New Zealand rugby health study: motor cortex excitability in retired elite and community level rugby players

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ABSTRACT

AIMS: Rugby union is a high contact sport in which players frequently experience brain injuries. Acute brain injury is associated with altered corticomotor function. However, it is uncertain if long-term exposure to rugby is associated with any alterations in corticomotor function. The aim of the study was to assess measures of corticomotor excitability and inhibition in retired rugby players in comparison to retired non-contact sport players.

METHODS: The design was a cross-sectional study with three groups of retired athletes: elite rugby (n=23), community level rugby (n=28) and non-contact sport control (n=22). Assessments of corticomotor excitability were made using transcranial magnetic stimulation.

RESULTS: Resting motor threshold was significantly higher and long-interval intracortical inhibition was greater in the elite rugby group compared to the control group. Participants in the two rugby groups had sustained significantly more concussions than the control group.

CONCLUSIONS: We provide some evidence of altered corticomotor excitation and inhibition in retired elite rugby players in comparison to retired non-contact sport players. Given the absence of findings in the community rugby group, who had experienced a similar number of concussions, the association with previous brain injury is unclear.

The long-term effects of head injuries in professional athletes have become a high-profile topic as evidence accumulates of the potential negative effects of repeated concussion on brain function. In contact sports such as rugby and American football, where high velocity impacts are a common feature of the game, concussions are frequently encountered. The incidence rate in adult rugby players has been reported at 3–23% in a single season,[1] while a recent meta-analysis determined a concussion incidence of 4.73 per 1,000 player match hours, or approximately one in every five games.[2]

Numerous studies using transcranial magnetic stimulation (TMS) have shown that corticomotor excitability, or the excitability of the pathway from the primary motor cortex to the motoneurons, is altered in the acute phase following concussion.[3–8] Consistent findings are an elevated motor threshold and a prolongation of the cortical silent period. The cortical silent period in particular, reflects an enhancement of gamma-aminobutyric acid B ($GABA_B$) receptor mediated intracortical inhibition. Enhanced $GABA_B$-inhibition has been shown to be associated with impaired motor performance,[7,9,10] suggesting that this could be a clinically relevant finding. The outcomes are less consistent in studies that have examined the long-term (>12 months) effects of concussion on corticomotor excitability. While some show a continued prolongation of the silent period,[10,11] others have shown no differences[12] or a relative shortening of silent period duration.[13] Similarly, long-interval intracortical inhibition

(LICI), a further measure reflecting $GABA_B$ receptor activation, has been reported to be reduced,[13] enhanced[9,14,15] or unchanged[12] in the long term, following concussion. Thus, there are inconsistencies in the studies to date on the potential long-term effects of concussion on corticomotor function.

The cumulative effect of multiple concussions may be more influential than the effect of a single concussion. The term "chronic traumatic encephalopathy" has been used to describe the progressive neurodegenerative effects of concussions, and it is particularly common in athletes who have experienced multiple blows to the head.[16] De Beaumont and colleagues[11] provide some evidence that athletes with multiple concussions have exaggerated alterations in corticomotor excitability compared to those with a single concussion.

The goal of the current study was to examine corticomotor excitability in athletes retired from competitive rugby union in comparison to athletes retired from non-contact sport. Given the potential differences in playing time, player size and skill, and data from previous studies showing differences in concussion rates among different levels of competition, separate groups of participants were recruited from retired elite (national and international) and community (club) level players.

Methods

Participants

Participants were 73 males who had previously played competitive rugby, hockey or cricket in New Zealand. They were a cohort from a larger study investigating the health of retired rugby and non-contact sport athletes who volunteered to participate in an additional session assessing corticomotor excitability. Participants were separated into three groups: elite rugby (n=23), community rugby (n=28) and a non-contact sport control (hockey and cricket; n=22). Elite rugby players were required to have played at international or national level, while the community level group played at club level. Non-contact sport players were club, national and international level. All participants were required to be male, aged 30–65 years and to be retired from competitive sport for at least five years. They were excluded if they had any contraindications to or did not want to undertake TMS (*n*=4), or were taking medication known to influence corticomotor excitability (*n*=1).[17] Ethical approval was obtained from the Auckland University of Technology Ethics Committee and informed consent was obtained prior to participation.

Protocol

Participants were asked to complete an online questionnaire assessing general physical and mental health, including concussion history. A detailed description of the assessment and the relevant findings is provided elsewhere.[18] Participants then completed an additional test session using TMS to assess intracortical and corticomotor excitability.

