Queering Epidemiology

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Shame and Stereotypes

Ecofeminism (Silvey 1998) and Queer Ecology (Gandy 2012) highlight relations among gender, sexuality, and nature. The agenda of 'queering ecology[, …] opening up […] environmental understanding to explicitly non-heterosexual forms of relationship, experience, and imagination as a way of transforming entrenched sexual and natural practices towards […] queer […] ends' (Mortimer-Sandilands and Erickson 2010, 30), resonates within Medical Geography and Epidemiology. This essay shows how we might track the effects of entrenched homophobia within the geographical framing of disease by examining one important set of epidemiological writings, those in which AIDS was first registered as a new mortality. I show how homophobic stereotypes shaped scientific writings, and how, in related but different ways, they pervaded the public geographies of AIDS circulating in the mass media. Finally, I will show how activists tried to undo the murderous homophobia of AIDS discourses building understandings of HIV vulnerability that were accepting of sexual diversity, effectively queering epidemiology.

Homi Bhabha (1983, 18) has highlighted the ambivalence of stereotypes, 'a vacillation between what is always "in place", already known, and something that must be anxiously repeated.' With repetition, the stereotype is confirmed but is never challenged by evidence. Again and again, the stereotype gives the unexamined Self the pleasure of feeling superior to the derided Other. The Self obsesses about the Other, and yet avoids confronting its own place in a world that includes the subjugation of this Other. This separation has its problems. In the first place, the Self is all too likely to project onto the Other repressed aspects of the Self which conjure the stereotype as a fantasy object (Stallybrass and White 1986). Alongside the pleasures of superiority and innocence, then, the Self is also able to enjoy disavowed elements of itself by dwelling on the Other's exemplary depravity, but this relish activates the guilty secret of identification. Secondly, it is terribly difficult for the Self to find in the Other no spark of humanity at all. The stereotype, then, is 'an "impossible" object' (Bhabha 1983, 33). Herein lies the violent energy of abjection, 'the powers of horror' (Kristeva 1982) that attend any threat to the distinction between Self and Other.

I want to propose that Epidemiology makes stereotypes in precisely these ways, through repetition, projection, and abjection. Epidemiology is a particularly suggestive vehicle for stereotyping (Craddock 2000; Sothern 2007) because of the ways the Other is produced out of the Self's horror

and disgust of its own corporeality. Nussbaum (2004, 74) has suggested that 'disgust embodies a shrinking from contamination that is associated with the human desire to be nonanimal, it is frequently hooked up with various forms of shady social practice, in which the discomfort people feel over the fact of having an animal body is projected outwards onto vulnerable people and groups.' Disgust, then, 'is typically unreasonable, embodying magical ideas of contamination, and impossible aspirations to purity, immortality, and nonanimality' (Nussbaum 2004, 14). These 'magical' ideas of contamination invite Epidemiology as both metaphor and justification. In this way, Geography and Sociology are collapsed, since the risk is at once both a contagion and a pariah. These discourses of disease are among the most resonant of public geographies but there have been few studies of their role in reproducing heteronormativity (Brown 1995; Hubbard 2000).

The Self's horror of its animality and mortality, is paradoxically often also expressed as an insistence that it and not the Other is properly natural (Romanillos 2011; Barnett 2012). The harmony between Nature and Self is asserted most strongly with respect to sex for, as Gaard (1997, 118) has observed, there is in Western culture, 'a fear of the erotic so strong that only one form of sexuality is overtly allowed.' Among the disallowed sexualities, '[i]n Euroamerican-dominant contexts, two kinds of sex have been (are) said to be toxic to nature: reproductive sex between non-white people, and sex between men' (Gosine 2010, 149). Both sorts of sex were implicated in the

epidemiological understanding of AIDS but it was sex between men that first drew the attention of epidemiologists. Homophobia proved to be the stable prejudice around which were arranged, like iron filings in a magnet field, the early epidemiological findings about AIDS, seemingly regardless of contradictions and conflicting results. In epidemiological terms, it seemed that the crucial question was whether AIDS was caused by some distinctive element of a gay lifestyle or rather by an infectious agent. Yet, in the epidemiological writings, both hypotheses rested upon assumptions and invited attitudes that tightened the screw of homophobic prejudice. Public reporting about AIDS rested upon multiple iterations of these prejudices in different fora, giving shame and stereotypes many opportunities to adjust the aim of AIDS science back towards its 'homosexual' target. In this it was so successful that AIDS became, for the general public, a marker of homosexual identity, 'outing' as gay those who were infected. I conclude by describing how this dismal science was challenged and devalued by queer activists, queering epidemiology themselves.

