Montmorency Cherry Juice Reduces Muscle Damage Caused by Intensive Strength Exercise

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ABSTRACT

BOWTELL, J. L., D. P. SUMNERS, A. DYER, P. FOX, and K. N. MILEVA. Montmorency Cherry Juice Reduces Muscle Damage Caused by Intensive Strength Exercise. *Med. Sci. Sports Exerc.*, Vol. 43, No. 8, pp. 1544–1551, 2011. **Purpose**: Montmorency cherries contain high levels of polyphenolic compounds including flavonoids and anthocyanins possessing antioxidant and anti-inflammatory effects. We investigated whether the effects of intensive unilateral leg exercise on oxidative damage and muscle function were attenuated by consumption of a Montmorency cherry juice concentrate using a crossover experimental design. **Methods**: Ten well-trained male overnight-fasted athletes completed two trials of 10 sets of 10 single-leg knee extensions at 80% one-repetition maximum. Trials were separated by 2 wk, and alternate legs were used in each trial. Participants consumed each supplement (CherryActive® (CA) or iso-energetic fruit concentrate (FC)) for 7 d before and 48 h after exercise. Knee extension maximum voluntary contractions (MVC) were performed before, immediately after, and 24 and 48 h after the damaging exercise. Venous blood samples were collected at each time point, and serum was analyzed for creatine kinase (CK) activity, nitrotyrosine, high-sensitivity C-reactive protein, total antioxidant capacity, and protein carbonyls (PC). Two-way repeated-measures ANOVA were used for statistical analysis of the data. **Results**: MVC force recovery was significantly faster (24 h: CA 90.9% ± 4.2% of initial MVC vs FC 84.9% ± 3.4% of initial MVC; 48 h: CA 92.9% ± 3.3% of initial MVC vs FC 88.5% ± 2.9% of initial MVC (mean ± SEM); $P < 0.05$) after CA than FC consumption. Only serum CK and PC increased significantly from baseline, peaking 24 h after exercise ($P < 0.001$). The exercise-induced increase in CK activity was not different between trials. However, both the percentage (24 h after: CA 23.8% ± 2.9% vs FC 82.7% ± 11.7%; $P = 0.013$) and absolute (24 h after: CA 0.31 ± 0.03 nmol·mg^{-1} protein vs FC 0.60 ± 0.08 nmol·mg^{-1} protein; $P = 0.079$) increase in PC was lower in CA than FC trials. **Conclusions**: Montmorency cherry juice consumption improved the recovery of isometric muscle strength after intensive exercise perhaps owing to the attenuation of the oxidative damage induced by the damaging exercise. **Key Words:** ANTIOXIDANT, PROTEIN CARBONYL, OXIDATIVE DAMAGE, MUSCLE STRENGTH

Novel or unaccustomed intensive exercise results in muscle damage, the symptoms of which are long-lasting (2–5 d) reductions in muscle strength and muscle soreness. The decreased muscle force–generating capacity has been attributed to myofibrillar disruption and structural damage to the muscle as evidenced by the increase in the blood concentration of large intracellular muscle proteins such as creatine kinase (CK) and lactate dehydrogenase. These changes have been directly associated with increased permeability of the damaged sarcolemma (18). Impaired excitation–contraction coupling related to altered intracellular calcium homeostasis has also been implicated in the reduced functional capacity of muscle after eccentric exercise. This may be due to damage to the ryanodine receptors of the sarcoplasmic reticulum resulting in elevated intracellular calcium ion concentration (for review, see Westerblad et al. (38)) and altered membrane potentials. Increased intracellular calcium may also contribute to muscle damage through activation of calcium-dependent proteolytic pathways and increased muscle protein degradation (36).

The exact mechanisms by which muscle damage occurs are not yet fully understood but are thought to involve both mechanical and metabolic pathways, with the relative contribution presumably varying according to the mode, intensity, and duration of exercise (for reviews, see Close et al. (9), Howatson and van Someren (15), Powers and Jackson (28), Proske and Morgan (30), and Smith et al. (34)). The initial phase of damage during exercise is suggested to occur because of both the mechanical forces to which the muscle fibers are exposed and oxidative stress due to exercise-induced increases in reactive oxygen species (ROS) and nitric oxide (NO) derivatives that exceed the antioxidant defense capacity. Acute high-intensity resistance exercise (>60% one-repetition maximum (1RM)) has been shown to induce increases in lipid hydroperoxides and protein carbonyls (PC), blood markers of oxidative damage (16). A

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second phase of damage occurs owing to the inflammatory response to muscle injury (for review, see Smith et al. (34)). This is characterized by neutrophil migration to muscle occurring within several hours of exercise and lasting for up to 24 h and the presence of macrophages within damaged muscle from 24 h to up to 14 d after exercise. These immune cells contribute to the degradation of damaged muscle by releasing ROS and NO derivatives as well as proinflammatory cytokines.