Motor cortex excitability assessment

Procedure

Participants were seated in a comfortable chair with their self-reported dominant arm resting on a pillow positioned over their lap. TMS was applied to the contralateral brain hemisphere. Magnetic stimuli were applied using a Bistim 200[2] (Magstim Co) and a 70mm figure-of-eight coil. The coil was held perpendicular to the scalp with the handle directed posteriorly at approximately 45^0 to induce a posterior-anterior current in the motor cortex. The "hot spot" for the participant's dominant first dorsal interosseus (FDI) muscle was found by moving the coil around the scalp and locating the site that elicited the largest motor evoked potentials (MEPs) in the FDI. This site was marked with a felt pen and all further stimuli were delivered with the coil over this location. The resting motor threshold (RMT) was established as the lowest intensity that elicited at least four MEPs>50 μV in a train of eight consecutive stimuli.[19]

Electromyography

The FDI muscle was selected as the target muscle, as it has almost exclusively been used in other studies assessing the impact of concussion on corticomotor excitability in sporting populations, and thus would enable comparison with previous work in the field.[10,11,13,14,20] Self-adhesive bipolar surface

electrodes (Norotrode, USA) were applied over the belly of the FDI of both hands. The skin was shaved and cleansed with alcohol prior to application of electrodes. A ground electrode was applied over the dominant styloid process or 5th carpometacarpal joint. Electromyography (EMG) signals were amplified (AMT-8, Bortec Biomedical, Canada), filtered (10–1000Hz) and sampled at 5,000Hz using a data acquisition board (Micro 1401, CED, UK) and Signal software (CED, UK).

Corticomotor excitability

Corticomotor excitability was assessed using stimulus-response curves. To obtain the curves, participants were given magnetic stimuli at eight intensities from 90–160% RMT in 10% steps. Ten stimuli were given at each intensity with the FDI muscle relaxed. Eighty stimuli were delivered 5–7 s apart with the order of intensity randomised.

Intracortical inhibition and facilitation

Measures of intracortical excitability reflect excitability of cortical interneurons, providing a specific measure of neural excitability within the brain itself. To examine short-interval intracortical inhibition (SICI), conditioning and test stimuli were delivered with an interstimulus interval of 2ms. The test stimulus intensity was set to elicit a MEP of 1mV amplitude (TS_{1mV}). Two intensities of conditioning stimulus were provided at 70% and 80% RMT.

To examine short-interval intracortical facilitation (SICF), a conditioning stimulus was delivered at TS_{1mV} followed by a test stimulus at 90% RMT. Two interstimulus intervals of 1.4 and 2.8ms were used.

To examine long-interval intracortical inhibition (LICI), a conditioning stimulus was delivered at 120% RMT followed by a test stimulus at TS_{1mV}. The interstimulus interval was 99ms.

Ten stimuli were given for each condition described above with the FDI muscle completely relaxed. An additional 10 stimuli were provided at TS_{1mV}. The 60 stimuli were delivered 5–7 s apart with the order of condition randomised.

Silent period

The silent period induced by single-pulse TMS was examined by delivering 10 stimuli at 120% RMT while the dominant FDI was activated at 10% of maximum isometric voluntary contraction (MVC). To determine MVC, participants performed an isometric abduction of the target index finger against a loadcell (Precision Transducers Ltd, New Zealand) for 5–10 s, and the maximum level of force identified. To generate a contraction of 10% MVC, participants were provided with visual feedback of the target force and their actual force.

Transcallosal inhibition

Transcallosal inhibition is a measure of efficacy of connections between the two primary motor cortices. It was assessed by recording responses in the non-dominant FDI while stimulating over the hot spot for the dominant FDI. Ten stimuli were delivered at 80% of maximum stimulator output while the non-dominant FDI was activated at 50% MVC. The MVC of the non-dominant FDI was determined in similar manner to that described for the dominant FDI. During stimulation, participants were provided with visual feedback of the target force and their actual force.

