

GRID = Gay Related Intestinal Dysbiosis?
Explaining HIV/AIDS Paradoxes in Terms of Intestinal Dysbiosis

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One thing that those who reject the HIV/AIDS hypothesis agree on is that HIV is not the cause of AIDS. But when it comes to alternative theories of causation, disagreement abounds. And some of the most vexing questions surround the earliest cases of AIDS, those that were initially dubbed Gay-Related Immune Deficiency (GRID). Why did it originate in some gay communities? Why did this happen in the late 1970s and early 1980s? Why in the particular form of PCP (*Pneumocystis carinii* pneumonia), candidiasis, KS (Kaposi's sarcoma)? And why still do gay men so often test "HIV+"? Why do some "HIV+" people thrive without medication while others get ill?

Here's a suggestion that answers all those questions in a coherent way.

INTESTINAL MICROFLORA: FUNCTION AND DYSFUNCTION

The normal functioning of the intestinal tract depends upon the presence of huge numbers of hundreds of species of beneficial bacteria. These bacteria are in a symbiotic relationship with humans and other mammals: they help digest food, produce vitamins such as B-12 and K, synthesize crucial amino acids and afford protection against harmful microbes. Gut flora are so vital to our health—yet so overlooked—that they have even been called “the forgotten organ” (1).

Inadvertent alteration of these intestinal microflora may eliminate beneficial bacteria while simultaneously promoting the proliferation of harmful microbes. This state, intestinal dysbiosis, can lead to a series of problems, problems which taken together may explain much of what is called AIDS, at least with respect to some groups of gay men.

First, the lining of the gut may become more porous than normal, a condition known as increased intestinal permeability or leaky gut (2). This then allows microbial translocation—a flood of antigens into the blood stream—to occur, which in turn sets off a systemic immune response and the production of large numbers of antibodies to lots of different things. This condition, hypergammaglobulinemia—too many antibodies to too many things—is a known cause of false-positive reactions on the “HIV tests” (3). Dr. Rodney Richards, an organic chemist who worked on the development of the first such tests, asserts that this condition, rather than infection with HIV, is what causes reactive results on HIV antibody tests (4).

Second, an imbalance of intestinal microflora has serious immunological implications. Beneficial flora play an essential role in our immune system (5). They stimulate immune cells to produce cytokines—chemical messengers—that not only have defensive properties against pathogenic microbes but also maintain the optimal number and distribution of lymphocytes (6) (7) (8). Friendly flora also compete for space in the lining of the gut with potentially pathogenic microbes, keeping their numbers in check (1)(2).

Third, the depletion of intestinal microflora affects nutrition. Since we rely on bacteria to help us digest food and produce various nutrients, a reduction in their number or a change in their nature means that the gut is less able to nourish the body. If this dysbiotic state becomes severe enough the gut becomes increasingly unable to take in nourishment—this is called malabsorption—and malnutrition is a predictable consequence (2).

In his 1996 paper “Destruction of normal resident microflora as the main cause of AIDS” (9) and his 1998 piece, “What causes a positive test for HIV-antibodies” (10), Vladimir Koliadin put forth essentially the same ideas, that the destruction of gut microflora is the reason gay men tend to test positive and develop AIDS. In the 1998 paper Koliadin writes:

“These [gut] flora are of vital importance for digestion of food as well [as] for stifling various opportunistic pathogenic microorganisms. Suppression of the friendly flora of the gut by antibiotics results in serious problems with digestion of food and in development of opportunistic intestinal infections. Such intestinal abnormalities frequently cause increased permeability of the gut walls (“leaky gut syndromes”). Proteins are essential parts of our diet, and they are very powerful foreign antigens. Why don’t they cause antigen overload in healthy individuals? Normally, proteins are digested into short fragments which are not antigens (cannot induce immune response), and only these non-antigen short [fragments] permeate the gut walls and run into the blood stream. Abnormally high permeability of the gut walls makes it possible for molecules of proteins themselves to run into the blood stream and to become a powerful factor of antigen overload. This mechanism provides a plausible explanation for the epidemiological association between promiscuity, diagnosis of AIDS, and HIV-seropositivity...”

