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1 **Title**

2 Montmorency tart cherry (*Prunus cerasus* L.) supplementation accelerates recovery from
3 exercise-induced muscle damage in females.

4 **Running head**

5 Tart cherry juice and recovery in females.

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Abstract

Tart Montmorency cherry concentrate (MC) has been reported to attenuate the symptoms of exercise-induced muscle damage (EIMD) and to accelerate exercise recovery, which has been attributed to its high anti-inflammatory and antioxidant properties. Although these data are promising, there are no data regarding exclusively female populations. Therefore, the aim of this investigation was to examine the efficacy of MC on recovery following EIMD in females. In a randomised, double-blind, placebo-controlled study, twenty physically active females (mean ± SD age 19 ± 1 y; stature 167 ± 6 cm; body mass 61.4 ± 5.7 kg) consumed MC or a placebo (PL) for eight days (30 mL twice per day). Following four days of supplementation, participants completed a repeated-sprint protocol and measures of muscle soreness (DOMS), pain pressure threshold (PPT), limb girth, flexibility, muscle function, and systemic indices of muscle damage and inflammation were collected pre, immediately post (0 h) and 24, 48 and 72 h post-exercise. Time effects were observed for all dependent variables ($p<0.05$) except limb girth and high sensitivity C-reactive protein. Recovery of countermovement jump height was improved in the MC group compared to PL ($p=0.016$). There was also a trend for lower DOMS ($p=0.070$) and for higher PPT in the MC group at the rectus femoris ($p=0.071$). The data demonstrate that MC supplementation may be a practical nutritional intervention to help attenuate the symptoms of muscle damage and improve recovery on subsequent days in females.

Key words

Repeated sprint, muscle soreness, algometry, muscle function, sour cherry

43 **Introduction**

44 Initial muscle damage is thought to be caused by a combination of mechanical disruption to
45 the myofibrils and oxidative stress; the latter owing to an increase in the production of reactive
46 oxygen and nitrogen species (RONS) and nitric oxide (NO) derivatives which may exceed
47 antioxidant capacity (Powers & Jackson, 2008). Moreover, the secondary inflammatory
48 response to muscle injury involves the degradation of damaged muscle by immune cells which
49 release pro-inflammatory cytokines and further RONS and NO derivatives; exacerbating
50 muscle damage (Clarkson & Hubal, 2002). The role of RONS and NO derivatives in the
51 oxidative stress, inflammatory, and muscle damage responses which manifest during and
52 following exercise has raised substantial interest in antioxidant supplementation. Of particular
53 interest is tart Montmorency cherry concentrate (MC), which has been proposed to be an
54 effective recovery aid due to its high anti-inflammatory and antioxidant properties (Bell et al.,
55 2014; Bell et al., 2015; Keane et al., 2015a; Kirakosyan et al., 2015; Seeram et al., 2001; Wang
56 et al., 1999). Montmorency tart cherries and their derivatives contain numerous polyphenols
57 that include flavonoids; for example, the flavonol quercetin and anthocyanins (Kim et al., 2005;
58 Kirakosyan et al., 2009). Certainly, the polyphenolic compounds that MC contain result in
59 higher oxygen radical absorbance capacity (ORAC) values compared to several other
60 antioxidant beverages such as Concord grape, acai, and blueberry juice (Bell et al., 2013;
61 Howatson et al., 2010; Seeram et al., 2008).

62 To date, research in exercise and recovery paradigms has demonstrated that MC can improve
63 recovery from damaging bouts of exercise in isolated muscle groups by attenuating decrements
64 in muscle strength and/or soreness and pain (Bowtell et al., 2011; Connolly et al., 2006; Levers
65 et al., 2015). Additionally, following damaging running activity, research has identified MC to
66 be beneficial in reducing pain (Kuehl et al., 2010) and improving indices of inflammation,
67 oxidative stress, antioxidant status and muscle function (Howatson et al., 2010). More recently,

68 MC has also been shown to facilitate recovery following cycling (Bell et al., 2014; Bell et al.,
69 2015) and an adapted Loughborough Intermittent Shuttle Test (LIST) protocol (Bell et al.,
70 2016).