Data processing

Stimulus-response curve and intracortical facilitation and inhibition data were visually inspected, and any responses containing unwanted muscle activation were removed prior to further analysis. The maximum peak-to-peak amplitude of each MEP was measured and then averaged across each intensity level or condition. For the stimulus-response curve data, a Boltzman equation was fitted for each participant and the overall group data using the following formula:[21]

$$\text{MEP amplitude (S)} = MEP_{max} / (1 + \exp((S_{50} - S)/m))$$

where MEP_{max} represents the maximum MEP amplitude or plateau, S is stimulus intensity, S_{50} is the stimulus intensity at which the MEP amplitude is 50% of MEP_{max} and m is a slope parameter. The maximal steepness of the function at S_{50} is directly proportional to the inverse of the slope parameter (ie, 1/m). In addition, the variability of MEP amplitude at 120% RMT (MEP_{CV}) was determined by expressing the standard deviation of response amplitude relative to the mean MEP amplitude. This provides a measure of the fluctuations in corticomotor excitability.

Responses obtained during the assessment of the silent period and transcallosal inhibition were rectified and averaged. The mean background EMG activity 80ms pre-stimulus was determined in the averaged response. The duration of the silent period was defined as the time from stimulation to the point at which the rectified EMG activity returned to the background mean level following the MEP (Figure 1). The onset and offset of transcallosal inhibition were defined as the time points at which rectified EMG activity dropped below and returned to the background mean level, respectively (Figure 1). The duration of transcallosal inhibition was the difference between these two time points.

General health assessment

The full online questionnaire took approximately 40 minutes to complete. Data were collected on participant demographics, injuries and illnesses, current physical and mental health, sleep quality, drug and alcohol use, and beliefs regarding sport and health. Participants were asked if they had ever experienced a concussion while playing or training for their sport. The following definition of a concussion was provided:

"A concussion is a blow to the head followed by a variety of symptoms that may include any of the following: headache, dizziness, loss of balance, blurred vision, 'seeing stars', feeling in a fog or slowed down, memory problems, poor concentration, nausea or throwing-up. Getting 'knocked out' or being unconscious does NOT always occur with a concussion."

The severity of concussions was assessed using the Rivermead Post Concussion Symptoms Questionnaire (RPQ).[22] Scores from the questionnaire were separated into two components reflecting predominantly early (RPQ-3) and late (RPQ-13) symptoms of brain injury.[23]

To evaluate current levels of physical activity, participants were also asked the number of days in the previous week that they undertook exercise.

Statistical analysis

Using the Kolgomorov Smirnov test, it was determined that all outcome variables except for the silent period duration, transcallosal inhibition duration and two of the Boltzmann equation coefficients were not normally distributed in at least one of the three groups. Therefore, corticomotor and intracortical excitability variables (RMT, MEP_{CV}, SICI, SICF, LICI, silent period duration, transcallosal inhibition duration, Boltzmann equation coefficients) and concussion data were compared among groups using Kruskal-Wallis tests. Significant findings were investigated using the Mann-Whitney *U* test, with the alpha level adjusted using a Bonferroni correction. Categorical data were compared among groups using Chi-Square tests. Effect size data were also determined for comparisons between the two rugby groups and the control group. Effects sizes for continuous and numerical data are expressed as Cohen's *d*. Effect sizes for categorical data were converted from the odds ratio.[24] Statistical analyses were undertaken using SPSS V23 (IBM, USA) and Comprehensive Meta-Analysis (Biostat, USA). An alpha level of 0.05 was adopted.

Results

Demographic and concussion information for the groups is shown in Table 1. Concussion information was missing from one elite rugby and one community rugby participant. The groups were matched for age, the numbers of years active in sport and current frequency of exercise, but there were significant differences in height, weight and body mass index (BMI). Participants in the elite rugby group were taller and heavier than the other two groups (all $P<0.001$; adjusted alpha = 0.012). Community rugby players were also heavier than the controls ($P=0.004$). Overall, participants in the two rugby groups had a significantly higher BMI than the control participants (both $P\leq0.002$), but the difference between the rugby groups was not significant ($P=0.09$).

There were also differences among groups in concussion history. All rugby players except one had experienced at least one concussion during their playing career, while only six of the control group had experienced concussions. Therefore, there were significantly more control participants who had not sustained a concussion compared to the two rugby groups (both $P<0.001$), and significantly less who had sustained three or more concussions (both $P<0.001$).

Table 1: Demographic, general health information for the three groups. Data are mean (standard deviation) or effect size (95% confidence interval).

