

# Anti-doping cheating? The manufacture of truth in the war against drugs in sport

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“Among the calamities of war may be jointly numbered the diminution of the love of truth, by the falsehoods which interest dictates and credulity encourages”. Samuel Johnson in *The Idler* n. 30, 11 November 1758, quoted in Knowles, 1999: 409.

## Introduction<sup>1</sup>

The Danish sports scholar Verner Møller has labelled the anti-doping campaign “a crusade that must not fail” (2010: 72), one in which seemingly “the ends justifies the means” (2010: 77). He advances the hypothesis that the officials who are leading it might be regarding “the fight against doping as a war in which a form of emergency situation prevails” (2010: 84). This is not certainly an improbable stance, as one of the favourite metaphors of the anti-doping campaigners themselves is the one which compares or equates their efforts to eradicate doping with a war –one which must be won at any cost because if not, “then sport –to use the dominant discourse of the area- will be at the mercy of the needle” (2010: 73).

It could actually be argued that nobody launches a war thinking that it might be lost –victory is the only plausible outcome for warriors of any sort and place when entering the battleground. In such an ordeal, as Møller points out, very often exception becomes the norm and the ends justify the means. The value of empirical evidence, historical rigour and honest research, for instance, might be easily overthrown for the sake of the cause. As the cliché goes on, truth is the first casualty of war, and the war on doping is no exception in this point: the supreme cause of saving sports’ purity from the scourge of *drug cheats* and other related moral monstrosities might well be worth some academic laziness, some more or less conscious distortion of facts or even some outright inventions.

This article is heavily inspired by another of Møller’s contributions, his piece “Knud Enemark Jensen's Death During the 1960 Rome Olympics: A Search

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<sup>1</sup> The research in which this paper is based was in part conducted in Sydney, Australia, in late 2009 and early 2010 thanks to an honorary Research Visitor appointment kindly offered to the author by the Centre for Cultural Research at University of Western Sydney. I give warm thanks to CCR’s Director, Prof. Brett Neilson and the Centre’s efficient support staff for their hospitality and help, as well as to Prof. David Rowe for his academic guidance and his friendship.

for Truth?” (2005), as well as by the sports historian Paul Dimeo’s book *A history of drug use in sport* (Dimeo, 2007). The article by Brian Denham (1999) has also served as inspiration. These three texts are underpinned by a deliberate, open desire to unveil the historical truth concerning some of the most often repeated stories about the use of doping substances in sport since the late nineteenth century. In a daringly and unfashionable modernistic approach, these authors claim that things did actually happen one way and not another, and for some reasons and not other, that there is one truth to be discovered (or at least approached) in any account of these and any other past events, and that it *does* matter to reconstruct them as close as possible to their original factuality in order to make sense of them and make them better illuminate our current debates and concerns.

Møller’s article presents a thoroughly researched and very convincing revision of the circumstances surrounding the death of the Danish cyclist Knud Enemark Jensen in the Rome Olympics of 1960, a central event in the history of anti-doping as it “placed the anti-doping campaign on the agenda of sports politics –and, indeed, of politics as a whole” (2010: 37). Jensen’s decease being soon and almost unanimously attributed to amphetamine intake, his lifeless body became “proof of the health risks of doping” (Dimeo, 2007: 55). There is almost no historical approach to doping in the academic literature which fails to mention Jensen’s death and its link with the dangers of drug use by athletes. But, as Møller has demonstrated, all of these accounts fail to provide the essential evidence needed to substantiate such serious allegations, a demeanour which might be rightly considered as “scientifically irresponsible” (Møller, 2005: 462) and which almost degrade the resulting claims to the “category of gossip” (2005: 463).

Dimeo does a similar (if less developed than Møller’s) deconstruction of another of the accounts of a “doping casualty” usually put on the table by the anti-doping literature in its attempts at warning us about the fatal effects of doping: the case of the English cyclist Arthur Linton in the late nineteenth century, who allegedly died due to “an overdose of drugs” (Houlihan, 1999: 34). After revising the available evidence, which none of the authors sustaining this claim have done so far, Dimeo concludes that

we cannot say that his death was due to drug use because there is no evidence either way. We definitely cannot say that it was ‘recorded’ as such, or that it was in 1886 (...) Therefore we cannot interpret the fact of his death as proof of the continuity and health risks of doping in history (Dimeo, 2007: 8).

Denham (1999) deconstructed the dominant journalistic account on another alleged, if less mentioned in the academic literature, drug-related death, the one of American football player Lyle Alzado in 1992. Despite the fact that “medical science did not demonstrate a cause-effect relationship” between his brain lymphoma and steroid intake, “many journalists in print and broadcast media assumed a connection -and perhaps still do” (1999: 2), probably as a result of “the dramatic manner in which Alzado revealed his condition” (1999: 3) in a popular television show and in the magazine *Sports Illustrated*. This case analysis, according to the author, “addresses the manner in which media contribute to panic and hysteria with regard to drug use in Western society” (1999: 4).

The uncovering of the mythical nature of these stories is really bad news for the anti-doping campaign, because athletes, policymakers, the media and the public opinion could barely believe “the scaremongering tactics of [these] authoritarian discourses” (Dimeo, 2007: 110) if there would be shortage of victims to be put in the account of doping. It is indeed “astonishing” (Møller, 2010: 33) that the campaign has been in big trouble to put up a list of doping casualties, at least until the late eighties, which can live up to the dramatic claims concerning the lethal effects of performance-enhancing substances. In fact, most of the *expert* literature mentions little more than two recorded victims in the history of doping up to 1960, Jensen’s and Linton’s, despite the fact that it unanimously acknowledges that substance use (or “abuse”, as the campaigners often put it) is as old as sport itself and therefore goes back as far as the Olympics in ancient Greece (Møller, 2010: 33). To these names it is usually added the one of the British cyclist Tom Simpson, deceased during the dispute of the 13th stage of the 1967 Tour de France<sup>2</sup>. But that’s it.

The ethnocentric nature of most of this literature (Dimeo, 2007), and the narrowness of its source range, has resulted in it missing some possible victims to add to the list, namely those occurring in the fringes of rich, developed Europe. For instance, the Spanish cyclists Joaquín Polo and Raúl Motos, who died during the course of the second stage of the Volta a Portugal the 3rd August 1958 (*El Mundo Deportivo*, 4-8-1958). The Spanish press reports of the time mentioned sunstroke as the cause of these tragic deceases, but had them been known by the anti-doping campaigners, one can bet that they would have been attributed to the intake of amphetamines or any other sort of stimulant. Two deaths due to sunstroke in the same race and the same day, almost at the same time, could be easily considered an “incredible statistical aberration” which “continue[s] to defy any objective explanation” (Wadler, 1994: 440). “Suspicious (...) very suspicious” (hematologist Joseph

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<sup>2</sup> This tragedy has become one of the favourite stories of the anti-doping literature, once again despite the weakness of the available evidence concerning the allegations that it was caused by amphetamine intake.

Eschbach, quoted in Leith, 1991), one could claim. But, as in Jensen's and Linton's cases, there is not any available evidence which could support the link of these deaths with drug intake.

The quotes of Wadler and Eschbach above do not refer, of course, to the Volta a Portugal's tragedy, but to the alleged "sudden spate of deaths from heart failure among professional cyclists" (Waddington, 1990: 179) in Belgium and Holland between 1987 and 1990, or between 1988 and 1992, or in the late eighties and the early nineties, or between 1998 and 2000. In fact, there is a great variety of time spans being quoted for these deaths in academic and journalistic sources, as will be seen below. A total of 18 cyclists (or of 12, or of 5, or of 24, depending of the source), the claim goes on, passed away in "mysterious" circumstances (Kimmage, 2007: 245), in a short time span and "in just two countries" (Ed Berg, a doctor of sports medicine at the Olympic Centre, Colorado Springs, quoted in Leith, 1991). "The overwhelming probability" (Waddington, 2000) is that a new drug, recombinant human erythropoietin, rhEPO or EPO more plainly, would be involved in these deaths. This sensational story, if it were true, would solve at once and forever the problems of lack of credibility of the anti-doping campaign which might arise from the mentioned shortage of victims in the list of doping casualties, in such a sensitive point for the campaign as the allegations concerning the deleterious effects of doping substances. This might explain why it is one of the most repeated claims in the academic and journalistic literature concerning the issue, together with the one concerning the allegedly amphetamine-related death of Simpson.

But it is not true. Or at least, it is not truer than the claims about Linton's and Jensen's deceases. This article aims at demonstrating that it is actually another myth of the anti-doping campaign, its *flagship myth*, one could say, one which succeeded in actually scaring the athletes themselves (see for instance Skibby, 2006: 127, quoted in Møller, 2010: 47) and generating outcry and concern among scientists, journalists, policymakers and the public opinion worldwide. A new drug had appeared which could be convincingly charged with being the culprit of a "spate of sudden deaths among healthy, young athletes". The anti-doping campaign could at last not be presented as a war to only preserve the integrity or the spirit of sport, an argument which in itself doesn't seem to have the potential to awake the indolent masses and the lazy politicians to the horrific dangers of doping: it is actually about saving the very athletes' lives. As Randy E. Eichner, a haematologist in the University of Oklahoma and one of the first experts, if not the very first, to ring the alarm, dramatically put it: "I am not so much concerned with getting the athlete to the finish-line first, but with getting him there alive" (quoted in Powell, 1990).

## The multiple versions of the myth

The journalist and anti-doping campaigner Paul Kimmage, a former professional cyclist, opens the epilogue of his enthralling autobiography *Rough Ride* with this sensational story: “One month after the final chapter of *Rough Ride* was delivered to the publishers in 1990, professional cycling was rocked by a spate of sudden and mysterious deaths (...) In the six years since the death of Johannes Draaijer [a Dutch cyclist deceased in 1990], the sport had edged its way to the brink of the abyss”. Kimmage recounts this horror story contained in a report on EPO use in Italy in the early 1990s by the Italian doctor Sandro Donati:

There was the confession of the top Italian ‘Y’ and his explanation of how he had narrowly cheated death after a stage of the Tour of Italy. Boosted before the stage by an injection of EPO, he had gone to bed that night and slept peacefully for two hours, unaware that the oxygen-enhanced blood, flowing through his veins, was rapidly thickening to treacle. EPO is transformed into a lethal cocktail, not during a race when the blood is pumped around the body by a 180 beats-per-minute, high-revving, super-fit, heart rate but at night when the revs drop way below the norm. As Y’s pulse dropped to a low of twenty-five beats per minute, his blood began to clot and his heart began to stall. Had he not been sharing with a teammate, there is every chance they would have found him dead in the morning (...) Y lived to tell the tale. Others were not so fortunate (Kimmage, 2007: 246).

Robin Parisotto, a scientist who acted as principal researcher in the EPO 2000 Project at the Australian Institute of Sport which developed the first ever blood tests to be used at the Olympic Games (<http://www.sportingo.com/authors/robin-parisotto>), is no less sensational in his account of these events. In his book *Blood sports: The inside dope on drugs in sport* he lets us know:

Between 1987 and 1990, 18 cyclists died tragically and suddenly all from heart attack or stroke. EPO was known to thicken the blood –the common cause of heart attack or stroke. Many victims developed clots that broke off and travelled to their hearts or brains; others died of simple cardiac arrest, the organ struggling to pump blood the consistency of oil (Parisotto, 2004: 36).

A still more authoritative voice in the scientific field is, presumably, the one of Dr. David Gerrard, an Associate Professor of Sports Medicine, Chair of the New Zealand Sports Drug Agency and

member of the Board of the World Anti-Doping Agency (Gerrard, 2005: 7). Concerning the facts under discussion he explains:

During the 1980s and early 1990s the emergence of EPO as a performance-enhancing drug of choice by cyclists drew international attention when a number of young cyclists in Belgium and Holland died suddenly and inexplicably (...) When the affected arteries were those that supplied the brain or the heart, formerly fit young athletes were literally dropping dead from massive strokes and heart attacks. Post-mortem studies revealed the extent of the damage. How tragic that it took such graphic consequences to highlight the recklessness of such acts of drug abuse? (2008: 461-462).

These three testimonies have been selected as they can be considered to be representatives of the journalistic (Kimmage), the scientific-vulgarizing (Parisotto) and the scientific-academic (Gerrard) discourses. All three are coincident in the sensational and dramatic picture they offer concerning the facts under discussion, whose truth they do not seem to doubt in the less, but also in the utter lack of evidence to sustain their claims. Kimmage and Parisotto fail to even quote a single source, neither for such amazing contentions as that blood thickness “is the common cause of heart attack”, or that EPO works at such speed that hours after getting a shot of it your blood thickens “to treacle”. But this might be explained (although not excused) taking into account the divulging aim of the respective books. On the other hand Gerrard, in his academic paper published in *Sport in society*, does provide a source for his claims: a chapter of the book *Clinical Sports Medicine* (Clisby, 2001).

