renew its longstanding commitment to maintain open access services, and provided valuable data to augment our efforts to expand consultant numbers.

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1 Report prepared by Public Health Laboratory Service Communicable Disease Surveillance Centre, Association for Genitourinary Medicine and The Medical Society for the Study of Venereal Disease. *Survey of Patient Access to GUM Clinics in the United Kingdom.* (Submitted for publication.)

Ethnicity and STIs: more than black and white

Because of its close links to behaviour, the epidemiology of sexually transmitted infections (STIs) involves forays into social science research. One of the most vexing problems has been defining the relation between ethnicity and STI risk. Defining these associations, even when methodologically carefully performed, is problematic because of the historical context of discrimination in both the United States and Europe. However, not dealing with these issues in a forthright manner may have profound public health consequences.

Population based cross sectional studies in the United States have demonstrated increased rates of gonorrhoea, chlamydia,[1] and genital herpes[2] in African-Americans. The herpes studies are particularly instructive because they were based on a national sample—and the differences persist when controlled for socioeconomic status and other demographic variables. The differences are also stable over time. In the United Kingdom, studies have shown that gonorrhoea rates in Leeds,[3] Birmingham,[4] and south London[5] and chlamydia rates in Coventry[6] and Birmingham[4] were substantially higher in black residents, again after controlling for socioeconomic status, and in an environment (in contrast with the United States) where there is universal access to free health care.

Commenting on the papers by Low *et al*[5] and Lacey *et al*,[3] Raj Bhopal[7] cautioned us to be prudent in using ethnicity data because of the historical propensity to marginalise and discriminate against minorities, but reminded us not to shirk from our responsibilities in protecting public health. Ethnic classification systems invented for one purpose, such as census monitoring, may not be adequate to explain differences in health. Pfeffer developed a trenchant critique of this essentialist view of ethnicity, where culture is presented as a fixed product and all members of a defined group are assumed to share a stereotypical "true" identity and biology: "black" versus "white." This is as problematic for the dominant group as for recognised minorities.[8] For example, in the United Kingdom an apparently homogeneous "white" ethnic group conceals many minorities subject to discrimination and disadvantage, such as the Irish.[9] In the United States the development of an integrated syphilis elimination programme is a model for an appropriate response. This programme is based on integrating community based organisations, religious leaders, and outreach programmes with medical providers, and creating multiple forums for the sharing of epidemiological data, community concerns, perceptions, and ideas.

With this background, we must welcome the paper by Low *et al* in this issue of *STI* (p 15), which attempts to go beyond studies on "black everybody" to the "black specific" and provide epidemiological data which highlight Bhopal and Pfeffer's concerns. Specifically, they demonstrate that in terms of gonorrhoea risk, black ethnicity is a complex variable. People with Caribbean ancestry have substantially higher risk than those with African ancestry—even in multivariate analyses. These are resonant themes. For example, one would argue that in terms of HIV risk, the ratios would be reversed.

Yet the issues they raise are complex and constrained by venue and history. A study by DeHovitz *et al* conducted in Brooklyn, New York, in the early 1990s[10] demonstrated that African-Americans who were Caribbean immigrants (or first generation) had substantially *lower* rates of STDs, and drug using behaviour, compared with native born US African-Americans. DeHovitz's work shows the complex interaction of ethnicity and socioeconomic status in a world of global migration spanning several centuries. For example, if Low *et al* had conducted their study in an area where the African immigrant population has been predominantly from east Africa rather than from west Africa, then lumping all people born in Africa as "black African" would have combined African born "blacks" with those of "south Asian" origin who may, or may not, have similar sexual behaviour. Moreover, you cannot classify the latter with "south Asians" of Asian origin—at least not when it comes to behaviour. A Trinidadian of Asian origin three or more generations ago may have more in common with a Trinidadian of "African" origin than an Indian in India. Finally, what has a villager from Bangladesh in common with a middle class professional from Dacca?

The diversity of results in Low *et al*'s study confirms the traditional public health approach—that we cannot look at disease in isolation from the social and cultural context of our patients' lives. The whole issue of ethnicity is more complicated than we imagined![11] Yet, having defined the problem in a number of model settings, we need to be able to expand our ability to define sociological/behavioural risk and to develop sensitive and appropriate intervention strategies. Before we can do this, however, we have to define more clearly the mechanism of the relation between social groups and STIs. In this expanded model ethnicity is one factor alongside many others determining sexual behaviour.[12]

With the possible exception of bacterial vaginosis (which actually may be a behavioural-cultural effect rather than true biological susceptibility),[13] there is no known biological susceptibility differences between ethnic groups. Traditional scalar measures such as demographics, number of sexual partners, and socioeconomic status do not explain the difference either. Geographic residence appears to be a factor—although careful studies beyond empirical descriptive analyses have not been done.

Laumann and Youm[1] and Rothenberg *et al*,[14] and Stoner *et al*[15] have to date provided the most coherent explanation for these phenomena. Rothenberg *et al*, in a series of

elegant social network studies, demonstrate that the immediate social network is the key determinant for STI risk. They suggest that in groups with high STI risk, partner concurrency is higher, even though the number of partners over time may be similar. This *qualitative* difference—that is, partner concurrency, leads to the more efficient transmission of STIs within a social network when those infections are introduced into one of the constituent relationships. Laumann and Youm, using national survey data from the United States, contend that the STI risk is related to partner concurrency and the intergroup mixing pattern. Interventions to address these types of transmission modes must therefore incorporate innovative approaches to screening (in terms of identifying truly marginalised people) and partner notification. Community involvement and cooperation in these approaches are absolutely necessary.

What to make of all this? In many ways this mirrors the condom debate (abstain—but if you don't, use condoms). We need to recognise the complexity and the occasional irrationality of the ethnicity variable. We should beware of simplistic "ethnic groups," forcing clients to self identify into a group that is irrelevant to their situation, ensuring instead that non-standard responses are also allowed and are used to inform the future construction of ethnic categories.[16] Sexual behaviour is perhaps the most culturally delimited behaviour. The use of skin colour as surrogate for ethnic-cultural sexual behaviour is clearly problematic. However, ignoring the relation between ethnic group, disease incidence, and social structure denies the important first step in developing prevention interventions—understanding the descriptive epidemiology. Bhopal,[7] Fullilove,[17] and others[8 9 16] have provided important historical perspective, philosophical guidance, and the admonition to understand the issues without stigmatisation or discrimination. Having assimilated these, Low *et al* and the other researchers in this field have provided important guidance in the field approach to the problem.

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