

Tainted Glory — Doping and Athletic Performance

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Is it possible for the “natural” athlete who competes without chemical assistance to achieve record-breaking performances in sports requiring strength, power, speed, or endurance? Because doping tests are infrequently positive in international sports, it has been widely believed that the answer is yes — and that few athletes competing in major sporting events, including the Olympic Games and the Tour de France, use performance-enhancing drugs. But multiple sources of evidence, including personal testimony^{1,2} and an ever-increasing incidence of doping scandals, suggest the opposite: that widespread use of performance-enhancing drugs has fundamentally distorted the upper range of human athletic performance.^{1,3-5} Unfortunately, a global code of silence has kept the problem hidden from public view.^{4,5}

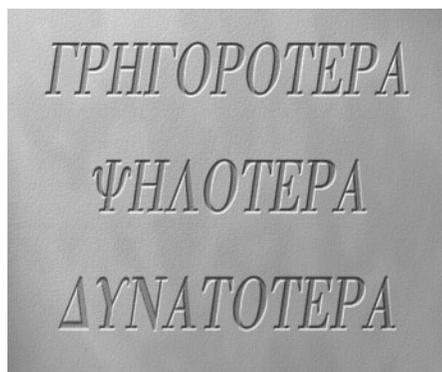
Drugs have been in sports for a long time. In the earliest modern Olympic Games, the drugs of choice included strychnine, heroin, cocaine, and morphine,⁴ which were probably more harmful than helpful. The first “effective” performance-enhancing drugs, the amphetamines, which were used widely by soldiers in the Second World War, crossed over into sports in the early 1950s.⁴ These drugs — nicknamed *la bomba* by Italian cyclists and *atoom* by Dutch cyclists — minimize the uncomfortable sensations of fatigue during exercise. By setting a safe upper limit to the body’s performance at peak exertion, these unpleasant sensations prevent bodily harm. The artificial manipulation of this limit by drugs places athletes at risk for uncontrolled overexertion. The first cases of fatal heatstroke in athletes using *atoom* were reported in the 1960s. In the 1967 Tour de France, elite British

cyclist Tom Simpson died on the steep ascent of Mont Ventoux, allegedly because of amphetamine abuse. The precise extent to which amphetamines enhance athletic performance is unknown, since, as with all performance-enhancing drugs, there are few modern studies quantifying their effects. The convenient absence of such information represents further evidence of a hidden problem. A popular opinion is that *la bomba* can turn the usual Tour de France *domestique*, or support rider, into a stage winner.

Since amphetamines must be present in the body to be effective, the sole method of avoiding the detection of their use during competition is to substitute a clean urine sample for the doped specimen. A multitude of innovative techniques have been developed to accomplish this swap.²

Cortisone, a potent but legal performance-enhancing drug used to dampen inflammation, also reduces the discomfort of heavy daily training and competition and lifts the mood. It is also widely abused by professional cyclists.²

Testosterone propionate (Testoviron), the prototype of the anabolic steroids, the second major group of potent performance-enhancing drugs, was synthesized in 1936 and appeared in sport sometime after the 1948 Olympic Games. The subsequent synthesis of methandrostenolone (Dianabol) in the United States in 1958 and oral chlordehydromethyltestosterone (Turinabol) in East Germany after 1966 marked the beginning of the “virilization” of modern sport.⁴ By increasing muscle size, these drugs increase strength, power, and sprinting speed; they also alter mood and speed the rate of recovery, permitting more intensive training





Photograph by Damien Meyer/AFP/Getty Images.

and hence superior training adaptation. For maximal effect, anabolic steroids are used in combination with other hormones that have similar activity, including insulin, growth hormone, and insulin-like growth factor. They have multiple side effects, some of which are serious, including premature death. The true extent of the use of performance-enhancing drugs is uncertain for a variety of reasons: athletes avoid detection by using scheduled testing for illicit drugs to plan their drug use^{1,3,5}; those conducting “out-of-competition” testing of athletes may intentionally avoid testing known drug users^{1,3,5}; hormones such as testosterone and insulin are initially undetectable, since they are so similar to the naturally produced substances, and designer drugs such as tetrahydrogestrinone (THG) are initially developed specifically to elude detection by all the current testing protocols; and positive tests are often not reported, and even proven drug users are generally not prosecuted.¹⁻⁵

The exact magnitude of benefit from the use of combined anabolic agents is unknown. Previously secret East German records indicate that anabolic steroids alone reduce 100-m sprinting time by as much as 0.7 second and improve performance in the 400-m, 800-m, and 1500-m running events by 4 to 5, 5 to 10, and 7 to 10 seconds, respectively.^{1,3} Equivalent benefits have been found among swimmers.³ Effects in throwing events are also substantial: a gain of 2.5 to 5 m in the shot put, 6 to 10 m in the hammer throw, 8 to 15 m in the javelin throw, and 10 to 20 m in the discus throw.^{1,3} Benefits are greatest in women, since the natural secretion of testosterone in young women is negligible.