As a consequence of the apparent role for ROS and NO derivatives in muscle damage, there has been considerable interest in the efficacy of antioxidant supplements such as vitamins C and E in ameliorating exercise-induced muscle damage. However, evidence is equivocal, with some studies showing that vitamin C and/or E supplementation decreased eccentric exercise–induced muscle damage (decreased CK and delayed onset muscle soreness (5); decreased CK (24); decreased malondialdehyde and PC (11)), but others found no change in CK (2) or even increased CK and lactate dehydrogenase (LDH) (8). The conflicting evidence can be attributed in part to the variation in exercise mode, intensity, and duration used to induce damage as well as the dose and duration of vitamin supplementation.

More recently, there has been interest in the potential of fruit-derived phytochemicals with both antioxidant and anti-inflammatory properties to improve recovery from exercise-induced muscle damage (10,13,35). Connolly et al. (10) found that consumption of Montmorency (tart) cherry juice for 4 d before repeated single-arm elbow flexor eccentric contractions resulted in a quicker recovery of isometric force generating capacity than in a placebo trial; however, no biochemical measures of muscle damage were taken. Howatson et al. (13) found quicker recovery of knee extensor maximal isometric force after running a marathon when participants consumed Montmorency cherry juice rather than placebo for 6 d before the race. Although there was no difference in serum CK or LDH between trials, markers of inflammation (interleukin 6 (IL-6) and C-reactive protein (CRP)) and oxidative damage (thiobarbituric acids reactive substances (TBARS)) were significantly lower in the cherry juice trial. In similar fashion, Trombold et al. (35) found that pomegranate-derived ellagitannin consumption improved recovery of elbow flexor isometric strength after repeated eccentric contractions in resistance exercise–naive participants. However, there was no difference between trials in serum markers of muscle damage (CK and myoglobin) or inflammation (IL-6 and CRP). It seems, therefore, that fruit-derived polyphenolic compounds have the potential to improve recovery from damaging exercise, although the mechanism is, as yet, unclear in part because there is only limited published evidence available. It is not clear how far these existing findings can be generalized. Although cherry juice consumption has been shown to enhance functional recovery from elbow flexor eccentric exercise in recreationally active participants (10), it is unclear whether such a supplement would be similarly effective for knee extensor recovery of well-trained individuals who are relatively resistant to damage. Howatson et al. (13) quantified the effects of cherry juice consumption on markers of oxidative damage and inflammation, as well as muscle function after marathon exercise. However, these data are not available for recovery from resistance exercise where at least the initial phase of damage will be more mechanical in nature. Therefore, in the present study, the effect of supplementation with Montmorency cherry juice concentrate on functional recovery from intensive knee extensor resistance exercise in well-trained individuals was investigated in parallel with changes in serum markers of oxidative damage and inflammation.

METHODS

Participants. Ten well-trained male participants (age = 27.8 ± 1.6 yr, single-leg 1RM = 73 ± 4 kg, weight = 81.3 ± 4.3 kg, height = 1.76 ± 0.03 m) completed the study. Participants all competed in high-intensity intermittent sports (rugby, football, or taekwondo) and regularly performed resistance training. The study was approved by the local university ethics committee and was conducted in compliance with the World Medical Association's Declaration of Helsinki (2008). All participants were informed verbally and in writing of the experimental procedures and associated risks before completing a medical health questionnaire and giving their written informed consent. Exclusion criteria included cardiorespiratory and neuromuscular problems and acute knee/ankle injuries and pain.

Baseline strength testing. Participants were fully familiarized with completing single-leg knee extension maximum voluntary contractions (MVC), as well as the other experimental procedures and measures. During the week before the first trial, the single-leg knee extension 1RM was determined for each leg. Subjects were seated on the knee extension machine (TechnoGym UK Ltd., Bracknell, Berkshire, United Kingdom), and the backrest and bar levels were adjusted for each subject and kept constant during all subsequent testing. A lapbelt restraint was used to exclude contribution from the hip musculature during the knee extension exercise. Subjects performed a standard ramp test to identify the 1RM (12). Each weight lift was assessed by the investigator and considered successful if performed with proper technique, within the metronome-guided time interval (2.5 s) and going through the full range of knee motion of the exercise (0.7 rad). The maximum weight lifted was identified as the 1RM. Verbal encouragement was given to each participant throughout.

