VDR or HIV?

Abstract
Vitamin D is a steroid hormone produced through sunlight (UVB) exposure. It is both an immunostimulant [1] and an immunosuppresant. [2] Deficiency of, or insensitivity to this substance can be classed as a type of immunodeficiency.

The literature proposes that vitamin D supplementation is beneficial to people 'infected' with 'HIV', [3] however, ample evidence suggests that the way in which vitamin D works provides strong support to dissident viewpoints that HIV either does not exist, or exists but cannot be the cause of AIDS. It may not even be exogenous. [4]

Debate
In 1984 the then US Secretary of Health announced by press conference that the "probable cause" of acquired immunodeficiency syndrome (AIDS) had been found: a virus called the human immunodeficiency virus (HIV). [5] A vaccine was scheduled to arrive within 2 years, but 26 years later there still hasn't been any notable developments. Current treatment for HIV/AIDS patients is the administration of antiretrovirals to control disease progression.

The most visible dissident of the HIV/AIDS hypothesis is Peter Duesberg who alleges that HIV is not the cause of AIDS but a subsequent benign infection arising from immunodeficiency initiated by toxic substance abuse and/or malnutrition. [6] Later, The Perth Group went a step further and claimed that HIV has never been purely isolated so may not even exist. They also state that what HIV test kits identify is hypergammaglobulinemia (an abundance of antibodies to many antigens), which is only indicative of immunodeficiency, not causative. [7]

Because of the amount of time, passion and money spent on AIDS research, Duesberg's and The Perth Group's claims have appeared controversial and been maligned by the mainstream. This however does not automatically nullify their arguments. May it also be noted that Kary Mullis, a Nobel laureate, - who invented the polymerase chain reaction (PCR) which is used to detect HIV "viral load" [8] - is amongst the growing group of voices called 'AIDS denialists'. [9]

Acquired Intestinal Dysbiosis Syndrome?
In 2008 a long-term non-progressing HIV+ gay man named Tony Lance penned an unpublished paper that pondered whether HIV positivity and subsequent development of AIDS in gay men was caused by intestinal dysbiosis ("leaky gut syndrome"). [10] The paper expanded on the writings of Vladimir Koliadin [11] [12] and painted a logical connection to The Perth Group's theory in that a compromised gut will release a stream of antigens into the blood to which a mass of antibodies are produced. This is hyergammaglobulinemia and Lance may have identified a cause, if not the root cause of AIDS.

Additionally, Lance points out that disturbed intestinal microflora leads to multiple micronutrient deficiencies which can account for the development of many AIDS associated illnesses. The paper's observations can be equally applied to abusers of intravenous drugs (recreational and some prescribed) as it has been found that cocaine addicts can suffer from intestinal tearing, [13] as can their babies developing in their womb. [14] In the case of the Sub-Saharan African epidemic, stereotypical malnutrition and parasitic assaults would lead to a similar intestinal environment. The cause of intestinal dysbiosis in gay men though - who may or may not be drug abusers - has been candidly suggested by Lance as being attributed to *excessive* anal sex, zealous douching or use of certain lubricants. Other than gay or bisexual men, women who frequently participate in receptive anal intercourse are seen as the most likely to be affected by HIV/AIDS. [15]

Vitamin D & the gut
Wherever there are vitamin D receptors (VDR) there will be a use for vitamin D, just like every lock will have a key to open it. Our bodies are riddled with vitamin D receptors [16] so it is no surprise that they can be found in a significant area such as the gut.

In mid 2010 scientists discovered that the VDR is a key player in maintaining gut flora balance. [17] The study was designed to shed light on general inflammatory bowel disorders, but their finding also appears to support the intestinal dysbiosis theory of HIV/AIDS.

In this murine study, mice were studied who were ether a) normal, b) had the VDR disabled or, c) free from germs. All were then infected with a harmful strain of Salmonella. It was found that in the mice with the VDR disabled, to quote a press article, "These mice showed higher levels of activity of inflammatory molecules, and they lost weight more quickly and were much more likely to die in response to infection." [18] Baring in mind that recurrent Salmonella infection is classed as an AIDS defining illness, [19] it is easy to see that they may have created stereotypical AIDS patients - albeit non-human ones. I therefore agree that people who suffer from HIV/AIDS if caused by intestinal dysbiosis are suffering directly because of behaviour that has modified or damaged the gut's VDR.

