Sexually Transmitted Infections

Editorial

Patterns of sexual mixing: mechanisms for or limits to the spread of STIs?

Prevalence and incidence of sexually transmitted infections (STI) vary across subpopulations defined by age and race ethnicity.[1–5] Some ethnic groups—for example, African and Caribbean blacks[6] in the United Kingdom and African-Americans[1] in the United States, have higher rates of STI including human immunodeficiency virus (HIV) infections, while other ethnic groups—for example, Asians in both the United Kingdom and the United States, have lower incidences of STI and AIDS. The reported incidence and prevalence rates often exaggerate the race ethnicity differentials in STI. In the United States the majority of the population seek STI related health care through private healthcare providers,[7] while the number of STI cases reported from public sources exceeds that reported by private providers by a wide margin.[1] To the extent that minority race ethnicity subpopulations seek STI related health care through public facilities, STI cases among these groups are overreported in the national data. Surveillance systems tend to collect data on either race ethnicity or socioeconomic status; thus, often it is impossible to analyse the effects of both sets of variables on STI incidence. Moreover, the multicollinearity between race ethnicity and socioeconomic status makes it difficult to delineate the independent contribution of ethnicity to differentials in STI rates even in those rare cases where data on both socioeconomic status and race ethnicity are available. Nevertheless, it seems clear that even after controlling for the effects of overreporting and confounding by socioeconomic status, racial ethnic differentials in STI rates persist.

The mechanism of action for the association between race ethnicity and STI is difficult to identify. Delayed healthcare seeking and failure to use condoms do not seem to account for the observed pattern.[8] Differentials in numbers of sex partners tend to suggest either no race ethnicity difference in STI or a differential in reverse direction from that observed,[2 3 9] at least among women. Sexual mixing patterns have been suggested as a possible mechanism of action. Some have suggested that "in the presence of raised levels of undetected sexually transmitted diseases, assortative mixing may make ethnicity an important determinant of incidence of sexually transmitted diseases" and sexual mixing patterns may account for the higher rates of STI among racial ethnic minorities.[6] In light of recent data that show high rates of asymptomatic incident STI in the United States,[10] this suggestion may be relevant in many epidemiological contexts. Others have shown that,[11] based on analyses of sexual behaviour data collected on nationally representative samples of the American population, (a) the relatively high frequency of sexual contacts between the African-American core and its periphery help spread STI into the entire African-American population—referred to as "the intraracial network effect," and (b) STI remain inside the African-American population because African-Americans are sexually segregated from other racial ethnic groups—referred to as "the interracial network effect." These findings support the view that assortative mixing fuels the spread of STI in the subpopulations; they also suggest that assortative mixing prevents the spread of STI to other subpopulations.

These conclusions may seem counterintuitive. A number of studies in the literature, many of which did not measure biomedical markers of STI, suggest that mixing across subpopulations may contribute to spread of STI in the population, particularly across subpopulations.[12–14] The purely behavioural measures employed in these studies may not be sufficient to support their main thesis that mixing across high and low prevalence subpopulations would facilitate faster spread of STI through the population. Some studies have demonstrated, in empirical terms, the contribution of sexual mixing to faster spread of HIV. For example, mixing between older and younger age groups of men who have sex with men (MSM) has been shown to contribute to faster spread of HIV.[15] The exact nature of the impact of sexual mixing across subpopulations on the rate of spread of STI may depend, in part, on the demographic characteristics of the subpopulations (for example, relative sizes). In addition, whether the sexual mixing takes place between members of the core groups in each subpopulation or between members of the periphery in each subpopulation may also affect the impact of mixing on the rate of STI spread. Thus, sexual mixing across racial ethnic subgroups may not have the same impact on rate of spread of STI as sexual mixing across age groups.

In a recent study conducted in Seattle we found that most of the disease burden for gonococcal and chlamydial infections in both high prevalence and low prevalence subpopulations was attributable to mixing within the subpopulations.[16] A relatively smaller burden of disease was attributable to direct mixing between high and low prevalence subpopulations, with the proportion of infection attributable to indirect mixing, or so called "bridge populations," being remarkably small.[16] While we found that sexual mixing between particular racial ethnic subpopulations increased the risk of STI significantly, the proportion of the population engaging in sexual mixing, and the numbers of sex partners reported by individuals engaging in sexual mixing across racial-ethnic subpopulations were too low for this increased risk to play a major part in disease burden.

The literature on racial-ethnic differentials in STI rates and the role of racial ethnic mixing on the spread of STI is emergent; many questions still remain unanswered. In order to define the enhancing or limiting role of sexual mixing in the spread of STI in specific epidemiological contexts, we need to know the answers to many, if not all, of the following:

- How big is the differential in STI rates between the sub-populations concerned?
- How much sexual mixing takes place between the high and low prevalence subpopulations? In both absolute and relative (to size of population) terms?
- What proportion of sexual mixing is direct? What proportion is indirect, through sexual bridges?
- What are the relative risks associated with distinct types of sexual mixing?
- What is the population attributable risk associated with distinct types of sexual mixing in different epidemiological and social contexts?

As future research further improves our understanding of the role of sexual mixing across racial-ethnic subpopulations in fuelling or limiting the rate of spread of STI, it is important to remember that the main programmatic goal is to eliminate the racial-ethnic disparities in the prevalence and incidence of STI.

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