

EDITORIAL

Has the United Kingdom averted an epidemic of HIV-1 infection among drug injectors?

The United Kingdom has not seen the major spread of HIV-1 infection among drug injectors that was anticipated in the mid-1980s. Although cities in many countries have seen rapid spread of HIV-1 infection to epidemic levels (40% prevalence and above), the United Kingdom is one of several countries that experiences low and stable prevalence rates. Since the mid-1980s there have been major changes in syringe-sharing risk behaviour, and evidence that specific preventive interventions have been successful: a plausible case can be made for the success of public health interventions in averting a potential public health disaster.¹

Ten years have passed since the first reported outbreak of HIV-1 infection among drug injectors in the United Kingdom. In 1985 it was discovered that at least 50% of injectors tested in Edinburgh were HIV positive,^{2,3} and about 40% in Dundee;⁴ there were reports of HIV-1 infection among injectors in other areas, and evidence of high-risk syringe-sharing behaviour.^{5,6} Heroin use and injecting had spread in the previous decade in many major cities in the United Kingdom, with estimates of about 100 000 injectors throughout the United Kingdom.⁷ By 1986 HIV-1 prevalence rates among drug injectors ranged between 44% and 76% in eight European cities, and between 50% and 72% in New York and New Jersey.⁴ The situation in 1986–87 suggested that if the Edinburgh experience were repeated throughout the United Kingdom there might be potentially 50 000 injectors with HIV-1 infection.

This has not happened. The cumulative numbers of injecting-related HIV-1 infections known from voluntary testing in the United Kingdom to the end of March 1995 totalled 2703 (of which 1054 were in Scotland, 1633 in England and 16 in Wales).⁸ Latest projections (including esti-

mates of the untested population) for England and Wales are for about 1800–2300 living injecting-related HIV-1 infected people to the end of 1994.³⁰ Studies undertaken in various sites, including samples in and out of treatment, show that in England (outside London) the prevalence rate among current injectors is less than 1%.⁹ In London, multisite studies of current injectors indicate a stable rate of about 7%.¹⁰ In Glasgow the rate is 1%.¹¹ Previous high levels in Edinburgh have now been succeeded by rates of about 20% in subsequently tested samples.¹² Low prevalence of HIV-1 infection has been sustained even though HIV-1 infection has been present in many areas since the early 1980s. By the end of March 1995 there had been a cumulative total of 614 cases of AIDS attributed to drug injecting in the United Kingdom (of which 229 were in Scotland).⁸

Elsewhere, the period up to 1995 saw the continuation of high prevalence rates in New York and in many European cities.^{13,14} South East Asian cities have reached a prevalence rate of 40% or more within 1 year of identification of first infections, and with some cities having peak prevalence rates of over 80%.¹⁵ Rapid spread of HIV-1 infection to high prevalence rates within 1 year of introduction is not historically unique. It then raises the question of why the United Kingdom has avoided this scenario.

Most of the UK preventive interventions were introduced when prevalence was low (except in Edinburgh and Dundee). The key period was 1987–77.¹ The first report on *AIDS and Drug Misuse*⁷ from the Advisory Council on the Misuse of Drugs brought together and legitimized new ideas about working with drug-injecting populations. The key target was current injectors, who were unable or unwilling to stop injecting. Ideas of service accessibility, flexibility of

service delivery, multiple and intermediate goals for treatment, and—most significantly—of harm minimization, were introduced.¹⁶ There was a rapid development of syringe distribution and exchange, an expansion of methadone treatment and an expansion of outreach to less accessible populations. Some of these interventions were evaluated. Use of syringe exchange was associated with lower risk behaviour;^{17,18} outreach was shown to be useful in reaching target populations;¹⁹ no studies of methadone treatment were undertaken but extrapolation of evidence from the United States and Australia indicates that appropriate delivery of methadone treatment can be effective in reducing risk behaviour.²⁰

