

Incongruous Age Distributions of HIV Infections and Deaths from HIV Disease: Where Is the Latent Period Between HIV Infection and AIDS?

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ABSTRACT

HIV/AIDS theory postulates a latent period of roughly 10 years between infection by human immunodeficiency virus (HIV) and the appearance of symptoms of acquired immune deficiency syndrome (AIDS). Progressively improved treatments have supposedly extended the latent period for asymptomatic HIV-positive persons as well as the lifespan of AIDS patients. However, neither the age distribution of first HIV-positive test nor that of death from AIDS changed between 1987 and 2004. Both distributions still peak between ages 35 and 44. These data are incompatible with the conventional view of HIV/AIDS.

Black and Hispanic Americans have higher rates of HIV positivity and death from AIDS, yet their age of death is appreciably older than that of whites, Amerindians, or Asian Americans with HIV/AIDS. It seems incongruous that a race-associated factor should both predispose to disease, and also permit longer survival with the disease.

Expected Course of HIV/AIDS, Before and After Antiretroviral Therapy

Infection by human immunodeficiency virus (HIV) is said to be followed by an asymptomatic latent period averaging 10 years. Once actual illness sets in, the victim becomes a patient with acquired immunodeficiency syndrome (AIDS)—though in recent years it is fashionable to speak of early and later stages of “HIV disease.”

In the early 1980s, a new diagnosis of AIDS was typically followed by death within a few months, perhaps a year or two.

The first anti-HIV drug was AZT (also known as azidothymidine, zidovudine [ZDV], and Retrovir), which the Food and Drug Administration (FDA) approved in 1987 for treatment of AIDS, and in 1990 as a prophylactic in HIV-positive people to delay the onset of AIDS. Early dosages were as much as 2,400 mg/day, despite common severe side effects such as anemia: “The unavailability at that time of alternatives to treat AIDS affected the risk/benefit ratio, with the certain toxicity of HIV infection outweighing the risk of drug toxicity.”¹ Increasingly since the mid-1990s, the favored treatment regimen has consisted of “cocktails” of several antiretroviral drugs (highly active antiretroviral treatment, HAART) that often include AZT at much lower doses, typically 300 mg twice a day.²

Up to 1987, HIV-positive people were expected to experience AIDS on average 10 years after infection, and to die within a year or two after that; so the average time between infection and death would

Table 1. Death Rates from HIV Disease. Deaths per 100,000 population, overall and by sex, selected years 1987-2004.

Ages (years)	1987	1990	1995	2000	2002	2003	2004
All persons							
All ages, age-adjusted	5.6	10.2	16.2	5.2	4.9	4.7	4.5
All ages, crude	5.6	10.1	16.2	5.1	4.9	4.7	4.4
≤ 1	2.3	2.7	1.5	*	*	*	*
1-4	0.7	0.8	1.3	*	*	*	*
5-14	0.1	0.2	0.5	0.1	0.1	0.1	0.1
15-24	1.3	1.5	1.7	0.5	0.4	0.4	0.5
25-34	11.7	19.7	28.3	6.1	4.6	4.0	3.7
35-44	14.0	27.4	44.2	13.1	12.7	12.0	10.9
45-54	8.0	15.2	26.0	11.0	11.2	10.9	10.6
55-64	3.5	6.2	10.9	5.1	5.1	5.4	5.4
65-74	1.3	2.0	3.6	2.2	2.2	2.4	2.4
75-84	0.8	0.7	0.7	0.7	0.8	0.7	0.8
Males							
All ages, age-adjusted	10.4	18.5	27.3	7.9	7.4	7.1	6.6
All ages, crude	10.2	18.5	27.6	7.9	7.4	7.1	6.6
≤ 1	2.2	2.4	1.7	*	*	*	*
1-4	0.7	0.8	1.2	*	*	*	*
5-14	0.2	0.3	0.5	0.1	*	*	*
15-24	2.2	2.2	2.0	0.5	0.4	0.4	0.5
25-34	20.7	34.5	45.5	8.0	5.9	5.1	4.5
35-44	26.3	50.2	75.5	19.8	18.8	17.5	15.7
45-54	15.5	29.1	46.2	17.8	17.7	17.2	16.3
55-64	6.8	12.0	19.7	8.7	8.5	9.1	9.0
65-74	2.4	3.7	6.4	3.8	3.9	4.0	4.0
75-84	1.2	1.1	1.3	1.3	1.4	1.5	1.4
Females							
All ages, age-adjusted	1.1	2.2	5.3	2.5	2.5	2.4	2.4
All ages, crude	1.1	2.2	5.3	2.5	2.5	2.4	2.4
≤ 1	2.5	3.0	1.2	*	*	*	*
1-4	0.7	0.8	1.5	*	*	*	*
5-14	*	0.2	0.5	0.1	*	*	*
15-24	0.3	0.7	1.4	0.4	0.4	0.4	0.4
25-34	2.8	4.9	10.9	4.2	3.3	2.8	2.8
35-44	2.1	5.2	13.3	6.5	6.7	6.5	6.2
45-54	0.8	1.9	6.6	4.4	4.8	4.8	5.2
55-64	0.5	1.1	2.8	1.8	1.9	2.1	2.0
65-74	0.5	0.8	1.4	0.8	0.8	1.0	1.0
75-84	0.5	0.4	0.3	0.3	0.3	0.3	0.5