71 Collectively, these lines of investigation have application to athletic populations that would
72 benefit from reduced symptoms of muscle damage following strenuous activity. However, the
73 effects of MC beyond isolated muscle, running and cycling activity are limited, and
74 conceptually other sports and activities could benefit from this intervention. In addition, whilst
75 females have been included in mixed-sex populations (Howatson et al., 2010; Kuehl et al.,
76 2010), there are no data regarding exclusively female populations, largely due to the potential
77 for oestrogen to influence outcome variables (Kendall & Eston, 2002). A growing body of
78 evidence suggests that oestrogen has antioxidant properties (Tiidus et al., 2005; Wolf et al.,
79 2012) and may help to maintain muscle membrane integrity consequent to muscle damage. As
80 a result, the initial physiological stress and ensuing recovery associated with exercise-induced
81 muscle damage (EIMD) in females is likely to differ compared to male populations. Indeed,
82 recent evidence suggests that EIMD and recovery may differ between menstrual cycle phases
83 given the fluctuating oestrogen concentrations (Markofski & Braun, 2014). Moreover, given
84 their structural similarities to oestrogen, polyphenolic secondary plant metabolites (including
85 the flavonoids that MC contains) appear to exert oestrogenic effects (Miksicek, 1995), and thus
86 modulate and affect the bioavailability of endogenous oestrogens (Ward & Kuhnle, 2010). As
87 such, since oestrogen is thought to play a key role in the observed sex differences in EIMD,
88 currently the lack of studies investigating the supplementation of MC in a female only
89 population is surprising and warrants research.

90 Therefore, the aim of this investigation was to examine the efficacy of MC on recovery from
91 EIMD in females. It was hypothesised that indices of EIMD would be attenuated by the
92 consumption of MC.

93 **Methods**

94 **Participants**

95 Twenty physically active females (mean ± SD age 19 ± 1 y; stature 167 ± 6 cm; body mass
96 61.4 ± 5.7 kg; BMI 22.1 ± 1.9 kg·m^{-2}) were recruited from a university dance team and gave
97 written informed consent. The sample size was determined by completing a power analysis
98 (power=0.8, α=0.05) based on isometric strength data from Bowtell et al. (2011). This
99 determined a sample size of five in each group would provide statistical power above 80%,
100 with an alpha level of 0.05. Exclusion criteria were; epilepsy, bronchitis, severe asthma, cardiac
101 complaints, bacterial or viral infection in the 2 weeks preceding, injury or recovering from an
102 injury sustained in the preceding 4 weeks, pregnancy, food allergy relating to the study
103 supplements (as discussed with the investigator), or anything that may prevent them from
104 successfully completing the study that was described. Participants had been training in dance
105 regularly for 13 ± 4 years and were currently exercising for 8.3 ± 5 h per week; with no
106 significant differences between groups (independent samples *t*-test *p*=0.567 and *p*=0.598,
107 respectively). A single self-reported menstrual cycle questionnaire (as used previously by
108 Brown et al., 2016a) identified the current contraceptive use of participants; nine were using
109 an oral combination pill (all monophasic; *n*=6 in Montmorency cherry concentrate (MC) group
110 and *n*=3 in placebo (PL) group), six were using a progesterone only pill/implant/injection (*n*=3
111 in both MC and PL groups), and five were menstruating normally (*n*=1 in MC group and *n*=4
112 in PL group). This also estimated menstrual cycle phase and allowed testing days to be
113 assigned; all data collection took place during the early to mid-luteal phase, or where applicable
114 in the 14 days before a withdrawal bleed. For 24 h prior to, and for each of the testing days,
115 participants were asked to avoid strenuous exercise, alcohol, caffeine, nutritional supplements,
116 and any anti-inflammatory drugs or alternative treatments. Participants completed a weighed
117 food diary (analysed using dietary analysis software (Nutritics Ltd, Swords, Ireland)) and

118 activity log throughout all trial periods. Aside from the restrictions outlined previously,
119 participants were not restricted in their consumption of polyphenolic rich foods and were
120 instructed to consume their habitual diets. However, portions of foods thought to contain
121 antioxidants were totalled for each day and averaged across the experimental period (Howatson
122 et al., 2012; Howatson et al., 2010). The study was conducted according to the guidelines of
123 the Declaration of Helsinki and all experimental procedures were approved by the Faculty of
124 Health and Life Sciences Ethics Committee at the University of Northumbria.

