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#### ORIGINAL ARTICLE



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# Mind over muscle? Time manipulation improves physical performance by slowing down the neuromuscular fatigue accumulation

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#### **Abstract**

While physical performance has long been thought to be limited only by physiological factors, many experiments denote that psychological ones can also influence it. Specifically, the deception paradigm investigates the effect of psychological factors on performance by manipulating a psychological variable unbeknownst to the subjects. For example, during a physical exercise performed to failure, previous results revealed an improvement in performance (i.e., holding time) when the clock shown to the subjects was deceptively slowed down. However, the underlying neurophysiological changes supporting this performance improvement due to deceptive time manipulation remain unknown. Here, we addressed this issue by investigating from a neuromuscular perspective the effect of a deceptive clock manipulation on a single-joint isometric task conducted to failure in 24 healthy participants (11 females). Neuromuscular fatigue was assessed by pre-to post-exercise changes in quadriceps maximal voluntary torque (T<sub>max</sub>), voluntary activation level (VAL), and potentiated twitch (T<sub>TW</sub>). Our main results indicated a significant performance improvement when the clock was slowed down (Biased:  $356 \pm 118$  s vs. Normal:  $332 \pm 112$  s, p = .036) but, surprisingly, without any difference in the associated neuromuscular fatigue (p > .05 and BF < 0.3 for  $T_{max}$ , VAL, and T<sub>TW</sub> between both sessions). Computational modeling showed that, when observed, the holding time improvement was explained by a neuromuscular fatigue accumulation based on subjective rather than actual time. These results support a psychological influence on neuromuscular processes and contribute significantly to the literature on the mind-body influence, by challenging our understanding of fatigue.

#### KEYWORDS

EMG, false-clock paradigm, nerve stimulation, perceived time, time deception, time to failure

Robin Baurès and Julien Duclay contributed equally to this work.

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#### 1 | INTRODUCTION

Why do we reach task failure during physical exercise? Limits of physical performance have long been considered from a physiological perspective (Hill et al., 1924), investigating, in turn, cardiovascular, respiratory, metabolic, and neuromuscular mechanisms (McKenna & Hargreaves, 2008). However, many experiments suggest that physical performance can be influenced by psychological factors, such as motivation or placebos (Noakes, 2011). For instance, in the presence of a high monetary reward (Cabanac, 1986) or after ingesting a pill thought as beneficial (Beedie & Foad, 2009), you are more likely to beat your personal best. A suitable paradigm to study the influence of psychological factors on physical performance is manipulating them without participants' knowledge (Jones et al., 2013; Williams et al., 2014). Remarkably, a few studies showed a consistent improvement in cycling performance (i.e., a decrease in the time required to cover a given distance) when participants are competing against an avatar who is moving, unbeknownst to them, 2% faster than a previously established personal best (Ansdell et al., 2018; Ducrocq et al., 2017; Stone et al., 2012). Interestingly, other deceptive types of feedback can be used, such as intensity (e.g., set resistance), distance, and time (Williams et al., 2014). In particular, time is a fundamental dimension of life (Buhusi & Meck, 2005) as it is universally and continuously experienced (Matthews & Meck, 2016), constituting a relevant variable to manipulate unbeknownst to the participants to improve their performance (Thönes et al., 2018). For example, one study (Morton, 2009) showed a performance improvement during a pedaling task performed to failure (i.e., an increase in the sustain time of the effort at a given load) when the clock was deceptively slowed down by 10% compared to a condition where the clock was running normally.

These studies raise an interesting yet unresolved question: How can psychological factors improve physical performance? The underlying neurophysiological changes that support performance improvement induced by psychological factors remain unclear. During acute exercise, the neurophysiological changes involved in the physical activity may be translated into neuromuscular fatigue, defined as a decrease in maximal force or power production capacity (Bigland-Ritchie & Woods, 1984; Gandevia, 2001). Considering deceptive paradigms, one can intuitively assume that improved performance leads to increased fatigue. However, deception studies investigating cycling time trial tasks (i.e., race on a pre-defined distance) provide conflicting evidence for this statement. On the one hand, one study found indeed an increase in generated neuromuscular fatigue (Ducrocq et al., 2017),

quantified (i) as a larger decrease in the ability of the central nervous system (CNS) to recruit motor units (i.e., central fatigue) and (ii) as a further recovery impairment of mechanisms occurring downstream of the neuromuscular junction (i.e., peripheral fatigue) (Bigland-Ritchie, 1981; Sesboüé & Guincestre, 2006). On the other hand, another study found no evidence in favor of changes in neuromuscular fatigue for a similar cycling task (Ansdell et al., 2018).

The current study aimed to address this relevant question by applying deceptive clock manipulation during a single-joint isometric task to failure. At first, this allowed us to assess the clock manipulation effect on participants' performance in this type of task for the first time. Then, we determined the underlying neurophysiological adaptations associated with deceptive time manipulation, which have not been investigated in previous studies. Importantly, this paradigm minimizes critical confounding factors, such as pacing strategy (Amann et al., 2008) and muscular compensation, while optimizing neurophysiological recordings (Bigland-Ritchie, 1981). Overall, we expected an increase in performance - defined as real holding time - when the clock is deceptively slowed down, in line with previous studies using related paradigms (Ansdell et al., 2018; Ducrocq et al., 2017; Morton, 2009). Concerning the underlying neurophysiological adaptations associated with the manipulation, our initial hypothesis was to observe a rise in the generated neuromuscular fatigue, consistent with the increase in holding time when the clock is slowed down (Ducrocq et al., 2017). However, our results refute this intuition, showing a similar amount of generated neuromuscular fatigue despite increasing holding time when the clock is slowed down (Ansdell et al., 2018). We then implemented fitting computational models to deepen our understanding of the mechanisms that underlie these findings. Remarkably, we found that neuromuscular fatigue accumulation depends on subjective rather than real time for the subjects involved in this performance improvement.

