses of variance, with Group as a between-participant factor and Environment as a within-participant factor.

Each neurophysiological measure (N20–P25 amplitude, N20 amplitude, P25 amplitude, PPI30, and PPI100) was analyzed using a separate three-way mixed-design analysis of variance, with Group as a between-participant factor and Environment and Phase as within-participant factors. Post hoc comparisons following significant interactions were based on estimated marginal means from linear mixed-effects models with the same factorial structure, and p values were adjusted using the Holm method. Associations between behavioral and neurophysiological measures after motor adaptation in the land and water conditions were examined using Pearson product-moment correlations. The Benjamini-Hochberg false discovery rate (FDR) procedure was applied, and Bonferroni adjustment was additionally used as a sensitivity analysis.

Use of generative artificial intelligence

OpenAI GPT-5 was used to assist with identifying potentially relevant literature, manuscript preparation, and Japanese-to-English translation. The authors verified all outputs and references and take full responsibility for the manuscript.

Results

Behavioral data

To examine speed modulation skill in swimmers and non-swimmers under land and water conditions, a trial-level linear mixed-effects model was applied to APE, with Group, Environment, Target, and all interactions as fixed effects and participant-specific random intercepts and random slopes for Environment. Satterthwaite-approximated degrees of freedom were used to test fixed effects. Significant Environment × Target ($F(4, 3010.08) = 9.461$, $p < .001$) and Group × Target interactions ($F(4, 3010.00) = 17.572$, $p < .001$) were observed. The main effect of Target was also significant ($F(4, 3010.00) = 220.329$, $p < .001$). In contrast, the main effects of Environment ($F(1, 30.14) = 0.597$, $p = .446$) and Group ($F(1, 30.56) = 0.146$, $p = .705$), the Environment × Group interaction ($F(1, 30.14) = 0.189$, $p = .666$), and the Environment × Group × Target interaction ($F(4, 3010.08) = 0.342$, $p = .850$) were not significant (Fig. 5).

Following the Environment × Target interaction, environmental differences at each target speed were tested using estimated marginal means from the same model, with Holm adjustment. At the 10% target,

APE was 3.54 percentage points higher in water than on land (Land: 16.17%; Water: 19.71%; 95% CI [1.34, 5.75]; t(98.12) = 3.189; p_{Holm} = .010). At the 30% target, APE was 3.10 percentage points higher in water (Land: 9.85%; Water: 12.95%; 95% CI [0.91, 5.29]; t(95.21) = 2.813; p_{Holm} = .024). No significant environmental differences were observed at the 50%, 70%, or 90% targets (p_{Holm} ≥ .183). In the Bonferroni sensitivity analysis, environmental differences remained significant at the 10% ($p_{Bonferroni}$ = .010) and 30% targets (pBonferroni = .030), whereas the difference at the 90% target was not significant ($p_{Bonferroni}$ = .305).

Holm-adjusted comparisons following the Group × Target interaction showed that swimmers had an APE 4.70 percentage points higher than non-swimmers at the 10% target (95% CI [1.95, 7.46]; p_{Holm} = .006) and 3.97 percentage points lower at the 70% target (95% CI [-6.72, -1.22]; p_{Holm} = .021). No significant group differences were observed at the other target speeds. Because the Environment × Group × Target interaction was not significant, these group differences could not be considered specific to either environment.

Polynomial trend analysis of the Water–Land APE difference revealed a significant linear trend (t(3009.15) = -5.898; p_{Holm} < .001). The Water–Land difference decreased by approximately 1.54 percentage points for each 20% increase in target speed. Quadratic and higher-order trends were not significant (p_{Holm} ≥ .571). Group-specific linear trends were significant in both non-swimmers (t(3012.05) = -3.844; pHolm < .001) and swimmers (t(3010.65) = -4.525; pHolm < .001). However, the Environment × Group × Target interaction was not significant (p = .850), providing no evidence that the linear trend differed between groups (Fig. 6).

AUC from the speed adaptation task was analyzed using a Group × Environment mixed-design analysis of variance. No significant main effect of Environment (F(1, 31) = 0.013, p = .909), main effect of Group (F(1, 31) = 0.386, p = .539), or interaction (F(1, 31) = 0.633, p = .432) was found. Similarly, for Trials to Adapt, neither the main effect of Environment (F(1, 31) < 0.001, p = .995), the main effect of Group (F(1, 31) = 0.428, p = .518), nor the interaction (F(1, 31) = 0.556, p = .461) was significant.

Neurophysiological data

N20–P25 amplitude, PPI30, and PPI100 were analyzed using separate three-way mixed-design analyses of variance, with Group as a between-participant factor and Environment and Phase as within-participant factors (Figs. 7 and 8). N20–P25 amplitude showed a significant main effect of Environment (F(1, 31) = 18.193, p < .001) and was lower in water than on land. The main effects of Group and Phase and all interactions for N20–P25 amplitude were not significant (all p ≥ .163). Results of the supplementary analyses of N20 and P25 amplitudes are reported in Online Resource 1.

PPI30 showed significant Group × Environment (F(1, 31) = 7.002, p = .013), Environment × Phase (F(1, 31) = 7.817, p = .009), and Group × Environment × Phase interactions (F(1, 31) = 5.196, p = .030). In Holm-

adjusted between-group comparisons, PPI30 was 20.26 percentage points higher in swimmers than in non-swimmers after motor adaptation in water (95% CI [6.50, 34.03]; p_{Holm} = .021). No other between-group comparisons or group-specific Environment or Phase comparisons remained significant after adjustment.

PPI100 showed a significant main effect of Environment ($F(1, 31) = 5.271$, $p = .029$) and a significant Group × Environment × Phase interaction ($F(1, 31) = 5.602$, $p = .024$). The Group × Environment interaction did not reach statistical significance ($F(1, 31) = 3.227$, $p = .082$), and no other main effects or interactions were significant (all $p \geq .165$). After Holm adjustment, PPI100 was 27.92 percentage points lower in water than on land after motor adaptation in swimmers (95% CI [-46.94, -8.91]; p_{Holm} = .018). After motor adaptation in water, PPI100 was also 30.68 percentage points lower in swimmers than in non-swimmers (95% CI [-52.08, -9.29]; p_{Holm} = .026). No other comparisons were significant.