The latter reads as follows: “More recently, the use of the drug erythropoietin has allegedly become widespread among endurance sportspeople and may have contributed to the death of a number of European cyclists” (Clisby, 2001: 873). This author doesn’t quote any source for her claim, and unlike Gerrard, neither gives details of the nationality of the victims nor the period when these alleged deaths occurred. She is also more cautious concerning their relationship with EPO intake (“may have contributed”). And she doesn’t mention having accessed the “post-mortem studies” which “revealed the extent of the damage”. As in the case of Houlihan’s account of Jensen’s death (Houlihan, 1999: 36, quoted in Møller, 2010: 37-38), Gerrard is obviously adding data from its own (blurred) knowledge of these events, apparently quoting from hearsay rather than solid scholarship.

This lack of concretion and of evidence concerning these deaths is in fact the norm for the reviewed 31 academic texts which refer to these deaths: either they fail to quote the source of their claim, or they quote another scholar who fails to do so, or instead they (or the quoted academic source) refer

to a journalistic source which at its turn doesn't mention any solid source (or any source at all) to substantiate the claim. This has resulted in a quite unscholarly imprecision concerning the facts alluded to: number of victims (ranking from an indeterminate "several", to up to "about 20", with intermediate figures of five, twelve and eighteen being provided as well), countries of origin (the victims being referred to as European, or Dutch, or Dutch and Belgian, or even Scandinavian) and time span of the deaths ("during the 1980s and early 1990s", or "between 1997 and 2000", or "in the early 1990s", or "between 1987 and 1991") (see Table 1). The most repeated figures, though, refer to 18 Dutch and Belgian cyclists having passed away in a period spanning from 1987 to 1990-91.

A non exhaustive research of journalistic texts reporting on these facts (a total of 25: 23 articles published in some of the most prestigious western newspapers, an Associated Press wire plus a book chapter) reveal an even higher degree of dispersion and imprecision (Table 2). The number of victims rank from "half a dozen" to "around forty" (other figures mentioned: 7, 14, 15, 16, 17, 18, 24 and 34); the most often quoted countries of origin are again Holland and Belgium, but Spain, Germany and Poland are also mentioned, with many texts just referring to "European" cyclists; and the time span can be as broad as 1970-1990, and as narrow as 1988-90.

As for the link of these deaths with EPO intake, it is noteworthy that all but one of the reviewed academic texts refer to it in one form or another: either more cautiously (EPO "may have contributed", "was a key suspect", "presumably", "rumoured") or showing a high degree of security (EPO was involved "in all probability", they died of "erythropoietin-induced heart failure", "as a result of rEPO", "EPO was everywhere"). If one expects the academic literature to use and present data more cautiously and thoroughly than the media reports, one cannot but get struck by the fact that the journalistic texts analyzed here do show a higher degree of circumspection and prudence than the academic ones when it comes to establish a cause-effect link between these deaths and EPO intake, as all of them use conditional expressions like "might be", "linked anecdotally", "believed", "suspicious", "widely feared" (see tables 1 and 2).

This contrast between the academic and the journalistic texts is also evident when it comes to present counter-evidence or to further qualify the strength of the link deaths-EPO. Only 13 of the 31 analyzed academic texts qualify or somehow downplay it, referring for instance to the evidence being "circumstantial" or that the link "was never proven". Whereas up to 18 of the 25 journalistic texts present some kind of counterevidence or qualify the strength of the mentioned evidence, some of them openly ruling out the possibility of doping being the culprit of these deaths, and/or

mentioning other more plausible causes, like congenital cardiac ailments, arrhythmias, “irrational training methods” (Deblander, 1994) or the extreme efforts required by high competition.

### **In search of evidence**

A source analysis reveals that only two of the 56 reviewed texts (including academic and journalistic pieces) quote more or less *solid* original sources for the figures they provide, and both are newspaper, not academic, articles:

- Deblander, 1990a (*Le Soir*), quotes Ad Nuijten, “spokesperson of the Dutch federation”, who mentions seven deaths in Holland “in the last two years”.
  
- Deblander, 1998 (*Le Soir*), quotes Chris Goossens, a Belgian sports physician, who just mentions 17 deceases without any further chronologic or geographic detail.

Two other newspaper articles mention the same Randy E. Eichner mentioned above as the source for the data, but nothing is said concerning him having accessed the original European sources. And another one mentions a TV programme from the ABC network, neither an original source. The remainder of the 56 texts either don’t quote any source, or they refer to another article which doesn’t quote any. The obvious conclusion is that the claim about this “sudden spate of deaths”, which features in no less than 31 academic articles and 25 journalistic pieces from the most prestigious newspapers in the developed world, is very poorly substantiated, if it is in any way. No official statistical source is quoted other than the statements of a sports physician from one of the relevant countries, and an official of the Dutch cycling federation. And by the way, the figures they provide are non-coincident.

So what is the truth concerning the bare facts of these deaths (who, how many, were, when, why)? In the absence of a central register which recorded at that time this kind of events (Wadler, 1994), reaching this truth seems mission impossible. But one can try to approach it by means of other sources, for instance the information possibilities provided by the internet. Granted, this is not the most reliable source, but it is far more solid than mere hearsay. Table 3 gathers all the information about cyclists as victims of sudden death a thorough web search, crossing data from the Lexis Nexis database of newspaper articles, other newspaper archives, cycling sites and fora, and personal blogs, has yielded. The time span of the recorded deceases goes from 1970 to 2009.

The total figure is 54 cyclists: 27 Belgian, 12 Dutch, 3 French, 4 Italian, 2 British, and one each Australian, Colombian, Norwegian, Polish, Spanish and Swedish. Between 1987 and 1990, references to eight Dutch, three Belgian and one French rider have been found. Between 1991 and 1992, three Belgian, one British and one Dutch casualty have been mentioned. The total figures for the period 1987-1992 (six years) are: nine Dutch, six Belgian, one British, and one French. Taking into account the evident lack of statistical representativity and value of those figures, which are clearly biased by the “Dutch and Belgian” focus of the media reports of the time, it would seem reckless to draw any strong conclusion from them. Although this opinion is not shared by some experts quoted in the reviewed literature, who talk with little hesitation about a “statistical aberration” (Wadler, 1994; Fine et al, 1998) concerning figures whose reliability they utterly ignore.

Recipe to create an “statistical aberration”: you aggregate all these victims together in a single series and add some more to the actually occurred, to reach the figure of 18, 20 or even “two dozen” (Longman, 1998), and then you go on to refer loosely to the time span when they occurred as “recently” or “in the late eighties” and stress that they happened “in just two countries” (Randy E. Eichner, quoted in Leith, 1991). The resulting picture is quite impressive, indeed, and one must acknowledge that it even gives ground to be “suspicious”. But let’s put it in another, less sensational way: one Dutch victim in 1987, two Dutch in 1988, two Dutch in 1989, three Dutch, three Belgian and one French in 1990, one British and one Belgian in 1991, one Dutch and two Belgian in 1992. These sudden deaths were for sure a tragic event for the families concerned, but presented this way, they don’t seem to be such an estatistica aberration. Or are they?

Van Teeffelen et al. (1991, quoted in Carrière, 1992) estimated the incidence of sudden death in sport in the Netherlands at around 150 deaths yearly in the early 1990s. And this in just one country. Can in this context one or two deceased cyclists per year in Holland be considered a statistical aberration? Between 1995 and 2006, a total of 180 sudden deaths of athletes were officially recorded in Spain (Manonelles et al, 2007: 27) -an average of 15 a year<sup>3</sup>. The yearly figures were highly variable: from a maximum of 20 in 1998, to a low of 4 in 2006, 15 deaths having been recorded only one year before. 42 victims (23.3%) had 20 years or less. Of course, these are figures for the ensemble of the age brackets, levels of practice and sporting activities: cycling alone recorded a remarkable 39 deaths for the whole period, 21.66% of the total, but was second to

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<sup>3</sup> Note that the average figure is much lower than the one estimated for the Netherlands years ago. A possible explanation is that the medical safety measures (preventive health checks, availability of cardiac resuscitation equipments in the places where competitions are being held) might have been improved precisely as a reaction to these previous high figures. It is also noteworthy the fact that an official registry of sudden death in athletes was only set up in Spain in 1995.

football, which recorded 40 victims. In the Spanish case, none of these deaths was officially attributed to drug (let alone EPO) intake, but to a wide range of cardiovascular ailments, including coronary aetomathosis, arrhythmogenic cardiomyopathy, hypertrophic cardiomyopathy, congenital coronary anomalies, idiopathic left ventricular hypertrophy and aortic valvular esthenosis (Manonelles et al, 2007: 29).

Nonetheless, the Spanish official figures include a dose of mystery: the doctors failed to establish a cause for a total of 14 deaths, all of them of athletes below 30. Distrustful people could use this figure to speculate about the health dangers of doping, but it could also just raise a perplexed reflection concerning the persistence<sup>4</sup> of such a mysterious and tragic event as the sudden death of young (sometimes as young as babies in their cots), “otherwise healthy” (Scott and Phillips, 2005) persons. According to Manonelles et al., “it has been proven that in some cases [of sudden death involving people with a structurally normal heart] the alteration is to be found at a molecular level, in the electrolytes’ channels which determine the cardiac beat” (2007: 33; own translation from Spanish). Nothing is said about doping.

Given the sulphurous reputation of Spain as a *doping paradise* during the late nineties and the early XXist century, it might be the case that these official figures would not meet with much credibility among distrustful people. Let’s move then to the United States. Maron (2003) reports 387 cases of sudden death in young athletes recorded by the registry of the Minneapolis Heart Institute Foundation. Of these, only four were attributed to “drug abuse”, slightly over 1% of the total. 26.4% were caused by hypertrophic cardiomyopathy, 19.9% by *commotio cordis* (unleashed by a violent chest blow), 13.7% by coronary artery anomalies, etc. (other 16 cardiovascular ailments are mentioned).

On a broader geographic and chronologic framework, Bille et al. published a “systematic review of the literature” (2006: 859) concerning sudden cardiac death in young athletes, mentioning 1.101 reported cases in athletes under 35 years between 1966 and 2004. They do not provide a break down of this figure by countries, but one could presume that they include mostly western developed nations, whose populations are usually the object of the kind of research involved here. According to these authors, “forty percent [of the deaths] occurred in athletes under 18 years, 33% under 16 years” (2006: 859). Another intriguing finding is that cycling doesn’t feature among the three sports with a higher number of recorded deaths, which are “soccer (30%), basketball (25%) and running

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<sup>4</sup> An academic paper published in *The Lancet* mentions Pheidippides, the soldier who died in 490 BC immediately after delivering in Athens the news of the Greek victory in the battle of Marathon, as one of the first recorded victims of sudden death in athletes (Corrado, 2005).

(15%)” (2006: 859). And concerning the causes, Bille et al. report that “underlying cardiac diseases account for approximately 90% of exercise-related sudden deaths” (2006: 868). This brings these authors to dismiss doping as a cause of sudden death as “unlikely”, despite it being a common explanation among “the media and lay people” (2006: 268). And, one might add, among many scientists and scholars, as this research shows.

It is in the light of these geographically and chronologically broader, evidence-based scientific studies, that the figure of the 15 Dutch and Belgian cyclists who reportedly died of cardiac failure between 1987 and 1992, should be assessed. And this serene and unprejudiced assessment leaves little room for speaking about statistical aberrations. It is already a cliché that, when one is looking to statistics with the aim of finding a particular result, one is in serious danger of actually *finding* it. Therefore, shrewd researchers might find a new “suspicious statistical aberration” in the 12 deaths recorded in our own research between 2003 and 2004 (including five Belgian and four Italians), while *only* 12 have been recorded between 1993 and 2002. And it is indeed remarkable that, after all the hue and cry surrounding the alleged 18 Dutch and Belgian victims of the late eighties, no one has done so, probably because one such *series* was enough to make the point about the deadly dangers of EPO. Does this new “sudden spate of deaths” in cycling mean that EPO *abuse* in cycling suddenly skyrocketed around 2004 after years of *moderation*? And this in spite of all the efforts and the money poured into the WADA and the multiplying National Anti-Doping Organisations? Not probable...

### **Is EPO to blame for these deaths? The anecdotal evidence**

As it doesn't seem probable at all to attribute to EPO intake all or even a majority of the mentioned 15 Dutch and Belgian victims reported between 1987 and 1992. Let's previously leave aside the fact that it seems quite incongruous to attribute, as an article published in the *Los Angeles Times* does, the death of 15 “competitive cyclists from The Netherlands” between 1987 and 1990 to a drug which was not already approved in Holland as late as June 1990 (Almond, 1990). Peter Janssen, a former doctor of PDM, the team of Johannes Draaijer, wrote in his 2001 book *Lactate threshold training* the following concerning the possibility of EPO having caused these deaths:

this scandalous media story is simply untrue. These tragic deaths all occurred during the late 1980s and early 1990s, a time when EPO was not used yet (...) But if EPO was really as dangerous as alleged, many riders would have died from 1991 to 1997, because the use of

EPO was unlimited and uncontrolled before January 1997 (...) The 18 deaths would have nothing to do with this EPO story if they were not constantly used as an argument to intensify the campaign against doping in general and EPO in particular (Janssen, 2001: 190).