Source: *Health, United States, 2007*,³ Table 42, p 236.

* Rates based on fewer than 20 deaths, considered unreliable

have been about 10–12 years. During the next few years, the expectation was no change in the latent period but a delay in death after the onset of AIDS, for survival times of perhaps as much as 3 or 4 years rather than 1 or 2, extending the average time between infection and death to perhaps 12–14 years. After 1990, AZT as prophylaxis was supposed to have delayed the onset of AIDS to make the latent period greater than 10 years, and also to have delayed death by at least an equal interval; thus the time between infection and death should have risen to more than 15 years. Following the introduction

Table 2. All-cause Mortality. Deaths per 100,000 population in selected years

Ages (years)	1980	1990	2000	2003	2004
All ages, age-adjusted	1,000	940	870	830	800.8
All ages, crude	880	860	850	840	816.5
≤ 1	1,290	970	740	700	685.2
1-4	64	47	32	32	29.9
5-14	31	24	18	17	16.8
15-24	120	99	80	82	80.1
25-34	140	140	100	100	102.1
35-44	230	220	200	200	193.5
45-54	580	470	430	430	427.0
55-64	1,400	1,200	990	940	910.3
65-74	3,000	2,650	2,400	2,300	2,164.6
75-84	6,700	6,010	5,700	5,500	5,275.1

Source: *Health, United States, 2007*,³ Table 35, p 197. Numbers are rounded to two significant figures except for 2004.

Table 3. HIV Deaths as a Percentage of All Deaths, by Age, 2004

	HIV deaths, 2004	All-cause deaths, 2004	RATIO (%)
All ages, age-adjusted	4.5	800.8	0.56
All ages, crude	4.4	816.5	0.53
≤ 1	*	685.2	---
1-4	*	29.9	---
5-14	0.1	16.8	0.6
15-24	0.5	80.1	0.6
25-34	3.7	102.1	3.6
35-44	10.9	193.5	5.6
45-54	10.6	427.0	2.5
55-64	5.4	910.3	0.6
65-74	2.4	2,164.6	0.1
75-84	0.8	5,275.1	≤ 0.1

of HAART in the mid-1990s, and its use even with asymptomatic HIV-positive people with low immune-system cell counts, the onset of AIDS should have been further delayed, to extend the average latent period well beyond 10 years. Thus, from about the mid-1990s on, the average time between HIV infection and death should have become 20 years or more. That expectation underlies what has become a fairly common assertion, that HIV/AIDS is a chronic but manageable disease.

Age Distribution of HIV/AIDS Diagnosis and Mortality

Actual data on HIV infections and deaths from HIV disease do not show the expected extension of latent period and lifespan. Rather, the age distribution of deaths from HIV disease has not changed appreciably since 1987; the highest risk of dying was initially, and remains now, among people aged around 40; it has not moved to older ages. Moreover, the age distribution of people infected with HIV is about the same: the highest rates of HIV infection are also among people aged around 40. Since the age distributions of deaths and of infections superimpose, there is no sign at all of a latent period.

These data are incompatible with the prevailing theory of HIV/AIDS. On the other hand, they are consistent with what many dissenters from HIV/AIDS theory have long held, that “HIV tests” are not specific for HIV, and that a multitude of conditions—tuberculosis, malaria, various vaccinations—can cause a positive test.