Relationship between behavioral and neurophysiological data

A significant positive correlation was found between PPI100 and 50% APE after motor adaptation in water for all participants ($r = .627$, p_{FDR} = .010, $n = 33$). A similarly strong positive correlation was observed in swimmers ($r = .764$, p_{FDR} = .012, $n = 18$). No other correlations remained significant after FDR adjustment. In the Bonferroni sensitivity analysis, these two correlations also remained significant ($p_{Bonferroni}$ = .010 and .024, respectively) (Fig. 9).

Discussion

Main findings

This study examined whether the effect of the water environment on speed modulation skill differed across target speeds, how the water environment influenced S1 neural activity, and whether these responses were associated with long-term motor experience in water. The main findings were as follows. First, the effect of the water environment on speed modulation skill was target-speed dependent, with greater impairment at lower target speeds and little evidence of impairment at higher speeds. Second, water immersion altered S1 excitability and paired-pulse processing, with some inhibitory responses differing between swimmers and non-swimmers. Third, the association between S1 inhibitory processing and speed modulation performance was limited to a specific condition rather than being consistently observed across measures. Together, these findings indicate that the effects of the water environment on sensorimotor control are not uniform but depend on movement demands and the specific neural processes examined.

Speed modulation skill

The speed-dependent pattern of impairment suggests that immersion affects speed modulation selectively, rather than uniformly. Specifically, the detrimental effect of the water environment was evident primarily at lower target speeds and became progressively smaller as target speed increased, with a similar trend observed in both swimmers and non-swimmers.

In water, hydrodynamic resistance increases with movement speed (Kudo et al. 2013; Koga et al. 2022), accompanied by changes in somatosensory input arising from water flow and skin deformation. Consequently, the sensory and mechanical cues available for estimating movement speed in water are likely to differ across target speeds. At lower target speeds, movement-related hydrodynamic resistance and water flow are relatively small, potentially providing weaker speed-related sensory cues. In contrast, at higher target speeds, greater hydrodynamic resistance may provide more salient cues about movement speed, thereby attenuating the negative effect of the water environment on speed modulation skill. This interpretation is consistent with the observed linear decrease in the Water–Land APE difference as target speed increased.

Comparisons between swimmers and non-swimmers revealed target-speed-dependent group differences in speed modulation skill, but these differences were not specific to the water environment. Neither the Environment × Group nor the Environment × Group × Target interaction was significant; thus, our hypothesis that swimmers would show greater speed modulation skill in water than non-swimmers was not supported. Previous work has shown that swimmers outperform non-swimmers in hand-angle modulation in water (Sato et al. 2020). This discrepancy may reflect differences in the motor demands of the tasks used to assess specific motor skill. Competitive swimming requires swimmers to capture water and generate propulsion through precise control of hand and forearm position and orientation, together with multijoint coordination (Toussaint and Beek 1992; Cappaert et al. 1995; Payton et al. 1999; Kudo et al. 2019, 2025). In particular, stabilizing the wrist and appropriately controlling hand orientation and angle of attack according to movement direction contribute to propulsion during actual swimming (Caty et al. 2007; Koga et al. 2022). By contrast, the present task required participants to match the speed of an isolated wrist flexion movement to a target value and therefore may not have adequately captured the movement features specifically refined through competitive swimming. Therefore, the absence of a water-specific behavioral group difference in this study should not be interpreted as evidence that competitive swimming experience is unrelated to motor control in water; rather, its behavioral effects may depend on whether the task engages movement features specifically developed through swimming training.

Excitatory and inhibitory function in the S1

Single-pulse SEP N20–P25 amplitude was reduced in the water environment. N20–P25 amplitude reflects the magnitude of the initial somatosensory-evoked response in S1 and is used as an index of S1 excitability (Allison et al. 1989; Allison et al. 1991; Cruccu et al. 2008). Accordingly, the present finding suggests that water immersion attenuated the initial cortical response in S1 to median nerve stimulation. This result is consistent with previous work showing that water immersion reduces short-

latency SEPs, particularly P25 amplitude (Sato et al. 2012). In water, hydrostatic pressure and contact with water provide sustained somatosensory input to the skin. Short-latency SEPs are also attenuated when additional tactile stimulation, distinct from the median nerve stimulation, is applied (Jones and Power 1984; Kakigi and Jones 1985; Kawashima et al. 1991; Jones et al. 1992). Thus, sustained somatosensory input in water may alter the background sensory state of S1, thereby attenuating the initial cortical response evoked by median nerve stimulation.

PPI30 showed a Group × Environment × Phase interaction, with higher values in swimmers than in non-swimmers after motor adaptation in water, indicating weaker paired-pulse suppression in swimmers under this condition. Paired-pulse suppression can be modulated by sensory stimulation, motor learning, and long-term skill experience (Höffken et al. 2007; Saito et al. 2018; Hirano et al. 2025). The group difference observed after motor adaptation may therefore reflect experience-dependent differences in short-interval somatosensory processing associated with long-term motor experience in water. However, neither group showed a significant change from before to after adaptation in PPI30. Accordingly, the interaction should not be interpreted as evidence that motor adaptation itself reduced suppression in swimmers or increased it in non-swimmers. Rather, the finding indicates that the two groups differed specifically in the post-adaptation state in water.

The group difference in PPI100 was in the opposite direction to that observed for PPI30. Swimmers had lower PPI100 than non-swimmers after motor adaptation in water, indicating stronger suppression of the second response. Paired-pulse suppression depends on the ISI, and responses at a 100-ms ISI are likely to involve neural processes that differ from those operating at shorter ISIs, including differences in inhibition and recovery of the evoked response (Lüders et al. 1984; Höffken et al. 2010; Liu et al. 2021). PPI100 can also change after motor learning (Pham et al. 2022). Taken together, the opposite group differences at PPI30 and PPI100 suggest that long-term motor experience in water may influence paired-pulse somatosensory processing in an ISI-dependent manner rather than producing a uniform increase or decrease in suppression. However, paired-pulse SEP measures do not permit identification of the specific neurotransmitters or inhibitory circuits underlying these effects.

