**MIGHT HAART HELP?**

Benefits from blocking opportunistic infections, not HIV

Protease inhibitors labeled “anti-HIV” directly block the opportunistic infections that make-up the “AIDS” definition. This fact has emerged quietly, without press attention, several years after the popular and scientific media in 1997 uncritically trumpeted corporate proclamations that these “HAART” drugs specifically targeted HIV while producing benefits, and thus confirmed the official HIV-causes-AIDS model. Two recent studies now flatly falsify any claims of HIV specificity, and offer a sensible explanation for beneficial effects that have nothing to do with HIV. This means that any evidence of HAART benefits cannot automatically bolster the HIV model, even among scientists who believe that HIV causes AIDS, and that HIV tests indicate HIV infections.

by Paul Philpott

**Protease inhibitors directly block P. carinii and Candida**

Does the popular protease inhibitor (PI) combination therapy benefit people who test HIV-positive? And, if so, how does it work?

Mainstream scientists — those who view AIDS as a distinct condition caused by HIV infection — say patients benefit dramatically from this “cocktail” therapy, also known as HAART (Highly Active Anti-Retroviral Therapy). American AIDS mortality has declined since HAART’s introduction, they point out, and many patients report getting better — rising from the dead like Lazarus, the press accounts say — while on it.

Coupling these points with studies that correlate HAART with reduced HIV “viral load,” and their assumption that HIV inhibition represents the HAART PI’s sole capacity, these scientists believe they have confirmation for their HIV model of AIDS: HAART helps people who test HIV-positive, HIV tests indicate HIV infections, HIV infections cause AIDS, and HAART blocks HIV replication; therefore, HIV causes AIDS.

The dissident scientists — who blame AIDS on non-HIV factors and dismiss HIV as a dud — refute this conclusion, and all of the assumptions and claims that lead up to it.

They recognize that American AIDS incidence and mortality figures both began their declines before HAART’s introduction. They realize that despite all the Lazarus testimonies, so many patients become worse that no scientific study has demonstrated a clinical benefit (increased life span or improved symptoms) for HAART. As for the labeling of PIs as “HIV-specific,” a growing list of serious “side effects” disproves that. The dissidents point out that the HIV scientists — the only ones who receive funds to research these issues — have failed to consider a wide scope of data that simultaneously refute the HIV-AIDS model while implicating non-HIV factors.

The dissidents conclude that these data exonerate HIV of any AIDS-causing capacity, and demonstrate that the HIV tests neither indicate nor measure active HIV infections. They substantiate these conclusions regularly in the pages of *RA* and in such books as Peter Duesberg’s *Inventing the AIDS Virus*, both of which rely on references to scientific journals.

Though this dissident view explains more of the facts than the HIV model, its advocates have thus far failed to effectively address the persistent claims by some HIV-positive people that HAART relieved their AIDS conditions. If HIV tests don’t indicate HIV infections, and HIV infections don’t cause AIDS, then how can HAART — designed as it was to block “HIV replication” — relieve the AIDS symptoms of some people who test HIV-positive?

Until now, the dissidents had little data to convincingly answer this question.

Two relatively new scientific reports, published in the *Journal of Infectious Diseases*, demonstrate that the protease inhibitor drugs used in HAART strongly and directly inhibit two of the most prevalent opportunistic microbes that define AIDS: *Pneumocystis carinii* and Candida. Aside from representing additional evidence against the “HIV-specific” assumption for HAART PIs, these studies offer a plausible, non-HIV reason for why these drugs might indeed benefit some patients: by blocking non innocuous HIV replication, but by blocking the pathogenic replication of true disease-causing microbes.