EVIDENCE FOR INTESTINAL DYSBIOSIS IN HIV+ GAY MEN

Conditions suggestive of intestinal dysbiosis such as increased intestinal permeability, malabsorption and systemic immune activation are commonly seen in gay men who are reactive on the “HIV test.” A 1995 study of 88 “HIV+” gay men published in *GUT* found that “Malabsorption... was prevalent in all groups of patients with AIDS but not in asymptomatic, well patients with HIV. Malabsorption correlated ... with the degree of immune suppression and with body mass index. Increased intestinal permeability was found in all subgroups of patients” (11). This is exactly what the intestinal dysbiosis hypothesis would predict. In an “HIV+” population, particularly one composed of gay men, increased intestinal permeability would be widespread but malabsorption, an indicator of more severe dysbiosis, would be less prevalent and correlated with the level of immune dysfunction.

Mainstream HIV/AIDS researchers in the last couple of years have begun to publish articles acknowledging the importance of the gut in the development of “HIV disease.”

And these pieces contain strong parallels with what is being argued here, albeit from an HIV-is-the-cause perspective.

In 2006 Brenchley et al. wrote: “Chronic activation of the immune system is a hallmark of progressive HIV infection and better predicts disease outcome than plasma viral load, yet its etiology remains obscure. Here we show that circulating microbial products, probably derived from the gastrointestinal tract, are a cause of HIV-related systemic immune activation.” They go on to say “These data establish a mechanism for chronic immune activation in the context of a compromised gastrointestinal mucosal surface...” (12).

An article from *Nature Medicine* quotes one researcher, who argues that a leaky gut is an important aspect of AIDS, as saying “We think microbial translocation causes systemic immune activation and therefore progression in HIV disease” (13). And a 2006 piece from Aidsmap began with this lead: “Leakage of microbes from the gut as a result of HIV-related damage to the wall of the gut may be one of the major causes of the systemic immune activation that drives the HIV disease process” (14).

POSSIBLE INFLUENCE OF GAY SEXUAL PRACTICES ON GUT MICROFLORA

If intestinal dysbiosis plays a causative role in AIDS, the obvious question is why in the late ‘70s and early ‘80s would sizable numbers of gay men suddenly become afflicted with the condition to the point of becoming seriously ill and dying. In his papers Koliadin points to the widespread and sometimes prophylactic use of broad-spectrum antibiotics which destroy bacteria indiscriminately. Although these drugs undoubtedly played and continue to play a big role, there are other factors that deserve serious consideration.

First, bear in mind the connection between receptive anal sex and the propensity to test “HIV+” which has been noted since AIDS began. The Perth Group in a letter to David Rasnick, citing the Multicenter AIDS Cohort study and the Padian study, stated “The only sexual act, in both gay and heterosexual sex, which is related to the appearance of AIDS and a positive antibody test is receptive anal intercourse.” And “The frequency of this practice, by either sex, and not the number of partners (promiscuity) is the risk factor for the development of AIDS and of a positive antibody test.” They go on to say, “...for AIDS to appear a very high frequency of receptive anal intercourse over a long period is necessary” (15).

It doesn’t take much imagination to connect receptive anal sex with a disturbance of the microflora found in the mucosal lining of the intestinal tract. Trauma associated with anal penetration and the effects of sexual lubricants are two factors that might plausibly be implicated. But another practice likely plays a significant role in intestinal dysbiosis, at least as far as many gay men are concerned: douching.

It’s easy to see how one might completely overlook that this common practice could be a health risk. Cleanliness through washing is a shibboleth; and anyway, how dangerous could a little water be? But the highest concentration and greatest diversity of intestinal microflora are found in the colon (1) (2), precisely the section of the lower intestine that is most impacted during rectal douching. Not surprisingly, the practice has been identified from the very beginning as a factor correlated with testing “HIV+.” A 2007 study of rectal douching in gay men found that “...53% of HIV-negative and 96%

of HIV-positive men douched in preparation for sex, most of them frequently or always...” (16). Anecdotal accounts indicate that some gay men douche daily, and reports of some doing so multiple times a day can be found (17).

