Blood Doping
Infusions, Erythropoietin and Artificial Blood

E. Randy Eichner
Health Sciences Center, University of Oklahoma, Oklahoma City, Oklahoma, USA

Abstract

As science marches on, athletes and coaches march close behind. Researchers have long been interested in how red cell mass and blood volume affect exercise capacity. Interest in blood doping soared after the 1968 Mexico City Olympics. Studies in the 1970s and 1980s suggested that transfusing red cells could speed endurance performance. Diverse athletes of the time were accused of blood doping. In the late 1980s, recombinant human erythropoietin (EPO) began to supplant transfusion for doping. EPO use is a suspect in nearly 20 deaths in 4 years in European cyclists. In the 1998 Tour de France, a team was ejected for using EPO and six other teams quit the race. The beat goes on; in recent years, diverse endurance and sprint athletes have been caught or accused of using EPO. Tests to detect EPO are improving but are not yet foolproof. As EPO tests improve, blood transfusion is back in vogue and some athletes may have infused artificial blood. Tests for detecting artificial blood also exist, but it seems it will take widespread, year-round, unannounced, out-of-competition testing and stern penalties to deter blood doping.

What is the ideal haematocrit to win the marathon? The answer is unclear. Epidemiological evidence suggests that a low haematocrit, say 40%, is good for a long life. Yet, if you want to climb Mount Everest without supplemental oxygen, research suggests you may want a high haematocrit, say 60%. But, a high haematocrit confers a risk of grave problems from blood clots, a known danger in mountaineering. Much evidence suggests that some marathons (and other athletes) are blood doping to boost their haematocrit to 50% or more. Some of them are dying to win.1

1. Infusions

As science marches on, top athletes and coaches, forever seeking an edge, march close behind. And athletes, like the rest of us, are fascinated by blood. Just as gladiators of yore drank the blood of foes for courage, Olympians of today infuse the blood of friends for stamina.

Researchers have long been interested in how red cell mass and blood volume affect exercise capacity. The first study of blood doping was in 1947; it suggested that boosting the haematocrit to 55% or so by homologous transfusion made exercise at altitude easier.2 Interest in blood doping soared after the 1968 Mexico City Olympics (7300 feet), where most winners of endurance footraces hailed from the highlands. Kenya’s Kip Keino, for example, won the 1500m race, leaving America’s Jim Ryun gasping in his wake. The premise that drove the ensuing research on blood doping was that athletes from altitude had ‘thick blood’ that helped them win in ‘thin air’.

A noted study of doping appeared 4 years after those Olympics.3 Three men who had 800mL of
their own blood reinfused 4 weeks after it had been drawn had a 13% increase in haemoglobin level and a 9% increase in maximal oxygen uptake (\(\text{VO}_2\text{max}\)). On a brief, all-out treadmill run, their run time to exhaustion increased 23%. This and similar studies of the time were designed not to improve athletic performance but to probe the determinants of \(\text{VO}_2\text{max}\). Even so, and although this study was uncontrolled, one can imagine the fervor it stirred in the sports world.

Three studies in the 1980s that used freeze-preserved autologous red cells are noteworthy. In one, doping increased \(\text{VO}_2\text{max}\) by 5% and brief, all-out run time to exhaustion by 35%. In the second, doping cut mean 5-mile run time by 45 seconds. In the third, doping cut mean 10km race time by 69 seconds.[4]

None of these studies are ideal; each has confounders. Yet, these and other studies suggest doping works. At the elite level, a 10km runner from sea level may gain a few seconds by doping, especially racing at altitude. A few seconds separate first place from sixth place.

It was quickly noted that blood doping seemed to work. Blood doping was used (by a Finnish steeplechaser) as early as the 1972 Olympic Games. In the 1984 Olympics, seven US cyclists doped with blood from relatives or friends. In 1987, US skier Kerry Lynch admitted blood doping. The list goes on. German and Italian marathoners, among others, have been accused of transfusion doping, as have, for example, Soviet and Finnish cross-country skiers. However, proof is often lacking.[1]

In 1987, recombinant human erythropoietin (EPO) appeared in Europe and soon antiquated transfusion doping. However, recently, with EPO being detected by testers, transfusion may be returning. At the 2002 Olympic Winter Games, blood-transfusion equipment was found in a house used by Austrian skiers. US cyclist Tyler Hamilton has been banned for 2 years for transfusing the blood of another person, despite Hamilton's novel excuse to explain his two populations of red cells as a "vanishing twin." And several top racers withdrew from the 2006 Tour de France, tied to a doping scandal involving a haematologist in Spain and, according to media reports, the finding of EPO and 100 bags of frozen blood.