Relationship between behavioral and neurophysiological indices

Associations between behavioral and neurophysiological measures were limited. After correction for multiple comparisons, significant associations were confined to PPI100 and 50% APE after motor adaptation in water. Because the 50% target was identical to the speed repeatedly practiced during the adaptation task, this finding may indicate an association between S1 inhibitory function assessed at an ISI of 100 ms and stabilization of the practiced speed condition. Somatosensory processing and S1 neural activity have been shown to change after motor learning (Ostry et al. 2010; Vahdat et al. 2011; Pham et al. 2022), and the present findings similarly suggest that post-adaptation S1 inhibitory function may be related to subsequent stabilization of motor skill. However, because the association was limited

to the 50% target and was not observed at other target speeds, it cannot be generalized as the neural basis underlying speed modulation skill in water.

Limitations

This study has several limitations. First, because the study used a cross-sectional group comparison, it cannot determine whether the neurophysiological differences between swimmers and non-swimmers resulted from long-term motor experience in water or reflected pre-existing individual differences. Second, single-pulse and paired-pulse SEPs are indirect indices of S1 excitability and inhibitory function and do not directly identify specific neurotransmitters or neural circuits. Third, SEPs and PPI were measured at rest and therefore did not directly assess S1 neural activity during performance of the speed modulation task. Fourth, hydrodynamic resistance, water flow, and cutaneous sensory input during movement were not quantitatively controlled; therefore, changes in speed modulation skill cannot be attributed to a specific sensory or mechanical component of the water environment. Finally, because speed modulation skill was evaluated using APE, a relative error normalized to maximal speed, the possibility that individual differences in maximal speed influenced this error metric cannot be excluded. Future studies should independently manipulate the sensory and mechanical components of the water environment, assess neural activity during movement, and use longitudinal intervention design to clarify the relationship between sensorimotor control and S1 function in water.

Conclusion

The effect of the water environment on speed modulation skill depended on target speed, with lower modulation accuracy in water at lower target speeds. S1 excitability was reduced in water, and paired-pulse processing after motor adaptation in water differed between swimmers and non-swimmers. These findings suggest that the effects of the water environment on sensorimotor control and S1 function are not uniform but depend on movement demands and, for paired-pulse somatosensory processing, on the interstimulus interval and long-term motor experience in water.

Declarations

Funding

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Competing interests

The authors declare no competing interests.

Ethics approval

This study was approved by the Research Ethics Committee of the Faculty of Health and Sport Sciences, University of Tsukuba (approval no. Tai 025-91) and was conducted in accordance with the Declaration of Helsinki and its later amendments.

Informed consent

Written informed consent was obtained from all individual participants included in the study.

Data availability

The de-identified datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request, subject to the conditions of the ethics approval and participant consent.

Author contributions

A.I. conducted the experiments, analyzed and interpreted the data, and drafted the original manuscript. Y.S. contributed to data interpretation and critically reviewed and revised the manuscript. D.S. conceived and supervised the study, contributed to the interpretation of the findings, and critically reviewed and revised the manuscript. All authors read and approved the final manuscript.

Supplementary Information

Online Resource 1 Supplementary analyses of N20 and P25 amplitudes and target-speed comparisons with the practiced 50% reference speed

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Figures

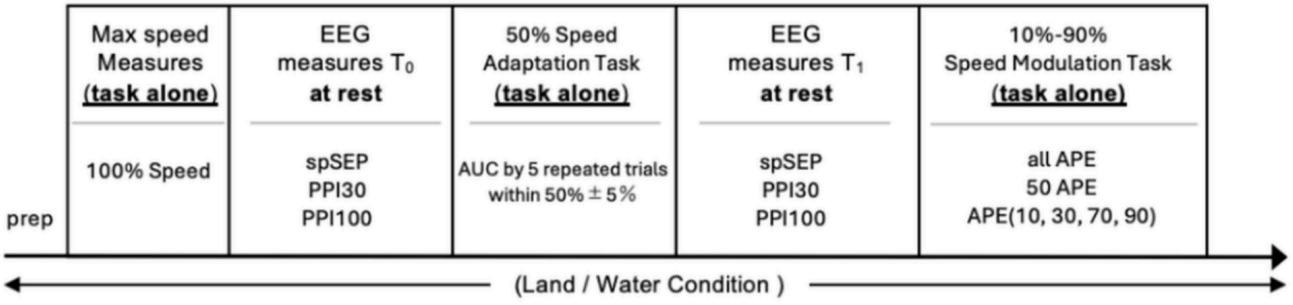


Figure 1

Experimental protocol

After a preparation period (prep), participants first performed the maximal speed task (100% speed). Resting EEG measurements were then obtained at T0, including single-pulse somatosensory evoked potentials (spSEP), paired-pulse inhibition at an interstimulus interval of 30 ms (PPI30), and paired-pulse inhibition at an interstimulus interval of 100 ms (PPI100). Next, participants performed the 50% speed adaptation task alone, and task performance was evaluated using the area under the curve (AUC). Resting EEG measurements were then repeated at T1, including spSEP, PPI30, and PPI100. Finally, participants performed the 10%–90% speed modulation task alone. Overall absolute percentage error (all APE), APE at 50% speed (50% APE), and APE for each speed condition (10%, 30%, 70%, and 90%) were calculated