Homosexual diseases?

Pneumocystis carinii pneumonia (PCP) is caused by a protozoa-like organism that is a common infection. It rarely causes sickness unless a person's immune system is seriously compromised. On 5 June 1981, the Centers for Disease Control (CDC) in Atlanta, Georgia, published a communication in

their *Morbidity and Mortality Weekly Report*, '*Pneumocystis* Pneumonia–Los Angeles.' The note began: 'In the period October 1980-May 1981, 5 young men, all active homosexuals, were treated for' PCP (CDC 1981a). The homosexualization of the disease was evident with this very first sentence, yet the phrase 'active homosexual' is not explicated. Later in the short piece we find that '[t]he 5 did not have comparable histories of sexually transmitted disease,' and that only '[t]wo of the 5 reported having frequent homosexual contacts with various partners.' The editorial comment appended to the piece drew a preliminary conclusion: 'The fact that these patients were all homosexuals suggests an association between some aspect of a homosexual lifestyle or disease acquired through sexual contact and *Pneumocystis* pneumonia in this population.' Note that two alternative causes are sketched: lifestyle factors or a sexually-transmitted infection (STI). Pariah and contagion were immediately evoked.

In the *Los Angeles Times*, Wayne Shandera, a CDC epidemiologist, explained that: 'The best we can say is that somehow the pneumonia appears to be related to gay life style' (Nelson 1981a, B3). Officially, then, the CDC was entertaining both an STI and a lifestyle explanation, but in speculating to a journalist in Los Angeles, this official tilted towards a lifestyle explanation. Crucial in this respect was researchers' puzzlement over the fact that 'there have been no reported cases of this type of pneumonia among gay women or heterosexuals of either sex' (*ibid.*, B25).

This article also reported that in addition to the cases in Los Angeles, there were also '[a]nother half dozen cases […] under investigation in San Francisco, along with an undetermined number in New York, Toronto and Florida (*ibid.*, B3). The cases in New York were not written-up in the CDC report because an article was in preparation for a medical journal and thus the findings were embargoed (Shilts 1987, 67).

In May, that is before any CDC report, a gay newspaper, the *New York Native*, had reported on a strange disease among gay men in Manhattan. Seeking confirmation from the New York City Department of Health, Lawrence Mass was told that 'the rumors are for the most part unfounded' (Mass 1981). 'For the most part,' is a strange way to deny a rumour, but the phrasing was perhaps motivated by a wish not to alarm gay men. Shilts learned that, as submitted, the first CDC article on PCP in Los Angeles had borne the title, '*Pneumocystis* pneumonia in homosexual men–Los Angeles,' but the CDC was a beleaguered institution in Reagan's America (Harden and Rodriguez 1993) and it had no wish to advertise its connection with gay issues. It was also reluctant to fuel prejudice against gay men, especially as gay men had been the main clinical volunteers in the CDC programme developing a vaccine for hepatitis B. Shilts suggested that, in dropping the reference to 'homosexual men' from the title of the article and placing the article on page two rather than as the cover story, the CDC was animated by twin desires: 'Don't offend the gays and don't inflame the

homphobes' (Shilts 1987, 69). Without speculating too far about the motives of the epidemiologists and journalists, it is clear that the 'homosexual' label was handled as more a matter of public-relations than of clinical precision. It was the pervasive homophobia that made this seem necessary.

When the research from New York was published in the summer of 1981, it extended the picture of 'exotic' disease in significant ways. The article was about Kaposi's sarcoma (KS), a slow cancer among elderly men, but now manifesting as an aggressive, widely distributed and fatal condition among some young gay men. The authors proposed an infectious explanation: 'This sudden, very high incidence of the condition in male homosexuals suggests an epidemic and raises the possibility of an infectious cause, especially because homosexuals are now known to have high incidence of many infectious diseases, e.g. venereal diseases and viral hepatitis' (G. Gottlieb 1981, 111). The known cases of PCP and KS were brought together in a further report from the CDC, this time on the front page of their *Morbidity and Mortality Weekly Report* and referring explicitly to 'homosexual men' in its title (CDC 1981b). This focus was modified a little in the editorial note appended to the article: 'Although it is not certain that the increase in KS and PC pneumonia is restricted to homosexual men, the vast majority of recent cases have been reported from this group' (CDC 1981b, 307). There was no mention of the infectious agent hypothesis in

this summary article and in its first report on these new 'homosexual' diseases, the *New York Times* went so far as to assert that 'there is as yet no evidence of contagion' (Altman 1981). James Curran, head of the Venereal Diseases unit at the CDC and chairing its new Kaposi's Sarcoma and Opportunistic Infections Task Force (KSOI), assured the *Times* that 'there was no apparent danger to nonhomosexuals from contagion. "The best evidence against contagion," he said, "is that no cases have been reported to date outside the homosexual community or in women"' (*ibid.*).

