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Evidence of altered corticomotor inhibition in older adults with a history of repetitive neurotrauma. A transcranial magnetic stimulation study

Alan J. Pearce [a,*], Dawson J. Kidgell [b], Ashlyn K. Frazer [b], Billymo Rist [a,c], Jamie Tallent [d]

[a] College of Science, Health and Engineering, La Trobe University, Melbourne 3086, Australia
[b] Faculty of Medicine Nursing and Health Science, Monash University, Melbourne 3800, Australia
[c] Australian Football League Players Association, Melbourne 32067, Australia
[d] School of Sport, Rehabilitation and Exercise Sciences, University of Essex, Essex CO4 3SQ, UK

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ABSTRACT

International concern continues regarding the association between the long-term neurophysiologic changes from repetitive neurotrauma associated with contact and collision sports. This study describes corticomotor changes in retired contact/collision sport athletes and controls, between the ages of 30 and 70 years. Retired athletes (n = 152; 49.1 ± 8.5 years) and controls (n = 72; 47.8 ± 9.5 years) were assessed using single and paired-pulse transcranial magnetic stimulation (TMS) for active motor threshold (aMT), motor evoked potential and cortical silent period duration (expressed as MEP:cSP ratio), and short- and long-interval intracortical inhibition (SICI and LICI). Motor threshold, MEP:cSP, SICI and LICI for both groups were correlated across age. Controls showed significant moderate correlations for MEP:cSP ratios at 130% (rho = 0.48, $p < 0.001$), 150% (rho = 0.49, $p < 0.001$) and 170% aMT (rho = 0.42; $p < 0.001$) and significant small negative correlation for SICI (rho = −0.27; $p = 0.030$), and moderate negative correlation for LICI (rho = −0.43; $p < 0.001$). Group-wise correlation analysis comparisons showed significant correlation differences between groups for 130% ($p = 0.016$) and 150% aMT ($p = 0.009$), specifically showing retired athletes were displaying increased corticomotor inhibition. While previous studies have focussed studies on older athletes (>50 years), this study is the first to characterize corticomotor differences between retired athletes and controls across the lifespan. These results, demonstrating pathophysiological differences in retired athletes across the lifespan, provide a foundation to utilise evoked potentials as a prodromal marker in supplementing neurological assessment for traumatic encephalopathy syndrome associated with contact/collision sport athletes that is currently lacking physiological biomarkers.

1. Introduction

Concerns regarding contact sports and the long-term neurophysiological effects of repetitive head impacts underpinning neurological function and risk of neurodegenerative disease continues. While studies have mostly focussed on the effects of concussion, repetitive subconcussive impacts are suggested to contribute to increased risks of neurological deficits [17], and increase the risk of neurological impairments and neurodegenerative disease [15,19]. While definitive evidence of neurodegenerative disease only exists post-mortem [4,21], some attempt has been made to investigate pathophysiological changes in retired contact sport athletes using transcranial magnetic stimulation (TMS) [6,14,29,30,32]. With TMS showing promise as a prodromal marker for Alzheimer's disease or frontotemporal dementia [1–3,20,24] that may contribute towards a physiological biomarker for traumatic encephalopathy syndrome or TES [11].

Research investigating the neurophysiology of healthy aging [16] and retired contact sport athletes [6,29,30,32] has utilised TMS motor evoked potentials (MEPs) and cortical silent period (cSP), as well as paired-pulse short-interval intracortical inhibition (SICI) and long-interval intracortical inhibition (LICI). Data reported by McGinley et al. [16] and Opie et al. [22] shows that increased inhibition would be expected with healthy aging as their studies showed increased inhibition in healthy older individuals, comparted to healthy younger individuals. Conversely, Pearce et al. [32] in retired older athletes (<50 years) reported reduced inhibition in older retired athletes. However, these studies have been limited to older retired players only to the exclusion of younger retired athletes aged in their 30s and 40s. Moreover, studies

* Corresponding author at: College of Science, Health and Engineering, La Trobe University, Kingsbury A venue, Bundoora, Melbourne, Victoria 3086, Australia.
E-mail address: alan.pearce@latrobe.edu.au (A.J. Pearce).

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have focussed on associating TMS variables to concussion history, rather than the wider issue of lifetime sub-concussive exposure.

