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Elite swimmers possess shorter telomeres than recreationally active controls

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- 1 **Elite swimmers possess shorter telomeres than recreationally active controls**
- 2 Nickels M, Mastana S, Denniff M, Codd V, Akam E

ABSTRACT

Purpose: Elite athletes are reported to possess longer telomeres than their less active counterparts. ACE gene (Insertion/Deletion) polymorphism has been previously associated with elite athletic performance, with the deletion (D) variant appearing more frequently in short distance swimmers. Additionally, the D allele has been reported to have a negative effect on telomere length. The aim of this study was to investigate the telomere length of elite swimmers and its potential association with ACE genotype. **Methods:** Telomere length was measured by real-time quantitative PCR and ACE I/D genotypes analysed by standard PCR and electrophoresis in 51 young elite swimmers and 56 controls. **Results:** Elite swimmers displayed shorter telomeres than controls (1.043 ± 0.127 vs 1.128 ± 0.177 , $p = 0.006$). When split by sex, only elite female swimmers showed significantly shorter telomeres than their recreationally active counterparts ($p = 0.019$). ACE genotype distribution and allelic frequency did not differ between elite swimmers and controls, or by event distance among elite swimmers only. No association was observed between telomere length and ACE genotype in the whole cohort. **Conclusions:** Elite swimmers possessed shorter telomeres than recreationally active controls. Our findings suggest a negative effect of high-level swimming competition and/or training on telomere length and subsequent biological aging, particularly in females. However, this significant difference in telomere length does not appear to be attributed to the D allele as we report a lack of association between telomere length and ACE genotype frequency in elite swimmers and controls.

Keywords: Athlete, Aging, Exercise and Genetics

INTRODUCTION

Telomeres are repeat nucleotide sequences (5'-TTAGGG_n-3') located at chromosome ends that confer genomic stability. Telomeres progressively shorten with each round of cell division and are considered to be a biomarker of biological aging (Huda et al., 2007). Shorter telomeres are associated with several age-related diseases (e.g. cardiovascular disease, cancer, diabetes) and an increased risk of mortality (Rizvi et al., 2015). Lifestyle factors including nutrition, sleep, stress management and physical activity (PA) have all been proposed to influence telomere length (TL) (Starkweather, 2014). For example, middle-aged and older individuals with higher levels of PA typically display longer telomeres than their sedentary counterparts (Cherkas et al., 2008; Saßenroth et al., 2015), although the effect of PA on TL in young individuals is less clear. The majority of the literature reports no association of PA with TL in young individuals (LaRocca et al., 2010; Østhus et al., 2012), although recent studies have provided evidence that PA-mediated effects on telomere maintenance may begin in adolescence/young adults (Mofrad and Ebrahim, 2018; Zhu et al., 2011). The lack of association between PA and TL in young individuals may partly be explained by an absence of significant age-related telomere attrition and/or insufficient exposure to PA levels (e.g. activity years). Nevertheless, a cohort of young elite athletes (27.2 ± 4.9 y) have been reported to possess longer telomeres than sedentary age-matched controls (Muniesa et al., 2017), suggesting that extremely high levels of exercise training, such as those performed by elite athletes, may be required in order to influence TL in young individuals.

Angiotensin converting enzyme (ACE) converts the hormone angiotensin I to the active vasoconstrictor angiotensin II. Angiotensin II has numerous actions, including promotion of a pro-inflammatory environment and increased oxidative stress (Fyhrquist and Saijonmaa, 2008), both of which are proposed to shorten TL (Correia-Melo et al., 2014). A 287-base *AhuI* polymorphism in intron 16 has been described (Insertion (I) / Deletion (D)) within the ACE

gene, resulting in three possible genotypes (II, ID and DD) (Rigat et al., 1990). The presence of the D allele has been linked with increased circulating ACE activity levels compared with II homozygotes (Rigat et al., 1990), which may lead to enhanced inflammation and oxidative stress. Interestingly, hypertensive patients with left ventricular hypertrophy with ID or DD genotypes display shortened leukocyte telomeres, suggesting a potential negative effect of the D allele on TL (Fyhrquist et al., 2013). It is well documented that the ACE D allele is associated with elite short distance swimming, and the ACE I allele with elite middle- and long-distance events (Costa et al., 2009; Nazarov et al., 2001; Tsianos et al., 2004; Woods et al., 2001). However, no study has investigated the notion that elite short distance swimmers may possess shortened telomeres compared with their middle- and long-distance counterparts due to the potential negative effect of the ACE gene D allele.

