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REVIEW ARTICLE

Demographic Characteristics of HIV: II. What Determines the Frequency of Positive HIV Tests?

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Abstract—HIV tests are supposed to detect the human immunodeficiency virus, but the accumulated results of 2 decades of HIV tests in the United States are not consonant with that supposition.

Newborn babies test HIV-positive about 4 times more often than do children from about 1 year of age to the pre-teen years. Male children always test positive more frequently than do female children. The frequency of positive HIV-tests, F(HIV), varies in regular fashion with age among widely different sectors of the population: blood donors, military personnel, drug users, and others. F(HIV) increases from the teenage years into the middle adult years and then declines again toward old age. These regularities and trends mark HIV tests as indicators of a physiologic process and not indicators of a sexually transmitted infection.

F(HIV) also varies from group to group, in a manner that reflects the general state of health of that group: repeat blood donors test positive most rarely, first-time donors somewhat more frequently, military personnel even more frequently, members of the Job Corps considerably more frequently, and medical patients being treated for reasons unconnected to HIV or AIDS nevertheless test HIV-positive more often than do healthy people—even when the medical condition is psychiatric. These variations again mark a positive HIV-test as indicating, not anything specific to HIV but something non-specific about health in general, for example, the degree of physiologic or oxidative stress.

These and other aspects of the data confirm the conclusion reached in Part I of this series, that HIV tests do not track a sexually transmitted agent. The most significant corollary is that newborns who happen to test HIV-positive should no longer be treated with the highly toxic anti-retroviral drugs.

Keywords: HIV tests—physiological stress—oxidative stress—demographics of HIV—HIV in newborns—anti-retroviral treatment of newborns

Introduction

The results of positive HIV-tests are often described as ''the prevalence of HIV''. This presupposes that what is detected by ''HIV tests'' is the human immunodeficiency virus. But the tests are for ***antibodies***, whose presence has been presumed to indicate actual infection by HIV. The distinction becomes of direct importance when considering the data from tests on newborns, as discussed below. To avoid confusing the results of positive HIV-tests with the presence of HIV, I use the term F(HIV) to denote the frequency of positive HIV-tests.

Part I of this series [1] reviewed the chronology and geography of the distribution of F(HIV) in the United States, concluding that if it measures the prevalence of HIV, then HIV must be endemic, not epidemic. It is not a readily transmitted sexual infection (STI). Therefore it could not have caused the AIDS outbreaks of the early 1980s.

But if HIV tests do not track an infection, what then is the significance of a positive HIV-test?

F(HIV) changes in regular, characteristic fashion with two kinds of variable:

1. variables particular to individuals: age, sex, race;
2. variables particular to activities and institutional settings: gay or heterosexual; healthy or in hospital; urban or rural.

The observed correlations of F(HIV) with these variables are explicable if F(HIV) is understood to be a fairly non-specific response to certain health challenges, as long argued by the Perth Group[1]. Different activities and institutional settings entail characteristically different health risks; age, sex, and race modify individual responses to these challenges.

This article collates data from HIV tests on a wide variety of social groups, between which F(HIV) varies enormously—by nearly 3 orders of magnitude. Repeat blood donors test as low as 1 positive in 100,000. Gay men (MSM—***m***en who have ***s***ex with ***m***en) test highest[2], at 40% or even more. Injecting drug users (IDU) sometimes test as high as, or close to, the levels in MSM. Other groups fall between those extremes, in a way that marks the level of F(HIV) as a marker of the degree of challenged health or actual illness in the sampled population.

Individual Demographic Variables

In all studied groups, F(HIV) varies in remarkably uniform fashion with age, race, and sex, as already noted in Part I [1]. Since several factors independently influence the magnitude of F(HIV), the precise influence of any one of them can only be found through a multivariate analysis, or by comparing groups that differ only in the magnitude of a single one of the variables. Most of the available data do not satisfy these requirements. There are only a handful of studies in which multivariate analysis was attempted, and even in these it is not clear that all the relevant variables could have been known and taken properly into account. Therefore, one cannot expect precisely quantitative replication of any given

observation when different social groups are compared, or a particular social sector for different periods of time. Even where testing has been mandatory for all members of a group (as with military cohorts, Job Corps, and blood donors), those tested are still only samples of volunteers drawn from the general population. In samples of any group at any given period of time, there will be random fluctuations in the distributions by age, race, and sex, and correspondingly there will be random variations in F(HIV). For example:

Almost every study shows F(HIV) increasing with age from the teens up to middle age before decreasing again at greater ages, the changes being more pronounced with men than with women. Among younger teenagers, F(HIV) is apparently higher among females than among males, while below and above the teenage years and into middle age, F(HIV) is higher among men than among women. But in several reports on prison inmates, not specified to be teenagers, the overall F(HIV) was greater for females than for males [9, 10]. Without more information than provided in those reports, one cannot know whether this is a genuine contradiction of the widely observed uniformities or whether age differences are confounding the data. Another possible confound is that a higher proportion of female inmates than males tend to be confined because of drug-related actions; since F(HIV) is usually much higher among those who abuse drugs than among those who do not, this would show up as a higher level of F(HIV) in females than in males who are in prison—but the conclusion would not be valid, that females are ***inherently*** more prone than men to test positive. The overwhelming mass of accumulated data suggests that in samples matched for IDU behavior and age and race, males will always test HIV-positive more frequently than females, except for a short period during the early teens.

Again, ''One of the most striking observations from these surveys is the marked race/ethnicity differences in HIV prevalence. In nearly all of the populations, prevalence was substantially higher among blacks than among whites. Although data from Hispanics were less consistent, prevalence among Hispanics was lower than among blacks and slightly higher than among whites in most populations'' (p. 38 in [11]). Those generalizations are illustrated in many individual studies cited in Part I [1], which also show Asian subjects always lower than white and Native Americans closer to white Americans than to any other racial group. But exceptions are occasionally found among prisoners [10, 12] and among patients at clinics for sexually transmitted diseases (STD) [13], which, again, might well be owing to confounding influences: the racial sub-groups in these samples are unlikely to have been matched for age, sex, and use of drugs.

Thus some exceptions must be expected whenever F(HIV) is compared between groups and over time. It is therefore striking that very few exceptions are actually encountered in practice, particularly not in the generally healthy groups where F(HIV) is no more than a few percent: there are robust regularities by age, sex, and race, reported in scores of official reports and peer-reviewed publications. Unwilling to believe that an STI[3] could show such demographic uniformity and constancy, I consulted the Centers for Disease Control and Prevention. They

responded, ''Your data 'regularities' appear to be true, and we agree that they are not 'artifacts'''[4]. Discussion in Part I [1] also showed that the regularities in the data could not be explained in terms of artifacts or false positives.

The dependence of F(HIV) on race will be discussed in Part III. Here, the only individual variables considered in any detail will be age and sex.

Group Variations of F(HIV)

Copious data on HIV tests are available for several groups: military personnel and potential recruits; blood donors; members of the Job Corps; childbearing women; and those attending a variety of public testing sites.

As noted in Part I [1], there has been a general decline in F(HIV) since 1985 within all observed groups. Therefore, comparisons between groups should be made for similar years. The most ready data for this purpose come from several review articles (Table 1).

These data are all consistent with a large number of individual studies, cited in Table 2. (Studies obviously drawn on by the review articles in Table 1 were not included in Table 2).

The lowest numbers come from repeat blood donors, where fewer than 1 in 10,000 test HIV-positive. First-time blood donors typically test higher, at several parts per 10,000. Military cohorts test higher again, between a few parts per 10,000 and several parts per 1000. For Job Corps members the numbers are, on average, significantly higher than for military cohorts. Patients at STD clinics and in hospitals are higher by an order of magnitude, parts per hundred (several percent) instead of parts per 1000. IDU and MSM show rates up to several tens of percent.

Some of these numbers underscore directly the conclusion reached in Part I [1], that F(HIV) does not track some sort of STI:

- F(HIV) for TB patients is about the same as, perhaps even higher than, for those visiting STD clinics (Table 1). That makes no sense if HIV is contracted sexually.
- Those visiting STD clinics and not specifically HIV clinics presumably know that their behavior may have exposed them to syphilis or gonorrhea but also believe that they did not put themselves at risk for HIV; yet F(HIV) among clients at STD and at HIV clinics is quite similar (Table 1)[5] [1, 19, 54, 61].
- Patients who are in hospital because of illnesses unrelated to HIV have a level of F(HIV) that is often about ten times that of such generally healthy populations as military cohorts—and even a hundred times greater than the level among repeat blood donors (Tables 1 & 2).
- Prostitutes should be at high risk for STIs. Yet a European study found that prostitutes who did not use drugs showed an F(HIV) level of only 1.5%, while those who did use drugs had a level of 32% [62]. Which then is the risky behavior, sex or drugs?

TABLE 1
Comparison of F(HIV) (%) between Various Groups, from Several Review Articles

Type of site	I 1995–1998 from public testing sites, averaged [14–16]	II 1991 review [17]	III 1991 review of studies of adolescents [18]	IV 1994 review of studies of adolescents [19]	V 1986–1988 review; table 6.3 in [20]
Blood donors					0.01
Applicants for military service, recruits		0.12			0.14
Soldiers					0.13
Childbearing women		0.15			
Reproductive health clinics		0.2 (2.6–0)			
College students				0.2	
Family planning clinics	0.23			0.18 (0.24–0.11)	
Job Corps		0.36			
Adolescent clinic			0.37–0.56	0.61 (1.3–0.37)	
Pre-natal and obstetric clinics	0.67				0.84
Newborns					1.5, 0.20, 0.18
Non-HIV hospital patients		0.7 (7.8–0.1)		3.3, 2.7, 0.5	0.32
STD clinics	1.4		1.2, 2.2	1.5 (2.2–0.18)	
TB clinics	1.6	>5 (58–0)			
HIV counseling and testing sites	1.8				
Abortion clinics			2.5		
Prisons	2.5			3.8 (5.2–2.3)	
Drug-treatment clinics	2.5				
Prostitutes					5
Runaway homeless youths			7	4.7 (5.3–4.1)	
IDU		4 (48–0)			55, 25, 5
MSM		36 (65–15)		11–9.4	45, 25
Hemophiliacs					60

Note: F(HIV) = frequency of positive HIV-tests; STD = sexually transmitted disease; IDU = injecting drug users; MSM = men who have sex with men.