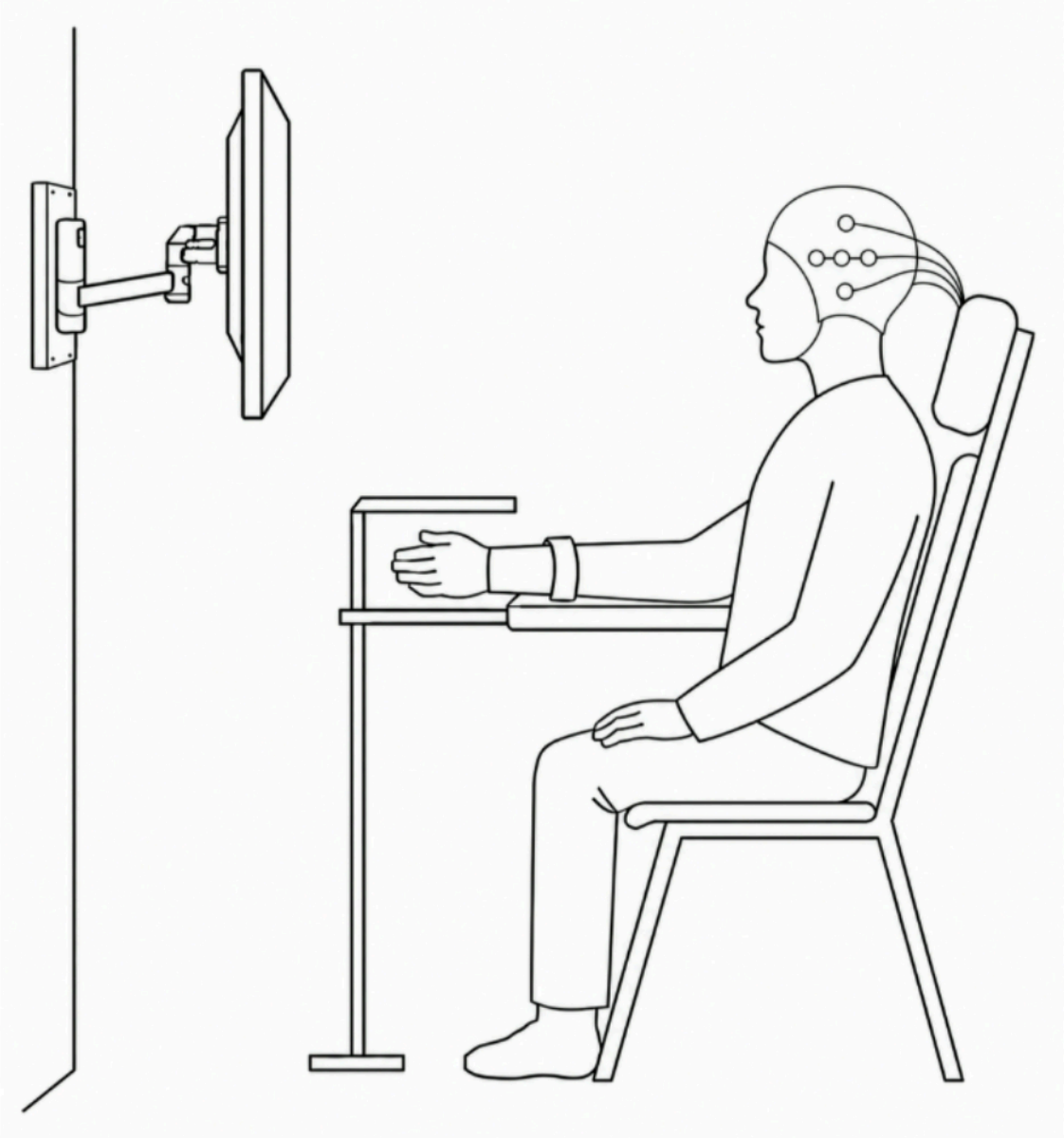


Figure 2

Experimental setting

Participants were seated in a chair with a backrest while wearing an EEG cap, with the right arm positioned on a support-mounted task apparatus. In the water condition, participants were immersed to the axillary level. During the tasks, participants used visual information presented on a monitor in front of them. The figure schematically illustrates the participant’s posture and the arrangement of the apparatus

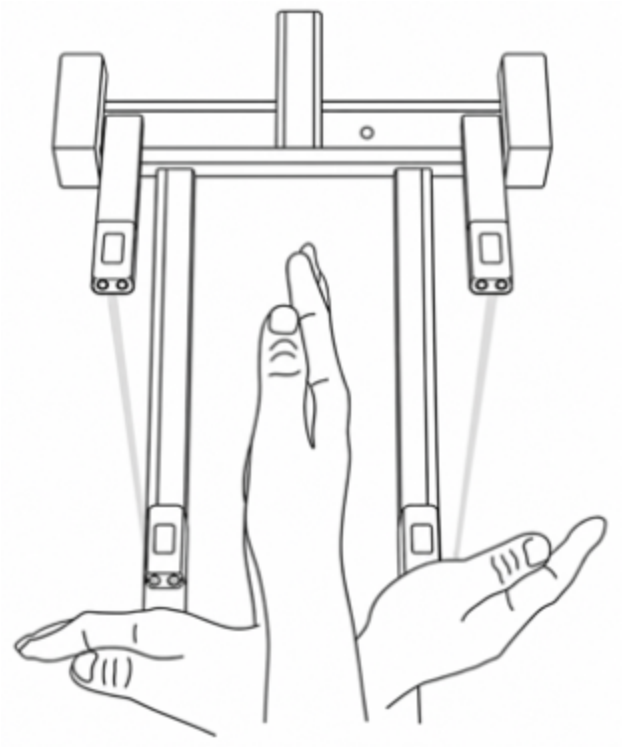


Figure 3

Wrist movement speed modulation task. The participant’s forearm was secured on a support platform, and movement was restricted to the wrist joint. Wrist movement speed was calculated from the time required for the hand to pass between the two sensors. The illustrated hand positions indicate the range of motion from the starting position to the end position