The primary aim of the present study is to investigate TL and ACE genotype in elite swimmers and recreationally active controls.

1. MATERIALS AND METHODS

2.1. Ethics Approval

Approval was obtained from the ethics committee of Loughborough University (R18-P175). The procedures used in this study adhere to the tenets of the Declaration of Helsinki. Informed consent was obtained from all individual participants included in the study.

1.2. Participants

A complete health assessment was completed for each participant using a structured questionnaire. Exclusion criteria for the study were known metabolic disorders, smoking,

cardiovascular disease, use of anti-inflammatory medication or steroids, any condition or disease that may interfere with the primary outcome measurement.

The study population comprised 51 elite young swimmers (characteristics in Table 1.), reporting an average training distance of $43,196 \pm 9,408$ m/week, reaction time of 0.69 ± 0.07 s and FINA point score of 805 ± 82 . All swimmers were currently competing at a minimum of national level, ranging up to Olympic standard (66% National, 2% European Championships, 8% Commonwealth Games, 14% World Championships, 10% Olympics). Swimmers were further categorised into three distinct categories based on their main event distance (41% sprint (≤ 100 m), 39% middle (≤ 200 -800m), and 20% long (≥ 800 m)).

A cohort of 56 healthy, age-matched control subjects were recruited. Control subjects were recreationally active and participated in a minimum 150 minutes of moderate PA, or 75 minutes of vigorous PA, per week. However, they were not involved in any structured training programme or competitive sports.

1.3. Anthropometric measurements

Height was measured using a Seca 213 stadiometer (Seca GmbH, Germany). Weight, body fat percentage (BF%), fat free mass (FFM) and body mass index (BMI) were quantified using the Tanita BC-418 MA segmental body composition analyser (Tanita Corporation, USA).

Table 1. Characteristics of elite swimmers and controls

	Female controls	Female swimmers	Male controls	Male swimmers
	<i>n</i> = 29	<i>n</i> = 22	<i>n</i> = 27	<i>n</i> = 29
Age (years)	19.6 ± 2.2*	20.5 ± 1.4	20.3 ± 4.7	20.6 ± 1.7
Height (cm)	165.1 ± 6.3*	171.4 ± 5.9	178.4 ± 6.0*	183.5 ± 5.3
Weight (kg)	61.7 ± 10.8	65.5 ± 6.3	72.4 ± 12.2*	77.7 ± 6.8
BMI (kg/m ²)	22.7 ± 3.4	22.3 ± 1.8	22.7 ± 3.5	23.0 ± 1.4
Body fat (%)	27.5 ± 6.0*	20.4 ± 3.9	14.0 ± 4.1*	11.1 ± 2.8
FFM (kg)	44.4 ± 4.5*	52.1 ± 5.0	62.0 ± 8.8*	69.0 ± 5.6

Data are means ± SD; *n*, no. of subjects. BMI, body mass index. FFM, fat free mass.

*Significant group effect.

1.4. Sample collection and DNA extraction

Participants collected buccal samples themselves using Isohelix DNA buccal swabs (Cell Projects Ltd, UK) following visual and verbal instructions. Samples were immediately placed in the microcentrifuge tube provided and kept at room temperature for a maximum of 2 hours, before being stored at −80°C until DNA extraction. DNA was extracted from buccal cells using the ReliaPrep™ gDNA Tissue Miniprep System (Promega, USA) according to the manufacturer's instructions. The quantity and quality of genomic DNA was assessed by spectrophotometry using a NanoDrop 2000 (ThermoFisher, USA), producing acceptable A260/280 values (1.88 ± 0.16).

