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## The Effect of Superoxygenated Water on Blood Gases, Lactate, and Aerobic Cycling Performance

Lars R. McNaughton, Steve Kenney, Jason Siegler, Adrian W. Midgley, Ric J. Lovell, and David J. Bentley

**Context:** Recently, superoxygenated-water beverages have emerged as a new purported ergogenic substance. **Purpose:** This study aimed to determine the effects of superoxygenated water on submaximal endurance performance. **Methods:** Eleven active male subjects,  $VO_{2max}$   $52.6 \pm 4.8$  mL · kg<sup>-1</sup> · min<sup>-1</sup>, height  $180.0 \pm 2.0$  cm, weight  $76.0 \pm 7.0$  kg, age  $24 \pm 1.0$  y (mean  $\pm$  SD), completed a 45-min cycle-ergometry exercise test at 70% of their previously predicted maximal power output with a 10-min rest period, followed by a 15-min time trial (TT). Thirty minutes before the exercise test subjects consumed 15 mL of either superoxygenated water (E) or placebo (P; water mixed with low-chlorine solution). Subjects then completed the test again a week later for the other condition (double-blind, randomized). The physiological variables measured during exercise were  $VO_2$ ,  $VCO_2$ , respiratory-exchange ratio (RER),  $V_E$ ,  $PO_2$ ,  $PCO_2$ , blood lactate (bLa<sup>-</sup>), and heart rate (HR). Mean distance covered and the average power output for the 15-min TT were also measured as performance indicators. **Results:** There were no significant differences in  $VO_2$ ,  $VCO_2$ , RER,  $V_E$ , bLa<sup>-</sup>,  $PO_2$ , and HR ( $P > .05$ ) during the exercise tests. Neither were there any significant improvements in the total distance covered (P  $9.01 \pm 0.74$  km vs E  $8.96 \pm 0.68$  km,  $P > .05$ ) or the average power output (P  $186.7 \pm 35.8$  W vs E  $179.0 \pm 25.9$  W,  $P > .05$ ) during the 15-min TT. **Conclusion:** Based on these results the authors conclude that consuming 15 mL of superoxygenated water does not enhance submaximal or maximal TT cycling performance.

**Key Words:** exercise, supplements, ergogenic aids, blood lactate

Nutritional supplements are often used by athletes in an attempt to enhance performance.<sup>1-3</sup> One such nutritional supplement is hyperoxygenated or superoxygenated water, which might contain a more than 9 times higher oxygen concentration than normal tap water.<sup>4</sup> The idea that athletes might use an ergogenic aid containing extra dissolved oxygen (O<sub>2</sub>) came about because it had been shown that breathing O<sub>2</sub> during exercise significantly improved performance.<sup>5</sup> There are now a number of superoxygenated waters on the market, and they appear to be popular with athletes,

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especially at the elite level. Previous work in this area has analyzed the intestinal absorption of these products and found it to be minimal,<sup>6</sup> and in a recent article, Piantadosi<sup>7</sup> supplied a number of rational and logical reasons why such products cannot work. Primary among such factors is that the amount of additional oxygen delivered by these products is minimal, only 3 mL per dose, depending on the level of oxygen found in the product. Clearly such a dose would have a negligible effect on oxygen transport to the tissues given the amounts transported through the respiratory and circulatory systems.