Experimental design. Participants completed two main trials separated by a 2-wk washout period (Fig. 1). Participants consumed 30 mL twice per day for 10 d of either Montmorency cherry juice concentrate (CherryActive® (CA)) or an isoenergetic fruit concentrate placebo (FC). On each occasion, participants completed the single-leg intensive knee extensor training session on day 8 of the supplementation period, with different legs used for each trial to minimize

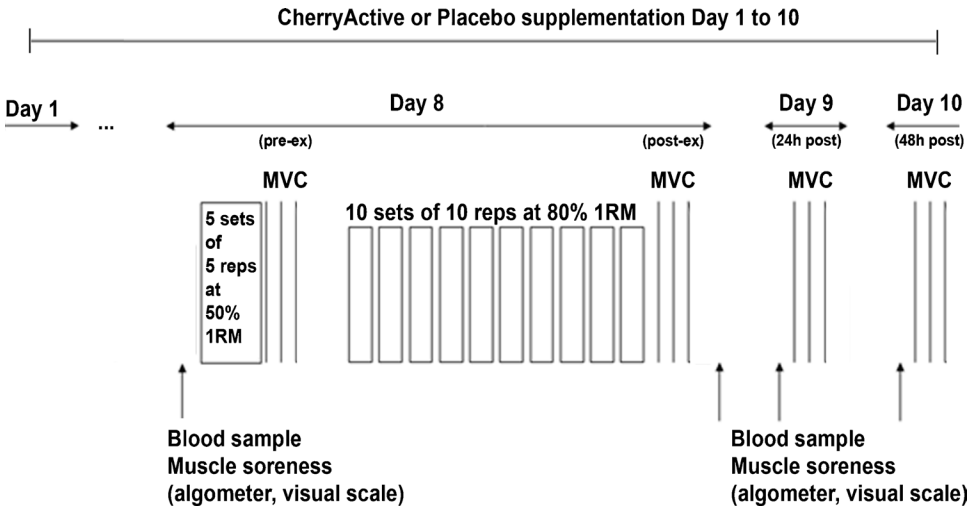

FIGURE 1—Experimental protocol.

any repeated bout effect. Trials were allocated by systematic rotation to counterbalance the study for trial order and leg dominance, with participants and investigators blinded to treatment. Participants were instructed to consume their habitual diet and continue normal training activities for the first 5 d of the supplementation period but to refrain from strenuous physical activity for 48 h before and after the intensive exercise protocol. Participants recorded their diet for 48 h before the first main trial and then repeated this diet before the second trial.

Nutritional supplements. Each 30-mL serving of the Montmorency cherry juice concentrate contained 20-g CHO and provided 96 kcal. High performance liquid chromatography (HPLC) analysis (6) of the concentrate performed by Atlas Bioscience, Inc. (Tucson, AZ), found that total anthocyanin content was 9.117 $mg \cdot mL^{-1}$, with malvidin (4.696 $mg \cdot mL^{-1}$) and cyanidin (3.346 $mg \cdot mL^{-1}$) being the most prevalent. The typical oxygen radical absorbance capacity (ORAC) of CA is 275 $mmol \cdot L^{-1}$ Trolox equivalents (Brunswick Laboratories, Southborough, MA), which compares favorably with reported ORAC values (9.1–31.7 $mmol \cdot L^{-1}$ Trolox equivalents) for other commercially available juices such as grape, pomegranate acai, blueberry, and cranberry (33) and competitor Montmorency cherry juice products (CherryPharm, Inc., Geneva, NY; 55 $mmol \cdot L^{-1}$ Trolox equivalents) (13).

The placebo was an isoenergetic synthetically derived fruit concentrate that was designed to have similar consistency and color but without the phytochemical content of the cherry juice concentrate. Participants were instructed to take one serving in the morning and one in the afternoon after training.

Experimental protocol. Participants arrived at the laboratory after an overnight fast and after weighing rested in the supine position while a 10-mL resting blood sample was taken from an antecubital vein (Fig. 1). Participants were then seated on the knee extension machine, and pressure pain threshold over vastus lateralis, rectus femoris, and vastus medialis muscles was measured using an algometer (Wagner Instruments, Inc., Greenwich, CT) as an index of muscle soreness. Measurements were made by the same investigator for each subject on each occasion.

Participants then completed a warm-up consisting of three sets of five repetitions of single-leg knee extension exercise at 50% 1RM each separated by a 2-min rest period. Participants then completed three single-leg knee extension MVC at 70° knee flexion angle (quadriceps muscle stretched) and separated by 2 min of rest. After a 5-min rest, participants completed 10 sets of 10 single-leg knee extensions at 80% of their 1RM with elongated eccentric phase (lasting 3 s); each set was separated by 2 min of rest. If subjects were unable to maintain the workload, the load was decreased by 10%, and this was then matched during the second trial. After completing the 10 sets, participants were asked to repeat the three MVC, with the first performed immediately after the last set; thereafter, each MVC was separated by 2 min of rest. Participants received verbal encouragement to perform maximally throughout the exercise protocol. Pressure pain threshold was reassessed after completion of the MVC, and a further blood sample was obtained 10 min after completion of the last MVC.

