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1 **Title:** Increased Oxidative Stress in Injured and Ill Elite International Olympic Rowers

2 **Submission type:** Original investigation.

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22

23 **Abstract (250)**

24 Identifying strategies that reduce the risk of illness and injury is an objective of sports science and
25 medicine teams. No studies have examined the relationship between oxidative stress (OS) and
26 illness or injury in international athletes undergoing periods of intensified training and
27 competition. **Purpose:** We aimed to identify relationships between illness, injury and OS.
28 **Methods:** A longitudinal, observational study of elite male rowers (n=10) was conducted over
29 18-weeks leading into World Championships. Following a recovery day and a 12-hour fast,
30 hydroperoxides (FORT) and total anti-oxidant capacity (FORD) were measured in venous blood,
31 with the ratio calculated as the oxidative stress index (OSI). At all study time points, athletes were
32 independently dichotomized as ill or not ill, injured or not injured. OS data were compared
33 between groups using independent t-tests. A Cox proportional hazard model was used to assess
34 the association of OS with injury and illness while adjusting for age and body mass index. **Results:**
35 FORD was lower ($p<0.02$) and OSI was higher ($p<0.001$) with illness than without illness. FORT
36 and OSI were higher with injury than without injury ($p<0.001$). FORD exerts a protective effect
37 on illness with $0.5 \text{ mmol}\cdot\text{L}^{-1}$ increase related to a 30.6% illness risk reduction ($p=0.014$), and OSI
38 exerts a harmful effect on illness risk with a 0.5 unit increase in OSI related to an 11.3% increased
39 risk ($p=0.036$). **Conclusion:** OS is increased in injured and ill athletes. Monitoring OS may be
40 advantageous in assessing recovery from, and in reducing injury and illness risk given the
41 association.

42

43 Introduction

44 Loss of training time due to illness and injury, is a major determinant of performance goal
45 success or failure for elite international athletes competing in individual Olympic sports¹.
46 Exceedingly high training loads and/or spikes in training load are a recognised risk factor for injury
47 and illness, and fatigue². Furthermore, illness and accumulating fatigue may result in the
48 development of chronic underperformance³; all of which can derail the athlete's season and may
49 finish careers. Finding ways to reduce the risk of injury and illness is therefore a primary objective
50 of the high-performance support team.

51 Oxidative stress (OS), historically and simply defined as a disturbance in the pro- to anti-
52 oxidant balance in favour of the former⁴, is evident in athletes diagnosed with overtraining
53 syndrome (OTS^{5,6}). Indeed, increases in biomarkers of OS correlate strongly with increases in
54 training volume^{7,8}. Furthermore, elite athletes designated as "impaired performers" had a twofold
55 greater total peroxide load (reported in H₂O₂ equivalents) and were more prone to infections⁹.
56 Such findings suggest that the longitudinal monitoring of OS biomarkers in the elite endurance
57 athlete may provide an indicator of excessive training load, increased risk of illness and injury,
58 and therefore allow for improved optimisation of workload¹⁰.

59 International rowers are exposed to high volume training and are at a higher risk of injury
60 than many non-contact sports, and some contact sports¹¹. However, due to the non-contact nature
61 of rowing, competition periods may pose less risk for injury; rowing being one of the sports with
62 the lowest incidence of injury at the Rio Olympic Games¹². In contrast, competition periods pose
63 a greater risk of illness in elite athletes^{13,14}. To the authors knowledge no studies have investigated
64 the relationship between OS biomarkers and injury in athletes, and only one study has investigated
65 the relationship with illness⁹.

66 We have previously shown the clinical point-of-care OS test, known as free oxygen
67 radicals test (FORT; a measure of hydroperoxides) and free oxygen radicals defence (FORD; a
68 measure of plasma antioxidant capacity), to be repeatable¹⁵, clinically useful in elite sport^{6,16}, and
69 possess validity in terms of capturing acute changes in OS in elite endurance athletes¹⁷. In this
70 observational study we analysed longitudinal data from elite international Olympic rowers.
71 Rowing is a sport in which some of the largest physiologically relevant changes in OS have been
72 reported¹⁸; reviewed in Lewis et al.¹⁰, and we have previously shown evidence of substantial OS
73 in an elite international rower diagnosed with unexplained under performance
74 syndrome/overtraining syndrome using the FORT and FORD⁶.