Quite a reasonable opinion, but it lacks empirical evidence and is not the one very likely to be believed by distrustful journalists and scholars who would probably be suspicious of one of the men who would be to *blame* if the EPO story were true, despite that, on the other hand, he would be one of the best positioned to know the truth for sure.

According to several sources, EPO began to circulate in Europe in 1987 (Leith, 1991; Longman, 1998), at a time when three of the recorded victims were already retired since long before. This reduces the list of potentially *suspect* deaths to 14. The case of Bert Oosterbosch should also be withdrawn from the list: the alleged deleterious effects of the drug, if he would have taken it during his professional career, would have faded long before dying, as he had retired one year before<sup>5</sup>. The list shrinks to 13. Four more victims had died between October and February, in the cycling off-season, when there is no point in “charging up” with such an expensive drug<sup>6</sup>, and one so cutting edge and therefore difficult to get hold of, so one can safely retire them from the list: it gets down to 9. Up to 7 victims were amateur riders, which for the same reasons just stated above would be very unlikely consuming the drug (See Christiansen, 2006a, for a discussion of why amateur riders are not prone to dope themselves). This leaves the list in just two potentially suspicious riders: one low-profile young pro (only 23 when he died) and a cyclo-crossman, none of the two the kind of usual suspect<sup>7</sup> for such a high-tech practice as EPO doping would be at that time. These data show therefore little, if any, trace of the “about 20 world-class Dutch and Belgian cyclists” killed by “rhEPO-induced erythrocytosis” (Jelkmann, 2002: 37). All this is not of course watertight scholarship, but at least one can safely say that it is based in (better or worst) empirical data and rational analysis, which is not the case for most of the academic and journalistic claims concerning these deaths.

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<sup>5</sup> According to most of the analyzed articles, death would be a direct consequence of blood turning into “mud”, “ketchup”, “oil” or “sludge”, to use only some of the dramatic expressions used in the reviewed literature, as a more or less immediate consequence of EPO intake, one which would quickly disappear once the effects of the drug had faded.

<sup>6</sup> Between \$4,000 and \$6,000 in the US for a year's treatment and 50 per cent more in Europe, according to Daniel Vapnek, senior vice-president of Amgen, the American manufacturer of the drug, quoted in Powell, 1990. Another source mentions a cost of between US\$ 5,000 and 10,000 (Specter, 1990).

<sup>7</sup> Patrice Bar was in his first year in the professional ranks. Neo-professionals are very seldom paid the high salaries the stars get, and which are needed in order to afford the most expensive doping products. Cyclo-cross is not an endurance discipline, as the races seldom last for longer than one hour. There is little point, therefore, for cyclo-crossmen consuming the endurance-booster EPO.

But let's put speculation aside for a while and listen to what science has to tell us concerning the causal link between EPO and sudden death. One of the experts most often quoted in the reviewed journalistic and academic literature is the mentioned Randy E. Eichner, "a hematologist at the University of Oklahoma Health Sciences Center" (Noden, 1990). Eichner is the author of possibly the most repeated metaphor on the dangers of EPO: if you abuse it, your "blood becomes mud" (quoted in Noden, 1990). The German current affairs magazine *Der Spiegel* liked it so much that used it to title an article on the issue: "Schlamm in den adern" (10-6-1991, p. 191). A prestigious hematologist, Eichner could be praised as well for his outstanding communication abilities, as shown as well in the ear-catching titles he chose for some of his papers and conferences: "Better dead than second" (Eichner, 1992b), or "Dying to win"<sup>8</sup>, a title which pre-dated by eight years its namesake, the well known book by the anti-doping scholar Barrie Houlihan (1999). And who have already heard about his concern with getting the athletes alive to the finish-line. In his 2007 paper he lets us know in striking but not very scientific fashion that "athletes, like the rest of us, are fascinated with blood. Just as gladiators of yore drank the blood of foes for courage, Olympians of today infuse the blood of friends for stamina" (2007: 389). Although he left also traces that he might not be as good as a cardiologist or a sports historian, for instance when he was quoted in *The Independent* as saying that "cycling is not dangerous for your heart. It's healthy for your heart (...) people have been cycling for 500 [sic] years without anything like this [sudden deaths] happening" (quoted in Leith, 1991).

Eichner didn't seem to be in much doubt concerning the causes of these deaths. According to *The Independent*, the wife of Johannes Draaijer, who had died in his sleep in January 1990, stated that "her husband was against doping, that he was well known in the peloton as a non-user", and that "the [post-mortem] investigations showed nothing" which could relate Draaijer's death with doping (Leith, 1991). But Eichner, thousands of kilometres away in his Oklahoma City headquarters, seemed to know better, as he didn't hesitate to dismiss Lisa Draaijer's opinions as "just a cover-up (...) just something they've brainwashed her with" (quoted in Leith, 1991). Still more arrogant were the statements to *The Globe and Mail* of Norman Gledhill, an exercise physiologist at York University in Toronto who seemed to be much better informed about the causes of these deaths than the very Dutch doctors and officials involved in cycling. Replying the opinion of Frans Stoele, a spokesman for the Netherlands Centre for Doping Affairs, that "there weren't any EPO-related deaths here", Gledhill was quoted as saying: "there's either been a massive coverup or the people who did [the investigations] were massively incompetent. I think the coverup is the likely answer"

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<sup>8</sup> A conference he delivered in May 1991 to the Congress of the American College of Sports Medicine concerning the dangers of EPO, according to *Der Spiegel*, 10-6-1991: 198.

(Fine et al., 1998). Others, like Ed Berg, a doctor of sports medicine at the Olympic Centre, Colorado Springs, and Joseph Eschbach, a haematologist at the University of Washington Medical School, Seattle, were quoted (Leith, 1991) at that time as joining the choir of North-American *experts* apparently suspicious of the explanations of their Dutch counterparts.

### **Is EPO to blame for these deaths? The scientific evidence**

Such a derogative attitude towards the opinions which denied or downplayed the possibility of these deaths being related to EPO could only be reasonably explained by the fact that the dismissive doctors would be in possession of a deep scientific knowledge, therefore including sufficient amount of empirical evidence, pointing in the opposite direction. But neither Gledhill nor Eichner mention any such evidence in their dramatic statements to the press, of the kind: “the use of EPO by athletes can result in ‘blood clots and obstructs the vessels, turning it into a mass as solid as stone’” (Eichner, quoted in Jollymore, 1992). The wise words of the then president of the Dutch Cycling Federation, Frank Buddels, that “there needs to be a lot of research before you can say concrete things” (quoted in Leith, 1991), didn’t seem to have impressed them much.

Nonetheless, Eichner did not limit himself to speak to the press. We approach an academic paper he published in 1992 with the hope of finding in it a more evidence-grounded justification for his emphatic claims:

For athletes abusing rEPO, this potential rise in exercising blood pressure, along with the expected rise in hematocrit in blood viscosity, has obvious and ominous implications (...) It seems plausible that, for example, a cyclist taking rEPO might start a race with a hematocrit of 55-60%. Also plausible is that, because of sweating, dehydration, and hemoconcentration, he might end the race with a hematocrit of 60-65%. Surely, the combination of high hematocrit, elevated blood viscosity, and dehydration –along with the hours on the saddle– would increase the risk of thrombosis in the veins of the pelvis or legs. This could lead to a fatal pulmonary embolus. These same elements, plus an accentuated rise in exercising blood pressure, may also increase the risk of stroke or, possibly, heart attack (...) Athletes and coaches must learn this: Abusing rEPO can kill you. What price glory? (Eichner, 1992: S317).

This seems at first sight a convincing explanation closed with some impressive rhetoric, but a second glance reveals that what is still missing here is empirical evidence. This piece belongs to the “I think that...” kind of scholarship, one which can hardly be labelled as scientific, or at least one which should not expect to raise much credulity, being based only in expressions like “potential”, “obvious”, “plausible”, “surely”, “might”, and “would”. It might be the case that such an evidence was not available at that time (but there was some, as will be seen below, although it pointed in the opposite direction), so let’s move forward in time to an article Eichner published in 2007, where he reproduced the same metaphor that made him almost famous in the early 1990s: “too much EPO can drive the haematocrit too high for safety by turning the blood to ‘mud’ that easily clots” (2007: 390). This time he quoted a source to sustain his claim: an article by Scott and Phillips (2005).

These authors do indeed state that “hyperviscosity associated with high hematocrit levels increases the risk of thrombotic events such as stroke and myocardial infarction. The unexplained death of 18 otherwise healthy cyclists between 1997 and 2000 have [sic] been linked to rEPO by some sources” (2005: 225). But they also fail to quote any scientific evidence or source for these claims.

Interestingly, this is not the case for other less dramatic side effects of the drug they mention, like “fever, nausea, headache, anxiety, and lethargy”, as well as “hypertension (...) in patients of hemodialysis” (ibid.), for which they refer to Denker (2004). This author mentions these side effects, but it also states that “early studies revealed conflicting findings for increases in vascular access thrombosis” (Denker, 2004: no page numbers). Of the two sources Denker provides for this claim, one doesn’t actually refer to vascular access thrombosis (Evans, 1990), and the other one, an empirical research involving 79 patients during 5.25 years, demonstrates that “epoetin [EPO] does not increase vascular access clotting” (Besarab et al, 1990, stress added).

To sum up, if one goes down the chain of quotations started by Eichner himself in his 2007 article, one finds not only a lack of empirical evidence for his claims about the fatal effects of EPO, but even an article based in original research which clearly refutes Eichner’s contention about the casual link between EPO and blood clotting. One which was published the same year his alarming claims were being aired by the *sudden spate* of newspaper reports of the early 1990’s which dramatically reported about the mentioned deaths (see for instance Almond, 1990; Reuter, 1990; Powell, 1990; Noden, 1990; Anonymous, 1991; Leith, 1991; Jollimore, 1992).

This lack of scientific evidence is not only a feature of Eichner’s statements, though. There have been found 25 academic texts providing some kind of explanation concerning the deleterious effects of EPO abuse, most of them in line with Eichner’s contention, as in fact some of them have Eichner

himself as their original source. For instance, Tokish et al. (2004: 1548) state that “raising one’s hematocrit beyond physiologically normal levels leads to an increase in blood viscosity, thrombogenic potential, and myocardial infarction risk”. The source these authors provide as the basis for this claim is Ramotar (1990), who quotes... Eichner, without mentioning any further empirical evidence<sup>9</sup>.

A telling example of the poor empirical basis of the scientific claims about the health dangers of EPO can be found in the article by Deligiannis et al (2006), a literature review on the cardiac dangers of doping whose scientific credibility becomes immediately under suspicion when one reads in its abstract that one of the authors’ purpose is to “discourage individuals from using drugs during sports” (2006: 687). They nonetheless honestly warn that “only sparse data and isolated clinical cases are actually available” and therefore “it is difficult to know the real hazard and effects of doping drugs and methods upon the cardiovascular system, particularly during acute exercise” (2006: 688). This is evident in their own explanation of the adverse effects of EPO. They contend that “the misuse of rHuEPO causes increased viscosity of the blood which, in combination with the elevated hematocrit, leads to increased risk of thrombosis and embolisms”. They quote Vergouwen et al. (1999) as the source of this claim, but these authors don’t mention these side effects in their article. According to Deligiannis et al., “a fatal reduction of heart rate during the night has also been reported” as a consequence of EPO “misuse” (2006: 690), a claim for which they quote Noakes (2004), who actually says the same (2004: 848) but doesn’t provide any empirical evidence nor quote for this claim. Finally, Deligiannis et al. also state that “increased afterload, elevated blood viscosity and the loss of hypoxia-induced vasodilation lead to arterial hypertension and perhaps to cardiac dysfunction” (2006: 690), quoting Wagner et al. (2001) as their source. But the same claim cannot be found in this article, rather the opposite: in their original research with transgenic mice they found that “most surprisingly (...), despite a hematocrit of 0.80, no increase in blood pressure and/or a reduction of cardiac output in the erythrocytotic mice was observed” (Wagner et al., 2001:

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<sup>9</sup> It is interesting to note that Eichner was a member of the editorial board of the journal where Ramotar published her article, *The Physician and Sportsmedicine*, one of the first, if not the very first, scientific publications to ring the alarm of the alleged health dangers of EPO abuse by athletes: in its August 1989 issue, prior to the mentioned *sudden spate* of press reports, Thomas H. Murray, another member of the journal’s editorial board and a member of the U.S. Olympic Committee’s panel on substance abuse research and education (Chen, 1990), published an article with the title “Erythropoietin: another violation of ethics” (Murray, 1989). He warned that EPO, together with other drugs, could be a “danger to the athlete’s health and life” (1989: 39), again as a self-evident truth without any empirical support. In fact Murray’s article, as shown in its title, doesn’t deal with the health consequences of doping, but almost entirely with its alleged moral wrongness, and does so in the typical evangelical tone of most of the anti-doping literature (Dimeo, 2007), for instance when he dismisses the sports system that tolerates doping as “corrupt and corrupting” (1989: 39) or asks for life bans to be dished out to “officials, coaches, physicians, and others who tolerate or encourage drug use” (1989: 42).