The potential danger associated with douching becomes strikingly evident when one examines the manner in which it’s done by the subset of gay men that has historically been recognized to be most at risk, those who practice more hardcore forms of anal sex such as fisting. Before the receptive partner participates in such activity, it is de rigeur that he clean himself out thoroughly. And this is not typically done with ordinary enemas. Instead, a device commonly called a shower shot--essentially a hose attached to a showerhead with a nozzle on the other end--is inserted inside the colon so that the user can direct a steady stream of water inside. And it is generally used multiple times over a period ranging from hours to days to achieve the desired level of cleanliness.

Some have remarked on the danger of these devices, pointing out that not only do they strip away the mucosal lining of the gut, but that the force of the spray they generate can be sufficient to cause tissue damage, including perforation of the colon, and that the danger of exposing colon tissue to dangerously high water temperatures exists unless great care is taken (18). Yet shower shots remain popular among this subset of gay men, and they can be readily purchased online or in sex shops in major cities catering to this clientele. Remarkably enough, they were introduced into common usage among this segment in the early to mid-’70s, 5-10 years before AIDS began.

Curiously, it was around the same time that Gay Bowel Disease, an unpopularly named condition that was officially recognized in 1976, began appearing, reaching epidemic levels in New York City and San Francisco (19). Gay Bowel Disease was considered by Casper Schmidt to be a “penumbral syndrome” for AIDS (20).

EXPLAINING HIV/AIDS ILLNESSES IN TERMS OF INTESTINAL DYSBIOSIS

Most of the diseases and disorders associated with AIDS can be connected, in some cases strikingly so, to intestinal dysbiosis. These connections strengthen the foundation for this simple, plausible explanation for the etiology of the syndrome based on the destruction of gut microflora. That contrasts starkly with the paradox-generating theory that HIV causes AIDS.

Pneumocystis carinii (PCP), candidiasis (thrush) – These two fungal conditions were among the first AIDS-defining illnesses to be recognized (PCP was initially thought to be bacterial but was later found to be caused by a fungus; it was renamed *Pneumocystis jiroveci* but the old name continues to be used). Normally, beneficial bacteria help keep these ubiquitous microbes in check. But in the absence of such flora, they emerge as opportunistic infections (OIs). In addition, gut microflora stimulate the production of cytokines including alpha and gamma interferon (21) (22). Gamma interferon is recognized for its fungus-fighting properties in general (23), and for its effect against PCP in particular (24). Clearly then, an increase in the incidence of fungal infections would be expected to accompany a decrease of beneficial bacteria, both as a result of vacated space in the gut lining being colonized by fungi and as a consequence of a drop in fungal-fighting agents produced by the microflora.

Glutathione deficiency – AIDS patients are characteristically deficient in glutathione, the body's master antioxidant. Numerous studies over more than a decade have documented this fact (25). Several prominent dissidents, including the Perth Group (26) and physician Heinrich Kremer (27) have based their own hypotheses or treatment protocols in large part around this observation. Although glutathione can be absorbed directly from the food we eat, it is chiefly manufactured intracellularly from amino acids, including glutamine and cysteine (28). What researchers on both sides of the fence almost always fail to point out is that these amino acids are produced in the gut by microflora including various strains of *Lactobacillus* (29) (30). Obviously, if one suffers from a lack of such intestinal flora, glutathione synthesis will be inhibited because fewer bacteria would be in place to produce its constituent building blocks, glutamine and cysteine. Backing up this point, a team concluded in 1999 that the results of their study "...suggest that the [glutathione] deficiency of HIV infection is due in part to a reduced synthesis rate secondary to a shortage in cysteine availability" (31).

T-cell abnormalities – There appears to be a connection to be elucidated between gut dysbiosis, glutathione deficiency, and T-cell anomalies thought to be characteristic of HIV/AIDS. Culshaw has discussed the relation between the Th0, Th1, and Th2 subsets of T-cell populations and the balance between reduced and oxidized forms of glutathione (32). A direct connection between the composition of gut microflora and the balance of Th-type cells has been reported by several authors:

"Certain strains of lactobacillus induce high levels of the Th1 skewing cytokines (IL-12 and TNF-a), while other strains of lactobacillus along with the bifidobacterium genus induce high amounts of IL-10 (a tolerogenic cytokine), but low levels of the Th1 skewing cytokines" (33).