1.5. Telomere length analysis

Measurement of buccal cell TL was conducted at the University of Leicester using the qPCR protocol previously described (Nickels et al., 2020). Telomere primer sequences were Tel1b 5'-CGGTTTGGTGGGTTTGGGTTTGGGTTTGGGTTTGGGTT-3'; Tel2b 5'-GGCTTGCCTTACCCTTACCCTTACCCTTACCCTTACCCTTACCCT-3', both diluted to a final concentration of 300nM. Primer sequences for the single-copy gene (36B4) were 36B4F 5'-CAGCAAGTGGGAAGGTGTAATCC-3'; 36B4R 5'-CCCATTCTATCATCAACGGGTACAA-3', both diluted to a final concentration of 500nM. Final reaction volume was 25µL, consisting of 22µL of qPCR master mix (SensiMix™ SYBR® No-ROX Kit, Bioline) and primers (targeting either the telomere or single copy gene) and 3µL of gDNA template (10ng/µL). Telomere (T) PCR cycling conditions consisted of 95°C for 10 min, 20 cycles of 95°C for 15 sec and 58°C for 1 min. Single-copy gene (S) PCR cycling conditions consisted of 95°C for 10 min, 30 cycles of 95°C for 15 sec and 58°C for 1 min. Telomere and single copy gene runs were performed separately, starting with former and followed immediately by the latter to keep conditions uniform. To ensure intra-run reliability each sample was run in duplicate. Any duplicate samples which had take-off values exceeding 0.2 of a cycle difference were excluded and re-run. To ensure inter-run reliability all PCR reactions were performed twice on two separate days using a Rotorgene Q (Qiagen, Germany). Alongside the samples, each run also contained a calibrator sample (pooled DNA) and no template control in duplicate. The average intra-plate coefficient of variation for calculated T/S ratio was 3.3%. The average inter-plate coefficient of variation for calculated T/S ratio was 4.1%. Relative T/S ratios were calculated using the Qiagen Rotorgene comparative quantitation software (Qiagen, Germany).

1.6. ACE genotyping

ACE genotype was determined using the following primers; forward, 5' CTGGAGACCACTCCCATCCTTTCT 3'; and reverse, 5' GATGTGGCCATCACATTCGTCAGAT 3', which were designed to amplify the polymorphic region from human genomic DNA. The PCR of 35 cycles, each with denaturation at 95°C for 30 seconds, annealing at 60°C for 90 seconds, extension at 72°C for 30 seconds, followed by a final cycle of 10 minutes at 66°C was conducted. PCR products were electrophoresed on 2% *w/v* agarose gel detected as 190-bp fragments in the absence of the insertion (D allele) and a 490-bp fragments in the presence of the insertion (I allele). 15% of samples were randomly repeated to check the validity of the genotyping. No errors were observed.

1.7. Statistical analysis

The normality and homogeneity of data were verified by the Shapiro-Wilk and Levene tests, respectively. The independent *t*-test or analysis of variance (ANOVA) was used to examine differences in mean TL, anthropometrics, genotype and performance data between groups. An analysis of covariance (ANCOVA) was also used to compare differences between groups when necessary. All associations were tested by two-tailed Pearson correlation. Allele frequencies were estimated by gene-counting method. Genotype distribution and allele frequencies between groups were compared by Chi-Square test, as were estimates of Hardy–Weinberg equilibrium. All analyses were performed using SPSS 23.0 (IBM Corporation, USA) with *p*-values < 0.05 considered significant.

2. RESULTS

3.1. Telomere length

Data analysis revealed control subjects possessed longer telomeres (1.128 ± 0.177) than elite swimmers (1.043 ± 0.127) ($p = 0.006$, calculated effect size = 0.55) (Figure 1). This group effect remained significant after adjusting for all covariates ($p = 0.031$). When analysed by sex, female swimmers displayed significantly shorter telomeres (1.049 ± 0.115) than female controls (1.154 ± 0.176) ($p = 0.019$, calculated effect size = 0.70), which remained significant after adjusting for all covariates ($p = 0.012$). There was no difference in TL between male swimmers (1.039 ± 0.136) and male controls (1.099 ± 0.176) ($p > 0.05$). There was no significant sex difference in TL for all participants after adjusting for covariates (males: 1.068 ± 0.158 , females: 1.109 ± 0.160 , $p = 0.114$).

2.2. Lack of association between telomere length and swimming performance

TL was not associated with swimming performance (FINA points) ($n = 50$, $p = 0.420$, $r = -0.117$), reaction time ($n = 33$, $p = 0.398$, $r = 0.152$), average weekly training distance ($n = 51$, $p = 0.613$, $r = -0.073$), main event distance ($p = 0.661$, $r = 0.063$) or highest level of competition ($p = 0.487$, $r = 0.099$) in swimmers overall. The lack of associations remained when swimmers were analysed by separate groups based on their sex ($p > 0.05$).

Fig. 1 Buccal cell TL for female swimmers (FS), female controls (FC), male swimmers (MS), male controls (MC), all swimmers (AS) and all controls (AC) (mean $\pm$ SD). *: different from FC; #: different from AC. Statistical significance defined as p-values <0.05 .

