

# Rethinking AIDS

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## DARBY DEBUNKED

# Pro-HIV Hemophiliac Study Actually Points Towards Non-Contagious AIDS

by Paul Philpott

The September 7, 1995 issue of *Nature* presented an article that remains often-cited by those promoting AIDS as a contagious syndrome and caused by HIV. Oxford cancer epidemiologists and hemophiliac researchers headed by Sarah Darby authored the study. Darby's team attempted to test the noncontagious AIDS theory proposed by retrovirologist Peter Duesberg of the University of California at Berkeley. Darby, et al., collected mortality data among Britain's hemophiliac population from 1977 through 1992, and documented a startling climb in mortality beginning sometime between the years 1984 and 1986 that exclusively affected subjects testing HIV-positive (see accompanying charts). At first glance, Darby seems justified in concluding that her data does "demonstrate particularly clearly the enormity and specificity of the effect of HIV-1 infection on mortality in this population." But like all conclusions favoring the HIV explanation of AIDS, this one collapses when carefully examined.

### Duesberg's View

Duesberg proposes that the original outbreak of AIDS mortality (cases occurring before HIV testing became available in 1985 to identify patients as positive or negative for HIV antibodies) did not include hemophiliacs. Rather, he contends, the outbreak of AIDS that inspired some researchers to search for a "new" virus occurred within the injection- and gay-drug cultures that had only grown to appreciable numbers a few years earlier. Indeed, 96% to 100% of the original gay AIDS patients admitted to consuming speed, cocaine, the new "designer" drugs, or the equally new "popper" drugs [Marmor, *Lancet*, May 15, 1982; Jaffe, *An. Int. Med.*, August, 19983; Havarkos, *STD*, Oct/Dec, 1985: 97%].

Furthermore, when researchers introduced HIV testing in 1985, approximately 12% of these AIDS-diagnosed patients tested negative for HIV antibodies, and 66% tested negative for active HIV infections [Gallo, *Science*, May 4, 1984]. Meanwhile, positiveness for a variety of other microbes proved even more prevalent than HIV among these patients [Fauci, *JAMA* 257:19, May 15, 1987, p2617-2621].

When researchers looked outside the original narcotics-using patients, they examined such other generally-unhealthy populations as hemophiliacs, transfusion patients, and impoverished residents of developing nations. They found immune suppression and other AIDS conditions, and many patients who tested positive for HIV. But these patients also tended to test positive for the same non-HIV

microbes that the injection and gay drug users did, and many tested HIV-negative, just like the injection and gay drug users.