540). They state as well that “continued exposure to hypoxia causes vasoconstriction”, not vasodilation (2001: 536).

15 out of the 25 reviewed articles fail to provide empirical evidence nor quote any source for some or all of their claims concerning the fatal side effects of EPO intake, while two of them just mention, as their only source, the statements or the opinions, lacking empirical evidence, of anti-doping experts like Eichner, Don Catlin (director of the Olympic Analytical Laboratory at the University of California, Los Angeles) and Robert O. Voy (medical director of the Las Vegas Institute of Physical Therapy and Sports Medicine and former director of medicine and sport science for the US Olympic Committee) (Cowart, 1989). Six more articles refer to other pieces which either fail to quote a single source, do not mention these side effects or do quote some of the mentioned experts. And one article (Martínez Lage et al, 2002) could hardly be considered a scientific research, as it provides evidence for a single case of an elite cyclist in which it was found a coincidence of thrombosis and intake of EPO and growth hormone, with no further proof of the casual link between the two being mentioned, despite the authors claiming that “the use of EPO to increase PCV in athletes suffering from other predisposing factors to thrombosis, such as erythrocythemia due to dehydration during exhausting exercise, could lead to serious side effects” (2002: 665). This leaves in fact the list down to two articles based in original research, and one more quoting three articles based in original research (see table 4).

1. Wagner et al. (2001) report on own research in which they studied the effects of chronic high haematorit on cardiovascular functions in experimental transgenic mice. In these animals, with a hematocrit of 0.80 (wild type: 0.47), “cardiac preload was increased, echocardiography revealed a dilated left ventricle, and the histologic alterations of the heart indicate cardiac dysfunction developed left and right ventricular hypertrophy and cardiac oedema. Their life expectancy was greatly reduced (...) High hematocrit without accompanying hypoxemia appears sufficient to damage the heart, most likely because of the increased blood viscosity” (Wagner et al., 2001: 542).

2. Ekblom (2002) quotes original research articles (Berglund and Ekblom, 1991; Ekblom, Wilson and Astrand, 1976; Huang, Davis and Johns, 1993) to sustain his claim that “during submaximal exercise at 200 W (...), there was a marked increase in arterial systolic B[lood] P[ressure] from an average of 177 to 191 mm Hg” in experimental subjects having been treated with rhEPO (2002: 105). Interestingly, though, according to this author these studies show that “compared with the pre-EPO-administration period, arterial systolic and diastolic

blood pressure (BP) at rest remained unaltered after an rhEPO administration period” (2002: 105).

3. Besarab et al. (1998) “studied 1233 patients with clinical evidence of congestive heart failure or ischemic heart disease that were undergoing hemodialysis”. One half received EPO treatment to keep their hematocrit up to 42%, while the other half received EPO to maintain their hematocrit to 30%. The result was that the group with a higher hematocrit recorded a higher tax of myocardial infarction with a “risk ratio for the normal-hematocrit group as compared with the low-hematocrit group [of] 1.3”.

This is all concerning the real scientific evidence mentioned in the 25 reviewed texts to sustain the claims about the fatal effects of EPO abuse by athletes. And even these three articles are not without their problems for the champions of the theory of EPO as an “undetectable killer” (Chen, 1990).

The research by Wagner et al. has little transportability to the case of athletes using EPO, as their high hematocrits are not structural but only occur at punctual moments in the sporting season, when EPO is administered coinciding with major sporting events (Voet, 2002), and then go down to natural levels as soon as the treatment is discontinued and reach the original values only one month after the last EPO injection (Birkeland et al., 1999: 1240). On the other hand, I have not found in the reviewed literature references to actual hematocrit levels in humans of up to 80%, the highest *hypothetical* (not actually recorded) figure being the one provided by Eichner (1992): 60-65% in dehydrated athletes. It seems rather unlikely that a figure of 80% might be reached in humans by means other than those used in the unfortunate mice of Wagner and colleagues’ experiment.

The article by Besarab and colleagues refers to hemodialysis patients with structurally low hematocrits and diagnosed cardiac ailments, none of which apply to elite athletes. And Ekblom provides with empirical evidence of an increase in blood pressure in EPO-treated subjects, but only during “submaximal exercise”, not at rest, which poses a major problem for the theory that sudden death while sleeping, recorded in no less than nine cases in my own preliminary research (see table 3), might be due to EPO-induced hypertension.

Let’s turn now to the available scientific evidence which belies or somehow contradicts the theory of EPO as an “undetectable killer”. My literature review has spotted 10 articles providing such counter-evidence, all of them based in original research or quoting articles based in original

research, and one article presenting an exhaustive literature review of sudden death in young athletes:

1. Manonelles et al. (2007) reviewed the causes of 180 recorded cases of sudden death of young athletes in Spain between 1995 and 2006, without attributing a single one to drug/EPO intake.
2. Lundby et al. (2007) report about an experiment involving “eight healthy subjects receiving 5000 IU recombinant human Epo (rHuEpo) for 15 weeks at a dose frequency aimed to increase and maintain haematocrit at approximately 50%” (2007: 309). The results show that “*mean arterial pressure was minimally affected by rHuEpo treatment, while cardiac output and systemic vascular conductance remained close to their pre-Epo respective values. This corroborates previous studies showing that in the range of haematocrits observed in this study (from 42 to 49%), a small increase of haematocrit has no major impact on resting mean arterial pressure and hence on systemic vascular conductance (...). In agreement, this study shows that the flexibility of the red blood cells remained unchanged. Based on this, blood viscosity may be assumed to be relatively unchanged*” (stress added).
3. Bille et al (2006) carried out a systematic literature review on sudden death in athletes and concluded that “underlying cardiac diseases account for approximately 90% of exercise-related sudden deaths” (2006: 868) (1101 reported cases between 1966 and 2004), which makes them dismiss “the general idea that most sudden deaths in sports are related to doping” (2006: 872).
4. Denker (2004) quotes Besarab et al. (1998) and Abels (1992), both research articles, to sustain the claim that “there is no correlation between hypertension and the rHuEPO dose or achieved hemoglobin concentrations and hypertension is **not** seen in rHuEPO-treated patients without renal disease” (original stress) (Denker, 2004, no page numbers provided).
5. Maron (2003) states that “sudden unexpected death, nonfatal stroke, and acute myocardial infarction in trained athletes have been attributed to the abuse of cocaine, anabolic steroids, and dietary and nutritional supplements”, but EPO is not mentioned. He refers to three research articles to sustain this claim (Lange and Hillis, 2001; Samenuk et al, 2002; Valli and Giardina, 2002). On the other hand, he shows through own original research that of 387 sudden deaths of young athletes recorded in the register of the Minneapolis Heart Institute Foundation, only four (roughly 1%) were attributed to “drug abuse” (HTML version consulted, no page numbers provided).
6. See above for Ekblom’s (2002) claim concerning blood pressure at rest remaining unaltered in subjects having been administered EPO.

7. Boraita (2002) quotes two research articles (Jensen-Urstad, 1995; Weslen, 1996) to sustain her claim that “in American series, the main causes of [sudden] death in this age group [young athletes] are hypertrophic cardiomyopathy and congenital anomalies of the coronary arteries”, while in European statistics “arrhythmogenic right ventricular cardiomyopathy and myocarditis (...) [are] the most frequent pathologies. Other, much rarer, causes of death in young athletes are arrhythmogenic syndromes, cerebral vascular malformations, bronchial asthma, and *commotio cordis* syndrome”. Boraita doesn’t mention drug, let alone EPO intake, as a cause of sudden death in young athletes.
8. Wagner et al (2001), in addition to providing the evidence mentioned above which supports the theory that chronically high hematocrit damages the heart, reported as well on results from their experiment with transgenic mice which contradict the occurrence of some of the most often quoted alleged secondary effects of EPO intake, mainly hypertension. They explain that “most surprisingly (...), despite a hematocrit of 0.80, no increase in blood pressure and/or a reduction of cardiac output in the erythrocytotic [transgenic] mice was observed” (2001: 540). They go on to conclude that “this observation strongly argues against a direct hypertensive effect of EPO” (2001: 541). They furthermore report on an original research with rats by Petit et al. (1995) in which these authors found that “when hematocrit was raised over a 3-week period to a value of 0.63 by the administration of EPO 500 units thrice weekly, mean arterial pressure, left ventricular end-diastolic pressure, right ventricular peak pressure, and cardiac output did not differ from the values in control animals (hematocrit 0.47)” (Wagner et al., 2001: 540).
9. Pluim et al (1996) ground in own original research their claim that “the left ventricular hypertrophy in cyclists [one of the most frequent causes of sudden cardiac death], showing normal left ventricular functional and metabolic parameters, suggests physiological hypertrophy rather than a pathophysiological adaptation”. That is, it hardly could be attributed to drug/EPO intake as it is not adaptative. In their view, sudden deaths raise “questions about the longterm health aspects of extreme physical training” –doping is not mentioned (1996: 1277).
10. And finally, Besarab et al (1990) present own original research to conclude that “epoetin [EPO] does not increase vascular access clotting” (see above).

## Conclusions

If anti-doping is indeed conceived as a war on drugs in sport, as many of its supporters like to say in public, right and wrong is to be found in both sides, and not just right in the one (anti-doping's) and wrong in its opposite (Dimeo, 2007). Therefore, alongside "doping cheats" and "drug abusing athletes", one might expect to find as well among their enemies some anti-doping cheating and "drug abusing journalism" (Christiansen, 2006b) and scholarship. This article aims at joining other academic efforts (Denham, 1999; Møller, 2005; Dimeo, 2007) in the task of uncovering and exposing some of this abuse, in the form of distortion or outright invention of historical facts which have provided ammunition for the propaganda campaign which, as in any kind of war, is to be found underpinning and preceding most of the anti-doping institutions and policies which have been set up in the last 20 years. Of course, not every scholar or journalist who has reproduced these distortions or inventions is to be accused of deliberate falseness, as many of them have simply picked up and reproduced a story which, after having circulated for years in the media and the academic discourses, has *fossilized* and acquired the category of truth. But every myth has an origin and, very often, a concrete and calculating authorship.

This article has demonstrated that the story about the 18 Dutch and Belgian cyclists who allegedly died between 1987 and 1990 due to EPO abuse has no empirical basis. Even more, the available empirical evidence points precisely in the opposite direction: that this series of deceases has been artificially concocted and even inflated in absolute terms, and, most importantly, that EPO had nothing to do with it, for several reasons, the more important being that there is no empirical evidence at all that it causes sudden death. It should therefore be considered more of a myth or an invention than a historical fact. A myth which in the last years has played a central role in the "scaremongering tactics" (Dimeo, 2007: 110) of the anti-doping campaigners, who until the early 90s were quite short of casualties to be charged to doping *abuse*. Two of these deaths, by the way, have been proven to be falsely attributed to doping: Arthur Linton's (Dimeo, 2007) and Knud Enemark Jensen's (Møller, 2005).

56 academic and journalistic texts have been reviewed which mention these deceases. Very few actually quote a solid source from the relevant countries (a government registry, a healthcare or cycling official, a newspaper report listing the deaths in some detail) for the data provided: number of victims, nationality, age when died, level of practice of the sport and concrete date of each decease. Most of these data are actually missing in the reviewed texts. In the absence of an official registry of sudden deaths of athletes in the concerned countries, I carried out a web-based research of newspaper archives (mostly Dutch and Belgian, through the Lexis Nexis database) and cycling websites, which are certainly not the most reliable sources but are better than nothing. This research

has recorded 17 sudden deaths among cyclists being reported in the six years between 1987 and 1992, an average of 2.6 per year, including 9 Dutch, 6 Belgian, one British and one French. I have argued that, given the lack of statistical reliability of these data and the “Dutch and Belgian” bias resulting from the search premises themselves, it cannot be reasonably said that they constitute a “suspicious statistical aberration”. But even if they were awarded credibility, comparing them with data from other, scientific researches on sudden deaths in athletes conducted in Holland, Spain and the United States plainly refutes this idea.

Arguably, then, what has actually been going on here is a double process of invention. The one concerns the series of deaths and has been achieved through aggregation of isolated cases from two different countries and during an elastic time span, to which some more not documented deaths have been added in order to reach the magic figure of 18 “in just two countries” and in a seemingly short period of time. This already yields a “suspicious sudden spate of deaths” for which an explanation needs to be found. And here comes the second invention: EPO is isolated as a “key suspect” on the basis that it started to be released by the time this “spate of deaths” was allegedly taking place. No further evidence is brought forward other than this coincidence and the common sense-based contention that if EPO thickens the blood, too much of a thick blood “will kill you” (Eichner, quoted in Reuter, 1990).

When going down into further details, most of these experts (and the journalists relaying their opinions) explain that EPO intake might cause blood clotting, hyperviscosity and hypertension. But the truly scientific literature, the one based in experimental case studies, I have accessed doesn't support the contention that EPO causes blood clotting (Besarab et al, 1990) or hyperviscosity (Lundby et al, 2007). And concerning the increase in blood pressure, it has been only found during “submaximal exercise” but not at rest (Lundby et al., 2007; Denker, 2004; Ekblom, 2002; Wagner et al., 2001), which poses a problem for the theory that EPO is to blame for the sudden deaths of athletes while sleeping or out of competition. Epidemiologic studies on sudden death in Holland, Spain, the United States and, more broadly, in the developed world show that the immense majority of these deceases are caused by congenital cardiac ailments, while only a residual minority of them (1% in the American research, for instance) have been attributed to drug intake (Maron, 2003). And EPO is not mentioned among the culprits, by the way.