Participants then returned to the laboratory at the same time of day 24 and 48 h later in an overnight fasted state. On each occasion, a 10-mL resting blood sample was obtained from an antecubital vein while participants rested in a supine position, and pressure pain threshold was reassessed. Participants then repeated the warm-up and, after 2 min of rest, completed three single-leg knee extension MVC separated by 2 min of rest.

For the second main trial, the protocol was repeated, but the exercise was performed with the contralateral leg to minimize the repeated bout effect. The experimental design was counterbalanced for trial order and for leg dominance.

Blood analysis. At each time point, fasting blood samples were collected into a 10-mL vacutainer containing no anticoagulant and left at room temperature for 1 h and then

centrifuged at 4500 rpm for 15 min at 4°C. Serum samples were aliquoted into Eppendorf tubes and stored at −80°C until analysis.

Serum samples were analyzed for CK, high-sensitivity CRP (hsCRP), total nitrotyrosine, PC, and total antioxidant capacity. Serum CK and CRP concentrations were determined using colorimetric and turbidometric assays on a Siemens Advia 2400 autoanalyzer and using commercially available reagents (hsCRP (PZ Cormay, Lublin, Poland) interassay coefficient of variation (CV) = 3.34%; CK (Siemens Medical Solutions Diagnostics, Ltd., Berks, United Kingdom) interassay CV = 3%). Total antioxidant status (TAS) was assessed using a colorimetric assay (Randox Laboratories, Ltd., Antrim, United Kingdom; interassay CV = 3.65%). Serum nitrotyrosine (Millipore, Billerica, MA; interassay CV = 8%) and PC (Oxiselect™; Cell Biolabs, Inc., San Diego, CA; interassay CV = 8%) were quantified using commercially available ELISA kits. Changes over time were quantified as both percentage and absolute differences from preexercise values.

Biomechanical recordings. The knee extension force was measured continuously during the experimental protocol using an inline force transducer (MCL; RDP Ltd., Wolverhampton, United Kingdom). The transducer was calibrated in the range from 0 to 100 kg using standard weights, and the force was recalculated and displayed online in newtons. Data were recorded and digitized simultaneously via an analog-to-digital converter (CED 1401power, Cambridge, United Kingdom), using Spike2 data acquisition software (CED), with a 200-Hz sampling frequency.

Offline data analysis was performed using custom-made software developed in the script language Spike2 ver. 6.1 for CED. The maximal isometric force was calculated as the average force over 1-s periods during the force plateau of each MVC contraction. This period did not include the first segment of the contraction (lasting about 0.5 s) to exclude the period of force development. The highest of three MVC completed before exercise was accepted as the initial MVC force. MVC force at each time point was normalized to the preexercise value for the specific condition. Work done during the exercise protocol was calculated by integrating the force over time trace, and data were normalized to the corresponding 1RM value to eliminate interindividual and interleg variability.

Pressure pain threshold. Pressure pain threshold (PPT) was measured using a handheld algometer (Wagner Instruments, Inc.) before, immediately after, and 24 and 48 h after completion of the exercise protocol. Muscle site contact was made with a cylindrical metal probe with flat head diameter of 10 mm. The investigator applied a steadily increasing pressure to the muscle until the participant indicated that the point of discomfort had been reached. The applied pressure was recorded. Measurements were made over the muscle belly of rectus femoris (distally to the midpoint of the line connecting the central superior aspect of the patella and anterior superior iliac spine), vastus lateralis (distal half of the muscle along the line connecting the lateral superior aspect of the patella and the head of the greater trochanter), and vastus medialis (4–5 cm medial to the superior aspect of the patella). Data were normalized to preexercise values to reduce interindividual variability.

Statistical analysis. All data are reported as mean ± SEM for preexercise, postexercise, day 1 recovery, and day 2 recovery for 10 participants unless otherwise stated. Data were analyzed by two-way repeated-measures ANOVA (treatment (two levels: CA and FC) vs time (four levels)) to determine whether there were any statistically significant effects of time or treatment. The Mauchly sphericity test was used to check homogeneity of covariance for all ANOVA analyses; violations of the assumption of sphericity were corrected using the Greenhouse–Geisser adjustment. Where appropriate, the effect size statistic (η^2) was also calculated. The overall acceptable significance level of differences for all statistical tests was set at $P \leq 0.05$. The statistical analyses were performed in SPSS 14 (SPSS, Inc., Chicago, IL) and Origin version 6.0 (MicroCal Software, Inc., Piscataway, NJ) package software. Intraclass correlation analysis was used to assess the test–retest reliability of the muscle function (MVC force) measurement. A one-way random-effects single-measure model [1,1] was applied on the data from repeated MVC tests to calculate the intraclass correlation coefficients.

