Saved by the Cell

Sean’s CD4’s are kickin’ ass. Can he kiss his prophylaxis goodbye?

July 1, 1998 By Interview by Lark Lands

Laboratory analyses of blood and other medical measurements, which help health practitioners make diagnoses and detect toxic effects of medication, can also help people with HIV track their health. Fred Valentine, MD, professor of medicine at New York University and a leading HIV immunologist, analyzes the lymphocyte proliferation assay results of POZ *founder Sean O. Strub.*

Sean’s highly active antiretroviral therapy (HAART -- currently Crixivan [indinavir], delavirdine [Rescriptor] and d4T [Zerit]) has produced a dramatic increase in his CD4 cell counts, from 1 in 1995 to this spring’s 357 (not to mention a viral load reduced from 3.3 million to 28 copies). But CD4 numbers alone can’t tell us how well his immune system is restoring itself. The return of immune competence is a function not only of increased CD4 cells but of how well they work. An important marker is the recovery of previously lost immune responses to specific pathogens (bacteria, fungi and viruses). Sean’s results on the test used to measure these responses -- the lymphocyte proliferation assay (LPA) -- show that his immune cells are doing well.

To do an LPA, the patient’s lymphocytes (CD4, CD8 and B cells -- see “Coming to Terms” below) are placed in a tissue culture to which various antigens (portions of pathogens) are added. If the lymphocytes “recognize” them, the immune cells proliferate -- rapidly divide -- creating many more cells to fight that pathogen. This process takes place in anyone with competent immune function, but is lost in HIV disease progression, creating increased susceptibility to pathogens. The hope is that, with sufficient time on HAART, this immune-cell response will be restored to normal, increasing resistance to infections. In the research done so far, we’ve seen that with viral suppression some individuals do indeed devel-op improved responses to a variety of antigens. But this does not necessarily occur in all individuals, or with all antigens.

Sean’s cells are functioning well, at least for most of the tested pathogens. He had a strong response to CMV, MAC, PCP and *Candida* antigens. While his cells did not seem to respond to antigens from the pathogens that cause toxoplasmosis, streptococcal infections or tetanus, this may be due to a lack of exposure to these organisms. If the body hasn’t encountered them, the immune-cell proliferation would not be programmed in. What we hope further research will tell us is whether we can use LPA results to determine who can discontinue prophylactic medications (such as Bactrim for PCP). Our experience to date tells us that a strong response to an antigen may indicate that the body can again protect itself against that infection, perhaps making

prophylaxis unnecessary.

Still, the failure of immune cells to respond to a pathogen may indicate the need to continue protective meds, even if CD4 counts rise above 200. I ran an LPA on someone who had developed PCP after discontinuing prophylaxis when his CD4 count climbed above 400. I discovered that although his cells were able to rapidly respond to CMV, MAC and toxo, they could not mount detectable responses to PCP. A single case can't give us a rule to follow, but it does raise a warning flag, and points to the need for further research to confirm whether the presence or absence of immune-cell responses to specific antigens is associated with susceptibility.

While the LPA has been around for some time, our lab is working to fine-tune the measurements of the antigens that cause the most problems for PWAs, including MAC, CMV, toxo, PCP and *Candida*. We haven't yet been able to add *cryptococcus* (the cause of cryptococcal meningitis) and *Cryptosporidia* (the cause of cryptosporidiosis), but this is still in progress. Once this type of broad-spectrum LPA is completed and validated through clinical trials, it can be developed commercially, so that physicians can gain a powerful new diagnostic tool for PWAs.

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