Many younger contact sport athletes are medically retired prematurely in their careers due to excessive neurotrauma from physical impacts experienced in training and competition, [34]. To date, no TMS research has included younger athletes (<50 years of age), within a retired player cohort, who have retired from competitive sport. Therefore, this descriptive study presents data comparing TMS single and paired pulse measures in retired contact sport athletes across the lifespan, compared to similar age-ranged control cohort of individuals who have never participated in contact sport. Our research question was to describe the long-term effects on those who participate in contact/collision sports on corticomotor physiology compared to age-matched controls with no history of exposure to neurotrauma from contact sports.

2. Methods

2.1. Participants

Retired contact sport athletes and controls ranging between 30 and 70 years were recruited for this study. Retired athletes ($n = 152$; 2 female; 49.1 ± 8.5 years) were recruited from a number of different sports including Rugby union and league ($n = 40$), Australian football ($n = 95$), boxing ($n = 2$), motor racing (n = 2) and cycling (BMX jumping, $n = 13$). Controls with no history of playing contact sports ($n = 72$; 2 female; 47.8 ± 9.5 years) were recruited from the general public following word of mouth and recruitment advertisements. Table 1 outlines participant details.

Individuals participating in the study did not report any neurological condition, specific sleep disorders (e.g., obstructive sleep apnoea), or psychiatric disorders, nor any brain injury outside of contact sports participation (e.g., car or workplace accidents, fights or falls). Participants provided voluntary written informed consent prior to data collection. TMS protocols were approved by the University Human Research Ethics Committee (HEC18005) conforming to the guidelines set out by the Declaration of Helsinki.

2.2. Materials and methods

2.2.1. Transcranial magnetic stimulation and electromyography recording

Applying previously described TMS protocols [25–27], TMS was applied over the contralateral primary motor cortex with surface electromyography (sEMG) recording 500 ms sweeps (100 ms pre-trigger, 400 ms post-trigger; PowerLab 4/35, ADInstruments, Australia). The sEMG activity was recorded using bipolar Ag/AgCl electrodes, with an intra-electrode distance of 2 cm positioned over the first dorsal interosseous (FDI) muscle of the participant's dominant hand adhering to the Non-Invasive Assessment of Muscles (SENIAM) guidelines for sEMG [8].

TMS was delivered using a MagPro R30 stimulator (MagVentre, Denmark) using a C-B70 butterfly coil (MagVenture, Denmark). For reliability of coil placement participants wore a snugly fitted cap (EasyCap, Germany), positioned with reference to the nasion-inion and interaural lines. The cap was marked with sites at 1 cm spacing in a latitude-longitude matrix to ensure reliable coil position throughout the testing protocol [28].

Following identification of the 'optimal site', defined as the site with the largest observed MEP [12,36], active motor threshold (aMT) was determined, during a controlled, low-level voluntary contraction of the FDI muscle at 10% of Maximal Voluntary Contraction (MVC). The aMT was identified by delivering TMS stimuli (5% of stimulator output steps, and in 1% steps closer to threshold) at intensities from a level below the participant's threshold until an observable MEP of at 200 μV and associated cSP could be measured in at least five of ten stimuli [36]. Stimuli were delivered in random intervals (between 4 and 8 s) at intensities of 130%, 150% and 170% of aMT. Twenty stimuli were presented in random intervals of four sets of five pulses per set, with a break of 30 s provided between sets and intensity levels to reduce the possibility of muscular fatigue [37].

MEP amplitudes were measured from the peak-to-trough difference of the waveform. Duration of the cSP was calculated from the onset (deflection) of the MEP waveform to the return of uninterrupted EMG [37].

Paired-pulse MEPs for short latency intracortical inhibition (SICI) and long intracortical inhibition (LICI) were measured with the FDI using an interstimulus interval (ISI) of 3 ms and 100 ms respectively. SICI was undertaken with a conditioning stimulus of 80% aMT and a test stimulus of 130% aMT, while LICI was completed using a suprathreshold conditioning and test stimulus at 130% aMT; stimulation ratios and ISIs which we have previously published in post-concussion population groups [29,31]. Twenty sweeps were delivered, in four sets of five, at random intervals between 8 and 10 s between stimuli and 30 s between sets to reduce fatigue.

2.3. Data and statistical analyses

Single-pulse MEPs were expressed as a cSP:MEP ratio to reflect a balance between excitatory and inhibitory mechanisms, previously shown to reduce between-participant variability [23,35]. SICI was calculated as a ratio of the paired-pulse MEP at 3 ms to the single-pulse MEP at 130% aMT. LICI was calculated as a ratio of the suprathreshold test stimulus to the conditioning stimulus [7].