RESULTS

There was no difference between trials in the amount of relative work completed by subjects during the intensive exercise protocol (326 ± 31 vs 335 ± 27, arbitrary units; $P = 0.6$). Knee extension MVC force decreased on average to 64% of preexercise levels after completing the intensive exercise protocol (main effect of time, $P < 0.001$). Force recovery was significantly faster during the CA trial than during the FC trial (interaction effect, $P = 0.04$; $\eta^2 = 0.36$), with levels returning to 90.9% ± 4.0 % (vs 84.9% ± 3.2%, FC trial) after

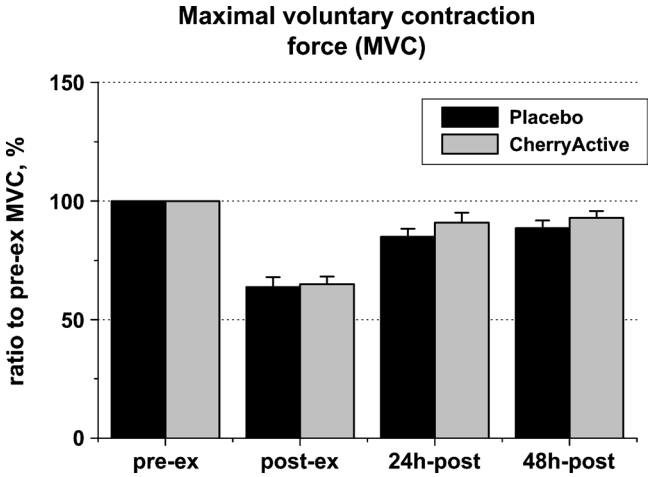

FIGURE 2—MVC force normalized to preexercise values. There was a main effect of time ($P < 0.001$) and a significant interaction effect ($P = 0.04$) with enhanced MVC force recovery in the CA trial.

TABLE 1. Serum markers of muscle damage, oxidative damage, and inflammation.

Outcome Measure	Trial	Before Exercise	After Exercise (Δ Before to After)	24 h After Exercise (Δ Before to 24 h After)	48 h After Exercise (Δ Before to 48 h After)
CK activity ($IU \cdot mL^{-1}$; $n = 10$)**	CA	437.7 ± 84.9	470.2 ± 90.7	763.5 ± 152.9	509.6 ± 102.4
		(32.5 ± 8.5)	(325.8 ± 109.6)	(71.9 ± 72.3)	
	FC	243.2 ± 35.9	257.3 ± 35.1	445.2 ± 58.6	370.0 ± 47.1
		(14.1 ± 3.2)	(202.0 ± 62.8)	(126.8 ± 40.3)	
PC ($nmol \cdot mg^{-1}$ protein; $n = 8$)**	CA§§	1.35 ± 0.05	1.48 ± 0.04	1.67 ± 0.04	1.64 ± 0.04
		(0.13 ± 0.02)	(0.31 ± 0.03)	(0.29 ± 0.04)	
	FC	0.75 ± 0.03	0.98 ± 0.05	1.35 ± 0.07	1.13 ± 0.14
		(0.23 ± 0.07)	(0.60 ± 0.08)	(0.38 ± 0.15)	
Nitrotyrosine ($nmol \cdot L^{-1}$; $n = 10$)	CA	539.6 ± 128.9	617.1 ± 104.3	578.8 ± 185.9	644.5 ± 111.4
	FC	630.7 ± 158.6	657.7 ± 170.9	644.7 ± 176.1	652.6 ± 172.5
TAS (mM; $n = 10$)	CA	1.72 ± 0.05	1.79 ± 0.04	1.67 ± 0.06	1.70 ± 0.0
	FC	1.78 ± 0.07	1.76 ± 0.06	1.73 ± 0.04	1.71 ± 0.04
hsCRP ($mg \cdot L^{-1}$; $n = 10$)	CA	1.8 ± 1.1	1.9 ± 1.1	1.6 ± 0.9	1.6 ± 0.8
	FC	2.8 ± 1.5	2.8 ± 1.6	2.4 ± 1.7	2.3 ± 1.7
MVC*,† (N; $n = 9$)	CA§	894.9 ± 70.9	578.0 ± 50.7	813.1 ± 73.9	834.6 ± 72.6
		(−318.1 ± 138.9)	(−137.0 ± 98.8)	(−92.6 ± 84.2)	
	FC	841.7 ± 56.5	523.6 ± 28.7	704.6 ± 48.5	749.0 ± 64.1
		(−316.9 ± 115.0)	(−81.8 ± 127.4)	(−60.3 ± 92.3)	

Data are presented as mean ± SEM, with absolute change from baseline indicated in parentheses.
There was a main effect of time for CK, PC, and MVC (** $P < 0.001$).
The absolute increase in PC tended to be lower during CA than FC trials (trial main effect, $P = 0.079$), but the reverse was true for CK (trial × time interaction effect, $P = 0.063$).
The absolute decrease in MVC force was smaller during CA than FC trials (trial × time interaction effect, † $P = 0.047$).
However, CK, MVC, and PC were higher during the CA trial than during the FC trial (trial main effect, $P = 0.055$, § $P = 0.03$, §§ $P = 0.001$, respectively). There was no effect of time or trial on serum nitrotyrosine, hsCRP, or TAS.