#### 2 | METHOD

Participants came to the laboratory five times (each visit being labeled a session) and performed during each session the same isometric contraction task of the quadriceps against a fixed resistance for as long as possible ("Time To Failure," TTF). Their performance, corresponding to their holding time, was displayed to them on a computer screen throughout the task by a digital clock continuously available. The maximum holding time of the first three sessions was retained for each subject, constituting the Baseline. During one of the last two sessions (counterbalanced

and randomized through the participants), the clock was slowed down to 90% of its actual speed unbeknownst to the subjects. In these last two sessions, we distinguished the Biased session, when the clock was slowed down, and the Normal session, when the clock calibration was not

modified. Neuromuscular fatigue was assessed before, during, and after the TTF task in Normal and Biased sessions. Inter and intra-sessions procedures are described in Figure 1. Data presented in the text and the tables are expressed as mean  $\pm$  SD.

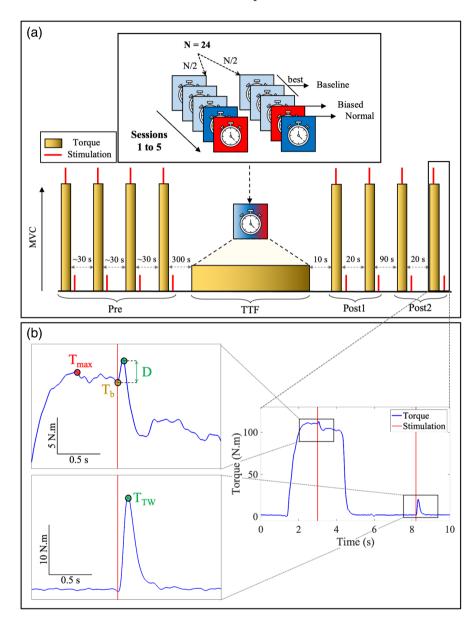


FIGURE 1 Experimental Protocol. (a) The lower part of the panel represents the course of a session. Each session started and ended with four Maximal Voluntary Contractions (MVC) of the right leg extensors, divided into Pre-Test, Post-Test1, and Post-Test2. The Time To Failure task (TTF) consisted of maintaining an isometric leg extension as long as possible against a fixed load corresponding to 20% of MVC recorded in the Pre-Test during the first session. The performance corresponds to the holding time performed. The box above describes the sequence of the five sessions (each spaced 3 to 10 days apart). All the subjects started with three sessions where the clock was not manipulated. We retained their best time from these three sessions, which constituted the Baseline. Then, the clock was either normal (Normal) in session four and then slowed down (Biased) in session five or vice versa (counterbalanced between participants). Torque and time were recorded during each session. In addition, during Normal and Biased sessions, we recorded electromyographic data and performed neurostimulations (vertical red bars). (b) A detailed view of one MVC shows how our main neuromuscular dependent variables were measured.  $T_{max}$  is the maximal voluntary torque reached during the MVC,  $T_{TW}$  is the amplitude of the potentiated twitch measured at rest (3 to 4s after the MVC), and VAL is the voluntary activation level. This last one was computed as follows:  $VAL = 100 - D^*(T_b/T_{max})/T_{TW}^*100$ , with D the amplitude of the superimposed twitch measured during MVCs and  $T_b$  the voluntary torque recorded just before the superimposed twitch. Pre-Post changes in  $T_{max}$ , VAL, and  $T_{TW}$  allow us to assess the general, central, and peripheral fatigue, respectively.

# 2.1 | Participants

Twenty-four healthy volunteers (13 men and 11 women, age:  $24 \pm 4$  years, mass:  $69 \pm 10$  kg, means  $\pm$  SD) with no history of neurological injuries or diseases participated in this study. They all practiced physical activity on a regular basis, that is, one to several times a week. Participants were informed that the study was interested in fatigue and cessation of exercise. Assessment of multiple sessions, presented as strictly identical, was justified as allowing to validate the measurements on several occasions, to override familiarity or goal effects, and to look for potential variability in the neurophysiological mechanisms involved. No further details were given, and subjects were informed about deception only after the completion of the whole study. The study protocol was approved by the French Ethical "Comité de Protection des Personnes Ile de France VI" (number 2022-A00032-41). All participants provided signed informed consent before enrolling in the study.

The sample size was determined using  $G^*Power$  (Faul et al., 2007) according to pilot experiments that showed 24 participants were sufficient to achieve a power of .80 in the current experiment.

# 2.2 | Experimental protocol

Participants performed a TTF task in a strictly identical way during the five sessions. For the same subject, each session (spaced 3 to 10 days apart) was scheduled at the same time of day to account for diurnal neuromuscular variations (Tamm et al., 2009). Participants were asked to avoid strenuous physical activity 24 h before each session and to come in the same conditions of sleep and nutrition.

The TTF task consisted of isometric holding of a right leg extension (knee at 90°) against a fixed resistance set at 20% of the average quadriceps torque recorded upstream during the first session (T<sub>PRE-S1</sub>, see below). As mentioned in the introduction, the single-limb isometric design allowed to minimize critical confounding factors. Indeed, only one muscle group was mobilized in a standardized position against a fixed resistance, making any pacing strategies and compensation from other muscles minimal. Furthermore, the neurophysiological assessment was performed in the same position as the task to failure, avoiding confounding factors due to the muscle length, for example, and allowing to reduce the delay of post-test neurophysiological assessment, thus preventing the interference of recovery on the latter (Place & Millet, 2020).

During the TTF task, participants had a screen in front of them displaying a digital clock informing them about their holding time online; and a counter that indicated the torque produced in real time and the torque targeted (with a tolerance range set at 90% of the 20% T<sub>PRE-S1</sub>). From the second session, the best time previously achieved by the subjects was reminded to them, and they were asked to perform as well as possible, trying at least to beat their previous best performance. The maximum holding time of the first three sessions was retained for each subject, constituting the Baseline (Ducrocq et al., 2017). During one of the two last sessions (fourth or fifth), in a counterbalanced and randomized order, the clock (custom-made application) was slowed down at 90% of its normal speed without participants' knowledge (100 s displayed = 111 s real time). In these last two sessions, we distinguish the Biased session, when the clock is slowed down, and the Normal session, when the clock calibration is not modified. Note that the 90% magnitude was chosen in accordance with the literature to maximize the effects of the manipulation while remaining undetectable (Ansley et al., 2004; Morton, 2009).

During each session, strong verbal encouragement to continue was provided frequently (at least every 30 s (Andreacci et al., 2002), but without a fixed frequency to avoid any temporal cue), and almost continuously when it became difficult. Similarly, milestone times were orally provided throughout the task as intermediate objectives to maximize performance. Indications about the time displayed on the clock were also provided regularly (at least every 30 s, without a fixed frequency) to maximize the orientation of attention to the time cue. All these verbal indications were standardized and were always delivered by the same experimenter.