Data were analysed using Jamovi software (Version 2.3, Sydney, Australia). Age, height, and weight were compared using independent *t*-tests. As TMS data was found to be non-normally distributed, relationships between TMS variables and age were explored using Spearman's *rho*. Interpretations of correlations used the following descriptive criteria: <0.1 = *trivial*, 0.1 = *small*, 0.3 = *moderate*, 0.5 = *large*, 0.7 = *very large*, 0.9 = *nearly perfect*, 1 = *perfect* [9]. Comparison of correlations between groups was completed using the method described by Cohen et al. [5]. Data is presented as means (±SD) and alpha was set at 0.05 apart from cSP:MEP where alpha was corrected as 0.017 due to comparisons at 130%, 150% and 170% aMT.

3. Results

No differences were observed between groups for age ($t_{200} = 0.997$, $p = 0.319$), height ($t_{222} = 1.856$, $p = 0.074$),weight ($t_{222} = 1.621$, $p = 0.106$) or BMI ($t_{222} = 0.255$., $p = 0.392$; Table 1). Active motor threshold (aMT) and single-pulse cSP:MEP ratios for stimulus intensities 130% aMT, 150% aMT and 170% aMT are presented in Figs. 1 and 2 respectively. No correlations were found between age and aMT in both groups (Fig. 1a and b).

3.1. Single pulse TMS

Correlations between cSP:MEP ratios and age showed large positive significant correlations for the control group for 130% aMT (*rho* = 0.482; $p < 0.001$, Fig. 2a), 150% aMT (*rho* = 0.486; $p < 0.001$, Fig. 2c) and 170% aMT (*rho* = 0.425; $p < 0.001$, Fig. 2e). Conversely small correlations were found for the retired athlete group at each stimulus

Table 1
Participant characteristics. Mean (± SD).

	Age (years)	Height (cm)	Mass (kg)	BMI	Time since retirement (years)
Retired athletes (n = 152)	49.1 (±8.5)	183.4 (±10.5)	92.8 (±15.9)	27.7 (±2.5)	18.5 (±8.9)
Controls (n = 72)	47.8 (+9.5)	179.7 (±7.6)	89.3 (±12.2)	27.6 (+3.2)	n/a

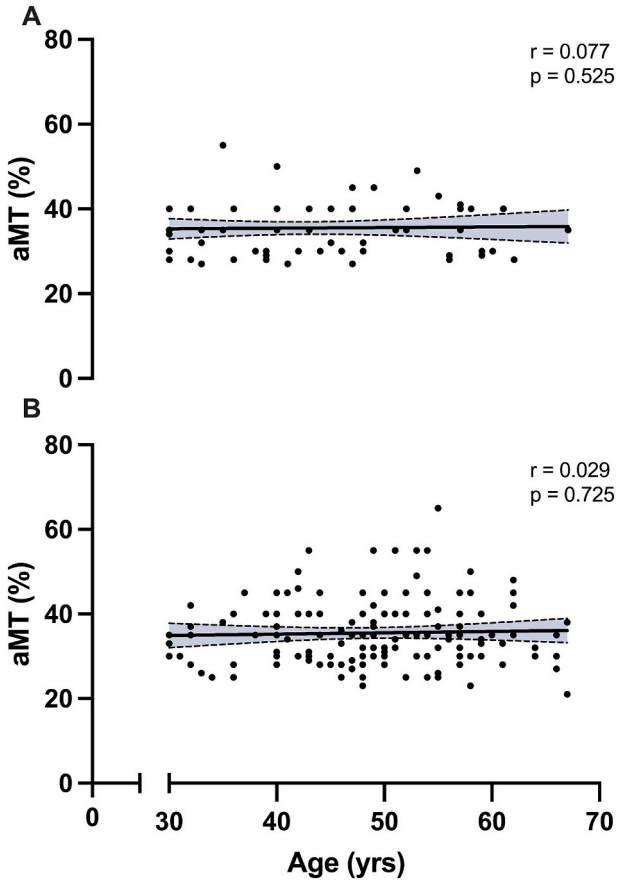

Fig. 1. Scatterplots between age and active motor threshold for control (A) and retired athletes (B).

intensity (130% aMT *rho* = 0.149; $p = 0.069$ Fig. 2b; 150% aMT *rho* = 0.178; $p = 0.058$ Fig. 2d, 170% aMT *rho* = 0.126; $p = 0.201$ Fig. 2f), suggesting that with age the ratio was driven by decreased inhibition in this group at all levels of stimulus intensity Comparisons between groups showed no significant difference in correlation for aMT ($Z = 0.328$, $p = 0.743$). Comparison between groups showed significant correlation differences in the control group compared to the retired athlete group for cSP:MEP ratios and between groups for 130% aMT ($Z = 2.547$, $p = 0.009$), 150% aMT ($Z = 2.409$, $p = 0.016$), but not 170% aMT ($Z = 1.974$, $p = 0.024$).

