Pathogenesis of HIV disease. More on co-infections.

June 26, 2010 By Joseph Sonnabend, MD

A few posts ago I wrote about interactions between HIV and some other infections caused by a variety of microorganisms, including viruses, bacteria and worms. Some of these co-infections can enhance HIV replication and HIV in turn can accelerate many of them, setting the stage for a vicious cycle or a positive feedback interaction, as I tried to describe in a previous post.

The deadly combination of TB and HIV disease is perhaps the clearest example of this mutually enhancing effect.

The importance of immune activation in driving HIV disease is well established, and as I described many infections that promote HIV replication can contribute to immune activation, although there are other mechanisms by which some enhance HIV replication.

We began to understand that HIV replication could be enhanced by some other infections in the late 1980s, and the particular relevance of some tropical infections was emphasized as early as the 1990s. As noted in previous posts on endemic infections in Africa and herpes infections worldwide, HIV replication can be modified by some concurrent infections, some of which in turn are modified by HIV.

The reason I'm returning to this topic is that today I saw an article in the current Lancet Infectious Diseases entitled **"Effect of treating co-infections on HIV-1 viral loads: a systematic review".**

Here is the abstract:

Co-infections contribute to HIV-related pathogenesis and often increase viral load in HIV-infected people. We did a systematic review to assess the effect of treating key co-infections on plasma HIV-1-RNA concentrations in low-income countries. We identified 18 eligible studies for review: two on tuberculosis, two on malaria, six on helminths, and eight on sexually transmitted infections, excluding untreatable or non-pathogenic infections. Standardised mean plasma viral load decreased after the treatment of co-infecting pathogens in all 18 studies. The standardised mean HIV viral-load difference ranged from -0 04 log_{10} copies per mL (95% CI

-0.24 to 0.16) after syphilis treatment to -3.47 log_{10} copies per mL (95% CI -3.78 to -3.16) after tuberculosis treatment. Of 14 studies with variance data available, 12 reported significant HIV viral-load differences before and after treatment. Although many of the viral-load reductions were 1.0 log_{10} copies per mL or less, even small changes in plasma HIV-RNA concentrations have been shown to slow HIV progression and could translate into population-level benefits in lowering HIV transmission risk.

This review makes it quite clear that we cannot look at HIV/AIDS in isolation from other infections, and therefore from those conditions that facilitate their acquisition and promote their progression.

The Lancet Infectious Disease review deals with published studies that looked at the effect on HIV of treating several different co-infections. There is nothing on herpes viruses other than herpes simplex virus. As noted in my post on positive feedback interactions, herpes viruses, and CMV in particular are probably major contributors to the pathogenesis of HIV disease worldwide, as are a number of endemic infections in parts of Africa.

The Lancet Infectious Diseases article is explicit in suggesting that control of common endemic infections could make an impact in our efforts to control HIV.

Traditional public health measures, such as the provision of sanitation and clean water are therefore perfectly appropriate in the fight against AIDS, and of course will independently improve the health of populations.

I'll end with something I wrote elsewhere.

The connection of so many endemic infections with AIDS in Africa is also a connection of poverty with AIDS. The acquisition of many endemic infections is facilitated in impoverished populations resulting in lives that are ravaged and shortened.

Many of these infections also interact with HIV to compound the devastation they cause. Poverty, multiple endemic infections and HIV are intimately intertwined and in many instances reciprocally affect and reinforce each other.

If our goal is to improve the health of populations, where multiple infections commonly co-exist, we cannot adequately manage any one of them without also considering the other infections that may be present, both in the individual and in the community.

© 2026 Smart + Strong All Rights Reserved.
http://beta.docker.poz.com/blog/pathogenesis-of-hiv