Numerous reports point to reductions in syringe-sharing risk behaviour (summarized in^{5,6}). Even though all studies rely on self-report, triangulation of data from studies using different methods and from various sites suggest that major risk reduction has occurred. There has been increased use of sterile syringes and syringe cleaning, reductions in the frequency of equipment-sharing and the number of sharing partners, and a restriction in the range of sharing partners (the last two are important for epidemic dynamics because they reduce the level of population mixing).⁵ The main changes in risk behaviour probably occurred between 1986 and 1990, with less reduction since; safer injecting practices appear to have become the “norm” in most contexts. Although risk reduction has occurred it has not been eliminated; syringe-sharing continues between sexual partners and close friends, and occurs even among HIV-1 positive injectors;²¹ “non-sharing” behaviours may carry risk of transmission (e.g. indirect sharing through other drug use paraphernalia;²² and some settings are riskier than others (e.g. prisons^{23,24}). There has been little change in sexual behaviour.²⁵

Difficulties must be acknowledged in inferring links between epidemic dynamics, population behaviours, preventive measures and policy direction, particularly bearing in mind that we are still at an early stage in the unfolding of HIV epidemics world-wide. On the face of it, there is evidence to support the hypothesis that preventive interventions facilitated behaviour change, and that this in turn led to the maintenance of low and stable HIV-1 prevalence rates. Various other hypotheses may be entertained.^{1,26} Genetic variation in population susceptibility to HIV-1,

or difference in virulence of strains of HIV-1, seems an unlikely explanation given the experience of the outbreak of infection in Edinburgh. Population sampling has been sufficiently varied and widespread to rule out sampling bias. Questions can be raised about the interpretation of prevalence rate data, which are affected by population dynamics (changes in the size of the drug injecting population), by new cases of infection and by loss of existing cases of infection. There is no information on whether the size of the population of injectors has increased in this period (Addicts Index notifications show more injectors, but these data bear an unknown relationship to the total population).²⁷ Even a two-fold increase in population size would only mask a doubling of the absolute prevalence of infection, other things being equal. This would still leave the United Kingdom with extremely low prevalence of infection.

While prevalence rates for HIV-1 infection are low, those for hepatitis B (at about 40%) and hepatitis C (at about 60%) are much higher.²⁸ This raises questions about the behaviour change hypothesis and calls for further research on the interaction between viruses, behaviour and epidemic dynamics. Differences in prevalence rates might be accounted for by: dates of viral introduction (hepatitis B has been around for longer than HIV-1, as is probably the case for hepatitis C); dates of peak prevalence in relation to introduction of risk reduction; and levels of infectiousness and of transmissibility. Reductions in risk behaviour (though not risk elimination) may be sufficient to reduce spread of HIV-1 infection in a situation of low prevalence, where there are few highly viraemic individuals (HIV viraemia being highest immediately following infection), and with a virus of relatively low transmissibility. Greater risk reduction is required when prevalence, viraemia and transmissibility are higher. In the United Kingdom, HIV preventive interventions occurred when prevalence rates were low in most areas. The United Kingdom probably benefits by an interaction between lowered risk behaviours and low prevalence. It has avoided that interaction between high risks and high prevalence which sustains HIV-1 epidemics at high levels in other locations.^{15,29}

Caution in the interpretation of data is still needed, because we have only a short history of responding to this virus, and still know relatively little about the interaction between epidemic

dynamics, social behaviour and prevention policy. Further international comparative work is needed to compare cities with different levels of epidemic spread, risk behaviour and prevention strategies. If the case proposed in this editorial should prove to be correct, then the international significance of the UK experience would point to the importance of HIV-1 prevention investment before or at an early stage of viral diffusion. However, HIV-1 prevention is not a one-off activity. The message to the UK government is *not* that the epidemic never happened and that resources may be directed elsewhere; rather, that evidence to date suggests that prevention investment has paid off, that public health prevention works with this population, and that this needs to be sustained in order to preserve the beneficial situation in which we find ourselves.

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