Several lines of evidence indicate that a positive HIV test is more likely under life-threatening conditions than under less serious ones. It would then be expected that some percentage of deaths from a variety of causes would be associated with positive HIV tests and would therefore be misreported as deaths from HIV disease.

Table 1 shows that from 1987 through 2004, the highest risk of death from HIV disease was among people aged 35–44; perhaps towards the lower end of this range among women and toward the upper end among men. In neither case is there a significant shift to later ages over the years, as would have been expected under HIV/AIDS theory, which predicts an increase in the latent period and the length of time during which AIDS patients could survive owing to antiretroviral therapy.

It is unusual for an infectious disease to preferentially kill people in the prime years of adulthood. Usually, children and seniors are considered to be at highest risk, and are urged to be vaccinated against influenza and pneumonia. All-cause death rates begin to rise gradually from the teens and markedly only after the mid-thirties—see Table 2.

Combining data from Tables 1 and 2 reveals that not only is the death rate from HIV disease highest at ages 35-44, but also the proportion of all deaths attributed to HIV disease is clearly at a maximum in that age range (Table 3). That underscores the curious phenomenon that people in the prime years of adulthood are the most likely to succumb to this infectious disease.

Racial Disparities

Another puzzle is the racial disparities found persistently whenever diverse groups undergo HIV tests. The rates of testing positive always increase in the same order: Asian < white < Amerindian < Hispanic < black. Moreover, the disparities are so regularly found that quantitative relations among them are frequently cited. Thus the black-to-white ratio is given in a recent review⁴ as 6.9 for males and 20.5 for females.

That same order and that same ratio apply to deaths from HIV disease (Table 4).

Racial disparities in death rates are the same as the disparities in testing HIV positive—in other words, in the infections that supposedly, according to the HIV/AIDS theory, bring on subsequent death. The respective age distributions, however, seem incongruous. On the one hand, black and Hispanic Americans are much more prone to become HIV positive than are members of other racial groups, and to the same extent more prone to die subsequently of HIV disease; yet appreciable numbers of these more-affected people survive the disease to greater ages than the supposedly less-affected others. Among HIV-positive individuals, some 11%–13% of black and Hispanic men survive beyond age 54, while a negligible number of men of other races do; and 30%–35% of black and Hispanic women survive beyond age 44 whereas a negligible number of women of other races do.

That disparity in survival rates is the opposite of what one would expect. Black and Hispanic Americans are acknowledged to be less well served—on average—by the medical system. Whether that be part of the reason or not, the overall life expectancy of black Americans is considerably shorter than that of white Americans: in 2004, life expectancy at birth was 6.2 years longer for white than for black American men, and 11.3 years longer for white than for black women.^{3, p175}

Furthermore: what could make some ethnic or racial groups both more prone to infection yet also better able to stave off the infection's fatal consequence?

Age of Infection and Age of Death

That the age distribution of deaths from HIV disease has remained the same over the years, and that the highest rates are still in the same age range, is incompatible with the notion of a latent asymptomatic period whose duration has increased over the years, or with a progressively longer extension of the subsequent life span of people actually ill with AIDS.

A direct estimate of the putative latent period could be made by comparing quantitatively the age distribution for positive HIV tests with the age distribution for deaths from HIV disease. For such a comparison, one needs HIV-test data for the population as a whole, since the death data in Table 1 are also for the population as a whole. The most appropriate and readily available data sets are those published for 1995–1998 by the Centers for Disease Control and Prevention (CDC) for all public testing sites (clinics for drug abuse, family planning, HIV, prenatal care, sexually transmitted diseases (STDs), tuberculosis, and more, as well as prisons, colleges, and some private medical practices). These comprise data from nearly 10 million tests (Table 5).⁸⁻¹⁰

The HIV-test data and the death data are, unfortunately, reported for 10-year ranges that are not superposable. In Table 5, the highest

Table 4. Number of Deaths and Relative Rates of Death from HIV Disease by Ethnicity, 2002-2004.