Is supplementation the answer?
In this instance vitamin D supplementation would have limited or no effect depending on the severity and progress of the condition; which in turn determines if someone with HIV remains an infinite long term non-progressor.

Activated vitamin D (known as 1,25D or calcitriol, opposed to supplemental or sun-exposure created calciferol or 25-D which needs to be converted by the body), however, could be considered as a treatment but with caution as it is infinitely less safer than letting the body activate ordinary vitamin D when it can. Ease of toxicity through high blood calcium levels (hypercalcemia) is the risk of administering the active form. [20] With normal supplementation there is little observed risk of toxicity in the literature, which even then can be attributed to rare production mishaps where someone may have inadvertently taken millions of units a day for a long

period of time. But even then permanent organ damage or death is not a necessity with good follow-up care. [21]

Regardless, since vitamin D deficiency is a worldwide problem owing to the majority of humans covering most of their body in clothing for most of their lives, adhering religiously to safe sun advice, and living and working indoors, maintaining a healthy calciferol level is a must for people who are HIV+ or otherwise. However, given the possible complete malabsorption problems of some sufferers this may need to be administered as the active form by injection, or alternatives to the by-products of vitamin D need to be sought out instead.

The reason why vitamin D deficient people – opposed to the insensitive - may not always test positive is down to the fact simple deficiency itself would not automatically lead to a mass of antibodies, plus there may be just about enough activated vitamin D being utilised even in deficiency.

Certain vaccines though, such as for flue, may produce a response that at least temporarily flags as 'false-positive' HIV infection in such people. [22]

It is important to note that there is some controversy about the amount of vitamin D to give a person and the level they can achieve. 400IU is the recommended daily allowance of many governments worldwide based on knowledge that this is enough to prevent rickets in children and osteomalacia in adults, but this level is woefully inadequate for satisfying over 2000 receptors in the body. [23] Reinhold Vieth has demonstrated that humans can generate up to 10-25,000IU per day through optimal UVB exposure. [24] Many physicians also do not discriminate between the preferred form of supplement (Vitamin D3) versus the commonly prescribed inferior form (D2). [25]

In England the highest accepted vitamin D level on the NHS appears to be 128 nmol/L (50 ng/mL), but there are several - primarily American recommendations - that allow going up to 200 nmol/L (80 ng/mL). [26] Very few people, however, have anywhere near these levels due to modern living, therefore the conservative recommended dose is over-cautious. It is the blood level achieved rather than the dose that matters.

As vitamin D is an important immunomodulator it stands to reason that even in absence of intestinal dysbiosis that vitamin D deficiency is indeed a form of AIDS that most of us suffer to a minor or major degree, dependent on seasonal UVB and antigen exposure; be it as simple winter colds or cancer.

Benefits and alternatives

Lance's paper mentions that HIV/AIDS patients are deficient in the detoxifier glutathione. This correlates with vitamin D deficiency or insensitivity since vitamin D up-regulates glutathione. [27] Glutathione deficiency may lead to an inability to absorb one form of vitamin B12 effectively, [28] and it's been shown that B12 deficiency is associated with T-cell abnormalities [29] which is a hallmark of HIV/AIDS. This can be remedied by administering injectable or high dose sub-lingual B12 therapy; the latter can bypass gut malabsorption as 1% of a dose is absorbed through an alternative pathway. [30] B12 has also been used to successfully treat AIDS-related dementia, [31] and is showing promise in typical dementia cases.

Fungal infections such as candida are also frequently found in HIV/AIDS patients and the small efficacy of standard antiretroviral treatment could be down to their anti-fungal activity alone. [32] Vitamin D is anti-fungal through helping to mass produce a broad-spectrum antibiotic called cathelcidin. [33] Given that long-term prescribed antibiotics can upset a gut it seems logical that an antimicrobial that the body produces with natural help is a much better option. Additionally, the downside of common antiretrovirals like zidovudine (AZT) is that they reduce vitamin D levels. [34] It is notable also that a touted cure for HIV/AIDS is a macrophage activating factor called GcMAF which also upregulates cathelicidin. [4]

Another way of making the conditions of the gut more favourable is to introduce more friendly bacterial products, as suggested by Lance.