One might be inclined to suspect that these HIV tests are being confounded by antibodies produced against other infections than HIV, so that these numbers would be artifacts. But—as argued at length in Part I [1]—the data themselves exclude false positives or artifacts as explanations:

- The same uniform trends by age, sex, and race are seen in all groups and cohorts. Thus any false positives or other artifactual influences would have

TABLE 2
Comparison of F(HIV) (%) between Various Groups, Collected from Individual Articles

Group	Years covered	Prevalence	Incidence per 100 person-years	Sources
Repeat blood donors	1991–2002		0.003–0.0016	[21–25]
First-time blood donors	1985–2002	0.04–0.012		[21–25]
Marines	1986–1988		0.028	[26]
Sailors	1986–1988		0.068	[26]
Active-duty Army	1985–2004	0.26–0.022	0.28–0.010	[27–33]
Army Reserve and National Guard	1985–2004	0.16–0.017	0.12–0.009	[27, 28, 34]
Applicants for military service	1985–2004	0.15–0.028		[11, 19, 27, 28, 35–38]
Applicants for military service, teenaged	1985–1989	0.71–0.034		[18, 39, 40]
Job Corps	1987–1997	0.39–0.23		[11, 18, 37, 41–43]
Health and nutrition survey of households	1988–1991	0.39		[44]
Prisons	1985–1988	2.6–2.1		[45]
Psychiatric hospital patients	1988–1991	5.5, 5.3		[46, 47]
Non-HIV hospital patients	1988–1995	7.8–1.3		[48, 49]
STD clinics	1987–1996	5.8–1.1		[50–54]
IDU	1985–1997	37–1 or 65–5	1.9 [55]	[11, 37, 52, 55] or [56]
MSM	1985–2000	56–4.4	1.4 [55]	[11, 52, 53, 57–60]

Note: F(HIV) = frequency of positive HIV-tests; STD = sexually transmitted disease; IDU = injecting drug users; MSM = men who have sex with men.

the same regularities. That possibility is exceedingly farfetched; but even if it were true, it would not vitiate inferences from those regularities.

- All the data come from official reports and peer-reviewed publications. If these data are unreliable, then so are ***all*** official data about HIV and all the conclusions based on them.
- The Centers for Disease Control and Prevention assured me that the data are not artifacts and that the regularities are genuinely there[4].

General Health and Physiological Stress

The variations of F(HIV) by population group cannot be reasonably explained as different rates of infection by an STI. But they do indicate that F(HIV) is higher, the lower is the presumptive fitness of the members of the group: F(HIV) in critically ill emergency-room patients was found to be 4%, much higher than for the least ill patients [63]. Conversely, the fitter the group, the lower is F(HIV). Tables 1 and 2 illustrate this correlation. Repeat blood donors

have been screened most intensively not only against current illnesses but also against former ones as indicated by the presence of certain antibodies, and have the lowest F(HIV). The fitness of first-time blood donors will generally be only slightly less than that of repeat donors: most people who offer to give blood believe themselves to be in good health. Applicants for military service know that they will be tested for drugs, so this group represents something like the average healthy general population. Those applicants who are recruited to active duty have passed tests of good health and would be expected to be fitter, on average, than the pool of applicants; and indeed, for the last half-a-dozen years, F(HIV) for active-duty soldiers has averaged about half of that among potential recruits (0.15 and 0.3 per 1000 respectively) [27]. A traditional belief holds that among the Armed Services, the Marine Corps is outstanding in its fitness. Taking F(HIV) as a marker of general health would support that stereotype: the incidence of F(HIV) among Marines is less than half that among sailors [26] or soldiers [27–33].

But without venturing into such fine (not to say controversial) detail, it is clearly the case that F(HIV) is lowest among the groups that are undoubtedly the healthiest, namely, blood donors and military personnel. It is clearly higher among the Job Corps, whose members are disadvantaged, unemployed youth—typically high-school drop-outs [41], enrolled even if they have a history of drug use [19]. In one comparison for comparable years, F(HIV) in the Job Corps was found to be more than 8 times that among applicants for military service [43]. The Centers for Disease Control and Prevention noted that in prisons, ''Most routine screening programs have yielded seroprevalence rates higher than those estimated for the general population but much lower than those seen in groups composed of persons at increased risk'' [63]. Hospitals and outpatient clinics report significantly higher F(HIV) than any of those groups, and in- or out-patients are by definition in ill health to some degree. It should be emphasized that these are patients—for example, psychiatric patients—whose health concerns have nothing to do with HIV or AIDS or risk factors for either of those.

That F(HIV) is a marker of challenged health was claimed already long ago on the entirely different and independent grounds of physiology and molecular biology. According to Eleni Papadopulos-Eleopulos, Valendar Turner, and their colleagues of the Perth Group[1], F(HIV) reflects oxidative stress resulting from an impending or actual illness[6]: the tests that supposedly detect antibodies specific to HIV are actually detecting signs of non-specific physiological stress. That hypothesis seems capable of explaining all the differences between groups just mentioned, and all those in Tables 1 and 2:

- It seems reasonable to expect that women who are already carrying a child are experiencing, on average, somewhat more physiologic stress than those at family-planning or reproductive-health clinics (Table 1).
- Again, those who have chosen to have an abortion (Table 1) are likely to be more stressed than those who carry a child to birth, either because of the

medical condition that made abortion seem desirable or through the psychosomatic stress of an experience that no one would freely choose. Could it be coincidental that in Paris, too, F(HIV) was found to be 2 or 3 times higher among women having abortions than among childbearing women [17]?

- People with TB and those with an STD might well experience comparable levels of physiologic stress.
- People being treated for drug abuse, and those in prison, are plausibly under more stress than those who are not in prison and not using drugs.
- Runaway homeless youths (Table 1) are plausibly in a more precarious state of health even than prison inmates.

A number of studies have noted that F(HIV) is high among psychiatric patients [46, 64], for example, 5.5% [46, 47]. Being a psychiatric patient makes it more likely, by an odds-ratio of 3.1, that one will test HIV-positive, while the odds-ratio is only 2.2 for a history of STD [64]. This makes no sense if F(HIV) detects an STI, but is quite consonant with the view that F(HIV) measures physiologic stress, in this case not only psychosomatic but also side effects of the powerful medications used to treat mental illness.

All the data, then, are quite consonant with HIV as an indicator of stress, but puzzling if HIV were an STI. Such an indication of stress might be thought of as similar perhaps to an inflammation, or to a long-lasting fever[7], or to the release of histamine in an allergic reaction, or the release of adrenalin or testosterone. Those are all ***reversible***, making these analogies consonant with the data cited in Part I [1] which revealed that ''HIV-positive'' is not necessarily a permanent condition.

These considerations also offer a plausible explanation for what is otherwise quite perplexing: newborn babies show a higher level of F(HIV) than do older children.

The Influence of Age

From Birth into Childhood

That newborn babies show a higher level of F(HIV) than do children aged between about 1 and the teenage years has been reported not only in the United States (Tables 3 & 4) but also in Africa (Table 5).

Before inferences are drawn from these data, two points need to be considered:

1. These are not observations over time on a given cohort. Since different age groups refer to different individuals, the results might not reflect the course of F(HIV) over time for an individual.
2. Does F(HIV) in newborns signify active infection or merely ''passive'' antibodies transferred from the mother?

TABLE 3
Variation of F(HIV) (%) with Age, 1995–1998, from Public Testing Sites

Age	F(HIV)			Ratio of male to female
	Overall	Males	Females	
0–4	3.25	4.15	2.48	1.7
5–12	0.84	0.99	0.73	1.4
13–19	0.26	0.34	0.22	1.5
20–29	1.00	1.56	0.61	2.6
30–39	2.58	3.65	1.53	2.4
40–49	2.67	3.49	1.60	2.2
≥50	1.96	2.36	1.27	1.9

Note: F(HIV) = frequency of positive HIV-tests. Data were averaged over 1995–1998; results were very similar in each individual year [14–16].

On the first point, it should be noted that Table 3 represents results for 4 separate years, each of which shows the same trend with age, and Tables 4 and 5 add two entirely independent groups. Either the results reflect F(HIV) values truly characteristic for individuals of the given ages, or half-a-dozen independent samples somehow captured groups in which individuals of a given age just happened to have the same F(HIV) relative to other age groups. The latter is hardly credible; it requires, for example, that in each sample, the newborns just happened to be 4 times as infected as children from about 1 to teenage. The only reasonable conclusion is that the data reflect a genuine and characteristic change of F(HIV) with age.

As to the second point, active infection by HIV via the mother has been guessed to account for perhaps 30–50% of all HIV-positive newborns [66]. For the studies reported in Tables 4 and 5, that would reduce the percentages

TABLE 4
Variation of F(HIV) (%) with Age among 3500 Hospital Patients, Newark, NJ, 1988 [48]

Age (years)	F(HIV)
<1	10.0
1–4	2.7
5–14	2.4
15–24	3.3
25–44	14.2
45–64	6.0
≥65	0.7

Note: F(HIV) = frequency of positive HIV-tests. Owing to the small numbers of patients in each category, undue weight should not be given to some of these percentages; for example, the ≥65 group included just two HIV-positive individuals, one male and the other female. But the point at issue is supported robustly: the drop in F(HIV) from newborns (25 positive out of 249) to 1-to-4 year-olds (6 out of 222) and 5-to-14-year-olds (9 out of 381).