In its own report, then, the CDC treated the concentration of cases among homosexuals as evidence in favour of an infectious agent, whereas in its comments to the newspaper the same evidence was offered as evidence against contagion. A contagious disease is one that is relatively easily transmitted between persons, so that both statements could be true: the disease is caused by an infectious agent but is not contagious. Nevertheless, the choice of the first statement for the technical epidemiological report and the second for the newspaper is a matter of public relations rather than public health. The second inconsistency is between, on one side, both the reporting only of cases among 'homosexual men' in the CDC article together with the statement to the *Times* that there were 'no cases' outside this group, and, on the other, the editorial note that claimed merely that the 'vast majority' of recent cases of PCP and KS had been among homosexual

men. The last was more accurate because, even at this early stage, the CDC knew of cases that did not fit the homosexual pattern.

The sickness that is homosexual difference

The 'homosexualization' of the disease was relentless and, while (as I will show below) it could be served both by the infectious agent hypothesis and by the lifestyle hypothesis, the latter carried moral opprobrium more easily while reassuring straight people that they had no cause for anxiety. In its own article on the CDC report, the *Los Angeles Times* admitted that '[r]esearchers are still unable to explain why male homosexuals appear to be especially vulnerable' to PCP and KS but it added that 'Friedman-Kien [of the New York University Medical Center had] said […] that all of the victims have been exceptionally promiscuous' (Nelson 1981b). Friedman-Kien himself told the *New York Times* that 'most cases had involved homosexual men who have had multiple and frequent sexual encounters with different partners, as many as 10 sexual encounters each night up to four times a week' (Altman 1981). Again, 'most' and 'up to' hinted at a diversity that ill served the lifestyle hypothesis.

In contrast, the infectious agent argument did not need to (although it could) pathologise gay sexuality. With a new STI, the majority of the early cases would very likely be among people with the most exposure, people having unprotected sex with many partners, who in turn themselves have

unprotected sex with many partners. This is a matter of probability rather than morality. Even among the first five cases of PCP, recall that only two of them self-reported as 'having frequent homosexual contacts with various partners' (CDC 1981a). Another of the early cases was a thirty-seven year old man with KS, 'currently in a stable homosexual relationship' and with 'no past history' of STIs (Bokovic and Schwartz 1981, 902). When the author of the very first CDC report, Michael Gottlieb, wrote up the clinical features and blood-work results for four cases of PCP for the *New England Journal of Medicine*, he reported significant diversity within the group: 'Patient 3 was highly sexually active and frequented homosexual bars and bathhouses. Patient 1 had lived with one partner for seven years, and Patients 2 and 4 had regular partners' (M. Gottlieb *et al.* 1981, 1429). Even if one would expect to find 'promiscuous' people among the early cases, one would also expect that focus to fade as an STI became more widely dispersed. Yet the pathologization of gay sexuality led people to project AIDS as a disease of and for the 'promiscuous' gay male, offering false security to all other sexually active people, gay or straight.

Within six months of the first reports, there was already significant evidence that this new condition of immune failure was not associated only with gay men. As early as July 1981, one month after its first report, the CDC was investigating cases of PCP among injecting drug users and, while some researchers assumed that these men must be lying about not having

sex with other men, the field researcher, Mary Guinan, who had conducted the interviews found them credible (Shilts 1987, 83). In December 1981, one study from New York City described eleven men with PCP, of whom seven were injecting drug users and six were homosexuals, including two reporting themselves as homosexuals who injected drugs (Masur *et al.* 1981). The profile of the epidemic provided by the CDC in January 1982 likewise noted that of 158 cases of KS, PCP or other serious opportunistic infections with no known cause for immune suppression, twelve were among men understood to be exclusively heterosexual (CDC 1982a, 251). Yet the early researchers continued to try to understand the condition as a manifestation of something specific to gay men; the gay lifestyle hypothesis required nothing less. In 1982, there were reports of 'Gay-related immunodeficiency' (GRID) (M. Gottlieb *et al.* 1982; Horowitz *et al.* 1982). A communication of September 1982 to the *British Medical Journal*, referred to 'gay compromise syndrome' (Oswald *et al.* 1982) as had a letter published in December 1981 (Brennan and Durack 1981).