75 The aim of the present study was to identify the relationship between changes in OS
76 biomarkers and injury and illness incidence during a competitive phase of the season in a
77 prospective, longitudinal study of elite international rowers.

78

79 **Methods**

80 **Subjects**

81 Eight openweight (Age 26.9 ± 2.2 years, height 192.3 ± 3.1 cm, weight 93.1 ± 4.9 kg) and two
82 lightweight (Age 28.6 ± 0.2 years, height 186.0 ± 1.4 cm, weight 73.3 ± 0.4 kg) male rowers
83 (including World and Olympic medalists) were recruited to participate and provided written
84 informed consent. Athletes were free living and attending a national training centre, were not
85 taking any medications and were subject to United Kingdom Anti-doping controls and testing
86 procedures. Data were collected weekly at the same time of day and included the following:
87 oxidative stress via the point of care blood test, prescribed training volume and intensity and
88 subjective assessments of wellness and sleep quality. All procedures were approved by the Internal
89 Review Board of the English Institute of Sport. All athletes were tested in the competition phase
90 of the annual cycle up until the World championships, including the World Cup series and two
91 altitude training camps ranging from 2,000-2,300m. Athletes were classified as injured and ill by
92 the medical staff, and for reasons of confidentiality, the specific details of the injuries and illnesses
93 are not disclosed.

94 **Design**

95 This was a longitudinal observational study to establish the relationship between biomarkers of
96 oxidative stress, injury and illness. Over the course of 122 days, 15 repeated measurements of
97 oxidative stress were taken from these rowers from venous blood samples, plasma hydroperoxides
98 and plasma antioxidant defence, along with the ratio of the two, the oxidative stress index (OSI).
99 Testing was carried out between 6 a.m. and 9 a.m. in a fasted, hydrated, and rested state. In the 24
100 hours prior to testing, training was kept to either an aerobic training day of low to moderate
101 intensity or a rest day. We present simple summaries of all biomarkers across illness and injured
102 status, not adjusted for repeated measures. Each illness and injury was diagnosed and documented
103 by the sports medicine team in the environment on the day of testing. In total there were eight
104 episodes of illness in six of the rowers across the study period, of which three required antibiotics
105 for treatment.

106

107 **Methodology**

108 *Blood sampling for oxidative stress tests*

109 Venous blood samples were taken from an antecubital vein using a 5 ml lithium heparin
110 vacutainer tube (BD system; New Jersey, USA), with 50 μ L and 20 μ L of blood transferred into
111 heparinized capillary tubes for the analysis of FORD and FORT respectively, in line with the
112 manufacturer's instructions (Callegari SpA, Catellani Group, Parma, Italy). Intra- and inter
113 assay coefficients of variation for FORT and FORD were < 5% and 7%, respectively.

114 *FORT assay*

115 We have described the details of the assay previously¹⁵. Briefly, reactive oxygen species
116 (ROS) activity was determined downstream through the measurement of hydroperoxides via the
117 FORT test. FORT is a colourimetric assay based on the capacity of transition metal ions (Fe^{3+})

118 /Fe²⁺) to catalyze the breakdown of hydroperoxides (R-OOH) into derivative radicals [alkoxyl (R-
119 O[•]) and peroxy radicals (R-OO[•])] within the biological sample. The application of an acidic buffer
120 to the 20µL blood sample, releases the transition metals from associated proteins, which react with
121 the hydroperoxides present in the sample, producing the alkoxyl and peroxy radicals. The
122 derivative radicals are trapped through the addition of a buffered chromogen (reagent; an amine
123 derivative, CrNH₂) and develop into a radical cation in a linear based reaction at a controlled
124 temperature of 37°C, photometrically detectable at 505nm.