There are many other evidences that rule out or downplay the possibility of EPO being a cause of these sudden deaths, for instance the fact that the drug had not already been released in Holland by mid-1990 (Almond, 1990), or that the immense majority of the deaths occurred between 1988 and

1992 actually recorded in the press would have very unlikely taken EPO if it would have been available. All this doesn't mean, of course, that there is *absolute* proof that EPO didn't contribute to the deceases of these or any other athletes, but that the *existing* evidence doesn't support this contention, rather the opposite. To date, therefore, the casual link between EPO and sudden death doesn't seem to be more scientifically substantiated than the one between masturbation and blindness, a classical example of a false health argument put at the service of a moral campaign.

Finally, a point might be made about the origins of this myth. Where was it born and who confected it? The information provided by this research points at one publication and one person as its initial disseminators: the journal *The Physician and Sportsmedicine*, and the hematologist at the University of Oklahoma, Randy E. Eichner. It seems that the first time the health and moral dangers of EPO were mentioned in the expert literature was in the August 1989 issue of the journal. And one of the members of its editorial board, Dr. Eichner himself, was among those who more actively contributed during 1990 and 1991 to spreading the word about the alleged lethal effects of the drug, delivering papers in conferences and speaking to the press in effective and dramatic style. So effective that his statements were relayed by such prestigious newspapers as the *Los Angeles Times*, *The New York Times*, *The Times*, *The Independent*, *The Globe and Mail*, *Der Spiegel* and *Sports Illustrated*. One such conference was "rhEPO and athletics: playing a dangerous game", which took place in New York in 1990 and was sponsored by Amgen, the Californian company which patented rhEPO in the mid 1980s (Wadler, 1994: 442).

Speculation about the true goals of these and other anti-doping campaigners, companies and media outlets when contributing to spreading the story of the 18 Dutch and Belgian cyclists that EPO *killed* in the late eighties is quite pointless here. It might well be the case that their intention was the best one: to save athletes' bodies, and/or souls, and to save sport's reputation and/or purity from this new *scourge*. But this research shows that, rather than being standing on solid ground they were in fact "fighting the fog" (Møller, 2010: 12). As a result, what they actually achieved was, on the one hand, creating a fearsome, "silent killer" ghost, and on the other, a much more tangible outcome: a dramatic increase in the public awareness about EPO, of which can safely be said that enormously boosted its legal –and illegal consumption throughout the nineties. As Møller has aptly pointed out concerning some of the anti-doping campaigners' tactics, "the road to Hell is paved with good intentions" (2010: 12).

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**Table 1. Mentions in the reviewed academic literature to cases of sudden death among cyclists in Belgium and Holland**

Article / book chapter	Number of deaths mentioned	Origin of victims	Time span	Evidence of link with EPO	Source quoted	Counter-evidence of link with EPO
Savulescu and Foddy, 2010	“nearly twenty”	Europe	“In the four years after EPO became available in Europe”	Occurred after the release of EPO in Europe	→Eichner, 2007	-
Catlin et al. 2008	“a number”	Europe	“in the late 1980s”	“the prevailing hypothesis is that they had overdosed on rHuEPO”	Catlin et al. 2006 (not accessed)	-
Gerrard, 2008	“a number”	Holland, Belgium	“during the 1980s and early 1990s”	during “the emergence of EPO as a performance-enhancing drug of choice”. These deaths were the result of “the recklessness of such acts of drug abuse”	→Gerrard, 2005; →Clisby, 2001	-
Stewart and Smith, 2008	18	Europe	“between 1988 and 1990”	deaths caused by “erythropoietin-induced heart failure”	→Parisotto, 2004	-
Cooke, 2007	“a rash”	-	“in the early 1990s”	Caused by EPO	→Leith, 1991 ( <i>The Independent</i> , 14-7-1991)	-
Eichner, 2007	“nearly 20”	European	“Within 4 years after EPO appeared in Europe”	“EPO was a key suspect”	→Scott and Phillips, 2005	-
Gerrard, 2005	“cyclists”	Holland, Belgium	-	“relationship between blood viscosity and catastrophic thrombo-occlusive events” due to EPO use	→Clisby, 2001	-
Scott and Phillips, 2005	18	-	“between 1997 and 2000”	“linked to rEPO by some sources”	None	“no concrete evidence has ever proved that rEPO abuse caused those deaths”
Tokish et al. 2004	5	Holland	1987	Occurred after the release of EPO in Europe in 1987	None	-
	18	-	“between 1997 and 2000” [sic]			
Savulescu et al, 2004	“several”	Holland	“in the early 1990s”	“attributed to high levels of EPO doping”	→Cazzola, 2000	-
Parisotto, 2004	18	-	“Between 1987 and 1990”	Caused by EPO. “EPO was at least in part responsible”; “EPO was everywhere”.	→Fisher, 1991 ( <i>The New York Times</i> , 19-5-1991)	-
Armstrong and Reilly, 2003 <sup>10</sup>	18	Europe	-	“linked (...) with rumours of EPO abuse”	Leith, 1992 ( <i>Athletics</i> magazine) (not accessed)	-
Cahsmore and Cashmore, 2003.	“deaths” (mentions Draaijer, Oosterbosch and Halupczok)	-	-	“rumored to have been related to dope”	None	-
Ekblom, 2002	“a number”	-	“during the past 10 years”	“In some cases, EPO use was confirmed”	→Adamson and Vapnek, 1991; “media reports”	media reports, but “not scientific reports”
Houlihan, 2002: 43	“a large number” “eighteen cyclists”	-	-	“associated with rEPO use”; “died as a result of rEPO”	→Armstrong and Reilly, 1996 (see Armstrong and Reilly, 2003)	-
	“twelve”	Holland	between 1987 and 1991			

<sup>10</sup> Third edition of same book as Armstrong and Reilly, 1996,

Jelkmann, 2002: 37	“about 20”	Holland, Belgium	-	“suspicion that rhEPO-induced erythrocytosis caused” these deaths	None	“it was never proven that any of these received rhEPO”
Jurith and Beddoes, 2002: 440, 442, 470	18	Holland and Belgium; “four Scandinavian [sic] countries (Belgium, Holland, Denmark and Sweden)”	“in the late 1980s and early 1990s”, beginning in 1987”	EPO “widely thought to have contributed”	→Wadler, 1994; →Fine et al, 1998 ( <i>The Globe and Mail</i> , 7-11-1998)	“there are no parallel data readily available indicating that there has been any excess morbidity, particularly coronary events or strokes, in elite Scandinavian orienteers or cyclists”
Kammerer, 2001: 16	“deaths”	-	“recent”	“presumably due to the abuse of erythropoietin”	→Leith, 1991 ( <i>The Independent</i> , 14-7-1991)	-
Clisby, 2001	“a number”	Europe	-	EPO “may have contributed”; EPO is “directly related”	None	-
Janssen, 2001	18	Holland, Belgium	-	-	None	“this scandalous media story is simply untrue. These tragic deaths all occurred during the late 1980s and early 1990s, a time when EPO was not used yet”
Waddington, 2000	“a sudden spate of deaths”, 14 + 4	Holland, Belgium	“Between 1987 and 1990”	Occurred after the release of EPO in Europe in 1987	→Leith, 1991 ( <i>The Independent</i> , 14-7-1991)	-
Cazzola, 2000	“professional cyclists”	Holland	“in the early 1990s”	“speculation” that EPO was “involved”	→Adamson and Vapnek, 1991	-
Sawka et al, 1996	18	Europe	“recently”	“the lay press has speculated” that EPO “may have contributed”	→Fisher, 1991 ( <i>The New York Times</i> , 19-5-1991); Woodland, 1991 ( <i>Bycycling magazine</i> ) (not accessed)	“there is no evidence that these cyclists were under the influence of EPO”
Gareau et al., 1996	18	-	-	“related to erythropoietin administration”	→Anonymous, 1991 ( <i>Der Spiegel</i> , 10-6-1991)	-
Wadler, 1994	“an alarming epidemic of deaths” 18	“fourth Scandinavian [sic] countries (Belgium, Holland, Denmark and Sweden)” Holland, Belgium	“Beginning in 1987”	“possibility that drug abuse may have been a significant contributing factor”	Escher and Maierhofer, 1992 (not consulted)	“assertions in the Netherlands that most of these deaths were <i>postviral</i> or from <i>coronary</i> causes”
Rogol, 1993	“a number”	-	-	“have occurred close in time to the availability of erythropoietin”	Cowart, 1989 (no reference to those deaths); Wadler and Hainline, 1989 (no reference to those deaths)	“there is no substantial credible evidence that they can be specifically attributed to abuse of biosynthetic erythropoietin”
Eichner, 1992	19	Holland, Belgium	“between 1987 and 1990”	Those deaths are “evidence of [EPO] abuse”. “It seems likely that at least some of these deaths were linked somehow to abuse of rEPO”	None	The evidence is “circumstantial”
Carrière, 1992	14	Holland,	“in de periode 1988-	“That makes thoughts swiftly to	None	“On the other hand, a number of

		Belgium	1990"	be turn to doping"*		deceased cyclists were known to have used no drugs".*
Spalding, 1991	18	Europe	"in the last four years"	"EPO may have contributed"; "the word is out that EPO can kill if you self-medicate. People are extremely frightened"	None	"Don Leggett, a compliance officer at the U.S. Food and Drug Administration (...) in fact, has run across so little EPO that he doesn't know what it's selling for on the black market"
Adamson and Vapnek, 1991	"cyclists"	Holland	-	EPO "may have been involved"	→Chen, 1990 ( <i>Los Angeles Times</i> )	"There is speculation, although no direct evidence"
Ramotar, 1990	4 13 other	Holland -	"in the past 2 years" "since the synthetic hormone was developed"	"widespread speculation" that they are related to EPO intake	Don Catlin, chief of the Division of Clinical Pharmacology and director of the Olympic Analytical Laboratory at the University of California Los Angeles; Juri Mainus, national team director of the US Cycling Federation (no empirical evidence)	"no absolute cause of death has been determined"
	-	-	-	-	Rinus Verboom, former coach of the Women's Cycling Team in the Netherlands (no empirical evidence)	"There was no EPO involvement to my knowledge"

\* Own translation from Dutch.

[→] means that the quoted source has been also reviewed

**Table 2. Mentions in the reviewed journalistic literature to cases of sudden death among cyclists in Belgium and Holland**

Article / book	Number of deaths mentioned	Origin of victims	Time span	Evidence of link with EPO	Source quoted	Counter-evidence of the link with EPO	Headline of article
Deblander, 1990a ( <i>Le Soir</i> , 26-3-1990)	7	Holland	"during the last two years"*	"might be linked to EPO intake".*	Ad Nuijten, "spokesperson of the Dutch federation"*	None	Lilholt, Maassen et Fignon révisent leurs copies
Specter, 1990 ( <i>The Washington Post</i> , 2-4-1990)	7	Holland	"over the past two years"	"EPO is among the chief suspects"	Royal Dutch Cycling Federation (not clear)	None	'Supermen' on new blood drug endanger sports and themselves
Chen, 1990 ( <i>Los Angeles Times</i> , 22-05-1990)	"a handful", "half dozen or so"	Europe, Holland	-	EPO "is widely feared to have (...) killed" these cyclists.	None	"sports officials and researchers concede they may never know for sure"	Undetectable dialysis drug is tied to athletes' deaths
Almond, 1990 ( <i>Los Angeles Times</i> , 2-6-1990)	15	Holland	"in three years"	"Some physicians are concerned that the drug (...) is somehow linked to the deaths"	"some physicians"	"but research does not indicate that"; Rob J. Pluijmers, a sportsmedicine doctor involved with Dutch cyclists for 15 years, (...) denied that any of the 15 deaths could be attributed to erythropoietin"; "EPO is not yet registered in the Netherlands"; "research has not shown any link to sudden cardiac arrest"; "In the Netherlands, Pluijmers said that autopsies did not offer any clues in the rash of deaths"; "Officials [in the Netherlands] finally called for an investigation after a hue and cry in the Dutch press. "The investigation is ruling out any kind of drug use""; "Cardiologists agree with Pluijmers that EPO may not be the culprit".	Dutch looking for link in deaths
Smith, 1990 ( <i>Sunday Mail</i> [Queensland], 5-08-1990)	15	Holland	-	EPO, "a killer super-drug", "has been linked" to these deaths. "Unless athletes know for certain that they will be caught if they use EPO, people will be dropping dead of it just like the Dutch cyclists," Black said"	"Former Queensland senator John Black, former chair of the Senate inquiry into drugs in sport"	None	Deadly superdrug gets the go-ahead
Deblander, 1990b ( <i>Le Soir</i> , 11-10-1990)	"around thirty", "thirty-four"* 23 + 10 + 1	Holland, Belgium, Spain	"in 20 years"*	EPO's "anarchical and totally uncontrolled administration would be the cause for all".*	"medical and pharmaceutical sources"*	"medical and pharmaceutical sources have little doubt that EPO is responsible for the death of several cyclists". "everybody is already limited to hypotheses. It is difficult, and will remain in	Les cyclistes meurent trop d'arrêts cardiaques. Le hasard a bon dos: haro sur