During each session, isometric maximal voluntary contractions (MVC) of the right leg extensors (quadriceps) were performed on the isokinetic ergometer before and after the TTF task. Prior to the task, subjects performed four 3 to 4s MVCs with a recovery time between 20 and 40 s between each repetition. 10s after the TTF ending, subjects performed two MVCs again, spaced 20 s apart, and then two more 2 min after the TTF end, with the same pattern (Figure 1a). Through sessions 4 and 5, during each MVC, an electrical stimulation was performed on the torque plateau and then one 3 to 4s after contraction on the relaxed muscle (Figure 1b). Two types of torque variations associated with these stimulations can be distinguished: (i) the superimposed twitch, observed during the application of the stimulation during the contraction, (ii) the potentiated twitch, recorded during the application of the stimulation at rest, 3 to 4s after the contraction. These twitches allow to calculate the voluntary activation level (VAL) (Merton, 1954), that is, the proportion of motor units recruited by the central nervous system during the contraction. The potentiated twitch (T<sub>TW</sub>) also allows learning about a possible alteration of the peripheral mechanisms (Kufel et al., 2002; Taylor & Gandevia, 2008). To specify these measures of central and peripheral fatigue at the superficial quadriceps muscles, the compound muscle action potential (M-wave), a characteristic electromyographic (EMG) response that reflects sarcolemma excitability (Rodriguez-Falces & Place, 2018), was also recorded for each muscle following stimulation. We distinguish (i)  $M_{\rm sup}$ , evoked during contraction, which allows normalizing the EMG signal recorded during the MVCs to specify central fatigue of each muscle studied (normalization allows us to exclude peripheral changes) (Millet et al., 2011) and (ii)  $M_{\rm max}$ , evoked at rest.

#### 2.3 | Measurements

During each whole session, subjects were seated on an isokinetic ergometer (Biodex, Shirley, NY) with both knees and hips flexed at 90° (0°=full extension). Their right leg was firmly attached to the ergometer accessory at the ankle, just above the malleolus, with a non-compliant strap. Participants were fastened to the ergometer by two shoulder harnesses and one abdominal harness to minimize body movements and to limit the contribution of other muscle groups aside from the quadriceps. All settings were recorded during each subject's first session and maintained for subsequent sessions to guarantee an identical subject position.

#### 2.3.1 Torque and EMG

Torque was recorded at 2000 Hz using a Biopac MP150 system and Acknowledge software. In synchronization with mechanical data, EMG data were recorded (gain ×500) at a sampling frequency of 2000 Hz, only during the Normal and Biased sessions (sessions 4 and 5). In accordance with the SENIAM recommendations, the skin was previously prepared to obtain a low impedance ( $<5 \text{ k}\Omega$ ), then three pairs of electrodes (AG-Cl, diameter: 11 mm, interelectrode distance: 2 cm) were placed on the muscle belly of Vastus Medialis (VM), Vastus Lateralis (VL), and Rectus Femoris (RF). If needed, electrodes placements were adjusted to obtain the greatest M-wave amplitudes (Place et al., 2006; Rodriguez-Falces & Place, 2013) in response to femoral nerve stimulation (see below). The reference electrode was placed on the patella of the contralateral knee.

#### 2.3.2 Motor nerve stimulation

In sessions 4 and 5, percutaneous stimulations were applied on the right femoral nerve with a single rectangular pulse delivered by a constant current stimulator (pulse duration:  $500\,\mu s$ ; Digitimer stimulator, model DS7, Hertfordshire,

UK). The cathode (AG-Cl, 11 mm diameter) was positioned on the femoral triangle (Verges et al., 2009), in the location that elicited the maximum quadriceps twitch amplitude at rest. The anode (5 cm  $\times$  10 cm, Medicompex SA, Switzerland) was placed midway between the right great trochanter and the iliac crest. The optimal stimulation intensity was determined as the minimum current that elicited maximum quadriceps twitch amplitude at rest. To ensure a supra-maximal stimulus, the intensity was then increased by 20% (Neyroud et al., 2014; Vagg et al., 1998). The stimulation intensity was similar between sessions (Normal:  $114\pm30\,\text{mA}$ , Biased:  $118\pm30\,\text{mA}$ , p=.089, Intraclass Correlation Coefficient (ICC)=0.92).

# 2.4 Data processing

The data set was processed using custom-made MATLAB software (MathWorks, USA).

#### 2.4.1 Mechanical data

Torque was filtered using a low-pass 4th order Butterworth filter at a cutoff frequency of  $10\,\mathrm{Hz}$ . The baseline torque, taken at rest, was systematically subtracted from the data set. During MVCs, maximal torque  $(T_{max})$  was recorded for each contraction (before stimulation when present). The four  $T_{max}$  recorded before the TTF task were averaged to constitute  $T_{max\text{-PRE}}$ . The two  $T_{max}$  recorded  $10\,\mathrm{s}$  and  $30\,\mathrm{s}$  after the task were also averaged  $(T_{max\text{-POST1}})$ . The same averaging was applied to the two  $T_{max}$  recorded 2' and 2'20 after the task  $(T_{max\text{-POST2}})$ .  $T_{max\text{-PRE}}$  measured in the first session constitutes the reference  $(T_{PRE\text{-S1}})$  used to calculate the fixed resistance (20% of  $T_{max\text{-PRE}})$  for each participant during the TTF task (resistance that was therefore identical for each participant respectively for the five sessions, see above).

Voluntary activation level (VAL) was computed using the following equation (Strojnik & Komi, 1998):

$$VAL = 100 - \frac{D * (T_b / T_{\text{max}})}{T_{\text{TW}}} * 100$$

where D is the amplitude of the superimposed twitch measured during MVCs,  $T_b$  is the voluntary torque collected just before the superimposed twitch,  $T_{max}$  is the maximal voluntary torque reached during the contraction, and  $T_{TW}$  is the amplitude of the potentiated twitch measured at rest. In the same way, as  $T_{max}$ , VAL and  $T_{TW}$  were averaged separately over the three defined time windows constituting VAL<sub>PRE</sub>, VAL<sub>POST1</sub>, VAL<sub>POST2</sub>,  $T_{TW-PRE}$ ,  $T_{TW-POST1}$ , and  $T_{TW-POST2}$ , respectively.