"...healthy gut flora keeps the two major arms of the immune system, the Th1 and Th2 immunity, in proper balance . . ." (34).

"The beneficial bacteria in the gut ensure appropriate production of different immune cells, immunoglobulins, and other parts of the immune system. But most importantly, they keep the immune system in the right balance. What typically happens in a person with gut dysbiosis is that two major arms of their immune system, Th1 and Th2, get out of balance with underactive Th1 and overactive Th2. . ." (35).

"There's something about lactobacilli and bifidobacteria, perhaps a protein on the bugs' cell surfaces, that stimulates TH1 immune cell function rather than TH2 function" (36).

Digestive disorders, wasting – Problems with the gastrointestinal tract and progressive weight loss are another hallmark of HIV/AIDS. Intestinal dysbiosis is, by definition, a digestive disorder and the malabsorption induced by severe cases of it offers a direct, uncomplicated explanation for the wasting often seen in AIDS patients.

Selenium deficiency – Since 1994 researchers have remarked upon the fact that low levels of selenium are observed in AIDS patients (37). And some have gone so far as to argue that selenium deficiency is the major factor in disease progression. In his book, *The Origin, Persistence and Failings of HIV/AIDS Theory*, Dr. Henry Bauer notes: “Geographer Harold Foster argues that a selenium deficiency could cause AIDS. He claims a correlation between the prevalence of HIV in Africa and soils deficient in selenium, and finds the level of selenium in serum to be a better predictor of AIDS mortality than are CD4 cell counts (38).”

In 2007 Hurwitz et al. concluded that “Daily selenium supplementation can suppress the progression of HIV-1 viral burden and provide indirect improvement of CD4 count. The results support the use of selenium as a simple, inexpensive, and safe adjunct therapy in HIV spectrum disease (39).”

Citing the Hurwitz paper, George M. Carter, director of the Foundation for Integrative AIDS Research (FIAR)—a group that sponsors and promotes clinical trials of herbal and nutritional treatments for people with HIV/AIDS, affirmed the benefit of selenium in a *POZ* special report from September 2007. He went on to say, “No matter how much food one eats, gut dis-regulation [disruption of digestive absorption caused by HIV] means there will be an inadequate uptake of nutrients (40).”

What Hurwitz, Carter, and others fail to note is that selenium uptake is directly affected by *Lactobacilli*. Calomme et al. reported in 1995 that “...species of the lactic acid bacteria are able to concentrate selenium intracellular[ly] as seleno-cysteine, which could be applied in supplementation studies (41).” And in her book, *The Immune System Cure*, Lorna R. Vanderhaege makes the same observation, and recommends supplementation with *L. acidophilus* as a method of ensuring adequate levels of selenium (42).

Therefore, progressive dysbiosis offers an uncomplicated explanation for the correlation observed between declining selenium levels and illness in AIDS.

Kaposi’s Sarcoma (KS) – Most dissidents believe KS, once considered the hallmark disease of HIV/AIDS, to be caused by the nitrite inhalants (poppers). Although evidence for this connection appears strong, there is also a link between intestinal dysbiosis and KS that’s worthy of consideration. The conventional view of KS is that it is caused not by HIV (as originally thought) but by a herpes virus, HHV-8. The first FDA-approved treatment for KS was alpha interferon, which is documented to block reactivation of HHV-8 (43). As noted above, intestinal microflora favor the production of alpha interferon. Is it possible that a depletion of intestinal flora, a corresponding decrease in the production of alpha interferon resulting in unchecked HHV-8, and a contributing factor such as poppers, are what enables KS to emerge as an AIDS-defining disease?

Other AIDS-defining illnesses – Connections can be found between such diseases as lymphoma (44), toxoplasmosis (45) (46) (47), and oral hairy leukoplakia (caused by Epstein-Barr virus) (48) (49) and the cytokines stimulated by intestinal flora.