125 **Experimental protocol**

126 Participants were allocated to either tart Montmorency cherry concentrate (MC; *n*=10) or
127 placebo (PL; *n*=10) supplementation in a double-blind manner using stratified randomisation
128 to ensure that groups were matched and counterbalanced for muscle function. A pre-
129 supplementation blood sample (baseline) was taken in order to detect any changes in total
130 creatine kinase (CK) and high sensitivity C-reactive protein (hsCRP) with preload
131 supplementation. Participants fasted for ≥10 h prior to each visit, except for water (consumed
132 *ad libitum*) and the morning supplement. On arrival at the laboratory, baseline measures of
133 dependant variables were recorded. Participants completed the exercise protocol, and after a 2
134 min rest, measurement of dependent variables was repeated. Before leaving the laboratory,
135 participants were reminded to consume a supplement prior to their evening meal.
136 Supplementation and measurement of dependent variables were then repeated at the same time
137 of day (± 1 h) for the following 3 days (24, 48 and 72 h post EIMD).

138 **Supplementation**

139 Participants consumed their habitual diets during all trial periods. Participants were provided
140 with eight days of supplementation along with instructions on ingestion frequency and timing.
141 This period was for four days prior to muscle-damaging exercise, the day of exercise, and for

142 three days of recovery. The daily dose was two servings of the MC or PL; one dose taken prior
143 to breakfast (or 1-2 h prior to laboratory visits), and one dose prior to evening meal (except for
144 the final day where only one supplement was consumed before the final visit). This is based on
145 previous work showing a positive effect on recovery following strenuous exercise (Bell et al.,
146 2016; Bell et al., 2014; Bell et al., 2015).

147 The MC beverage was prepared with 30 mL of concentrate (CherryActive, Sunbury, UK)
148 diluted in 100 mL of water. According to the manufacturer's information, a 30 mL dose of
149 concentrate is equivalent to approximately 90 whole cherries and has been previously reported
150 to contain a total anthocyanin content of 73.5 $mg \cdot L^{-1}$ of cyanidin-3-glucoside, a total phenolic
151 content of 178.8 gallic acid equivalent$\cdot L^{-1}$ and an antioxidant capacity (TEAC) of 0.58 trolox
152 equivalents$\cdot L^{-1}$ (Keane et al., 2016). The PL was prepared with 25 mL of a synthetically
153 derived fruit flavoured concentrate with negligible phytochemical content (Kia-Ora, Uxbridge,
154 UK) in 100 mL of water and was fortified with flavourless maltodextrin (Myprotein,
155 Manchester, UK) and flavourless whey protein powder (Arla Foods, Amba, Denmark). This
156 was in order to match test beverages as closely as possible for volume (130 ml), consistency,
157 colour, and macronutrient (24.5 g carbohydrate and 1.1 g protein) and energy content (102 kcal
158 and 103 kcal for the MC and PL beverages, respectively).

159 **Exercise protocol**

160 Following a standardised warm up, participants completed a repeated-sprint protocol which
161 comprised 15 x 30 m maximal sprints with a rapid 10 m deceleration phase, each separated by
162 60 s rest (Brown et al., 2016a; Brown et al., 2016b; Howatson & Milak, 2009; Keane et al.,
163 2015b). Rate of perceived exertion (RPE; Borg, (1982)) and heart rate (HR; Model RS-400,
164 Polar, Kempele, Finland) were collected after each sprint effort. Sprint times were recorded

165 using timing gates (Brower telemetric timers, Brower timing systems, Draper, USA) to
166 determine total sprint time, mean sprint time, and rate of fatigue (Fitzsimons et al., 1993).

167

168 **Dependant variables**

169 ***Muscle soreness***

170 Subjective ratings of muscle soreness (DOMS) were measured using a 200 mm visual analogue
171 scale. Participants were required to indicate the level of perceived active lower limb soreness
172 felt during a 90^0 squat. Pain pressure threshold (PPT) was measured with a digital algometer
173 (Model FDX, Wagner Instruments, Greenwich, USA) at three muscle locations on the right
174 leg; the rectus femoris (RF), the vastus lateralis (VL), and medial head of the gastrocnemius
175 (GM) (Clifford et al., 2016). To determine PPT, participants were asked to verbally indicate
176 when the pressure applied to the muscle (at an approximate rate of 5 $N \cdot s^{-1}$) became too
177 uncomfortable to tolerate. Intra-trial and inter-trial percentage coefficient of variation (%CV)
178 were <8% for all locations.

