

Andrew Moss
212 - 663 - 0656

11-4-93

Darrow was 1st person
to intro me to
AIDS pat.
That was spring '82
Chelsea clinic
pat. was high class
cellist, classy guy
was caught in nightmare
I saw there was the
man caught in the
world he never could
have imagined
Darrow was secretive...
he wanted to work in
SF

all pat. zero was wrong

He wanted an ally in SF
he was very middle
class guy, didn't really
fit in
He was better than
Curran & Jaffe --- "it's
all our attitude"

His 1st study:

only data was case data
Duty & COE

So I went down w/ Dennis
& organized Selma, put

in a meeting system →
got it going

We also ripped her data off --
fished her off

We did little study

~ 1:300 gay men
had AIDS already
(end 1982) and little came
going up

UCSF group: we had
vote PV vs. JZ on who
run as PI for res.
group. Paul won
about 8 of us

Jan '83 started w/ Michael
Gorman & we went
out & gave the data
to gay groups, HltH
dept., highly placed gay
officials (Bill Krause,
etc.).

~~Also~~ People were really
stunned. Mouths fell open

Spring '83: Big power
struggle what to do w/
the data. Everybody
wanted it (incl. shells)

M. Corant gave a talk.
= NY spring '83 &
gave the data
Lang Kramer was there

Then we leased it to Skitt
- He did a story -
Wrote it as letter to
Lancet

Counted all cases; zip coded
divided SF into 3 areas
(by gay areas)

↳ if used never-married
men by census in
area (it is an over-
estimate of gays)
& overlaid cases &
computed

see Lancet May 83(?)

OK effect was amazing
it got huge media
attn, even in
Playboy

It was useful for me
in terms get \$ for res.
We noted the gay
political clubs (Tobias
H. Muth) →

We got to all the SF supervisors,
gay advisers →
got \$60,000 from
city in July '83
Which was big bucks
the
amazing business

~~Coto~~

Pat zero:

they got it wrong
Was pub'd in a crappy
study

If you look at these
clusters

↳ he asked: did you
have sex in "X" in
last year

(we all thought there
was a 1 year ^{incubator} cluster)
So the links in the
cluster are imaginary

Selma & Darrow believed it
So did Randy

I wrote NY Rev. Books
a critique

Dugas got crucified for
it.

Why did they give Randy
his name?

What should have done is
a case controlled study
on freq. of sex activity

CDC Annals of Int Med '84 study
they said no assoc w/
anal sex
↳ crappy study
↳ the controls were
from STD clinic &
were some of the
were (+)

Campbell He was a nurse
worked SF general
was sort of sleezy
not a hero

Grim
white
IVDU

Mrs. Proffit ← she must have gotten infected early
old man black
she had babies in
w/ AIDS (had 2)?
she was demented

they
never
could
get
him
to

We did a prostitute study
in Tenderloin -- she
was only one who was (+)

Did mathematical stats.
@ Stanford

very
early
HIV
case

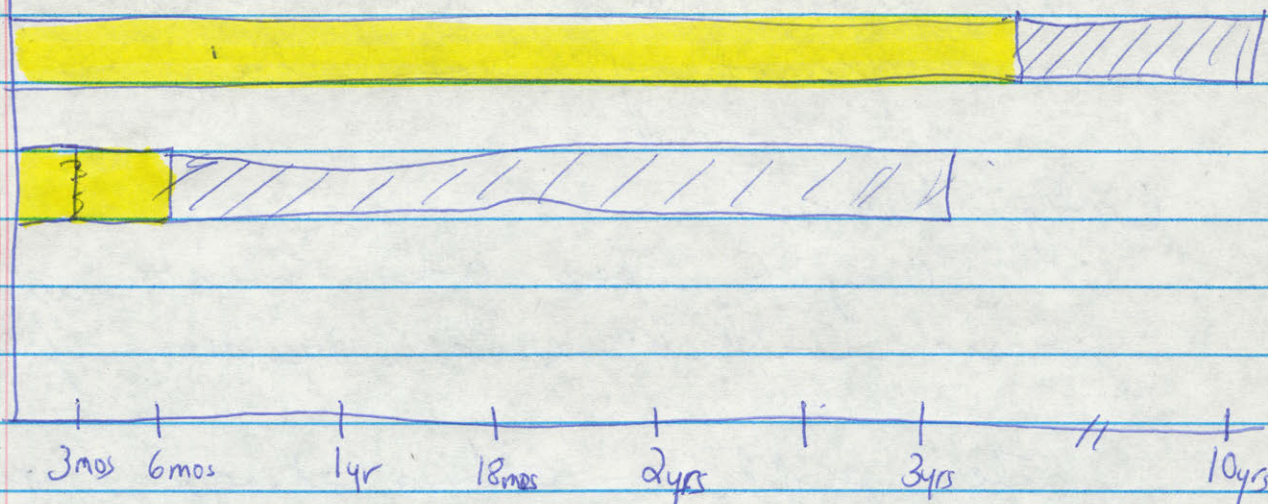
If you're outside the
CDC they're no fun.
Competition
don't cooperate
no fun
you have to resign to
being treated like
shit

There's still no reason HIV
started w/gays here
- look at Thailand &
Brazil --- huge TV
HIV stuff
and little S. Europe

AIDS TIMELINE

High Risk
Homosexual
MEN

Hemophiliacs
and indiv.
who get
infected
from blood
products



- Maximum documented time documented during which people are "silently infected"
- Average amt. of time the virus incubates before causing full-blown AIDS

Amer. Red Cross

212 - 787-1000

202 - 737 - 8300 (♀) Ansley Harkness
Dr. ^{Lewellys} F. Barker → Shr. VP Amer. Red Cross
Bld. Servs.

"You know... the people involved are all described as donors. we in high risk..." "these people we do everything in our power not to collect bld. from."

"We know it's not 100% effective, so we test for Ab to HIV."

During that 4 yr. period (of test) CDC has only doc. 2 cases of AIDS that were transf. assoc."

over
2000
Circ
before
testing

"We don't think this paper."

"We have known there was a period of time before."

Only homos?

"Anyone that gets infected w/ the virus will have a window period."

"The people in this study, none of them would be old doors. Homosex, $\Rightarrow$ have been very coop."

"We don't live in a perfect world."

"If we had a better test, we would be interested."

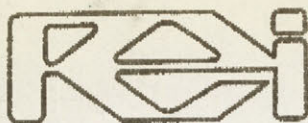
Ag test?

"I haven't seen any evid. to supp. it would be a helpful option."

"What we're interested in is an improved Ab test."

"I think it's unlikely there will be a test that will pick up everything but we'll look at all test that come along."

Need educ. people about not donating



®

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Harbor-UCLA Medical Center

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FRANK J. DE SANTIS, CAE
President

May 31, 1989

NOTE: EMBARGOED FOR RELEASE 6 p.m, Wednesday, May 31.

Homosexual men at very high risk may be infected with the human immunodeficiency virus type 1 (HIV-1) for as long as 35 months before antibodies to HIV-1 can be detected by standard available blood tests. These findings are the result of tissue culture studies and polymerase chain reaction (PCR) tests which were conducted to amplify small amounts of HIV DNA. PCR is a procedure that amplifies the genetic material of the virus several thousand fold, providing enough DNA to find "footprints" of the AIDS virus.

"The degree of infectiousness of these individuals is unknown at the present time, but these studies reemphasize the importance that men who are engaged in high risk sexual practices should not donate blood," said Dr. David T. Imagawa. Imagawa is the primary author of a research paper being published in the June 1 issue of the New England Journal of Medicine.

Between February and October 1986, cultures of peripheral blood lymphocytes were performed from 133 seronegative men (those who tested negative on a standard blood test for presence of HIV-1). The 133 men were selected for the study because they continued to engage in high risk sexual practices. The HIV-1 was isolated in tissue culture samples from 31 of the 133 men (23 percent).

Four of the 31 men seroconverted (tested positive) 11 to 17 months after the initial isolation of the virus, but 27 of the 31 men had not seroconverted for as long as 35 months after the positive tissue culture. PCR tests have yet to be conducted on these 27 men.

-more-

Imagawa/AIDS
2-2-2

Imagawa directs a team of investigators who conduct AIDS research at the Research and Education Institute (REI), Inc., Harbor-UCLA Medical Center in Torrance. The research is part of the Multicenter AIDS Cohort Study, a collaborative program involving researchers from REI, UCLA, Johns Hopkins University in Baltimore, the University of Pittsburgh, and Northwestern University in Chicago. Dr. Roger Detels of the UCLA School of Public Health is the principal investigator for the research being conducted in the Los Angeles area at UCLA and REI. The project is supported in part by grants from the National Institute of Allergy and Infectious Diseases.

The research indicates that a prolonged period of latency in HIV-1 infections may be more common than previously recognized. Imagawa said that there is a need for more sensitive antibody tests to confirm HIV-1 infection. He emphasized that this research is being conducted on a group of men selected specifically because they continued to engage in high risk practices; therefore, the study may not be reflective of other gay populations. The investigators continue to study the 133 men.

Many questions remain unanswered, according to Imagawa. One might speculate on the possibility that some people harbor viruses but never seroconvert. Another important question is whether the men were infected more than once with different strains of HIV-1 and whether seroconversion occurred only when they were infected with a virulent strain. Proof of this last hypothesis needs to be obtained in further studies.

The study cannot provide an estimate of the proportion of HIV-1 infections that do not stimulate measurable antibodies. Additional studies, such as PCR, are needed.

Mike Bérn
David Imegawa
Harbor Med., Long Beach
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Jean PR

Haselton

617 - 732 - 3068

Regina - 617 - 732 - 3323

Alan Brownstein
Natl Hemoph. Fdn.

Hemophilia
212-219-8180

"We usually don't see anything beyond 6 months."

"This poten. of silent trans-underscores the import. of having the most effective virus isol. processes in place to safeguard the old. prods. that are used by people w/ hemophilia."

already is very expensive
"The safety imperative is critical because of this poten. window of vulnerability."

"This sends a chill up my spine that we're going to get a whole new wave of seroconv."

virus also may be silent
because of ↑ed CD8 levels

"This implies that the
body recog. the fact
~~there~~ is an infection
and is trying to suppress it."

"It's poss., and if that's
the case, if we can
enhance the supp., maybe
these indivs. can emi
well."

MM: — "High risk indivs.
should not donate bld.
and these men should
practice safe ex."

David
Imagawa

VAIDS I

US copy

old camp, as far as 1984
35 mos. is furthest back
in Stockholm

Steve Wolinsky reported
that one case PCR+ @ 42 mos.

Unique

"At the present time
we cannot generalize. Our
studies were among sero-
who contin. to have high
risk activities."

Spec.

"There is a poss. that
these $\rightarrow$ may get multiplying
infected, and they drift sero-
until they get a virulent strain."

— "It might be poss. these
men get infected & they
overcome the infect."

— Concern for old bands?

"Of course there is a concern,
but you must remember old
bands are ethnic individuals who
are high risk."

"It's a concern, but I personally
don't think it's much of a concern."

— Heteros:

"I cannot answer"
No data avail.

Some people may carry the AIDS virus, ^{silently} for ~~up to~~ over 3 years, ~~without developing~~ ^{without} turning up positive on an AIDS blood test, according to a study ~~research today~~ appearing in today's N Eng J Med.

Researchers at Harbor-UCLA Med. Cntr., in Long Beach, California report that ~~up~~ a quarter of the homosexual men in their study are infected but are not making antibodies against HIV-1, the virus responsible for AIDS. The AIDS bld test measures the presence of such antibodies.

The findings, ^{warned} ~~said~~ Harvard's Dr. William Haseltine in a companion editorial, "raises the sobering possibility that HIV-1 infections may be transmitted by blood & organ donors who are silently infected".

"This sends a chill up my spine," said Alan Brownstein, Pres. of the Natl. Hemophilia Fdn. Because Hemophiliacs have a blood-clotting disorder that ^{renders} ~~makes~~ them highly dependant upon donated blood, and many hemophs. have contracted AIDS ~~since~~ since the epidemic began, Brownstein explained in an interview that

most doctors & hemophiles have assumed ~~that~~ current bld. testing is keeping ~~AID~~ HIV-infected contaminated blood out of the system. "But this ~~problem~~ of silent ~~transmission~~ ^{infects}," said Brownstein, "underscores the import. of having the most effective viral isol. processes in place to safeguard the bld. prods. that are used by people w/ hemophilia."

~~In this latest study)~~
Los Angeles researchers analysed blood samples drawn ~~from~~ ^{from} 133 gay men in 1986 from 133 gay men who tested negative in the standard AIDS test. Using sophisticated ~~new~~ ^{new} laboratory techniques, Dr. David Inagawa and his colleagues from UCLA

Such tests were used in this Univ. of Calif. study, finding the virus, itself, rather than antibodies against the virus. ~~The~~ Blood was drawn at regular intervals, beginning in 1984, from 133 gay men who tested negative in the standard AIDS bld. test. According to study coordinator Dr. David Inagawa, ~~31 of those~~ virus was found in 31 blood samples.

On the last 3 yrs.
~~to date~~, only 4 of these 31
have developed Abs and tested positive
in the standard ~~blood~~ blood test. The
remaining 27 men have low levels
of HIV-1 in their blood, are
well, and continue to test
negative in bld. tests.