Drug use and testing “HIV+” – In addition to the illnesses discussed above, the intestinal dysbiosis theory offers an explanation, at least with regard to some gay men, for

the connection seen between drug use and the propensity to be reactive on the “HIV test.” The orthodox rationale for this observation is that drug use makes unsafe sex acts more likely to occur, putting drug users at increased risk of contracting HIV; and that injecting drug users infect one another by sharing dirty needles. Some dissidents, most notably Peter Duesberg, argue for a direct link between drug use and immune deficiency (50). While such a linkage certainly exists, there is another aspect of this correlation that supports the intestinal dysbiosis hypothesis. As Duesberg notes, some gay men use drugs to facilitate receptive anal sex. But the type of drug use typically varies with the kind of anal sex a gay man might partake in. For example, a run-of-the-mill instance of receptive anal intercourse rarely involves more than nitrate inhalants, if that. But the more extreme varieties of anal sex are almost invariably accompanied by the use of harder drugs such as crystal methamphetamine that not only facilitate but actually fuel these behaviors.

A 2002 article in *POZ* dealt with the connection between methamphetamine (crystal) use and the risk of becoming “HIV+”. The piece includes this quote: “I started as a top [the insertive partner in anal sex], but the more I used [crystal] -- you know, it turns everybody into a raging bottom,” says ‘Mike,’ a 36-year-old New Yorker ...who still uses. “With crystal, all roads lead to fisting. It’s like some weird mystical thing between you and the other guys” (51).

Jad Adams, a medical journalist and the author of *AIDS: The HIV Myth*, wrote in 1989: “Looking at homosexual men with and without the disease it looked clear that the AIDS patients were mainly those who had a large number of sexual contacts (and ‘large’ often means twenty a week); those who were receptive to anal intercourse and those who practised... brachioproctal intercourse [fisting]”.

As noted earlier, extensive douching, often with a shower shot, usually precedes more extreme types of anal sex. Therefore, heavy drug use, extreme forms of anal sex, and extensive douching can all be connected in the particular subset of gay men that have historically been seen to be most at risk for being reactive on the “HIV test” and for developing AIDS. Consequently, cause and effect in this group should not be reduced simply to drugs=AIDS.

EXPLAINING HIV/AIDS PARADOXES IN TERMS OF INTESTINAL DYSBIOSIS

Correlated continuums of cause and effect - Intestinal dysbiosis surely exists on a continuum. That is, most people who have it suffer mild cases, while those on the other end of the spectrum--say, those with AIDS--have severe cases. The rest fall somewhere in between. Once you reach a particular point along that continuum, you will have sufficient levels of circulating antibodies to trigger a reactive result on the “HIV test.” The *GUT* study referenced above, which found that levels of malabsorption, an indicator of more severe gut dysbiosis, correlated with degree of immune suppression, bolsters this view.

But if such a continuum exists, then it would also include on one extreme “HIV-negative” individuals who are at risk and who display immune abnormalities not seen in “negative” individuals not at risk. This, too, is supported by the literature. (Duesberg and David Rasnick made this observation in their 1997 paper, *The Drug-AIDS Hypothesis* [52].) Keeping in mind the aforementioned connection between intestinal microflora and

the production of alpha and gamma interferon, the 1986 study by Bergstrom et al. becomes especially illuminating (53). They measured levels of alpha and gamma interferon in 90 “asymptomatic” gay men and found that 41 of these men produced less gamma interferon than any of their 19 healthy, male, heterosexual controls. But only eight of the 90 gay men were found to be “HIV+”—and these men produced “significantly less alpha and gamma interferon than did the HIV-seronegative homosexuals.”

Such findings are exactly what the intestinal dysbiosis theory would predict and it suggests two parallel continuums: degree of disturbance of the intestinal microflora and a continuum from showing immune abnormalities while testing negative to testing “HIV+” through moderate illness to manifest AIDS. (It should be noted here that there is no reason to assume that illnesses arising from dysbiosis must always come after the point on the continuum at which one would be reactive on the “HIV test.” It’s reasonable to expect that illnesses commonly seen in “negative” individuals—herpes, for instance—might appear with greater frequency prior to becoming “HIV+”.)

The HIV/AIDS theory cannot satisfactorily account for such an observation in which effect (immune abnormality) precedes cause (HIV). This leads to such explanations as the one offered by Bergstrom et al.: “It is concluded that the significantly lower production of both alpha and gamma interferon in asymptomatic homosexual males may play an important role in susceptibility to viruses, including HIV.”