	Elite Rugby (*n*=23)	Community Rugby (*n*=28)	Control (*n*=22)	*P*-value	Effect size ER v Control	Effect size CR v Control
Age (years)	43 (7)	45 (8)	44 (9)	0.77	0.18 (-0.40 to 0.77)	0.03 (-0.53 to 0.59)
Height (cm)	188 (7)	178 (6)	178 (5)	<0.001*[a,b]	1.64 (0.95 to 2.33)	0.05 (-0.61 to 0.7)
Weight (kg)	107 (17)	88 (9)	81 (10)	<0.001*[a,b,c]	1.83 (1.12 to 2.55)	0.76 (0.09 to 1.44)
BMI (kg/m^2)	31 (5)	29 (4)	26 (3)	<0.001*[a,c]	1.35 (0.70 to 2.00)	0.88 (0.26 to 1.49)
Years of sport	23 (8)	23 (7)	24 (9)	0.92	0.08 (-0.53 to 0.70)	0.04 (-0.52 to 0.6)
Exercise days	2.9 (1.0)	2.8 (1.2)	2.8 (0.9)	0.38		
Reported concussions						
0 concussions (*n*)	0 (0%)	1 (4%)	16 (75%)	<0.001*[a,c]	NA	2.34 (1.12 to 3.55)
1–2 concussions (*n*)	3 (13%)	3 (11%)	5 (21%)	0.50	0.37 (-0.49 to 1.24)	0.47 (0.39 to 1.33)
≥3 concussions (*n*)	20 (87%)	23 (85%)	1 (4%)	<0.001*[a,c]	2.72 (1.43 to 4.02)	2.64 (1.39 to 3.89)
RPQ-3	3.5 (2.1)	4.5 (2.9)	2.5 (1.0)	0.21	0.59 (-0.33 to 1.51)	0.93 (0.03 to 1.83)
RPQ-13	11.8 (8.4)	11.7 (10.9)	8.3 (11.8)	0.41	0.34 (-0.57 to 1.25)	0.30 (-0.60 to 1.19)

ER = elite rugby; CR = community rugby; BMI = body mass index; RPQ = Rivermead Post Concussion Questionnaire; * = significant difference among groups; [a] = significant difference between control and elite rugby groups; [b] = significant difference between community and elite rugby groups; [c] = significant difference between control and community rugby groups.

Motor cortex excitability

Example MEPs from single- and paired-pulse stimulation and group stimulus response curves are shown in Figures 1 and 2, respectively. The mean MEP amplitude obtained during stimulation at TS_{1mV} was 0.97±0.95mV, 1.02±1.10mV and 1.02±0.63mV for the elite rugby, community rugby and control groups, respectively. This indicates adequate matching of test MEP amplitude for the assessment of paired-pulse data.

Summary information for all of the dependent variables is provided in Table 2. There was a significant difference in RMT among the groups. RMT in the elite rugby group was significantly higher compared to the control (*P*=0.004) group. There was also a difference in LICI among the groups, with the elite rugby group showing significantly more LICI (reduced MEP size) in comparison to the control group (*P*=0.005). There were no other significant differences among groups in the other motor cortex excitability or inhibition outcome measures.

Discussion

We found some evidence for altered corticomotor excitability and intracortical inhibition in retired elite rugby players in comparison to retired non-contact sport players. Resting motor threshold was elevated in the elite rugby group, reflecting reduced excitability, and LICI was increased, reflecting enhanced inhibition. Both of these findings follow some of the previous research on the impact of concussion on measures of motor cortex excitability. However, although both rugby groups had experienced a greater number of concussions compared to the control group, there were no similar differences in RMT and LICI in the community rugby group. Therefore, the association with concussion history is unclear.