~~Imigawa~~

"It is not clear," said
Imigawa in an interview, "why
these men have silent infections."
~~It is They may carry less~~
~~virulent strains of the AIDS virus,~~
~~or have an exceptionally~~
possible explanations, he said,
include:

- the viruses these men carry
are less lethal.
- these men are actually getting
off infection, without relying
on antibodies; and ~~may~~ a
"ray of hope in this epidemic"
said Imigawa, <sup>"because it might be
poss. these men have overcome
their infections."</sup>
- other factors are necessary to
trigger AIDS, and those factors
will eventually appear; a guess
possib. that could mean
"silent infections" are ^{both} contagious
and life-threatening.

Imigawa ~~does~~ downplayed concern about
the safety of the American bld. supply,
noting that blood banks are gay ~~right~~

advocacy organizations actively discourage
homosex. men from donating blood.
"It's a concern," he said, "but I
don't personally think it's much
of a concern."

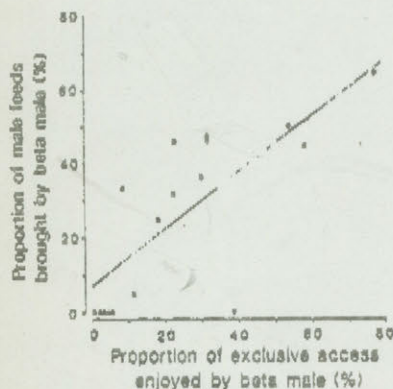
Of greater concern, he said,
~~was~~ is the poss. such indivs.
may practice unsafe sex, passing
the virus onto their partners.

Because this study focused
on high risk individuals, said
Omigawa, "it's imposs. to generalize"
~~about~~ about the ~~rate~~ poss. of
silent HIV infections among
IV drug users, their sexual
partners, and ~~other~~ ~~users~~

polygyny where we recorded chick feeding, the female fed the young at all 30 nests, helped by both males in 14 cases, by the alpha male only in 11 cases (in one of these the beta male died during incubation), by the beta male only in 4 cases (one of these the alpha male died) and in one case the female alone fed the young (mean observation time per nest = 4.35 h, 45).

NA fingerprinting showed that males were more likely to feed the young if they had paternity (Table 2). Two results are that this association does not arise because males can recognize their own offspring. First, in two cases the alpha male fed the young even though he had no paternity and in four cases the beta male did so. (In two cases both helped to feed a single young.) Second, after fledging, broods were often divided among parents with each adult taking sole care of some young for the next 2 weeks until they became independent. There was no tendency for males to pick out their own young. We observed independent fledglings, from 12 broods where both alpha and beta males had helped to feed the nestlings; 11 young were sired by the alpha male, of which 5 were fed by their father and 6 by the beta male, whereas 17 were sired by the beta male, of which 8 were fed by their father and 9 by the alpha male.

Now, then, does the association between chick feeding and paternity come about? A male's access to the female gives a prediction of his chances of paternity (Table 2), and males recently use mating access to determine whether they feed the young. Including data from all polyandrous and polygynous mating systems observed in 1981-88, beta males fed the young in 22 of the 27 cases where they had some exclusive access during the mating period, compared with only 1 out of 5 cases where they were not observed to have access ($P < 0.001$). Furthermore, beta males brought a greater proportion of the feeds where they had had a greater proportion of the exclusive access time during the mating period (Fig. 2). Beta males benefit by copulating with two males because chicks are fed more, and more survive with the help of two males rather than one²². An alpha male, however, would need at least 60-70% exclusive paternity in trio-fed broods for co-operative polyandry



Relationship, for polyandry and polygyny, between the proportion of exclusive access time with the female by the beta male during the mating period, and the proportion of male feeds brought by the beta male to the young. Exclusive access time is the total time that either the alpha or beta male was the only male within 10 m of the female. Data from 1981-88 cases where beta males gained some exclusive access ($y = 5.75 + x$; analysis on log-transformed data: $r = 0.775$, 15df, $P < 0.01$; the slope does not differ significantly from 1, $t = 1.33$, 15df). In polygyny, the provisioning of the young at one nest may be influenced by the presence of other females in the mating system,^{10,22} so cases where other males were laying or had young simultaneously were excluded.

56.9% sired by the alpha male). This suggests that alpha males do not achieve the critical proportion above which it would pay them to share the female, and explains why although females encourage the presence of beta males, alpha males attempt to drive them away²³.

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Incubation period of AIDS in San Francisco

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In a closed population, the distribution of AIDS diagnoses over time is the convolution of the distributions of human immunodeficiency virus (HIV) infections and the incubation period. This has motivated estimates of the infection distribution, assuming known diagnosis and incubation distributions¹, but the usefulness of this method is limited by uncertainty about incubation²⁻⁴. The large amount of information on the distribution of HIV infections in San Francisco's gay community suggests the opposite approach—estimating the incubation distribution, assuming known infection and diagnosis distributions. A non-parametric implementation of this strategy produced an estimate with a median at 9.8 years, increasing hazard rates, and less uncertainty than previous estimates.

Data are available for seroconversions rather than infections, so we estimated the time from seroconversion to AIDS diagnosis⁵. (Seroconversion may occur later than infection⁶.) Figure 1a shows estimated seroconversion rates from three sources, and Fig. 1b shows the overall seroconversion and diagnosis patterns that we used. The dramatic drop in seroconversion by 1984 was paralleled by rectal gonorrhea rates among males at the same time⁷, presumably because of behaviour change. The effect of such change on HIV infections would have been enhanced by the saturation of high-risk subgroups⁸. This further

CDC -
gays/bisex,
men rate
is slack
off - The
trend is
in gay
men
rate of
this is
while rate
is high
IVDA

CDC
Act UP
Mike
212-979-7529
NYC
Joseph
212-566-7150
PR = Sam Friedman
212-566-8148
5802

TABLE 1 Estimates of the incubation period of AIDS

Estimate	Median (yr) (95% CI)	Per cent with AIDS by 9 yr (95% CI)
Present study	9.8 (8.4, ∞)	45 (42, 47)
Penalized 15% for uncertainty in seroconversion pattern	(8.6, ∞)	(36, 54)
Parametric analysis of US transfusion data ²	7.3 (4.6, ∞)	Not given
Parametric analysis of data from 84 men in the San Francisco City Clinic Cohort ²	7.8 (4.2, 15.0)*	65 (not given)
Nonparametric analysis of data from 181 men in the San Francisco City Clinic Cohort ¹⁶	Not given	42 (30, 54)†

* Estimated mean and 90% confidence interval (CI).

† Confidence interval does not reflect uncertainty resulting from imputing exact seroconversion times from interval-censored data.

corroborates the drop shown by all three sources in Fig. 1a. Mathematical models of epidemic spread in a susceptible population⁸ corroborate the early rapid rise.

Treating the distributions in Fig. 1b as known allows estimation of the incubation distribution. In particular, we wish to estimate parameters h_k , the hazard rates for developing AIDS k months after seroconversion. With 10 years worth of data, h_k can be estimated for $k=0$ to 120. Let $d_{j,k}$ be the number of persons who seroconverted in month j and were diagnosed in month k . If all the $d_{j,k}$ were known, then the maximum likelihood estimate of h_k would be

$$\hat{h}_k = e_k / r_k \quad (1)$$

where $e_k = \sum_{j=0}^{120-k} d_{j,k}$ is the number of persons diagnosed exactly k months after seroconversion, and r_k is the number of

persons still free of AIDS ($k-1$) months after seroconversion. Although the $d_{j,k}$ are unknown, we can use the EM algorithm⁹ to estimate the h_k . The algorithm begins with arbitrary initial values of the h_k and calculates the expected values of the $d_{j,k}$ conditional on the initial h_k and the known seroconversion and diagnosis patterns. The expected values are used in place of the unknown $d_{j,k}$ in equation (1) to yield refined estimates of the h_k . The refined h_k s lead to new expected values, which yield new estimated h_k s, and so on. The algorithm continues until the estimates converge.

Because it is biologically reasonable that the hazard changes smoothly over time, we used a modification of equation (1):

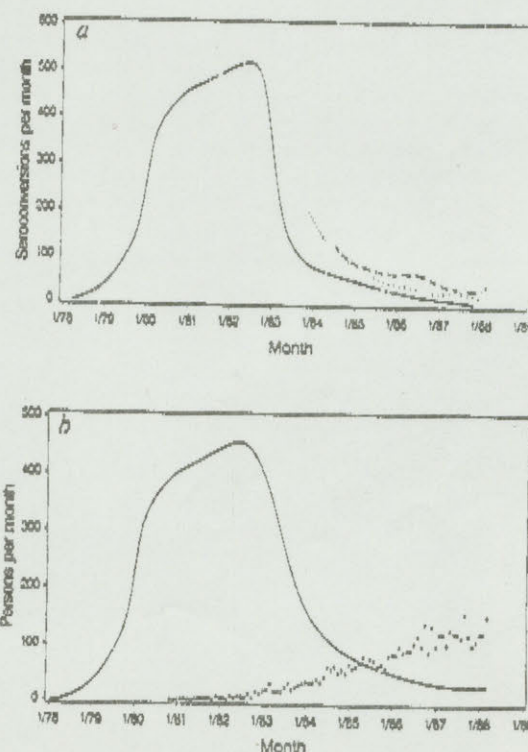
$$\hat{h}_k = \frac{\sum_{i=k-6}^{k+6} e_i}{\sum_{i=k-6}^{k+6} r_i} \quad (2)$$

This is a local likelihood estimate¹⁰ with a span of 13. (This span was the smallest that produced acceptably smooth estimates.) In addition to ensuring reasonable smoothness, this modification also reduced the estimate's variability and led to faster convergence, while still being non-parametric. Figure 2 shows the estimates that resulted from applying this method to the distributions in Fig. 1b. The estimated probabilities of developing AIDS during the first to tenth years following seroconversion were 0.002, 0.007, 0.022, 0.043, 0.061, 0.073, 0.082, 0.081, 0.074 and 0.067, respectively. The estimated distribution is an average for all gay men in San Francisco. It may well differ for subgroups of this population, and it may change as prophylaxis of seropositives becomes more widespread before diagnosis.

The confidence intervals in Fig. 2 do not reflect the uncertainty that results from estimation of the seroconversion pattern. Multiplying the seroconversion curve in Fig. 1b by a factor (or dividing the diagnosis curve by a factor) would divide the curve in Fig. 2a by the same factor. As the estimated number of seroconversions may be too high or too low by 10% (ref. 11), the curve in Fig. 2a could plausibly be multiplied by 0.9 or 1.1.

FIG. 1 Data used for estimating the distribution of times from HIV seroconversion to AIDS among gay men in San Francisco. a, HIV seroconversions estimated from the hepatitis B vaccine trial (solid line), the San Francisco Men's Health Study¹¹ (long dashes), and the San Francisco General Hospital Study¹² (short dashes). b, Overall estimates of HIV seroconversions (solid line) and AIDS diagnoses (dots).

METHODS. a, For the hepatitis B vaccine trial, a smooth curve was fitted to match estimated yearly totals¹⁶ and scaled to total 20,260 seroconversions through September 1984. The total 20,260 is an estimated 8,757 seropositive but AIDS-free men from a random sample of part of San Francisco¹¹, divided by the proportion of AIDS cases from October 1984 until July 1988 who came from the sampled area (1,708 of 3,767, or 45%; this proportion was fairly stable over the period) plus 800 AIDS cases diagnosed up to September 1984. For the other cohorts, Turnbull's EM algorithm¹³ was applied to interval-censored seroconversion data, with local-likelihood smoothing¹⁰ in the M-step. The curves were scaled by the same factor as the vaccine trial estimate. b, Monthly diagnosis totals up to March 1988 are from surveillance data as of July 1988, adjusted for several sources of error. Based on data reported of November 1987 until July 1988, the proportionate increase in monthly totals arising from reports more than four months after diagnosis was modelled as a quadratic function of difference between report and diagnosis month, plus a quadratic function of calendar month of report (to allow for diminishing influence of the change in case definition⁵). This yielded corrections ranging from +14% for diagnoses in March 1988 to less than +1% for March 1987 and before. The San Francisco Department of Public Health has estimated under-reporting as ~2% before 1986 and ~5% more recently, and has also estimated that about 4% of San Francisco residents with AIDS are diagnosed elsewhere (unpublished data). Follow-up of the San Francisco General Hospital Cohort¹², in which 3 of 69 AIDS cases (4%) were diagnosed after moving from the city, suggested a correction of +4% for persons included in the seroconversion totals who were not residents when diagnosed. The seroconversion curve is a compromise among the curves from (a) that retains the overall shape from the hepatitis B vaccine trial and agrees closely with the other curves, while remaining smooth and totalling 20,260 seroconversions up to September 1984.



New cases of infection

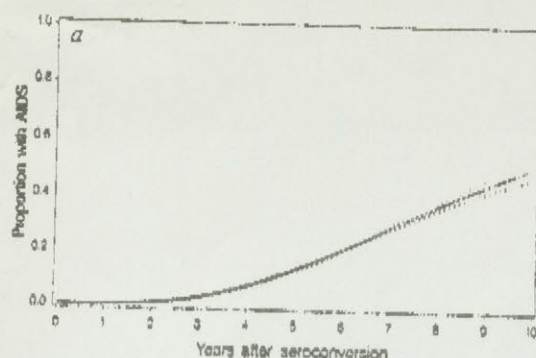
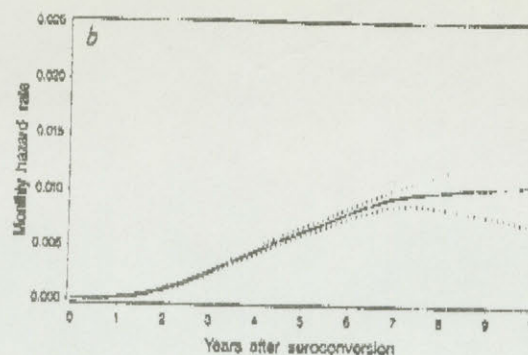


FIG. 2 Estimated distribution of time from HIV seroconversion to diagnosis of AIDS for gay men in San Francisco, 1978-1988. a, Cumulative distribution function (solid line), with approximate 95% confidence intervals (dashed lines).

b, Hazard function (solid line), with approximate 95% confidence intervals (dashed lines).