TABLE 5
Variation of F(HIV) (%) with Age among 6000 Healthy Subjects, Kinshasa (Zaire), 1984–1986 [65]

Age (years)	F(HIV)
0–0.9	8.2
1–1.9	1.9
2–14	1.6
15–19	9.8
20–29	7.6
30–39	6.6
40–49	5.6
>50	4.6

Note: F(HIV) = frequency of positive HIV-tests.

of actively infected newborns respectively to between 5 and 7 or to between 4.1 and 5.8, which remain 2 or 3 times greater than for children aged $\geq$1. If this really is a measure of infection, then this infection reverses itself spontaneously in the majority of infants in Zaire (Table 5), in New Jersey (Table 4), and indeed across the United States (Table 3). As noted in Part I [1], there is a variety of other evidence showing that such reversion does indeed occur.

As an alternative to the chain of inference just set out, one might suggest that a much higher proportion of F(HIV) newborns than earlier estimated are not actually infected but simply harbor antibodies transferred from the mother. All three cited studies would then suggest that $\geq$75% of F(HIV) in newborns represents such passive antibodies, and only $\leq$25% active infection. But this would also require that some of the passive antibodies persist for years, for the lowest level of F(HIV) is shown for ages 13–19, 5–14, and 2–14 respectively in the three studies; and that contradicts what has been found in actual studies of passively transferred antibodies, namely, that they disappear after about 9 months: ''any child who has a positive HIV antibody test beyond 9 months should remain positive for the remainder of his or her life'' under the official view that a positive HIV-test indicates permanent infection (p. 45 in [67]).

In several ways, then, these data confirm that F(HIV) measures not an active infection but something that is characteristic of a given age. And not only of a given age but also of sex: the ratio of F(HIV) among males and females (Table 3) shows such a smooth variation with age as to demand an explanation in terms of physiology rather than sexual behavior; in particular, the difference of F(HIV) between boys and girls below teenage can hardly reflect differences in sexual behavior. By contrast, these variations with age and sex can be explained quite reasonably if F(HIV) is an indication of physiological stress. For example, male babies are indeed at somewhat greater risk of illness than are female ones—''sudden infant death syndrome'' afflicts 50% more boys than girls[8]. Overall rates for all cancers are also higher for men than for women[9].

From the Teens to above Middle Age

For ages from the teens onwards, much more data are available. For both sexes, F(HIV) increases with age from the low teens up to what one might call middle age (somewhere between 30 and 50), in all groups: the military, the Job Corps, university students, patients at publicly funded testing sites, blood donors, applicants for marriage licenses [11, 14–16, 18, 19, 25, 29, 35, 36, 38, 40, 41, 43, 50, 52, 68–70].

Above the middle years, F(HIV) decreases again, though the age at which the decrease begins varies. In several military cohorts, prevalence increases with age only up to about 30, and then declines markedly at higher ages [26, 29, 30, 35]. Among non-military blood donors, prevalence increased into the 30s for first-time donors but only into the 20s for repeat donors, before declining again [25].

These changes of F(HIV) with age are far more pronounced among men than among women (Figure 1).

Among people who initially tested HIV-negative—for example, repeat blood donors [25], the incidence of new HIV-positive tests varies with age in a similar manner as does the average or total F(HIV) [29]. Again, this makes sense if F(HIV) measures a reversible physiologic response.

Where the data are reported in sufficient detail, the same variation with age is evident in all racial sub-groups [21, 26, 30, 36, 41] (Figures 2 & 3).

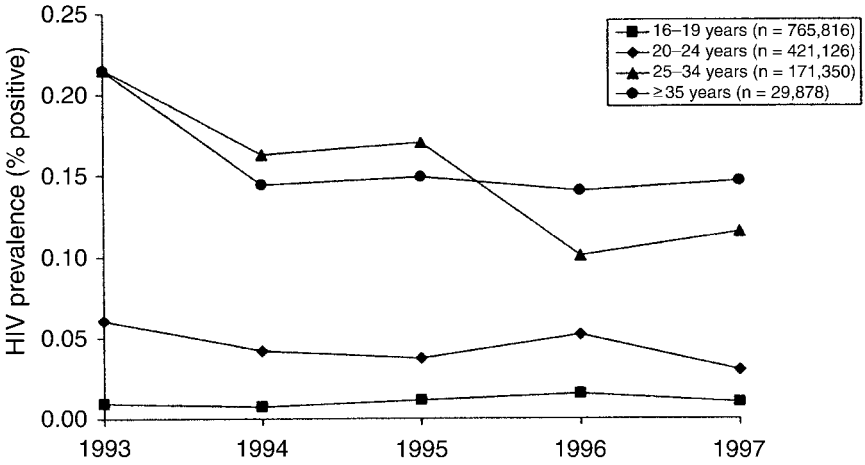
The same variation with age is seen at all overall levels of F(HIV), from the lowest to the highest: among blood donors [25], military personnel [18, 26, 30, 32, 35], applicants for marriage licenses [69], patients at clinics [18, 50, 60, 71, 72] and in hospitals [48, 49], prison inmates [9, 10, 12, 45, 73], at specifically HIV clinics [74], and in communities of MSM [13, 57, 75].

That age is so constant an independent variable speaks against any behavioral interpretation of F(HIV). Specifically, the fact that adolescents have a lower F(HIV) than do young and middle-aged adults is the opposite of the situation with sexually transmitted diseases: ''Adolescents and young adults have very high rates of STDs compared with older adults'' [60]; rates of chlamydia, gonorrhea, and syphilis infections among females aged 10–19 were higher than for older groups—''the highest rates occur among adolescents, despite the impression that STDs are a problem particularly endemic to the adult population'' [76].

The Younger Teens

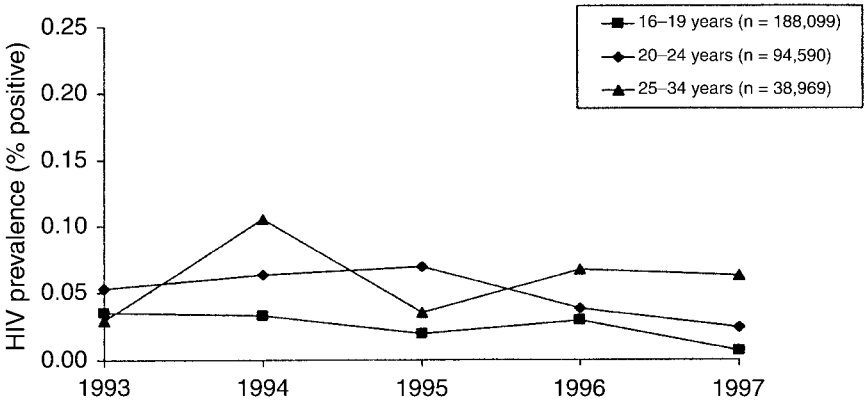
Two studies [39, 41] have reported that, in the lower teen years, F(HIV) is higher among females than among males; see, for example, Figure 4.

All these observed variations with age are compatible with the hypothesis that F(HIV) measures physiologic stress, in other words, a response to a health challenge. The capacity to generate such a biochemical response might well increase from the teenage years into the middle years only to decline again into old age: many biological capacities vary in that manner. In the lower teen years, higher levels of F(HIV) among females than among males might



Note. Standardized to 1993 population of military applicants by region, race/ethnicity, and metropolitan statistical area.

Source of data: U.S. Department of Defense.



Note. Standardized to 1993 population of military applicants by region, race/ethnicity, and metropolitan statistical area. Data for women ≥35 years of age excluded because of small numbers.

Source of data: U.S. Department of Defense.

Fig. 1. Variation of F(HIV) with age is much more pronounced among males (upper figure) than among females (lower); from [11].

reflect the stresses attendant to menarche, the onset of menstruation, by comparison to the milder physiologic changes experienced by boys during puberty. Newborns, of course, have just experienced the considerable physiologic stress of being born.

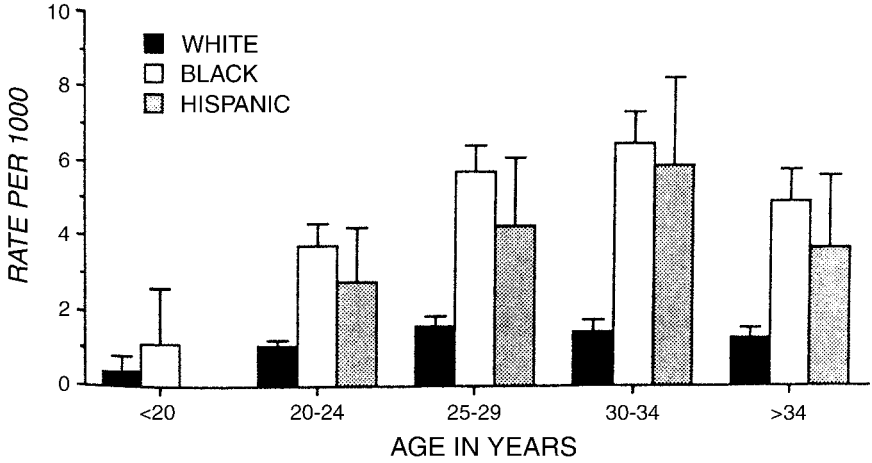


Fig. 2. The variation of F(HIV) with age is similar among white, black, and Hispanic Americans. Data for active-duty soldiers; upper 95% confidence bounds are indicated [30].