2. Erythropoietin

If transfusion doping works, so does EPO. In one study,[5] 20 male endurance athletes were given EPO or placebo for 4 weeks. Those who got EPO had a rise in haematocrit (43% to 51%), a 7% rise in \(\text{VO}_2\text{max}\) and a 9% increase in time to exhaustion in a brief, incremental cycling test. The ergogenic benefits lasted up to 3 weeks after EPO was stopped.[5]

However, too much EPO can drive the haematocrit too high for safety by turning the blood to 'mud' that easily clots. Within 4 years after EPO appeared in Europe, nearly 20 top European cyclists died, suddenly and unexpectedly. EPO was a key suspect in many deaths.[6] Cyclists at first denied using EPO, but at the 1998 Tour de France, a masseur for the Festina team was caught with EPO and other banned drugs. Festina was ejected from the race, six other teams quit and eventually seven of nine Festina cyclists admitted doping. Marco Pantani won that 1998 race, but was ejected from a race in Italy the next year for signs of EPO use (media reports claim in a 1995 race his haematocrit was 60%). He died in 2004.

The beat goes on. Chinese runners, swimmers and rowers have been caught on EPO. Russia's top female cross-country skier and two other skiers were caught using a long-acting EPO, darbepoietin, in the 2002 Winter Olympics. Six of Finland's top skiers and two top German runners were caught using hydroxylstarch as a blood-diluter, likely to mask EPO use. Russian and American female runners, even sprinters, have been suspected of EPO; one sprinter, Kelli White, admitted EPO. Lance Armstrong has always denied EPO, but two of his teammates from the 1999 Tour de France, the first of Armstrong's record seven wins, admitted they used EPO for that race. EPO has even been used in American football, to increase haematocrit in thalassemia minor.[7]

Detecting EPO use is not easy. Pre-race haematocrit checking, with a cutoff that prevents
racing, may prevent deaths, but may encourage athletes to 'dope up to the line' and/or dilute their blood via saline or HES. Blood profiles to suggest recent EPO use are problematic.\textsuperscript{[8]} A urine test, immunodetection of blotted EPO after isoelectric focusing, can detect exogenous EPO,\textsuperscript{[9]} but recent events suggest it is not foolproof. If a reliable test is used year-round in unannounced, out-of-competition testing, it may cut EPO use.

3. Artificial Blood

Reports of athletes using artificial oxygen carriers ('artificial blood') are few and poorly documented. One Swiss cyclist spent 10 days in intensive care in 1998 with a mysterious illness said to be from infusing a perfluorochemical (PFC). Rumors are that other athletes have infused PFCs to increase endurance. But, PFCs are toxic and make no sense for athletes, because to function well as oxygen carriers, PFCs require high percentages of inspired oxygen, obtainable only with a tight oxygen mask, as in the hospital setting.\textsuperscript{[10]} Haemoglobin-based oxygen carriers such as Hemopure\textsuperscript{®} \textsuperscript{1} are less toxic than PFCs and deliver oxygen better, but they too have can have noxious adverse effects and have no place in sports.\textsuperscript{[10]} Alas, too many athletes, with high but false hopes, use new chemicals that may be unsafe. So, just in case, detection methods for haemoglobin-based oxygen carriers have been developed.\textsuperscript{[11]}

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E. Randy Eichner is a consultant to Gatorade Sports Science Institute.

References


Correspondence: Professor E. Randy Eichner, Health Sciences Center, University of Oklahoma, 5505 N. Stonewall Drive, Oklahoma City, OK 73111, USA.
E-mail: Reichner1@cox.net

\textsuperscript{1} The use of trade names is for product identification purposes only and does not imply endorsement.