	White	Black	Hispanic	Asian	Amerindian
MALES					
5-9		15		1	
10-14		16			
15-19		24		1	
20-24		159	52		1
25-34	1,403	1,685	554	29	25
35-44	5,886	5,481	1,679	96	85
45-54	4,874	5,258	1,365	79	17
55-64		1,798	445		
TOTALS	12,163	14,436	4,095	206	128
Relative rates	1.0 (reference)	7.4	1.9	0.3	0.84
FEMALES					
5-9		6			
10-14		25	2		
15-19		36	13		
20-24		157	13	1	
25-34	407	1,330	163	11	3
35-44	1,312	2,968	503	9	27
45-54		2,143	347		
TOTALS	1,719	6,665	1,041	21	30
Relative Rates	1.0 (reference)	24	3.4	0.22	1.4

Source: National Vital Statistics Reports.⁵⁻⁷ Numbers reported for the 10 most common causes of death in each age group were pooled from three separate years to minimize stochastic fluctuations in the smaller numbers. Rates for each racial group were calculated from 2005 census data. U.S. population percentages were: white 80.2%, Hispanic 14.4%, black 12.8%, Asian 4.5%, Amerindian 1%. Relative rates were then calculated using the rate for white Americans as reference.

Table 5. Frequencies (Percentages) of Positive HIV Tests, 1995-1998

AGE	0-4	5-12	13-19	20-29	25-34	30-39	35-44	40-49	45-54	≥ 50
ALL TESTS	3.3	0.85	0.25	1.0		2.6	XX	2.7	X	2.0
ALL males	4.2	1.0	0.35	1.6		3.7	X	3.5	XX	2.4
ALL females	2.5	0.75	0.2	0.6		1.53	XXX	1.6		1.3
W males	3.5	0.4	0.15	1.1		2.5	XXX	1.8		1.2
W females	0.9	0.3	0.1	0.25		0.6	XXX	0.55		0.35
B males	4.3	1.3	0.5	2.0		4.7	XX	5.3	X	4.0
B females	3.6	1.05	0.4	1.15		2.6	XXX	3.2		2.8
H males	4.4	1.3	0.4	2.1		5.4	XXX	5.6		3.6
H females	2.8	1.1	0.25	0.7		1.9	XXX	2.3		2.0
ASN males	0.75	0.75	0.17	0.7		1.7	XXX	1.4		0.8
ASN females	0.6	---	0.05	0.15	XXX	0.3		0.47		0.7
NAT-AM males	---	---	0.25	1.4		2.8	XXX	1.7		1.3
NAT-AM females	---	---	0.1	0.4		0.8	XXX	1.04		0.3

Ages of highest frequencies of positive HIV tests (as percentage of tests done) are in bold. "X's" represent ages of highest rates of deaths from HIV disease from Tables 1 and 4 (see text). W = white, B = black, H = Hispanic, ASN = Asian, NAT-AM = Native American (Amerindian)

rates for positive HIV tests are highlighted for comparison with the ages of the highest rates of death from HIV disease; where the rates for neighboring age groups are closely similar, both are highlighted. (The fact that newborns and children in the first few years of life tend to test positive for HIV at higher rates than middle-aged adults supports the view that a positive HIV test is a nonspecific response to physiologic stress, such as the stress associated with birth, but deaths from HIV disease in those age groups occur at negligible rates.) Where the highest death rate falls clearly within an age range, it is denoted by "XXX." Where the death rates for neighboring age groups are closely similar, "X" and "XX" represent the slightly lower and slightly higher rates, respectively.

Despite the fact that the age ranges for reporting deaths and positive tests are not the same, it is clear enough that the highest rates of testing HIV positive (numbers in bold) and the highest death-rates (X, XX, and XXX) are indistinguishably the same within the unavoidable uncertainty of comparing data averaged over non-superposable 10-year ranges: the peak rates overlap or straddle one another in 12 of 13 instances, and the exception is for a group whose very small numbers make it least reliable. There is thus a quantitative correspondence between the ages of maximum probability of testing HIV positive and the ages of maximum rate of dying from HIV disease. There is no indication of the latent period of about a decade followed by some years of living with AIDS that is a staple of HIV/AIDS theory; indeed, these data appear to be a disproof of the hypothesis of a latent period.

Furthermore, there is no indication that the time between infection and death has increased over the years as treatments were introduced and improved, particularly since the mid-1990s, when "life-saving" HAART supposedly extended the life spans of HIV-positive people significantly. The peak ages for deaths from HIV disease in 2002–2004 (Tables 1 and 4) are the same as the ages at which infections were most common in 1995–1998; yet most of the people infected in 1995–1998 should have had the benefit of HAART and would have been expected to survive for some 20 years

or so, perhaps on average at least to 2015. This adds a quantitative element to the general argument made earlier, that the peak age range for deaths from HIV disease has remained the same from 1987 to 2004.