To date there have been very few studies that have analyzed the performance benefits of superoxygenated water in humans. As far as we can tell, the first study was undertaken by Duncan<sup>8</sup> at Texas Women's University. That study reported a mean decrease of 15 seconds in 5-km time-trial performance when subjects consumed superoxygenated water compared with ingestion of regular tap water. Furthermore, the ergogenic effect was significantly greater in the more highly trained subjects ( $\text{VO}_2\text{max} > 54 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ). Oxygen saturation was measured by pulse oximetry but did not change in the experimental trial. Manufacturers, however, claim that this is the physiological mechanism by which these superoxygenated beverages improve endurance performance.<sup>9</sup> In contrast, in a later study, significant differences in  $\text{SaO}_2$  values were reported when comparing superoxygenated water with regular tap water.<sup>10</sup> According to Piantadosi,<sup>7</sup> however, the changes in  $\text{SaO}_2$  could not possibly be accounted for by the amount of superoxygenated water given to the subjects. It is interesting that in the Jenkins et al study<sup>10</sup> no significant improvements in endurance performance occurred when comparing subjects who consumed superoxygenated water with those who consumed tap water. Mielke et al<sup>11</sup> reported similar findings for submaximal and maximal cycle-ergometry performance. This is supported by the most recent work in the area, which found no significant ergogenic effect of consuming superoxygenated water on submaximal or maximal metabolism or on lactate kinetics during cycle ergometry. Again, the authors of that study concluded that these products do not enhance performance.<sup>12</sup>

Willmert et al<sup>13</sup> investigated the effects of superoxygenated water and standard tap water on responses of blood pressure, heart rate (HR), maximal aerobic capacity ( $\text{VO}_2\text{max}$ ), and blood lactate ( $\text{bLa}^-$ ) to exercise and recovery. The results indicated that superoxygenated water had no effect on HR, blood pressure,  $\text{bLa}^-$ , or  $\text{VO}_2$  values when subjects exercised at either submaximal or maximal levels. The findings from this study are in contrast with those of other studies that found benefits of consuming superoxygenated water.<sup>8,10</sup>

A more recent study<sup>14</sup> analyzed the responses of  $\text{SaO}_2$  and HR during hypoxia after subjects consumed superoxygenated water. Twenty subjects randomly completed 2 trials in which they consumed either 20 oz of superoxygenated water or 20 oz of standard tap water. They then rested for 30 minutes before inhaling a 10% oxygen-rich gas mixture for 3 minutes, followed by inhalation of room air for a further 5 minutes. This procedure was repeated 3 times. Throughout all trials HR and  $\text{SaO}_2$  were recorded by pulse oximetry, from which rates of oxyhemoglobin desaturation and resaturation were calculated. Results showed no differences for either mean HR response or  $\text{SaO}_2$  at maximum for both the superoxygenated water and the standard-water conditions. Those researchers concluded that consuming 20 oz of superoxygenated water has no effect on HR or  $\text{SaO}_2$  during induced hypoxia.

On the basis of previous work in the area, the aim of this study was therefore to investigate the use of superoxygenated water as an ergogenic aid during cycling performance. We also wished to investigate the effects of this drink on some physiological parameters before and after exercise.

## Methods

### Subjects

Eleven active male subjects took part in this study. They had the following (mean  $\pm$  SD) physical characteristics:  $\text{VO}_2\text{max}$   $52.6 \pm 4.8 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ , height  $180.0 \pm 2.0 \text{ cm}$ , weight  $76.0 \pm 7.0 \text{ kg}$ , and age  $24 \pm 1.0 \text{ years}$ . All subjects were injury free and regularly participating in aerobic training  $4 \pm 1 \text{ d/wk}$ . All testing took place in an environmentally controlled laboratory (temperature  $22^\circ\text{C} \pm 1^\circ\text{C}$ ). The experimental procedures had previously been reviewed and approved by the departmental ethics committee. After being informed of potential risks and discomforts of participation, subjects gave written informed consent.