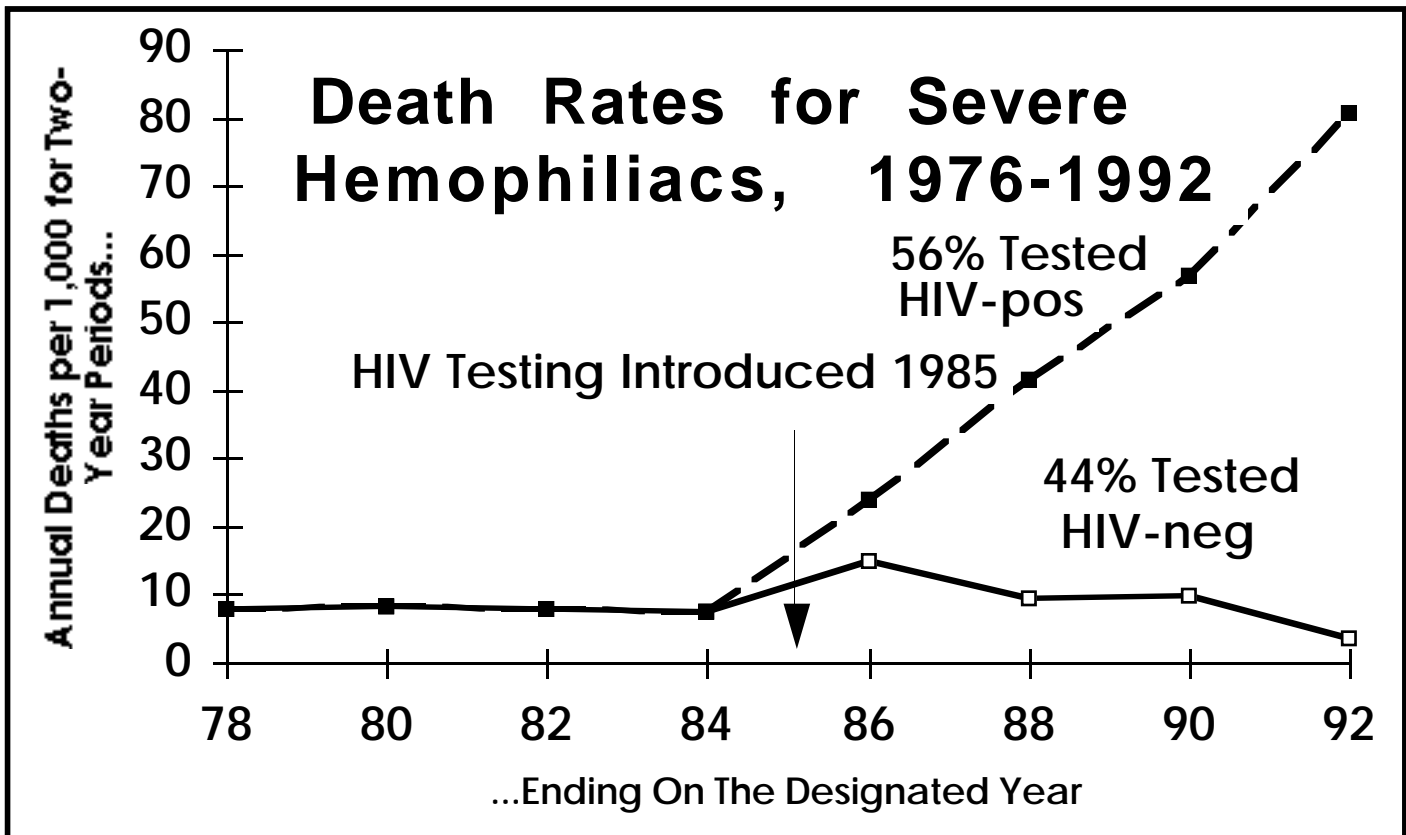
For these and other reasons, Duesberg concluded that the health factors — including the medical treatments — that define these groups offer the most likely explanation for their AIDS conditions [Duesberg, "Inventing the AIDS Virus", and "Infectious AIDS: Have We Been Misled?"]. But other scientists somehow interpreted these data as implicating HIV as the cause of AIDS. They predicted that hemophiliacs and blood recipients would — like the gay and injection drug users — experience increased mortality. Their medical response to this prediction, Duesberg believes, unintentionally and ironically made this prediction come true.

### Darby's Data

In order to test what they interpreted as Duesberg's non-infectious AIDS model, Darby and her team members obtained annual mortality rates for 4,043 British hemophiliacs who "received potentially [HIV] infected treatments" between the years 1977 and 1992, documented at two-year intervals. Their data (presented here in the accompanying charts) revealed that between the years 1977 and 1984, annual mortality was stable and low, at about 4 deaths per 1,000 for patients with mild-to-moderate hemophilia, and twice as high for patients with severe hemophilia, at about 8 deaths per 1,000.

HIV testing was introduced in 1985, and administered to most of the hemophiliacs composing Darby's data pool by the end of that year. The next year for which mortality data was collected, 1986, Darby's team members found it to have tripled to about 24 per 1,000 for severe hemophiliacs who had tested HIV-positive, and to have increased by about five times to 20 per 1,000 for mild-to-moderate hemophiliacs who had tested HIV-positive. According to Darby, the HIV-negative hemophiliacs did not experience any increased mortality.

The mortality among HIV-positive hemophiliacs increased each data year through the course of the study. By the last data year, 1992, both severe and mild-to-moderate hemophiliacs who tested HIV-positive showed nearly the same mortality: about 80 per 1,000, which represented roughly a ten-fold increase for severe hemophiliacs and roughly a 20-fold increase for mild-to-moderate hemophiliacs over their pre-1986 levels. Meanwhile annual mortality rates for both severe and mild-to-moderate hemophiliacs who tested HIV-negative remained at their low pre-1986 levels.



"During 1985-'92," Darby writes, "there were 403 deaths in HIV seropositive patients, whereas 60 would have been predicted from rates in seronegatives, suggesting that...[the 343 excess] deaths in seropositive patients were due to HIV infections." Darby could reach this conclusion only by ignoring one obvious feature of her data: there was no detectable mortality increase prior to the introduction of HIV testing in 1985. Both charts accompanying this article, constructed from data presented in Darby's paper, particularly her Table 2, show that the explosion in HIV-positive mortality occurred as if cued to do so by the massive HIV screening that immediately proceeded it.