There seems to be no way to reconcile these data with the conventional view of HIV/AIDS.

Alternative Explanations of the Meaning of “HIV Positive” and Deaths from “HIV Disease”

HIV Tests Are Nonspecific

Many “HIV/AIDS dissidents” have argued that positive HIV tests do not identify the presence of particles of retrovirus and are therefore not reliably diagnostic of infection by HIV. Perhaps the most comprehensive discussion is in a monograph from the Perth Group.¹¹ Concise explanations for the general reader have been offered by Hodgkinson.^{12,13} A host of relevant quotations with source references has been assembled by David Crowe.¹⁴ The very pamphlets in HIV test kits state that the tests do not in themselves constitute an accurate diagnosis of HIV infection.¹⁵

The reason is that a successful isolation of whole virions of HIV directly from an AIDS patient or an HIV-positive person has never been accomplished or published.¹⁶⁻¹⁸ Both antibody and polymerase chain reaction (PCR) tests purporting to be specific for HIV are based on assumptions about the proteins and RNA genome thought to constitute HIV particles—assumptions that have not been verified by the only certain means, namely, examination of authentic virions.

An empirical basis for holding HIV tests to be nonspecific is the array of publications reporting false positives from a large variety of conditions and infections,¹⁹ even including vaccination against influenza.^{20,21}

Corollary evidence comes from comparing the frequencies of positive HIV tests in various social groups:^{16, p 83} there appears to be an inverse correlation between general fitness or overall health, and the rate of positive tests among people who are not in the groups judged to be at high risk of becoming HIV positive. This does not exclude the possibility that testing HIV positive actually represents an infection, but it suggests as more or at least equally likely that testing HIV positive is a nonspecific response to physiologic stress—oxidative stress, according to the Perth Group.¹¹

First-time blood donors typically test positive at a rate between 1 in 1,000 and 1 in 10,000. Among repeat blood donors, the rate of positive tests is less than 1 in 10,000. The latter group is more highly selected for fitness and the absence of any symptoms of illness, even allergies.

Applicants for military service test positive at rates up to 1 in 100. Active-duty military personnel have positive rates up to a few per 1,000; the less fit are screened out before entering service.

The population-based National Health and Nutrition Survey reports an overall average of a few positives per 1,000. Higher rates are found in prisons and STD clinics, among homeless youths, and in hospitals among non-HIV/AIDS patients, including psychiatric patients—all settings associated with high stress levels. A progression from lower to higher rates is found from family planning clinics to prenatal clinics to abortion clinics, consistent with the hypothesis that HIV positivity increases with physiologic stress per se.

Very high rates of testing HIV positive occur in three groups: homosexual men, tuberculosis patients, and drug abusers. The latter two groups are obviously experiencing considerable physiologic stress, if not outright ill health. The first group is probably self-selected for high suspicion of HIV infection because of a lifestyle that is generally stressful and unhealthy, and that places devotees at increased risk of many types of bacterial, viral, and fungal infections.

Data from autopsies and hospitals, especially emergency rooms, support a correlation between physiologic stress and HIV positivity. Positive rates as high as 1.9% and 3.7% are found in autopsied patients, with the rate directly correlated with the severity of trauma.²² One San Francisco study showed a rate as high as 18% in patients who had no evidence of AIDS.²³ Patients admitted to hospital for reasons unconnected to HIV/AIDS had positive HIV positive tests at between 0.1% and 7.6%,²⁴⁻²⁷ varying with age in the same manner as in Table 5.²⁸ Emergency room patients were positive at a rate of 5%–6%,^{29,30} an order of magnitude higher than in the population-based National Health and Nutrition Survey.

Deaths from HIV Disease

Anyone found HIV positive before death or at autopsy is likely to be reported as a death from HIV disease, because infection by HIV is regarded as a likely *underlying* cause of death; for example, some deaths from diabetes have “HIV disease” listed as underlying cause.³¹ The rationale for this is the belief that HIV destroys the immune system; therefore, HIV causes people to succumb to other diseases more readily than they otherwise would. One of the curiosities often cited by HIV/AIDS dissidents is that tuberculosis patients often test positive, and when they do are said to be suffering from HIV/AIDS rather than tuberculosis, and are treated with antiretroviral drugs as well as antibiotics.