125 The intensity of the sample colour correlates with the quantity of radical compounds and
126 therefore the concentration of hydroperoxides in the biological sample, according to Lambert-
127 Beer's law. The results are expressed as equivalent concentrations of H₂O₂ mmol·L⁻¹.

128 *FORD assay*

129 The FORD test (Callegari, Catellani, Italy) determines the presence of plasma antioxidants
130 via a colourimetric assay based on the capacity of the sample to reduce a preformed radical cation.
131 In the presence of an acidic buffer and a suitable oxidant (FeCl₃), the chromogen that contains 4-
132 amino-N,N-diethylaniline sulfate forms a stable and coloured radical cation, photometrically
133 detectable at 505 nm. The antioxidant compounds present in the plasma sample reduce the radical
134 cation of the chromogen, quenching the colour, and causing a discolouration of the sample,
135 proportional to the concentration of antioxidants present. The absorbance values generated are
136 compared to standard curves derived from Trolox (6-hydroxy-2,5,7,8-tetramethylchroman-2-
137 carboxylic acid), a derivative of vitamin E with enhanced water solubility. FORD values are
138 reported as Trolox equivalents, mmol·L⁻¹, linearity ranged from 0.25 to 3.0 mmol·L⁻¹ Trolox.

139 *Screening blood tests*

140 Additional blood draws via the antecubital vein were conducted on two occasions, with 3 x 5 ml
141 venous blood samples collected in a serum separator (SST) and 1 x 5 ml blood sample via an
142 EDTA (ethylenediamine tetraacetic acid) vacutainer tube (BD system; New Jersey, USA). Full
143 blood counts, iron status (Ferritin, Iron, Transferrin saturation), thyroid function (Thyroid
144 Stimulating Hormone), nutritional status (vitamin C, vitamin D, alpha-tocopherol, and the
145 carotenoids; lutein, alpha-carotene and beta-carotene), muscle damage and inflammation (Creatine
146 Kinase, C-Reactive Protein), were measured at the beginning of the study period and again prior
147 to the final training prior to the world championships.

148 We have previously described the methodology and assay performance of the aforementioned
149 venous and capillary biomarkers ¹⁵.

150 *Training Assessment*

151 Training was prescribed by coaching staff in accordance with aims of the national programme and
152 was deliberately not influenced by the study design or the research personnel. For ease of
153 interpretation, prescribed training is characterised using the adopted terminology as either
154 “<UT2”; a training intensity below the onset of blood lactate accumulation or “>UT2”; an intensity
155 above the onset of blood lactate accumulation. These are well established for each rower, based
156 on regular and routine physiological testing conducted by sports scientists.

157 *Wellness and Sleep*

158 Subjective assessments of wellbeing and sleep quality were recorded daily using a smartphone
159 application developed by the English Institute of Sport. The app contained 5 questions; ratings of
160 perceived energy level (0-10 scale), perceived “shape” (0-10 scale), perceived freshness (0-10
161 scale), perceived sleep quality (0-10 scale), and perceived sleep duration (minutes).

162

163 **Statistical analysis**

164 All statistics were carried out using R software. The distributions of all variables were
165 assessed for normality and the presence of outliers with box-plots. To determine differences
166 between means for healthy rowers and injured and ill rowers independent T-tests were used, with
167 Cohen’s d effect sizes (d) used to calculate the magnitude of the standardised difference in means
168 where significant, and reported as 0.2 (small), 0.5 (moderate), 0.8 (large), and 1.3 (very large).

169 There are two main outcomes for analysis, time to illness and time to injury. We treat both
170 as time-to-event variables, namely that we have both an event (e.g. ill/not ill) and a time of illness
171 (day). The time of illness for those athletes who were never ill is set to the length of the study, i.e.
172 these athletes are said to be censored at the end of the study. Illness and injury are both modelled
173 using a Cox proportional hazards model [2], which is the most commonly used approach for time-
174 to-event variables. For each variable under examination (FORD, FORT and OSI) we estimate a
175 hazard ratio for a 0.5 unit increase in FORD, FORT and OSI, since a 1-unit change is not realistic.
176 A hazard ratio of 1 represents no effect of the variable being tested, below 1 represents a protective
177 effect and above 1 represents a harmful effect. A further complication with these data analysis of
178 these data is that athletes can become ill or get injured multiple times. This repeated measure of
179 illness or injury is correlated over time, and we must account for this correlation (i.e. that an athlete
180 who gets ill may be more likely to get ill again) as well as the correlation between OS biomarkers
181 over time. To account for such repeated measures of OS, illness and injury in this study we
182 included a so-called frailty term in the Cox model for each athlete ID. This term controls for the
183 correlation that is likely present in the outcomes over time. To control for potential confounding
184 effects, we included age and BMI (kg/m^2) in models of both illness and injury.