Latency period (variable lag between “infection” and the onset of illness); Long-term non-progressors: Moderate dysbiosis yields a positive “HIV test” but not necessarily illness, but prolonged insults to the intestinal flora lead to illness and AIDS. The variability is connected largely to individual differences as to receptive anal sex. Non-progressors (or “elite controllers”) can be accounted for as well: their lifestyle never leads to more than slight or moderate dysbiosis. Similarly, the increasing tendency of gay men to test positive from the teens into middle age: were “HIV+” a sexually contracted condition, one would expect adolescents to be at greatest risk, as they are with classic STDs.

Oral sex: Truly sexually transmitted diseases such as syphilis and gonorrhea are easily passed on via oral sex, yet oral sex carries little or no risk for the transmission of HIV. Naturally not: intestinal dysbiosis is not transferable through oral contact.

Transient infection: According to HIV/AIDS theory, infection by HIV is permanent—once “HIV+” one remains “HIV+”. However, many independent pieces of evidence testify that some “HIV+” individuals have reverted to negative (54) (55). If “HIV+” results from intestinal dysbiosis sufficiently mild that it can be reversed, then seroreversion would have a straightforward explanation.

It’s known that an “HIV+” reaction may be produced by any of a variety of conditions that may not be seriously threatening, like flu vaccination (56) (57). Intestinal dysbiosis seems to be yet another one.

Research failures: The search for an HIV vaccine has been a total bust despite billions of dollars being spent in the endeavor. If the “HIV tests” are actually detecting

hypergammaglobulinemia resulting from intestinal dysbiosis, then the quest for a vaccine would be predicted to fail.

Other notable failures include the microbicides nonoxynol-9 (58) and Ushercell (59). Both of these sexual microbicides were anticipated to provide new ways to block the transmission of HIV because they killed HIV in vitro. But instead of reducing the rate of “HIV+” they actually increased it! This result is consistent with the dysbiosis hypothesis.

HETEROSEXUALS AND HIV/AIDS

Though the focus here is on the observations concerning HIV/AIDS with regard to gay men, intestinal dysbiosis (and perhaps vaginal dysbiosis) may offer insights into the syndrome with respect to heterosexuals as well.

HIV/AIDS and heterosexual women: A 1999 study concluded that the presence of abnormal vaginal flora was associated with an increased risk of HIV infection (60) and a 2006 paper found that vaginal douching among African sex workers increased their risk of acquiring HIV (61). The conventional explanation for these observations is that douching disturbs the protective mucosal lining of the vagina, making it easier for HIV to reach the underlying tissue and gain entry into the body. But the mucosal lining is also where the beneficial flora reside. So if the mucosa are damaged then it follows that the flora it harbors are being disturbed as well.

HIV/AIDS is equally distributed between the sexes in Africa:

AIDS in Africa subsists in the general population, not largely among high-risk groups such as gay men; and unlike in the Western world where AIDS afflicts more men than women in all tested groups, HIV/AIDS in Africa does not discriminate based on gender. This would be predicted by the intestinal dysbiosis theory. Conditions of poverty such as malnutrition, poor sanitation, limited access to health care, widespread parasitic infections, and the lack of clean water would likely contribute to and exacerbate intestinal dysbiosis. Indeed, intestinal problems are endemic in much of Africa. And intestinal dysbiosis resulting from living conditions would not skew toward one sex or the other but would instead be evenly divided between the two. This would be in sharp contrast to the Western world where such Third World-like conditions would be found only in unusual circumstances.

In the film “The Other Side of AIDS,” Richard MacIntyre says “With gay men the problem is even worse than with heterosexuals because of anal sex. The organisms that are spread through anal sex live outside the body for long periods of time. And if you’ve got everybody having 200 or so partners per year, what you created is a situation that’s very similar to Third-World countries where the water is contaminated by feces. And so we’re having this in San Francisco, in New York. In many gay urban areas, we have Third World–like conditions. Dysentery was endemic in San Francisco right before AIDS hit. There were epidemics of dysentery both in San Francisco and New York.”