“In Vitro Activity of HIV PIs Against *Pneumocystis carinii*” (Atzori, 181:1629-1634) appeared in the May 2000 issue of *JID*, while...
"In Vitro and In Vivo Anticandidal Activity of HIV Protease Inhibitors" (Cassone, 180: 448-53) ran in the August 1999 issue. Interestingly, the authors of both studies live in Italy, far away from the billions of annual USA tax dollars devoted to the HIV model of AIDS. Neither study cited any sponsor, such as drug companies, US universities, or agencies with links to that money. Nor did the reports receive any of the celebrity-like media hoopla heralding the many corporate-funded studies claiming to demonstrate HIV-based benefits for HAART.

Both reports clearly proclaimed direct and significant inhibitory effects of these drugs on two of the main opportunistic infections comprised by the AIDS definition: Pneumocystis carinii, the microbe involved in P. carinii pneumonia (PCP), and Candida, the fungus involved in thrush. According to the HIV model of AIDS, these non-HIV infections erupt because HIV replication has weakened the host immune system. According to the dissident view, these opportunistic infections erupt because non-HIV factors — such as narcotics consumption or even HIV medications among affluent westerners, or poverty among poor Africans — have weakened the host's immune system.

When HAART coincides with suppression of some symptoms as PCP or thrush, HIV scientists have always confidently attributed the favorable result to HAART’s presumed inhibition of HIV. These JID papers represent the first time that funded scientists have formally considered alternative explanations.

But even these authors make sure to declare unsubstantiated allegiance to the presumptions that HAART generally benefits people who test HIV-positive, and that the benefits derive at least in part from anti-HIV effects. According to the PCP article: "Since the introduction of HIV PI's, dramatic declines in all OIs [Opportunistic Infections], including PCP, have been observed. This favorable outcome and the decline in PCP incidence is clearly due, in large part, to [immune] cell reconstitution induced by HAART," by which the authors mean anti-HIV effects.

According to the Candida article: "observational reports clearly indicate a beneficial effect of PI's" that "has been remarkably efficacious against HIV replication, AIDS progression, and mortality." The authors even make sure to present their own anti-Candida findings as an additional benefit to the "expected immunorestoration," by which they mean anti-HIV effects.

But the data from both papers make it possible to explain HAART benefits without involving the HIV-AIDS model at all. The PCP investigators found that each of the four PI's they examined (indinavir, ritonavir, nelfinavir, and saquinavir) directly suppress PCP as effectively as standard PCP medications. The Candida investigators concluded both of the PI's they examined (indinavir and ritonavir) exerted in vivo anti-Candida effects that were "particularly remarkable as they compared with the curative effects of fluconazole, a well-known antifungal agent."

"No specific evidence that [an anti-HIV effect] is the only or the predominant effect of HAART on OIs has been provided," the Candida report states. So where are the data that compel these or any authors to attribute any observed drug benefits to "anti-HIV" effects?

These articles clearly support the dissidents, indirectly in their view that AIDS can be explained without HIV, and directly in their view that important non-HIV factors have been overlooked. The new data should embolden the call for expanding the list of variables and hypotheses considered by funded researchers.

Can narcotics help explain AIDS in Africa?

The USA's National Institute on Drug Abuse (NIDA, a division of the National Institutes of Health) now officially recognizes an overlap between Africa's "AIDS" patients and narcotics consumers. The NIDA's director, Dr. Alan I. Leshner, on June 30 declared, "Drug use is a contributing factor in the rising HIV infection rates in South Africa and other countries." His comments came in an NIDA press release, reported by the "PRNewswire," issued just before the International AIDS Conference convened July 9 - 14 in Durban, a major port city in South Africa.

The announcement sought to draw attention to a separate series of three sequentially slated mini-conferences, all held in Capetown, all sponsored by the NIDA, and all devoted to such topics as "HIV Prevention in Drug-Using Populations." The first conference convened July 1 and the third closed on July 9, just as the mega-convention began. The NIDA in its press release bragged that it funds "more than 85% of the world's research on the health aspects of drug abuse and addiction."

Funded researchers consider narcotics just an ancillary factor in explaining AIDS, which they attribute exclusively to HIV. Narcotics, they say, contribute to AIDS only in terms of transmitting HIV, either directly via shared needles in the case of injected drugs, or indirectly by causing "high" people to disregard "safe sex" measures.