METHODS. Confidence intervals were determined by a bootstrap method⁴⁴.



The estimated incubation distribution was used to generate simulated diagnosis data assuming the seroconversion pattern shown in Fig. 1b. Estimates were made on 200 such sets of simulated data to assess the variability resulting from random variation in how well the diagnosis pattern reflects the incubation distribution. At each month, 95% of the estimates fell between the dashed lines.

Shifting the seroconversion curve relative to the diagnosis curve would expand or contract the initial period of virtually no progression to AIDS. Applying the estimation method to a seroconversion pattern identical to the one in Fig. 1b, but with 10 seroconversions per month from December 1976 to May 1978, would change the estimate in Fig. 2a by less than 0.006 at all time points, so the possibility of an unobserved long, slow start to the epidemic does not invalidate the estimate.

Figure 3 compares fitted monthly AIDS diagnosis totals with adjusted surveillance data. The fitted curve matches the data well. The projected curves reflect the range of distributions shown in Fig. 2b. They are not influenced by uncertainty in the magnitude of the seroconversion curve, because any error would result in an opposite error in the incubation estimate, with no net influence on projected totals. The projections show the sharp drop in seroconversions in Fig. 1b manifesting itself as a much more gradual drop in AIDS diagnoses 6 to 8 years after the drop in seroconversions. For each of the three projections, the time between the peaks in seroconversions and projected diag-

noses is approximately equal to the time of the peak in the density of the incubation. The gradualness of the drop in projected diagnoses is due to the large variability in incubation times⁶.

Four factors may bias the estimate of Fig. 2 upwards when compared with other studies: use of the more inclusive 1987 definition of AIDS⁵, use of seroconversion rather than infection times, inclusion of cases who seroconverted outside San Francisco in diagnosis totals but not in seroconversion totals, and possible downward bias in the magnitude of the seroconversion curve resulting from over representation of seropositives from the sampled area¹¹ among diagnosed AIDS cases. Despite these factors, Fig. 2 shows slower progression to AIDS than previous estimates^{2,3} (Table 1). The large uncertainty in previous estimates, however, makes them quite compatible with Fig. 2. In addition, analyses based on blood-transfusion data deal with a very different population and only estimate the incubation period that is conditional on AIDS developing.

The incubation period of AIDS is difficult to study. Prospectively followed incident seroconverters are rare, so estimates based on them are highly uncertain², and data from transfusion-associated AIDS cases are inherently limited³. The method used here is an alternative approach. It is possible because of the availability of information on seroconversions among gay men in San Francisco and because the drop in seroconversions makes the pattern of diagnoses quite informative about the incubation. Its dependence on an accurate estimate of seroconversions is a drawback, but it does not rely on parametric assumptions, and Table 1 shows that the estimate is more precise than others, even allowing for uncertainty in estimating seroconversions.

4 factors affect data reliability

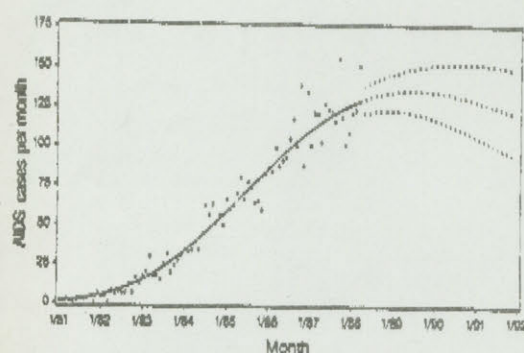


FIG. 3 Fitted (solid line) and projected (dashed lines) AIDS diagnoses among gay men in San Francisco. Projections are based on incubation distributions corresponding to the estimate in Fig. 2b, with the hazard remaining constant beyond year 10 (middle projection), and corresponding to the upper and lower confidence limits in Fig. 2b, with the hazard extrapolated linearly beyond year 10 (upper and lower projections). The adjusted surveillance totals from Fig. 1b (dots) are shown for comparison. Note that the vertical scale represents corrected rather than raw surveillance totals.

METHODS. Given v_j seroconversions in month j and probability p_k of developing AIDS k months after seroconversion, the expected number of diagnoses in month i is $\sum_{j=0}^{i-1} v_j p_{i-j}$. Fitted and projected monthly diagnosis totals were calculated by this formula with the v_j s from the seroconversion curve in Fig. 1b (with constant extrapolation through 1991) and the p_k s from the incubation distributions described above.

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NEWS

FROM
SFGH/UC-SAN FRANCISCO

UCSF

San Francisco General Hospital Medical Center, 1001 Potrero Avenue, San Francisco, CA 94110

Carol Fox, News Director

Source: Sanna Craig (415) 476-2655 or 821-5310

EMBARGOED FOR RELEASE

WEDNESDAY, MARCH 15, 1989, 6 PM EST

UC-SAN FRANCISCO RESEARCHERS ESTIMATE TIME FROM AIDS INFECTION
TO DIAGNOSIS IN GAY MEN IS 2-3 YEARS MORE THAN PREVIOUSLY THOUGHT,
BUT PREDICT DECLINE IN NEW CASES

By 9.8 years after infection with the AIDS virus, one half of the people infected would have developed the clinical disease of AIDS, according to a new study of San Francisco male homosexuals by UC-San Francisco researchers at San Francisco General Hospital.

The new timeframe exceeds by two to three years previous estimates of a seven-to-eight-year average time from infection to AIDS diagnosis.

This same study also predicts that the number of new AIDS cases is already leveling off and will begin dropping by as much as 15% by the end of 1991, and will continue to decline significantly in San Francisco gay men.

The estimate, the most precise to date, is being reported in the March 16, 1989 issue of the British scientific journal Nature.

Using stored blood specimens, some dating as far back as 1978, researchers Peter Bacchetti, PhD, and Andrew Moss, PhD, compared patterns of antibody development that linked human immunodeficiency virus (HIV) infection to patterns of new AIDS cases diagnosed in San Francisco. The current study is the first to link the observed incidence rates in gay men with the drop in new infections believed to have happened between 1982-84.

The UCSF researchers predict new AIDS cases among San Francisco gay men will peak this year, followed by a gradual decline in incidence. In fact, the peak may have already occurred, says Moss, UCSF associate adjunct professor of epidemiology and international health and director of the AIDS epidemiology group at SFGH.

The decline is due to a dramatic drop of more than 80 percent in new infections in 1982-84, says Bacchetti, assistant adjunct professor of epidemiology and international health at SFGH. During that time, the number

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scSFGH/AIDSIncub

of new HIV positive findings dropped to less than 50 a month from an estimated monthly high of 450 for the peak period in mid-1982. This finding was corroborated by a similar downward turn in rectal gonorrhea reported in the San Francisco gay male community at that time. The reason, says Bacchetti, is that there appears to have been a major change in gay men's sexual behavior.

Blood specimens were used from three San Francisco based studies of homosexual men: a hepatitis B vaccine trial begun in 1978, and homosexual male volunteers who agreed to be followed for incidence of HIV infection and development of AIDS from the San Francisco General Hospital Cohort Study, begun in 1983, and the San Francisco Men's Health Study, begun in 1984. The researchers also used data on new AIDS diagnoses from the San Francisco Department of Public Health.

"Use of antiviral treatment with AZT and improved treatment of pneumocystis carinii pneumonia, may have reduced AIDS incidence below that predicted by our model," Moss notes, "making the drop in AIDS cases even larger."

A flattening in AIDS incidence rates in homosexual males has also been noted in New York and Los Angeles, the researchers point out.

The downswing in new AIDS cases may not affect the total number of AIDS patients in San Francisco because people are surviving longer and require medical care for related diseases, says Bacchetti.

Bacchetti and Moss note that the rate of AIDS diagnosis is rising rapidly in intravenous drug users in San Francisco. One implication of the current research, they say, is to focus more attention on this second group at risk for AIDS.

###

AIDS INCIDENCE IN SAN FRANCISCO
FIRST QUARTER 1983

1. INTRODUCTION

This report presents summary analyses of Acquired Immune Deficiency Syndrome (AIDS) incidence in San Francisco as of March 31, 1983.

The incidence data on which these analyses are based were collected in metropolitan San Francisco by the Bureau of Communicable Disease Control of the San Francisco Department of Public Health, for reporting to the Centers for Disease Control. AIDS as reported includes Kaposi's Sarcoma and some lymphomas limited to the brain, Pneumocystis Carinii pneumonia, and certain opportunistic infections.¹ Surveillance of San Francisco data up through March 31st was estimated to have been 85-90% complete at the time the current analyses were carried out. Thus incidence estimates given here can be assumed to be somewhat below the true incidence of the disease.

AIDS incidence data is presented here using two different denominators: never-married men aged 15 and over, and all men. Incidence rates among all men are also given for the two ten-year age groups in which ninety percent of San Francisco AIDS cases are concentrated, ages 25-34 and 35-44. Thus all rates given in this report and in the Appendix tables are presented in a fourfold fashion: (a) among all never-married men; (b) among all men; (c) among all men aged 25-34; (d) among all men aged 35-44.

AIDS in San Francisco is still primarily a disease of gay men. The 146 cases analysed here are all among men believed to be either gay or bisexual.² In metropolitan San Francisco only 3 other adult cases have been reported as of March 31, 1983.³ Incidence rates given for never-married men aged 15 and above may be regarded as approximations to the incidence rates among gay men in areas which are largely gay. These rates are progressively less accurate estimators of the rates among gay men in progressively more heterosexual neighborhoods.

AIDS incidence data are given here first for the City as a whole, then for fifteen subareas of San Francisco. These subareas were obtained by dividing the five health service delivery areas of the San Francisco Department of Public Health into between

-
1. The squamous cell tumors of the mouth and tongue and Burkitts-like lymphomas which have been observed in gay men in San Francisco are not included.
 2. By physician report. CDC surveillance interviewing is not yet complete on all reported cases.
 3. One Haitian male; one transient male about whom nothing is known; one 85 year-old male believed to be a transfusion recipient.

Source: Dritz

two and four subareas each--subareas which reflect the demography of San Francisco (Figure 1).

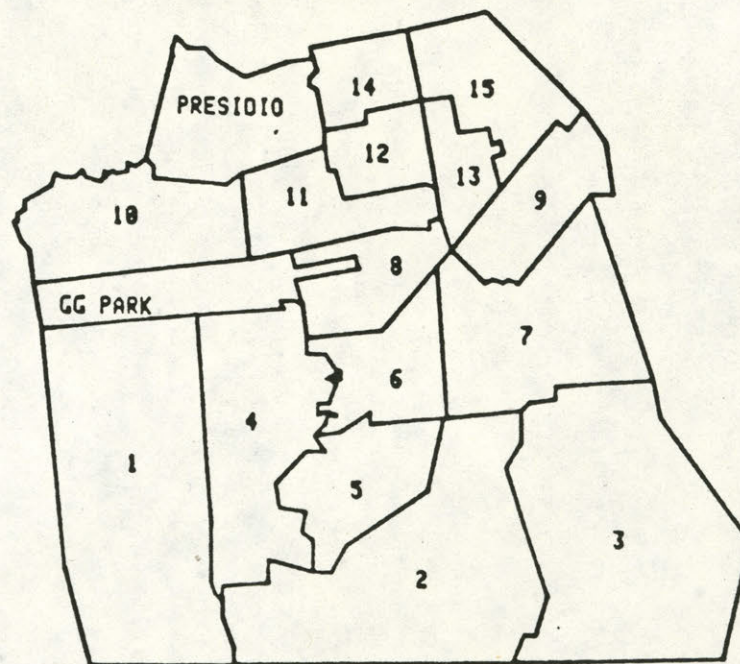


FIGURE 1.
San Francisco Districts

AIDS incidence data are given in the form of cumulative totals of cases, and also as annualized rates. These rates are given for the City as a whole, by quarter, from the beginning of the epidemic through the first quarter of 1983.

Finally, since the fifteen subareas are too small to calculate annualized incidence rates, areas 6 and 8 have been lumped ("Area X") and subareas 9, 12, and 13 have been lumped ("Area Y") and annualized rates are given for Area X, Area Y, and the rest of San Francisco.⁴

Appendix tables 1-4 give cumulative incidence data for the fifteen subareas of San Francisco. Appendix tables 5-8 present annualized incidence rates for the City as a whole. Appendix tables 9-12 present annualized rates for Area X, Area Y, and the rest of San Francisco.

4. Areas X and Y are based on subareas derived from the Health Department's service delivery areas and are similar to, but not quite comparable with, the Areas A and B used in a previous report.

2. CUMULATIVE AIDS INCIDENCE IN SAN FRANCISCO

Figure 2a shows AIDS cases reported by quarter from the beginning of the epidemic, in the last quarter of 1980, until the present. (Each quarter is labeled by the middle month.) Of the total of 146 AIDS cases, 46 were reported in the first quarter of 1983, as compared with 23 in the last quarter of 1982. Thus the incidence of the disease continues to increase in an apparently geometric fashion.