3.2. Paired-pulse TMS

SICI showed moderate significant negative correlations for control participants (*rho* = −0.334; $p = 0.01$, Fig. 3a) but were not observed in retired athletes (*rho* = −0.005; $p = 0.956$, Fig. 3b) suggesting decreased inhibition with age. Similarly, LICI showed moderate significant negative correlations for control participants (*rho* = −0.423; $p < 0.001$, Fig. 3c) but not in retired athletes (*rho* = 0.107, $p = 0.243$, Fig. 3d) also suggesting decreased inhibition with age.Comparison between groups showed significant correlations in the control group compared to the retired athlete group for SICI (Z = -1.965, $p = 0.049$), and LICI (Z = -3.734, $p < 0.001$),

4. Discussion

Previous TMS studies have shown inhibitory changes only in older retired contact-sport athletes (<50 years) with a history of repetitive neurotrauma from concussion and sub-concussion impacts [6,29,30,32]. However, TMS studies to date have not included data of retired younger athletes (<50 years) within the overall cohort. The aim of this descriptive study was to compare TMS corticomotor and intracortical measures across the lifespan (30–70 years) in retired sports athletes (minimum five years) with a history of repetitive neurotrauma to controls with no history of playing contact sports. Supporting previous TMS work in healthy aging [16,22], we observed increased MEP:cSP ratios indicating greater intracortical inhibition measures in older individuals. This was not found in the retired athlete cohort, suggesting that across the lifespan retired contact sport athletes with progressing age illustrated less inhibition.

The findings in this study provide further evidence of alterations in GABAergic receptor activity resulting from a history of repetitive neurotrauma. By what mechanisms repetitive trauma affects GABAergic activity we can only speculate. Following acute physical trauma, it has been shown that physical trauma alter depolarizing actions of GABA contributing to maladaptive signal transmission [33]. It is plausible that repetitive physical neurotrauma experienced in contact sports over many years may contribute to maladaptive long-term potentiation changes as shown with single and paired pulse TMS. While further research is required to explore this in cohorts such as contact sport athletes, the data in this study illustrates that retired athletes showing altered inhibition measures suggests a potential prodromal temporal biomarker as part of overall clinical criteria for traumatic encephalopathy syndrome (TES) [11]. Indeed, TMS evoked potentials have demonstrated the efficacy of prodromal temporal biomarkers in identifying pathophysiology in Alzheimer's and frontotemporal dementia versus aged-matched controls [3,20,24].

Despite this, we acknowledge that intracortical inhibition changes in older individuals are not uniform across studies in both healthy and those with a history of brain injury. As suggested by McGinley et al. [16] these differences may be attributed to the target muscle and TMS stimulus protocols. In single-pulse TMS, another reason for disparities seen in inhibition changes across previous studies may also be attributable to cSP duration being measured in isolation and not part of the overall excitation/inhibition balance. Despite being well-known that the intensity of TMS is an influencing confounding factor on MEP and cSP duration [10,37], whereby increased cSP duration is observed at higher intensities of stimulation until a plateau in duration occurs at high intensities of stimulus [13]. Orth and Rothwell [23], and Škarabot et al. [35] have argued that the relationship between stimulus intensity and cSP duration may also be due to the influence of the preceding MEP, which also increases with stimulus intensity until plateau, affecting cSP duration [23]. Consequently, the present study sought to present data as cSP:MEP ratio to reflect a balance between excitatory and inhibitory mechanisms. Further to mitigate any influence of differences in motor threshold on changes in cSP duration [13] our study completed a modified stimulus-response curve which also allowed for the comparison of associated MEP amplitude/cSP duration with TMS intensity [13]. Our data showed that with increasing TMS intensities MEP amplitude and cSP duration increased in the control group (Fig. 2), but this was not the case in the retired athlete cohort that showed with increasing MEP amplitudes there was not a concurrent lengthening in the cSP suggesting increased net excitability through decreased inhibitory influences [23,35]. We found similar results in both SICI and LICI ratios (Fig. 3) suggesting a general change in GABAergic intracortical inhibition.