Dissident scientists, though, doubt or dismiss altogether any role for HIV, and cite the NIH's own research in showing that narcotics consumption directly places users at risk for the diseases that define "AIDS," even when the users test negative on the HIV tests. But articulating this fact in plain language, and acknowledging that it points away from HIV in explaining AIDS, disqualifies scientists from receiving grants, even from the NIDA.

Scientists who consider non-HIV explanations for AIDS in Africa usually focus on one plausible factor that most patients there have in common: the abject poverty that produces many AIDS-defining illnesses even in Africans who test HIV-negative. But like all the factors known to cause these diseases, this one doesn't cover all patients diagnosed as having "AIDS," even in Africa.

This new official revelation of a narcotics culture in Africa could help develop a comprehensive explanation for African AIDS, and suggest a rational and effective course of prevention and treatment.

The NIH and its subsidiaries should support qualified scientists interested in examining the long term immunological and other health effects of narcotics consumption and Third-World poverty. These factors and the consequences of their cumulative life-time exposure merit the same exhaustive scrutiny that NIH funding has provided to cigarette smoking (lung cancer, high blood pressure, emphysema), alcohol consumption (cirrhosis, hepatitis), and high-fat diets (colon cancer, high blood pressure).

— P. P.
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outh African president Thabo Mbeki and his Health Minister faced down journalists pressuring them to publicly pledge allegiance to the HIV model of AIDS.

A *Time* magazine interviewer in the September 11 issue asked Mbeki: "You've been criticized for playing down the link between HIV and AIDS. Where do you now stand on this very controversial issue?"

Mbeki replied: "Clearly there is such a thing as acquired immune deficiency. The question you have to ask is what produces this deficiency? A whole variety of things can cause the immune system to collapse. Now it is perfectly possible that among those things is a particular virus. But the notion that immune deficiency is only acquired from a single virus cannot be sustained. Once you say immune deficiency is acquired from that virus your response will be antiviral drugs. But if you accept that there can be a variety of reasons, including poverty and the many diseases that afflict Africans, then you can have a more comprehensive treatment response."

The reporter then asked if Mbeki was prepared to acknowledge that there is a link between HIV and AIDS.

Mbeki replied: "No, I am saying that you cannot attribute immune deficiency solely and exclusively to a virus. There may very well be a virus. But TB, for example, destroys the immune system and at a certain point if you have TB you will test HIV positive because the immune system is fighting the TB which is destroying it. Then you will go further to say TB is an opportunistic disease of AIDS whereas the fact is TB is the thing that destroyed the immune system in the first place. But if you come to the conclusion that the only thing that destroys immune systems is HIV then your only response is to give them antiretroviral drugs. There's no point in attending to this TB business because that's just an opportunistic disease. If the scientists say this virus is part of the variety of things from which people acquire immune deficiency, I have no problem with that. But to say this is the sole cause, therefore the only response to it is anti-retroviral drugs, I am saying we'll never be able to solve the AIDS problem."

Before *Time* published Mbeki's interview, his Health Minister, Dr Manto Tshabalala-Msimang, sat for a live Radio 702 appearance with Johannesburg talk host John Robbie. According to a September 6 wire report by the South African Press Association (SAPA), Robbie repeatedly tried to pin Tshabalala-Msimang down as to whether or not she believed that HIV was the cause of AIDS.

Robbie's interest derived from Mbeki's controversial embrace of the RA Group scientists who doubt or dismiss a role for HIV in AIDS, and instead blame assorted other factors, such as the wide-spread rural and urban poverty in South Africa. The controversy had gained fresh attention when the press reported that Tshabalala-Msimang distributed a document which her office received that apparently refuted the HIV explanation of AIDS.