179 ***Limb girth***

180 An anthropometric tape measure (Bodycare Products, Warwickshire, United Kingdom) was
181 used to determine calf (measured at its largest girth at baseline) and mid-thigh (located as
182 midway between the inguinal fold and the superior border of the patella) girths of the right leg.
183 Calf and mid-thigh girth intra-examiner %CVs were <1%.

184 ***Flexibility***

185 Hamstring stiffness and flexibility was measured using the sit and reach test. The knees were
186 fully extended with the feet together against the sit and reach box. Participants were instructed

to stretch as far as possible (but not to the point of pain) and to hold their ‘best stretch’ for approximately 2 s (American College of Sports Medicine, 2013) (recorded to the nearest 0.5 cm). Intra-trial and inter-trial %CV were <5%.

Muscle function

Participants completed three countermovement jumps (CMJ) and three drop jumps (for measurement of reactive strength index (RSI)) using a light timing system (Optojump, Microgate, Bolzano, Italy), keeping their hands on their hips throughout. For CMJ, participants were asked to squat down and jump vertically and maximally. For RSI (jump height (cm) ÷ ground contact time (s)), participants were instructed to drop from a height of 30 cm and upon landing, to perform a two-footed jump maximally with minimum contact time. Each effort was separated by 60 s rest and the peak CMJ and RSI were used for analysis. Intra-trial and inter-trial %CV were both <4% and <12% for CMJ and RSI respectively.

Maximum voluntary isometric contraction (MVC) of the right knee extensors was measured using a strain gauge (MIE Digital Myometer, MIE Medical Research Ltd, Leeds, UK). While in a seated position, the knee joint angle was standardised at 90^0 of flexion before each contraction. The peak force (Newtons, N) of three MVCs (of 3 s duration interspersed with 30 s rest) was used for analysis. Intra-trial and inter-trial %CV were <4%.

Participants completed a single maximal effort 30 m sprint, and sprint time was recorded (Brower telemetric timers, Brower timing systems, Draper, USA). Both intra-trial and inter-trial %CV were <2%.

Blood sampling and analysis

209 Blood samples (10 mL) were collected via venepuncture from the antecubital fossa area into
210 serum gel vacutainers. Samples were rested at room temperature for 20 min, and centrifuged
211 for 15 min (4˚C) at 3000 RCF (Allegra X-22 Centrifuge, Beckman Coulter, Bucks, UK).
212 Aliquots of serum were stored at -80°C for later analysis of CK and hsCRP; determined
213 spectrophotometrically using an automated system (Roche Modular, Roche Diagnostics,
214 Burgess Hill, UK). Due to technical issues, 5 blood samples (<5%) were not collected. When
215 lower detection limits were not reached for hsCRP, the lowest detectable concentration was
216 used (0.15 $mg \cdot L^{-1}$). Inter-assay and intra-assay %CV for both total CK and hsCRP were <9%.

217 **Statistical analysis**

218 For the purpose of data analysis, all dependant variables except for DOMS, CK and hsCRP are
219 expressed as a percentage relative to pre muscle damage values to account for inter-individual
220 variability. Statistical software (IBM Statistical Package for Social Sciences (SPSS) V22 IBM,
221 Armonk, USA) was used for inferential analysis and statistical significance accepted at the
222 $p \leq 0.05$ level *a priori*. Mixed factor repeated-measures analysis of variance were performed for
223 each dependent variable. Mauchly's test assessed the sphericity of the data and, where
224 appropriate, violations were corrected using the Greenhouse–Geisser correction. Significant
225 main effects were analysed using the Least Significant Difference test for adjustment for
226 multiple comparisons. Paired samples *t*-tests were conducted to assess differences between
227 total CK and hsCRP levels pre-supplementation (baseline) and pre-exercise, in order to detect
228 any changes in systemic indices with preload supplementation. Independent samples *t*-tests
229 were conducted on peak HR, peak RPE, fatigue, and total and mean sprint time to examine
230 differences in exercise intensity during the repeated-sprint protocol between groups. Where
231 appropriate, Cohen's *d* effect sizes (ES) were calculated with the magnitude of effects
232 considered small (0.2), moderate (0.5) and large (>0.8).