24 h of recovery and to 92.9% ± 2.8% (vs 88.5% ± 3.1%, FC trial) after 48 h of recovery, respectively (Fig. 2). A similar pattern was observed for nonnormalized MVC force data, with both absolute change (trial × time interaction, $P = 0.047$, $\eta^2 = 0.32$) and absolute MVC force (main trial effect, $P = 0.03$, $\eta^2 = 0.47$; Table 1) data significantly higher in the CA trial than in the FC trial. An intraclass correlation coefficient of 0.955 was calculated from the repeated MVC tests performed by three participants during a preliminary trial and the main trial, indicating high reliability of the MVC force measurement.

There was a significant reduction in the PPT after 24 and 48 h of recovery from the exercise protocol in all three muscles ($P < 0.005$), indicating the development of muscle soreness. Similarly, when a summed response across all muscles was considered, there was a significant reduction in pressure pain threshold after 24 and 48 h of recovery (interaction effect, $P < 0.001$). However, there was no significant difference between trials in PPT of either individual muscles or the summed response (Fig. 3).

There was a significant increase in serum CK activity after the exercise protocol (main effect of time, $P < 0.001$; Table 1). The percentage increase in serum CK activity from baseline was not statistically different between trials after 24 h (108.3% ± 56.5% CA; 127.6% ± 54.3% FC) and 48 h (38.2% ± 33.2% CA; 73.7% ± 31.2% FC) of recovery from the exercise protocol (Fig. 4). However, both total serum CK activity (trial main effect, $P = 0.055$; Table 1) and the absolute increase in serum CK activity tended to be higher during the CA trial than during the FC trial (interaction effect, $P = 0.063$; Table 1). PC content also increased significantly after the exercise protocol (main effect of time, $P < 0.001$; Table 1). Both the percentage (main trial effect, $P = 0.013$, Fig. 5) and the absolute (main trial effect, $P = 0.079$; Table 1)

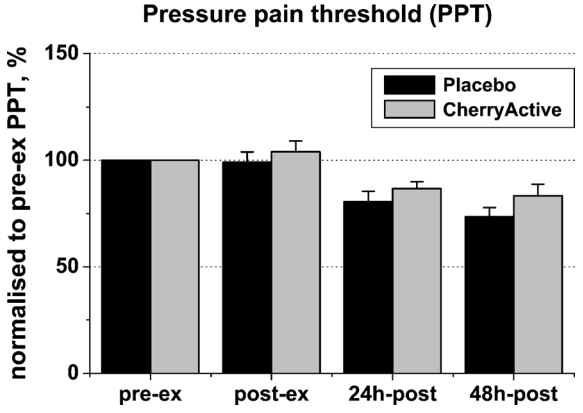

FIGURE 3—PPT normalized to preexercise values was decreased after the exercise intervention ($P < 0.001$), but there was no statistically significant effect of trial or trial × time interaction for the summed response.

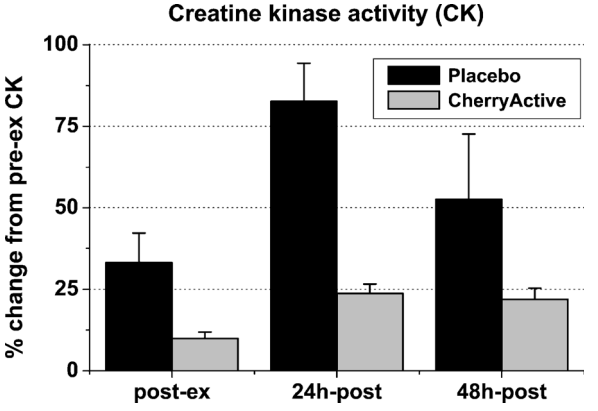

FIGURE 4—Percentage change in serum CK activity from preexercise values. There was a main effect of time ($P = 0.031$).