### Experimental Design

***VO<sub>2</sub>max Testing.*** Every subject undertook a baseline  $\text{VO}_2\text{max}$  test on the SRM cycle ergometer (SRM, Julich, Germany) a week before undergoing performance testing. Before the actual testing, the gas analyzer and ventilation of the metabolic cart (Cosmed Quark b<sup>2</sup>, Cosmed, Rome, Italy) were calibrated according to the manufacturer's specifications. The  $\text{VO}_2\text{max}$  protocol began at a power output of 50 W/min, with increments of 25 W/min until the subject reached volitional exhaustion and could no longer turn the pedals. Throughout the test, gas analysis was monitored and HR was recorded through use of a Polar HR monitor (S810i, Polar Electro Oy, Finland). Subjects were deemed to have provided a maximal effort with a respiratory-exchange ratio  $>1.13$  and a maximum HR of  $\pm 10 \text{ beats/min}$  of their age-predicted maximal HR ( $220 - \text{age}$ ).

***Submaximal and Time-Trial Performance.*** Subjects, who were all nonsmokers, were asked not to consume any carbohydrates or water 2 hours before testing and to refrain from any caffeine intake in the 12 hours before testing. All testing equipment was set up appropriately 1 hour before testing, and the metabolic system (Cosmed Quark b<sup>2</sup>) was calibrated just before each subject began the test. All testing started with a standardized warm-up consisting of 5 minutes of light exercise on the cycle ergometer (SRM, Julich, Germany) followed by 5 minutes of stretching the predominant muscles to be used during the cycling exercise.

The performance-test protocol was a double-blind crossover design in which a counterbalance randomization procedure was applied.<sup>15</sup> All subjects were therefore randomly assigned either 15 mL of superoxygenated water (Oxyshot; E) or placebo (P; water mixed with low-chlorine solution, 2.86 ppm, pH 7.64). After consuming their drinks, the subjects remained seated for 30 minutes, which is the manufacturer's recommended time for the E to be beneficial to performance. Each subject then exercised at 70% of his power-output-related  $\text{VO}_2\text{max}$  for 45 minutes on the cycle ergometer, and breath-by-breath gas-exchange variables (Cosmed Quark b<sup>2</sup>) and

HR (S810i, Polar Electro Oy, Finland) were recorded throughout the test. Once this was completed the subject stopped cycling and remained seated on the cycle ergometer for 10 minutes. The subject then carried out a 15-minute time trial (TT) in which he was instructed to cover as much distance as possible during the allotted time period. The distance (km) and the average power output (W) were recorded for all subjects at the end the 15-minute TT. A week after the first test, each subject then undertook a second test, in which he consumed the alternate solution.

**Blood Lactate and Blood Gases.** Capillary blood samples were taken aseptically, with the subjects at rest, 30 minutes after consumption of the water or placebo and during exercise at 10, 20, 30, 40, and at 45 minutes. Further blood samples were taken before and at the cessation of the TT, as well as at 3 and 7 minutes postexercise. Capillary blood was analyzed for  $\text{PO}_2$ ,  $\text{PCO}_2$  (OMNI 4, Roche Diagnostics Ltd, Lewes, UK), and  $\text{bLa}^-$  (YSI 1500 Sport, YSI Inc, Yellow Springs, OH) in 200- $\mu\text{L}$  blood-gas capillary tubes (Roche Diagnostics).

## Data Analysis

All data are presented as mean ( $\pm$  SD) and were analyzed using SPSS for Windows software (release 11.5.0, SPSS Inc, Chicago, IL). Two-way analysis of variance (ANOVA) with repeated measures on both factors (trials  $\times$  time) was conducted to determine whether there were any statistically significant differences between the E and P conditions. Paired-samples Student *t* tests allowed statistically significant differences to be reported for both distance covered and average power output for the E and P conditions. Statistical significance was accepted as  $P < .05$ .

## Results

The gas-analysis variables are reported in Table 1. There was no statistically significant difference between  $\text{VO}_2$  scores for E versus P ( $P > .05$ ). For  $\text{VCO}_2$ , however, for both P and E there was a statistical significance between time and increase in  $\text{VCO}_2$  for the 2 combined conditions ( $P < .001$ ). There was, however, no significant difference between the  $\text{VCO}_2$  scores for the 2 conditions ( $P > .05$ ).