233

234 **Results**

235 There were no differences in the total energy intake and macronutrient intake presented as a
236 percentage of total energy intake (all $p>0.05$, ES>0.47), and the number of portions of foods
237 containing antioxidants consumed by participants in both treatment groups (6 ± 2 in both MC
238 and PL groups; p=0.731) during the supplementation period. Following all data collection
239 periods, only *n*=4 participants correctly identified which supplement they had consumed. There
240 were no differences between MC and PL groups for total (80.74 ± 4.02 vs 81.69 ± 3.67 s) and
241 mean (5.38 ± 0.27 vs 5.45 ± 0.24 s) sprint time, fatigue (5.23 ± 2.02 vs 4.54 ± 2.16%), peak
242 HR (176 ± 15 vs 178 ± 8 bpm), and peak RPE (17 ± 2 vs 18 ± 1) during the repeated-sprint
243 protocol (all $p>0.05$); demonstrating that the exercise stimulus was comparable between
244 groups. All raw data not illustrated in figures are presented in Table 1.

245 Muscle soreness increased post-exercise ($p<0.001$), peaking at 24 h and remained elevated
246 throughout recovery in both groups (Figure 1); however, there was a trend and moderate effect
247 for lower DOMS in the MC group ($p=0.070$, ES=0.58). At all three locations, PPT was reduced
248 post-exercise (all $p<0.001$), reached lowest levels at 24 h and then increased throughout
249 recovery. There were no group differences in PPT at the VL or GM, but a trend and moderate
250 effect for higher PPT in the MC group at the RF was observed ($p=0.071$, ES=0.59).

251 Thigh and calf girths were unaffected post-exercise and there were no differences between
252 treatment groups (all $p>0.05$). Flexibility was reduced for 48 h post-exercise but returned to
253 baseline levels at 72 h in both groups ($p=0.022$) with no group differences.

254 All measures of muscle function (CMJ, RSI, MVC and 30 m sprint time) were reduced post-
255 exercise and progressively recovered throughout recovery (all $p<0.05$). While recovery of these

256 measures appeared to accelerate with MC, a group effect was only evident with CMJ (p=0.016,
257 ES=0.66) (Figure 2).

258 Total CK and hsCRP concentrations were not different between baseline and pre-exercise time-
259 points for both MC and PL groups (all p>0.05). Both groups experienced an increase in
260 circulating CK, which peaked 24 h post-exercise and remained elevated for 72 h post-exercise
261 (p<0.001) with no differences between groups (Figure 3). Circulating hsCRP was unaffected
262 by exercise and was not different between groups.

263

264 **Discussion**

265 This study sought to examine the efficacy of 8-day MC supplementation on recovery from
266 EIMD in females. The data demonstrate that MC supplementation accelerated the recovery of
267 CMJ and was associated with trends of reduced muscle soreness.

268 The clear acceleration in recovery of CMJ following MC supplementation supports a recent
269 study demonstrating an improvement in CMJ (Bell et al., 2016), and a number of studies
270 demonstrating an accelerated recovery in other measures of muscle function with MC
271 consumption (Bell et al., 2015; Bowtell et al., 2011; Connolly et al., 2006; Howatson et al.,
272 2010). The improvement of CMJ with MC may be explained by a protection against oxidative
273 injury to the type II fibres recruited for such activity. Eccentric exercise is thought to
274 preferentially damage type II muscle fibres (Macaluso et al., 2012) and this has implications
275 on the muscles' force-generating capacity (Byrne et al., 2004). Interestingly, evidence suggests
276 that mitochondrial ROS production and/or release is potentiated in type II fibres (Anderson &
277 Neufer, 2006) and the activity of endogenous antioxidant enzymes including superoxide
278 dismutase (SOD) (Criswell et al., 1993; Powers et al., 1994) and glutathione peroxidase (GPX)

279 (Lawler et al., 1994; Powers et al., 1994) are lower compared with type I fibres in rodent
280 models. During periods of increased oxidant production (for instance intense exercise), both
281 enzymatic and non-enzymatic antioxidants collectively provide some protection to muscle
282 fibres from oxidative injury (Powers & Jackson, 2008). Therefore, conceptually the
283 supplementation of non-enzymatic antioxidants contributed to the free radical scavenging
284 capacity of type II fibres and facilitated recovery of CMJ. However, the functional measures
285 in the present investigation all require type II fibre recruitment, so intuitively, we would have
286 expected the measures to be equally affected by MC. Indeed, the lack of an accelerated
287 recovery of MVC with MC is in contrast to a number of previous studies (Bell et al., 2015;
288 Bowtell et al., 2011; Connolly et al., 2006; Howatson et al., 2010). Certainly, the role of MC
289 in accelerating the recovery of muscle function in this population following EIMD in the
290 current study remains unclear.