The Influence of Other "Risk Factors"

One of the first puzzles about "AIDS"—**A**cquired **I**mmune-**D**eficiency **S**yndrome—was that people were ill and dying from such a variety of different diseases. The hypothesis became that HIV was destroying the immune system, leaving the victim helpless against the many opportunistic infections that are

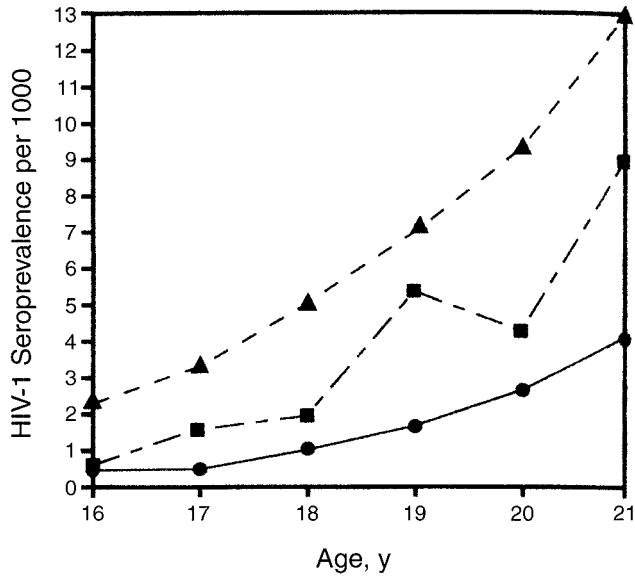


Fig. 3. The variation of F(HIV) with age is the same among white (♦), black (▲), and Hispanic Americans (■); data for teenagers and young adults in the Job Corps [41].

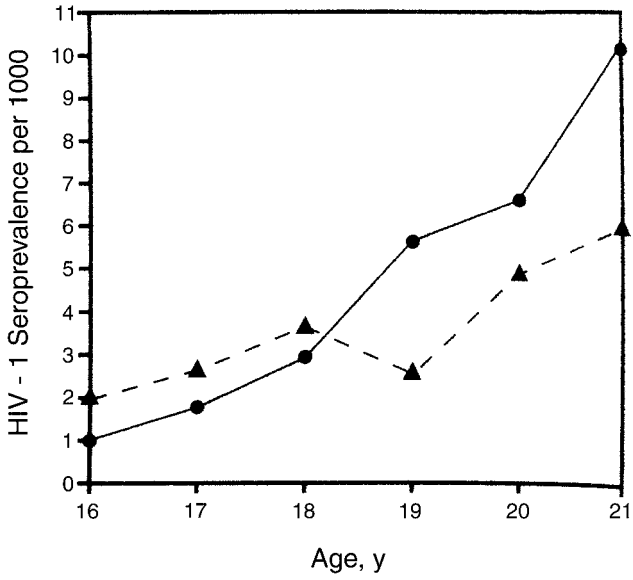


Fig. 4. F(HIV) is higher among females (▲) than males (●) at ages 16–18; Job Corps, 1987–1990 [41].

endemic but normally kept under control. An apparent association of these illnesses with positive HIV-tests was taken as proof that HIV was ***the cause*** of the immune-deficiency.

However, the correlation between AIDS and HIV was never tight:

- It has become increasingly clear over the years that only a very few HIV-positive people ever become ill [77]. Conversely, only ***some*** AIDS patients, by no means all, test HIV-positive (*passim* in [78]).

  To preserve the view that HIV causes AIDS, the term ''idiopathic CD4-T cell lymphopenia'' was coined for cases whose symptoms are identical to those of AIDS but where HIV is absent [79]. Prior to the announced discovery of HIV in 1984, cases of ''idiopathic CD4-T cell lymphopenia'' were simply AIDS cases. Since the Centers for Disease Control and Prevention introduced the presence of HIV as a pre-requisite for a diagnosis of AIDS, HIV and AIDS became associated ***by definition*** (pp. 59–62 in [78]; pp. 209 ff. in [80]; [81]).
- An early asserted geographic association between AIDS and HIV was shown in Part I (Appendix, [1]) to be invalid.
- Pre-AIDS symptoms were reported in 12.9% of HIV-positive MSM but also in 8.4% of HIV-negative MSM, and generalized lymphadenopathy in 48.8% of seropositive MSM but also in 11.4% of the seronegative MSM [82].

- The first publication from Robert Gallo's group in support of the theory that HIV causes AIDS reported finding HIV in 26 of 72 victims of AIDS, in 18 of 21 people with pre-AIDS symptoms, and in 3 of 4 ''clinically normal mothers of juveniles with AIDS''; but only in 1 of 22 normal male homosexuals and in none in 115 normal heterosexuals [83].

It is a separate question, which needs and deserves to be addressed elsewhere, why finding HIV in only 26 of 72 AIDS patients was ever taken as evidence that HIV caused AIDS. Here, the information is useful in confirming that a positive HIV-test indicates the presence of some sort of health challenge. Note that 18 of 21 ''pre-AIDS'' patients—86%—tested positive, but only 26 of 72 people—36%—who actually had full-blown AIDS. This makes no sense if HIV is the cause of AIDS. It does make sense if HIV appears as a response by the immune system to some sort of health challenge. In pre-AIDS, the immune system is still functioning sufficiently to generate this response; but when the immune system has been so damaged as it is with full-blown AIDS, then it is no longer capable of generating the HIV-response.

Another strike against HIV as the cause of AIDS is the matter of Kaposi's sarcoma (KS). This cancer, obvious as purple patches on the skin, was so characteristic of the AIDS outbreaks of the 1980s that the investigation by the Centers for Disease Control and Prevention to determine the cause of AIDS was named the Kaposi's Sarcoma and Opportunistic Infections Task Force. However, for more than a decade it has been recognized that HIV does not cause KS, in part because so many KS patients do not test HIV-positive, in other part because KS has become quite rare even among people with AIDS (pp. 382–84 and 463 in [80]; also [84, 85]).

Returning to the question of HIV itself, the suggestion that exposure to gonorrhea, syphilis [53, 54, 64, 86], or some other undoubted STD is a risk factor for contracting HIV has already been shown to be without merit[5]: since infection with HIV is supposed to be permanent, any ***history*** of ''risky behavior'' should correlate with being HIV-positive; yet the drug, sexual, and STD histories of HIV-positive and HIV-negative adolescents showed no significant differences [61]. On the other hand, many people ***currently*** infected with STDs—people attending STD clinics (Tables 1 & 2)—do have a higher F(HIV) than, say, blood donors or military personnel. Those two facts are compatible with the present hypothesis that HIV is a ***reversible*** indicator of physiologic stress: a sexual infection, like many other illnesses, is associated with a higher F(HIV). The reported positive associations between F(HIV) and hepatitis B [54], IDU, syphilis, hepatitis, and having had transfusions, a tattoo, paid sex, or sex with bisexual men [12] are at the same time associations with a less than fully healthy lifestyle. Many risk factors for sexual and other diseases were associated with high odds-ratios for being HIV-positive, in one of the early groups of MSM to be studied [82].

As to the fact that F(HIV) is typically high among IDU, it should be obvious enough that resort to ''recreational'' drugs, be they injected or taken orally, has biochemical consequences that the body seeks to resist, and to which the body generates a tangible response—part of that response being the generation of whatever it is that shows positive on an ''HIV test''. That IDU are at high risk for F(HIV) is simply a reflection of the fact that drugs are not good for health. The notion that F(HIV) among IDU results from the sharing of infected needles has not stood the test of actual observation: F(HIV) can be higher among those who do ***not*** share needles than among those who do [74, 87]. It is the content of the needles, not the sharing of dirty needles, that is so hazardous to health.

This is further confirmed by observations on rehabilitated IDU [55]. Among those who had completed treatment and remained drug-free, F(HIV) was less than half that among addicts who had just begun detoxification treatment. For those who had remained drug-free for more than a year, F(HIV) was a quarter of that among former IDU who had remained drug-free for less than a year. Those studies also provide yet further evidence that HIV-positive is a reversible condition, analogous perhaps to an inflammation.

That the content of the needles is the problem and not the unclean injecting is further illustrated by the fact that different drugs are associated with different levels of F(HIV). Relative risk-ratios were reported [55] of 0.9 for intravenous (IV) amphetamine, 1.3 for IV heroin, and 2.3 for IV cocaine; but for ***non-injected*** crack, it was highest of all, at 3.2. Furthermore, those who use different drugs also contract different diseases [88]. That these drugs are bad for health, and indeed cause symptoms just like those of AIDS, was noted by Gordon Stewart in the 1960s (p. 103 f. in [89]). It is also worth noting that the AIDS epidemic in the United States came at the same time as the epidemic of ''recreational'' drugs [88].

The great variety of illnesses taken as ''AIDS-indicator diseases'' (Table 6) has been further added to since the presence of HIV became a requirement for a diagnosis of AIDS. For example, in December 1992, a letter from the Centers for Disease Control and Prevention to State Health Officers added to the list, CD4 cell-count below a certain value; pulmonary TB; pneumonia recurring within 12 months; and invasive cervical cancer (p. 423 in [5]).