2.3. ACE I/D genotype and allele frequency

Genotypic frequencies in both swimmers and controls was consistent with Hardy–Weinberg equilibrium. There were no statistical differences for genotype and allele distributions between swimmers and controls overall (Table 2) or when analysed by distinct groups based on sex ($p > 0.05$). Furthermore, no statistical differences were observed for genotype distributions and allelic frequencies when the swimmers were stratified into three groups based on main event distance, or when the control group was compared with each individual swimming distance ($p > 0.05$) (Figure 2).

Table 2. ACE genotype distribution and allele frequencies of swimmers and controls

Subjects	<i>n</i>	ACE genotype			Allele frequency	
		DD	ID	II	D	I
Swimmers overall	50	13 (0.26)	25 (0.50)	12 (0.24)	0.51	0.49
Sprint	21	6 (0.29)	11 (0.52)	4 (0.19)	0.55	0.45
Middle	19	7 (0.37)	7 (0.37)	5 (0.26)	0.55	0.45
Long	10	3 (0.3)	7 (0.7)	0 (0.0)	0.65	0.35
Controls	54	12 (0.22)	29 (0.54)	13 (0.24)	0.49	0.51

Fig. 2 ACE I/D genotype frequencies amongst swimmers overall (SO), sprint swimmers (SS), middle-distance swimmers (MS), long-distance swimmers (LS) and controls (C).

2.4. Telomere length and ACE I/D genotype

In the whole cohort, TL was not associated with ACE I/D genotype ($p > 0.05$). Furthermore, TL was not associated with ACE I/D genotype when the cohort was stratified into distinct groups of swimmers, controls or split by sex ($p > 0.05$). There was also no association between TL and ACE I/D genotype when swimming groups were individually analysed by main event distance ($p > 0.05$).

3. DISCUSSION

The main findings of the present study are that: 1) elite swimmers display shorter TL compared with their recreationally active peers, which appears specific to females; 2) buccal cell TL is not associated with ACE I/D genotype; 3) swimming main event distance is not associated with ACE I/D genotype distribution.

There is a progressive loss of telomeres with each round of cell division and they eventually reach a critically short length, culminating in replicative senescence (Olovnikov, 1973). However, engagement in moderate levels of PA is associated with longer telomeres and may preserve their length (Cherkas et al., 2008; Saßenroth et al., 2015). Conversely, middle-aged and older individuals with extremely low or high PA levels appear to exhibit shorter telomeres than their moderately active counterparts (Cherkas et al., 2006; Savela et al., 2012). To highlight, individuals with extremely high levels of PA have been reported to display telomeres comparable in length to their low PA level counterparts (Ludlow et al., 2008). As such, an ‘inverted U’ relationship between PA level and TL has been proposed, in which low and extremely high levels of PA may be detrimental to TL and lead to accelerated shortening (Ludlow et al., 2008).

In the present study, the relative TL of young recreationally active control subjects was 8.1% greater than the elite swimmers. When that data was analysed split by sex, female controls displayed telomeres 9.9% longer than elite female swimmers. The lack of difference in TL observed between male controls and swimmers indicates that participation in elite level swimming training/competition may be detrimental to telomere maintenance and subsequent biological aging in females. We cannot be certain that these findings are biologically relevant at such as young age, but short buccal cell telomeres have been associated with age-related disease (Broberg et al., 2005; Thomas et al., 2008). These major findings are in contrast with previous data that reported young high-level international athletes possess longer leukocyte TL (LTL) than healthy controls (Muniesa et al., 2017). The discrepancy in these findings may be explained by the fact that the elite athletes used in the study by Muniesa *et al.* were recruited from a wide variety of sport specialties (running events >1500 m, triathlons, weightlifting, judo, gymnastics, canoeing, basketball, soccer, taekwondo), each of which generates different metabolic demands (Muniesa et al., 2017). Several other studies investigating TL and PA in young individuals have involved non-elite athletes and have reported no difference in TL between active and sedentary individuals (LaRocca et al., 2010; Østhus et al., 2012; Werner et al., 2009). This may be because young individuals have not accumulated enough training years/volume, or they are yet to experience any significant age-related telomere attrition. Nonetheless, we are the first to demonstrate that extremely high training loads can be detrimental to TL in elite young athletes, specifically females, a finding which may offer support to the hypothesis of an ‘inverted U’ relationship between PA and TL. In combination with the results reported by Muniesa *et al.*, it appears that the physical demands of elite level sports competition/training may provide a novel stimulus that is able to both positively, and negatively, influence telomere dynamics in young individuals.