Figures 2b and 2c give the same data for Kaposi's sarcoma (KS), and for all other presenting diagnoses respectively. (The category "All Other Diagnoses" includes Pneumocystis Carinii pneumonia, other opportunistic infections, and cases with both KS and opportunistic infections.) While the number of cases presenting with KS increased only slightly in the first quarter of 1983, there was a rapid jump in the number of AIDS cases presenting with other diagnoses. Of the 46 new cases in the quarter, 17 presented with KS only and 29 with other diagnoses.

Comparing the first quarter of 1983 with previous quarters, it is clear that casefinding differences are in part responsible for the large increase in non-KS cases. AIDS infections became reportable in the first quarter of 1983,⁵ while KS, as a neoplasm, has always been reportable in the Bay Area. In retrospect, there was a major increase in the number of KS cases reported in the third quarter of 1982, reflecting the increased publicity given to the disease last summer, and probably also reflecting a centering of academic interest in KS rather than opportunistic infections, following the announcement of the National Cancer Institute's research awards. After last summer's rapid increase, KS case-finding remained relatively stable for the next six months. It appears likely that comparable attention was given to opportunistic infections only in the first quarter of 1983, resulting in the recent jump in case-finding. At least part of the jump probably represents poor casefinding and reporting in the preceding quarters. However, it should also be pointed out that with the jump in the number of opportunistic infections reported, the one-to two ratio of KS to non-KS cases in San Francisco has now begun to approximate the relationship that prevails among homosexual men who are AIDS cases in the U.S. as a whole. Thus this pattern may now persist in San Francisco.

Why?! It should also be noted that cases of Pneumocystis and other opportunistic infections are drawn from a different demographic subpopulation than cases of Kaposi's sarcoma. Men presenting with infections are about five years older, on average, and tend to live in different parts of San Francisco (Area Y and the rest of the City, rather than Area X). Thus the recent change in the distribution of cases may represent

☆ ← 5. Strictly speaking the announcement of reportability was made. AIDS did not become legally reportable until the second quarter of 1983.

FIGURE 2a

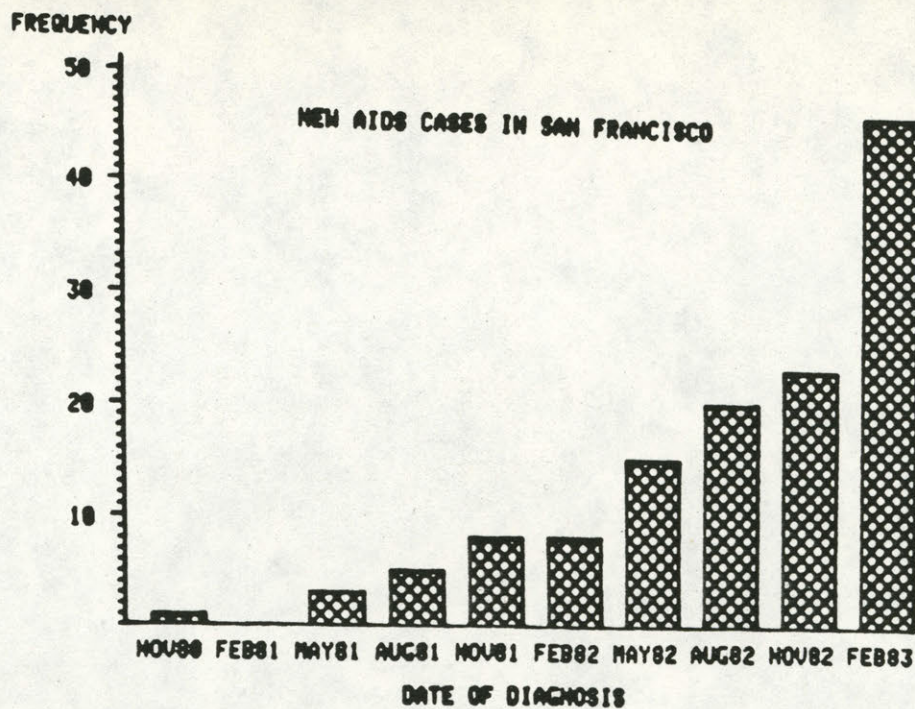


FIGURE 2b

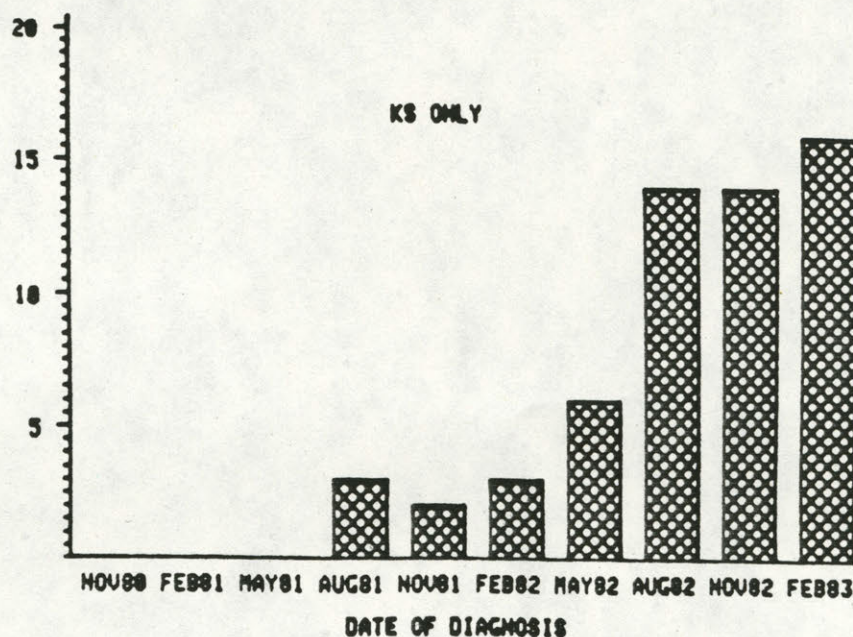


FIGURE 2c

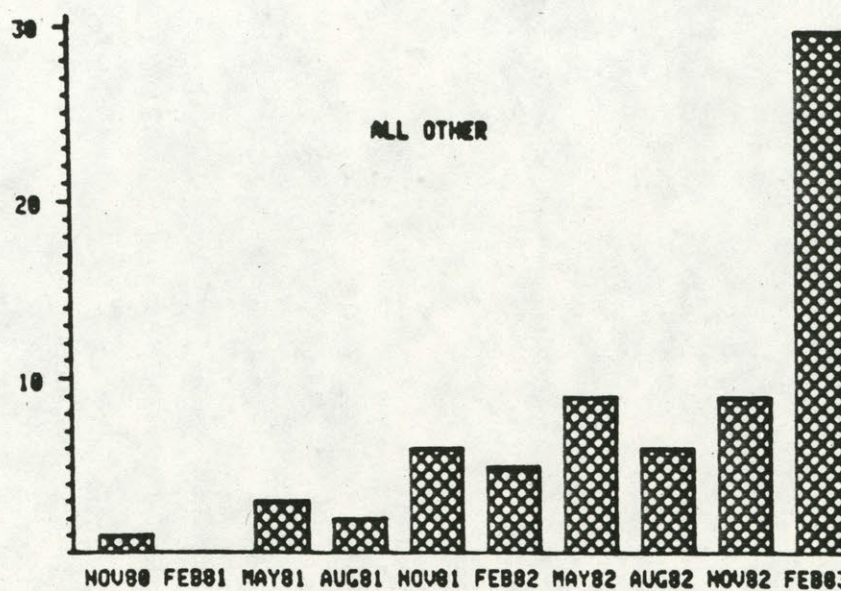


Figure 2.
New AIDS Cases in San Francisco: (a) All Cases; (b) Kaposi's Sarcoma Only; (c) All Other Diagnoses

a movement of the epidemic into different subgroups. Since infections among New York AIDS cases are commonly associated with IV drug use, some of the recent cases in San Francisco may be among gay men who are IV drug users. If so, it is to be expected that cases will also present among heterosexual drug users, with the possibility of a second epidemic.

Finally, Figure 3 shows new AIDS cases by month up through March 1983, confirming the geometric increase in incidence. Table 1 gives the breakdown of cases by admitting institution. Sixty-eight of one hundred and forty-six cases, or about half the total, were admitted to UCSF or San Francisco General hospitals.⁶

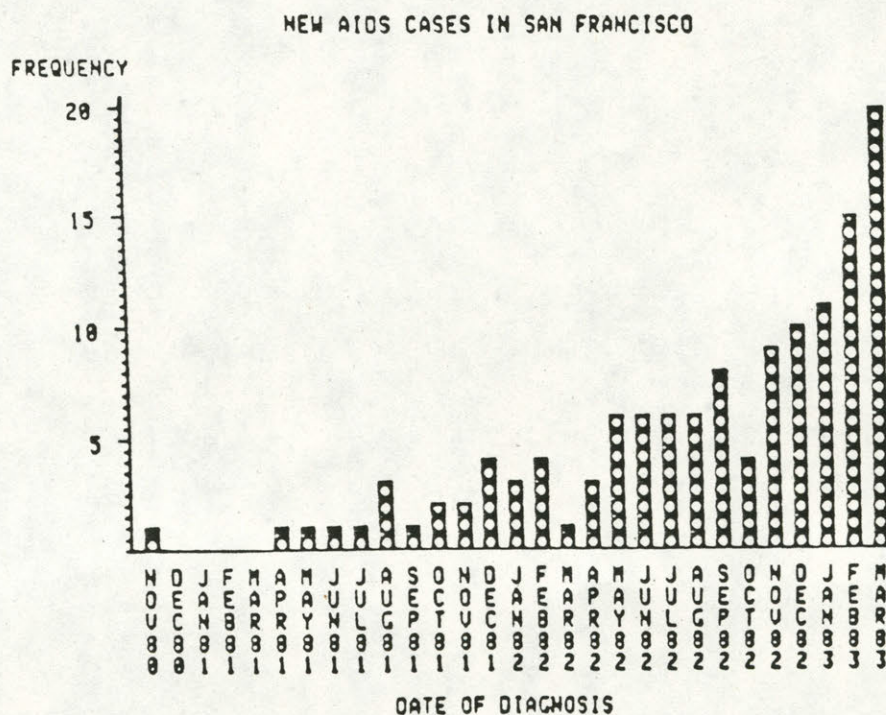


FIGURE 3. New AIDS Cases by Month of Diagnosis

3. CUMULATIVE AIDS INCIDENCE FOR FIFTEEN SUBAREAS OF SAN FRANCISCO

Table 2. gives numbers of cases reported among men living in the fifteen subareas shown in Figure 1. Numbers of cases are given for the period 1980-1982, and also for the first quarter of 1983. Cases have now been reported in all fifteen subareas, with the exception of the Marina (Area 14) and Downtown-North Beach-Russian Hill (Area 15). Seven cases have now been reported in Pacific Heights (Area 12) three

6. Because only AIDS cases as defined by the CDC are included here, this table under-represents the contribution of some hospitals in the current epidemic.

TABLE 1

SAN FRANCISCO AIDS CASES
BY ADMITTING INSTITUTION

UCSF/SF GEN	68
STANFORD	2
VA SF	5
ST. FRANCIS	7
PMC	11
KAISER SF	21
RK DAVIES	12
CHILDREN'S	5
MARSHALL HALE	2
LETTERMAN	1
MARY'S HELP	1
KAISER S. SF	1
OTHER	5
UNKNOWN	5
	146

TABLE 2

AIDS CASES IN SAN FRANCISCO BY AREA

<u>AREA</u>	<u>1980-82</u>	<u>AIDS CASES 1ST QUARTER</u>	<u>TOTAL</u>
1	-	3	3
2	3	1	4
3	-	1	1
4	3	1	4
5	5	2	7
6	40	8	48
7	6	2	8
8	24	13	37
9	4	2	6
10	-	1	1
11	1	2	3
12	3	4	7
13	10	6	16
14	-	-	0
15	-	-	0
UKN.	1	-	1
TOTAL	100	46	146

in the Sunset (Area 1), and four in the Outer Mission (Area 2). These numbers confirm the fact that the AIDS epidemic in San Francisco is not localized in the central gay neighborhoods north and south of Castro and Market. In fact, of the 46 new cases reported in the first quarter of 1983, only 8 were in Area 6 (Noe, Castro, Eureka), a much lower proportion than in the period 1980-82. 13 were reported in Area 8 (Duboce Triangle, Haight, Western Addition), about the same proportion as in 1980-82.

Appendix Tables 1-4 give cumulative incidence rates for the fifteen areas. The incidence data are presented in the fourfold fashion described in section 1 above. Appendix Table 1 shows that in Areas 6 and 8, cumulative incidence rates among never-married men aged 15 and over were 356 per 100,000 and 256 per 100,000 respectively as of March 31, 1983. In Area 5 (Glen Park), and in Areas 9, 12, and 13, (South of Market, Polk Street, Pacific Heights), the rates were of the order of 120 per 100,000. In all other areas the cumulative incidence rates were appreciably lower.