This list and these additions illustrate how the prior assumption that HIV causes AIDS led to portentous confusion. Ailments already known long before the advent of AIDS, and by no means always rare ones, have become classified as ''AIDS'' whenever HIV can be detected: pneumonia, TB, multiple or recurrent bacterial infections in children, and more, have come to be defined as AIDS whenever HIV is detected, while ***the very same illnesses, with the same symptoms***, are not defined as AIDS if HIV cannot be detected. This confusion was possible because HIV is a rather non-specific indicator of ill health or challenged health, so it can be found in some patients who are ill for a wide variety of reasons. That ***all*** the so-called ''AIDS-indicator'' diseases had in fact

TABLE 6
AIDS-Indicator Diseases as of January 1992, from Table 11 in [90]

Bacterial infections (multiple or recurrent, in children only)	Kaposi's sarcoma[a]
Candidiasis of bronchi, trachea, or lungs	Lymphoid interstitial pneumonia or pulmonary lymphoid hyperplasia
Candidiasis of esophagus	Lymphoma, Burkitt's (or equivalent term)
Coccidioidomycosis, disseminated or extrapulmonary	Lymphoma, immunoblastic (or equivalent term)
Cryptococcosis, extrapulmonary	Lymphoma, primary in brain
Cryptosporidiosis, chronic intestinal	*Mycobacterium avium* or *M. Kansasii*, disseminated or intrapulmonary
Cytomegalovirus disease other then retinitis	*M. tuberculosis*, disseminated or intraplumonary
Cytomegalovirus retinitis	Mycobacterial diseases, other, disseminated or intrapulmonary
HIV encephalopathy (dementia)	*Pneumocystis carinii* pneumonia
Herpes simplex, with esophagitis, pneumonitis, or chronic mucocutaneous ulcers	Progressive multifocal leukoencephalopathy
Histoplasmosis, disseminated or extrapulmonary	Salmonella septicemia, recurrent
Isosporiasis, chronic intestinal	Toxoplasmosis of brain
	HIV wasting syndrome

[a] Note that Kaposi's sarcoma is no longer attributed to HIV—see the text.

been quite well known and characterized before AIDS ever appeared is demonstrated by the fact that they had all been described and named.

Not only are a wide variety of illnesses included under ''AIDS''; it is also well established that ''false-positive'' HIV tests can arise from many circumstances, for example a ***variety*** of viral infections including flu and flu ***vaccination***, interferon therapy, many antibodies, blood transfusions, autoimmune diseases, hemophilia, hepatitis, herpes simplex, leprosy, malaria, certain cancers, rheumatoid arthritis, TB, tetanus vaccination, and more—even pregnancy (for a fuller list, and references for each cited condition, see p. 11 in [77]). This is the same as saying that a positive HIV-test is a rather non-specific marker of challenged health.

Yet further HIV anomalies are explicable in similar fashion. The difficulty of generating a vaccine against HIV has sometimes been ascribed to an extraordinarily high rate of mutation of the virus. In other words, ''HIV''—or what the HIV tests detect—is not always exactly the same, in fact is rarely quite the same entity. A non-specific indicator of challenged health—compare a fever, or swollen lymph glands—is naturally not always comprised of the very same biochemical entities.

A supposed high mutation rate has also been suggested as the reason why anti-retroviral drugs soon lose their efficacy in a given individual. A plausible alternative is hormesis. Low levels of a number of substances, as also low levels of radiation [91], are known to be ***beneficial***, presumably because they stimulate

the immune system to fight harder against any health challenge. An antiretroviral drug may act beneficially in this manner when first used and then become harmful rather than helpful upon continued use.

Population Density

Several authors have noted a marked association between F(HIV) and population density. Multivariate analyses among applicants for military service yielded an adjusted odds-ratio of 1.04 or 1.05 for each additional thousand persons per square mile [36, 39]. A different mode of classification [35] found increases—above rural rates—by factors of between 1.16 and 1.29 for rural/urban areas, between 1.78 and 2.29 for urban/rural, and between 3.1 and 5.9 for urban locations[10]. In a study of the Job Corps [41], F(HIV) in metropolitan statistical areas (MSAs) of 50,000—249,000 was 1.18 times that in rural areas; in MSAs between ¼ and 1 million, the ratio was 2.1, and in MSAs above 1 million the ratio was 3.2; in another study [43], these ratios were reported as 1.08, 1.58, and 2.75 respectively.

Similar observations have come from other countries. F(HIV) was found to be twice as high in Quebec as in British Columbia. In the United Kingdom, rates were higher (among pregnant women and newborns) in London and Edinburgh than elsewhere; and higher for both MSM and heterosexuals at STD clinics in London than at STD clinics elsewhere.

One must not jump to the conclusion that an increase in F(HIV) with increasing population density indicates that HIV is sexually transmitted, because that is contrary to actual facts about STIs. Gonorrhea and syphilis break out periodically in various places, often chiefly in specific social groups, and not in proportion to population density. For example, genital herpes is more widespread in rural areas than in suburban ones [92]; this STD ‘‘affects an estimated 60 million Americans. Approximately 500,000 new cases of this incurable viral infection develop annually’’[11]. Population density, an independent risk factor for F(HIV), is not an independent risk factor for STIs.

Concluding Discussion

In Part I [1] of this series of articles, it was shown that the distribution of F(HIV) has been constant over time and in geography during 2 decades, utterly unlike any sexually transmitted infection. Here in Part II, it has been shown that F(HIV) varies among groups in a way that corresponds to the general level of health; it appears to be a rather non-specific, general, indicator, analogous to an inflammation or a fever. F(HIV) varies with age in the way that physiologic capacities or responses do. For example, fevers much above 100 are regarded as life-threatening for adults but not so in young children; analogously, F(HIV) among newborns—apparently healthy newborns—is about four times as great as for older children.

The last variable discussed, population density, is also consistent with the view of F(HIV) as a stress-response indicator. Increased population density does not indicate a greater risk of specifically sexually transmitted disease, but it certainly does correspond to a greater risk of other infectious diseases transmitted by other means—flu, say. Higher population density also presents a greater variety of challenges to good health through the greater presence of air- and water-borne pollutants and allergens in urban areas; just think of smog, say, or of lead before it was removed from gasoline. Thus a general measure of stress-response, a physiologic indicator of health challenges, would be expected to increase monotonically, as F(HIV) does, from rural into urban locations.

So the conclusions reached in Part I [1] are further strengthened: HIV is endemic, not epidemic. It is not a readily transmitted sexual infection. It was not the cause of the AIDS epidemics of the early 1980s.

In human terms, the most significant aspect of this conclusion concerns newborns. Apparently healthy babies test HIV-positive much more frequently than do the healthiest adults (repeat blood donors). Currently this is taken to indicate permanent infection by a deadly virus, and highly toxic anti-retroviral chemotherapy is the standard procedure, sometimes even against the wishes of parents [93]. But since F(HIV) appears to be a normal response to physiologic stress, treating newborns with anti-retrovirals constitutes iatrogenic harm. Even on the standard view of HIV/AIDS, however, such treatment would be unwarranted, for—as noted earlier—some 50–75% of F(HIV) in newborns represents passive antibodies transferred from the mother. Moreover, various studies have shown that 75% of HIV-positive babies revert to negative without medical intervention, and 90% of babies born to healthy HIV-positive mothers test negative without drug therapy (p. 24 in [77]). There is really no excuse for continuing to treat newborns with anti-retrovirals.

In terms of understanding what reports of F(HIV) signify, one more point needs to be made. Any given reversible indicator of a health challenge, for instance an inflammation, will appear and disappear in any given individual but will be continuously present, ***on average***, in some small proportion of the population. F(HIV), we have seen, is measurably above zero even among the most carefully screened groups, repeat blood donors. In groups that probably represent something like the average health of the population, say, applicants for military service (see Tables 1 & 2) or those surveyed in the National Health and Nutrition surveys of households (Table 2), F(HIV) is consistently at a level of a few per thousand. As noted in Part I [1], the same level has been found among low-risk populations in other countries: Canada, Germany, South Africa, United Kingdom. This evidently represents the ***normal*** level of ***reversible*** F(HIV), the ‘‘HIV’’ response to common infections, inflammations, allergies, and the like; it does not represent infection with a human immunodeficiency virus. Where the rates of incidence of new ‘‘HIV infections’’ are studied, as they have been, for example, in military cohorts and among sexual partners, a few per thousand should be expected to appear spontaneously; and therefore a rate of new

‘‘infections’’ on that order, a few per thousand, should be interpreted as ***no sign at all of any transmitted infection***. In point of fact, the observed rates in studies of ‘‘new incidence of HIV infection’’ have indeed been of this order of magnitude, a few per thousand [26, 29, 94–104]. This modifies the conclusion reached in Part I [1], where the rate of infection was found to be so low that it could not sustain an epidemic: actually, there is no evidence that ‘‘HIV’’ is transmitted at all.

Notes

[1] www.theperthgroup.com/index.shtml, accessed 2 April 2005.

[2] It should be borne in mind that information about HIV among people known to be MSM comes from surveys and studies on only a minority of gay men, typically those who practice a ‘‘liberated’’ ‘‘fast-lane’’ lifestyle that is rather obviously less than healthy—it is defined by incessant promiscuity and use of drugs. Insider descriptions of fast-lane gay behavior can be found in the memoirs of Michael Callen [2] and of Richard Berkowitz [3], in the novel *Faggots* [4], and in John Lauritsen’s essays (pp. 188–200 in [5]). Knowledgeable insiders have suggested that AIDS struck, and continues to strike, only those gay men who abuse drugs (pp. 191–3 in [5]).

While it is fairly common knowledge that the AIDS epidemic never spread to the general heterosexual population, I have not seen it pointed out that it did not sweep the general gay population either—or rather, I had not seen it pointed out until I was ready to send this manuscript for processing and discovered that the point had been cogently expounded by journalist Tony Brown in 1995 (p. 147 in [6]). By 2003, the cumulative total of AIDS cases among MSM, including MSM-IDU, was well under half a million [7]. Most estimates of the percentage of men who have sex with other men are between 2 and 10%—higher if one includes men who occasionally rather than exclusively have sex with other men. Say the figure is 5%; that would come to 7 million in the United States. Half a million among those represents only about 7% of all gay men. Double or treble that percentage to take account of men who are not of a sexually active age, and it still remains a minority among gay men.