Tshabalala-Msimang reportedly talked around the question without ever answering it, perhaps the way politicians do when they know that honesty will be unpopular (RA could not obtain a transcript, and relies only on the SAPA report's rendering of the interview). According to SAPA, a "furious row" ensued, with Tshabalala-Msimang insisting, "You will not force me into a corner into whether saying 'yes' or 'no.' You will not pressure me to answer that." Robbie declared, "I find your position to that question bizarre." He concluded with a parting shot that seems shockingly harsh given Tshabalala-Msimang's position: "Go away," SAPA quotes him as telling her. "I cannot take that rubbish any longer. Can you believe it... I have never in my life heard such rubbish."

SA PRES. MBEKI, HEALTH MINISTER CONTINUE AIDS SKEPTICISM

Frustrate reporters demanding embrace of popular HIV model of AIDS

According to a September 7, 2000 *Africa News* article, Mbeki and his Health Minister faced down journalists pressuring them to publicly pledge allegiance to the HIV model of AIDS.

The article says that Montagnier's study began with 600 prostitutes, of whom 29 tested HIV-positive — or just 5% — as compared to about 25% of their customers, whose HIV-positive rate reflected the general population living in the countryside. These findings surprised people who, like the author of the *Africa News* article, believe that HIV tests identify people with HIV infections, and that HIV infections cause AIDS and transmit via vaginal intercourse. These assumptions predict that prostitutes have a higher than average likelihood of testing HIV-positive and developing the various AIDS diseases.

The Montagnier article undermines this prediction, and thus the
assumptions that lead to it.

Montagnier’s “statement”, though, does not undermine an alternative, “dissident” perspective: that HIV tests don’t indicate any sort of active viral infections; that what scientists call HIV qualifies neither as a pathogen nor a vaginally transmitted agent; and that people who have been exposed to a variety of non-HIV, AIDS-causing factors, such as narcotics, the poor sanitation of abject poverty, and ordinary pathogens, like TB, cholera, and malaria, simply have an increased likelihood of testing positive for any virus, not just HIV. So Montagnier may simply have observed (through HIV-colored glasses) that urban prostitutes in Kenya enjoy a higher standard of living than their customers or people who live in the countryside. If so, it would make sense that unprotected vaginal intercourse with HIV-positive men would not correlate with their becoming positive, but that leaving the sex trade would. Examining this hypothesis would involve surveying and measuring the living standards of prostitutes, their customers, people living in the countryside, and former prostitutes. It would also involve testing these groups for a variety of microbes, to determine if the rate of HIV positivity merely reflects a trend of these people to experience exposure to many biological factors.

Montagnier’s "statement," as presented in the article, demonstrates how promoters of the HIV model — due to their obligation to interpret all data in accordance with the HIV model — face data that contradict their model not by reconsidering their model, but by inventing new absurdities to accommodate it. Take, for example, the prostitutes’ failure to become HIV-positive despite regular unprotected coitus with HIV-positive customers. Rather than reconsider the presumption that vaginal intercourse transmits whatever it is that “HIV” tests indicate, the article has Montagnier presuming that the regular exposure to "small doses" of HIV (in customer semen) had conferred to the women an “immunity” to HIV! And that because these women often became positive after retiring from prostitution, this “immunity” was false.

This involves three paradoxes: (1) Logically, Montagnier should advocate condomless intercourse — and lots of it — with people who test HIV-positive, as a way to prevent HIV transmission; (2) Montagnier not only redefines immunity to mean a lack of neutralizing antibodies (a positive viral test), but that when people produce such antibodies, rather than gaining immunity, they have lost it; and, of course; (3) although prostitutes have higher-than-average rates of non-HIV sexual infections, and although frequency of unprotected sex increases their chance of acquiring them, HIV qualifies as a sexually transmitted microbe even though its transmission not only eludes prostitutes, but increases when sexual frequency decreases.

The biggest paradox of all is that representatives of a supposedly free and independent press credulously report such a preposterous pronouncement from a powerful official involved with spending millions of public dollars and influencing public policy.

— P.P.