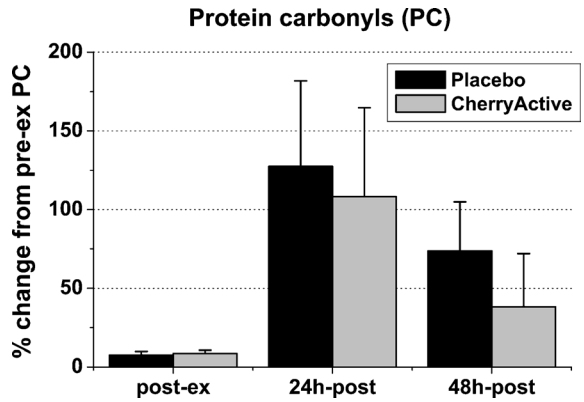

FIGURE 5—Percentage change in serum PC from preexercise values. There was a main effect of time ($P = 0.034$) and a significant effect of trial ($P = 0.013$).

increase in PC content from baseline was lower during the CA trial than during the FC trial. However, PC content was significantly higher throughout the experimental protocol in the CA trial than during the FC trial (trial main effect, $P = 0.001$; Table 1). There was no statistically significant effect of time or condition on total serum nitrotyrosine concentration or hsCRP, although there was a tendency for hsCRP to be higher during the FC trial than during the CA trial (Table 1). For 7 of the 10 subjects, hsCRP concentrations were close to the detection limit of the assay 0.1 $mg \cdot L^{-1}$. In the remaining three subjects, baseline hsCRP concentration was observed to be lower in the CA trial (5.1 ± 3.0 $mg \cdot L^{-1}$) than in the FC trial (9.0 ± 2.8 $mg \cdot L^{-1}$).

There was no significant change over time in serum total antioxidant capacity and no difference between trials (Table 1). The total antioxidant capacity values were at the top end of the reference range (1.3–1.77 $mmol \cdot L^{-1}$) for both FC and CA trials (1.74 ± 0.03 vs 1.72 ± 0.05 $mmol \cdot L^{-1}$).

DISCUSSION

The main finding of this study was that recovery of knee extensor maximum isometric strength was enhanced after consuming Montmorency cherry juice for 7 d before and 2 d after an intensive knee extensor resistance training session. This improvement in functional recovery was accompanied by a reduction in serum PC indicative of reduced oxidative damage. However, no other markers of muscle damage or inflammation were favorably affected by Montmorency cherry juice consumption. Also, there was no evidence of a significant reduction in muscle soreness because Montmorency cherry juice consumption had no effect on muscle pressure pain threshold.

These findings are in agreement with those of Connolly et al. (10) and Howatson et al. (13) who also found that Montmorency cherry consumption enhanced maximum isometric force recovery of the elbow flexors after eccentric exercise and of the knee extensors after completing a marathon, respectively. Interestingly, Howatson et al. (13) found that the magnitude of the immediate reduction in maximum isometric force production after completing the marathon was not different between trials. This was suggested to indicate that the cherry juice supplement did not prevent the initial muscle injury, which was presumably induced by a combination of mechanical disruption of the myofibrils and increased generation of ROS and NO species during exercise. Instead, the cherry juice was suggested to blunt the secondary muscle damage response associated with the local inflammatory response in the damaged muscle, and this was corroborated by the finding of reduced IL-6, CRP, and uric acid response to the marathon race. In the present study, the reduction in knee extensor maximum isometric force 4 min after completion of the exercise protocol was also very similar between trials (~36%), suggesting similar degrees of long-lasting fatigue immediately after exercise. However, hsCRP was not elevated in response to the single-leg knee extensor exercise, perhaps the smaller muscle mass involvement was not sufficient to elevate this marker of systemic inflammation, and unfortunately, no markers of local muscle inflammation were measured. Surprisingly, Connolly et al. (10) did not measure MVC force immediately after the eccentric elbow flexor exercise so data are not available for comparison.

Kuehl et al. (23) and Connolly et al. (10) reported that participants experienced less muscle soreness after the Hood to Coast relay race (mean 26-km run) and eccentric elbow flexion exercise when cherry juice was consumed for a period before and after the exercise. However, in common with Howatson et al. (13), we found no reduction in muscle soreness in the cherry juice trial, which, in the face of reduced inflammation, at least in the former trial, is perhaps surprising. Pressure pain threshold on the belly of the muscle was used as the measure of muscle soreness in the present study in an attempt to ameliorate the subjective nature of the visual analog scale measure of soreness. However, any measure of muscle soreness is, by its very nature, subjective and therefore subject to variability, although PPT reliability is maximized when measures are taken over the muscle belly, by the same investigator on each occasion, as in the present study (27). However, the rate of pressure development, which also introduces variability, was not directly controlled in the present study, although Kinser et al. (22) found that a single investigator has a high degree of reliability in rate of pressure development. The magnitude of muscle soreness reported by our subjects was modest (at maximum ~27% reduction in PPT) compared with that induced by 40-min downhill running (~50%). The repeated bout effect is well documented (25), thus it was perhaps unsurprising that we were only able to induce relatively modest muscle soreness in these participants who regularly performed intensive knee extensor resistance training. This small magnitude of change in PPT may have limited the ability of our experimental paradigm to detect any effect of the Montmorency cherry juice.