Rae *et al.* have previously reported that skeletal muscle TL was inversely correlated with the number of hours and years spent training in older endurance runners (Rae et al., 2010). Additionally, it has previously been reported that individuals with high PA (exercising 10–14 hours/week) possess shortened TL compared with moderately active individuals (Ludlow et al., 2008). At the time of data collection, swimmers were in competition season and training ~14 hours/week in the pool alone (excluding any additional training). Thus, despite a lack of association between the average weekly training distance and buccal cell TL amongst swimmers in the present study, the high number of training hours they performed may potentially explain their shortened telomeres compared with the recreationally active controls. Furthermore, highly trained middle-aged endurance athletes experiencing ‘exercise-associated chronic fatigue’ or ‘overtraining’ report significantly shorter skeletal muscle TL compared with age- and training-matched athletes free from these symptoms (Collins et al., 2003). Although we did not specifically diagnose whether the swimmers were ‘overtrained’ in the present study, we can only speculate that some of them may have been in such a physical state based on their reportedly high weekly training hours and average swimming distance. If this was the case, then the elevated resting levels of ROS and inflammation associated with overtraining (Margonis et al., 2007) and shortened TL (Correia-Melo et al., 2014) may explain the results of the present study. It is important to reiterate that whilst this is biologically plausible, we cannot deduce such a conclusion from our findings and that further research is required.

Due to time constraints and the nature of the study cohort, we were only permitted to collect DNA from the swimmers using non-invasive procedures. TL is typically measured in leukocytes extracted from venous blood, which would have permitted us to measure inflammatory and oxidative stress markers and potentially enable us to deduce biological causality for our findings. The rationale for using buccal cell-derived DNA in the present

study was based on the fact that although absolute leukocyte and buccal cell TL values vary, there is a significant intra-individual correlation across the two tissue types (Gadalla et al., 2010). Buccal cell TL measurements also show a significant association with both sex and age, as seen with LTL, providing further evidence of similarities in telomere dynamics between the tissues (Finnicum et al., 2017). Previous work in our laboratory also revealed a significant positive linear relationship between the LTL and buccal cell TL with a large magnitude of association ($r = 0.599$, $p = <0.0001$) in a group of young and middle-aged runners ($n = 69$) (supplementary data). In support of the findings by Finnicum *et al.* and Gadalla *et al.*, our supplementary data revealed that buccal cell TL (0.953 ± 0.133) is significantly longer than LTL (0.818 ± 0.121) ($p = <0.0001$) and both tissue types displayed the expected negative correlation with age ($p = 0.006$, $r = -0.331$ and $p = <0.0001$, $r = -0.432$, for leukocyte and buccal cell, respectively). Together, these studies demonstrate synchrony in the telomere dynamics of leukocyte and buccal cells, supporting the use of the latter as a viable biomarker of aging.

TL and shortening rate is tissue specific (Aubert and Lansdorp, 2008), with the variability in attrition across tissue types likely the result of divergent proliferative capacities; highly proliferative tissues (e.g. leukocytes and epithelial cells) typically display greater age-related telomere shortening than minimally proliferative tissues (e.g. skeletal muscle) (Kadi and Ponsot, 2010). Many of the studies referenced in the present paper have measured TL across a variety of tissues, which may lead to different associations with TL and exercise performance. However, Daniali *et al.* revealed that intra-individual age-related telomere attrition is similar across somatic tissues with divergent replicative status (leukocytes, muscle, skin and fat), despite variations in their absolute length (Daniali et al., 2013). This finding suggests that there may be a shared molecular mechanism regulating TL across somatic tissue types. Several other studies have also reported significant correlations in TL