When calculated among all men, incidence rates in the 25-34 year old group were 257 and 127 per 100,000 respectively for Areas 6 and 8, (Appendix Table 3). They were 407 and 609 per 100,000 respectively in the age group 35-44 (Appendix Table 4). Since these rates are calculated among all men, they must underestimate the rates among gay men; thus the rates among gay men aged 25-44 in areas 6 and 8 must be somewhat larger. These rates confirm unpublished data from the Centers for Disease Control which suggests that in some cohorts of gay men in San Francisco, AIDS incidence rates in the thirty and forty-year old groups are now of the order of 1-2 percent.⁷ Thus although the AIDS epidemic appears to be increasingly less concentrated in the central gay neighborhoods, incidence rates among some groups of men in these areas are already quite high.⁸

7. D. Francis, personal communication.

8. For comparison, incidence rates among never married men aged 15 and over in "Area A" and "Area B: of our previous incidence report (Lancet, April 23, 1983) have risen from 287 to 379 per 100,000 and from 111 to 170 per 100,000 respectively.

4. ANNUALIZED AIDS INCIDENCE RATES FOR SAN FRANCISCO AND THREE MAJOR SUBDIVISIONS OF THE CITY

Appendix Tables 5-8 present incidence data in the form of annualized rates, by quarter, from the beginning of the epidemic up through the first quarter of 1983. In the City as a whole, the annual incidence of AIDS reached 138 per 100,000 among never-married men aged 15 and over by the first quarter of 1983--about six times the rate in the first quarter of 1982. (Appendix Table 5).

Among all men aged 25-34 the rate was 104 per 100,000, up from 69 per 100,000 in the previous quarter and about ten times the rate in the first quarter of 1982. Among all men aged 35-44 the rate was 191 per 100,000, up from 82 in the previous quarter and about four times the rate a year earlier. The recent doubling in rates among men in the older group reflects the greatly increased incidence of opportunistic infections rather than Kaposi's sarcoma in the current quarter. AIDS incidence rates in San Francisco, among all men aged 25-44, are now running at about twice the rate for cancers of all sites combined.

Appendix Tables 9-12 present annualized incidence data for three separate subregions of the City: Area X, combining subareas 6 and 8, includes most of the central gay neighborhoods north and south of Castro and Market. Area Y, combining subareas 9, 12, and 13, includes the South of Market, Polk Street and Pacific Heights, three areas which have similar incidence rates.

Appendix Table 9 shows annualized incidence rates among never-married men aged 15 and over in the three areas. In Area X the rate was 301 per 100,000, up from 258 in the previous quarter. In Area Y the rate was 183 per 100,000, up from 46 in the previous quarter. In the rest of San Francisco the rate was 66, up from 10 in the previous quarter. While the annualized rate remains highest in Area X, the rates are climbing much faster in Area Y and the rest of San Francisco.⁸

Among all men aged 25-34, the annual incidence rate actually fell in Area X in the first quarter of 1983 (Appendix Table 11), while rising steeply in Area Y and the rest of San Francisco. Among all men aged 35-44 the rate rose in all three areas, most steeply in Area Y. Thus the current quarter has seen a tendency for incidence rates to even out between Area X and the rest of San Francisco. This phenomenon probably represents the aftereffect of a very high level of case-finding in the central gay neighborhoods in Area X in the last half of 1982, resulting from the high degree of public awareness in these neighborhoods. It seems likely that a similar level of alarm may have been achieved only recently in other parts of San Francisco.

4. CONCLUSION

Incidence data for the first quarter of 1983 suggests that the geometric rise in the incidence of AIDS in San Francisco continued unchecked through March 1983, as it did in the nation as a whole. San Francisco continues to have about ten percent of all AIDS cases nationwide.

The large rise in the number of opportunistic infection cases reported, as opposed to Kaposi's sarcoma cases, may be in part the result of case finding differences and reportability. Alternatively it is possible that the distribution of AIDS is shifting, as cases are reported among different subpopulations, and that the ratio of KS cases to infection cases in San Francisco is coming to match that in New York and the country as a whole.

The proportion of cases reported in some of the central gay neighborhoods of San Francisco appears to be declining. Among men aged 25-34 there was an absolute decline in the incidence rate in Areas 6 and 8 (Noe-Eureka-Castro-Haight-Duboce Triangle-Western Addition). This decline may be an after-effect of the early high level of awareness in this area, and the consequent detection of a large number of cases among its residents in 1982.

While the epidemic appears to be appearing in new population subgroups, it should be noted that among all men aged 25-34 and 35-44 in the central gay neighborhoods of San Francisco, annual incidence rates of AIDS are now running at approximately 200 per 100,000 and 400 per 100,000 respectively. Among gay men the rates must be somewhat higher.

In San Francisco as a whole, annual incidence rates among men ages 25-43 and 35-44 were 104 per 100,000 and 191 per 100,000 respectively in the first quarter of 1983. These rates are about twice as high as the incidence rates for cancer of all sites in the same age groups. They are about half as high as the reported rates for Hepatitis B in San Francisco in 1982 in the same age groups.

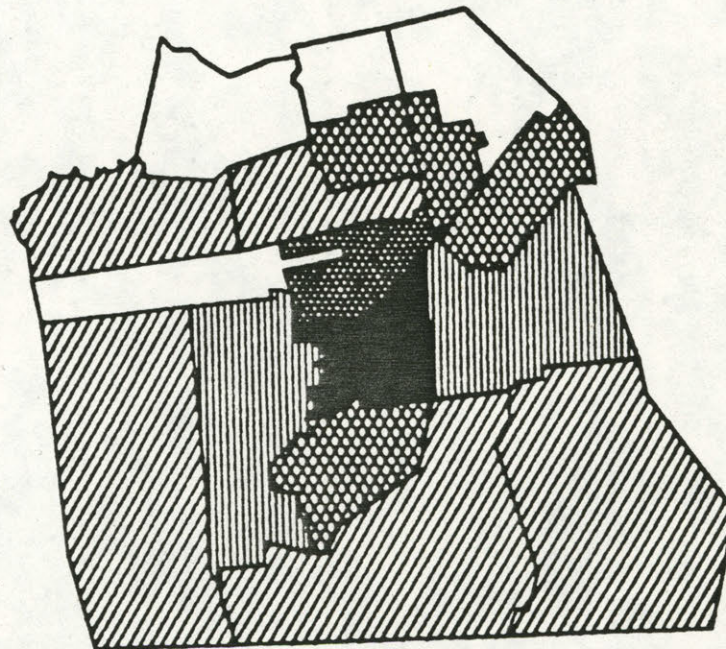
AIDS in San Francisco remains primarily a disease of gay men. However, the changing distribution of cases suggests that San Francisco may soon be seeing new cases among heterosexual drug users. Thus public awareness campaigns about AIDS should be directed at this group as well as at gay men.

There is no evidence of any spread of AIDS by casual transmission in the incidence data as reported so far.

APPENDIX TABLES

- Table 1. Cumulative incidence per 100,000 single men
- Table 2. Cumulative incidence per 100,000 men
- Table 3. Cumulative incidence per 100,000 men aged 25-34
- Table 4. Cumulative incidence per 100,000 men aged 35-44
- Table 5. Annualized incidence per 100,000 single men, by quarter
- Table 6. Annualized incidence per 100,000 men, by quarter
- Table 7. Annualized incidence per 100,000 men aged 25-34, by quarter
- Table 8. Annualized incidence per 100,000 men aged 35-44, by quarter
- Table 9. Annualized incidence per 100,000 single men, by quarter: three areas
- Table 10. Annualized incidence per 100,000 men, by quarter: three areas
- Table 11. Annualized incidence per 100,000 men aged 25-34, by quarter, three areas
- Table 12. Annualized incidence per 100,000 men age 35-44, by quarter, three areas

CUMULATIVE INCIDENCE PER 100,000 SINGLE MEN AGED 15 & UP



LEGEND: INCIDENCE

0
101-200

1-50
201-300

51-100
>300

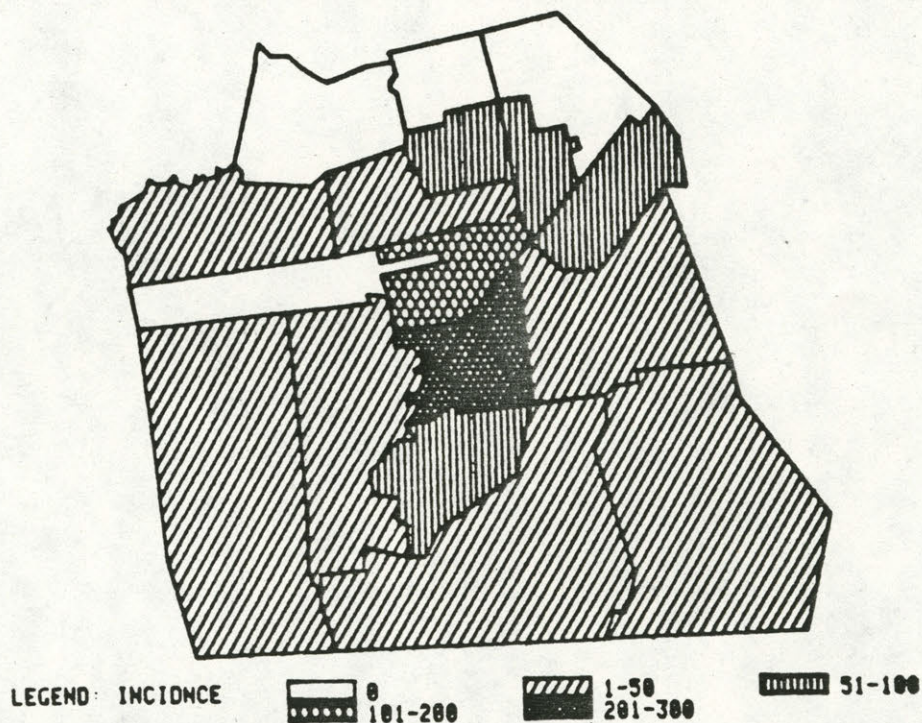
CUMULATIVE INCIDENCE PER 100,000 SINGLE MEN AGED 15 & UP

AREA	CASES	SINGLMEN	INCIDENCE
1	1	10290	29
2	4	15300	26
3	1	3030	33
4	4	7720	52
5	7	5420	129
6	48	13480	356
7	8	8170	98
8	37	14470	256
9	6	4910	122
10	1	8520	12
11	3	8620	35
12	7	6120	114
13	16	15160	106
14	0	2770	0
15	0	8550	0

TABLE 1

Cumulative Incidence per 100,000, Single Men

CUMULATIVE INCIDENCE PER 100,000 MEN AGED 15 & UP



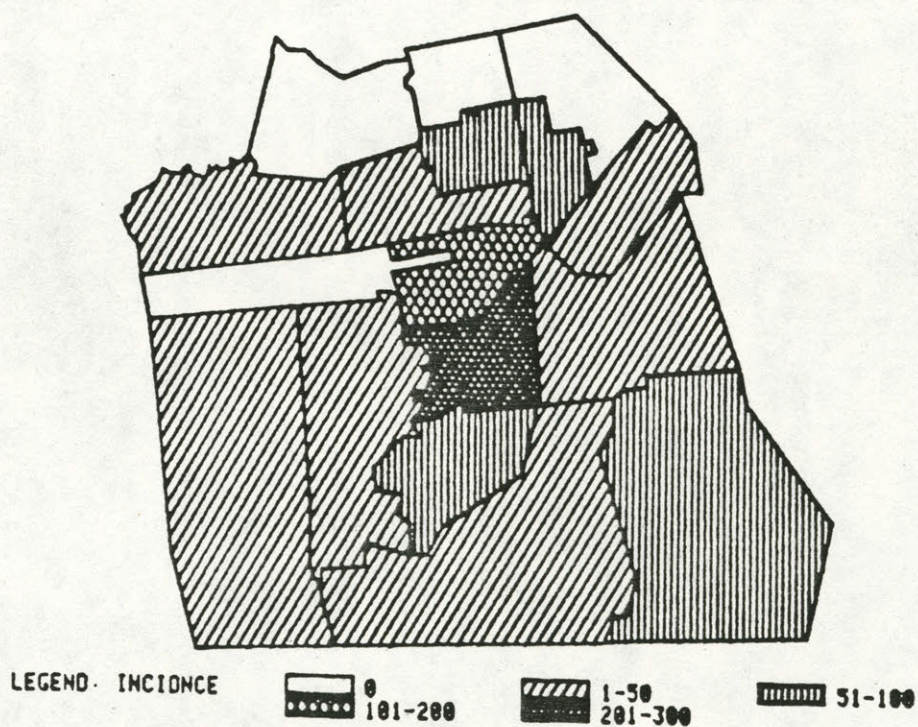
CUMULATIVE INCIDENCE PER 100,000 MEN AGED 15 & UP

AREA	CASES	MEN15UP	INCIDENCE
1	1	.	.
2	3	29470	10
3	4	41440	10
4	1	7940	13
5	4	20140	20
6	7	12620	55
7	48	22860	210
8	8	17590	45
9	37	22660	163
10	6	9230	65
11	1	21500	5
12	3	17370	17
13	7	12460	56
14	16	26580	60
15	0	6980	0
16	0	19490	0

TABLE 2.