# 2.4.2 | Holding time

The TTF task was delineated using a torque thresholding method. The start of the TTF task was taken as the first instant the actual torque exceeded 90% of target torque (20%  $T_{PRE-S1}$ ) for 3 s; and the end was determined as the first moment this was no longer the case, from which 3 s was then subtracted. Because the EMG signal was analyzed during the TTF task (see below), this subtraction allows us to avoid including EMG signals when the muscle is relaxed in our analysis. Holding time was defined as the duration between the start and end of the TTF task.

# 2.4.3 | Electromyographic data

EMG data were prefiltered during acquisition using a notch filter at a cutoff frequency of 50 Hz. M-wave was quantified by the peak-to-peak amplitude for VM, VL, and RF, individually.  $M_{max}$  were averaged independently for each muscle, as previously described ( $M_{PRE}$ ,  $M_{POST1}$ , and  $M_{POST2}$ ). EMG signal was then filtered using a bandpass Butterworth filter 4th order between 20 and 500 Hz. The filtered signal was quantified as Root Mean Square (RMS) for each MVC over an integration time of 500 ms before the application of stimulation. For each MVC and each muscle, this signal was normalized by the peak-to-peak amplitude of the associated  $M_{sup}$ . RMS/ $M_{sup}$  ratios were then averaged for each muscle, as described above (RMS/ $M_{PRE}$ , RMS/ $M_{POST1}$ ), and RMS/ $M_{POST2}$ ).

# 2.4.4 | Modeling fatigue accumulation

For participants who did a higher performance in the Biased session compared to the Normal session (real time in Biased session > real time in Normal session), the EMG signal corresponding to the TTF task was delineated for both sessions, corresponding to the holding time delimited above on the torque data. Then, for the same subject, the Biased session was truncated to match the Normal session time. The EMG signal was quantified as RMS over an integration period equivalent to 1% of the total time retained over the session, constituting 100 values for each subject and session. The RMS values obtained were normalized by the RMS<sub>PRE</sub> of the associated session and muscle. Then, the average of the RMS/RMS<sub>PRE</sub> from VM, VL, and RF was performed. These RMS/RMS<sub>PRE</sub> data are thought to reflect the central motor command increase that occurs during the task (Taylor et al., 2016), consequently to neuromuscular fatigue development. In this sense, they are commonly used as an indicator of neuromuscular fatigue accumulation (Kim et al., 2007; Merletti et al., 1991).

A second model was applied to account for the whole Biased session. To this end, based on the average progression of participants who strictly improved their holding time during the Biased session (+15% compared to the Normal session), the EMG signal was quantified as RMS over an integration period equivalent to 1% of the time of the Normal session, and thus constituting 115 values (each representing 1%) for the Biased session, for each subject. The same normalization and averaging were then established. All the modeling analyses were performed using Curve Fitting Toolbox (MATLAB). The power function selected to model the RMS evolution (see *Results* for details) was chosen according to physiological plausibility, visual discrimination, residual minimization, and parsimony criteria.

# 2.5 | Statistical analysis

The normality and sphericity of all data distribution were verified using the Kolmogorov–Smirnov test and Mauchly's W test, respectively. The RMS/M<sub>sup</sub> ratio of the VM and all M<sub>max</sub> waves exhibited moderate positive skewness that could be corrected by applying a square root transformation:  $x' = \sqrt{x}$ . The VAL had a strong negative skewness which can be explained by the characteristics of this variable (bounded at 100%). An arcsine transformation of the type:  $x' = 2*a\sin\left(\sqrt{x}\right)$  was applied to decorrelate the mean value from the variance. The distribution of these data was assessed as normal. Note that untransformed data have been kept for the graphs and tables to ensure their visualization. When sphericity was not verified, the Huynh–Feldt degrees of freedom correction was applied.

Repeated measures ANOVAs were performed to test differences between sessions (Session: Baseline, Normal, and Biased) separately for real and displayed holding time. Note that a sex group factor (Sex: Male and Female) had initially been added, but since the mixed ANOVA performed did not indicate a sex effect on holding time, we removed it and kept all subjects as one pool. Neurophysiological markers ( $T_{max}$ , VAL,  $T_{TW}$ , as well as RMS/ $M_{sup}$  and  $M_{max}$ for VM, VL, and RF) were analyzed individually using two-factor repeated-measures ANOVAs: clock calibration (Session: Normal, Biased) and period of measurement (Period: Pre-Test, Post-Test1, and Post-Test2). Significance was set at p < .05. ANOVAs' results are presented along with partial eta squared  $(\eta^2)$ , and the effect size measurement Cohen's f(f). For all ANOVAs, Bonferroni pairwise comparison tests were run post hoc when a significant main or interaction effect was observed. Cohen's  $d(d_7)$  (Lakens, 2013) and ICC (Koo & Li, 2016) were also

provided when necessary. Because we wanted to draw solid conclusions about a potential absence of neuromuscular fatigue difference, Bayes Factor (BF) were calculated on the main neuromuscular fatigue markers ( $T_{max}$ , VAL, and  $T_{TW}$ ) in the event of non-significant result to overcome the limitations of the frequentist approach regarding the acceptance of the null hypothesis (Masson, 2011). Repeated measures ANOVAs and BF were also computed on the main neuromuscular fatigue markers ( $T_{max}$ , VAL, and  $T_{TW}$ ) for the sub-group of subjects who improve their performance in Biased compared to Normal session. All analyses were performed in R (CranR, version 4.1.1), except BF, which were calculated in JASP (JASP Team 2022, default priors, analysis of effects across matched models).

#### 3 RESULTS

# 3.1 | Initial maximal voluntary torque

Before our main analyses, we controlled that participants came in a similar shape during the Baseline, Normal and Biased sessions. No significant difference was observed between maximal voluntary torque recorded in Pre-Test ( $T_{\text{max-PRE}}$ ) during these sessions (p=.127; Baseline:  $223\pm55\,\text{N.m}$ , Normal:  $218\pm57\,\text{N.m}$ , Biased:  $220\pm55\,\text{N.m}$ ; ICC=0.97). These results allowed considering the participants' similar state of strength during these three sessions.