Altogether, then, the data do not establish a connection between gay sex as such by contrast to MSM who are promiscuous and use drugs. Health-risk factors may well be exactly the same for gay men as they are for other people. Admittedly, it seems that promiscuity is more common among gay men than among heterosexual men; but that is not because being gay promotes promiscuity, it is because ***being male promotes promiscuity***. As Jad Adams (pp. 123 ff. in [8]) points out, among heterosexual men promiscuity is curbed by the lack of promiscuous tendency among the men’s female partners; in the gay community, that curb is missing. Nevertheless, the majority of gay men are not promiscuous, and it is the promiscuous minority that came down with AIDS.

[3] Though often used interchangeably, ''sexually transmitted infection (STI)'' is not strictly the same as ''sexually transmitted disease (STD)''.

[4] Letter to the author, dated 19 May 2005, from Shari Steinberg, Divisions of HIV/AIDS Prevention, National Center for HIV, STD, and TB Prevention, Centers for Disease Control and Prevention.

[5] It is sometimes suggested that people infected with syphilis or gonorrhea are more likely to contract HIV as well. But it was found that the drug, sexual, and STD histories of HIV-positive and HIV-negative adolescents showed no significant differences [61]. F(HIV) among non-MSM, non-IDU patients at STD clinics in New York was not associated with exposure to prostitutes, whereas one might expect such an association for an STI [54]. Annually, about 12 million Americans contract an STI [19]; if that enhanced infection with HIV, then there ought to be a much higher level of F(HIV) than there is, for the latest estimate from the Centers for Disease Control and Prevention, as in the 1980s, is that the ***total***, ***cumulative*** number of Americans infected by HIV is about 1 million (for detailed references, see Part I [1]).

[6] That oxidative processes, particularly those initiated by ''free radicals'', are involved in a variety of health-threatening or health-damaging biological processes is well established and has become well known. ''Anti-oxidant'' supplements have accordingly been widely marketed for years.

[7] I am grateful to Dr. Christian Fiala for suggesting this analogy.

[8] http://www.chclibrary.org/micromed/00066890.html, accessed 12 June 2005.

[9] http://www.cancer.gov/cancertopics/factsheet/cancerhealthdisparities, accessed 19 September 2005.

[10] The lower figures are from adjusted, the higher from unadjusted odds-ratios.

[11] www.niaid.nih.gov/factsheets/stdinfo.htm, dated July 1999, accessed 25 May 2005.

References

1. Bauer, H. H. (2005). Demographic characteristics of HIV: I. How did HIV spread? *Journal of Scientific Exploration, 19,* 567–603.
2. Callen, M. (1990). *Surviving AIDS.* HarperCollins.
3. Berkowitz, R. (2003). *Stayin' Alive: The Invention of Safe Sex.* Westview.
4. Kramer, L. (1978). *Faggots.* Random House (and many other editions).
5. Lauritsen, J. (1993). *The AIDS War: Propaganda, Profiteering and Genocide from the Medical-Industrial Complex.* ASKLEPIOS.
6. Brown, T. (1995). *Black Lies, White Lies: The Truth According to Tony Brown.* William Morrow.
7. Centers for Disease Control and Prevention. (2004). *HIV/AIDS Surveillance Report,* 2003, *15,* 1–46. Available at: www.cdc.gov/hiv/stats/hasrlink.htm. Accessed 30 June 2005.
8. Adams, J. (1989). *AIDS: The HIV Myth.* St. Martin's Press.
9. Vlahov, D., Brewer, T. F., Castro, K. G., Narkunas, J. P., Salive, M. E., Ullrich, J., & Muñoz, A. (1991). Temporal trends of human immunodeficiency virus Type 1 (HIV-1) infection among inmates entering a statewide prison system, 1985–1987. *Journal of the American Medical Association, 265,* 1129–1132.
10. Weisfuse, I. B., Greenberg, B. L., Back, S. D., Makki, H. A., Thomas, P., Rooney, W. C., & Rautenberg, E. L. (1991). HIV-1 infection among New York City inmates. *AIDS, 5,* 1133–1138.

11. Centers for Disease Control and Prevention. (2001). *HIV prevalence trends in selected populations in the United States—Results from national serosurveillance, 1993–1997.* 1–51.
12. Smith, O. F., Mikl, J., Truman, B. I., Lessner, L., Lehman, J. S., Stevens, R. W., Lord, E. A., Broaddus, R. K., & Morse, D. L. (1991). VI. Infection among women entering the New York State correctional system. *American Journal of Public Health, 81,* Supplement, 35–40.
13. Weinstock, H. S., Sidhu, J., Gwinn, M., Karon, J., & Petersen, L. R. (1995). Trends in HIV seroprevalence among persons attending sexually transmitted disease clinics in the United States, 1988–1992. *Journal of Acquired Human Immunodeficiency Syndrome and Human Retrovirology, 9,* 514–522.
14. Centers for Disease Control and Prevention. (1997). *HIV Counseling and Testing in Publicly Funded Sites—1995 Summary Report.* September.
15. Centers for Disease Control and Prevention.(1998). *HIV Counseling and Testing in Publicly Funded Sites—1996 Annual Report.* May.
16. Centers for Disease Control and Prevention. (2001). *HIV Counseling and Testing in Publicly Funded Sites—Annual Report 1997 and 1998.*
17. Dondero, T. J., & Gill, O. N. (1991). Large-scale HIV surveys: what has been learned? *AIDS, 5,* Supplement 2, S63–S69.
18. Gayle, H. D., & D'Angelo, L. J. (1991). Epidemiology of acquired immunodeficiency syndrome and human immunodeficiency virus infection in adolescents. *Pediatric Infectious Diseases Journal, 10,* 322–328.
19. Lindegren, M. L., Hanson, C., Miller, K., Byers, R. H., & Onorato, I. (1994). Epidemiology of human immunodeficiency virus infection in adolescents, United States. *Pediatric Infectious Diseases Journal, 13,* 525–535.
20. Kaslow, R. A., & Francis, D. P. (1989). *The Epidemiology of AIDS: Expression, Occurrence, and Control of Human Immunodeficiency Virus Type 1 Infection.* Oxford University Press.
21. Schorr, J. B., Berkowitz, A., Cumming, P. D., Katz, A. J., & Sandler, S. G. (1985). Prevalence of HTLV-III antibody in American blood donors. *New England Journal of Medicine, 313,* 384–385.
22. Petersen, L. R., & Doll, L. S. (1991). Human immunodeficiency virus Type 1-infected blood donors: epidemiologic, laboratory, and donation characteristics. *Transfusion, 31,* 698–703.
23. Glynn, S. A., Kleinman, S. H., Schreiber, G. B., Busch, M. P., Wright, D. J., Smith, J. W., Nass, C. C., & Williams, A. E., for the Retrovirus Epidemiology Donor Study (REDS). (2000). Transfusion-transmissible viral infections in US blood donors, 1991 to 1996. *Journal of the American Medical Association, 284,* 229–235.
24. Dodd, R. Y., Notari, E. P., IV, & Stramer, S. L. (2002). Current prevalence and incidence of infectious disease makers and estimated window-period risk in the American Red Cross blood donor population. *Transfusion, 42,* 975–979.
25. Zou, S., Notari, E. P., IV, Stramer, S. L., Wahab, F., Musavi, F., & Dodd, R. Y., for the ARCNET Research Group. (2004). Patterns of age- and sex-specific prevalence of major blood-borne infections in United States blood donors, 1995 to 2002: American Red Cross blood donor study. *Transfusion, 44,* 1640–1647.
26. Garland, F. C., Mayers, D. L., Hickey, T. M., Miller, M. R., Shaw, E. K., Gorham, E. D., Bigbee, L. R., & McNally, M. M. (1989). Incidence of human immunodeficiency virus seroconversion in US Navy and Marine Corps personnel, 1986 through 1988. *Journal of the American Medical Association, 262,* 3161–3165.
27. Army Medical Surveillance Activity. (2004). Update: Human immunodeficiency virus, Type 1 (HIV-1), antibody screening among active and reserve component soldiers and civilian applicants for military service, 1985–June 2004. *Medical Surveillance Monthly Report, 10*(4), 2–8.
28. Army Medical Surveillance Activity. (1996). Supplement—HIV in the Army. *Medical Surveillance Monthly Report, 6*(2), 12–14.
29. McNeil, J. G., Brundage, J. F., Wann, F., Burke, D. S., Miller, R. N., & the Walter Reed Retrovirus Research Group. (1989). Direct measurement of human immunodeficiency virus seroconversions in a serially tested population of young adults in the United States Army, October 1985 to October 1987. *New England Journal of Medicine, 320,* 1581–1585.
30. Kelley, P. W., Miller, R. N., Pomerantz, R., Wann, F., Brundage, J. F., & Burke, D. S. (1990). Human immunodeficiency virus seropositivity among members of the active duty US Army 1985–89. *American Journal of Public Health, 80,* 405–410.
31. McNeil, J. G., Brundage, J. F., Gardner, L. I., Wann, F. Z., Renzullo, P. O., Redfield, R. R., Burke, D. S., Miller, R. N., & the US Army Retrovirus Research Group. (1991). Trends of HIV