Up to 1997, the CDC published details of the various diseases being reported as AIDS. For 1997, among the total of 60,000 cases diagnosed with AIDS, no fewer than 47,000 did not manifest the opportunistic infections that had been the AIDS-defining conditions in the early 1980s; in particular, more than 36,000 had been diagnosed solely on the basis of a positive HIV test and low counts of immune-system cells—and *in the absence of any symptoms of illness*.³²

Consequently, it is plausible that a large proportion of so-called deaths from HIV disease in the United States are actually deaths from a wide variety of illnesses in which there is a false-positive HIV diagnosis. Since a certain number of autopsied or seriously ill patients test HIV positive, it is to be expected that a comparable number of all-cause deaths would be falsely identified as HIV-caused. The data are quite consistent with this expectation: Table 3 shows that deaths from HIV disease represent no more than a few percent of all-cause deaths, and they are more than 1% of all-cause deaths only among adults in the prime years of life, the same years in which people are most likely to test HIV positive.

This explanation accounts also for the racial disparities: blacks and Hispanics are much more likely to be classed as having died of HIV disease simply because they are much more likely to test HIV positive. The data in the National Vital Statistics Reports are for the 10 most common causes of death in each age group. At ages above about 45 for women and 55 for men, testing HIV positive is so rare among whites, Asians, and Amerindians that HIV disease is no longer listed

among the 10 most common causes of death. Since Hispanics and blacks test HIV positive so much more often than those in other ethnic groups, HIV disease still appears among the top 10 causes of death at the higher ages.

Conclusions

The ages of maximum death rates attributed to HIV disease and the ages of maximum probability of testing HIV positive are the same. That vitiates the notion of a latent period between becoming HIV positive and becoming ill and dying. Moreover, the latent period and the period between becoming ill and dying should have increased over the years as treatments were introduced and improved, yet the age distribution of deaths from HIV disease shows no obvious change between 1987 and 2004. HIV/AIDS theory fails to explain these data; indeed, one might venture that these data constitute disproof of HIV/AIDS theory.

On the other hand, the data are fully consistent with the view that testing HIV positive is a nonspecific marker of a challenge to health that is displayed, for example, by patients who are seriously ill for a variety of reasons.

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Potential Conflict of Interest: I am the author of a cited book, *The Origins, Persistence and Failings of HIV/AIDS Theory*, which claims to show that HIV is not the cause of AIDS.