Cumulative Incidence per 100,000 Men

CUMULATIVE INCIDENCE PER 100,000 MEN AGED 25 TO 34



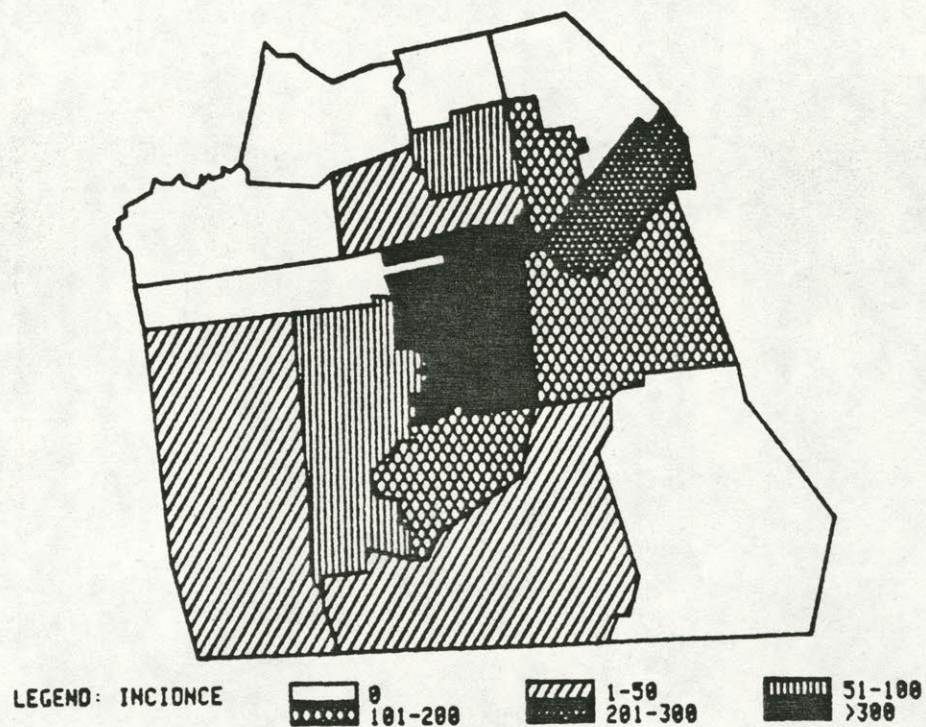
CUMULATIVE INCIDENCE PER 100,000 MEN AGED 25 TO 34

AREA	CASES	MEN25_34	INCIDENCE
	0	.	.
1	2	6340	32
2	2	8950	22
3	1	1330	75
4	1	5030	20
5	2	3610	55
6	24	9150	257
7	2	4840	41
8	12	9630	127
9	1	2480	40
10	1	5970	17
11	1	5000	20
12	3	4170	72
13	5	7140	70
14	0	2010	0
15	0	4430	0

TABLE 3.

Cumulative Incidence per 100,000
Men Aged 25-34

CUMULATIVE INCIDENCE PER 100,000 MEN AGED 35 TO 44



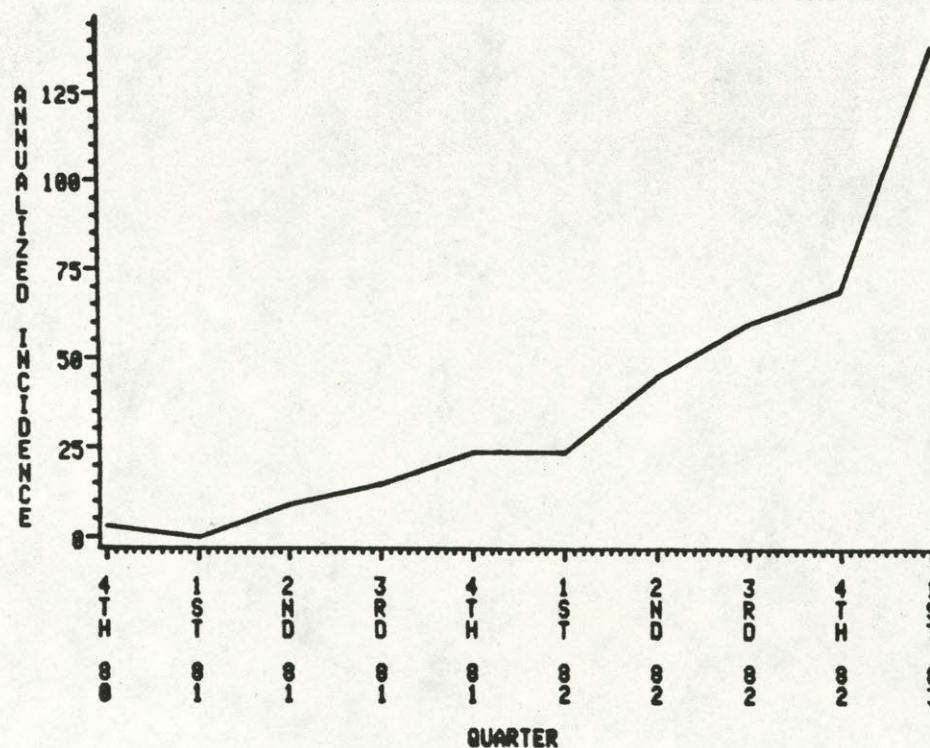
CUMULATIVE INCIDENCE PER 100,000 MEN AGED 35 TO 44

AREA	CASES	MEN35_44	INCIDENCE
	0		
1	1	3330	30
2	2	5750	35
3	0	850	0
4	2	2760	72
5	4	2240	179
6	19	4550	407
7	5	2900	172
8	24	3900	609
9	4	1300	290
10	0	2940	0
11	1	2400	40
12	2	2310	87
13	5	4200	119
14	0	1220	0
15	0	2830	0

FIGURE 4.

Cumulative Incidence per 100,000 Men aged 35-44

ANNUALIZED INCIDENCE PER 100,000 SINGLE MEN, BY QUARTER

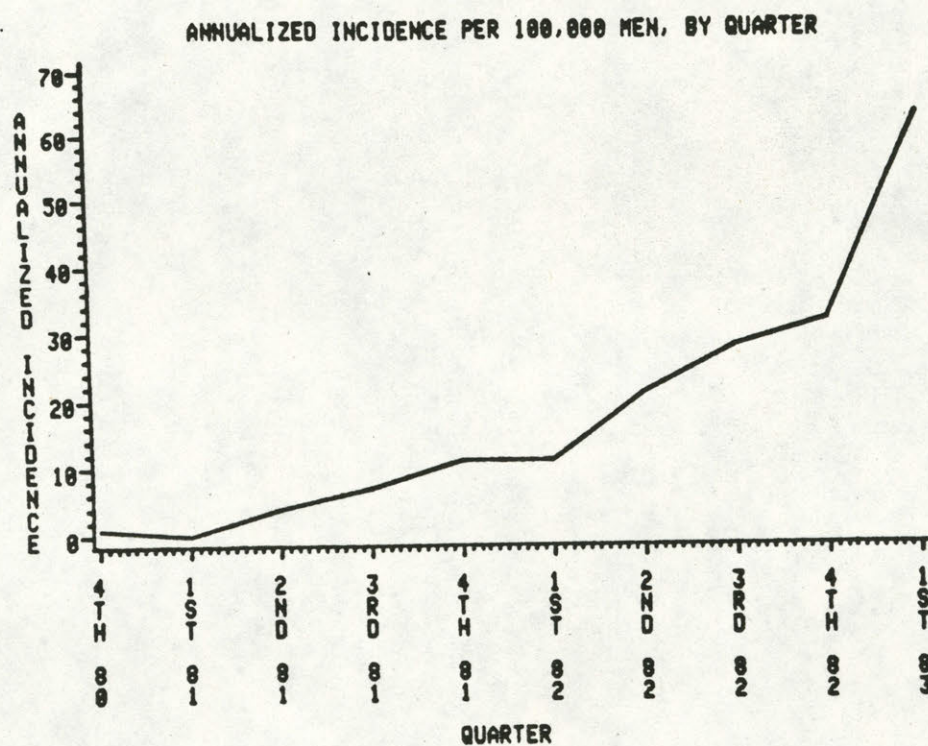


ANNUALIZED INCIDENCE PER 100,000 SINGLE MEN, BY QUARTER

QUARTER	CASES	SINGLMEN	INCIDNCE
4TH 80	1	133,170	3
1ST 81	0	133,170	0
2ND 81	3	133,170	9
3RD 81	5	133,170	15
4TH 81	8	133,170	24
1ST 82	8	133,170	24
2ND 82	15	133,170	45
3RD 82	20	133,170	60
4TH 82	23	133,170	69
1ST 83	46	133,170	138

TABLE 5.

Annualized Incidence per 100,000 single Men by Quarter



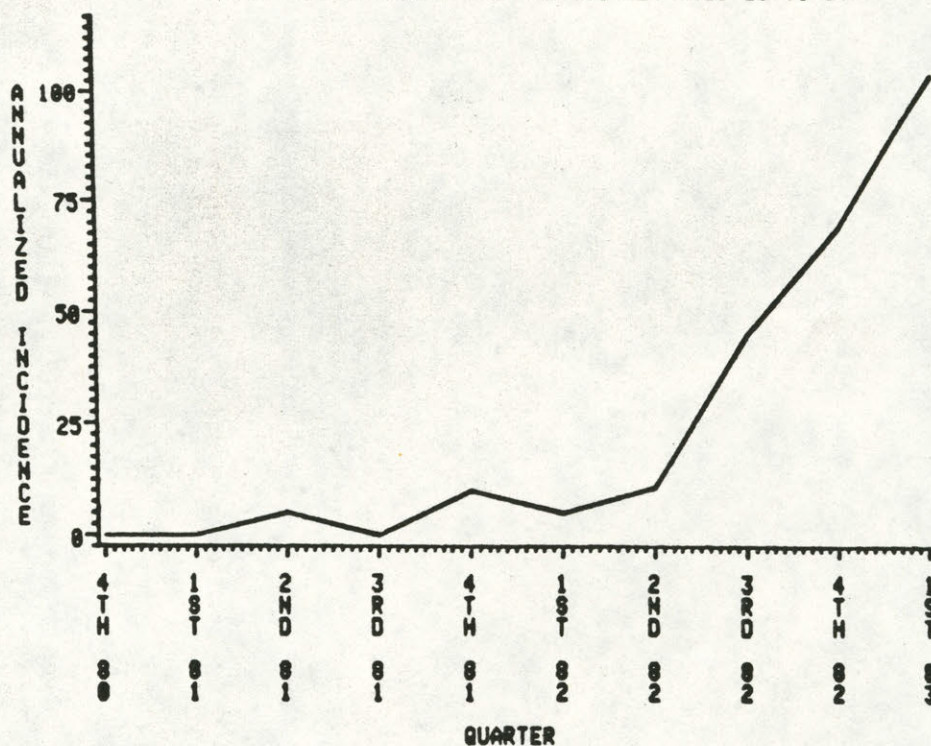
ANNUALIZED INCIDENCE PER 100,000 MEN, BY QUARTER

QUARTER	CASES	MEN15UP	INCIDNCE
4TH 80	1	290,450	1
1ST 81	0	290,450	0
2ND 81	3	290,450	4
3RD 81	5	290,450	7
4TH 81	8	290,450	11
1ST 82	8	290,450	11
2ND 82	15	290,450	21
3RD 82	20	290,450	28
4TH 82	23	290,450	32
1ST 83	46	290,450	63

TABLE 6.

Annualized Incidence per 100,000 Men, by Quarter

ANNUALIZED INCIDENCE PER 100,000 MEN AGED 25 TO 34

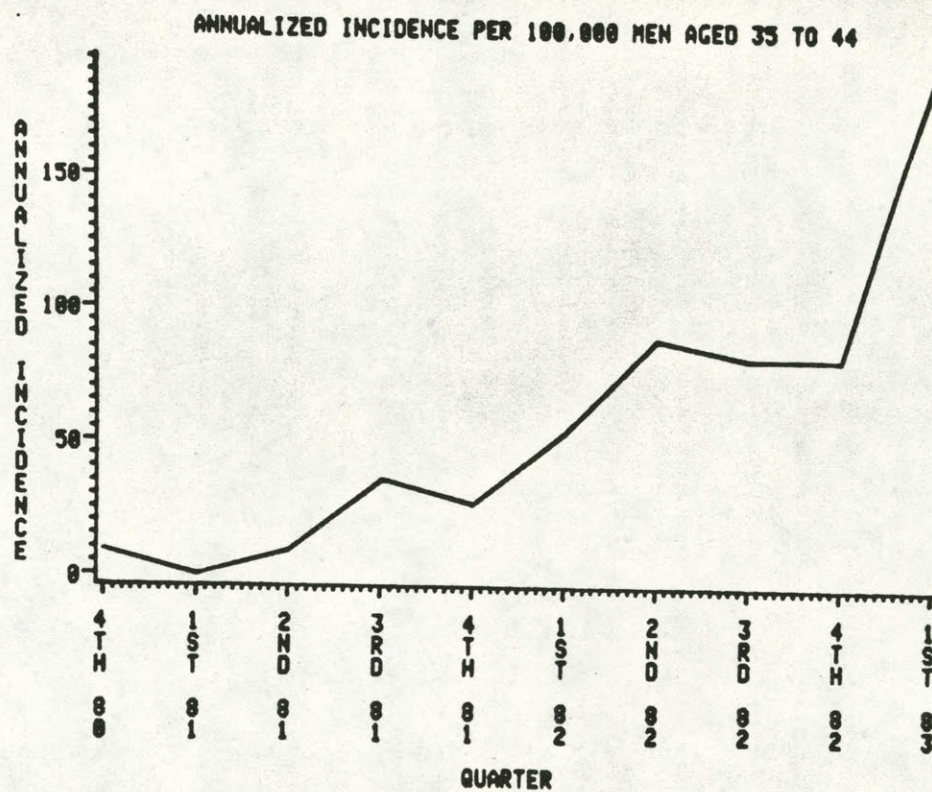


ANNUALIZED INCIDENCE PER 100,000 MEN AGED 25 TO 34

QUARTER	CASES	MEN25_34	INCIDNCE
4TH 80	0	80,840	0
1ST 81	0	80,840	0
2ND 81	1	80,840	5
3RD 81	0	80,840	0
4TH 81	2	80,840	10
1ST 82	1	80,840	5
2ND 82	2	80,840	11
3RD 82	9	80,840	45
4TH 82	14	80,840	69
1ST 83	21	80,840	104

TABLE 7.