185 We use 95% confidence intervals to provide evidence as to whether our sample results are
186 likely to infer population effects for all athletes represented by this sample. We also report p-
187 values, but do not use a stringent cut-off of 0.05 to determine a population effect. We use a strength
188 of evidence approach ¹⁹, whereby smaller p-values suggest stronger evidence for a population
189 effect. Data are presented as mean \pm SD.

190

191

192 **Results**

193 A total of 140 blood samples were drawn in the rowers across the competitive phase. OS testing
194 compliance was very good, at 93%. In contrast, compliance across the training period was poor
195 for the entry of the wellness and sleep data in the smart phone app. All 10 rowers entered data at
196 various time points throughout, however, compliance ranged from 7% to 100% on an individual
197 level, and 44% overall for the squad. Of the ten rowers, eight competed in the World
198 Championships at the end of the monitoring period.

199

200 **Training load**

201 The prescribed training load across the competition phase, through to the World Championships
202 is presented in Figure 1. The peak in training load occurred in week 13, totaling 227.5 km,
203 including 41 km performed above UT2 intensity.

204

205 Figure 1 here

206

207 **Injury and Illness**

208 All injuries were of rib, back or hip in origin. No additional medical details are included
209 with regards to the illnesses (i.e. infections) and injuries due to issues of confidentiality.

210 The results in Figure 2 do not account for the correlation of repeated measurements within athletes
211 over time but give a simple comparison of levels of biomarkers across health status. Lower FORD
212 ($p=0.02$), higher FORT ($p=0.063$) and higher OSI ($p=0.003$) were observed on days where an
213 athlete was ill, while higher FORT ($p<0.001$) and OSI ($p=0.017$) were observed in measurements
214 taken while an athlete was injured.

215

216 Figure 2

217

218 **Modelling**

219 The results of a Cox regression of the time to illness and injury are given in Table 3.

220

221 Table 1 here

222

223

224 **Modelling illness**

225 There was evidence for a protective effect of FORD on illness, with a 0.5 mmol·L⁻¹ increase in
226 FORD associated with a 30.6% risk reduction for illness in this sample (hazard ratio 0.694). A
227 95% confidence interval for this estimate suggests that in a population of endurance athletes, the
228 mean risk reduction is likely between 7% and 48.2% (hazard ratio 95% CI 0.518, 0.93; p=0.014).
229 OSI had a harmful effect on illness risk, with a 0.5 unit increase in OSI being associated with a
230 11.3% increased risk of illness in this sample (hazard ratio 1.113, 95% CI 1.007, 1.231; p=0.036)
231 on average.

232 **Modelling injury**

233 There was only weak evidence for a harmful effect of OSI, with a 0.5 unit increase associated with
234 a 8.3% increased risk of injury in this sample (hazard ratio 1.083). The 95% confidence interval
235 suggests that this estimate could range from a 1.3% reduction in injury risk to a 19% increased risk
236 (hazard ratio 95% CI 0.987, 1.19; p=0.094).

237

238 **Haematology and biochemistry**

239 See table 2.

240 Discussion

241 The aim of the study was to identify the relationship between injury, illness (i.e. infections)
242 and biomarkers of OS. We report for the first time, strong associations between redox biomarkers
243 and both illness and injury in elite male rowers. OSI was higher in both ill and injured athletes,
244 furthermore it was associated with increased risk of both illness and injury when analysed through
245 a Cox proportional hazards model. Given the low sample size these results merit further
246 investigation in a larger group.