REFERENCES

- 1 Anon. Zidovudine. <http://en.wikipedia.org/wiki/Zidovudine>. Accessed Mar 20, 2008.
- 2 DHHS Panel, a working group of the Office of AIDS Research Advisory Council. Guidelines for the Use of Antiretroviral Agents in HIV-1-Infected Adults and Adolescents; Dec 1, 2007.
- 3 National Center for Health Statistics. *Health, United States, 2007 with Chartbook on Trends in the Health of Americans*. Hyattsville, Md.; 2007.
- 4 Centers for Disease Control and Prevention. Racial/ethnic disparities in diagnoses of HIV/AIDS—33 States, 2001-2005. *MMWR* 2007;56:189-193.
- 5 Anderson RN, Smith BL. Deaths: leading causes for 2002. *Natl Vital Stat Rep* 2005;53(17).
- 6 Heron MP, Smith BL. Deaths: leading causes for 2003. *Natl Vital Stat Rep* 2007;55(10).
- 7 Heron M. Deaths: leading causes for 2004. *Natl Vital Stat Rep* 2007;56(5).
- 8 Centers for Disease Control and Prevention. *HIV Counseling and Testing in Publicly Funded Sites: 1995 Summary Report*. Atlanta, Ga. Centers for Disease Control and Prevention, U.S. Dept of Health and Human Services; September 1997.
- 9 Centers for Disease Control and Prevention. *HIV Counseling and Testing in Publicly Funded Sites: 1996 Annual Report*. Atlanta, Ga.: Centers for Disease Control and Prevention, U.S. Dept of Health and Human Services; May 1998.
- 10 Centers for Disease Control and Prevention. *HIV Counseling and Testing in Publicly Funded Sites Annual Report 1997 and 1998*. Atlanta, Ga.: Centers for Disease Control and Prevention, U.S. Dept of Health and Human Services; 2001.
- 11 Papadopoulos-Eleopoulos E, Turner VF, Papadimitriou JM, et al. *Mother to Child Transmission of HIV and its Prevention with AZT and Nevirapine*. Perth, Australia: The Perth Group; 2001.
- 12 Hodgkinson N. Why an HIV test may not provide proof positive at all. *The Business*, May 9/10, 2004:1,6.
- 13 Hodgkinson N. HIV diagnosis: a ludicrous case of circular reasoning. *The Business*, May 16/17, 2004:1,4.
- 14 Alberta Reappraising AIDS Society. Referenced quotations about HIV/AIDS tests and measurements. <http://aras.ab.ca/test.html>. Accessed Mar 23, 2008.
- 15 Alberta Reappraising AIDS Society. List of manufacturer test labels. aras.ab.ca/HIVTestInformation.zip. Accessed Mar 22, 2008.
- 16 Bauer HH. *The Origin, Persistence and Failings of HIV/AIDS Theory*. Jefferson, N.C.: McFarland; 2007.
- 17 De Harven E. Problems with isolating HIV. Address to European Parliament, Dec 8, 2003. English translation by de Harven. <http://hivskeptic.wordpress.com/2008/01/15/hiv-has-never-been-isolated-from-aids-patients/>. Accessed Mar 23, 2008.
- 18 Pease EP. *AIDS, Cancer and Arthritis: A New Perspective*; 2005: pp 124-131. ISBN 0-9550567-0-5. Available at: www.phyllis-evelyn-pease.com. Accessed Mar 23, 2008.
- 19 Johnson, C. Whose antibodies are they anyway? Factors known to cause false positive HIV antibody test results. *Continuum* 4(3), Sept/Oct,1996. www.virusmyth.net/aids/data/cjtestfp.htm. Accessed Jul 5, 2006.
- 20 Simonsen L, Buffington J, Shapiro CN, et al. Multiple false reactions in viral antibody screening assays after influenza vaccination. *Am J Epidemiol* 1995;141:1089-1096.
- 21 Erickson CP, McNiff T, Klausner JD. Influenza vaccination and false positive HIV results. *N Engl J Med* 2006;354:1422-1423.
- 22 Resnick D, Hellman F, Mirchandani H, Goodman DBP. Human immunodeficiency virus infection in cases presenting to the Philadelphia Medical Examiner's Office. *Am J Forensic Med Pathol* 1991;12:200-203.
- 23 Coleman DL, Luce JM, Wilber JC, et al. Antibody to the retrovirus associated with the acquired immunodeficiency syndrome (AIDS): presence in presumably healthy San Franciscans who died unexpectedly. *Arch Intern Med* 1986;146:713-715.
- 24 Lombardo JM, Kloser PC, Pawel BR, et al. Anonymous human immunodeficiency virus surveillance and clinically directed testing in a Newark, NJ, hospital. *Arch Intern Med* 1991;151:965-968.
- 25 St. Louis ME, Rauch KJ, Petersen LR, et al.; and the Sentinel Hospital Surveillance Group. Seroprevalence rates of human immunodeficiency virus infection at sentinel hospitals in the United States. *N Engl J Med* 1990;323:213-218.
- 26 Dondero TJ, Gill ON. Large-scale HIV surveys: What has been learned? *AIDS* 1991;5(suppl. 2):S63-S69.
- 27 Lindegren ML, Hanson C, Miller K, Byers RH, Onorato I. Epidemiology of human immunodeficiency virus infection in adolescents, United States. *Pediatr Infect Dis J* 1994;13:525-535.
- 28 Lombardo JM, Kloser PC, Pawel BR, et al. Anonymous human immunodeficiency virus surveillance and clinically directed testing in a Newark, NJ, hospital. *Arch Intern Med* 1991;151:965-968.
- 29 Kelen GD, Fritz S, Qaqish B, et al. Unrecognized human immunodeficiency virus infection in emergency department patients. *N Engl J Med* 1988;318:1645-1650.
- 30 Kelen GD, DiGiovanna T, Bisson L, et al. Human immunodeficiency virus infection in emergency department patients: epidemiology, clinical presentations, and risk to health care workers: the Johns Hopkins experience. *JAMA* 1989;262:516-522.
- 31 Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health. *Massachusetts Deaths 2005*; March 2007.
- 32 Centers for Disease Control and Prevention. *HIV/AIDS Surveillance Report* 1997;9(2):1-43.