In addition, there was a significant increase in  $\text{V}_E$  over time for both the P and the E trials ( $P < .05$ ), but with no significance between the  $\text{V}_E$  scores for the 2 conditions ( $P > .05$ ). The analysis of respiratory-exchange rate showed a time effect ( $P < .05$ ), but there was no statistical significance between the respiratory-exchange-rate scores for the 2 separate conditions ( $P > .05$ ).

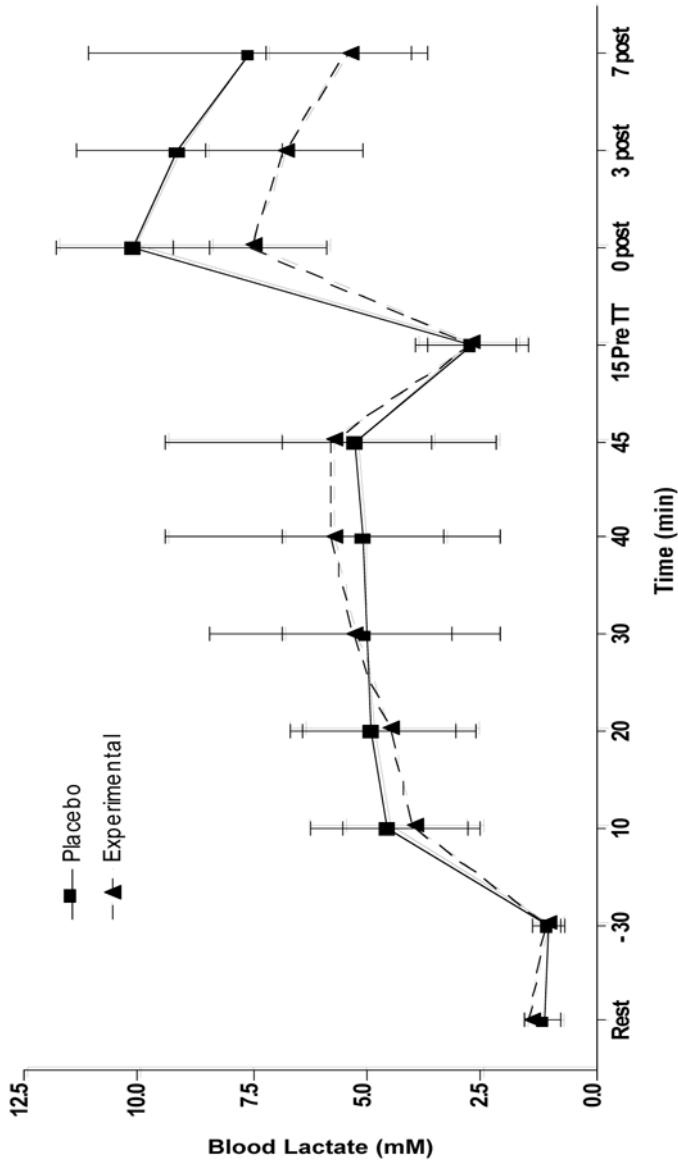
Mean  $\text{bLa}^-$  during the 45-minute prefatigue trial were similar for both conditions (see Figure 1), and there was no time  $\times$  trial interaction effect ( $P > .05$ ) and no main effect of group ( $P > .05$ ). There was, however, as expected, a significant main effect of time ( $P < .0001$ ).

After the 15-minute TT, there was no time  $\times$  group interaction effect ( $P > .05$ ), but there were a main effect of group ( $P < .05$ ) and a main effect of time ( $P < .0001$ ). A Tukey post hoc analysis indicated that in P,  $\text{bLa}^-$  was significantly higher ( $P < .05$ ) at 0 and 3 minutes post-TT but not at 7 min post-TT ( $P > .05$ ).