If the condition was not exclusive to gay men, then, forlorn must be the effort to isolate a specific element of gay behaviour as its essential cause. Yet, the search for a gay cause of AIDS continued long after the diversity of the condition was known. Even the earliest letter in the *British Medical Journal* to refer to a 'gay compromise syndrome,' had noted in its first paragraph that of the first 108 cases of KS and PCP that were reported, only '94% of

the patients were homosexual or bisexual' (Brennan and Durack 1981, 1338). Even as the terminology of the disease evolved, this tension persisted, between a gay disease and its non-gay sufferers. Shilts suggested that some epidemiologists thought they might avoid stigmatizing gay people if they referred instead to 'community acquired deficiency syndrome': 'The "community" […] was a polite way of saying gay' (Shilts 1987, 138). For example, Arthur Levine reported in June 1982 on the first 300 cases in the United States, of whom 242 were identified as gay or bisexual men, and then ignored the other 58 to insist that 'the syndrome is occurring mainly in a particular subset of the homosexual male population' and that 'this is the first documented epidemic of community-acquired immune dysfunction' (Levine 1982, 1392).

By focusing only upon the gay cases, the question could be posed: What is it about gay men that makes them sick? If the whole population of sufferers was comprehended, the question became: What vulnerability did this group of gay men have in common with this other group of people who are not gay men? But the second was far less efficient as a vehicle for stigma. This representation of a community somehow collectively acquiring a failure of immunity rendered homosexuality dangerously akin to biological suicide and the hint was taken in such homophobic commentary as that of Patrick Buchanan (1983): 'The poor homosexuals–they have declared war upon Nature, and now Nature is exacting an awful retribution.'

The incoherence that this notion of 'community' produced is evident even in the titles of some epidemiological articles–'Opportunistic infection in previously healthy women: Initial manifestations of a community-acquired cellular immune dysfunction' (Masur *et al.* 1982); 'Community-acquired opportunistic infections and defective cellular immunity in heterosexual drug abusers and homosexual men' (Small *et al.* 1983). In these examples, it is far from clear which 'community' is being imputed but the association of gay men with disease was often reinforced even when speaking of the vulnerability of other people. For many newspapers and magazines, the disease only became visible when it affected people who mattered more to the editors than did gay men. Thus, dozens of gay men had died but the reporting of 23 cases among people identifying as heterosexual broke down the reluctance of editors at the *Wall Street Journal* which published its first AIDS piece in February 1982, 'New, often-fatal illness in homosexuals turns up in women, heterosexual males' (Bishop 1982).

Shilts (1987, 126) was right: 'The gay plague got covered only because it finally had struck people who counted, people who were not homosexuals.' Yet, the natural home of AIDS among gay men was reinforced even in this early reporting of non-gay cases (Nelson 1982). Thus the first *Newsweek* article observed that 'the "homosexual plague" has started spilling over into the general population' (Keerdoja and Morris 1982). It was

as if the homosexual community was so saturated with disease that sickness was now moving beyond its natural limits, an impression reinforced in a subsequent *Newsweek* article that warned of AIDS 'creeping out of well--defined epidemiological confines' (Seligmann *et al.* 1983, 74). In its natural form, then, AIDS, then, is made to seem a gay disease, although the deceptively-titled 'general population' is warned that it might break out towards them–how unfair. As Jan Zita Grover (1987, 23) noted, the 'general population' denoted the part of society that 'is virtuously going about its business, which is not pleasure-seeking (as drugs and gay life are uniformly imagined to be), so AIDS hits *its* members as an assault from diseased hedonists upon hard-working innocents.' This homophobia both produced and was reproduced by the homosexualization of AIDS.

A Morbid Lifestyle?

Despite the diversity within the group of people sick with AIDS, the epidemiological focus upon gay men was relentless and it directed attention to what was different about gay men, pathologizing that difference. Friedman-Kien, who had spoken to the *New York Times* of the 'exceptionally promiscuous' patients he was seeing (Nelson 1981b), wrote up the cases in similar terms for a medical journal, describing nineteen patients as 'young homosexual men, highly sexually active' (Friedman-Kien *et al.* 1982, 697). This focus likewise directed the search for causes:

> The recent appearance of this disease may be associated with changes that have occurred over the last 15 years in the lifestyle of homosexual men living in large urban centers. There has been a marked increase in gay bathhouses, bars, and meeting places where multiple anonymous sexual encounters occur. […] Use of multiple recreational drugs, especially the inhalation of amyl and butyl nitrite […] is also an important aspect of this changing lifestyle (*loc. cit.*).