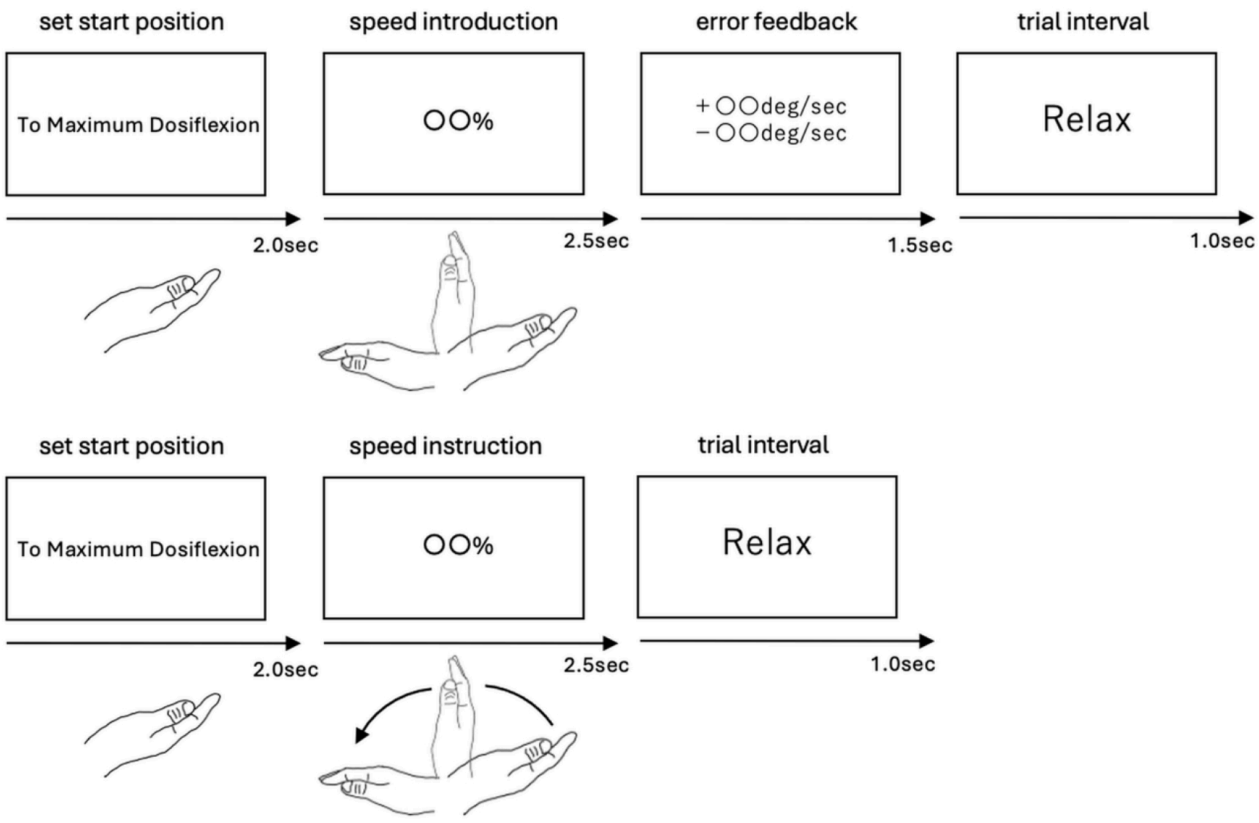


Figure 4

Flow of the speed adaptation and modulation tasks

The upper sequence shows the 50% speed adaptation task, and the lower sequence shows the 10%–90% speed modulation task. In both tasks, the target speed was presented after the starting position had been set, and participants performed wrist flexion from the maximally dorsiflexed position. During the 50% speed adaptation task, error relative to the target speed was presented after each movement. No error feedback was provided during the speed modulation task