298 between tissue types with different proliferative demands (Friedrich et al., 2000; Hiam et al.,
299 2020). To our knowledge, our supplementary data was the first to investigate the relationship
300 between LTL and buccal cell TL in athletes at the time of the present study. As we revealed
301 that TL is strongly correlated across the two tissue types in a group of young and middle-aged
302 runners, we are inclined to believe that buccal cell TL responds to exercise in synchrony with
303 leukocytes (inverted U relationship) and is impacted via the same exercise-induced
304 mechanisms (e.g. inflammation and oxidative stress). More recently, genomic DNA isolated
305 from buccal swabs revealed elderly ultra-trail runners display longer telomeres compared to
306 age-matched sedentary controls (Hernando et al., 2020). Similar studies have also previously
307 shown that ultra-endurance athletes have better preserved telomeres in both skeletal muscle
308 (Østhus et al., 2012) and leukocytes (Denham et al., 2013), suggesting a synchrony in TL
309 across tissue types in response to exercise. Thus, we speculate that the present results would
310 have been replicated in our cohort if TL had been measured in leukocytes (although absolute
311 values would be different). However, it must be stated that there is evidence to suggest that
312 the response of TL to exercise may be unique in skeletal muscle, which is likely caused by
313 their satellite cell content. As exercise promotes muscle damage and stimulates the
314 proliferation of satellite cells (Yin et al., 2013), and telomere attrition occurs with cell
315 division, exercise may cause telomere loss in skeletal muscle tissue of highly active
316 individuals. Chronic endurance running has therefore been reported to shorten skeletal
317 muscle TL due to the increased demand on satellite cells to proliferate and repair damaged
318 tissue (Rae et al., 2010). Long-term exercise also accelerates TL attrition in skeletal muscle in
319 mice, but attenuates age-related decreases in TL in cardiac and liver tissue (Ludlow et al.,
320 2012). Thus, the difference in TL between elite swimmers and controls in the present study
321 may be even more significant when measured in skeletal muscle. Additionally, no study has
322 investigated how exercise specifically impacts replicative stress in the mouth of humans and

further research is required to determine whether TL responds to exercise in a synchronous or tissue-specific fashion.

Another key finding of the present study is the lack of association between ACE genotype and TL in elite swimmers or controls. It has been previously reported that the presence of the D allele is associated with shortened TL in hypertensive patients with left ventricular hypertrophy (Fyhrquist et al., 2013). Additionally, several previous studies that have reported an excess of the D allele in elite short distance swimming (Costa et al., 2009; Nazarov et al., 2001; Tsianos et al., 2004; Woods et al., 2001). However, our results reveal that TL is not associated with ACE I/D genotype distribution in swimmers or controls. As such, the accelerated telomere attrition displayed by the swimmers in the present study cannot be attributed to a negative effect of the D allele and/or variations in ACE activity. We also observed a lack of association between swimming event distance and ACE I/D genotype. Although this finding is not novel, as the association between ACE I/D genotype and athletic performance is well studied amongst elite swimmers, the duplicability of such an association is contentious. For instance, studies have shown an increased frequency of the I allele with middle- (Nazarov et al., 2001) and long-distance swimming (Tsianos et al., 2004). However, our results reinforce previous reports that show a lack of association between elite long-distance swimming and ACE genotype (Nazarov et al., 2001; Woods et al., 2001) and also between controls and elite swimmers competing in events >400m (Costa et al., 2009). Further research is required to clarify our findings due to the ambiguity of the current literature and based on the limited sample size, the present data should be interpreted with caution.

The novel aspects of our study are that it is the first to specifically investigate the TL of elite swimmers, and to report that elite athletes display shortened telomeres compared to their recreationally active counterparts. Additionally, no study has previously explored the association between ACE genotype and TL in elite athletes. However, the present study is not

without limitations. For example, we did not control for known certain confounders of TL, such as psychological stress or nutrition. Although 36B4 serves as a commonly used reference gene in conventional qPCR for telomere measurement (Cawthon, 2002; Denham et al., 2013; Hiam et al., 2020), it may amplify pseudogenes on chromosomes 12 and 5. The present study also lacked an additional group of young sedentary controls, which would have permitted us to further explore the ‘inverted U’ hypothesis and determine whether the elite swimmers had comparable TL to their sedentary peers. As such, further studies investigating the associations between TL, oxidative stress and inflammation in young elite athletes compared with both recreationally active and sedentary subjects are required.

To conclude, the present study reveals that elite female swimmers, who routinely engage in high levels of competition and/or training, possess shorter telomeres than their recreationally active counterparts. Participation in elite level swimming training/competition may therefore be detrimental to telomere maintenance and subsequent biological aging in females, which may be significant in later life as short buccal cell telomeres are associated with age-related disease (Broberg et al., 2005; Thomas et al., 2008). However, as we did not observe an association between ACE I/D genotype distribution and TL in elite swimmers and/or controls, the accelerated telomere attrition detected in elite swimmers does not appear to be influenced by a potential negative effect of the D allele and/or variations in ACE activity.

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Declarations of interest: none

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