A number of cancers are classed as AIDS defining illnesses, and it's possible that physicians do not scrutinise their cause if they occur in HIV+ individuals. Another amazing benefit of vitamin D is its ability to make cancerous cells commit suicide (apoptosis). [35] Cells contain a 'button' that beg to be pushed by vitamin D when they turn awry and multiply uncontrollably. Indeed, research is even being directed to the development of patentable vitamin D analogues (synthetic clones) [36] as an alternative to current non-discriminatory cancer treatments that can damage healthy cells.

Beyond dysbiosis

Adding weight to the theory that the VDR plays a role in HIV/AIDS is the fact that intravenous drug abusers suffer from low cholesterol. [37] This is a critical finding because if the cholesterol precursor called 7-dehydrocholesterol is specifically being inhibited, this will also diminish vitamin D production since vitamin D is made when UVB meets the precursor in the skin. Subsequently when the body lacks glutathione and cathelicidin, the response to foreign threats is handled almost exclusively, though not necessarily successfully, through antibody production.

The fact that a well-known celebrity tested positive while a drug user, then negative while not [38] shows that in absence of possible dysbiosis, quitting drugs alone may re-establish the normal production of cholesterol which in turn re-enables vitamin D synthesis and clearance of material that flags as HIV.

Vitamin D & HIV/AIDS orthodoxy

A study on pregnant women has shown that a higher vitamin D level cuts the rate of HIV transmission. [39] What was not pondered was that the reason for HIV negativity could be purely down to the fact that such mothers simply enhanced their immunomodulatory status which is then passed on to the child. Bodies that can deal with threats through innate – non-antibody - immunity in most cases may not generate antibodies that flag as HIV. The reason babies may spontaneously lose their acquired HIV+ status could be attributed to gut sealing as suggested in Lance's paper and/or the fact that babies in developed countries are often quickly given cow's milk with some form of vitamin D fortification which would improve the immunomodulatory status of small bodies with ease. Breast milk from vitamin D deficient mothers would be of less help.

Pregnancy itself is commonly acknowledged as a source of false positive results. [40] In one recent popular media case of a young German popstar we see a HIV+ mother with a child who could be HIV- (as they have not been outed as HIV+, which would be expected as prosecution evidence), yet one of a *few* of the mother's subsequent sexual encounters yielded in HIV transmission. This is odd given that the baby is a product of her mother's cells while sexual fluid transmission is limited in intercourse. Benaissa herself is of dark complexion and a former drug addict [41] which could account for a series of positive results.

Other observations

From an American observation blacks and Hispanics are more likely in that order to test positive than Caucasians. [42] While poverty and social standing can play a factor in HIV positivity, if the VDR hypothesis is further true minus dysbiosis, then this fact can be easily explained: blacks are most likely to be vitamin D deficient due to having the most pigmentation which acts as a sunlight filter. Hispanics would be the second most affected as their skin is less lighter than Caucasians.

There is a spanner in the works, however, as other ethnic groups test less positive than Hispanics despite reasonably similar skin pigmentation. [43] It is therefore possible that either HIV tests give a positive result in accordance with specific genetic traits, [44] and/or that certain communities are hounded overall into testing because of perceived behaviours.

Age also appears to a play factor, with risk increasing in the middle aged [45] - paradoxically when alleged risky behaviours stereotypically diminish. This can be explained by the fact that 7-dehydrocholesterol transport to the skin declines with age, therefore reducing vitamin D synthesis. [46] If the decline is low enough it may produce signs and symptoms of HIV/AIDS.

Haemophiliacs are another group who test positive frequently and there could be two reasons for this: the first is that factor VIII treatment presents a similar assault to the body as recreational intravenous drugs, while secondly, impaired kidney function is a common finding in such sufferers. The kidney plays a part in converting inactive vitamin D to its active form. Another clue to the impairment of this process is that haemophiliacs with rickets can be typically non-responsive to the even low amount of vitamin D2 or D3 that can correct the problem. [47] If they are non-responsive to simply treating rickets they have worse problems to face. Hysterical media reports have erroneously blamed HIV contamination of blood products for haemophiliacs, but how this could have happened has never been elucidated. [48]

Penultimately, tuberculosis, which is a classic AIDS defining illness is also a classic vitamin D deficiency related illness that has historically been treated through sunlight exposure (heliotherapy), though with over-caution that led to its demise. [49] Lastly, osteoporosis which is linked to low vitamin D may not seem an unlikely candidate for being added as an HIV defining condition. [50] This would be the result if the gut cannot absorb calcium, which would be the case in VDR dysfunction or deprivation.