The lifestyle dimension was the preferred focus of epidemiologists, particularly 'nitrite exposure and promiscuity' (Levine 1982, 1394). But once the epidemiologist had identified gay men as the source, then, speculation was unbridled: 'It is also possible that a retrovirus is involved in these malignancies: moreover, as a consequence of intercourse with animals, an animal retrovirus might have been introduced into the homosexual population' (*loc. cit.*). No evidence was cited for this observation and yet it was acceptable in a refereed medical journal. Can one imagine such speculation being acceptable in the absence of pervasive homophobia?

Lifestyle was understood as implicated by the clustering of early cases, but, bedeviled by equifinality (Olsson 1969), such patterns are ambivalent. Thus while one epidemiologist could assert that '[t]he possibility that an infectious agent represents the ultimate cause stems from […] the geographic clustering of cases […], suggesting common sources of possible

primary infectious factors' (Quagliarello 1982, 447), others countered that '[t]he geographic clustering of cases suggests causal factors related to lifestyle or environment' (Haverkos and Curran 1982, 335).

One way to establish the significance of various lifestyle elements would have been to frame a case-control study for multidimensional comparisons between the sick and the well. From July 1981, the CDC wanted to conduct such a case-control study of the PCP and KS cases but the National Institutes of Health (NIH) advised that it would take three years to devise, recruit for, and complete such a study (Shilts 1987, 81). Shilts (1987, 96) reported that by September 1981 the KSOI had devised a questionnaire and were seeking controls for each KS or PCP case. Selma Dritz (1995, 16), an epidemiologist with the San Francisco Department of Health, recalled that she used 'the questionnaire on about 100 of the patients here' but that having 'gathered all the information and sent it all back to CDC, […] it took them two years to do a computer analysis of it […]. Jim Curran was crazy; he was wild: NIH wouldn't give him the money.' The findings of the case-control study were not published until August 1983 (Jaffe *et al.* 1983). The delay implied that it was enough to know that gay life was at fault. There was little urgency about facilitating its healthy flourishing.

Yet, the critical evidence against the lifestyle hypothesis was available within months of the first reports. Cases among heterosexual injecting drug users were published in December 1981 (Masur *et al.* 1981). In July 1982 the

CDC reported cases among Haitian men who denied having had sex with other men, but they also reported cases among Haitian women (CDC 1982b). In the same month, came reports of unexplained PCP among hemophiliacs (CDC 1982c). In November an article about unexplained PCP, KS and other opportunistic infections reported on 86 heterosexual men and 35 heterosexual women, and noted that of these about three-in-five reported injecting drugs (Allen and Mellin 1982). In December 1982, the CDC reported on AIDS transmitted by blood transfusion (CDC 1982d) and transmission *in utero* (CDC 1982e). At the start of 1983, the CDC described a set of cases among the female sexual partners of infected males (CDC 1983). Nothing about a gay lifestyle could explain any and certainly not all of these but as the CDC, in reviewing the early years of AIDS with the hindsight of 2011, observed of the reception of these sorts of findings which argued strongly in favour of an infectious agent as the cause of AIDS: 'Nonetheless, whether because of competing hypotheses or merely denial, many scientists and the public were skeptical of the infectious agent causation theory' (Curran and Jaffe 2011, 65).

Curran and Jaffe do not explain the nature of the 'denial' to which they refer. In an oral history of the epidemic, Marcus Conant, who founded the first KS clinic in San Francisco, was asked: 'Why did it take so long to accept the idea that the epidemic was caused by a transmissible agent, and to forget about poppers and all the other things that the CDC and other

people were looking at?' His response was almost as evasive as the vague reference to 'denial': 'Well, that's a very good and very complex question, and there's not a simple and easy answer. I guess if there were, this whole terrible epidemic in America would not have happened' (Conant 1996, 144). In other words, focusing upon the gay lifestyle was a way of attending to the epidemic that went hand-in-hand with systematic neglect. The implication is that by identifying AIDS as a gay disease, it was not felt to be urgent.

It's not enough to blame this on individual scientists although one epidemiologist suggested in 1992 that 'Harry Haverkos of the CDC invested his career in [the poppers hypothesis], pushed it very hard. He's still pushing on it' (Moss 1996, 248). He would not be the last, and Peter Duesberg (1987) returned to this hypothesis and from 1990 he promoted it to the South African government (Kalichman 2009). Another way of putting this question is to ask: Why did it seem so easy for scientists, pundits and members of the public to believe that there must be something about a gay lifestyle that was intrinsically harmful to health? With this belief, many people behaved like Pat Buchanan and were content to let nature run its course. Such savage insouciance drove Larry Kramer (1989) to speak of a gay holocaust engineered by an institutional failure to address the epidemic.