seroconversion among young adults in the US Army, 1985 to 1989. *Journal of the American Medical Association, 265,* 1709–1714.
32. Renzullo, P. O., McNeil, J. G., Wann, Z. F., Burke, D. S., Brundage, J. F., & the United States Military Medical Consortium for Applied Retroviral Research. (1995). Human immunodeficiency virus Type-1 seroconversion trends among young adults serving in the United States Army, 1985–1993. *Journal of Acquired Human Immunodeficiency Syndrome and Human Retrovirology, 10,* 177–185.
33. Renzullo, P. O., Sateren, W. B., Garner, R. P., Milazzo, M. J., Birx, D. L., & McNeil, J. G. (2001). HIV-1 seroconversion in United States Army active duty personnel, 1985–1999. *AIDS, 15,* 1569–1574.
34. Cowan, D. N., Pomerantz, R. S., Wann, Z. F., Goldenbaum, M., Brundage, J. F., Miller, R. N., Burke, D. S., Carroll, C. A., & the Walter Reed Retrovirus Research Group. (1990). Human immunodeficiency virus infection among members of the Reserve Components of the US Army: Prevalence, incidence, and demographic characteristics. *Journal of Infectious Diseases, 162,* 827–836.
35. Sateren, W. B., Renzullo, P. O., Carr, J. K., Birx, D. L., & McNeil, J. G. (2003). HIV-1 infection among civilian applicants for US military service, 1985 to 2000: Epidemiology and geography. *Journal of Acquired Immune Deficiency Syndromes, 32,* 215–222.
36. Burke, D. S., Brundage, J. F., Herbold, J. R., Berner, W., Gardner, L. I., Gunzenhauser, J. D., Voskovitch, J., & Redfield, R. R. (1987). Human immunodeficiency virus infections among civilian applicants for United States military service, October 1985 to March 1986. *New England Journal of Medicine, 317,* 131–136.
37. Centers for Disease Control and Prevention. (1998). *National HIV Prevalence Surveys—1997 Summary.* 1–25.
38. Brundage, J. F., Burke, D. S., Gardner, L. I., McNeil, J. G., Goldenbaum, M., Visintine, R., Redfield, R. R., Peterson, M., & Miller, R. N. (1990). Tracking the spread of the HIV infection epidemic among young adults in the United States: Results of the first four years of screening among civilian applicants for U.S. military service. *Journal of Acquired Immune Deficiency Syndromes, 3,* 1168–1180.
39. Burke, D. S., Brundage, J. F., Goldenbaum, M., Gardner, L. I., Peterson, M., Visintine, R., Redfield, R. R., & the Walter Reed Retrovirus Research Group. (1990). Human immunodeficiency virus infections in teenagers—Seroprevalence among applicants for US military service. *Journal of the American Medical Association, 263,* 2074–2077.
40. Brundage, J. F., Burke, D. S., Gardner, L. I., Visintine, R., Peterson, M., & Redfield, R. R. (1988). HIV infection among young adults in the New York City area: Prevalence and incidence estimates based on antibody screening among civilian applicants for military service. *New York State Journal of Medicine, May,* 232–235.
41. St. Louis, M. E., Conway, G. A., Hayman, C. R., Miller, C., Peterson, L. R., & Dondero, T. J. (1991). Human immunodeficiency virus infection in disadvantaged adolescents—Findings from the Job Corps. *Journal of the American Medical Association, 266,* 2387–2391.
42. Conway, G. A., Epstein, M. R., Hayman, C. R., Miller, C. A., Wendell, D. A., Gwinn, M., Karon, J. M., & Petersen, L. R. (1993). Trends in HIV prevalence among disadvantaged youths—Survey results from a national job training program, 1988 through 1992. *Journal of the American Medical Association, 269,* 2887–2889.
43. Valleroy, L. A., MacKellar, D. A., Karon, J. M., Janssen, R. S., & Hayman, C. R. (1998). HIV Infection in Disadvantaged Out-of-School Youth: Prevalence for U.S. Job Corps Entrants, 1990 through 1996 [Epidemiology]. *Journal of Acquired Human Immunodeficiency Syndrome and Human Retrovirology, 19,* 67–73.
44. McQuillan, G. M., Khare, M., Ezzati-Rice, T. M., Karon, J. M., Schable, C. A., & Murphy, R. S. (1994). The seroepidemiology of human immunodeficiency virus in the United States household population: NHANES III, 1988–1991. *Journal of Acquired Immune Deficiency Syndromes, 7,* 1195–1201.
45. Horsburgh, C. R., Jarvis, J. Q., McArthur, T., Ignacio, T., & Stock, P. (1990). Seroconversion to human immunodeficiency virus in prison inmates. *American Journal of Public Health, 80,* 209–210.
46. Cournos, F., Horwath, E., Guido, J. R., McKinnon, K., & Hopkins, N. (1994). HIV-1 infection at two public psychiatric hospitals in New York City. *AIDS Care, 6,* 443–452.
47. Cournos, F., Empfield, M., Horwath, E., McKinnon, K., Meyer, I., Schrage, H., Currie, C., & Agosin, B. (1991). HIV seroprevalence among patients admitted to two psychiatric hospitals. *American Journal of Psychiatry, 148,* 1225–1230.

48. Lombardo, J. M., Kloser, P. C., Pawel, B. R., Trost, R. C., Kapila, R., & St. Louis, M. E. (1991). Anonymous human immunodeficiency virus surveillance and clinically directed testing in a Newark, NJ, hospital. *Archives of Internal Medicine, 151,* 965–968.
49. St. Louis, M. E., Rauch, K. J., Petersen, L. R., Anderson, J. E., Schable, C. A., Dondero, T. J., & the Sentinel Hospital Surveillance Group. (1990). Seroprevalence rates of human immunodeficiency virus infection at sentinel hospitals in the United States. *New England Journal of Medicine, 323,* 213–218.
50. Quinn, T. C., Glasser, D., Cannon, R. O., Matuszak, D. L., Dunning, R. W., Kline, R. L., Campbell, C. H., Israel, E., Fauci, A. S., & Hook, E. W., III. (1988). Human immunodeficiency virus infection among patients attending clinics for sexually transmitted diseases. *New England Journal of Medicine, 318,* 197–203.
51. Weinstock, H., Sweeney, S., Satten, G. A., & Gwinn, M., for the STD Clinic HIV Seroincidence Study Group. (1998). HIV seroincidence and risk factors among patients repeatedly tested for HIV attending sexually transmitted disease clinics in the United States, 1991 to 1996 [Epidemiology]. *Journal of Acquired Immune Deficiency Syndromes, 19,* 506–512.
52. McCray, E., Onorato, I. M., & the Field Services Branch. (1992). Sentinel surveillance of human immunodeficiency virus infection in sexually transmitted disease clinics in the United States. *Sexually Transmitted Diseases, 19,* 235–241.
53. Quinn, T. C., Cannon, R. O., Glasser, D., Groseclose, S. L., Brathwaite, W. S., Fauci, A. S., & Hook, E. W., III. (1990). The association of syphilis with risk of human immunodeficiency virus infection in patients attending sexually transmitted disease clinics. *Archives of Internal Medicine, 150,* 1297–1302.
54. Rabkin, C. S., Thomas, P. A., Jaffe, H. W., & Schultz, S. (1987). Prevalence of antibody to HTLV-III/LAV in a population attending a sexually transmitted diseases clinic. *Sexually Transmitted Diseases, 14,* 48–51.
55. Moss, A. R., Vranizan, K., Gorter, R., Bacchetti, P., Watters, J., & Osmond, D. (1994). HIV seroconversion in intravenous drug users in San Francisco, 1985–90. *AIDS, 8,* 223–231.
56. Hahn, R. A., Onorato, I. M., Jones, S., & Dougherty, J. (1989). Prevalence of HIV infection among intravenous drug users in the United States. *Journal of the American Medical Association, 261,* 2677–2684.
57. Valleroy, L. A., MacKellar, D. A., Karon, J. M., Rosen, D. H., McFarland, W., Shehan, D. A., Stoyanoff, S. R., LaLota, M., Celentano, D. D., Koblin, B. A., Thiede, H., Katz, M. H., Torian, L. V., & Janssen, R. S., for the Young Men's Survey Study Group. (2000). HIV prevalence and associated risks in young men who have sex with men. *Journal of the American Medical Association, 284,* 198–204.
58. Centers for Disease Control and Prevention. (2001). *Morbidity and Mortality Weekly Report, 50*(21), 440–443.
59. Hessol, N. A., Lifson, A. R., O'Malley, P. M., Doll, L. S., Jaffe, H. W., & Rutherford, G. W. (1989). Prevalence, incidence, and progression of human immunodeficiency virus infection in homosexual and bisexual men in hepatitis B vaccine trails, 1978–88. *American Journal of Epidemiology, 130,* 1167–1175.
60. Wendell, D. A., Onorato, I. M., McCray, E., Allen, D. M., & Sweeney, P. A. (1992). Youth at risk: Sex, drugs and human immunodeficiency virus. *American Journal of Diseases of Children, 146,* 76–81.
61. Futterman, D., Hein, K., Reuben, N., Dell, R., & Shaffer, N. (1993). Human immunodeficiency virus-infected adolescents: The first 50 patients in a New York City program. *Pediatrics, 91,* 730–735.
62. Fiala, C. (2000). *Epidemiological evidence against heterosexual transmission of HIV and against prevention-campaigns.* www.virusmyth.net/aids/data/cfepidem.htm (25 June; accessed 10 March 2005).
63. Centers for Disease Control and Prevention. (1987). *Morbidity and Mortality Weekly Report, 36* Supplement 6, 1–20.
64. Altice, F. L., Mostashari, F., Selwyn, P. A., Checko, P. J., Singh, R., Tanguay, S., & Blanchette, E. A. (1998). Predictors of HIV infection among newly sentenced male prisoners. *Journal of Acquired Human Immunodeficiency Syndrome and Human Retrovirology, 18,* 444–453.
65. Quinn, T. C. (1987). AIDS in Africa: Evidence for heterosexual transmission of the human immunodeficiency virus. *New York State Journal of Medicine,* May, 286–289.