Research targets

If the intestinal dysbiosis via gut VDR dysfunction hypothesis is true, research should be aimed at trying to repair the VDR or seeing what the effect of introducing the exact or equivalent substances dependent on vitamin D has on HIV/AIDS patients. For this to happen, proponents of vitamin D and AIDS rethinkers need to form a symbiotic relationship to create pressure. It is possible that the relative unpopularity of the latter group may impede the higher positive mainstream progress of the former, though a widescreen understanding may also quickly raise the profiles of both parties in order to benefit public health worldwide.

This is a matter of urgency because if the virus does not exist, it can never be isolated and a vaccine cannot ever be developed. Mutations of the 'virus' may simply be soups of antibodies or cellular debris through inflammation that simply react with unreliable and non-specific test kits. If HIV, however, exists, potent antibody production may still not be the core issue.

In the mean time then, people are suffering from illnesses – current or potential – attributed to the wrong target.

Disclaimer: This review has not been written or endorsed by a scientist, medical professional or a HIV+ individual. It has neither been published in any medical journal. The author has no conflicts of interest to disclose.

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References

1. Cerezuela R, Cuesta A, Meseguer J, Angeles Esteban M. Effects of dietary vitamin D3 administration on innate immune parameters of seabream (Sparus aurata L.). *Fish Shellfish Immunol. 2009 Feb;26(2):243-8.*
2. Lemire JM, Archer DC, Beck L, Spiegelberg HL. Immunosuppressive actions of 1,25-dihydroxyvitamin D3: preferential inhibition of Th1 functions. *J Nutr. 1995 Jun;125(6 Suppl):1704S-1708S.* Review.
3. Villamor E. A potential role for vitamin D on HIV infection? *Nutr Rev. 2006 May;64(5 Pt 1):226-33.* Review.
4. Matteuzzi M. Endogenous retroviruses as confounding factors in the pathogenesis of AIDS. *University of Florence. 2010.* Retrieved December 1, 2010, from http://hivskeptic.files.wordpress.com/2010/10/tesi-chiara-matteuzzi.pdf
5. U.S. Announces 3 AIDS Discoveries. *Toledo Blade. 1984 Apr.*
6. Duesberg P, Koehnlein C, Rasnick D. The chemical bases of the various AIDS epidemics: recreational drugs, anti-viral chemotherapy and malnutrition. *J Biosci. 2003 Jun;28(4):383-412.*
7. Papadopulos-Eleopulos E, Turner VF, Papadimitriou J, Page B, Causer D, Alfonso H, Mhlongo S, Miller T, Maniotis A, Fiala C. A critique of the Montagnier evidence for the HIV/AIDS hypothesis. *Med Hypotheses. 2004;63(4):597-601.*
8. Bartlett JM, Stirling D. A short history of the polymerase chain reaction. *Methods Mol Biol. 2003;226:3-6.*
9. Foreword for 'Inventing the AIDS Virus'. *1993.* Retrieved December 1, 2010, from http://www.duesberg.com/news/Foreword.pdf
10. Lance T. GRID = Gay Related Intestinal Dysbiosis? Explaining HIV/AIDS Paradoxes in Terms of Intestinal Dysbiosis. *2008.* Retrieved December 1, 2010, from http://hivskeptic.files.wordpress.com/2008/02/gay-