Contagion

When the evidence for an infectious agent was overwhelming, the stereotyping and prejudice did not end, it took instead the form of contagion rather than pariah. In early 1982 the CDC's Epidemic Intelligence Service officer assigned to the Los Angeles County Department of Health Services was approached by a local gay man who knew there to have been sexual contact between a number of the earliest cases in southern California (Curran and Jaffe 2011, 65). By June 1982, the CDC felt able to report on a 'cluster' of patients in southern California. Of the 19 reported cases of KS or PCP reported from southern California by 12 April 1982, the eight still living were interviewed, as were close friends of seven among the eleven already deceased. From these fifteen cases, data on aspects of sexual histories were collected for thirteen of them. Taking a five-year period preceding the onset of KS or PCP, nine of these thirteen had some sexual contact with persons who either were already diagnosed with KS or PCP or would later be so diagnosed. At this time, very little was known about the latency period between HIV infection and the development of serious opportunistic infections and the CDC observed three patients who developed KS after sexual contact with someone already diagnosed with KS. In these cases the intervals were nine months, thirteen months, and twenty-two months respectively (CDC 1982f, 305). Among the sexual contacts detailed to the CDC two KS patients from Orange County and two PCP patients from Los Angeles County were among the sexual contacts detailed

by a non-Californian who was also interviewed. In rather low-key reporting, the *New York Times* reported on the cluster study as 'new evidence […] suggesting that the outbreak is linked to an infectious agent' (Altman 1982).

Very quickly, the non-Californian himself became identified as the infectious agent. With these early results from the cluster study, Selma Dritz approached the non-Californian:

> I told him, 'Look, we've got proof now.' I didn't tell him how scientifically accurate the information was. It wasn't inaccurate, but it wasn't actually scientifically proven. I said, 'We've got proof that you've been infecting these other people. You've got AIDS, you know. We know it's transmissible now, because you're transmitting it.' He was the active partner in all this gay business, anal-genital sex. 'You've just got to cut it out.' 'Don't be silly, I won't cut it out. It's my life. I'll do what I want' (Dritz 1995, 35-6).

Marcus Conant (1996, 166) recalled that, as soon as he heard about the cluster study, Randy Shilts, working for the *San Francisco Bay Chronicle*, 'went nuts trying to get the name out of me as to who the patient was.' Someone did leak the name and Gaétan Dugas, a Canadian airline steward, was soon facing personal threats, 'a group of gay men had decided to drive the "Orange County connection" out of town for so purposefully spreading the disease' (Shilts 1987, 208). At least one of these men, 'a Vietnam veteran, ex-

marine medic, who had Kaposi's sarcoma,' and who had named Dugas as one of his sexual contacts, claimed that he was 'waiting for Dugas to come back to San Francisco so he could kill him' (Moss 1996, 281). The personification of the disease and attributing base motives to the guilty men spreading it, became dominant themes in later press coverage, but they are found here already in the actions of epidemiologists who leaked names and invited mob justice.

The full cluster study would not be published until 1984 but in 1983 fears of contagion were incited by another epidemiological study. A study of AIDS among eight infants in New Jersey established that in each case 'our patients had in common household exposure to one or more persons with known risk factors for AIDS: IV drug abusers (seven), prostitutes (two), Haitians/Dominicans (two), and homosexuals/IV drug abusers (one). There was no evidence that our patients had been sexually abused or given illicit drugs' (Oleske *et al.* 1983, 2347). Of the eight children, six had mothers 'who represented a risk factor' and this suggested 'the possibility of vertical spread of disease' (*loc. cit.*). The article, therefore, implied that, for the other two women, having sex with a person with known risk factors was not in itself a putative risk. Thus, while infection within the womb was suggested as a possibility for six of the infants, for the other two, with mothers who not themselves in what were thought to be risk groups but who were living with

and presumably having sex with men who were in such groups, nothing more specific than 'household exposure' was offered.

In an editorial for this issue of the *Journal of the American Medical Association* (JAMA), Anthony Fauci (1983, 2375) from the NIH concluded that the widening set of groups presenting with AIDS, meant that 'the evidence for a transmissible agent being the cause of aids is about as strong as it can be, despite the fact that, up to this point, no agent has been identified or isolated.' Fauci noted that transmission in the womb was a possibility but he went on to suggest that: 'Perhaps more important is the possibility that routine close contact, as within a family household, can spread the disease' (*loc. cit.*). This speculation was promoted in a press release from the American Medical Association and although Fauci later claimed (Shilts 1987, 301) that this sensationalized his speculation, his editorial was certainly inflammatory. The *New York Times* published the Associated Press (1983) piece quoting Fauci's phrase about 'routine close contact' as a possible cause of AIDS. Within a few weeks of the publication of the JAMA article, its author was back in the pages of the *New York Times* trying to dampen the fears of contagion: 'although the disease can be acquired other than sexually–through blood products or the birth process–Dr. Oleske stresses that "casual contact will not transmit AIDS"' (Gardner 1983, 21).