Mother-to-child transmission – Some children are presumed infected with HIV perinatally or via breast milk. But perhaps dysbiosis is involved here. Babies are born with an immature mucosal immune system that develops following exposure to microbes transferred from the mother through contact and breastfeeding.

“Babies get their gut flora from the mother. So, if the mother does not have healthy bodily flora, then the baby will not have it either. . . The majority of mothers of immune-compromised children . . . show symptoms of Gut Dysbiosis...” (34).

In *Mucosal Immunity*, Cripps and Gleeson write: “The mucosal immune system is rapidly stimulated at birth by bacterial colonization of the mucosal and external surface...The initial bacterial colonization patterns in the gastrointestinal tract differ between breast- and formula-fed infants; hence, so do the degree and nature of antigenic stimulation of the mucosal immune system” (62).

Cripps and Gleeson also say, “The immediate postnatal period is characterized by increased intestinal permeability to intact macromolecules. Ingestion of colostrum promotes membrane maturation in the gastrointestinal tract, leading to closure within 48 hours of birth.”

Understanding this, perhaps the intestinal dysbiosis hypothesis provides a rational explanation for the paradoxical findings of Coovadia et al. in which they noted that children born to “HIV+” mothers were half as likely to test positive if they were exclusively breastfed than if they were given formula (63). This makes no sense within the context of the HIV/AIDS hypothesis. More exposure to a deadly virus should result in more infections; less exposure should lead to fewer infections.

This team also found that infants who received solid foods in addition to breast milk were 11 times more likely to test “HIV+”. According to a BBC News story, a possible explanation is that “...this higher risk is due to the larger, more complex proteins found in solid foods which may lead to greater damage to the lining of the stomach, allowing the virus to pass through the gut wall” (64).

A more sensible accounting for the observations of Coovadia et al. is that they merely reflect the degree of dysbiosis the infants acquired from their mothers, their corresponding level of unresolved intestinal permeability, and their degree of exposure to the larger and more complex antigen-inducing proteins in the solid food eaten before their mucosal immunity system was adequately developed.

WHY DO ANTIRETROVIRAL DRUGS SOMETIMES HELP AIDS PATIENTS?

One of the most common arguments voiced by defenders of the HIV/AIDS hypothesis is why, if HIV doesn't cause AIDS, do the anti-HIV drugs appear to be working? Though the idea that the drugs are “working” in the long run is debatable (65), reports of patients getting markedly better after having started HAART cannot be ignored. But this observation is easily explained if intestinal dysbiosis is a causative factor in AIDS. A central point is that the principal original AIDS-defining diseases, candidiasis and PCP, were fungal.

The intestinal dysbiosis hypothesis argues that beneficial flora are destroyed and replaced by aggressive colonizers, mainly fungi such as *Candida albicans*, and that much

of what is called AIDS is driven by these unchecked microbes. The protease inhibitors (PIs) introduced in the mid-'90s have strong antifungal properties. PIs target secretory aspartyl proteases (SAP), and fungi have SAP. Cassone et al. wrote, "...patients receiving PI therapy may benefit from a direct anticandidal activity of these drugs" (66). Atzori et al. stated: "...it is conceivable that PIs had other beneficial effects, including direct activity against Pneumocystis [PCP]" (67).

What's more, Bektic et al. noted: "Oropharyngeal candidiasis is one of the first and most commonly reported opportunistic infections of untreated AIDS patients. With the introduction of the new antiviral HAART therapy, including HIV protease inhibitors, this mucocutaneous infection is nowadays only rarely observed in treated patients. It was recently shown that HIV protease inhibitors have a direct attenuating effect on *Candida albicans* secreted aspartic proteinases (Saps), an investigation prompted by the fact that both Sap and HIV protease belong to the superfamily of aspartic proteinases and by the observation that *mucocutaneous infections sometimes resolve even in the absence of an immunological improvement of the host*" (emphasis added) (68). Which is to say the researchers noticed that patients sometimes got better even though their surrogate markers--CD4 counts, viral load--did not improve.

