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HIV and hepatitis C epidemics in Africa: continuing the debate

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Authors of recent communications (1-3) conclude that the apparent lack of parallel transmission between human immunodeficiency virus (HIV) and hepatitis C virus (HCV) supports the dominant view that the HIV burden in sub-Saharan Africa is driven by heterosexual transmission and that unsafe health care is not an important factor (4). They argue that because both viruses have similar parenteral transmission (i.e., by puncturing the skin), their epidemiologic patterns should be similar and that this is not the case in Africa (3,5). Here, we respond to the authors' criticisms.

The principal weakness of their argument lies with the assumption that the two viruses have similar parenteral transmission. The authors do not distinguish between different forms of parenteral exposure, i.e., intravenous, intramuscular, subcutaneous, or intradermal. Recent information suggests that HCV may not be efficiently transmitted intramuscularly. Conversely, HIV RNA (but not HCV RNA), possibly from interstitial fluid (6), has been recovered from syringes after intramuscular or subcutaneous injections of infected individuals (R. Heimer, personal communication, 10 June 2003). Should this be confirmed, one would not expect that a pathogen transmitted by contaminated intramuscular injections — the mode we suspect explains a substantial part of HIV transmission in Africa — would follow the same epidemiologic pattern as HCV transmitted by intravenous injection. Kallestrup and colleagues (1) note that HCV "has shown a remarkable capacity for spreading by unsafe injections" and advance supportive evidence in the "large HCV epidemic in Egypt associated with parenteral anti-schistosomal therapy," but they omit that therapy was administered intravenously (7). We are unaware of any major outbreak of HCV attributed to intramuscular, as opposed to intravenous, transmission.

Relying on an early estimate of HCV transmission efficiency (1.8% for needlestick exposure), the authors contend that HCV prevalence should be higher than HIV prevalence if both viruses were transmitted iatrogenically. This expectation seems unwarranted in light of the recently estimated

0.5% probability of HCV transmission from needlestick exposure, derived from a large international series (8). This revised estimate is similar to the 0.3% probability of transmission for HIV occupational exposures (9). In testing their expectation, Walker and colleagues (3) used data that are neither comparable nor representative. The African prevalence ratios they reported were constructed from reports that compared old and new data on dissimilar populations (3): The 1999 HCV estimates from the World Health Organization (WHO) are based on earlier published reports, mostly stemming from convenience samples surveyed between 1986 and 1995 (5), while the 1999 HIV estimates from UNAIDS (the Joint United Nations Programme on HIV/AIDS) stem from systematic and recent sentinel surveys. In addition, their presentation of stable low prevalence in South Africa between 1990 and 1999 (3), a period of rising HIV prevalence, is based on data from populations not comparable from year to year and excludes available information from other samples that report higher HCV prevalence (10-13).

No conclusions can be confidently drawn from dissimilar ratios of HIV to HCV prevalence alone. Ratios vary in different regions for various reasons, including the date that each virus was introduced, the local patterns of parenteral risk of exposure (e.g., injection drug use in developed countries, contaminated medical injections and other health care procedures in Africa, etc), non-parenteral transmission, frequent transience of HCV infection, and testing artifacts. For example, HCV antibody tests may have relatively low sensitivity for HCV genotypes that are common in Africa. In a recent study, HCV RNA was isolated from 13 of 173 South Africans, all 13 of whom were negative for HCV antibody on third generation enzyme immunoassays (13). HCV antibody tests have also been reported to miss as much as 39% of HCV infections in persons co-infected with HIV (14), which would attenuate the observed tendency toward co-infection (1). In a Seattle study, the majority of HCV infections caused by needlestick injuries to health care workers were quite short-lived, with no HCV RNA detectable a few months later (J. Schwarz, personal communication, 11 September 2003). Lastly, even among injecting drug users, the ratio of HCV to HIV prevalence varies substantially in different geographical locations, indicating again the loose association between HCV and HIV prevalences (15-16).

Health care providers administer hundreds of millions of unsafe injections to Africans each year (17). Among WHO regions, Africa has the highest prevalence of HCV (5.3%) (18), hepatitis B virus (HBV, 70%-95% lifetime exposure) (19), and HIV. Although we do not have sufficient data to accurately identify the different parenteral risk factors for HCV, HBV, and HIV infection in Africa, we do have sufficient data to warn Africans about skin-puncturing exposures in healthcare settings. Finally, the contribution of health care to HCV, HBV, or HIV transmission will not be clarified by retrospective, ecologic analysis of unsynchronized data in unconnected populations. New studies, with simultaneous attention to multiple blood-borne viruses and to specific parenteral and other risks in different African countries, would be of considerable value in elucidating modes of transmission for each virus.

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