291 Supplementation with MC resulted in a moderate effect for reductions in DOMS and increases
292 in PPT at the RF; which could have played a role in the observed improvements in CMJ. This
293 is in line with a number of investigations reporting reduced soreness and pain with MC
294 supplementation (Bell et al., 2016; Connolly et al., 2006; Kuehl et al., 2010; Levers et al.,
295 2015). However, these reductions previously reported have not always been accompanied with
296 improvements in muscle function, and vice versa (Bell et al., 2015; Bowtell et al., 2011; Levers
297 et al., 2015), and others have found no reduction in DOMS with MC consumption (Bell et al.,
298 2015; Bowtell et al., 2011; Howatson et al., 2010). The inconsistencies in the literature could
299 be explained by the disparities in exercise protocol employed. Indeed, muscle soreness has
300 been associated with increases in inflammation following exercise (Kraemer et al., 2004). In
301 the current investigation, hsCRP was not different between groups, and limb girth (an indirect
302 measure of inflammation, swelling and oedema (Smith, 1991)) was unaffected by the exercise.
303 Compared to marathon running (Howatson et al., 2010) and high intensity cycling exercise

304 (Bell et al., 2015), where CRP has been shown to increase 24 and 48 h post-exercise, the
305 repeated-sprint protocol does not appear to induce a large inflammatory response. It is
306 conceivable that the exercise stimulus was insufficient to detect larger magnitudes of change
307 in soreness and PPT with MC. Similarly, the CK values elicited by the exercise protocol in the
308 current study are lower than other damaging protocols, suggesting a moderate muscle damage
309 response, and likely influencing the ability to demonstrate differences. In addition, females
310 have been shown to demonstrate lower basal circulating concentrations of CK, and a lower CK
311 response following exercise compared to males (Wolf et al., 2012), owing in part to the
312 antioxidant properties of oestrogen (Tang, Abplanalp, Ayres, & Subbiah, 1996).

313 The data which demonstrate no group differences in hsCRP and CK do not wholly support the
314 literature which traditionally suggests attenuated symptoms of EIMD with MC is attributable
315 to reduced muscle damage and inflammation; at least following repeated-sprint exercise in
316 females. However, the current study was limited by a lack of measurement of oxidative stress
317 and antioxidant capacity. Indeed, some have identified differences in oxidative stress and
318 antioxidant status following strenuous exercise with supplementation of MC compared to PL
319 (Bell et al., 2014; Bowtell et al., 2011; Howatson et al., 2010). It is possible that this is
320 magnified in females given that oestrogen is also thought to have antioxidant properties (Tiidus
321 et al., 2005; Wolf et al., 2012) as oestrogens, similar to vitamin E, display a hydroxyl group on
322 their phenolic ring (Tiidus et al., 2001). It is thought that oestrogen donates the hydrogen atom
323 to lipid peroxyl radicals, limiting lipid peroxidation in the cell membrane (Kendall & Eston,
324 2002). Perhaps enhanced antioxidant status and redox balance may have contributed to the
325 improvements in CMJ and trends in reduced soreness observed with MC in this study. Future
326 research should include measurement of a variety of systemic indices to provide greater insight
327 into specific mechanisms influencing improved muscle function and pain with MC. Moreover,
328 more accurate determination of menstrual cycle phase (urine ovulation prediction tests for

329 example) and measurement of concentrations of oestrogen (particularly of oestradiol) in female
330 participants is warranted to assist with the interpretation of results.

331 This study demonstrated that 8-day MC supplementation alongside a habitual diet improved
332 recovery of CMJ and tended to lower muscle soreness compared to PL. This research adds to
333 the existing body of knowledge whilst providing new information for the potential application
334 of MC to wider groups. In particular, it would appear that female populations might benefit
335 from this nutritional intervention to help attenuate the symptoms of EIMD and improve
336 recovery on subsequent days.