relatedintestinaldysbiosis.pdf
11. Koliadin V. Destruction of normal resident microflora as the main cause of AIDS. Retrieved December 1, 2010, from *Continuum. 1996 Aug*. http://www.virusmyth.com/aids/hiv/vkmicro.htm
12. Koliadin V. What causes a positive test for HIV-antibodies? *Continuum. 1998 Apr.* Retrieved December 1, 2010, from http://www.virusmyth.com/aids/hiv/vktest.htm
13. Tiwari A, Moghal M, Meleagros L. Life threatening abdominal complications following cocaine abuse. *J R Soc Med. 2006 Feb;99(2):51-2*.
14. The' TG, Young M, Rosser S. In-utero cocaine exposure and neonatal intestinal perforation: a case report. *J Natl Med Assoc. 1995 Dec;87(12):889-91*.
15. Papadopulos-Eleopulos E et al. Perth group responds to Rasnick. *2000.* Retrieved December 1, 2010, from http://www.virusmyth.com/aids/hiv/epreprasnick.htm
16. Ramagopalan SV, Heger A, Berlanga AJ, Maugeri NJ, Lincoln MR, Burrell A, Handunnetthi L, Handel AE, Disanto G, Orton SM, Watson CT, Morahan JM, Giovannoni G, Ponting CP, Ebers GC, Knight JC. A ChIP-seq defined genome-wide map of vitamin D receptor binding: associations with disease and evolution. *Genome Res. 2010 Oct;20(10):1352-60.*
17. Wu S, Liao AP, Xia Y, Li YC, Li JD, Sartor RB, Sun J. Vitamin D receptor negatively regulates bacterial-stimulated NF-kappaB activity in intestine. *Am J Pathol. 2010 Aug;177(2):686-97*.
18. University of Rochester Medical Center (2010, July 8). Amid the murk of 'gut flora,' vitamin D receptor emerges as a key player. ScienceDaily. Retrieved December 1, 2010, from http://www.sciencedaily.com /releases/2010/07/100707141558.htm
19. Centers for Disease Control and Prevention. Appendix A: AIDS-Defining Conditions. *December 5, 2008 / 57(RR10);9* Retrieved December 1, 2010, from http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5710a2.htm
20. Valrance ME, Brunet AH, Welsh J. Vitamin D receptor-dependent inhibition of mammary tumor growth by EB1089 and ultraviolet radiation in vivo. *Endocrinology. 2007 Oct;148(10):4887-94*.
21. Cannell J. The Truth About Vitamin D Toxicity. *2009.* Retrieved December 1, 2010, from http://www.vitamindcouncil.org/vitaminDToxicity.shtml
22. Mac Kenzie WR, Davis JP, Peterson DE, Hibbard AJ, Becker G, Zarvan BS. Multiple false-positive serologic tests for HIV, HTLV-1, and hepatitis C following influenza vaccination, 1991. *JAMA. 1992 Aug 26;268(8):1015-7*.
23. Medscape Today. Postmenopausal women receiving inadequate vitamin D. *2005 Jun.* Retrieved December 1, 2010, from http://www.medscape.com/viewarticle/538233
24. Vieth R.Vitamin D supplementation, 25-hydroxyvitamin D concentrations, and safety. 1999. *Am J Clin Nutr. 1999 May;69(5):842-56.* Review.
25. Houghton LA, Vieth R. The case against ergocalciferol (vitamin D2) as a vitamin supplement. *Am J Clin Nutr. 2006 Oct;84(4):694-7*.
26. 25-Hydroxy Vitamin D and Calcium Reference Ranges Updated (American). *Legacy Laboratory Services. 2008 Jan*. Retrieved December 1, 2010, from http://bit.ly/1TDGCq
27. Garcion E, Wion-Barbot N, Montero-Menei CN, Berger F, Wion D. New clues about vitamin D functions in the nervous system. *Trends Endocrinol Metab. 2002 Apr;13(3):100-5.* Review.
28. Pacholok SM, Stuart J. Chapter 12: The Possible Role of B12 in Autism. Could it be B12?: *An Epidemic of Misdiagnoses. 2005. Quill Driver Books/Word Dancer Press, Inc.*
29. Tamura J, Kubota K, Murakami H, Sawamura M, Matsushima T, Tamura T, Saitoh T, Kurabayshi H, Naruse T. Immunomodulation by vitamin B12: augmentation of CD8+ T lymphocytes and natural killer (NK) cell activity in vitamin B12-deficient patients by methyl-B12 treatment. *Clin Exp Immunol. 1999 Apr;116(1):28-32*.