# 3.2 | Holding time

We first performed a repeated measures ANOVA considering *Session* (Baseline, Normal, Biased) as an independent variable. As hypothesized, repeated measures ANOVA on the TTF real holding times revealed a significant difference between the three sessions ( $F_{(2,46)}=11.15$ , p<.001,  $\eta^2=0.33$ , f=0.70). The real time in the Biased session was significantly higher than the time performed in the Normal session (p=.036,  $d_z=0.56$ ) and in the Baseline (p<.001,  $d_z=0.93$ ), while no significant difference was detected between real times performed during the Normal and Baseline sessions (p=.235,  $d_z=0.38$ , ICC=0.96) (Table 1). The high concordance between the holding time in the Baseline and Normal sessions and the counterbalanced order between the Normal and Biased sessions

**TABLE 1** Displayed and real holding time.

	Baseline	Normal	Biased
Displayed time (s)	$321 \pm 114$ [276:367]	$332 \pm 112$ [288:377]	$321 \pm 106$ [278:363]
Real time (s)	$321 \pm 114$ [276:367]	$332 \pm 112$ [288:377]	$356 \pm 118^{ab} [309:403]$

<sup>&</sup>lt;sup>a</sup>Different from Baseline: p < .001.

allow us to rule out a possible training effect induced by the repetition of the task. Finally, the displayed times appeared analogous between sessions (p=.208, ICC=0.93) (Table 1). Taken together, these results indicate a physical performance, assessed by holding time, relying on subjective (i.e., displayed) rather than real time.

# 3.3 Neuromuscular fatigue

Following the increase in performance during the Biased session compared to the Baseline and Normal sessions, we compared the neurophysiological measurements recorded via two-factor repeated measures ANOVAs, considering Period (Pre-Test, Post-Test1, and Post-Test2) and Session (Biased and Normal) as factors, to assess any difference in generated neuromuscular fatigue. First, repeated measures ANOVA performed on maximal voluntary torque (T<sub>max</sub>) reveals a significant *Period* effect  $(F_{(2.46)} = 81.41, p < .001,$  $\eta^2 = 0.78$ , f = 1.88). Pairwise comparisons indicated that T<sub>max-POST1</sub> and T<sub>max-POST2</sub> were significantly lower than  $T_{\text{max-PRE}}$  (p < .001, rate of change: -47 ± 16%; -32 ± 14%, respectively).  $T_{\text{max-POST2}}$  was higher than  $T_{\text{max-POST1}}$  (p < .001) (Figure 2a). These results indicate the presence of neuromuscular fatigue generated during the TTF task (measured in Post-Test1, i.e., 10s after the TTF task), which recovers partially 2 min after the task (Post-Test2). Surprisingly, there was no significant Session effect (p=.200) or interaction effect between Session and Period (p = .099), as confirmed via Bayesian repeated-measures ANOVA (BF < 0.210 for both effects). Overall, the amount of generated neuromuscular fatigue was not different between the Normal and Biased sessions, meaning that prolonged maintenance of effort during clock manipulation did not impact the magnitude of the neuromuscular fatigue generated or its short-term recovery.

#### 3.3.1 | Central fatigue

Neuromuscular fatigue can be divided into central and peripheral components (Bigland-Ritchie, 1981). In the current report, central fatigue was first quantified by the pre- to post-exercise changes of the voluntary activation level (VAL), a marker related to the proportion of motor units recruited by the CNS during the contraction (Merton, 1954). Consistently with  $T_{max}$  changes, a

<sup>&</sup>lt;sup>b</sup>Different from Normal session: p < .05; 95% CI of the mean are reported in square brackets.

FIGURE 2 Neuromuscular fatigue. Each panel's figure shows maximal voluntary torque (a), voluntary activation level (b), and potentiated twitch (c), which allows assessing general, central, and peripheral fatigue, respectively. Asterisks indicate significant differences with p < .001. The dots represent individual data (averaged for the same individual between the Normal and Biased sessions). The midline represents the median of the distribution, and the lower and upper ones are the first and third quartiles, respectively. The whiskers encompass points within 1.5 times the length of the Q2-Q1 or Q3-Q2 space. The figure shows that the values are significantly lower in Post-Test1 compared to Pre-Test irrespective of the variable, indicating the presence of neuromuscular fatigue. This fatigue recovers or not in a partial way in Post-Test2. Importantly, for each variable, we observe no difference within the same measurement period between the Normal and Biased sessions, revealing that neuromuscular fatigue is established in the same proportions regardless of the session.

TABLE 2 EMG assessment of neuromuscular fatigue.

	Pre-Test	Post-Test1	Post-Test2
$RMS/M_{sup} VM (\emptyset)$	$0.055 \pm 0.025 [0.045:0.065]$	$0.047 \pm 0.023^{a} [0.038; 0.057]$	$0.044 \pm 0.018^{a} [0.037; 0.051]$
$RMS/M_{sup} VL(\emptyset)$	$0.051 \pm 0.018  [0.044 : 0.058]$	$0.049 \pm 0.015 \ [0.043:0.056]$	$0.045 \pm 0.013^{a} [0.040:0.051]$
$RMS/M_{sup} RF(\emptyset)$	$0.059 \pm 0.020  [0.051:0.067]$	$0.046 \pm 0.016^{a} [0.040:0.052]$	$0.047 \pm 0.016^{a} [0.041:0.054]$
M <sub>max</sub> VM (mV)	$3.164 \pm 1.388$ [2.609:3.720]	$3.004 \pm 1.407$ [2.441:3.567]	$3.184 \pm 1.539$ [2.569:3.800]
$M_{max} VL (mV)$	$2.301 \pm 1.234  [1.807:2.795]$	$2.024 \pm 1.311^{a} [1.499:2.548]$	$2.240 \pm 1.284^{b}$ [1.727:2.754]
M <sub>max</sub> RF (mV)	$2.453 \pm 0.889$ [2.097:2.809]	$2.376 \pm 0.859$ [2.032:2.720]	$2.444 \pm 0.852$ [2.103:2.784]

Abbreviations: RF, Rectus Femoris; VL, Vastus Lateralis; VM, Vastus Medialis.