337

338

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471

472 **Tables**

473

474 Table 1. Values for dependent variables in response to muscle-damaging exercise, mean ±
475 SD.

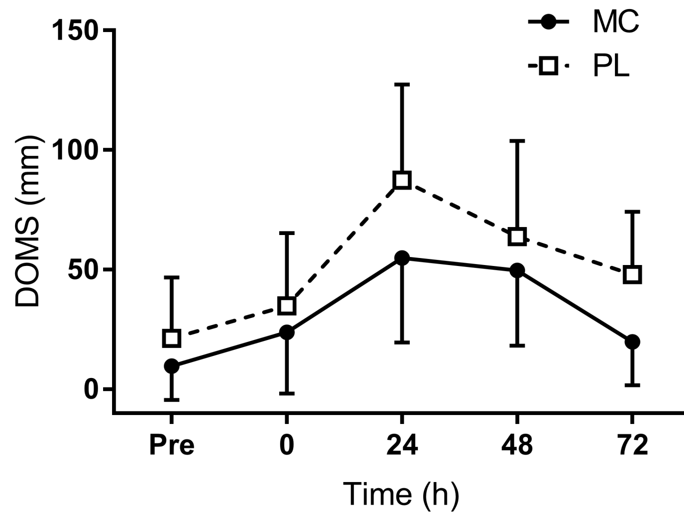
476

		Time post muscle-damaging exercise (h)				
Variable	**Group**	**Pre**	**0**	**24**	**48**	**72**
RF PPT, N	MC	48.1 ± 8.8	46.9 ± 11.6	42.5 ± 10.8	48.7 ± 12.9	56.0 ± 17.5
	PL	35.2 ± 12.4	30.8 ± 10.6	25.9 ± 6.9	28.7 ± 10.0	35.1 ± 8.8
VL PPT, N	MC	42.6 ± 9.8	47.3 ± 18.0	34.9 ± 13.6	41.6 ± 12.6	53.3 ± 23.3
	PL	30.2 ± 10.1	29.7 ± 11.8	25.5 ± 7.2	28.5 ± 10.2	32.5 ± 10.5
GM PPT, N	MC	41.4 ± 11.2	42.4 ± 15.5	31.8 ± 8.8	39.4 ± 9.4	50.2 ± 12.2
	PL	27.3 ± 12.1	25.0 ± 12.1	21.9 ± 8.7	24.9 ± 10.2	30.1 ± 9.9
Thigh girth, cm	MC	50.1 ± 3.1	50.4 ± 3.0	50.3 ± 3.0	50.3 ± 3.0	50.3 ± 2.8
	PL	50.6 ± 2.2	50.9 ± 2.6	51.0 ± 2.4	51.0 ± 2.5	50.9 ± 2.4
Calf girth, cm	MC	36.1 ± 2.3	36.2 ± 2.4	36.1 ± 2.4	36.1 ± 2.3	36.2 ± 2.2
	PL	36.2 ± 2.1	36.1 ± 1.9	36.2 ± 2.0	36.2 ± 2.2	36.2 ± 2.0
Flexibility, cm	MC	29.1 ± 5.4	28.3 ± 6.4	25.0 ± 7.2	26.4 ± 9.3	29.0 ± 7.8

	PL	20.3 ± 9.0	17.7 ± 9.0	14.5 ± 6.1	17.4 ± 8.3	17.6 ± 9.0
RSI, cm·s^{-1}	MC	102.8 ± 22.5	88.6 ± 21.5	97.2 ± 28.5	99.0 ± 22.2	107.0 ± 27.1
	PL	81.5 ± 17.6	74.7 ± 13.7	72.0 ± 13.8	73.0 ± 14.4	80.2 ± 20.1
MVC, N	MC	394.3 ± 59.3	347.1 ± 82.5	362.7 ± 87.1	381.3 ± 87.2	376.5 ± 73.6
	PL	392.2 ± 89.4	354.1 ± 72.7	355.4 ± 73.8	361.8 ± 70.2	375.9 ± 63.9
30 m sprint time, s	MC	5.32 ± 0.35	5.42 ± 0.36	5.39 ± 0.31	5.45 ± 0.29	5.37 ± 0.40
	PL	5.28 ± 0.26	5.46 ± 0.30	5.37 ± 0.27	5.58 ± 0.41	5.43 ± 0.29
hsCRP, mg·L^{-1}	MC	1.63 ± 1.99	1.79 ± 1.87	2.15 ± 0.24	2.13 ± 1.97	1.56 ± 1.35
	PL	1.81 ± 1.93	1.80 ± 1.87	1.73 ± 1.73	1.29 ± 1.14	1.71 ± 1.31