This latter point is notable, and strong support for this interpretation comes from the time scale of the improvement in health that is often cited. The largest study of HAART covering >22,000 individuals in 12 cohorts between 1995 and 2003 observed: "Virological response after starting HAART improved over calendar years, but such improvement has not translated into a decrease in mortality"; in other words, viral load was suppressed but the patients' health--or risk of dying--did not improve (69). Other studies have reported that suppression of viral load, or increase in CD4 counts, occur rather slowly (if at all) under HAART. Any improved health resulting from either of these sources would necessarily also be slow. But the most enthusiastic accounts of success of antiretroviral treatment describe rapid improvement in health, sometimes almost an immediate improvement. It is highly implausible that slow restoration of T-cell numbers and slow suppression of T-cell-killing virus could bring noticeably quick improvement in health; but antifungal treatment can produce favorable outcomes very rapidly.

IMPLICATIONS

If intestinal dysbiosis plays a significant role in AIDS, restoring a normal balance of microflora through the use of probiotics would be expected to offer some benefit. Support for this can be found in the literature.

Zareie et al. found that probiotics prevented bacterial translocation in rats (70). Other researchers noted that "Probiotics displayed the following effects in these studies: Involvement in production of essential nutrients of the colonic mucosa, beneficial effect on intestinal immunity, recovery of the disturbed gut mucosal barrier and prevention of microbial translocation..." (71).

"High levels of Th1 cytokines have been found repeatedly in the mucous membranes of long-term HIV/AIDS survivors. Strengthening these Th1 cytokines requires the patient to eat fresh, healthful food slowly, mixing it with saliva, and only when hungry.

Probiotics *Lactobacillus plantarum* and *L. casei* are exceptional stimulators of Th1 cytokines” (72).

Cornell immunologist Susanna Cunningham-Rundles found that 13 “HIV+” children experiencing “failure to thrive”—meaning they were low in weight and short in height for their age—improved when given probiotic bacteria in a fruit juice drink.

“The process of digestion, nutrient metabolism and activation of the immune system are linked in rather surprising ways to the specific composition of micro-organisms that comprise the gastrointestinal flora,” said Dr. Cunningham-Rundles. “Certain micro flora directly stimulate the immune system, and this may promote a healthy immune response.”

“The immune system is like a muscle, requiring constant stimulation to work in peak condition. Finding that immune cell products, called cytokines, are secreted in response to *Lactobacillus plantarum* 299v was all-important, since one of these, gamma interferon, has powerful defence capabilities,” she added (73).

Two papers released in 2008 offer further evidence that probiotics are useful to those who are “HIV+.” One asserts that *Lactobacillus* supplementation might reduce the viral load of vaginal secretions in “HIV+” women (74). And most remarkable of all, Dr. Kingsley Anukam, a Canada-based African researcher, published a study in the *Journal of Gastroenterology* which found that probiotic supplementation relieved diarrhea in “HIV+” women and stabilized CD4 counts. He wrote, “This is the first study to show the benefits of probiotic yogurt on quality of life of women in Nigeria with HIV/AIDS, and suggests that perhaps a simple fermented food can provide some relief in the management of the AIDS epidemic in Africa” (75).

CONCLUSIONS

Dysbiosis is capable of producing effects that can lead to testing “HIV+”, and it can lead to opportunistic fungal infections and other illnesses and disorders characteristic of AIDS. Certain sexual practices common among subsets of gay men seem designed to produce dysbiosis. Cessation or limitation of such practices coupled with the consumption of probiotic foods and supplements may arrest or reverse dysbiosis.

Author’s note: The foregoing clearly implies that paying attention to a normal balance of intestinal microflora could be of benefit in individuals who have tested “HIV+”, perhaps particularly (but not only) if they evidence no symptoms of actual illness, and especially if there are reasons to suspect intestinal disturbances. However, this discussion is intended to be informational and should not be interpreted as medically qualified advice. I’m neither a doctor nor a scientist, but I do have valuable real-life experience: I’ve been “HIV+” for at least 12 years; I’ve never taken HAART yet I continue to live in good health; I’ve read countless articles and studies on the subject; and, perhaps most important, I’ve observed the behaviors of a lot of gay men. What’s more, I’ve been regularly consuming probiotics of various kinds—kefir, yogurt, cultured buttermilk, sauerkraut, cultured vegetables, semi-hard cheeses—for more than a decade, and I’m convinced they are beneficial to my health.

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