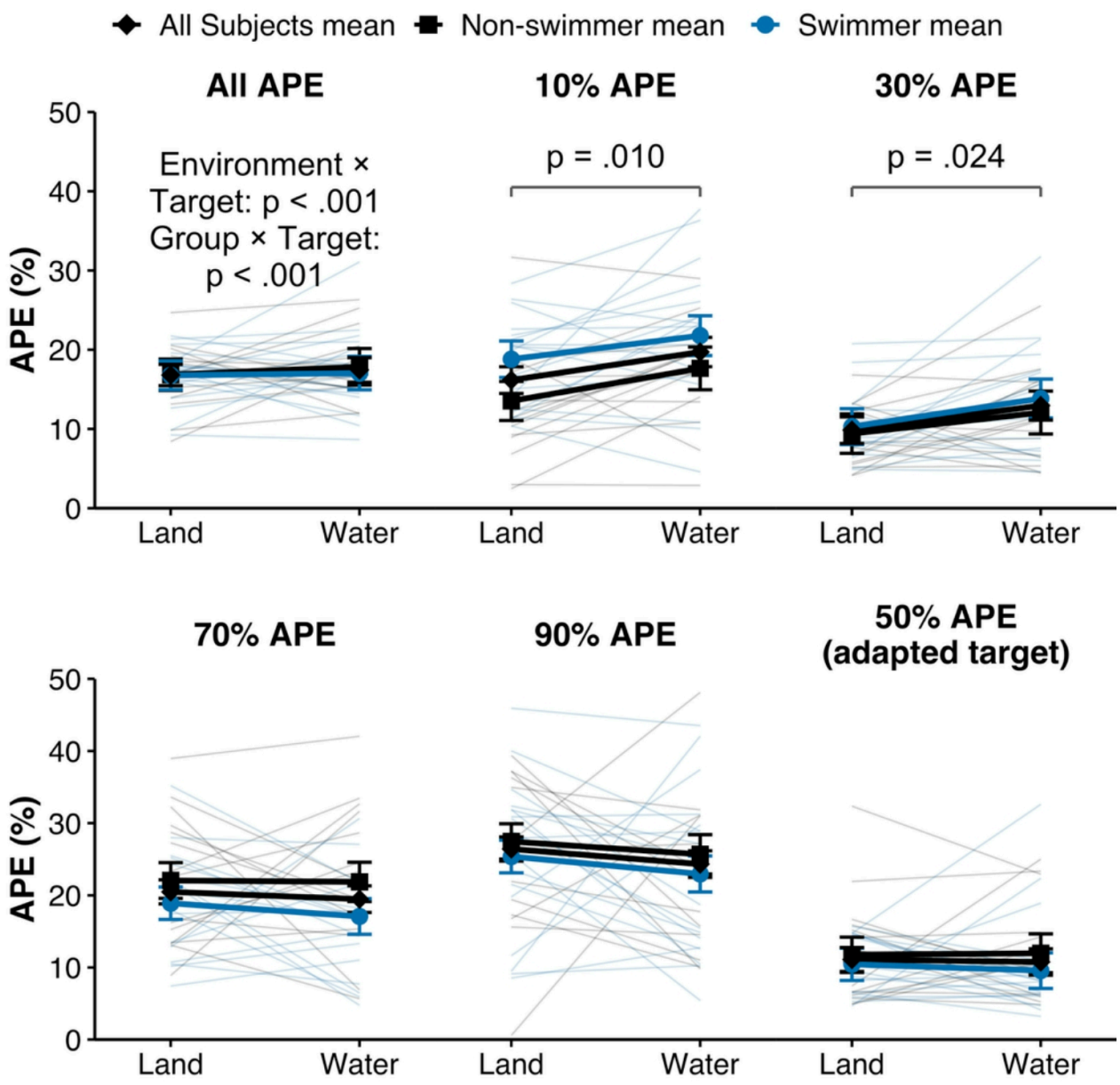


Figure 5

Speed modulation skill under land and water conditions

Each panel shows absolute percentage error (APE) averaged across all target speeds or at each target speed. Thin lines represent individual Land–Water changes. Thick lines and symbols represent estimated marginal means derived from the linear mixed model (LMM), and error bars indicate 95% confidence intervals. The interaction p values in the All APE panel are from the omnibus LMM tests. Brackets and p values indicate significant target-specific Land–Water comparisons after Holm correction

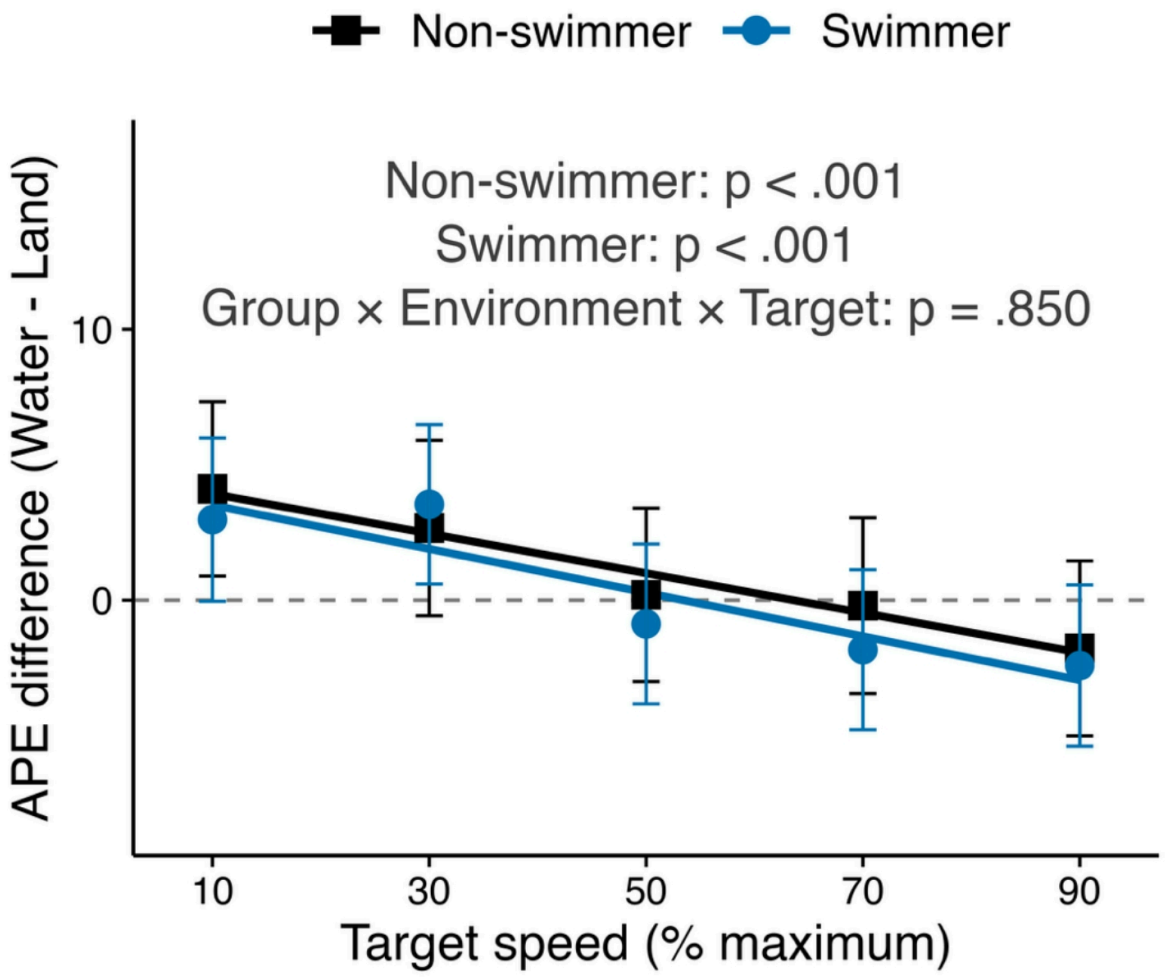


Figure 6

Target-speed-dependent environmental differences in APE

Each point represents the estimated Water–Land difference in APE for swimmers and non-swimmers at each target speed. Positive values indicate greater APE in water, whereas negative values indicate lower APE in water. Error bars indicate 95% confidence intervals, and solid lines represent linear trends across target speeds. Black squares represent non-swimmers, and blue circles represent swimmers. P values for the group-specific linear trends were adjusted using the Holm method. The p value for the Group × Environment × Target interaction is from the omnibus LMM test and was not adjusted for multiple comparisons