Motor threshold

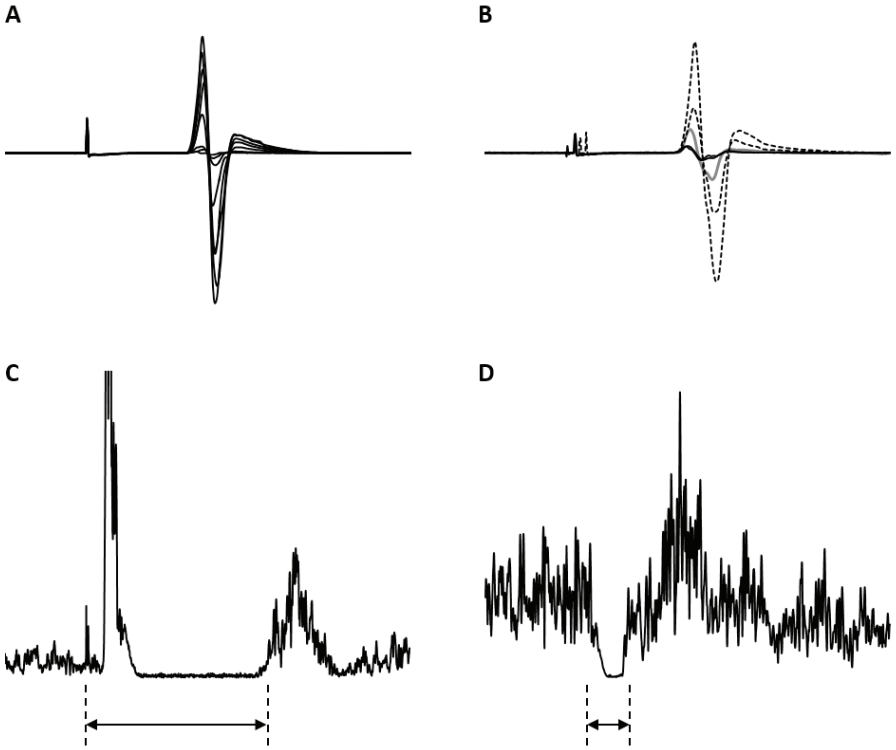
The elite rugby group had a higher resting motor threshold compared to the control group. An elevated motor threshold normally

Table 2: Group results of corticomotor and intracortical excitability data. Data are mean (standard deviation) or effect size (95% confidence interval).

	Elite Rugby (*n*=23)	**Community Rugby (*n*=28)**	**Control (*n*=22)**	***P*-value**	**Effect size ER v Control**	**Effect size CR v Control**
RMT (% MSO)	50 (7)	46 (10)	44 (8)	0.01*[a]	0.89 (0.27 to 1.50)	0.21 (-0.35 to 0.77)
MEP variability (%)	71 (24)	80 (54)	61 (22)	0.43	0.41 (-0.18 to 1.00)	0.46 (-0.11 to 1.02)
Boltzmann equation coefficients						
MEP_{max} (mV)	4.3 (2.0)	4.9 (3.3)	5.8 (3.5)	0.57	0.47 (-0.12 to 1.07)	0.21 (-0.35 to 0.77)
slope	8.4 (3.7)	8.0 (3.5)	7.3 (4.0)	0.52	0.23 (-0.36 to 0.81)	0.12 (-0.43 to 0.68)
S_{50} (%RTh)	126 (10)	126 (12)	126 (14)	0.77	0.02 (-0.56 to 0.61)	0.02 (-0.54 to 0.58)
Paired-pulse stimuli						
$SICI_{70}$ (mV)	0.33 (0.40)	0.37 (0.33)	0.44 (0.39)	0.20	0.27 (-0.31 to 0.86)	0.19 (-0.37 to 0.29)
$SICI_{80}$ (mV)	0.45 (0.67	0.51 (0.50)	0.44 (0.37)	0.27	0.02 (-0.56 to 0.61)	0.17 (-0.39 to 0.73)
$SICF_{1.4}$ (mV)	2.27 (1.43)	2.49 (1.79)	2.41 (1.42)	0.86	0.10 (-0.48 to 0.69)	0.05 (-0.51 to 0.60)
$SICF_{2.8}$ (mV)	1.51 (0.84)	1.73 (1.61)	2.07 (1.52)	0.39	0.46 (-0.14 to 1.05)	0.22 (-0.34 to 0.78)
LICI (mV)	0.22 (0.53)	0.31 (0.44)	0.50 (0.86)	0.03*[a]	0.40 (-0.2 to 0.99)	0.28 (-0.28 to 0.84)
Silent period (ms)	157 (33)	151 (30)	153 (28)	0.63	0.12 (-0.47 to 0.70)	0.07 (-0.49 to 0.62)
Transcallosal inhibition (ms)	41 (11)	48 (16)	39 (18)	0.13	0.13 (-0.46 to 0.72)	0.48 (-0.09 to 1.06)

ER = elite rugby; CR = community rugby; RMT = resting motor threshold; MSO = maximum stimulator output; MEP = motor evoked potential; S_{50} = stimulus intensity at which MEP amplitude is 50% MEP_{max}; SICI = short-interval intracortical inhibition; SICF = short-interval intracortical facilitation; LICI = long-interval intracortical inhibition. * = significant difference among groups; [a] = significant difference between elite rugby and control groups.