### If Not HIV, What?

Duesberg's risk-AIDS theory regards HIV as too harmless to cause any of the AIDS conditions (low T4 counts, various opportunistic infections, three specific cancers, dementia, and wasting). Among hemophiliacs, Duesberg asserts that AIDS conditions, above their background incidences within the general population, result from two factors:

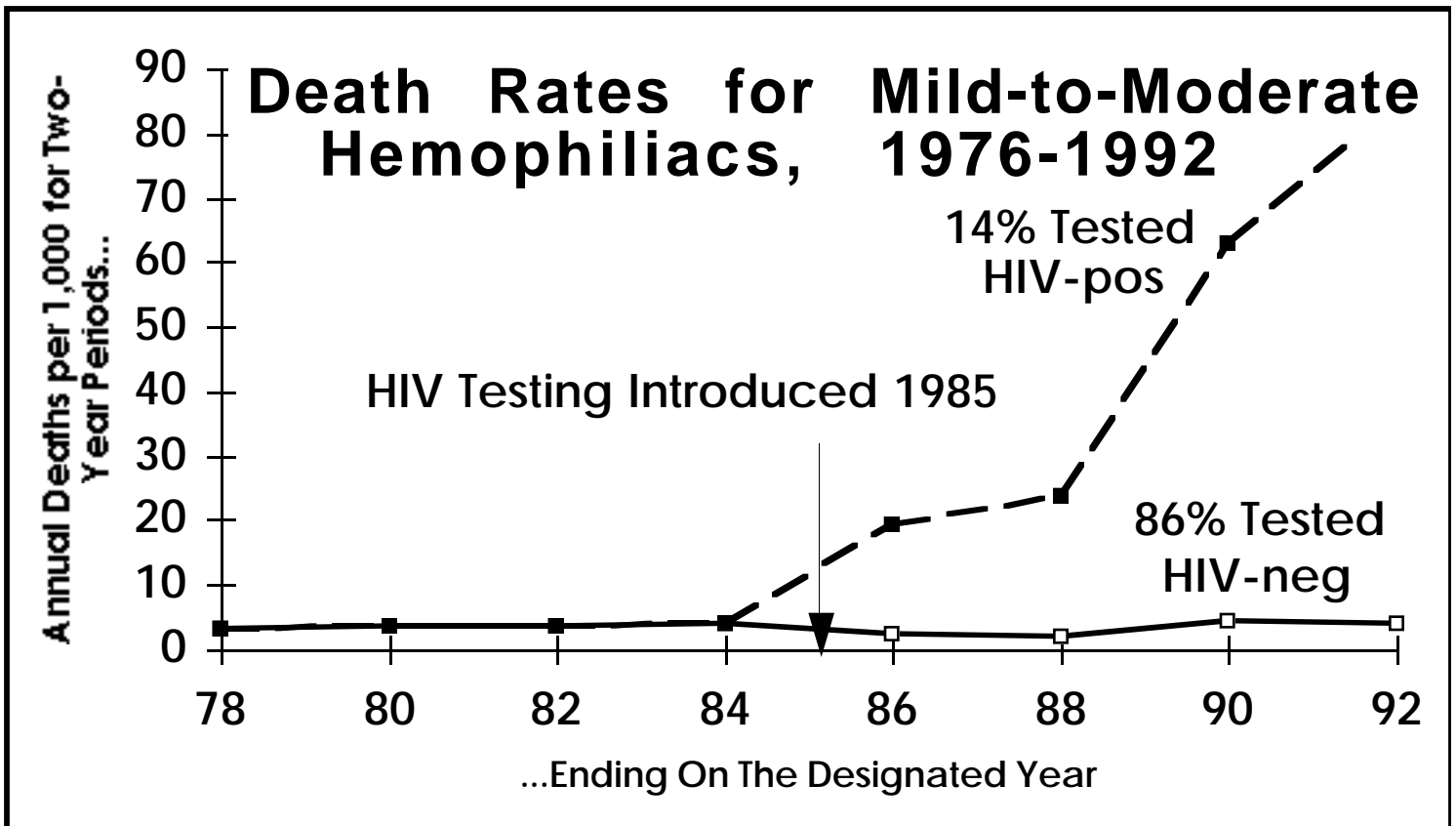
(1) Chronic immunological exposure to foreign blood proteins that contaminate un-purified Factor VIII injections. This can ultimately result in general immune suppression, including low T4 counts and opportunistic infections. A number of academic papers document those symptoms even in HIV-negative hemophiliacs, and also correlate immune dysfunction with doses of un-purified — but not contaminant-free — Factor VIII, even among HIV-positive subjects [Duesberg, *ibid.*].