When the cluster study was finally published in March 1984, Dugas was not named but he still featured, this time as 'Patient 0' who had given

the CDC the names of 72 of what he estimated were his 750 sexual partners during three years 1979-81: 'Eight of these 72 named partners were AIDS patients: four from southern California and four from New York City' (Auerbach *et al.* 1984, 489). Patient 0 had marked lymphadenopathy from December 1979 and KS lesions from May 1980, and five of his eight sexual contacts preceded any of these symptoms leading the study to propose the 'existence of an aysymptomatic carrier state of AIDS' (*ibid.*, 490). To estimate the latency (or asymptomatic) period for AIDS, the authors highlighted six cases where only one other patient was a sexual contact. They then assumed that this must be the occasion of infection and then were able to estimate that the latency period, from infection to diagnosis with an opportunistic infection, was on average ten months. The press reports highlighted Patient 0, the man 'who carried AIDS,' 'spreading it across the country' (Associated Press 1984).

In 1984 it was accepted by most scientists that the retrovirus that caused the failure of the immune system had been identified, and by 1985 there was a blood test available that could detect antibodies to this virus. At this point, a remarkable retrospective study was undertaken (Jaffe *et al.* 1985). In San Francisco, some 6,875 gay or bisexual men had taken part in a hepatitis B study in 1978-80 and their blood samples had been retained at the CDC. Taking a 50% sample from the earliest part of the hepatitis B study (sera collected January-May 1978) and a 6% sample from the later part

of the study (men whose sera was collected June 1978-December 1980), 474 men were selected for follow-up blood tests yielding estimates of HIV prevalence in this cohort for 1978 (4.5%), 1979 (12.6%), 1980 (24.1%) and 1984 (67.4%). Yet in 1984 only 2.4% of the cohort showed evidence of the opportunistic infections that would trigger an AIDS diagnosis. For the men known to have been infected already in 1978-80, the median time thereafter before they developed opportunistic infections was 43 months. By August 1985, then, the cluster study published in May 1984 was known to be highly misleading. The latency periods identified in that earlier study were simply far too short and thus all the sexual contacts listed in the study were between pairs of men already infected with HIV, and that included those attributed to Gaétan Dugas. By the time of the very first report on inexplicable PCP, in June 1981, there was already an appreciable pool of infected men in San Francisco, and probably also in Los Angeles and New York.

This should have been the end of the road for the cluster study, but it was not. When Shilts was writing up his AIDS journalism into the first history of the early years of the epidemic, he retained the focus upon Gaétan Dugas. For Shilts, Dugas exemplified one half of the story of AIDS, and that was the half that was about the complicity of gay men in the transmission of the virus. Recent debates over homonormativity (Duggan 2002; Puar 2006; Visser 2007; Browne and Bakshi 2011; Brown 2012;

Gieseking 2013; Nash 2013) are prefigured in the distinction, suggested by Shilts, between good gays who could restrain their sexuality and bad queers whose unbridled sexuality threatened not only the reputation but also the lives of the good gays. Throughout *And the band played on*, Shilts (1987) stressed the vanity of Dugas, having him repeatedly look into the mirror to exclaim 'I am the prettiest one' (p. 21), 'I'm still the prettiest one' (p. 47), and 'still the prettiest one' (p. 79). Siding with Apollonian Larry Kramer (1989) rather than Dionysian Michael Callen (Berkowitz and Callen 1983; Paglia 1990, 1994), Shilts used Dugas to make a general point about hedonism deflating the earlier radicalism of the gay movement: 'Success was spoiling gay liberation' (p. 15). Shilts implied that indulgence had displaced responsibility within the gay community and that Dugas' promiscuity epitomized the ways that the gay movement 'had become a victim of its own success' (p. 15). To present Dugas in this way in 1984 would have been an unfair use of confidential data, but to do so in 1987 was to moralize on the basis of hypotheses long since tested and rejected.