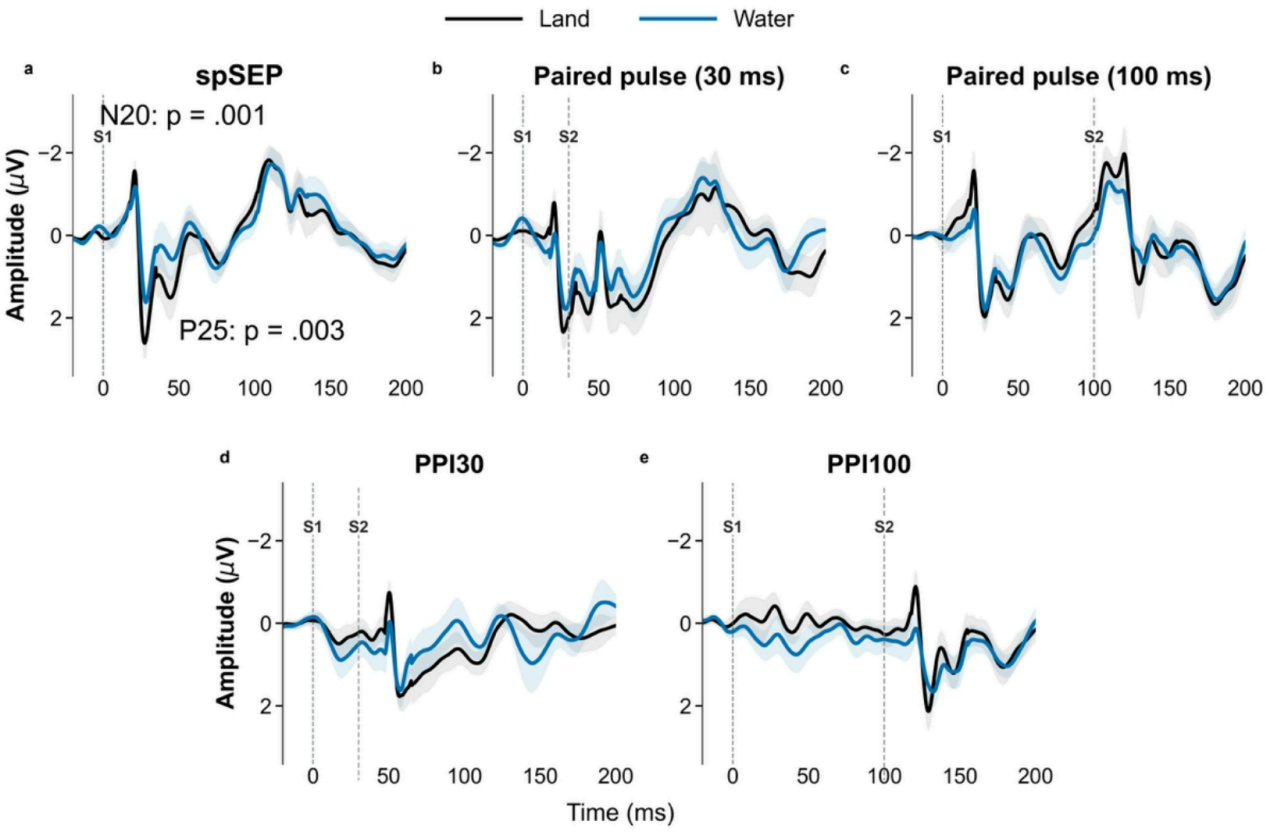


Figure 7

Grand-average waveforms recorded at CP3 after motor adaptation

The upper panels show the single-pulse SEP and paired-pulse waveforms at interstimulus intervals of 30 and 100 ms. The lower panels show the corresponding waveforms after subtraction of the single-pulse SEP from each paired-pulse waveform. S1 and S2 and the vertical dashed lines indicate the timing of the first and second stimuli, respectively. Black lines represent the land condition, blue lines represent the water condition, and shaded areas indicate 95% confidence intervals. Time is referenced to the first stimulus, and negative amplitudes are plotted upward. The p values indicate the main effects of Environment on the N20 and P25 amplitudes