4.1. Study strengths

This is the first study to describe corticomotor changes over the lifespan (30–70 years), rather than in older individual cohorts, in those with a history of repetitive neurotrauma from sub-concussive exposure in contact/collision sport. While previous studies have provided evidence of altered corticomotor and corticocortical evoked potentials associated with a history of concussions [6,14,29,30,32], this study has specifically aimed to present corticomotor data reflecting long-term outcomes in athlete cohorts experiencing repetitive sub-concussive

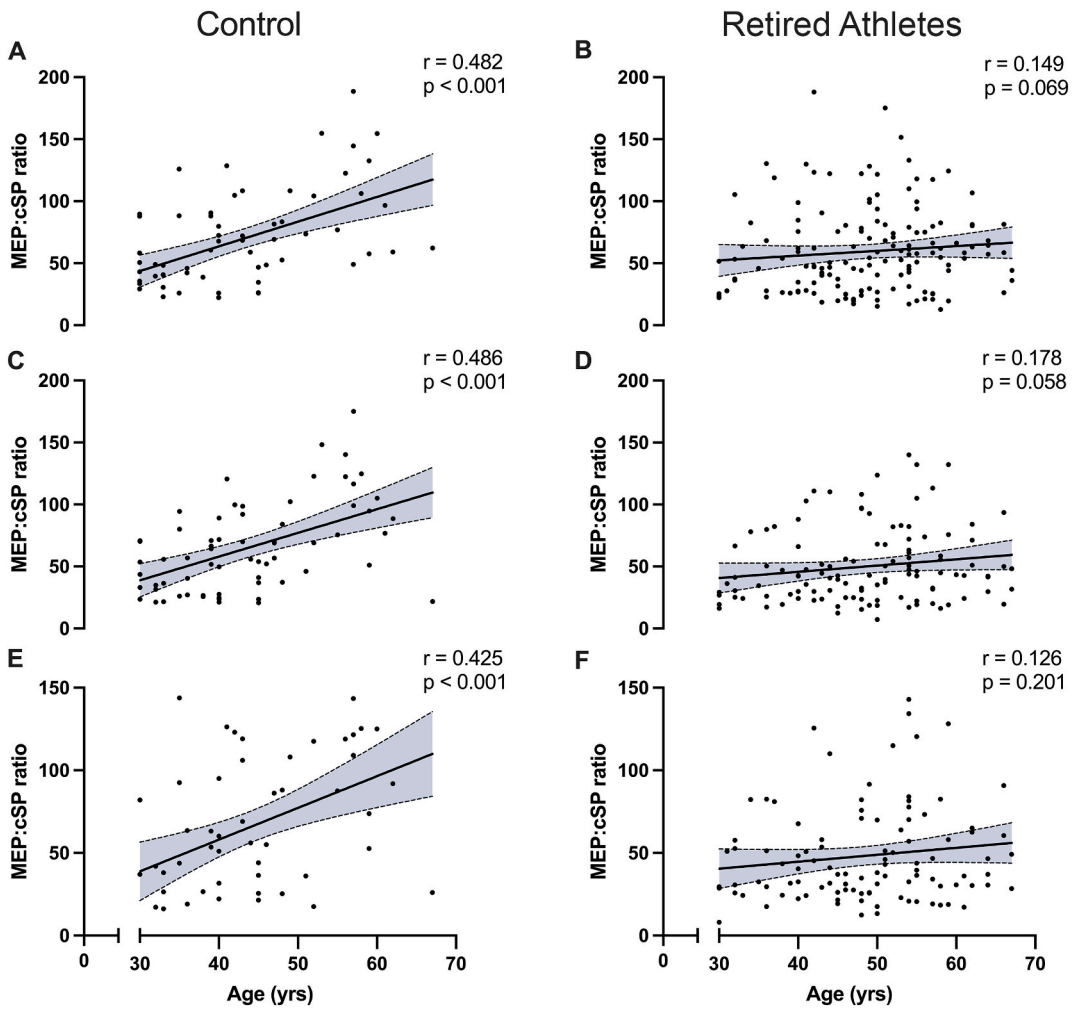

Fig. 2. Scatterplots between age and MEP:cSP ratios at 130%, 150% and 170% aMT for controls (A, C, and E respectively) and retired athletes (B, D, and F respectively).

impacts. Moreover, this study has included younger cohorts of retired athletes which have not yet been presented in studies.