						the future, to prove that a man died because having taken too much EPO (...) Currently, generalisation should be avoided, as well as the traditional targeting on cycling".*	I'EPO
Dreyfuss, 1990 (Associated Press wire, 15-10-1990)	"Several"	Holland	-	"EPO is suspected in the deaths"	None	"autopsy results have been inconclusive"	Experts fear new wonder drug may kill athletes
Reuter, 1990 ( <i>The globe and mail</i> , 3-11-1990)	6	Holland	-	"linked to the drug"	None	None	Doctor issues drug warning
Powell, 1990 ( <i>The Times</i> , 14-11-1990)	"a spate of mysterious deaths", "perhaps 15"	Holland	"in the past three years"	"might be linked to EPO"; "I am not so much concerned with getting the athlete to the finish-line first, but with getting him there alive." (Eichner)	Randy E. Eichner	"It is extremely expensive about \$4,000 to \$6,000 in the US (for a year's treatment) and 50 per cent more in Europe."	Blood doping risks revealed
Noden, 1990 ( <i>Sports Illustrated</i> , 26-11-1990)	16	Holland	"in the past two years"	"Some sports-medicine experts blame the deaths on rEPO abuse"	ABC's "World News Tonight"; "Some sports-medicine experts"	"although doctors for the Dutch cyclists acknowledge that the cyclists died from "some kind of heart failure," they deny any connection with rEPO".	A bad boost
Starkman, 1991 ( <i>The Toronto Star</i> , 27-4-1991)	18	Europe	"over the past four years"	these deaths have "placed a focus on erythropoietin". "It seems to be related" with EPO. "There is a life risk in taking this"	"Dr. Arne Ljungqvist of Sweden, a member of the medical commissions of the IOC and track and field's governing body".	"I think there are those who are saying at the moment there's a plague upon us, but I'm not sure there's any evidence of this", according to "Dr. Andrew Pipe, chairman of the (...) Canadian Anti-Doping Organization".	New wonder drug may speed athletes to the killing fields
Fisher, 1991 ( <i>The New York Times</i> , 19-5-1991)	18	Europe	"in the last four years"	"the drug [EPO] may be implicated (...) "Physicians say they believe athletes began using the drug almost with the beginning of clinical trials in 1986. Then the deaths began"	Randy E. Eichner; "physicians"	"Only anecdotal evidence links EPO to these deaths (...) "There is no absolute proof (...) " said Dr. Randy Eichner"	Stamina-building drug linked to athletes' deaths
Anonymous, 1991 ( <i>Der Spiegel</i> , 10-6-1991)	2 + 3 5 7 18 4	Holland, Belgium Holland Holland, Belgium Holland, Belgium  Germany	1990 1989 1987-1988 "in the last four years"** 1990	[Draaijer's] widow: "he has taken erythropoietin"*. Randy E. Eichner suspects that the cause is erythropoietin.	Randy E. Eichner	"Some of the dead were necropsied at the request of relatives. None was found anything unusual, apart from their big hearts, which are a common feature in cyclists". "On the death certificates of 18 young athletes are still all sorts of cardiac diagnosis as the cause of their respective unexpected and early end".**	Schlamm in den adern (Mud in the veins)
Leith, 1991 ( <i>The Independent</i> , 14-7-1991)	5 1 + 2 5	Holland Belgium, Holland Holland	1987 1988 1989	"You could say it was as if all these cyclists had taken something which made their	None	Lisa Draaijeer says that her husband was against doping, that he was well known in the	Cyclists don't die like this

	2 + 3	Holland, Belgium	1990	blood thicker (...) making their arteries silt up when they were at rest". These deaths are "very suspicious".		peloton as a non-user. She thinks that cycling might simply have overstrained her husband's heart. "(...) it could have something to do with taxing your body so much. Before his death, nobody could believe it wasn't doping. But the investigations showed nothing". "there needs to be a lot of research before you can say concrete things," he [Frank Buddels, the president of the Royal Dutch Cycling Federation] says. "We commissioned some research on the matter. There were no concrete findings on any drug, certainly not from EPO. The tests were very intensive". "This is what muddies the waters of the EPO issue. A lot of cyclists die of heart attacks anyway, EPO or not". "Of course, there is no evidence to link any of the mysterious deaths to the drug".	
Jollimore, 1992 ( <i>The Globe and Mail</i> , 5-3-1992)	18	Holland, Belgium	Since 1987	"suspected to have used EPO"	None	"the deaths were attributed to cardiac problems"	New test detects banned drug
Woldendorp, 1992 ( <i>Trouw</i> , 3-4-1992)	14	Holland, Belgium	"between 1988 and 1990"****	None	None	"the cause of death should not necessarily be sought in the use of doping". "The fact is that stimulants but also an extreme effort are significant risk factors".***	Plotse dood niet per definitie door doping (Sudden death not necessarily due to doping)
Deblander, 1994 ( <i>Le Soir</i> , 8-2-1994)	"Around forty"*	-	"in less than 20 years"*	"Doping, and EPO in particular, become the object of much talking".*	None	Cyclists "indeed adopt, still nowadays, training methods which, from a medical point of view, are sometimes heretical (...) Cardiac arrhythmia is fearsome (...) Rather than talking about doping, let's simply refer to irrational training methods (...) Halpuczok and Nelissen are probably not new victims of EPO".*	En marge du décès de Halupczok et de a retraite de Nelissen, les coureurs ont aussi leurs problèmes de coeur
Belga, 1994 ( <i>Le Soir</i> , 11-10-1994)	12 + 6	Belgium, Holland	"in the last five years"*	might be related to "EPO abuse".*	None	might be related to "wrong training methods (...) Necropsies have shown that, in many cases, the cause was a congenital cardiac ailment called hypertrophic cardiomyopathy (...) it seems that, in some	Etude sur les problèmes cardiaques de nos coureurs. Douze Belges et six Néerlandais victimes en cinq

						athletes, the problem had arisen at the level of the aorta, as some people have got only two arterial valves instead of three".*	ans
Deblander, 1995 ( <i>Le Soir</i> , 7-2-1995)	"deaths"**	-	-	None	None	The cause might be "certain training mistakes, too early focused in stamina [résistance] compared with endurance".*	La vulnérabilité du cycliste n'est sûrement pas démontrée
Deblander, 1998 ( <i>Le Soir</i> , 24-07-1998)	17	-	-	EPO "would be the responsible" of these deaths.*	Chris Goossens, belgian sports physician	"this research was useless. It would have been necessary to necropsy those men in order to know for sure the cause for their deaths, and we did not have the legal means".*	Chris Goossens Sur la question du dopage dans le cyclisme, en particulier, et dans le sport en général
Longman, 1998 ( <i>The New York Times</i> , 26-7-1998)	"approximately two dozen"	-	"since the late 1980's"	"have been linked anecdotally to the drug"	None	None	Backtalk; Lifesaving Drug Can Be Deadly When Misused
Blair, 1998 ( <i>Time</i> , 27-7-1998)	5	Holland	"In the late '80s"	"believed to have died from side effects of the drug"	None	None	Just say go
	15	-	"since then"				
Fine et al, 1998 ( <i>The Globe and Mail</i> , 7-11-1998)	18	Holland, Belgium	"in the late eighties"	"Suspicion focused on a new drug, erythropoietin (EPO)"	None	"Today there is a complete divergence of opinion in Europe and North America over the deaths. "There weren't any EPO-related deaths here," Frans Stoele, a spokesman for the Netherlands Centre for Doping Affairs, said in an interview. "In most cases people died of heart failure. One was a suicide. No autopsies were done because there was no suspicion about the cause of death.""	Canadian cyclist watches dream die
Romo, 2002 ( <i>El Mundo</i> , 23-2-2002)	16	Holland	"between 1987 and 1990"****	"The occurrence of thrombotic episodes linked to EPO intake is not new"****	None	None	Primer caso español de trombosis por consumo de EPO (First Spanish case of thrombosis due to EPO intake)
Kimmage, 2007	"lives which were lost"	-	"in the early 1990s"	"Rumours began to circulate about a new wonder drug called Erythropoietin"	None	"There was no factual evidence linking its abuse to any of the deaths"	-
	"a spate of sudden and mysterious deaths"		in 1990				

\* Own translation from French.

\*\* Own translation from German.

\*\*\* Own translation from Dutch.

\*\*\* Own translation from Spanish.

**Table 3. Sudden death of competitive cyclists between 1970 and 2009 mentioned in the press and in cycling sites, fora and blogs**

Name	Nationality	Date of death	Age	Level of practice	Cause of death	Where
Pierre Bellemans	Belgian	12-09-1972	23	Professional	n.a.	n.a.
Lionel Vandamme	Belgian	15-8-1974	32	Retired	n.a.	n.a.
Vicente López Carril	Spaniard	29-3-1980	37	Retired	n.a.	During a football match
Marc De Meyer	Belgian	20-1-1982	31	Professional	“malaise cardiaque”	In bed
Sture Pettersson	Swedish	26-6-1983	40	Retired	n.a.	n.a.
Louis Verreydt	Belgian	14-12-1983	32	Retired	n.a.	n.a.
Ludo Vanderlinden	Belgian	1983	n.a.	Retired	n.a.	n.a.
Cees Evers	Dutch	1985?	n.a.	Amateur	n.a.	n.a.
Reinier Valkenburg	Dutch	4-12-1987	25	Amateur	n.a.	n.a.
Ruud Brouwers	Dutch	3-4-1988	n.a.	Amateur	n.a.	In bed
Connie Meijer	Dutch	17-8-1988	25	Amateur	inflammation of the heart muscle	During a race
Arjan de Ridder	Dutch	Summer 1989	n.a.	Amateur	“he had serious heart rhythm disorders”, “had a heart surgery behind him”	In the beach (?)
Bert Oosterbosch	Dutch	18-08-1989	32	Retired	“ataque al corazón”	In bed
Johannes Draaijer	Dutch	27-2-1990	26	Professional	“aorta outflow disturbance (...) had traces of an echo in his heart two weeks before his death”.	In bed
Jef Lahaye	Dutch	12-4-1990	57	Retired	n.a.	n.a.
Eric Chanton	French	14-5-1990	26	Amateur	“problème cardiaque” (cardiac ailment)	In competition
Leo Duynham	Dutch	26-7-1990	42	Retired	n.a.	n.a.
Dirk de Cauwer	Belgian	12-8-1990	23	Amateur	n.a.	n.a.
Patrice Bar	Belgian	14-9-1990	23	Professional	“bradycardy”	In bed
Geert Reynaert	Belgian	3-10-1990	21	Amateur	n.a.	n.a.
Adrian Hawkins	British	20-5-1991	22	Amateur	n.a.	After winning a major race
Jürgen de Cock	Belgian	29-10-1991	21	Amateur	n.a.	n.a.
Bart Zoet	Dutch	13-5-1992	49	Retired	n.a.	n.a.
Philippe Van Coningsloo	Belgian	14-6-1992	24	Amateur	“arrêt cardiaque” (heart failure)	In competition
Wim Lambrechts	Belgian	16-8-1992	25	Cyclo-crossman	“malaise cardiaque” (cardiac ailment)	In competition
Geert De Vlaeminck	Belgian	9-10-1993	26	Cyclo-crossman	n.a.	In competition
Carmino Baelen	Belgian	27-10-1993	22	Cyclo-crossman		
Joachim Halupczok	Polish	5-2-1994	25	Retired	In autumn 1990 was diagnosed with a cardiac arrhythmia and had to retire from professional cycling	During the dispute of a soccer match

Chris Cox	Belgian	September 1994	n.a.	n.a.	“malaise” (ailment)	During the dispute of a soccer match
Philippe Casado	French	21-1-1995	30	Professional	n.a.	During the dispute of a rugby match
Jo Leysen	Belgian	26-3-1995	22	Amateur	n.a.	In bed
Marc Van Meensel	Belgian	11-8-1996	26	Amateur	n.a.	n.a.
Paul Haghedooren	Belgian	9-11-1997	38	Retired	Heart attack	n.a.
Bjørn Stenersen	Norwegian	12-09-1998	28	Retired	n.a.	In competition
Glenn Fockaert	Belgian	10-03-2001	20	Amateur	n.a.	n.a.
Kim Van Bouwel	Belgian	21-03-2001	21	Cyclo-crossman	n.a.	After a night out
Johan Mannaert	Belgian	29-08-2002	19	Amateur	n.a.	In bed
Denis Zanette	Italian	10-1-2003	32	Professional	“arrêt cardiaque”	In the dentist
Kenny Vanstreels	Belgian	2-03-2003	19	Amateur	n.a.	n.a.
Marco Ceriani	Italian	5-05-2003	16	Amateur	heart attack	In competition
Fabrice Salanson	French	3-6-2003	23	Professional	cardiac arrest	In bed
Marco Rusconi	Italian	14-11-2003	24	Amateur	heart attack	After birthday party
Michel Zanoli	Dutch	3-1-2004	35	Retired	cardiac arrest / suicide?	n.a.
Johan Sermon	Belgian	14-2-2004	21	Amateur	heart attack	In bed
Alessio Galetti	Italian	15-6-2004	37	Professional	“arrêt cardiaque” (cardiac arrest)	In competition
Stive Vermaut	Belgian	28-6-2004	28	Retired	“arrêt cardiaque”, “had ended his professional career in 2002 on the advice of his doctors, suffered a congenital heart disease”	On a training ride
Tim Pauwels	Belgian	26-9-2004	22	Cyclo-crossman	“arrêt cardiaque à la suite d’une chute”	In competition
Bert Heremans	Belgian	15-10-2004	25	Amateur	“arrêt cardiaque”	
Ubaldo Mesa	Colombian	9-10-2004	32 (31?)	Professional	“heart failure”	In competition
Arno Wallard	Dutch	28-2-2006	26	Professional	n.a.	While driving his car
Daniel Bennett	Australian	5-1-2007	23	Amateur	n.a.	While training
Gert Verheecke	Belgian	5-9-2007	36	Amateur	“malaise cardiaque”	In competition
Peter Bissell	British	29-12-2007	21	Amateur	“arrêt cardiaque”	After a night out
Fredriek Nolf	Belgian	5-2-2009	21	Professional	n.a.	In bed

Source: own work based on an exploitation of the Lexis Nexis database, other on-line newspaper archives and cycling sites, fora and blogs. Every reference has been checked with at least two independent sources.