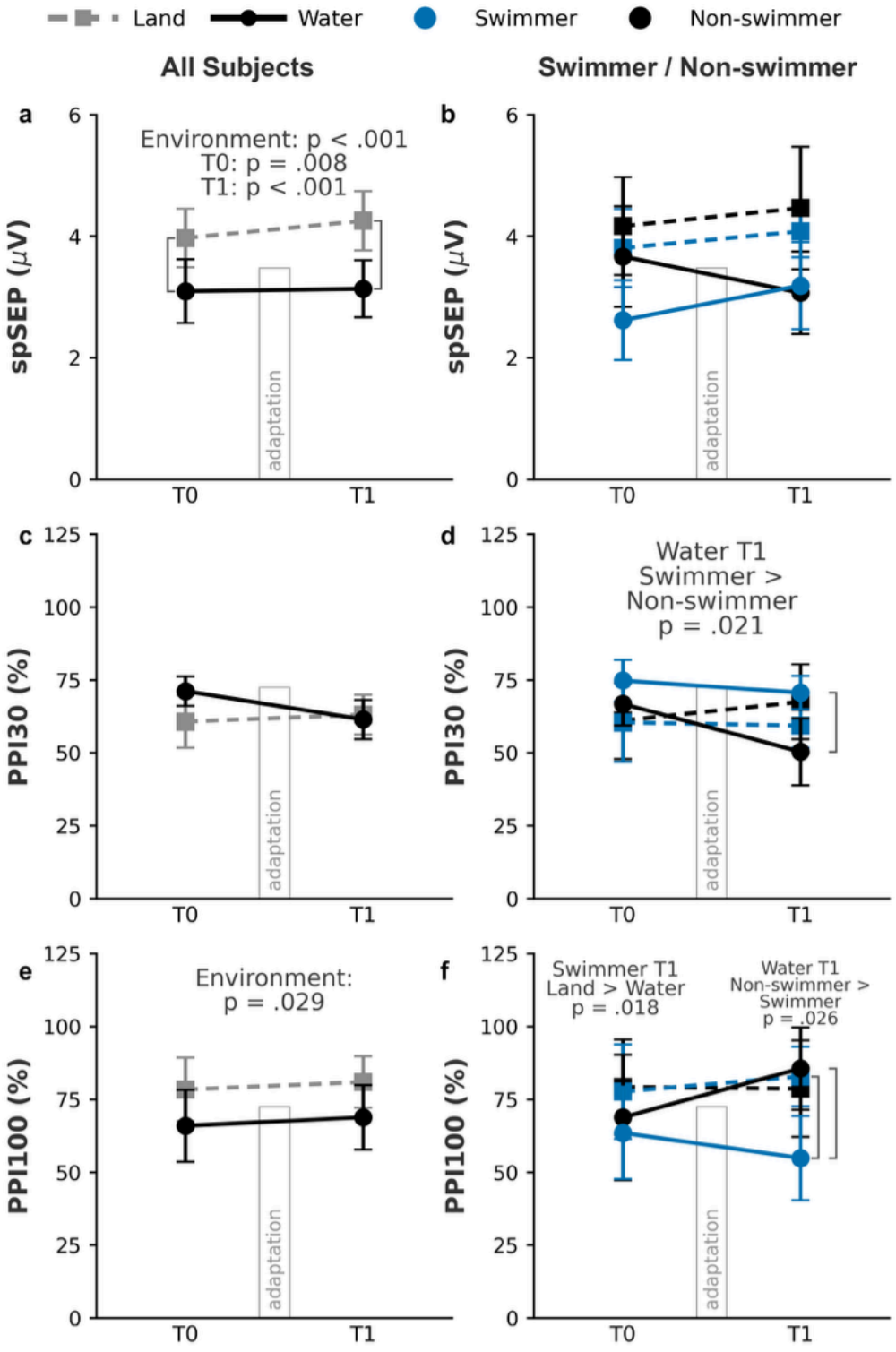


Figure 8

Somatosensory evoked potentials and paired-pulse inhibition before and after speed adaptation

The upper, middle, and lower panels show single-pulse N20–P25 amplitude, PPI30, and PPI100, respectively. The left column shows all participants, and the right column shows swimmers and non-swimmers separately. Dashed lines with squares represent the land condition, and solid lines with circles

represent the water condition. Symbols indicate estimated means, and error bars indicate 95% confidence intervals. P values for main effects and interactions are from the three-way mixed-design analyses of variance. Brackets and p values indicate condition comparisons after Holm correction

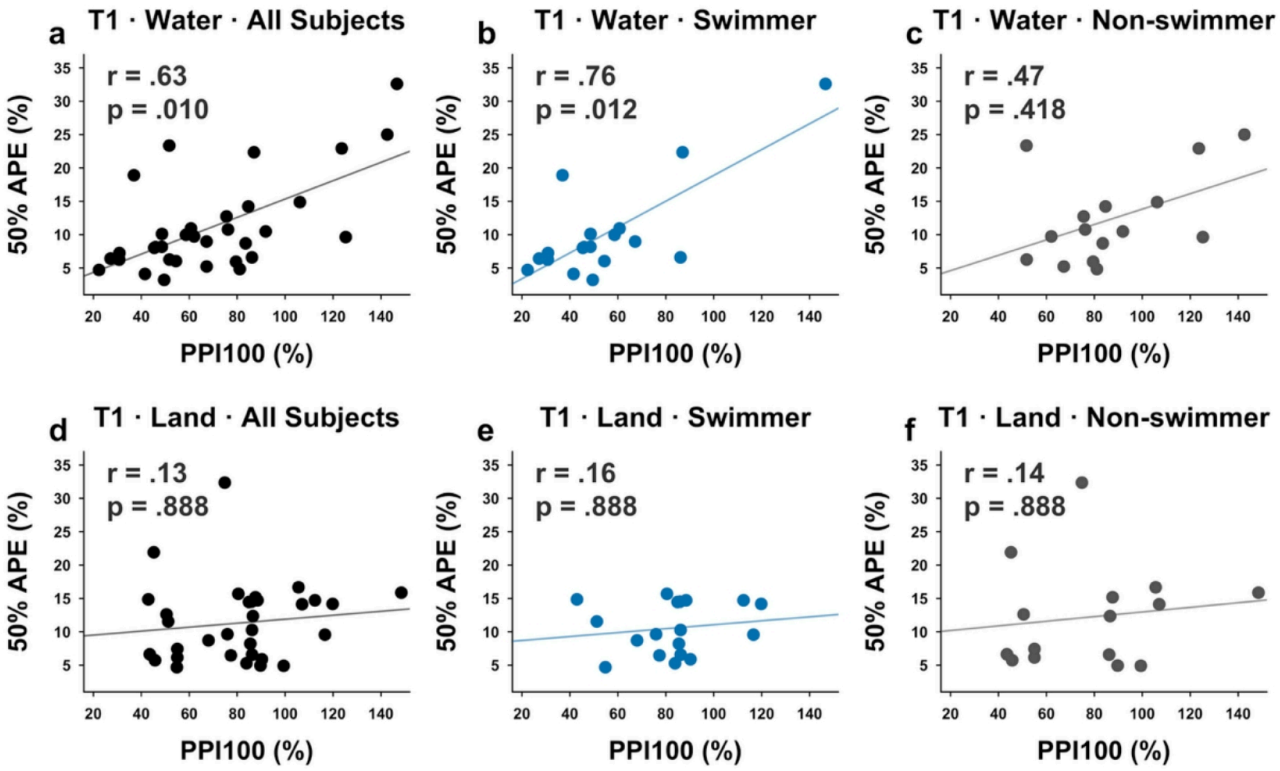


Figure 9

Relationships between PPI100 and 50% APE after speed adaptation

The upper and lower rows show the water and land conditions at T1, respectively. From left to right, the panels show all participants, swimmers, and non-swimmers. Each point represents one participant, and solid lines indicate least-squares regression lines. Pearson's correlation coefficients and FDR-adjusted p values are shown in each panel

Supplementary Files

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