Figure 1: Example motor evoked potentials (MEPs) from individual participants. Each response shown is an average of 10 stimuli.



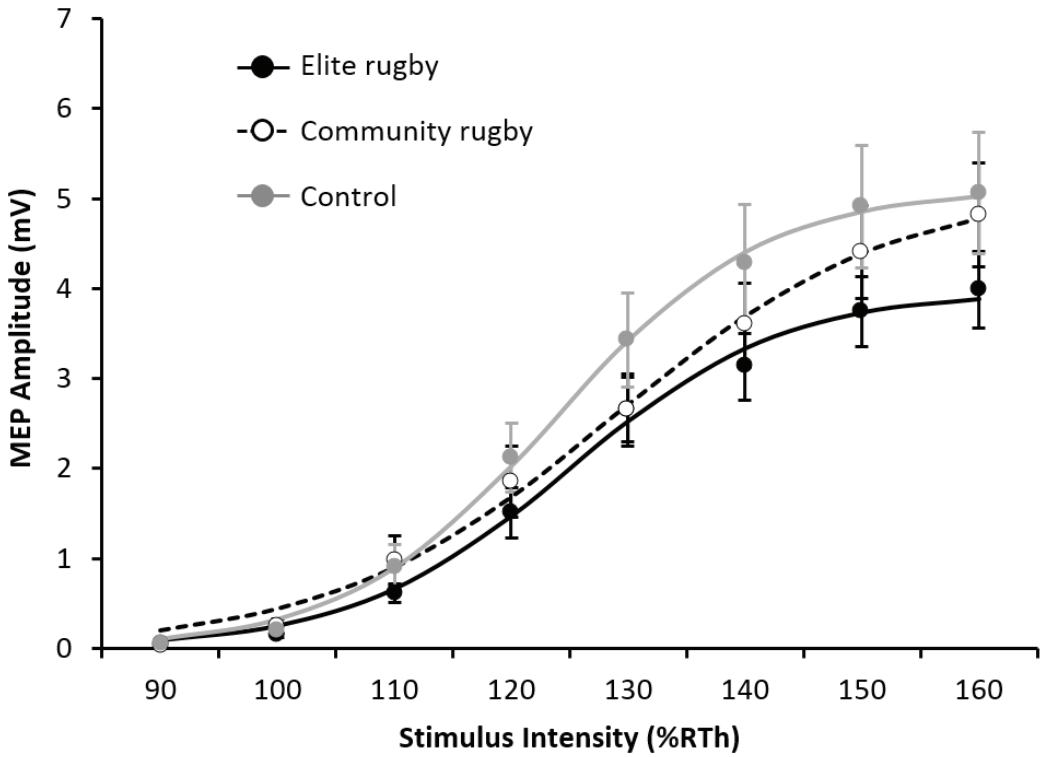
A. Overlaid stimulus-response curve MEPs from an elite rugby participant. Responses are from stimulus intensities from 90–160% of resting threshold.

B. Overlaid paired-pulse responses in a control participant. The non-conditioned (single-pulse) response is shown in grey. The black traces show the two short-interval intracortical inhibition conditions. The dotted traces show the two short-interval intracortical facilitation conditions.

C. Silent period in a community rugby player. The silent period duration is indicated by the arrow.

D. Transcallosal inhibition in a community rugby player. The duration of transcallosal inhibition is indicated by the arrow.

Figure 2: Stimulus-response curves showing mean motor evoked potential (MEP) amplitude for the three groups. The data have been fitted with Boltzmann equation curves. RMT = resting motor threshold. Bars are 1 standard error of the mean.



reflects impaired excitability threshold of cortical motor neurons and is one of the most common alterations evident in studies that have used TMS in people with acute concussion (<1 month).[3–5] Studies involving people with a longer post-concussion period have produced less consistent findings. While motor threshold was increased in one study[25] investigating the effects of mild brain injury sustained an average of five years previously, two similar studies found no significant differences.[10,26] The latter two studies involved people who were nine-months to two-years post-brain injury.