Pre Post1 Post2

significant effect for the VAL regarding the Period factor was found  $(F_{(2.46)} = 19.83, p < .001, \eta^2 = 0.46, f = 0.93),$ but no effect for Session (p=.923) or their interaction (p=.691), as confirmed via a Bayesian analysis (BF< 0.180 for both effects). Pairwise comparisons showed that, independently from the sessions, VAL<sub>POST1</sub> and VAL<sub>POST2</sub> were significantly lower than VAL<sub>PRE</sub> ( $p < .001, -7 \pm 9\%$ ;  $-6 \pm 6\%$ , respectively) without any difference between the two post-test measures (p=1.000, BF=0.20) (Figure 2b). As the global neuromuscular fatigue ( $T_{max}$ ), the decrease in the CNS capacity to recruit motor units (VAL) emerges in the same proportions regardless of the session, notwithstanding the performance improvement in the Biased session.

Further analyses were performed on RMS/M<sub>sup</sub> ratios to specify the central fatigue to each superficial quadriceps muscle (Millet et al., 2011). Repeated measures ANOVA revealed a significant Period effect for RMS/M<sub>sup</sub> ratios of each muscle: Vastus Medialis  $(F_{(2.46)} = 7.50, p = .002, \eta^2 = 0.25, f = 0.57)$ , Vastus Lateralis  $(F_{(2.46)} = 3.75, p = .031, \eta^2 = 0.14, f = 0.40)$ , and Rectus Femoris  $(F_{(2.46)} = 27.02, p < .001, \eta^2 = 0.54, f = 1.08).$ Differences from the pairwise comparisons are shown in Table 2. Globally, these ratios indicate that all three quadriceps muscles are involved in the macroscopically observed voluntary activation deficit and its short-term persistence.

Post1 Post2

<sup>&</sup>lt;sup>a</sup>Different from Pre-Test: p < .05.

<sup>&</sup>lt;sup>b</sup>Different from Post-Test1: p < .001; 95% CI of the mean are reported in square brackets.

# 3.3.2 | Peripheral fatigue

Neuromuscular fatigue can also be quantified in its peripheral component, assessed in the current study especially through pre-post changes of potentiated twitch (T<sub>TW</sub>), which corresponds to the muscle response to a maximal electrical motor nerve stimulation at rest (Kufel et al., 2002). Hence, changes in T<sub>TW</sub> directly reflect the amount of peripheral fatigue, which accounts for all mechanisms from the neuromuscular junction to the excitation-contraction coupling (Gandevia, 2001). Repeated measures ANOVA performed on T<sub>TW</sub> presents a significant *Period* effect  $(F_{(2,46)} = 87.65, p < .001, \eta^2 = 0.79,$ f=1.95). In the same way as  $T_{max}$ , pairwise comparisons show that both  $T_{TW-POST1}$  and  $T_{TW\_POST2}$  are significantly lower than  $T_{TW-PRE}$  ( $p < .001, -58 \pm 17\%; -34 \pm 15\%,$ respectively). T<sub>TW-POST2</sub> is also higher than T<sub>TW-POST1</sub> (p < .001) (Figure 2c). Repeated measures ANOVA also revealed a Session-Period interaction effect  $(F_{(2.46)} = 4.87,$ p < .012,  $\eta^2 = 0.17$ , f = 0.46) but no Session effect (p = .303, BF = 0.240). Despite the presence of an interaction effect, pairwise comparisons denoted no difference between sessions for the same Period (i.e., Pre-Test, Post-Test1, or Post-Test2, respectively) (p>.410) (Figure 2c). Furthermore, BF computed for Session-Period interaction effect in the Bayesian repeated-measures ANOVA was 0.161, showing again evidence in favor of the null hypothesis. All in all, the generated muscle fatigue (Kufel et al., 2002) and its short-term recovery appeared similar between the Normal and Biased sessions, despite a performance improvement in the latter.

We performed further analyses to investigate the causal origin of generated peripheral fatigue (cf. Period effect). Specifically, we investigated the compound muscle action potential electrically evoked at rest (M<sub>max</sub>), a typical EMG response reflecting sarcolemma excitability (Rodriguez-Falces & Place, 2018). A significant Period effect for M<sub>max</sub> was found only for Vastus Lateralis ( $F_{(2,46)} = 14.22, p < .001,$  $\eta^2 = 0.38, f = 0.79$ ). Differences from the pairwise comparisons are shown in Table 2. As T<sub>TW</sub> decreases in Post-Test and M<sub>max</sub> remains stable for Vastus Medialis and Rectus Femoris, peripheral fatigue originating from impaired calcium phenomena could be assumed (Allen et al., 2008; Lepers, 2010). The decrease in  $M_{max}$  of the Vastus Lateralis measured just after the task may also suggest an alteration in sarcolemma excitability contributing to peripheral fatigue (Fuglevand et al., 1993).

#### 3.4 | Control analyses

In the previous analyses, we found an improvement in performance when the clock was slowed down without increasing the associated neuromuscular fatigue. To rule out a possible increase in neuromuscular fatigue masked by participants who did not improve their performance when the clock was slowed down, the same analyses were performed on our main neurophysiological fatigue markers ( $T_{\rm max}$ , VAL,  $T_{\rm TW}$ ) only for participants who strictly improved their holding time in the Biased session (N=16/24). Interestingly, the same pattern of results was found, with a BF always less than or equal to 0.3 concerning *Session* and *Session-Period* effect, supporting evidence in favor of an absence of difference in neuromuscular fatigue between the Normal and Biased sessions.

# 3.5 | Modeling fatigue accumulation

As previously demonstrated, our data revealed that the generated neuromuscular fatigue was analogous in the Normal and Biased sessions, despite a performance improvement in the latter, even if we keep only the participants who improve their performance when the clock is slowed down. Therefore, to explain these results, one could suppose a modified neuromuscular fatigue accumulation during the TTF task for these participants (Hunter & Enoka, 2003). According to recent studies on mindbody influence (Park et al., 2016; Pomares et al., 2011), we could hypothesize an evolution of neuromuscular fatigue accumulation according to subjective rather than real time. To verify this hypothesis, we considered the RMS/RMS<sub>PRE</sub> data collected during the TTF task, which reflect the kinetic of neuromuscular fatigue (Merletti et al., 1991). Specifically, we fitted a power function to the average of the RMS/RMS<sub>PRE</sub> data, for subjects who improved their performance during the Biased session (N=16), as follows:

$$y(x) = a * x^b + c$$

In particular, we fitted the data to both the Normal and the Biased sessions, but the latter was truncated for each subject at the holding time performed during the Normal session. In the equation, x represents time, y(x) is the RMS/RMS<sub>PRE</sub> ratio as a function of time, and a, b, and c are constants optimized for each session, respectively.