247 *Illness*

248 The higher OSI (lower FORD and higher FORT) observed in the rowers with infections can be
249 explained by the increased production of reactive oxygen and nitrogen species that occurs with
250 activated leukocytes, namely the phagocytic, polymorphonuclear leukocytes, in the presence of a
251 bacterium or virus. We have not reported the types of infection encountered by the rowers (e.g.
252 whether bacterial or viral, or of upper respiratory, gastrointestinal tract in origin) for reasons of
253 confidentiality, however, both viral and bacterial infections can give rise to OS. Evidence of
254 increased OS in physically active individuals with infection and acute and severe infection has
255 previously been reported²⁰, and a relationship between performance, illness and OS has previously
256 been documented in elite athletes. For example, in a longitudinal study of elite alpine skiers, those
257 athletes classified as “impaired performers” had approximately double the total peroxide load
258 (H_2O_2 equivalents) and were more prone to infection than those skiers classified as “good
259 performers”⁹. The fact that a $0.5 \text{ mmol}\cdot\text{L}^{-1}$ increase in blood antioxidant capacity (i.e. FORD) in
260 the present study was associated with a ~30% risk reduction in illness, warrants further
261 investigation in other athletic populations. It is noteworthy that others have reported a diminished
262 plasma antioxidant capacity in the overloaded/overtrained athlete^{8,21}, and overtrained athletes are
263 reported to be more susceptible to illness²². Furthermore, in testing the rower’s plasma ascorbate
264 (vitamin C) (added to the profile as part of a nutritional screen; see Table 2), a number of the squad
265 were observed to have a marginal vitamin C status ($<23 \text{ umol}\cdot\text{L}^{-1}$), which may have contributed
266 to low FORD values and reported illness. The ability to track and quantify the athlete’s risk of
267 illness, would be of value to any sports science team, given that significant proportions of training
268 time lost to illness does occur. Indeed, UK Sport and the English Institute of Sport estimated that
269 for the 12 months prior to the Rio 2016 Olympics, 108,845 days and 17,173 days of training were
270 lost to injury and illness respectively, as a result of 4685 separate injury or illness incidents,
271 experienced by 1144 athletes across 38 sports (UK Sport, unpublished data). Clearly athletes who
272 are repeatedly ill, and therefore lose training time due to illness, are more likely to experience
273 competition failure¹.

274 It is of interest, that the doubling of antioxidant foods in elite endurance athletes training
275 at altitude (Sierra Nevada) increases plasma antioxidant capacity and attenuates inflammation
276 without impeding training adaptations (Koivsto et al. 2019). Whilst the administration of
277 encapsulated dried fruit and vegetable concentrates (EDFC) increase plasma antioxidant capacity
278 and circulating gamma T-cells leading to fewer ($p=0.07$) self-reported total illness symptoms in
279 healthy students (Nantz et al, 2006). Indeed, the same EDFC administered to special forces results
280 in a similar trend, with fewer duty days lost to illness ($p=0.06$) and a significant decline in
281 inflammation and oxidative stress (Lamprecht et al. 2007). To our knowledge, fruits and
282 vegetables (FV) (dried or whole) have not been shown to attenuate adaptations to exercise. Thus,
283 increasing FV consumption increases plasma antioxidant capacity, and may reduce infection risk,

284 and such advice should be given to athletes for illness prevention. Whilst speculative, increasing
285 FV whilst ill, may serve as to attenuate symptoms and lead to faster resolution of the infection
286 through moderating oxidative stress and immune responses. Finally, it is noteworthy, that despite
287 the rowers receiving World class performance nutrition support, we observed a marked decline for
288 some antioxidant nutrients (i.e. alpha-carotene, lutein, vitamin C; see table 2) in individual athletes
289 across the period of monitoring; with borderline nutrient status for vitamin C in 2 cases. Despite
290 very limited research in athletes, others have previously reported such findings in elite athletes for
291 carotenoids post altitude training (Pialoux et al.). Although the beyond the scope of this paper, we
292 feel this is an area that requires further research in view of the importance of antioxidant nutrients
293 for the health of the athlete.