The mean  $\text{PO}_2$  levels during the 45-minute prefatigue trial were also similar for both conditions, and no statistical significance between the 2 conditions was

**Table 1 The Gas-Analysis Variables (Mean  $\pm$  SD) for the 2 Conditions at Specific Times in the 45-Minute Prefatigue Trial**

Time	$\dot{V}O_2$ (mL $\cdot$ min $^{-1}$ $\cdot$ kg $^{-1}$ )		$\dot{V}CO_2$ (mL/min)		$V_E$ (L/min)		Respiratory-Exchange Ratio	
	Placebo	Experimental	Placebo	Experimental	Placebo	Experimental	Placebo	Experimental
Resting, 0 min	25.3 $\pm$ 3.2	33.2 $\pm$ 2.7	1561.7 $\pm$ 238.6	1454.8 $\pm$ 184.9	36.6 $\pm$ 5.9	34.2 $\pm$ 5.2	0.81 $\pm$ 0.09	0.83 $\pm$ 0.06
5 min	33.4 $\pm$ 2.8	33.0 $\pm$ 2.9	2624.2 $\pm$ 210.5	2567 $\pm$ 255.6	63.7 $\pm$ 7.4	59.9 $\pm$ 6.5	1.03 $\pm$ 0.03	1.03 $\pm$ 0.04
10 min	34.6 $\pm$ 3.3	35.0 $\pm$ 3.0	2614.5 $\pm$ 143.1	2694.2 $\pm$ 233.8	69.4 $\pm$ 10.3	69.5 $\pm$ 8.9	1.01 $\pm$ 0.02	1.02 $\pm$ 0.04
15 min	34.2 $\pm$ 3.5	25.2 $\pm$ 3.1	2505.2 $\pm$ 198.7	2658.2 $\pm$ 247.9	65.8 $\pm$ 9.0	68.3 $\pm$ 8.8	0.97 $\pm$ 0.02	1.00 $\pm$ 0.03
20 min	34.6 $\pm$ 3.3	35.2 $\pm$ 2.9	2578.8 $\pm$ 162.1	2687.7 $\pm$ 305.1	70.6 $\pm$ 9.5	73.4 $\pm$ 13.5	0.99 $\pm$ 0.03	1.01 $\pm$ 0.03
25 min	34.0 $\pm$ 3.8	36.0 $\pm$ 2.2	2492.7 $\pm$ 241.4	2591.5 $\pm$ 165.5	70.4 $\pm$ 14.6	68.3 $\pm$ 8.6	0.96 $\pm$ 0.05	0.97 $\pm$ 0.05
30 min	34.7 $\pm$ 4.1	35.2 $\pm$ 2.6	2559.3 $\pm$ 189.9	2592 $\pm$ 268.2	71.6 $\pm$ 10.8	71.4 $\pm$ 12.7	0.97 $\pm$ 0.03	0.97 $\pm$ 0.04
35 min	34.7 $\pm$ 3.3	34.4 $\pm$ 2.3	2405.8 $\pm$ 254.6	2480.7 $\pm$ 185.9	68.8 $\pm$ 11.4	68.0 $\pm$ 11.1	0.95 $\pm$ 0.03	0.96 $\pm$ 0.03
40 min	35.0 $\pm$ 3.4	34.1 $\pm$ 3.0	2581.3 $\pm$ 230.4	2460.3 $\pm$ 218.1	72.4 $\pm$ 10.3	67.8 $\pm$ 9.7	0.94 $\pm$ 0.04	0.96 $\pm$ 0.05
45 min	34.4 $\pm$ 5.7	33.6 $\pm$ 6.4	2503.8 $\pm$ 351.5	2456.7 $\pm$ 243.5	73.3 $\pm$ 16.0	69.6 $\pm$ 14.6	0.96 $\pm$ 0.02	0.97 $\pm$ 0.03