Annualized Incidence per 100,000 Men Aged 25-34, By Quarter

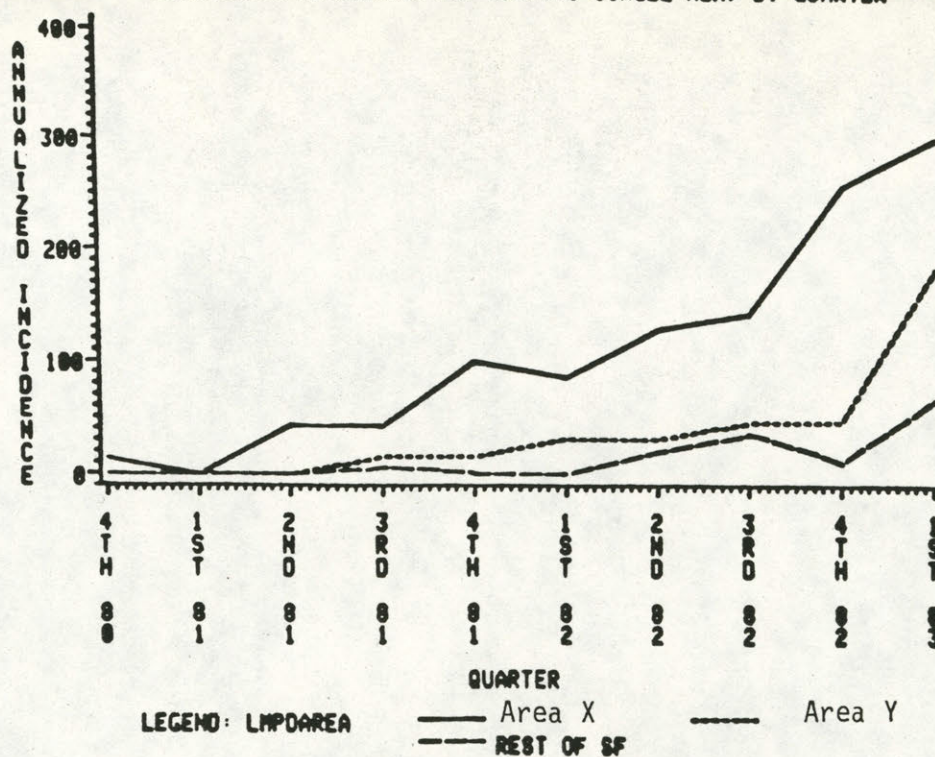


ANNUALIZED INCIDENCE PER 100,000 MEN AGED 35 TO 44

QUARTER	CASES	MEN35_44	INCIDNCE
4TH 80	1	44,040	9
1ST 81	0	44,040	0
2ND 81	1	44,040	9
3RD 81	4	44,040	36
4TH 81	3	44,040	27
1ST 82	6	44,040	54
2ND 82	10	44,040	89
3RD 82	9	44,040	82
4TH 82	9	44,040	82
1ST 83	21	44,040	191

TABLE 8.

Annualized Incidence per 100,000 Men Aged 35-44, By Quarter



ANNUALIZED INCIDENCE PER 100,000 SINGLE MEN, BY QUARTER

----- AREA=AREA X -----

QUARTER	CASES	SINGLMEN	INCIDENCE
4TH 80	1	27,950	14
1ST 81	0	27,950	0
2ND 81	3	27,950	43
3RD 81	3	27,950	43
4TH 81	7	27,950	100
1ST 82	6	27,950	86
2ND 82	9	27,950	129
3RD 82	10	27,950	143
4TH 82	18	27,950	258
1ST 83	21	27,950	301

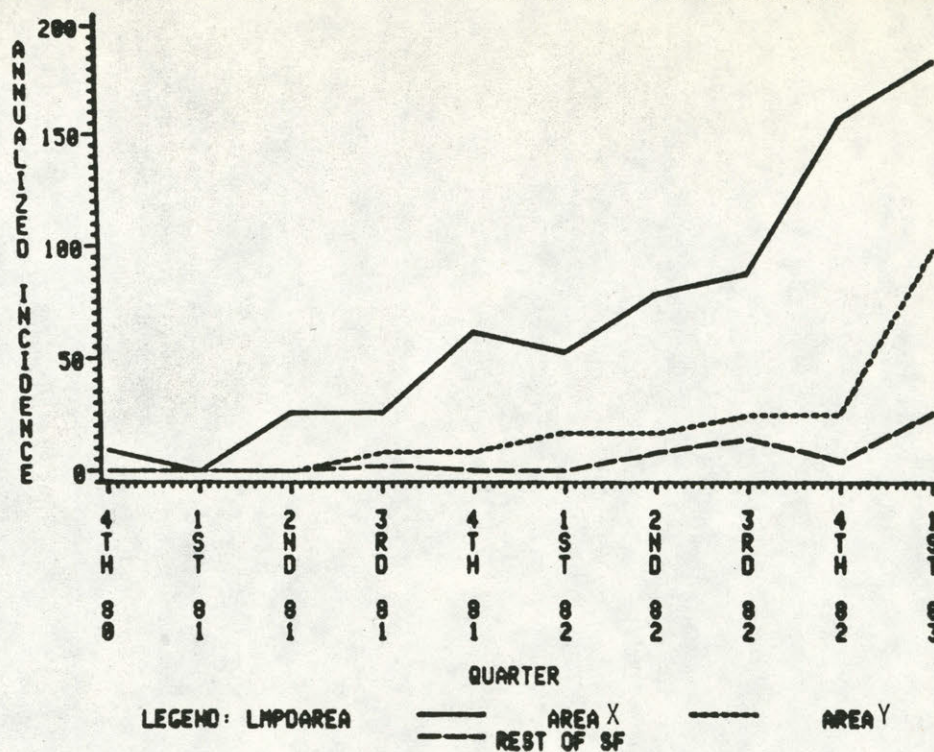
----- AREA=AREA Y -----

QUARTER	CASES	SINGLMEN	INCIDENCE
4TH 80	0	26,190	0
1ST 81	0	26,190	0
2ND 81	0	26,190	0
3RD 81	1	26,190	15
4TH 81	1	26,190	15
1ST 82	2	26,190	31
2ND 82	2	26,190	31
3RD 82	3	26,190	46
4TH 82	3	26,190	46
1ST 83	12	26,190	183

----- AREA=REST OF SF -----

QUARTER	CASES	SINGLMEN	INCIDENCE
4TH 80	0	79,030	0
1ST 81	0	79,030	0
2ND 81	0	79,030	0
3RD 81	1	79,030	5
4TH 81	0	79,030	0
1ST 82	0	79,030	0
2ND 82	4	79,030	20
3RD 82	7	79,030	35
4TH 82	2	79,030	10
1ST 83	13	79,030	66

TABLE 9. Annualized Incidence per 100,000 Single Men, By Quarter: Three Areas



ANNUALIZED INCIDENCE PER 100,000 MEN AGED 15 & UP, BY QUARTER

----- AREA=AREA X -----

QUARTER	CASES	MEN15UP	INCIDNCE
4TH 80	1	45,520	9
1ST 81	0	45,520	0
2ND 81	3	45,520	26
3RD 81	3	45,520	26
4TH 81	7	45,520	62
1ST 82	6	45,520	53
2ND 82	9	45,520	79
3RD 82	10	45,520	88
4TH 82	18	45,520	158
1ST 83	21	45,520	185

----- AREA=AREA Y -----

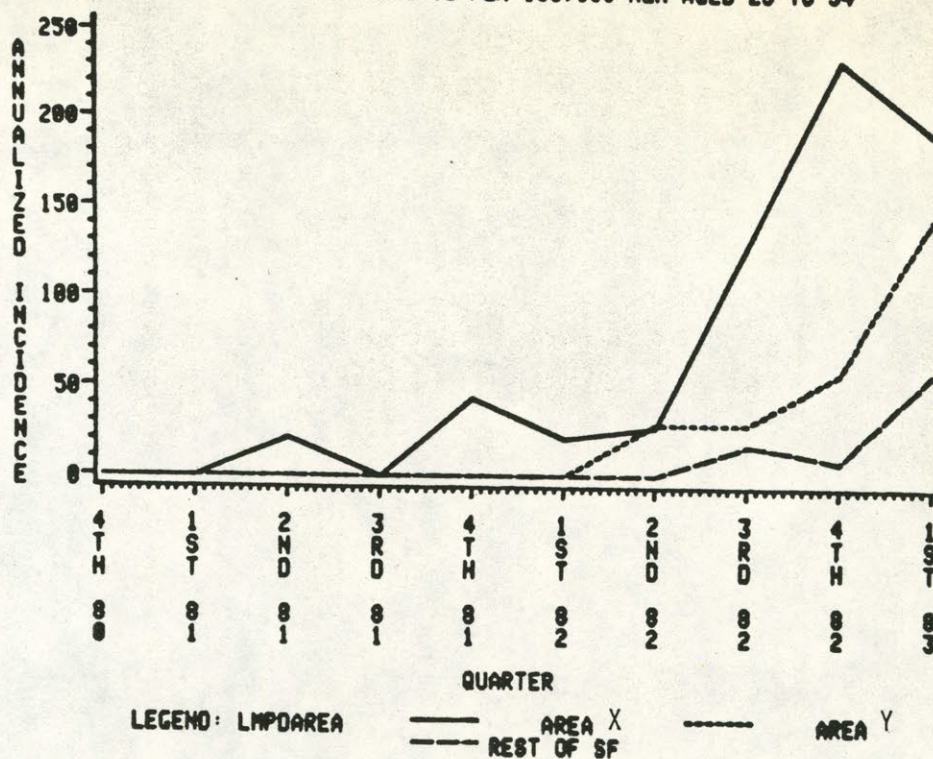
QUARTER	CASES	MEN15UP	INCIDNCE
4TH 80	0	48,270	0
1ST 81	0	48,270	0
2ND 81	0	48,270	0
3RD 81	1	48,270	8
4TH 81	1	48,270	8
1ST 82	2	48,270	17
2ND 82	2	48,270	17
3RD 82	3	48,270	25
4TH 82	3	48,270	25
1ST 83	12	48,270	99

----- AREA=REST OF SF -----

QUARTER	CASES	MEN15UP	INCIDNCE
4TH 80	0	196,660	0
1ST 81	0	196,660	0
2ND 81	0	196,660	0
3RD 81	1	196,660	2
4TH 81	0	196,660	0
1ST 82	0	196,660	0
2ND 82	4	196,660	8
3RD 82	7	196,660	14
4TH 82	2	196,660	4
1ST 83	13	196,660	26

TABLE 10. Annualized Incidence per 100,000 men, by Quarter: Three Areas

ANNUALIZED INCIDENCE PER 100,000 MEN AGED 25 TO 34



ANNUALIZED INCIDENCE PER 100,000 MEN AGED 25 TO 34, BY QUARTER

----- AREA=AREA X -----

QUARTER	CASES	MEN25_34	INCIDENCE
4TH 80	0	18,780	0
1ST 81	0	18,780	0
2ND 81	1	18,780	21
3RD 81	0	18,780	0
4TH 81	2	18,780	43
1ST 82	1	18,780	21
2ND 82	1	18,780	27
3RD 82	6	18,780	128
4TH 82	11	18,780	234
1ST 83	9	18,780	192

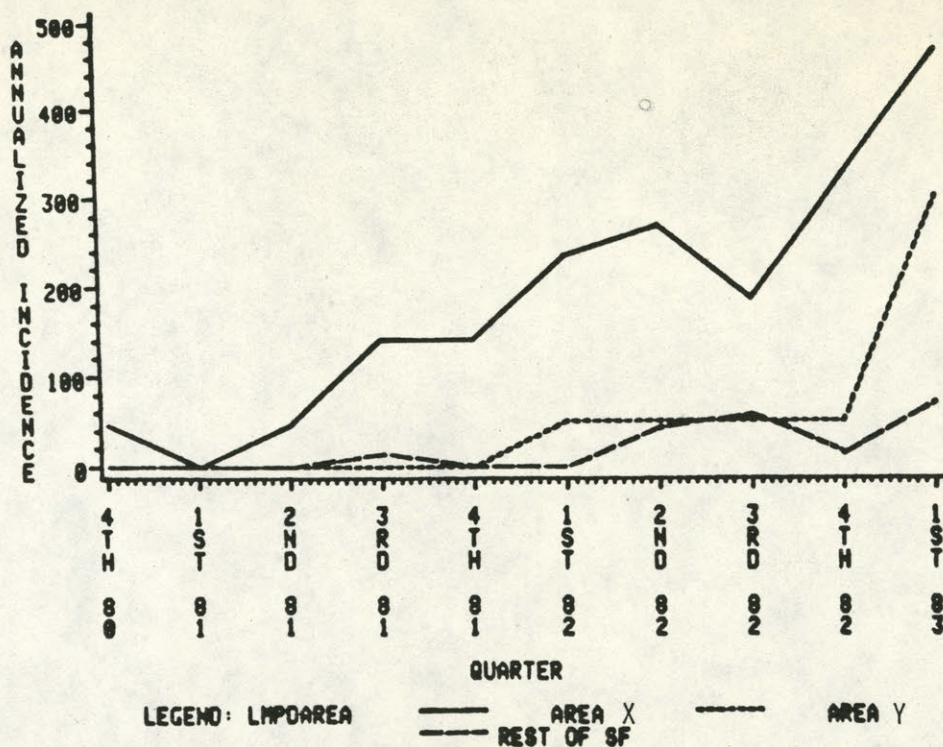
----- AREA=AREA Y -----

QUARTER	CASES	MEN25_34	INCIDENCE
4TH 80	0	13,790	0
1ST 81	0	13,790	0
2ND 81	0	13,790	0
3RD 81	0	13,790	0
4TH 81	0	13,790	0
1ST 82	0	13,790	0
2ND 82	1	13,790	29
3RD 82	1	13,790	29
4TH 82	2	13,790	58
1ST 83	5	13,790	145