Throughout his book Shilts presented as fact, the discarded guesses of 1983 and 1984. Shilts reported CDC officials discovering that one man developed symptoms 'some ten months after Gaétan spend the weekend with him on Thanksgiving 1980. Another Los Angeles man found his first Kaposi's sarcoma lesions thirteen months after he had slept with the French-Canadian during a trip Gaétan made to southern California in

February 1980' (p. 128). In the light of the science of 1985, these guesses from 1983 were just plain wrong. Both men were almost certainly infected long before 1980. Yet Shilts relies upon these speculations to establish his conclusion: 'Whether Gaétan Dugas actually was the person who brought AIDS to North America remains a question of debate and is ultimately unanswerable. The fact that the first cases in both New York City and Los Angeles could be linked to Gaétan, who himself was one of the first half-dozen or so patients on the continent, gives weight to that theory' (p. 439) Except that it does no such thing.

When St Martin's Press were trying to ginger press coverage for the book, they found indifference within the media, but decided, in the words of Michael Denneny, Shilts' editor, to descend to 'the worst kind of yellow journalism' and pitch the story to the media that Shilts had uncovered the man who had brought AIDS to America (Babineau 2001). Dugas became the story, given 'key role in spread of AIDS' (Associated Press 1987), and sensationalized as 'The appalling saga of Patient Zero' in *Time* (Henry 1987), and, even worse, achieving front-page notoriety with the *New York Daily Post* (1987) as 'The man who gave us AIDS.' Denneny had warned Shilts that 'You're not going to get on the "Today Show" with an attack on the Reagan administration' (Tiemeyer 2013, 175). In other words, the homophobia of the media was insatiable. But, as Crimp (1987) noted, Shilts' own conceptualization of the irresponsibility of promiscuous gay men was easily

assimilated into the homophobic agenda. McKay's (2014) study of Shilts' papers shows that the author chose not to use anecdotes about Dugas' consideration for others including one in which Dugas turned down sex after a dinner date, saying 'We can't. … It won't work out. I can't say any more' (p. 183). The elision of this story 'removed any ambiguity from Dugas's motivations, and strengthened the image of the flight attendant as a deliberate disease spreader' (*loc. cit.*).

Epidemiology and Stereotypes

As Treichler (1987) remarked, the early association between gay men and the new disease persisted despite the increasing diversity of the sufferers. The pervasive homophobia of US society in the early 1980s shaped how epidemiologists communicated their work producing a vagueness in terminology (bodily fluids, contact) that fed anxiety without developing coherent prevention strategies. The epidemiological obsession with the gay lifestyle, that persisted long after the unique focus upon gay men had ceased to be a plausible explanation for the dynamics of the epidemic, further prejudiced effective prevention policies. It fed such irresponsible reporting as the psychiatrist who, over two years after the CDC had clearly concluded that the virus could be transmitted 'from heterosexual men to their female sexual partners' (CDC 1985), told the readers of *Cosmopolitan*, that that there was no credible threat to heterosexual women from what he described as

sex in the 'missionary position' (Gould 1988). It was activism on the part of the Women's group within ACT UP New York that challenged this and that eventually forced the CDC (1992) itself to revise its case definition of AIDS so that female conditions such as invasive cervical cancer became recognized as relevant opportunistic infections (Shotwell 2013).

The presentation of sexual activity as lifestyle made it seem a relatively insignificant consumer choice and it delayed serious consideration of what people invest in sexual expression and of how to accommodate those desires and fears within effective prevention strategies. Here, again, it was activists leading the way in the development of effective epidemiological knowledge and within two years of the first cases, Berkowitz and Callen (1983, 1-2) had articulated the central theme of practical prevention, insisting that '[s]ex doesn't make you sick–diseases do,' they accepted that the 'challenge is to figure out how we can have gay, life-affirming sex, satisfy emotional needs, and say alive!' Such a pro-sex message was not compatible with the homophobia that treated gay men as sick by virtue of depraved choice.

While epidemiologists debated the relative merits of theories based either on lifestyle or on the presence of an infectious agent, the homophobic context meant that both were grist to the mills of prejudice. If it was lifestyle, this confirmed the dangers of homosexuality. If it was contagion, this confirmed the dangers of homosexual men. Truly, this was a distinction

without effective difference. Only this sickening homophobia could so conflate gay men with sickness that when Ryan White, an adolescent with hemophilia, contracted HIV from a transfusion following an operation to remove part of a lung damaged during a bout of pneumonia, not only did other parents try to exclude him from the school of their children, but he found his school locker defaced with such graffiti as 'fag' (White 1988).

Notions of sickness and wellness are irresistible as metaphors for all sorts of social issues. When a new medical crisis irrupted into the richest state on earth, epidemiology was so readily colonized by homophobia that the medical response was woeful and public debate toxic. We might speak of epidemiology having been homosexualized when instead it needed to be queered.

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