Once we modeled the accumulation of neuromuscular fatigue during the Normal session, we predicted the data in the biased condition (labeled "Prediction" in the following) if fatigue did indeed accumulate according to the subjective time (at 90% of real time). Concretely, we established our Prediction using the same model obtained with the Normal session (same constants a, b, and c) but changing only the time variable by adding a 0.90 factor on time (x). Then we compared the

Prediction and the Biased data (Figure 3a). As shown in Figure 3a, the curve modeling the Biased session overlaps considerably with the Prediction, as quantified by the minimal magnitude of the differences between these curves (NRMSE = 0.0079). This surprising result reveals, for participants who improved their holding time in the Biased session, an average accumulation of neuromuscular fatigue during the Biased session at a speed of 90% of the Normal session, that is according to displayed rather than real time.

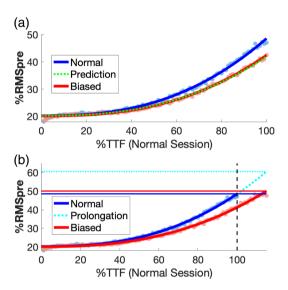


FIGURE 3 Modeling of neuromuscular fatigue accumulation. (a) The dots represent averaged RMS/RMS<sub>PRE</sub> data from the Normal session (blue) and the truncated Biased session (red). The solid curves represent the power models derived from these points. The green dashed curve represents the Prediction established from the equation of the blue curve (Normal session) by applying a coefficient of 0.90 to time. We see an almost perfect match between the predicted and observed data, thus highlighting a subjective time-dependent neuromuscular fatigue accumulation. (b) As in (a), dots represent the averaged RMS/RMS $_{\rm PRE}$  data of the Normal session (blue) and the complete Biased session (red), established based on averaged progression. The solid curves represent the power models derived from these points. The curve of the Normal session has been extended (cyan) to be equal to the mean time performed during the Biased Session. This panel shows that the actual threshold reached at the end of the TTF task in the Biased session (50%, in red) is very similar to the one reached in the Normal session (49%, in blue). Furthermore, the Biased threshold is considerably lower than the Prolongation one (61%, in cyan), which would be theoretically reached if participants performed an identical holding time than in the Biased session but with the same neuromuscular fatigue accumulation as in the Normal session. These results are fully consistent with the findings of a slowed neuromuscular fatigue accumulation in the Biased session, as presented in panel (a). See the text for further details on the models' construction.

Similarly, we modeled the complete Biased and Normal sessions with a theoretical "Prolongation" of the RMS/RMS<sub>PRE</sub> data curve of the Normal session. As shown in Figure 3b, the curves representing the Biased session and the Prolongation reach the average mean holding time performed in the Biased session by the participants who improved their performance in the latter (i.e., +15% compared to the Normal session). The purpose of this analysis was to estimate the maximal RMS/RMS<sub>PRE</sub> threshold reached by these participants if their holding time was identical to the Biased session but considering an identical neuromuscular fatigue accumulation as in the Normal session. Interestingly, this estimation reveals a considerably higher maximal threshold activity (61%) than the actual threshold reached (50%), and the effort cessation for the Normal and Biased sessions occurs for a very close RMS/RMS<sub>PRE</sub> value (49% and 50%, respectively, of maximal electromyographic activity). All in all, our results demonstrate that participants who improved their performance in the Biased session stopped their effort for a similar EMG threshold, irrespective of the time manipulation. This indicates that neuromuscular fatigue accumulation can be modulated by subjective factors (such as time perception) thus delaying such failure threshold.

# 4 | DISCUSSION

In the present study, we investigated the underlying neurophysiological adaptations that support a holding time improvement due to a deceptive clock manipulation. Our main results showed a significant performance improvement when the clock was slowed down without increasing the associated neuromuscular fatigue. Further analyses revealed that neuromuscular fatigue accumulation seems to evolve according to subjective rather than real time for participants who improved their performance when the clock was slowed down.

# 4.1 | Increasing performance without increasing fatigue

Participants' real holding time was significantly longer in the Biased session compared to the Baseline and Normal sessions, indicating an improved performance during the temporal manipulation. Our results are consistent with previous studies (Ansdell et al., 2018; Ducrocq et al., 2017; Morton, 2009) by relating an improvement in performance following a temporal manipulation. So far, studies focused exclusively on whole-body tasks (Williams et al., 2014), and ours is the first to extend the deception

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effect to a single-joint isometric task. Our approach notably allows minimizing confounding factors, such as muscular compensation, as responsible for the performance improvement. Curiously, it was previously observed that the increase in performance following temporal manipulation was specific to men (Morton, 2009). One proposed explanation was based on psychological mechanisms, especially the competitiveness of the participants (Morton, 2009). However, this sex differentiation was not observed in our study.

Our results support the findings that physical performance limits are not solely based on physiological principles. Psychological factors play a key role in setting physical performance limits (Marcora & Staiano, 2010; Noakes, 2011). Previous studies suggested that perceiving the current performance as slightly degraded compared to a previous one may act as a motivational factor (Ansdell et al., 2018). Some authors proposed motivational input as an excitatory signal that increases motor output to muscles, as opposed to an inhibitory pathway (Tanaka & Watanabe, 2012). A motivational state could therefore allow for prolonged motor responses (Robertson & Marino, 2016; St Clair Gibson et al., 2013), increasing the level of fatigue achieved (Ducrocq et al., 2017; Rube & Secher, 1981). However, surprisingly, no increase in neuromuscular fatigue, regardless of the component (central, peripheral) studied, was observed within our study. Furthermore, since the peripheral fatigue was deemed at the same level irrespective of the session, the improvement of holding time in the Biased session does not seem to be due to exceeding a "critical" threshold of peripheral fatigue (Amann et al., 2013) and drawing on contractile reserves (Ducrocq et al., 2017) as proposed before. In the following, we reviewed several explanations to account for our results.