30. Kuzminski AM, Del Giacco EJ, Allen RH, Stabler SP, Lindenbaum J. Effective treatment of cobalamin deficiency with oral cobalamin. *Blood. 1998 Aug 15;92(4):1191-8.*
31. Herzlich BC, Schiano TD. Reversal of apparent AIDS dementia complex following treatment with vitamin B12. *J Intern Med. 1993 Jun;233(6):495-7*.
32. Munro CA, Hube B. Anti-fungal therapy at the HAART of viral therapy. *Trends Microbiol. 2002 Apr;10(4):173-7*.
33. López-García B, Lee PH, Yamasaki K, Gallo RL. Anti-fungal activity of cathelicidins and their potential role in Candida albicans skin infection. *J Invest Dermatol. 2005 Jul;125(1):108-15*
34. Fox J, Peters B, Prakash M, Arribas J, Hill A, Moecklinghoff C. Improvement in Vitamin D Deficiency Following Antiretroviral Regime Change: Results from the MONET Trial. *AIDS Res Hum Retroviruses. 2010 Sep 21*.
35. Zinser GM, McEleney K, Welsh J. Characterization of mammary tumor cell lines from wild type and vitamin D3 receptor knockout mice. *Mol Cell Endocrinol. 2003 Feb 28;200(1-2):67-80.*
36. Guyton KZ, Kensler TW, Posner GH. Vitamin D and vitamin D analogs as cancer chemopreventive agents.*Nutr Rev. 2003 Jul;61(7):227-38.* Review.
37. Gettler JF. Hypocholesterolemia in substance abusers. *South Med J. 1991 Jul;84(7):937*.
38. Ozzy Osbourne 'was told he could be HIV positive by doctors'. *The Telegraph. 2009.* Retrieved December 1, 2010, from http://www.telegraph.co.uk/news/newstopics/celebritynews/6261061/Ozzy-Osbourne-was-told-by-doctors-he-could-be-HIV-positive.html
39. Mehta S, Hunter DJ, Mugusi FM, Spiegelman D, Manji KP, Giovannucci EL, Hertzmark E, Msamanga GI, Fawzi WW. Perinatal outcomes, including mother-to-child transmission of HIV, and child mortality and their association with maternal vitamin D status in Tanzania. *J Infect Dis. 2009 Oct 1;200(7):1022-30*.
40. Matemo D, Kinuthia J, John F, Chung M, Farquhar C, John-Stewart G, Kiarie J. Indeterminate rapid HIV-1 test results among antenatal and postnatal mothers. 2009. *Int J STD AIDS. 2009 Nov;20(11):790-2*.
41. HIV-positive pop singer Nadja Benaissa admits having unprotected sex. *The Times. 2010 Aug.* Retrieved December 1, 2010, from http://www.independent.co.uk/news/world/europe/hivpositive-pop-singer-nadja-benaissa-admits-having-unprotected-sex-2054528.html
42. Friedman SR, Sotheran JL, Abdul-Quader A, Primm BJ, Des Jarlais DC, Kleinman P, Maugé C, Goldsmith DS, el-Sadr W, Maslansky R. The AIDS epidemic among blacks and Hispanics. *Milbank Q. 1987;65 Suppl 2:455-99*.
43. HIV/AIDS: New HIV report turns up some surprises. *IRIN. 2010.* Retrieved December 1, 2010, from http://www.irinnews.org/report.aspx?Reportid=89677
44. Various posts of Henry Bauer at HIV/AIDS Skepticism blog. http://hivskeptic.wordpress.com/
45. Arie S. HIV infection is rising among over 50s across the world, figures show. *BMJ. 2010 Jul 27;341:c4064. doi: 10.1136/bmj.c4064*.
46. MacLaughlin J, Holick MF. Aging decreases the capacity of human skin to produce vitamin D3. *J Clin Invest. 1985 Oct;76(4):1536-8*.
47. Welt SI, Graham JB, Kossove DB, Kirkman HN. Vitamin D-resistant rickets and hemophilia A--lack of close linkage on the X chromosome. *Am J Hum Genet. 1973 Jan;25(1):105-7*.
48. Bayer Exposed (HIV Contaminated Vaccine). *MSNBC. 2006 Nov*. Retrieved December 1, 2010, from http://www.youtube.com/watch?v=wg-52mHljhs
49. Howson CR. HELIOTHERAPY IN PULMONARY TUBERCULOSIS-ITS POSSIBILITIES AND DANGERS. *Cal West Med. 1928 Jul;29(1):25-30.*
50. Paccou J, Viget N, Legrout-Gérot I, Yazdanpanah Y, Cortet B. Bone loss in patients with HIV infection. *Joint Bone Spine. 2009 Dec;76(6):637-41*. Review.