**Table 4. Alleged adverse effects of EPO mentioned in the reviewed academic literature**

Adverse effects mentioned	Text	Source quoted	Empirical/scientific evidence mentioned
“early uncontrolled and poorly monitored use of EPO resulted in excessive red cell production and an increased viscosity or stickiness of blood. Unavoidable blood clot formation in the arterial circulation occurred with acute, fatal consequences”	Gerrard, 2008	→Clisby, 2001	None
“The Rise in hematocrit can cause a significant worsening of blood viscosity, which can entail an increase in blood pressure and in the risk of thrombosis”	Shänzer and Thevis, 2007	None	None
“too much EPO can drive the haematocrit too high for safety by turning the blood to ‘mud’ that easily clots”	Eichner, 2007	None	None
“The misuse of rHuEPO causes increased viscosity of the blood which, in combination with the elevated hematocrit, leads to increased risk of thrombosis and embolisms”	Deligiannis et al, 2006	Vergouwen et al, 1999 (no mention to these side effects)	None
“increased afterload, elevated blood viscosity and the loss of hypoxia-induced vasodilation lead to arterial hypertension and perhaps to cardiac dysfunction”		→Wagner et al., 2001	None
“A fatal reduction of heart rate during the night has also been reported”		→Noakes, 2004	None
““Hypertension is the most common side effect of the drug when used in patients of hemodialysis, and can be found in one quarter to one third of patients receiving rEPO”	Scott and Phillips, 2005	→Denker, 2004	None
“The dangers of erythropoietin use include sudden death consequent to a fatal reduction in the heart rate, usually at night”	Noakes, 2004	None	None
“The most common side effect of erythropoietin treatment is the appearance or worsening of hypertension in about 25%-30% of treated patients. No single factor has consistently been linked to an enhanced pressor response”	Denker, 2004	→Besarab et al, 1998	None
“the more recent study of normal (42%) versus (30%) hematocrits in patients with cardiac disease did show a significantly increased incidence of vascular thrombosis in the normal hematocrit group”		→Besarab et al, 1998	None
“raising one’s hematocrit beyond physiologically normal levels leads to an increase in blood viscosity, thrombogenic potential, and myocardial infarction risk”	Tokish et al, 2004	→Ramotar, 1990	None
“The excess iron [necessarily associated with EPO use] is taken up in parenchymal cells and can cause an entire series of problems in various organs, not least in the liver”	Ekblom, 2002	None	None
“high EPO values can enhance thrombotic activity through endothelial and platelet activation”		None	None
“during submaximal exercise at 200 W (corresponding to an average of about 50% of VO2max in the fairly well trained subjects in these studies), there was a marked increase in arterial systolic BP from an average of 177 to 191 mm Hg” in experimental subjects having been treated with rhEPO”		Berglund and Ekblom, 1991 (original research); Bergström, 1993 (symposium results abstract); Ekblom, Wilson and Astrand, 1976 (original research); Huang, Davis and Johns, 1993 (original research); Maschio, 1995 (literature review)	None
“This concentration would cause significant increases in both systolic blood pressure and blood viscosity. In the short term there would be an increased risk of thrombosis and stroke. In the long term, cronicly elevated Htc and blood viscosity could lead to left ventricular hypertrophy and, ultimately, to left ventricular failure”	Armstrong and Reilly, 2003	Leith, 1992 ( <i>Athletics</i> magazine) (not consulted)	n.a.
“EPO-stimulated erythropoiesis vastly augments the demands of the sportsperson for ferrous iron from the synthesis of haemoglobin (...) leading to iron overload (...) These values are equivalent to those seen in congenital haemochromatosis. This condition is characterized by iron deposition in various tissues and organs leading to multiple organ failure, including cirrhosis. It also increases the risk of hepatic carcinoma”		None	None
“Erythrocytosis increases the risk to aquire myocardial infarction and stroke. Recently, we have studied effects of chronic erythrocytosis on cardiovascular functions in experimental mice transgenic for EPO. Compared to wild-type mice (hematocrit 0.47),	Jelkmann, 2002	→Wagner et al., 2001	None

the transgenic animals (hematocrit 0.80) developed left and right ventricular hypertrophy and cardiac oedema. Their life expectancy was greatly reduced as shown in the Kaplan-Meier-plot of survival"			
"The main risks of erythrocytosis with hematocrits (hct) > 0.55 include heart failure, myocardial infarction, seizures, peripheral thromboembolic events and pulmonary embolism. Endurance athletes are at increased risk during the competition, if their blood viscosity increases further due to the great loss of fluid associated with sweating"		None	None
"we cannot attribute the appearance of the thrombosis to a single agent, but to the effect of the doping drug cocktail": EPO, human growth hormone and Vitamins A and E supplements.	Martínez Lage, 2002	Description of a single case without any further empirical research	
"At hematocrits above 55%, the blood viscosity increases exponentially, thereby substantially increasing the risk of coronary or cerebral artery occlusions. Similarly, occlusions can occur in other blood vessels"	Jurith and Beddoes, 2002: 450	None	None
"A rise in hematocrit is associated with a greater blood viscosity that may cause hemodynamic and rheological problems.	Wagner et al., 2001	Editorial article, <i>The Lancet</i> 1989 2: 20-22 (not consulted)	n.a.
"It is believed that hematocrit values above 0.50 increase the risk to acquire hypertension, heart failure, myocardial infarction, seizures, and thromboses".		None	None
"The life span was markedly reduced in the tg mice investigated. Mean survival of tg [transgenic] mice was 7.4 months (...) In our study, cardiac preload was increased, echocardiography revealed a dilated left ventricle, and the histologic alterations of the heart indicate cardiac dysfunction. Cardiac reserve, typically found to be decreased in clinical cardiac insufficiency, was reduced, which became evident from the markedly lowered exercise endurance capacity (...) High hematocrit without accompanying hypoxemia appears sufficient to damage the heart, most likely because of the increased blood viscosity".		Own original research	
"the main side effect of EPO is hyperviscosity of the blood due to a raised hematocrit. There is a danger of clotting and cerebrovascular accident becomes even more problematic with dehydrating endurance exercise"	Clisby, 2001	→Cowart, 1989	None
"At that time, rHuEpo abuse was largely uncontrolled and Hct values in excess of 60% were presumably achieved. These polycythemic conditions compounded by dehydration during exercise readily predisposed athletes to thromboembolic complications"	Cazzola, 2000	None	None
"this type of iron overload [associated with EPO use] will eventually produce organ damage comparable to that occurring in genetic hemochromatosis, including the risk of developing hepatic carcinoma"		None	None
"EPO may well be the most dangerous, in health terms, of all the performance-enhancing drugs currently available"	Waddington, 2000	→Leith, 1991 ( <i>Independent on Sunday</i> , 14 July 1991)	None
"After 29 months, there were 183 deaths and 19 first nonfatal myocardial infarctions among the patients in the normal-hematocrit group and 150 deaths and 14 nonfatal myocardial infarctions among those in the low-hematocrit group (risk ratio for the normal-hematocrit group as compared with the low-hematocrit group, 1.3; 95 percent confidence interval, 0.9 to 1.9)."	Besarab et al, 1998	Own original research	
"Too low a packed-cell volume means a decrease of tissue oxygenation and too high carries the risk of thromboembolic events"	Marx and Senden, 1998	None	None
"Treatment of the anaemia of renal disease with recombinant human erythropoietin results in (...) an increased risk of thrombovascular accidents"	Kooistra et al, 1994	None	None
"rEPO (...) somehow accentuated the exercise-induced rise in systolic blood pressure (...) the combination of high hematocrit, elevated blood viscosity, and dehydration (...) would increase the risk of thrombosis (...) These same elements, plus an accentuated rise in exercising blood pressure, may also increase the risk of stroke or, possibly, heart attack".	Eichner, 1992	None	None
"as red-blood-cell counts rise, the blood thickens, raising the risk of blood clots that can cause heart attack or stroke (...) Levels above 55 percent impair blood flow, leading to clots (...) elevated levels may persist for months, increasing the risk of stroke or heart attack"	Spalding, 1991	None	None
"any form of induced erythrocythemia carries with it the potential medical complications which have been well described with polycythemia, including hypertension, congestive heart failure, and stroke"	Wadler and Hainline, 1989	None	None
"Excessive use of EPO raises the hematocrit to high levels, thereby making the blood viscous and leading to poor circulation, thrombosis, or even myocardial infarction"	Ramotar, 1990	Edward R. Eichner	None
"The effects of overadministration (...) may therefore not be apparent until it is too late and the athlete is dead."		Don Catlin	None
"if the hematocrit keeps going up, the blood will get thicker. At a certain point (...) an element of danger comes in. The thickened blood begins to move to vital organs more slowly. It also clots more quickly, thus increasing the risk of heart attack and stroke"	Cowart, 1989	None	None
"too much erythropoietin can in theory drive the hematocrit up to 80%"		Edward R. Eichner	None

““there seems to be no upper limit –the upper limit is toxicity””		Don Catlin	None
““We do’t know how much and how long erythropoietin will stimulate the system to produce RBCs (...) if it overshoots what is physiologically tolerable for the cardiovascular and pulmonary system, some athletes will develop heart failure and pulmonary edema. We may even see deaths”		Robert O. Voy	None

[→] means that the quoted source has been also reviewed

**Table 5. Alleged adverse effects of EPO mentioned in the reviewed journalistic literature**

Alleged adverse side effects of EPO mentioned	Article	Source quoted	Empirical/scientific evidence mentioned
"it could cause strokes or heart attacks"	Specter, 1990 ( <i>The Washington Post</i> , 2-3-1990)	"Bjorn Ekblom, professor of physiology at the Karolinska Institute".	None
"the danger is that an excessive number of red blood cells causes blood to thicken. This forces the heart to work harder and possibly leads to strokes and heart attacks, especially during vigorous exercise"	Chen, 1990 ( <i>Los Angeles Times</i> , 22-05-1990)	"Thomas Murray, a member of the U.S. Olympic Committee's panel on substance abuse research and education", and "Daniel Vapnek, Amgen's senior vice president for research".	None
"Physicians believe it can cause the blood to become so thick that the heart can no longer pump it. The result would be a heart attack, stroke or blood clot, doctors said"	Almond, 1990 ( <i>Los Angeles Times</i> , 2-6-1990)	"Physicians".	None
EPO "is powerful enough to kill athletes who misuse it (...) EPO-enhanced level may rise so high that the blood becomes too thick for the heart to pump properly (...) The experts say the blood can reach the viscosity of motor oil, increasing the possibility of clots forming in the bloodstream. And the body's loss of water through sweat during exercise makes the viscosity greater, they say. The result can be seizures, heart attack or stroke, the council's report said. "Unfortunately, the sky's the limit," said Randy Eichner (...) "You would have mud, not blood""	Dreyfuss, 1990 (Associated Press wire, 15-10-1990)	American Medical Association's Council on Scientific Affairs, "scientists", "experts", Randy E. Eichner.	None
EPO "can turn a person's blood to "mud and that will kill you""	Reuter, 1990 ( <i>The Globe and Mail</i> , 3-11-1990)	Randy E. Eichner.	None
"Beginning with hematocrits in the 50 to 55 per cent range, blood viscosity (thickness) rises sharply, increasing the chance of clotting, strokes and heart attacks.""	Powell, 1990 ( <i>The Times</i> , 14-11-1990)	"Dr John Adamson, president of the New York Blood Center".	None
"The higher the percentage of red blood cells, the thicker the blood. And the thicker the blood, the greater the chance of developing blood clots and therefore of suffering a stroke or a heart attack. When the red blood cell count gets too high (...) "the blood becomes mud"".	Noden, 1990 ( <i>Sports Illustrated</i> , 26-11-1990)	Randy E. Eichner.	None
"medical officials say that athletes who use EPO are exposing themselves to great risks (...) [They] are in danger of thickening their blood to a point where they could become susceptible to clotting and strokes. One American hematologist said an athlete's blood can literally turn to mud from misuse of the drug".	Starkman, 1991 ( <i>The Toronto Star</i> , 27-4-1991)	"medical officials", "one American hematologist" (Eichner?).	None
"Eichner in Orlando: "Statt Blut fließt dann Schlamm in den Adern"" ("Instead of blood, what runs in the veins is mud"). ""Das Blut verklumpt, verschliesst die Gefäße, und dann wird es ganz zu Stein"" ("The blood clots, closes the vessels, and then it gets entirely of stone").	Anonymous, 1991 ( <i>Der Spiegel</i> , 10-6-1991)	Randy E. Eichner.	None
"Having too many red cells at this point could be dangerous - the heart might not be strong enough to force thick blood through the arteries. You could say it was as if all these cyclists had taken something which made their blood thicker, something which altered the balance between the red and white cells, making their arteries silt up when they were at rest"	Leith, 1991 ( <i>The Independent</i> , 14-7-1991)	None	None
""The effect of EPO is very dramatic and very quick (...) It raises the red-cell count to the point where it turns the blood into sludge. (...) for healthy individuals, use of EPO could cause clots".		Ed Berg, a doctor of sports medicine at the Olympic Centre, Colorado Springs	None
"the use of EPO by athletes can result in "blood clots and obstructs the vessels, turning it into a mass as solid as stone. It is no longer	Jollymore, 1992 ( <i>The</i>	Randy E. Eichner	None