The repeated bout effect is a well-accepted phenomenon, which refers to the marked reduction in muscle damage

when repeated bouts of eccentric exercise are performed up to 6 months apart (26). The effect is generally considered to be peripheral in nature and isolated to the specific muscle/limb exercised. The repeated bout effect must be considered when designing studies to investigate the efficacy of strategies to counteract muscle damage. In this instance, we used single-limb exercise, with trials counterbalanced for supplement and leg dominance to avoid the response to exercise in trial 1 influencing results in trial 2. However, there is one published study that has demonstrated a small protective effect against eccentric elbow flexor damage and soreness in the contralateral arm (14). A second bout of single arm exercise was performed by both contralateral and ipsilateral arms 2 wk after performing the same exercise with one arm only. The repeated bout effect was much smaller in the contralateral than ipsilateral arm, and except for muscle soreness, the contralateral repeated bout effects were evident only at 96 h not 48 h after bout 2 of exercise. The observed differences in parameters during the first 48 h of recovery in the present study were not therefore confounded by any repeated bout effect from trial 1 in the contralateral leg. In line with previously published crossover phytonutrient studies, we adopted a 2-wk washout period between trials (10,35). In addition, the study was fully counterbalanced.

Despite the improvement in functional recovery with Montmorency cherry juice consumption, there does not seem to be any protective effect in terms of the extent of the structural damage to the muscle because the percentage change in serum CK was not different between trials. Indeed, the absolute concentration and absolute change in CK after the intensive exercise tended to be larger during the cherry juice trial. This finding also concurs with Howatson et al. (13) where, despite the improved functional recovery and reduced inflammatory response to marathon running after cherry juice consumption, the increases in CK and lactate dehydrogenase were not different between trials. Unfortunately, in many studies, markers of muscle damage have been measured in the absence of markers of oxidative damage and vice versa. However, in the present study and others, there is a dissociation between the extent of oxidative damage induced by exercise as indicated by a variety of measures, such as PC, isoprostanes (7,20,21), TBARS (13), and malondialdehyde (7), and the increase in serum CK and LDH. In our hands, the exercise-induced increase in PC was significantly attenuated by cherry juice consumption, whereas the increase in CK was, if anything, exaggerated in the cherry juice condition. There is considerable variation across studies in the oxidative damage response to resistance exercise with some groups finding evidence of oxidative damage (4,11,16, 31,32) and others not (1,3). This variation may be attributable to differences in the exercise mode, duration and intensity, training status of participants, as well as the use of a variety of indirect measures of oxidative damage to muscle. Veskoukis et al. (37) recently demonstrated that blood and muscle PC after swimming to exhaustion were closely correlated in rats and therefore suggest that blood PC, as well as catalase and reduced glutathione, provide reliable indicators of skeletal muscle redox status. In contrast, TBARS, xanthine oxidase, and total antioxidant capacity were found to be poorly correlated with changes in muscle redox status.

Although the extent of exercise-induced oxidative damage was attenuated in the Montmorency cherry juice trial, there was no difference between trials in total antioxidant capacity. This is in contrast to the findings of Howatson et al. (13), who found a greater increase in total antioxidant capacity after the race in the cherry juice trial. However, in the present study, blood samples were taken from overnight-fasted subjects at least 8 h after consumption of the previous dose of Montmorency cherry juice concentrate, whereas subjects in the study of Howatson et al. (13) were not fasted. Consumption of 28 g of sweet Bing cherries has been shown to result in a significant increase in plasma lipophilic ORAC score and reduction in plasma urate that was present up to 5 h after consumption (17,29). Chronic consumption of Bing cherries (280 $g \cdot d^{-1}$ for 28 d) has also been shown to reduce some serum markers of inflammation (CRP, NO, and RANTES) even in the fasted state (19). Although the dose–response to Montmorency cherries has not yet been studied, it is likely to be similar in time course if not in magnitude. This may explain the absence of any effect of CA consumption on total antioxidant capacity because blood samples were taken more than 5 h after consumption of CA (at least 8 h), and the period of supplementation was only 7 d not 28 d and may not therefore have been long enough to induce an increase in TAS through the day.

The elevated baseline measures of muscle and oxidative damage, which was statistically significant in the case of PC, is a potentially confounding factor. This presumably indicates a higher level of background oxidative damage in the CA trial. Although participants were instructed to perform the same training during the 7 d of supplementation before the controlled intensive exercise bout, it seems that for at least 7 of the 10 subjects this, was not the case. Despite this, we are unaware of any mechanism by which the significantly higher background serum PC would influence the subsequent response to the same exercise protocol. Although as a result, the data should only be generalized with caution.

In conclusion, consumption of Montmorency cherry juice concentrate for 7 d before, the day of, and 2 d after completing a bout of intensive knee extensor resistance enhanced recovery of isometric muscle strength. This improvement in functional recovery was accompanied by a reduction in oxidative stress presumably because of the anti-inflammatory and antioxidative effects of the phytochemicals within the Montmorency cherries.

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