Letters to the Editor

was categorized as low, moderate, high, or severe. We developed a 20-item scale to determine categories and severity of HIV risk. Risk categories included sexual, drug use, and condom use behaviors of the abuser and respondents' self-reports of STDs during their abusive partnerships.

Experiences of high to extreme levels of severity were estimated for physical (42%), psychological (70%), and sexual (53%) forms of abuse. Sixty percent of the women reported that their abusive partner was a current user of illicit (opiate-form) drugs, 63% reported that their partner had other sexual partners, 98% reported that their partner never or rarely used a condom, and 50% reported that their partner had infected them with at least 1 form of STD. Three quarters of the women were at multiple risk of HIV infection.

Multiple HIV exposure risk was strongly correlated with high to extreme levels of physical abuse ($\chi^2=176.5$, $P<.001$), psychological abuse ($\chi^2=261.0$, $P<.001$), and sexual abuse ($\chi^2=43.0$, $P<.001$). Measures of association demonstrate that psychological abuse was most strongly associated with multiple risk of HIV exposure.

Eighty-five percent of the women who were at "multiple" HIV risk did not perceive themselves to be at risk all. Eighty percent had "never" asked their abusive partner to wear a condom. The respondents' inability to realistically assess their vulnerability to this pandemic disease is consistent with the failure of individuals diagnosed with acute stress disorder to recognize their basic health and safety needs.

Our figures may suggest a stronger relationship between abuse and HIV risk than is the case with Wingood and DiClemente's findings. We hypothesize that HIV exposure is more evident among chronically and severely abused women electing to live in a battered women's shelter than among abused women who still live with their partners. It is essential that programs serving victims of domestic abuse respond to the threatening intersection between domestic battering and HIV transmission. Finally, it is imperative that this threat be recognized as one that transcends all racial and socioeconomic boundaries.

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Wingood and DiClemente Respond: Unanswered Questions Remain

Drs Molina and Basinait-Smith are to be applauded for addressing an issue that remains understudied, namely, the association between domestic abuse and HIV risk. The investigation by Molina and Basinait-Smith corroborates the findings from our research, which focused specifically on HIV risk taking among African-American women having a physically abusive primary partner. Also, their study extended our findings by examining HIV risk exposure in an ethnically and socioeconomicly diverse sample of women residing in battered women's shelters. In addition, the authors examined the effects of sexual and psychological abuse, as well as physical abuse, on women's HIV risk. The findings of Molina and Basinait-Smith indicate that high to extreme levels of physical, psychological, and sexual abuse are correlated with multiple HIV risk.

While Molina and Basinait-Smith's study augments our previous paper, the impact of abuse on HIV-related risk needs to be addressed among a much larger sample of women. For example, the independent effects of the severity, frequency, and chronicity of abuse and women's HIV risk are still unknown. The relationship between type of abuse and HIV risk among women of different ethnicities has not been fully examined. Equally important, there are scant empirical data on the factors that mediate the relationship between abuse and HIV risk. Furthermore, studies need to go beyond self-reported data and examine the influence of abuse on actual adverse health outcomes such as acquisition of HIV and other sexually transmitted diseases among women in abusive relationships.

Helping women deal with the threat and experience of partner abuse is an essential element in reducing their risk of HIV. The findings of Molina and Basinait-Smith, as well as our own research, challenge public health officials and researchers alike to begin greater surveillance of partner abuse and its adverse sequelae. Also, public health practitioners should earnestly address the need for designing interventions to reduce the risk of HIV among survivors of abuse and among women currently involved in abusive relationships. While many questions remain unanswered, it is clear that the intersection between abuse of women and their risk for HIV represents an important area for further prevention research.

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Salicylate Intake and Cardiovascular Disease: Ingster and Feinleib Respond to Hu and Willett

We are pleased to respond to the comments by Hu and Willett about our suggestion that the per capita intake of salicylates may have increased during the last 50 years to a level that may have affected the decline in cardiovascular disease mortality. First of all, the main thrust of our paper was the amount of synthetic salicylates produced and used in the food, drug, and cosmetic industries of the United States. The estimates by Swain et al. were in addition to our estimates. However, we agree with Hu and Willett that the salicylate content of foods remains controversial. We stress that the methodology to accurately measure the salicylate content of foods or to estimate salicylate exposure on the basis of urine samples appears to be still in its infancy.
Hu and Willett cite a study by Janssen et al., which is based on a very small sample of 17 volunteers, from 14 countries and 4 continents, who consumed highly varied diets. Such results must be approached with great caution. The data for the 4 volunteers reported by Janssen and colleagues indicate that salicylate intake estimates obtained by all methods except that of Swain et al. were accurate when urinary excretion measurements were low but were gross underestimates when urinary excretion levels were higher. Considering that Janssen et al. claim to have recovered 80% of orally administered aspirin or salicylic acid, the underestimate may be even greater.

It is inaccurate to claim that salicylates do not affect thromboxane \( B_2 \) formation and platelet aggregation. They do.\(^\text{5,6}\) The difference is that aspirin inhibits platelet aggregation \textit{irreversibly}, whereas the other members of the salicylate family have a reversible effect. Even Roth and Calverly\(^\text{7}\) note that distinction. Of interest to us are the findings of Williams et al.,\(^\text{8}\) which clearly demonstrate salicylate inhibition of platelet aggregation and also suggest that other, nonsalicylate additives convey \textit{additive} inhibition of platelet aggregation when combined with salicylates, albeit at higher concentrations (although the authors point out that the doses used are doses contained in many diets). Williams et al. suggest that other additives may bind to different sites on the cyclo-oxygenase enzyme, whereas salicylates compete with aspirin for the same site. The fact that the effect is reversible may be moot if one is exposed to a consistent, daily, low dose.

To the best of our knowledge, no one has tested the effects of the synthetic salicylates (amyl-, benzyl-, methyl-, etc.) on platelet aggregation. Yet even this information would not be conclusive, for as Fuster and colleagues state, “the powerful antithrombotic effect of aspirin seems out of proportion to the inhibition of the rather weak platelet thromboxane pathway.”\(^\text{9(p661)}\)

Much is still unknown. It may be the anti-inflammatory effect, the anti-bacterial effect, the anti-protein C effect, some combination of the above, or some as yet unknown property of aspirin that provides the bulk of the protection.

We feel that the role of salicylate intake in relation to mortality from cardiovascular disease is far from settled, and much more research is needed. 

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