While the elite rugby group had an elevated RMT in relation to the control group, it is difficult to make an association with concussion history given the similar number and severity of concussions experienced in the community rugby group, whose RMT did not differ from the controls. A potential confounding factor is the larger size of the elite rugby group, both in terms of weight and BMI. The elite rugby group were on average 25kg heavier than the control group. Stokes and colleagues[27] identified that a 1mm increase in the distance from the TMS coil to the motor cortex was associated with a 2.8% increase in motor threshold. Therefore, the potential for more adipose tissue and increased cranium thickness[28] in the larger elite rugby players may contribute to the increased resting motor threshold.

Corticomotor and intracortical excitability

The finding that corticomotor excitability, assessed using the stimulus-response curves, was equivalent to controls in the two rugby groups indicates that the excitability of the pathway from the corticospinal tract to the FDI muscle was not abnormal. Therefore, the strength of synaptic connections in the output pathway from the primary motor cortex was the same in the retired rugby players as the control group.

The intracortical excitability measurement of LICI was increased in the elite rugby group in comparison to the controls. Two previous studies have reported that athletes with multiple previous concussions (>12 months ago) had greater LICI than comparable athletes without concussion history.[9,15] LICI is mediated by $GABA_B$ receptors in the primary motor cortex.[29] Based on evidence

from drugs that block GABA re-uptake[30] or are GABA-receptor agonists,[31] it has been proposed that the inhibition arises through the generation of slow inhibitory post-synaptic potentials following activation of the $GABA_B$ receptor. Inhibitory circuits involving GABA receptors are implicated in the modulation of neural plasticity,[32] and increased inhibitory activity constrains the potential for neural plasticity and may consequently impair cognitive and motor skill learning tasks that are dependent on such plasticity.[9,33] An increased silent period duration, which also reflects over-activation of neural pathways that involve $GABA_B$ receptor,[29] also has been consistently reported in people with chronic brain injury or concussion.[9–11,14,15] However, a recent study that examined cortical excitatory and inhibitory function in American football players who had experienced a concussion (>10 months previously) reported no significant differences in LICI, silent period duration or any brain metabolites between concussed and non-concussed athletes.[12] We did not see any differences in the silent period duration among our groups. Other studies have previously indicated that LICI and the silent period duration are likely to reflect different aspects of $GABA_B$ receptor-mediated inhibition,[34,35] which may be explained in part by the spinal level contribution to the early part of the silent period.[36] Therefore, we provide partial evidence of dysfunctional $GABA_B$ receptor-mediated inhibition in retired elite rugby players compared to retired non-contact sport players, in that LICI was reduced in the elite rugby group but the silent period duration was not altered.

Measures of SICI and SICF were comparable among the groups. These two measures reflect $GABA_A$ receptor-mediated inhibition and excitatory descending volley interactions, respectively. As these measures were not clearly different among the three groups in our study, we find no evidence of altered function of these processes in retired rugby players.

Transcallosal inhibition

Transcallosal pathways are critical for inhibiting activation of muscles in the opposite side of the body during unilateral motor tasks, particularly fine motor skills.[37] For example, transcallosal inhibition is reduced in people with mirror movements who are unable to move the hands independently.[38] Conversely, an increase in transcallosal inhibition has been associated with higher performance on tests of unimanual dexterity.[39] There was no difference in transcallosal inhibition among groups in the current study, indicating that interhemispheric motor pathways in the two rugby groups were comparable to controls.

Limitations

There were several limitations to the study. Firstly, the recall of concussion history may not be accurate, particularly the symptoms assessed by the RPQ. We also did not assess the time since the last concussion, or assess concussions that were not related to sport. There is a large age range in the participant groups, which may contribute to the variability of the data and a reduction in study power. For example, there is evidence that some measures of corticomotor[40,41] and intracortical[42] excitability are altered in older people. Current levels of physical activity may be a confounding factor in the study, as it has been shown that exercise can influence the strength of GABA-mediated circuits.[43] However, there were no differences in the frequency of exercise reported in the previous week among the groups, so this is unlikely to have a large impact on the study findings.

Conclusion

We provide some evidence of altered cortical motor excitation and $GABA_B$ mediated-inhibition in retired elite rugby players in comparison to athletes retired from non-contact sport, although differences in body size could be a confounding factor in the measure of resting motor threshold. These findings were not present in retired community-level rugby players. Given that the community rugby group had experienced an equivalent number of concussions and severity of symptoms as the elite rugby group, it is difficult to form an association between altered corticomotor excitability and concussion history. However, these alterations should be investigated further given the large number of athletes who participate in rugby at the elite level.

Competing interests:
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