477 MC, Montmorency cherry group (*n*=10); PL, placebo group (*n*=10); Pre, pre-exercise; RF,
478 rectus femoris; VL, vastus lateralis; GM, medial head of the gastrocnemius; PPT, pain pressure
479 threshold; CMJ, countermovement jump; RSI, reactive strength index; MVC, maximal
480 voluntary isometric contraction; hsCRP, high sensitivity C-reactive protein.

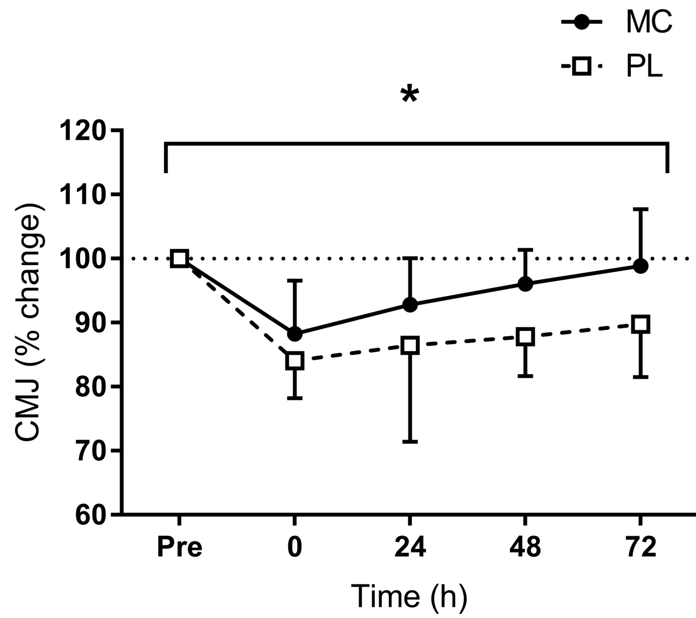
481



482

483 Figure 1. Muscle soreness (DOMS) post exercise-induced muscle damage in the Montmorency

484 cherry (MC) (*n*=10) and placebo (PL) (*n*=10) groups. Values presented as mean ± SD.



485

486 Figure 2. Percentage relative to pre-exercise (Pre) countermovement jump height (CMJ) post
487 exercise-induced muscle damage in the Montmorency cherry (MC) (*n*=10) and placebo (PL)
488 (*n*=10) groups. Values presented as mean ± SD. *denotes significantly higher CMJ in MC
489 group. Significance at $p<0.05$.

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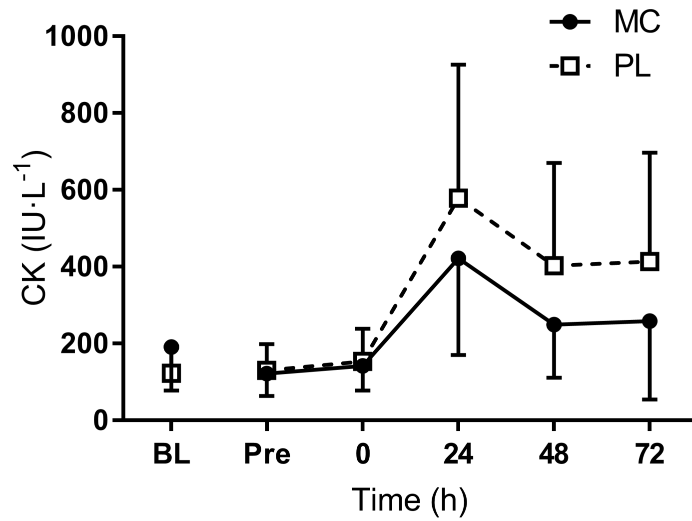

491

492 Figure 3. Total creatine kinase (CK) at baseline pre-supplementation (BL), before (Pre) and
493 post exercise-induced muscle damage in the Montmorency cherry (MC) (*n*=10) and placebo
494 (PL) groups (*n*=10). Values presented as mean ± SD.