----- AREA=REST OF SF -----

QUARTER	CASES	MEN25_34	INCIDENCE
4TH 80	0	48,270	0
1ST 81	0	48,270	0
2ND 81	0	48,270	0
3RD 81	0	48,270	0
4TH 81	0	48,270	0
1ST 82	0	48,270	0
2ND 82	0	48,270	0
3RD 82	2	48,270	17
4TH 82	1	48,270	8
1ST 83	7	48,270	58

ANNUALIZED INCIDENCE PER 100,000 MEN AGED 35 TO 44



ANNUALIZED INCIDENCE PER 100,000 MEN AGED 35 TO 44, BY QUARTER

----- AREA=AREA X -----

QUARTER	CASES	MEN35_44	INCIDNCE
4TH 80	1	8,450	47
1ST 81	0	8,450	0
2ND 81	1	8,450	47
3RD 81	3	8,450	142
4TH 81	3	8,450	142
1ST 82	5	8,450	237
2ND 82	6	8,450	272
3RD 82	4	8,450	189
4TH 82	7	8,450	331
1ST 83	10	8,450	473

----- AREA=AREA Y -----

QUARTER	CASES	MEN35_44	INCIDNCE
4TH 80	0	7,890	0
1ST 81	0	7,890	0
2ND 81	0	7,890	0
3RD 81	0	7,890	0
4TH 81	0	7,890	0
1ST 82	1	7,890	51
2ND 82	1	7,890	51
3RD 82	1	7,890	51
4TH 82	1	7,890	51
1ST 83	6	7,890	304

----- AREA=REST OF SF -----

QUARTER	CASES	MEN35_44	INCIDNCE
4TH 80	0	27,700	0
1ST 81	0	27,700	0
2ND 81	0	27,700	0
3RD 81	1	27,700	14
4TH 81	0	27,700	0
1ST 82	0	27,700	0
2ND 82	3	27,700	43
3RD 82	4	27,700	58
4TH 82	1	27,700	14
1ST 83	5	27,700	72

TABLE 12. Annualized Incidence per 100,000 men aged 35-44, by quarter: three areas

TESTIMONY TO THE HOUSE SUBCOMMITTEE
ON INTERGOVERNMENTAL RELATIONS

JULY 3, 1985

A.R. Moss Ph.D

Department of Epidemiology and International Health
University of California, San Francisco

Andrew Moss

Mr Chairman, members of the Committee:

Thank you for this opportunity to address the Committee
on AIDS.

I am an epidemiologist and I speak from the experience
of having the seen the AIDS epidemic in San Francisco grow
from a handful of cases in 1981 to the 1200 or so AIDS
cases we have today.

My experience has been much like the classic nightmare
in which you shout as loudly as you can about the coming
danger but nobody can hear you. Lewis Thomas said at the
first international AIDS meeting that we, in this
generation, had forgotten about infectious diseases -- we
had assumed that, in western countries at least, infectious
diseases had passed out of the ranks of great enemies of
the people. AIDS, he said, showed us we were wrong.

So one reason it was difficult for us to see what was happening in AIDS was that we have lost the collective memory of infectious disease jumping from one person to another-- people dying first in ones and twos, then in dozens, then hundreds, now thousands. That's what we have seen in the first decade of AIDS, the decade in which it was mostly a disease of homosexual men. Now, suddenly, we are facing the fact that we may see the same process not in homosexual men, or in the other "high risk" populations, but in plain old heterosexual americans.

So the first lesson that I learned from this era of AIDS, was that we have forgotten about infectious disease.

Secondly, we failed to deal with AIDS in homosexual men. They didn't believe it could be happening, and we -- researchers, doctors, public health people, politicians--we hoped it would go away. Because we couldn't deal, collectively, with a disease in a stigmatised population. Somehow the homosexuality of seventy percent of the cases froze the bureacracy, frightened off the funding agencies. It made us all reluctant to take the preventive measures that might have held the disease down to ten percent or fifteen percent of the gay population instead of the forty-five or fifty percent that have now met the AIDS virus, and are waiting

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to find out what's going to happen to them. The second lesson I have learned from the homosexual era of AIDS is that we weren't prepared to deal with disease in a stigmatised population.

But, members of the Committee, I have news for you. It isn't any easier to deal with AIDS in heterosexuals. AIDS is a venereal disease, at least in part, and the stigma of venereal disease is as great in the straight world as in the gay world. And AIDS is a disease of heroin addicts and probably will soon be a disease of prostitutes. This society has never been able to regulate either group, and does not know how to cope with an epidemic spreading from either group.

And, members of the Committee, as you probably know, AIDS is already in the wider heterosexual community, in considerable numbers. The tool we have now for dealing with the AIDS epidemic is the antibody test. That test has confirmed the other awful thing about AIDS besides its stigma, which is its slowness. In some ways, it's a very slow disease. When we saw the first homosexual case, AIDS had already been in the country for four or five years -- which is why I say we have just come to the end of the first decade of AIDS. And by the time we started

seeing sick people, there were many more infected people than we were seeing. In San Francisco we estimate that there are at least 25 times as many people with antibodies to the virus as people who are sick with AIDS. So in this small city there are maybe thirty thousand people in the real epidemic.

So now, as we start to see what we have learned to call "no risk factor cases" in heterosexuals, that epidemic is already four or five years old, and at least 25 times as big as what we see. There are for example 200 or so transfusion related AIDS cases, but perhaps five or ten thousand people who are seropositive from transfusions, and who may be infectious, and they don't even know who they are. And the same is true for sexual spread for the other primary risk groups -- from hemophiliacs and their spouses, from IV drug users, via prostitution, and, in from bisexuality.

AIDS spread very efficiently among homosexual men and drug users, and now it has begun to spread in the rest of the population. So the question in front of us is an empirical one. How fast will it spread? Who will it spread to? Will it damp out in the general population, like Hepatitis B, or will it spread explosively, like herpes? Shouldn't we know some of these answers already? Who is studying these questions? Who is responsible for national

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strategy on the spread and prevention -- on the public health aspects -- of AIDS? What is that strategy? Who is in charge? Don't tell me the CDC because I know that when it was time to close the baths in San Francisco it wasn't the CDC who had to do it. It was local government -- the Health Department, with its allies in the gay community and among researchers.

During the first decade of AIDS nobody was in charge. There was no strategy. There was no central direction of research effort. There was nobody who admitted to being responsible for actually stopping the epidemic. I suspect this is still true. There are upwards of three hundred thousand people in the US carrying the AIDS virus, most of them don't even know it, and so far as I know we have no proposal for doing anything about it. Vaccines won't help for several years. What we need is some kind of antiviral prevention. What do we have to give those three hundred thousand people, and how much of it, and how, not to cure them, but to stop them from spreading the virus? How do you find them? How do you get them to agree to treatment? Who's going to organize it? Who pays? What is the national strategy for prevention?

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A final sad lesson we learned from the first decade of AIDS, the homosexual decade of AIDS, was that the Federal government may not solve the problem. I am one of the lucky epidemiologists who was actually funded by the National Cancer Institute back in 1983, and what I get is twenty-nine thousand dollars a year, about a thirteenth of my research budget. I'm sorry to tell you that from our point of view in San Francisco, and I strongly suspect from New York and Los Angeles too, much of that money that you appropriate somehow stays back there close to Washington, where the government is, and doesn't make it out here, where the disease is. We've been doing AIDS epidemiology for three years now, and it was paid for by the City of San Francisco, god bless it, and by the State, organized by Willie Brown.

Finally, AIDS in its first decade was a homosexual disease. That's not going to be true in its second decade... Very soon AIDS in gay men is going to taper off --because there are only so many gay men, and because sexual behavior has really changed, at least in San Francisco.

Meanwhile, the number of heterosexual cases is going to rise, slowly, like everything in AIDS. Four or five or six years from now, there are going to be more heterosexual cases in the US than homosexual cases, and we will have learned to see AIDS as a venereal disease. But during that time, most of the sick people who we are going to see in the

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clinic are going to be gay men. So the antiviral therapies that the world is waiting for are going to be tested in clinical trial on sick gay men. And the prevention trials, if we manage to get them started, will be done only with the support of seropositive gay men. And the vaccine when it comes, like the hepatitis B vaccine, will probably be tested only with the cooperation and assistance of gay men. So I urge you to remember, as AIDS heads into its second, heterosexual, decade, that ironically the cure and the prevention that the whole world is waiting for are going to come only with the support of its first and most stigmatised group of victims.

5/31/88

Andrew Moss

Spring '82 he started working on it
Bill Rarrow 1st @ CDC

When realize big?

"We did this study that was
very simple in SF in '82... to
figure out an incidence rate
among gay men."

"We came out with 287/100,000...
it was a big number. And we
thought Christ! This is big.
That's when I began to have
fits of paranoia, in the fall of
'82."

"Very dramatic effect, on everybody."

"The study was very dramatic. I
said 3/yr 1000 gay men had
this disease. And we did this
curve that showed it rocketing
up... and you could hear people
go oooooh!"

Gov't impact?

On beginning nobody would give
you \$

"it was like an actual
injection... you know the
they is falling... you tell the

(2)

Moss

"its falling ... nobody listens"
Then in 1987 Fed. started throwing
\$ @ AIDS

Brontosaurus metaphor

"we're walking on the tail
in SF for 2 yrs and finally
the message gets through
to the head"

★
"And the truth is it's just
like that now with drug
users." another 2 yrs...

OTA report ← check it
~~~~~~~~~

Bell shaped curve?

"OK flat. The actual new cases  
are flat for gay men in SF."

"I actually bet will see a decline  
in gay cases next year. We're  
at the hump now."

(all early projects, assumed no behavior  
changes)

Big unknown is heteros ...

"I really do think it  
a big unknown"



③ Mrs

"We don't know what's going to happen in terms of Epidemic 3... people can't focus on it calmly."

NYC — — can't get agreement on what's happening ... too threatening

Ries, § : "it's done it. It's like cancer, went up to \$1 B per... then down" Has produced bad research ... lots of junk ... opportunism, Can't use that much \$ that fast.

War on Cancer metaphor

Research front → "the channels are a bit plugged now, and you have to compete with a lot of people now who are only in it for the \$ ... it's a machine now that has nothing to do with real world public health."

Cholera e.g. : They built an infrastructure @ local level to do it.

Real AIDS means delivering health care to poor people, "something the U.S. doesn't do well at all"



④ Moss

- we're talking about public health  
It's a 19th century construct

"It has to be reinvented  
to deal w/ AIDS"

- "The issue is how you deliver  
PH to poor people"

- africa, e.g., only hope is PH

- system is set up to do high  
intensity med... not PH

- "The S. Bronx is where it's at. One  
quarter of all hetero. cases are  
there. Where is the PH  
apparatus? It's not there. Zuck.  
You think we're going to do  
something about the S. Bronx? Forget  
it! We never will. That's the real  
~~the~~ U.S. Crisis."

- Cycles of response:

"The missing middle. There are only  
2 poss. rxns: the sky is falling,  
or it's not falling. The public  
never sees a middle."

• "Of you think about what America  
will do about the fact AIDS  
is a poor people's disease  
it's not ~~great~~ promising. Look  
at history."



⑤ Moss

He is personally shifting to drug  
users

Sometimes he burns out...

says it's possible, get out

We are trying to do P/H

NIDA just cut his grant ----

their reviewers don't understand  
AIDs

"this is just like 1983. got to  
watch the brontosaurus tail again  
to get \$."

- "do I really want to do this? go to  
battle again? And the answer is  
I don't know."

- Basic criteria is are we useful

- How far does altruism go?

"This isn't an altruism business  
any more. It's 1/3 \$, 1/3 <sup>Politics</sup> ~~altruism~~  
and 1/3 revenge. Now it's business  
as usual."

- AIDs is normalized. ~~It's~~ ~~normalized~~

"It has to be normalized"

"It's normal or nothing."

The real heads of AIDs are the guys  
who work S. Bronx & such. ~~AD~~  
"I'm pretty sick of it to tell you the truth"



(6) Moss

Why hang in there?

We got a lot of strokes. That  
keeps you going  
I don't care about AIDS politics...

Stockholm?

"a lot of flesh gets pressed."

"my buddies will be there"

- it's normalized too. Like AAAS mtg.

Compared to European Sci.....

more seminar collaboration science

"it's a convention"

like any professional convention