The third type of potent performance-enhanc-

ing drug is erythropoietin, the hormone that regulates the red-cell mass. A popular theory holds that performance during high-intensity exercise is limited by the rate of oxygen delivery to the exercising muscles. By increasing the red-cell mass and hence the oxygen-carrying capacity of the blood, erythropoietin should increase performance only during all-out exercise lasting a few minutes. Yet it spectacularly increases performance in events that last anywhere from minutes to hours² and in events in which oxygen delivery is not the primary determinant of performance. It therefore seems likely that erythropoietin has another type of action, with effects on the brain that may resemble those of amphetamines, cortisone, and anabolic steroids. Currently, erythropoietin can be detected in the urine for only a few days after the most recent injection, although the related blood changes (in particular, the increase in red-cell mass) last much longer. Indeed, the benefits of even a short course of erythropoietin may last for many weeks.

The dangers of erythropoietin use include sudden death consequent to a fatal reduction in the heart rate, usually at night, and the development of antierythropoietin antibodies, which may cause a paradoxical reduction in the red-cell mass (pure red-cell aplasia). Eighteen young professional cyclists died from unknown causes in the late 1980s, when erythropoietin was first introduced into the world of cycling. Eight additional unexpected deaths of professional cyclists have been reported since January 2003, including that, reportedly from an accidental cocaine overdose, of Marco Pantani, the winner of the 1998 Tour de France who was banned from cycling after testing positive for markers of erythropoietin use while leading the Tour of Italy. The widespread use of performance-enhancing drugs may be associated with an increase in the use of recreational drugs by some of the same athletes.²

Performance-enhancing drugs pose a great threat not only to the health of users, but to the moral integrity and hence the continued relevance of modern sport.^{4,5} For, when used by fully trained, elite athletes, these drugs can improve performance to a much greater extent than any combination of the most intensive, sophisticated, and costly non-pharmaceutical interventions known to modern sports science.¹⁻⁴ Scientifically based training regimens, special diets, and complex physiological and biomechanical measurements during exercise and recovery cannot match the enhancing effects

of drugs. The attraction of performance-enhancing drugs is simply that they permit the fulfillment of the mythical promise of boundless athletic performance^{1,4} — the hubristic “faster, higher, stronger” motto of the Olympic Games. An ethically based medical science cannot compete. Thus, drug use in a subgroup of athletes who — even in the absence of drugs — are able to compete at an elite level causes their separation into a distinct athletic population, distanced from “natural” humans by a margin determined by the potency of the drug combinations that are used. These athletes, quite simply, have moved off the natural bell-shaped curve of normal human performance.

In disclosing his own drug-enhanced performances, former Australian world discus champion Werner Reiterer, who chose to retire rather than risk winning a tainted medal in the 2000 Olympic Games in Sydney, has written, “There was something pathetically wrong with the fact that a packed home arena — an entire country — would urge me

on without any concept of the truth behind my ultimate athletic achievement, or of the sham of which they were unwittingly a part.”¹ Our burden is that no longer do we share this ignorance. We can no longer pretend that we do not know.

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Building a Bridge to Heart Transplantation

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He who saves one life, saves the world entire.

— Hebrew proverb, quoted in
Thomas Keneally, *Schindler's List*

End-stage heart failure, characterized by marked symptoms at rest or with minimal activity despite optimal therapy, is designated as stage D heart failure. Frequent, recurring exacerbations may often be treated successfully, but decline is inevitable and life expectancy with medical therapy alone is short (survival rates are below 50 percent at one to two years) (see Figure). The addition of palliative measures, such as continuous infusions of inotropic drugs and hospice-like care, may be considered. Cardiac transplantation or permanent mechanical circulatory support is possible only in a select few patients.

When candidates for heart transplantation have hemodynamic deterioration, metabolic, cellular, and nutritional compromise follow, and the likelihood of survival after transplantation diminishes.

Even among patients with moderately compromised function, the likelihood of post-transplantation survival exceeds 80 percent at one year. However, as hemodynamic compromise progresses from moderate to severe, not only is there an increase in the risk of dying before transplantation can be performed, but the results after transplantation also worsen. The timely use of mechanical circulatory support halts further deterioration; decreases the likelihood of death before transplantation can occur; and reverses metabolic, cellular, and nutritional compromise. The temporary use of such support thus permits heart transplantation with a greater expectation of long-term survival and a better quality of life.

The temporary use of mechanical circulatory support before transplantation, known as “bridging,” is not to be confused with mechanical circulatory support intended from the outset to be permanent treatment, known as “destination therapy.” Bridging is reserved for candidates for transplantation,