294

295 *Injury*

296 OS biomarkers correlate with training load and volume ^{7,8}, and exceedingly high training loads
297 and/or spikes in training load are a recognised risk factor for injury ². Furthermore, OS has been
298 suggested to play a role in the pathophysiology of overuse injuries ²³. We report a significant
299 difference between OSI and FORT biomarkers for injured versus healthy rowers, but not FORD
300 (Figure 2). In modelling the data, there was some evidence for a harmful effect of the OSI with
301 regards to injury, with a 0.5 unit increase associated with an 8.3% increased risk of injury in this
302 sample (hazard ratio: 1.083). In the present study, the rowers were monitored between May and
303 September in the competition phase, a period in which both the training volumes and the potential
304 for injury could be lower than the general preparation training phase in the winter. Indeed, a higher
305 injury rate has been reported in the winter months in international rowers, coinciding with periods
306 of highest training volumes ¹¹. Had a greater number of injuries resulted, stronger associations
307 with injury and OSI maybe have been evident. Further research is required to explore this
308 relationship in a larger cohort of elite athletes. Moreover, in order to test the validity of monitoring
309 redox biomarkers as a means of guiding strategies aimed at protecting the athlete and mitigating
310 against injury, the monitoring period should encompass the entire season to include the general
311 preparation phase.

312 *Strengths and limitations*

313 FORT and FORD data were repeatedly measured in the same athletes overtime, which
314 allows for stronger inferences to be made compared with a cross-sectional study. We used Cox
315 proportional hazard models to model time-to-event outcomes, while including a frailty term to
316 account for the within player correlation in FORT and FORD. A limitation was the lack of data
317 available on the study athletes, such as profile of mood states, sleep quality and muscle soreness
318 data, which may have provided more information on the relationship between illness/injury and
319 OS.

320 We have previously highlighted limitations associated with the blood-based OS biomarkers
321 used in this study ¹⁷, and others have recently reviewed the topic of redox biomarkers ²⁴. The FORT
322 (e.g. hydroperoxides) and FORD (e.g. plasma antioxidant capacity) biomarkers provide minimal
323 insight into the complexity of redox signaling and associated cellular changes. The fact that such
324 biomarkers provide little mechanistic insight into cellular redox pathways is of lesser importance
325 in the context of how these biomarkers are applied in the field or the clinic setting. Indeed, the

326 primary purpose of deploying such biomarkers in the field is to generate real time results (as
327 opposed to a delayed retrospective analysis) to monitor athlete “stress” and recovery, and assist in
328 determining the appropriate hormetic “stimulus” for adaptation in the context of the athletes own
329 historic data. Crucially, these point of care tests are useful to be able to inform athlete management
330 decisions being made at the time of sampling.

331 It should be recognised that the athletes in this study were part of the British elite sport
332 system and included athletes of the very highest caliber (i.e. Olympic Champion), with all athletes
333 being supported by sports science and medicine practitioners. All athletes were healthy on
334 commencing the study as evidenced in the biomarker wellness screen (Table 2). In addition, the
335 research can be considered to have high ecological validity, as the rowers did not change their
336 routines, diets, or planned training based on any of the monitoring data. In fact, it is clear the
337 rowers engaged and complied with the weekly blood monitoring, which contrasts with the
338 compliance to the subjective wellness data collection; despite entry and collection being simplified
339 through the use of a smartphone application and the questions being tailored to the rowers and
340 their environment.

341 *Practical considerations*

342 OS is increased in injured and ill athletes, and the monitoring of OS may be advantageous
343 assessing recovery from, and in reducing injury and illness risk given the association. In addition,
344 avoiding sustained OS may be advantageous in reducing injury and illness occurrence in individual
345 endurance athletes, however further research is needed. The data presented here provide
346 preliminary evidence that both infection and injury risk can be inferred with measurable changes
347 in OS. Given the inherent practical challenges that come with subjective data collection (e.g. long-
348 term athlete compliance, reliability of the data entered), these biomarkers provide an objective
349 means of quantifying “stress” in the clinic or in the field (e.g. an overseas training camp) for sports
350 science and medicine practitioners.