First, methodological limitations could be considered responsible for the absence of neuromuscular fatigue increase. Ansdell and colleagues (2018) previously suggested that the increase in fatigue (associated with the increase in performance) may be too negligible to present a significant difference. Furthermore, methodological temporal constraints may lead to a slight underestimate of the fatigue generated during the task (Place & Millet, 2020) (i.e., Post-Test1 consisting of two contractions, the first 10 s after task cessation and the second 30s after task cessation). Using doublet rather than single stimuli could also have increased the accuracy of VAL and T<sub>TW</sub> measures (Oskouei et al., 2003; Zarkou et al., 2017). However, all these methodological considerations do not seem sufficient to mask a potential difference in fatigue between sessions if there was one (Ducrocq et al., 2017). Moreover, since the measurement methodology is the same in both Normal and Biased sessions, the limitations identified appear identical

between sessions, reducing their potential effects. Second, we have considered the idea that the participants who did not improve could hide a neuromuscular fatigue increase when the clock was slowed down. However, the additional analyses performed only on the participants who strictly improved their holding time in the Biased session (N=16)allowed us to discard this hypothesis by showing the same effects as the whole subjects' pool. Our final hypothesis to explain an increase in performance without an increase in neuromuscular fatigue was that neuromuscular fatigue accumulates more slowly (Hunter & Enoka, 2003) when the clock is slowed down, as we'll discuss below.

# Mind's influence on neuromuscular processes

Recent discoveries emphasized the mind's influence on fundamental body functions (Crum et al., 2011; Langer et al., 2010; Thönes et al., 2018). Among them, several psychophysiological variables such as pain (Pomares et al., 2011), cognitive fatigue (Snyder et al., 1974), or even blood glucose levels (Park et al., 2016) have been shown to evolve according to subjective rather than real time. However, no studies have addressed whether the same influence may exist for neuromuscular fatigue. The RMS/ RMS<sub>PRE</sub> data related to the kinetics of neuromuscular fatigue accumulation (Edwards, 1981) appeared relevant to test the plausibility of slowed neuromuscular fatigue accumulation. Modeling the RMS/RMS<sub>PRE</sub> data allowed us (i) to draw predictions of what could be expected if neuromuscular fatigue accumulation was slowed or unmodified, (ii) to compare these predictions with the models established on our collected data. We showed in Figure 3a that for subjects who increased their performance when the clock was slowed down, the accumulation of EMG activity over time appears to depend on subjective rather than real time. Despite some limitations (Keenan et al., 2005) and without any impairment of the neuromuscular propagation, normalized surface EMG supposedly reflects the magnitude of central motor command reaching the muscle fibers (Taylor et al., 2016), which in turn reflects the spatial/temporal recruitment of motor units during the task (Moritani et al., 1986). Because here the sarcolemma excitability assessed in Pre- Post-Test was similar between sessions (cf. M<sub>max</sub>), we are confident that any difference in RMS/RMS<sub>PRE</sub> evolution between sessions was due to the central motor command. Therefore, we can speculate that the neural activation strategy (Hunter & Enoka, 2003) depends on perceived time rather than real time. The origins of this change remain hitherto unknown. Because clock manipulation primarily affects perceptive processes, a top-down influence from specific brain regions could be

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expected. Activation of interoceptive areas (directly involved in the perception of effort) (Craig, 2009; Meyniel et al., 2013) could be hypothesized as subjective timedependent. This hypothesis echoes previous reports that high monetary reward may decrease the activation slope of interoceptive areas (Meyniel et al., 2013) and, more generally, the placebo effect, which reduces insular and thalamic responses to pain stimulation (Wager et al., 2004). The existence of structural links between perceptual and motor structures (McMorris et al., 2018) may suggest an influence of perceptual processes on the generated central motor command, which could support this subjectivedependent neural activation strategy. In the current study, we did not assess the perceived effort (e.g., through Rating of Perceived Effort (RPE) measurement), as we noticed in pilot experiments that these measurements were not compatible with our design. Indeed, it was too challenging for the participants to focus on the torque feedback to maintain a stable force as well as on the clock while simultaneously assessing their perceived effort. We acknowledge that this may constitute a limitation of the current work that could be addressed by future studies.

We also demonstrated in Figure 3b that the effort cessation for the Normal and Biased sessions appears for a very comparable RMS/RMS $_{\rm PRE}$  value (49% and 50% of maximal electromyographic activity, respectively). However, if the accumulation of neuromuscular fatigue was established according to similar kinetics to the Normal session, the threshold reached to prolong the effort to an equivalent time in the Biased session should have been widely increased (61%). In other words, the subject stops his effort for the same stopping criterion, that is, when the RMS activity reaches a critical value. Yet, this criterion is reached depending on the subjective perception of time rather than the real one.

Taken together, our findings significantly extend the literature on mind-body influence in the presence of a biased time cue (Thönes et al., 2018) to the neuromuscular fatigue field. Our results revealed that performance depends on a critical neurophysiological threshold. However, a performance improvement seems to rely on a modified neuromuscular fatigue accumulation, following perceived rather than actual time. All in all, our findings introduce new elements in the complex quest for the limits of physical performance. Indeed, if "perception of effort" (Pageaux, 2016) has already been proposed as the "cardinal exercise stopper" (Marcora, 2008; Staiano et al., 2018), the materialization of this idea through a direct influence on the accumulation of neuromuscular fatigue has never been considered. By reconsidering our understanding of neuromuscular fatigue, our results open several perspectives, encouraging, on the one hand, the exploration of other top-down cerebral factors to see if their evolution

is also dependent on subjective rather than real time; on the other hand, our results hint at the possibility to explore possible translations in clinical conditions, such as pathological fatigue (Greenhouse-Tucknott et al., 2022), in order to improve medical care.

#### **AUTHOR CONTRIBUTIONS**

**Pierre-Marie Matta:** Conceptualization; data curation; formal analysis; investigation; methodology; project administration; software; visualization; writing – original draft. **Dorian Glories:** Investigation; writing – review and editing. **Andrea Alamia:** Formal analysis; supervision; writing – review and editing. **Robin Baurès:** Conceptualization; formal analysis; methodology; supervision; writing – review and editing. **Julien Duclay:** Conceptualization; methodology; supervision; writing – review and editing.

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#### CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

#### DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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