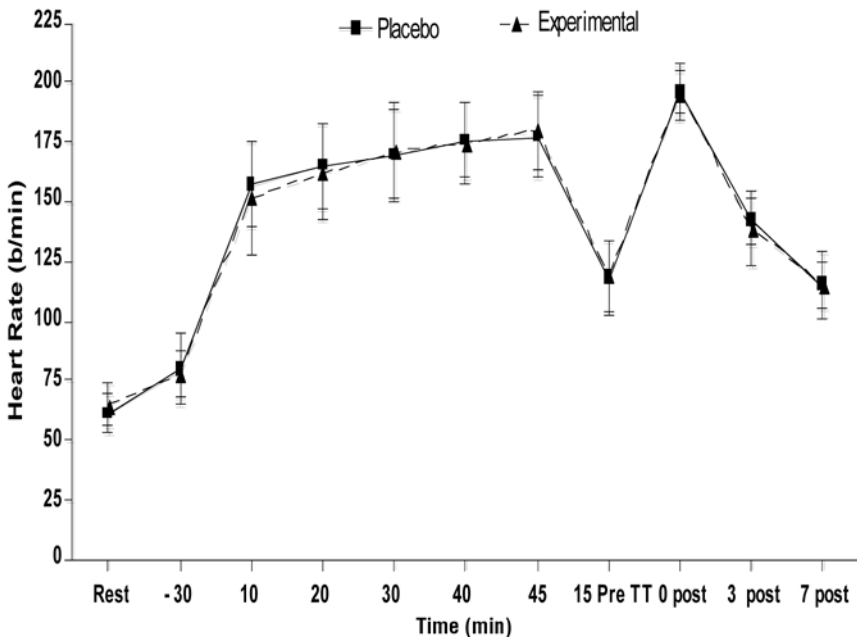
**Figure 1** — Blood lactate response in the 2 trials (mean  $\pm$  SD).

found ( $P > .05$ ). There was also no statistical difference ( $P > .05$ ) in  $PCO_2$  between the 2 conditions ( $P$  vs  $E$ ; Table 2).

HR increased significantly over time in both conditions ( $P < .001$ ), although there was no significant ( $P > .05$ ) difference between the  $P$  and  $E$  conditions (Figure 2).

**Table 2 The Blood-Gas Measurements (Mean  $\pm$  SD) for Both Conditions**

Time	$PO_2$ (mm Hg)		$PCO_2$ (mm Hg)	
	Placebo	Experimental	Placebo	Experimental
Resting, 0 min	78.6 $\pm$ 4.2	76.9 $\pm$ 8.8	39.0 $\pm$ 3.9	36.6 $\pm$ 2.2
30 min	79.2 $\pm$ 3.7	72.8 $\pm$ 8.4	39.1 $\pm$ 3.1	37.4 $\pm$ 2.7
10 min	78.8 $\pm$ 8.9	79.5 $\pm$ 4.5	37.0 $\pm$ 1.4	33.7 $\pm$ 2.5
20 min	79.2 $\pm$ 7.0	83.8 $\pm$ 2.5	36.6 $\pm$ 1.9	33.7 $\pm$ 2.0
30 min	82.6 $\pm$ 5.8	83.5 $\pm$ 5.8	34.8 $\pm$ 4.0	30.9 $\pm$ 1.9
40 min	82.9 $\pm$ 3.9	86.2 $\pm$ 3.1	35.2 $\pm$ 1.4	33.3 $\pm$ 3.7
45 min	83.3 $\pm$ 4.3	83.9 $\pm$ 6.8	37.2 $\pm$ 2.1	25.9 $\pm$ 3.7
15 min pre-time trial	80.1 $\pm$ 3.0	80.4 $\pm$ 6.5	35.3 $\pm$ 3.5	35.5 $\pm$ 3.1
0 min post-time trial	94.8 $\pm$ 7.6	87.8 $\pm$ 9.9	31.2 $\pm$ 3.1	33.2 $\pm$ 4.2
3 min post-time trial	91.4 $\pm$ 9.3	84.8 $\pm$ 4.4	32.4 $\pm$ 2.2	32.2 $\pm$ 2.9
7 min post-time trial	82.5 $\pm$ 6.0	78.3 $\pm$ 6.1	32.3 $\pm$ 2.8	33.3 $\pm$ 4.6