(2) "HIV medicine," which is administered to even the healthiest

symptom-free people who test positive for HIV, under the presumption that positive HIV tests indicate AIDS-causing HIV infections.

HIV medicine possesses both a chemical and a psychological component. Chemically, HIV medicine consists of aggressive prophylactic, therapeutic, and often experimental treatments with powerful antibiotics, antifungals, and even cancer chemotherapies such as AZT and ddI. Nearly every one of the AIDS conditions reside among the multitudinous "side effects" of these many drugs. The most famous and often-prescribed of these drugs, AZT, debuted as a leukemia treatment because of its ability to kill all replicating human cells, particularly those composing the immune system. AZT also kills intestinal cells, damages brain, nerve, and muscle cells, and even stands as a logical theoretical candidate for causing lymphoma, one of the three official AIDS cancers. For this reason, the 1994 Physician' Desk Reference (p.742-746) entry for AZT states: "It is often difficult to distinguish adverse effects possibly associated with AZT administration from underlying signs of [so-called] HIV disease."

Perhaps one-third [Ascher, *Science*, Feb 24, 1995, p.1080] to one-half [British Medical Journal, July 15, 1995] of all AIDS patients develop their first symptoms only after beginning AZT treatment as a result of testing "HIV-positive." Although clinicians introduced AZT as an "anti-HIV" treatment in 1987, a year after British hemophiliac mortality began the steep climb observed by Darby, AZT is far from the only harsh drug administered even to symptom-free people who test HIV-positive. Anti-fungal drugs against PCP, one of the AIDS pneumonias, are even more likely to be administered prophylactically than AZT [BMJ, *ibid.*]. Symptom-free HIV-positives are the first healthy humans to ever consume such medications indefinitely, and in such combinations. That such



people would display ever-growing mortality should not surprise thoughtful observers.

While some symptom-free people labeled as “HIV-positive” avoid toxic prophylaxis, few escape the psychological aspect of HIV medicine, which consists of intense anxiety from receiving a fatal and profoundly stigmatized diagnosis. The terror can be intense indeed. The Australian mathematician Mark Craddock has publicized the following startling fact: of 1,300 deaths among HIV-positive Australians over a recent two-year period, 500 (more than one-third) were medically-assisted suicides [“Doctors Admit Helping in AIDS Deaths”, Sydney Morning Herald, November 17, 1995, p.3]. This does not necessarily mean that many or even some of the deaths among Darby’s HIV-positive subjects resulted from suicide. But it does offer direct support for the contention that HIV terror represents a biologically relevant factor.

### Rethinking Darby

In order to judge the infectious versus non-infectious models of AIDS, Darby’s study must address the following points:

(1) Hemophiliac mortality increased only after the introduction of HIV medicine in 1985. Since about half of Darby’s 2,037 severe hemophiliacs were already unwittingly HIV-positive by this time, surely HIV-caused mortality should have exerted a detectable influence prior to 1985 in this group. Of the two models, only Duesberg’s provides a possible explanation for why the explosion of hemophiliac mortality should occur exactly on the heels of HIV testing: the increased mortality resulted from the pharmaceuticals and terror that typically accompany a positive HIV test.

(2) Of the 403 HIV-positive deaths recorded by Darby during the course of her study, records attributed 235 to AIDS. How many of these cases developed only after the patients received their HIV-positive diagnoses? Darby does not provide the data needed to answer this question directly, but the data she does present points in Duesberg’s favor: no detectable mortality increase manifested until after most of the HIV-positive subjects received their designation. This corresponds with Duesberg’s prediction that AIDS conditions which develop in hemophiliacs mostly do so in those who first receive an HIV-positive label and consequently experience HIV terror and consume anti-AIDS prophylaxis. However, Duesberg also expects a few hemophiliacs will develop AIDS symptoms (in the form of immune suppression from Factor VIII contaminants) prior to HIV screening. Of these, Duesberg predicts, those who test negative should usually recover since they would not receive HIV medicine, whereas those who test positive should usually deteriorate to “full-blown AIDS” as a consequence of HIV medicine. Too bad Darby didn’t understand Duesberg’s theory well enough to collect the data that would have tested this very obvious aspect of it.