4.2. Study limitations

Our descriptive study is limited by several factors. Firstly, single-pulse TMS is a measure of corticomotor physiology and while it has been argued that cSP represents intracortical networks, we caution on generalizing these findings to more global measures of intracortical inhibition. Secondly, SICI and LICI measures were limited to only one ISI (3 ms and 100 ms respectively), and we did not collect intracortical facilitation (ICF). The inclusion of greater ISI has been used to detect intracortical synaptic and cholinergic circuits impairments that have predicted cognitive decline in various dementias [3,20,24] as well as in previous studies in retired contact sport athletes [6] and based on the data in this descriptive study, we aim to include expanded paired-pulse ISIs in future work.

Finally, we also acknowledge the retrospective design of this study. Future research should aim to conduct prospective data in recently retired athletes as they age post retirement. However, it is important that current cohorts are not ignored. Retrospective and cross-sectional research designs are still important in lieu of prospective randomised controlled studies [18] and provide a strong foundation for future work.

5. Conclusion

In conclusion, this study has reported on single and paired pulse evoked potentials in retired athletes across the lifespan. Compared to age-matched controls, we found pathophysiological changes, specifically reduced inhibition, associated with aging of athletes. While further studies are required to determine the mechanisms of these differences, our findings concur with studies in dementia [20,24]. TMS evoked potentials provide a potential biomarker supplementing clinical assessment and neuropsychological batteries which currently lacks physiological biomarkers in the assessment for TES [11].

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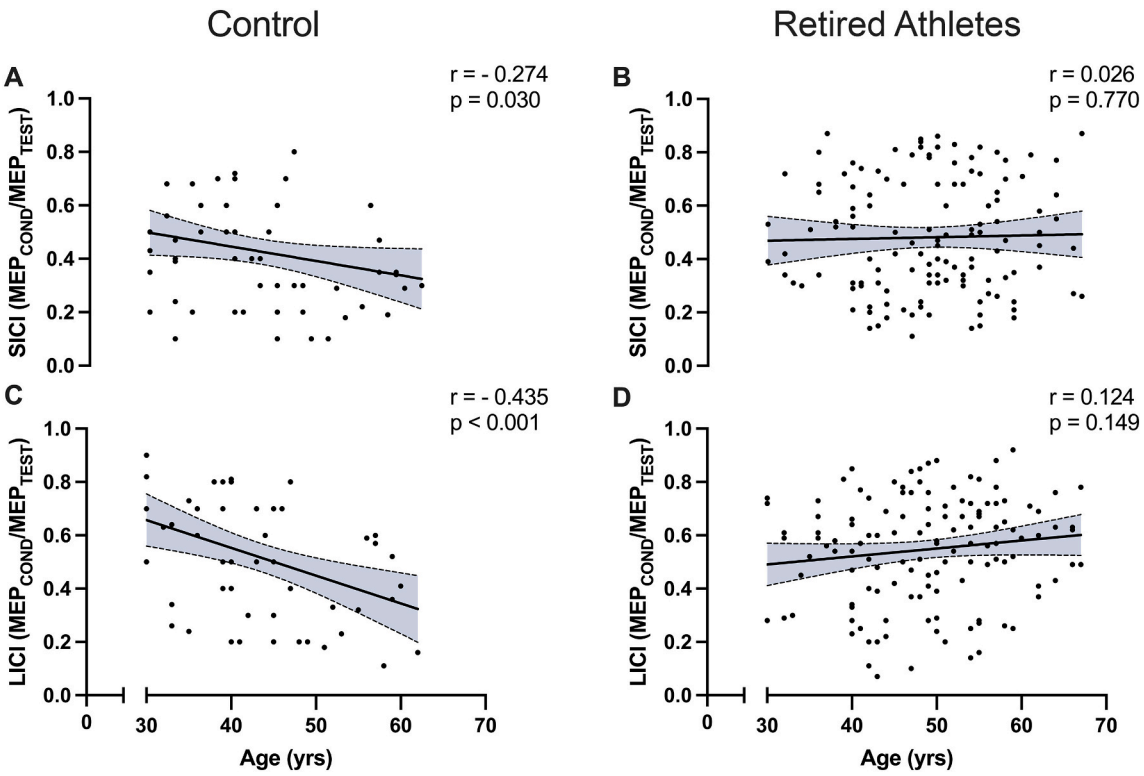

Fig. 3. Correlations across age with SICI 3 ms (A and B) and LICI 100 ms (C and D).

Declaration of Competing Interest

None.

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