**Figure 2** — Heart-rate response in the 2 trials (mean  $\pm$  SD).



**Table 3 Mean ( $\pm$  SD) Distance Covered and Average Power Output for Both Conditions for the 15-Minute Time Trial**

Measure	Condition		Significance (2-tailed)
	Placebo	Experimental	
Distance covered (km)	9.01 $\pm$ 0.74	8.96 $\pm$ 0.68	.906
Average power output (W)	186.7 $\pm$ 35.8	179.0 $\pm$ 25.9	.390

Two performance indicators were measured in the 15-minute TT: the distance covered (km) and average power output (W) sustained by the subjects. Table 3 presents the mean measurements for these 2 performance indicators for both conditions, which did not differ significantly ( $P > .05$ ).

## Discussion

The aim of this work was to determine whether a commercially available superoxygenated water product had any beneficial effect on submaximal or maximal aerobic performance. Furthermore, we wanted to determine whether any physiological parameters, namely, blood gases or blood lactate, were changed by ingestion of this product. Based on our data, there was no effect of consuming superoxygenated water with regard to either the performance measures or the physiological measurements.

These results are in agreement with the findings by most authors in the area.<sup>11-13</sup> In the work of Willmert et al,<sup>13</sup> their subjects completed 2 incremental tests using the modified Bruce protocol after consuming 500 mL of either superoxygenated water or tap water. The authors found no differences between the 2 drinks in maximal blood lactate concentration, rating of perceived exertion, or  $VO_2$ . Furthermore, these findings agree with those of Mielke et al,<sup>11</sup> who found no significant difference in the physiological response during submaximal exercise. They also found no significant improvement in  $VO_2$  values when subjects worked at a variety of intensities (60%, 80%, and 90% of their previously measured  $VO_{2max}$ ) after consuming 1200 mL of either oxygenated water or placebo every day for 3 days before the tests with 600 mL of either oxygenated water or placebo ingested 15 minutes before the protocol.

Blood analysis showed that lactate levels did not significantly decrease between the P and E trials throughout the 45-minute prefatigue exercise. Analysis showed that immediately and 3 minutes postexercise, blood lactate levels were statistically significantly lower for the E trial, but no significant differences were found at 7 minutes postexercise, although they approached statistical significance. These findings are therefore in contrast with the findings by others who concluded that blood lactate levels had no significant changes during recovery from either a submaximal or maximal exercise test after consuming the superoxygenated beverage.<sup>13</sup> Leibetseder et al<sup>12</sup> also found a lower lactate concentration at maximal work levels after a maximal effort but believed this difference to be the result of a regression toward the mean (a statistical effect) rather than a physiological effect. Given the discrepancies in experimental results, the effect of superoxygenated water on blood lactate in the postexercise period should be investigated further.

We found no changes in blood gases during the exercise bout, which is in contrast to the work of Jenkins et al.<sup>10</sup> They found significantly improved O<sub>2</sub> saturation levels but without any improvement in performance. Again, such a small amount of oxygen cannot explain the rise in O<sub>2</sub> saturation shown in this work.<sup>7</sup>

There is no doubt that any effect of superoxygenated waters cannot be caused by the amount of oxygen made available by such products.<sup>7</sup> The volume of liquid consumed is too small to allow any significant amount into the blood stream, a fact supported by the work of Willmert et al,<sup>13</sup> and the gut, unlike the lung, is not adapted to transport oxygen across the gut membrane.<sup>7</sup> If there is such an effect, it might be caused by other factors, but more work needs to be undertaken before any firm conclusions can be drawn.<sup>12</sup>

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