(3) Darby calculates that 343 more deaths occurred in her HIV-positive subjects than “would have been predicted from rates in seronegatives.” She concludes then that all of these excess deaths “were due to HIV infections.” Yet her Table 3, which itemizes the causes of deaths in this study, lists only 279 HIV-positive deaths as resulting from AIDS (235), and AIDS conditions (44 cases of dementia, non-hepatitis infections, pneumonia, and lymphoma) which, inexplicably, received no “AIDS” designation. But 279 still falls short 64 short of 343. The HIV explanation of AIDS can not explain 64 extra non-AIDS deaths among HIV-positive hemophiliacs. Duesberg’s theory can.

Since HIV constitutes a rare contaminant of Factor VIII, it takes many injections to finally become positive for it. Thus hemophiliacs who have become HIV-positive tend to be those who have acquired the most Factor VIII (and Factor VIII contaminants), which is to say they tend to have the most severe hemophilia. That could explain why 56% of Darby's severe hemophiliacs were HIV-positive, as compared to only 14% (a fourth as much) of her mild-to-moderate hemophiliacs. Duesberg's theory correctly predicts both more AIDS conditions (from excess amounts of Factor VIII contaminants and from HIV medicine) and more hemophilia-related illnesses (due to the predominance of severe hemophilia) in groups of HIV-positive hemophiliacs. Indeed, the proportion of severe hemophiliacs in Darby's HIV-positive population (about 80%) was roughly twice as high as the proportion in her HIV-negative population (about 40%). Darby's Table 3 agrees with Duesberg's view (but not the infectious AIDS model) by listing nearly twice as many hemophilia-related deaths among the HIV-positive patients (93 as compared to 56).

(4) Then there is the fascinating question inspired by the news report of suicides committed by HIV-positive hemophiliacs in Australia: How many of the deaths among Darby's HIV-positive patients were actually suicides resulting from HIV anxiety? She records only five deaths due to

"injury, poisoning, [or] suicide," the same as recorded for HIV-negative subjects. But it is important here to realize that Darby obtained her cause of death information by examining death certificates. And according to the press report from Australia, the suicides there discovered in HIV-positive hemophiliacs were not listed as such in the death certificates. Rather, the attending physicians tended to record the deaths simply as "AIDS related." If Darby were a thorough researcher, she would know about this news report and would have considered its implications in any study of HIV-positive mortality.

## HIV Acquitted: Searching For the Real Killers

The medical literature offers over 100,000 papers devoted to HIV. The data appear to justify Duesberg's conclusion that HIV possesses no capacity to cause any of the AIDS conditions. Its replication does not kill the cells that it infects, and even if it did, HIV typically infects only a few cells (and sometimes none at all) and is usually present only at trace concentrations (and sometimes not at all) in patients diagnosed as having AIDS [Duesberg, *ibid.*]. Meanwhile essentially all AIDS-diagnosed patients experience health factors that, unlike HIV, are biologically significant. Such factors include recreational drugs, immunological exposure to foreign proteins (from un-purified Factor VIII injections and other sources), anti-AIDS pharmaceuticals, HIV terror, and impoverished living conditions in developing nations [Duesberg, *ibid.*].

AIDS in hemophiliacs is remarkably different than AIDS in some other risk groups. Whereas increased mortality from AIDS diseases among hemophiliacs occurred only after the introduction of HIV medicine in 1985, other groups demonstrated increased mortality from AIDS conditions even before HIV-positiveness could be assigned. For example, the alarming increase in AIDS conditions among young gay men led to the establishment of this syndrome, and a search for a viral cause. But gays who were developing deadly AIDS before 1985 were exclusively those who participated in the new recreational drug culture that began in the 1970s [Duesberg, *ibid.*]. HIV medicine represented the second new biologically significant phenomenon introduced into this group. But for hemophiliacs, HIV medicine was the first biologically significant factor recently introduced to them prior to the increased mortality observed by Darby.

Darby's study suggests that, at least for hemophiliacs, HIV medicine is the deadliest factor affecting those diagnosed as having AIDS.

## Rethinking AIDS

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