351 **Conclusions**

352 We have demonstrated that plasma antioxidant capacity (i.e. FORD) may exert a protective effect
353 with regards to the risk of illness, and that OSI is associated with both injury and illness in a cohort
354 of elite rowers. Similar studies in other Olympic and professional sports are needed to corroborate
355 these findings and to prospectively deploy these blood tests to reduce injury and illness.

356

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360

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362

363 Authors contributions

- 364 NAL, RB, SM, GT, MH, AR. Conception and design of the study
- 365 SM, GT, MH, AR. Data collection
- 366 NAL, AJS, CRP, RB. Analysis and interpretation of the work
- 367 NAL, AJS, CRP, RB. Drafting of the manuscript
- 368 NAL, AJS, CRP, RB, SM, GT, MH, AR. Reviewed and approved the final version of the manuscript.
- 369 All authors approved the final version of the manuscript and agree to be accountable for all aspects of the
- 370 work. All persons designated as authors qualify for authorship and are listed.

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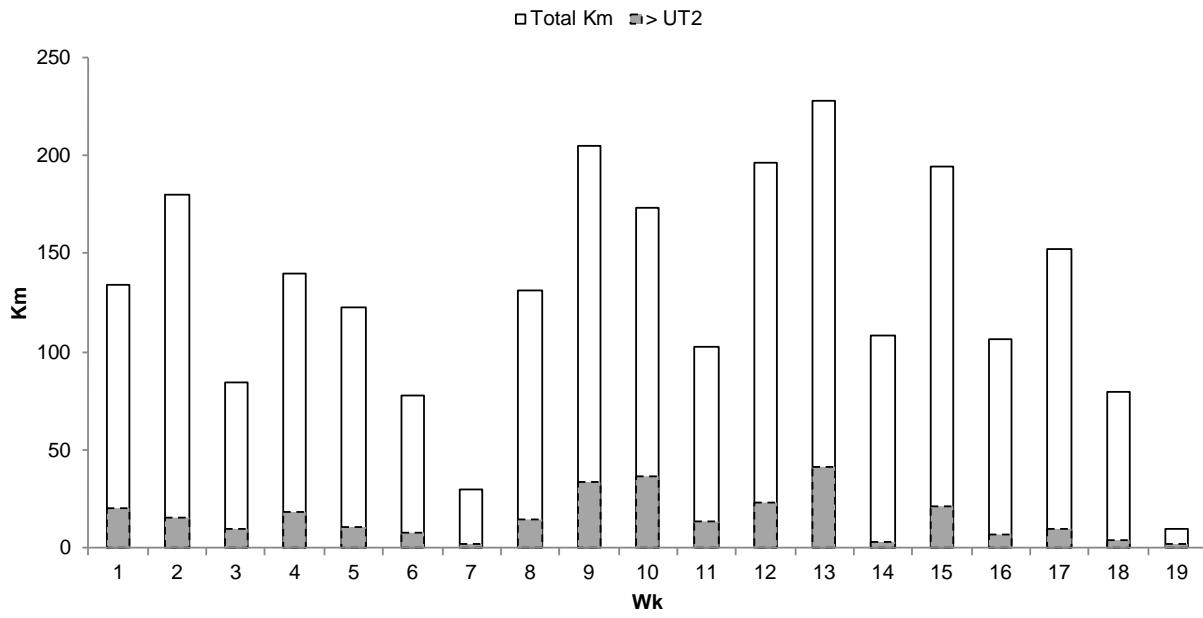
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450 Figures 1
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472 Figure 2

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475 Figure captions

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477 Figure 1. Weekly training volume measured in kilometres. Bars indicate total weekly training
478 including both on-water rowing and land-based rowing ergometry. Filled in (greyed out)
479 proportions of the bars indicate weekly training volume at an intensity greater than UT2 (above
480 lactate threshold).

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482 Figure 2: FORD, FORT and OSI measurements comparing healthy and ill individuals; and healthy
483 and injured individuals

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