66. Novick, L. F., Glebatis, D. M., Stricof, R. L., MacCubbin, P. A., Lessner, L., & Berns, D. S. (1991). Newborn seroprevalence study: Methods and results. *American Journal of Public Health, 81,* Supplement, 15–21.
67. Papadopulos-Eleopulos, E., Turner, V. F., Papadimitriou, J. M., Alfonso, H., Page, B. A. P., Causer, D., Mhlongo, S., Fiala, C., Miller, T., Brink, A., & Hodgkinson, N. (2001). *Mother to Child Transmission of HIV and its Prevention with AZT and Nevirapine: A Critical Analysis of the Evidence.* The Perth Group, Perth, Western Australia; ISBN 1-876763-72-8; available at http://www.theperthgroup.com/monograph.html (3.51 MB pdf), accessed 16 November 2005.
68. Gayle, H. D., Keeling, R. P., Garcia-Tunon, M., Kilbourne, B. W., Narkunas, J. P., Ingram, F. R., Rogers, M. F., & Curran, J. W. (1990). Prevalence of the human immunodeficiency virus among university students. *New England Journal of Medicine, 323,* 1538–1541.
69. Petersen, L. R, White, C. R. & the Premarital Screening Group. (1990). Premarital screening for antibodies to human immunodeficiency virus Type 1 in the United States. *American Journal of Public Health, 80,* 1087–1090.
70. Centers for Disease Control and Prevention. (1986). Human T-lymphotropic virus Type III/ lymphadenopathy-associated virus antibody prevalence in U.S. military recruit applicants. *Morbidity and Mortality Weekly Report, 35*(26), 421–424.
71. D'Angelo, L. J., Getson, P. R., Luban, N. L. C., & Gayle, H. D. (1991). Human immunodeficiency virus infection in urban adolescents: Can we predict who is at risk? *Pediatrics, 88,* 982–986.
72. HIV Seroepidemiology Unit, San Francisco Department of Public Health. (1998). *HIV Epidemiology Report—Data Available to 1998.* December 1998, revised 11 May 1999.
73. Vlahov, D., Brewer, F., Muñoz, A., Hall, D., Taylor, E., & Polk, B. F. (1989). Temporal trends of human immunodeficiency virus Type 1 (HIV-1) infection among inmates entering a statewide prison system, 1985–1987. *Journal of Acquired Immune Deficiency Syndromes, 2,* 283–290.
74. Krueger, L. E., Wood, R. W., Diehr, P. H., & Maxwell, C. L. (1990). Poverty and HIV seropositivity: The poor are more likely to be infected. *AIDS, 4,* 811–814.
75. Osmond, D. H., Page, K., Wiley, J., Garrett, K., Sheppard, H. W., Moss, A. R., Schrager, L., & Winkelstein, W. (1994). HIV infection in homosexual and bisexual men 18 to 29 years of age: The San Francisco Young Men's Health Study. *American Journal of Public Health, 84,* 1933–1937.
76. DiClemente, R. J. (1990). The emergence of adolescents as a risk group for human immunodeficiency virus infection. *Journal of Adolescent Research, 5,* 7–17.
77. Maggiore, C. (2000). *What If Everything You Thought You Knew about AIDS Was Wrong?* American Foundation for AIDS Alternatives, 4th rev. ed.
78. Root-Bernstein, R. (1993). *Rethinking AIDS—The Tragic Cost of Premature Consensus.* Free Press.
79. Root-Bernstein, R. (1995). The Duesberg phenomenon: What does it mean? *Science, 267,* 159–160.
80. Duesberg, P. (1996). *Inventing the AIDS Virus.* Regnery.
81. Papadopulos-Eleopulos, E., Turner, V. F., Papadimitriou, J. M., & Causer, D. (1995). Factor VIII, HIV and AIDS in haemophiliacs: An analysis of their relationship. *Genetica, 95,* 25–50.
82. Chmiel, J. S., Detels, R., Kaslow, R. A., Van Raden, M., Kingsley, L. A., Brookmeyer, R., & the Multicenter AIDS Cohort Study Group. (1987). Factors associated with prevalent human immunodeficiency virus (HIV) infection in the multicenter AIDS cohort study. *American Journal of Epidemiology, 126,* 568–77.
83. Gallo, R. C., Salahuddin, S. Z., Popovic, M., Shearer, G. M., Kaplan, M., Haynes, B. F., Palker, T. J., Redfield, R., Oleske, J., Safai, B., White, G., Foster, P., & Markham, P. D. (1984). Frequent detection and isolation of cytopathic retroviruses (HTLV-III) from patients with AIDS and at risk for AIDS. *Science, 224,* 500–503.
84. Cohen, J. (1994). Is a new virus the cause of KS? *Science, 266,* 1803–1804.
85. *Kaposi's Sarcoma (PDQ®): Treatment—Health Professional Version;* www.cancer.gov/ cancertopics/pdq/treatment/kaposis/healthprofessional#Reference1.1, last modified 29 September 2003; accessed 14 September 2005.
86. Torian, L. V., Makki, H. A., Menzies, I. B., Murrill, C. S., & Weisfuse, I. B. (2002). HIV infection in men who have sex with men. *Sexually Transmitted Diseases, 29*(2), 73–78.
87. Bruneau, J., Lamothe, F., Franco, E., Lachance, N., Désy, M., Soto, J., & Vincelette, J. (1997). High rates of HIV infection among injection drug users participating in needle exchange

programs in Montreal: Results of a cohort study. *American Journal of Epidemiology, 146,* 994–1002.
88. Duesberg, P., Koehnlein, C., & Rasnick, D. (2003). The chemical bases of the various AIDS epidemics: Recreational drugs, anti-viral chemotherapy and malnutrition. *Journal of Bioscience, 28,* 383–412.
89. Hodgkinson, N. (1996). *AIDS: The Failure of Contemporary Science.* Fourth Estate.
90. Centers for Disease Control and Prevention. (1992). *HIV/AIDS Surveillance Report*, January, 1–22, year-end edition; U.S. AIDS cases reported through December 1991.
91. Kauffman, J. M. (2003). Radiation hormesis: Demonstrated, deconstructed, denied, dismissed, and some implications for public policy. *Journal of Scientific Exploration, 17,* 389–407.
92. Johnson, R. E., Nahmias, A. J., Magder, L. S., Lee, F. K., Brooks, C. A., & Snowden, C. B. (1989). A seroepidemiologic survey of the prevalence of herpes simplex virus Type 2 infection in the United states. *New England Journal of Medicine, 321,* 7–12.
93. Scheff, L. (2004). Orphans on trial: Abandoned kids are force-fed experimental AIDS drugs at a Catholic children's home in Washington Heights. And the city wants it that way. *New York Press*, 13 July; available at http://nypress.com/print.cfm?content_id=10614, accessed 15 September 2005.
94. Royce, R. A., Sena, A., Cates, W., Jr., & Cohen, M. S. (1997). Sexual transmission of HIV. *New England Journal of Medicine, 336,* 1072–78, cited in Brewer, D. D., Brody, S., Drucker, E., Gisselquist, D., Minkin, S. F., Potterat, J. J., Rothenberg, R. B., & Vachon, F. (2003). Mounting anomalies in the epidemiology of HIV in Africa: Cry the beloved paradigm. *International Journal of STD & AIDS, 14,* 144–147.
95. Anon. (1986). New studies focus on AIDS transmission chances. *New York Times*, 1 October, B2.
96. Bernstein, N. (1999). For subjects in Haiti study, free AIDS care has a price. *New York Times*, 6 June, I1, I10.
97. Padian, N. S., Shiboski, S. C., Glass, S. O., & Vittinghoff, E. (1997). Heterosexual transmission of Human Immunodeficiency Virus (HIV) in Northern California: Results from a ten-year study. *American Journal of Epidemiology, 146,* 350–357.
98. Wiley, J. A., Herschkorn, S. J., & Padian, N. S. (1989). Heterogeneity in the probability of HIV-1 transmission per sexual contact: the case of male-to-female transmission in penile-vaginal intercourse. *Statistics in Medicine, 8,* 93–102.
99. Peterman, T. A., Stonebumer, R. L., Allen, J. R., Jaffe, H. W., & Curran, J. W. (1988). Risk of human immunodeficiency virus transmission from heterosexual adults with transfusion-associated infections. *Journal of the American Medical Association, 259,* 55–58.
100. Padian, N., Marquis, L., Francis, D. P., Anderson, R. E., Rutherford, G. W., O'Malley, P. M., & Winkelstein, W. (1987). Male-to-female transmission of human immunodeficiency virus. *Journal of the American Medical Association, 258,* 788–790.
101. Duerr, A., Xia, Z., Nagachinta, T., Tovanabutra, S., Tansuhaj, A., & Nelson, K. (1994). Probability of male-to-female HIV-1 transmission among married couples in Chiang Mai, Thailand. *Tenth International Conference on AIDS.* Yokohama, August [Abstract 105C]; cited in [104].
102. Shiboski, S. C., & Padian, N. S. (1998). Epidemiological evidence for time variation in HIV-1 infectivity. *Journal of Acquired Human Immunodeficiency Syndrome and Human Retrovirology, 19,* 527–535; cited in [104].
103. Hugonnet, S., Mosha, F., Todd, J., Mugeye, K., Klokke, A., Ndeki, L., Ross, D., Grosskurth, H., & Hayes, R. (2002). Incidence of HIV infection in stable sexual partnerships: A retrospective cohort study of 1802 couples in Mwanza Region, Tanzania. *Journal of Acquired Immune Deficiency Syndromes, 30,* 73–80.
104. Chakraborty, H., Sena, P. K., Helms, R. W., Vernazza, P. L., Fiscus, S. A., Eron, J. J., Patterson, B. K., Coombs, R. W., Krieger, J. N., & Cohen, M. S. (2001). Viral burden in genital secretions determines male-to-female sexual transmission of HIV-1: A probabilistic empiric model. *AIDS, 15,* 621–627.