blood running in their veins, but mud.””	<i>Globe and Mail</i> , 5-3-1992)		
“L'érythropoïétine, substance naturelle qui augmente le nombre de globules rouges, permettant, du même coup, un meilleur transport de l'oxygène et, donc, la production d'un plus gros effort, présente, prise en trop grande quantité, la particularité d'épaissir le sang et de provoquer des embolies”	Deblander, 1994 ( <i>Le Soir</i> , 8-2-1994)	None	None
“The danger with EPO is that an excessive number of red cells thickens the blood, especially with the dehydration that results from strenuous exercise, making it more difficult for the heart to pump blood through the body. This leaves athletes at risk of clotting, strokes and heart attacks, Catlin said”.	Longman, 1998 ( <i>The New York Times</i> , 26-7-1998)	Don Catlin, head of the Olympic drug testing laboratory at the University of California at Los Angeles and member of IOC's medical commission.	None
“flushed features betraying the heart's struggle to pump blood thickened to gel by the endurance-boosting drug erythropoietin, or EPO”.		None	None
“The EPO-triggered overproduction of oxygen-bearing red cells leaves athletes' blood "so viscous and thick that they end up with all sorts of volume-overload and clotting problems" (...) The result can be heart attacks or strokes, most commonly during sleep”.	Blair, 1998 ( <i>Time</i> , 27-7-1998)	Bob Goldman, president of the National Academy of Sports Medicine in Chicago.	None
EPO “boosted the production of red blood cells, but it also thickened the blood, forcing the heart to work harder to pump it out (...) As an athlete dehydrates, EPO can cause blood to thicken or clot, possibly causing heart attacks, strokes or kidney damage”.	Fine et al, 1998 ( <i>The Globe and Mail</i> , 7-11-1998)	None	None

**Table 6. Counter-evidence of the casual link between EPO intake and sudden death in athletes mentioned in the reviewed academic literature**

Counter-evidence concerning the casual link between sudden death and EPO intake	Article	Quoted source / evidence
“Between 74 and 94% of non traumatic deaths which happened while doing a sporting activity have got cardiovascular causes”.*	Manonelles et al, 2007	Three original research articles
“there is a high amount of deceases (42 cases) among people 20 years old or less (25.45% of the 165 known cases)”.*		Own original research
“Cerebral-vascular accidents are well known but not frequent causes of sudden death in athletes, and are typically provoked by a ruptured cerebral aneurysm”.*		Fann, 2000 (original research)
“In the deceases of people 30 years old or less [45 from a total of 115 in which information about the necropsic study is available], the etiology of sudden death remarkably differs from the previous group, as it is well known. In particular in our series the most frequent causes have been arrhythmogenic cardiomyopathy, in 7 cases (13.72%); hypertrophic cardiomyopathy, in 6 cases (11.76%); ongenital coronary anomalies, in 5 cases (9.8%); left ventricular hypertrophy, in 4 cases (7.84%), and aortic valvular esthenosis, in 3 cases (5.88%). To cases have been described of acute myocarditis and cardiac fibrosis (3.92%) (...) It is noteworthy that in this age group a majority of the deaths had an undeterminate cause (14 cases, 27.45%) despite a complete necropsic procedure having been applied”.*		Own original research
“The fact that 14 deceases (27.45%), all of them in people 30 years old or less, did not show any objective cause of sudden death during the complete necropsy procedure deserves a special attention (...) In this group, corresponding to the so-called cardiac or arrhythmic sudden death with structurally normal heart, it has been demonstrated that in some cases the disturbance is to be found at a molecular level, in the electrolytic channels which determine the cardiac beat”.*		None
“in our subjects, mean arterial pressure was minimally affected by rHuEpo treatment, while cardiac output and systemic vascular conductance remained close to their pre-Epo respective values. This corroborates previous studies showing that in the range of haematocrits observed in this study (from 42 to 49%), a small increase of haematocrit has no major impact on resting mean arterial pressure and hence on systemic vascular conductance (Berglund & Ekblom, 1991). In agreement, this study shows that the flexibility of the red blood cells remained unchanged. Based on this, blood viscosity may be assumed to be relatively unchanged”.	Lundby et al, 2007	Own original research and Berglund & Ekblom, 1991 (original research)
“SCD [sudden cardiac death] occurred in 1101 (1966–2004) reported cases in athletes under 35 years (...) SCD was reported in almost all sports; most frequently involved were soccer (30%), basketball (25%) and running (15%) (...) Doping is often considered to be the main cause of sudden death by the media and lay people, which seems unlikely, as underlying cardiac diseases account for approximately 90% of exercise-related sudden deaths”	Bille et al, 2006	Article based in “a systematic review of the literature” on sudden cardiac death.
“there is no correlation between hypertension and the rHuEPO dose or achieved hemoglobin concentrations and hypertension is <b>not</b> seen in rHuEPO- treated patients without renal disease” (original stress)	Denker, 2004	Besarab et al, 1998 (original research); Abels, 1992 (original research)
“The direct effects of the drug are, for the most part, minor (reported in 3%-11% of patients) and include body aches, headache, nausea, fever, lethargy, and anxiety (...) Early studies revealed conflicting findings for increases in vascular access thrombosis”		→Evans et al, 1990 (no mention to these effects); →Besarab et al, 1990 (original research)
“Sudden unexpected death, nonfatal stroke, and acute myocardial infarction in trained athletes have been attributed to the abuse of cocaine, anabolic steroids, and dietary and nutritional supplements” (EPO not metioned)	Maron, 2003	Lange and Hillis, 2001 (literature review); Samenuk et al, 2002; Valli and Giardina, 2002 (original research)
Of 387 sudden deaths of young athletes recorded in the US (data from the register of the Minneapolis Heart Institute Foundation), only four (roughly 1%) were attributed to “drug abuse”.		Own original research
“On the other hand, endurance athletes generally have reduced thrombolytic risk as a consequence of the positive medical effects of endurance training”	Ekblom, 2002	None
“Studies have shown that compared with the pre-EPO-administration period, arterial systolic and diastolic blood pressure (BP) at rest remained unaltered after an rHuEPO administration period”		Berglund and Ekblom, 1991 (original research); Bergström, 1993 (symposium results abstract); Ekblom, Wilson and Astrand, 1976 (original research); Huang, Davis and Johns, 1993 (original research); Maschio, 1995 (literature review)
“the causes of death are generally congenital and almost never of ischemic origin in young athletes. In American series, the main	Boraita, 2002	Jensen-Urstad, 1995; Weslen,

causes of death in this age group are hypertrophic cardiomyopathy and congenital anomalies of the coronary arteries. Nevertheless, statistics for the European continent indicate appreciably different results, arrhythmogenic right ventricular cardiomyopathy and myocarditis being the most frequent pathologies. Other, much rarer, causes of death in young athletes are arrhythmogenic syndromes, cerebral vascular malformations, bronchial asthma, and <i>commotio cordis</i> syndrome"		1996 (original research).
"Most surprisingly, however, despite a hematocrit of 0.80, no increase in blood pressure and/or a reduction of cardiac output in the erythrocytotic mice was observed. In fact, both mean arterial pressure and cardiac output were normal and exactly within the range of the wt, normocythemc control littermates". "we did not observe an increase in mean arterial pressure, despite continuously elevated plasma EPO levels. Moreover, this observation strongly argues against a direct hypertensive effect of EPO".	Wagner et al., 2001	Own original research
"erythrocytosis with a hematocrit of 0.80 is detrimental during physical exercise"		Petit et al, 1995 (original research)
"In rats, when hematocrit was raised over a 3-week period to a value of 0.63 by the administration of EPO 500 units thrice weekly, mean arterial pressure, left ventricular end-diastolic pressure, right ventricular peak pressure, and cardiac output did not differ from the values in control animals (hematocrit 0.47)".		
"the left ventricular hypertrophy in cyclists, showing normal left ventricular functional and metabolic parameters, suggests physiological hypertrophy rather than a pathophysiological adaptation". Deaths "raised questions about the longterm health aspects of extreme physical training".	Pluim et al, 1996	Own original research
"It is concluded that epoetin does not increase vascular access clotting"	Besarab et al, 1990	Own original research

\* Own translation from Spanish.

[→] means that the quoted source has been also reviewed

**Table 7. Counter-evidence of the casual link between EPO intake and sudden death in athletes mentioned in the reviewed journalistic literature**

Counter-evidence concerning the casual link between these deaths and EPO intake	Article	Source quoted	Empirical/scientific evidence mentioned
"Pluijmers said they speculate that Dutch cyclists are starting strenuous exercise too young. He said the top pros and amateurs begin at age 8 to 10 and continue until 30. "We think there is a very big, thin heart, a very slow heart rate," he said. "I don't think that's so good. During sleep, (abnormal) beats can develop. If you have a very relaxed, thin heart, it can sort of flutter . . . causing a dysrhythmia.""	Almond, 1990 ( <i>Los Angeles Times</i> , 2-6-1990)	Rob J. Pluijmers, a sportsmedicine doctor involved with Dutch cyclists for 15 years	None
"I would suggest something is going on, but I wouldn't think EPO is necessarily the factor"		Dr. Tim Noakes of the University of Cape Town in South Africa	None
"Pluijmers said preliminary results show the causes of deaths as: --Four ischemic cases, which are usually traced to coronary artery disease. --Six post-viral cases in which a cold is suspected of causing cardiomyopathy. --One Wolff-Parkinson-White syndrome that was diagnosed earlier. --One aorta outflow disturbance. --One suicide. --Two unknown.		Rob J. Pluijmers, a sportsmedicine doctor involved with Dutch cyclists for 15 years	None
"there needs to be a lot of research before you can say concrete things," he says. "We commissioned some research on the matter. There were no concrete findings on any drug, certainly not from EPO. The tests were very intensive"	Leith, 1991 ( <i>The Independent</i> , 14-7-1991)	Frank Buddels, the president of the Royal Dutch Cycling Federation	Unidentified research
"the cause of death should not necessarily be sought in the use of doping".*	Woldendorp, 1992 ( <i>Trouw</i> , 3-4-1992)	Dr. Else Carriere, medical physiology and sports medicine department of the University of Utrecht in the report "Sudden death in elite sport"	Research report <i>Plotse dood bij topsport</i> (Carriere, 1992).
"Professor [Mosterd] does not deny that EPO (blood doping) was "popular" in the period 1988-90. "It may have played a role, but it is too easy to put everything under the doping label"*. *		Wim Mosterd, professor in sports medicine	
"To date, only amphetamines, wich are banned in cycling, can be blamed for cardiac failure, according to Luc Van Brussel".**	Deblander, 1995 ( <i>Le Soir</i> , 7-2-1995)	Luc Van Brussel, sports physician of the Belgian Cycling Federation.	None
"At the request of the UCI, I once tried to conduct a reserach on the perverse effects of EPO, which would be to blame for the decease of seventeen cyclits. But this research yielded nothing. It would have been necessary to necropsy those men in order to know for sure the cause of their decease, and we did not have the legal means".**	Deblander, 1998 ( <i>Le Soir</i> , 24-07-1998)	Chris Goossens, belgian sports physician.	Unidentified research

\* Own translation from Dutch

\*\* Own translation from French.