"Oxygenated" water and athletic performance

C A Piantadosi

Ergogenic claims for oxygenated water cannot be taken seriously

A decade or so ago, the idea arose that athletes might gain a competitive edge by drinking water that contained extra dissolved oxygen (O_2). The notion stems from observations that O_2 breathing during exercise enhances athletic performance,^{1,2} but the connection of O_2 breathing during exercise with drinking "hyperoxygenated" water before exercise conflates physics and physiology in a struthonian visit to placebo land. Fuelled by bottled-water mavens, who collect testimonials for oxygenated waters, claims abound of ergogenic benefits of water advertised to hold up to 40 times more O_2 than plain water.

The issue of hydration aside, such claims have a flimsy rationale and no rigorous experimental support. On close inspection, three scientific problems immediately arise. Firstly, for all practical purposes, supplemental O_2 improves performance only during exercise, not before or between bouts.³ With normal lungs, breathing of even pure O_2 at sea level increases maximal O_2 uptake ($\dot{V}O_{2\text{MAX}}$) by only 5–10% because air breathing alone, except at high intensity work, keeps the arterial O_2 saturation (SaO_2) and content (CaO_2) very high.²

The next problem is that the solubility of O_2 in water (and plasma) is low. The solubility constant obeys Henry's Law: it is directly proportional to O_2 partial pressure at the air-liquid interface as well as inversely proportional to the temperature—cold water holds more O_2 than warm water.⁴ At sea level (760 mm Hg) and room temperature (20°C), O_2 solubility in water is ~0.68 ml/dl. The O_2 content of bottled water can be raised by increasing the O_2 partial pressure, but when the pressure returns to atmospheric—for instance, when the bottle is opened— O_2 off-gases just like the CO_2 fizz in a carbonated beverage. The aqueous O_2 content rapidly falls to the lower equilibrium.

Problem three is that it is not known whether O_2 is actually absorbed from drinking water, hyperoxygenated or not. Perhaps some O_2 enters the portal circulation, and then the venous circulation, to

raise venous PO_2 . But the intestine, unlike the lung, is not designed for gas exchange, and O_2 absorbed in this way would have a negligible effect visceraally or on systemic oxygen delivery (DO_2). At a normal CaO_2 of 20 ml/dl, only 0.3 ml/dl (1.5%) is dissolved in plasma. Basal DO_2 is ~1000 ml/min, and at a $\dot{V}O_2$ of 3.5 ml O_2 /kg/min (1 MET), the O_2 extraction ratio is ~0.25; therefore, to supply this amount for one minute to the average sized person, a drink would have to provide ~250 ml of rapidly absorbable oxygen.

How much O_2 is present in the oxygenated water sold commercially? Hampson *et al*⁵ measured the O_2 content of five brands of "hyperoxygenated" water and compared them with plain water. Four of five brands had values above plain water, and one was similar. The highest values in sealed glass bottles were nine times those of plain water and 80 ml O_2 /l (STPD). However, air is 20.9% O_2 , and a normal human tidal volume of 500 ml contains roughly 100 ml O_2 . Thus a breath of fresh air contains more O_2 than a litre of hyperoxygenated water.

Does this amount of O_2 influence exercise performance? Three controlled studies on oxygenated water and performance say not.^{5–7} Willmert *et al*⁶ measured resting, submaximal, and maximal $\dot{V}O_2$, circulatory variables, and blood lactate in 12 subjects who randomly consumed 500 ml of either oxygenated or plain bottled water. No differences were detected between conditions for any variable at rest or in exercise. A second part of the study found nothing to suggest that oxygenated water facilitated recovery from exercise. An independent, randomised, double blind crossover study of 11 subjects found no differences in $\dot{V}O_{2\text{MAX}}$ or associated cardiorespiratory performance variables after drinking oxygenated or deoxygenated water of the same brand five minutes before exercise.⁵ Finally, a study of nine male recreational cyclists showed that oxygenated water did not improve performance even in mild acute hypoxia (P_{bar}

= 641 mm Hg).⁷ These studies all meet the physiological expectation that oxygenated water would not improve incremental exercise to $\dot{V}O_{2\text{MAX}}$ or recovery from strenuous exercise.

One abstracted study also found no exercise performance benefit, but noted SaO_2 to be higher with oxygenated water (91.3% v 87.3%) at the end of a standard endurance test.⁸ The O_2 content of the water was not given, but if 80 ml O_2 as mentioned above instantaneously entered the circulation, basal DO_2 would rise to 1080 ml/min. This would not increase SaO_2 (as haemoglobin is already essentially saturated) but would raise SvO_2 for ~20 seconds before the O_2 was consumed. At 10 MET, however, 80 ml O_2 is consumed in about two seconds—too short a time to measurably increase SaO_2 or SvO_2 . Simply put, minute amounts of dissolved O_2 cannot explain an increase in SaO_2 in an endurance test.

In summary, oxygenated water fails both quantitative analysis and practical physiological tests of exercise performance and recovery. Only miniscule quantities of O_2 can be dissolved in drinking water compared with that required for exercise, and significant intestinal absorption of O_2 is unsubstantiated. Ergogenic claims for oxygenated water therefore cannot be taken seriously.

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Correspondence to: Dr Piantadosi, Box 3315, Duke University Medical Center, Durham, NC 27710, USA; piant001@mc.duke.edu

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