There are no translations available.

WePeA5599 Poster

Atherosclerosis and AIDS : Homology between atherogenic Marek's herpes virus and LDL receptor, between cytomegalovirus (CMV) and LDL-related receptor, and between HIV-1 VIF and phospholipase A2.

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Aids is characterized by the occurrence of severe atherosclerosis in very young patients. This prompted us to look for the physiopathology of atherosclerosis in this especially susceptible but unusual population. Our objective is to screen for viruses inducing atherosclerosis.

We looked on Marek's herpes virus (Fabricant C.G., 1978), which can induce in chicken coronary disease even with a diet without cholesterol. CMV is also a good candidate, as it enhances the recurrences of coronary stenosis after stent. Lastly, in transgenic mouse, phospholipase A2 is responsible of atherosclerosis (Ivandic B., 1999). By comparison of amino acid sequences, we found 3 significant homologies :

1. Marek's herpes virus 132bp repeat (sequence LHTS... TICRNK FLCLLP) ...is a rabbit LDL cholesterol receptor (repeat V)-like (CIHSSÉSLC-ss-CKNK... RQFVC-ss-CPVL) in tridimensional cysteine bridged [-ss-] structure.
2. CMV TRL6 (sequence WKTV...DQWLCNVTGIGNAT) is a LDL related receptor-like. (WHSV...QWLCD).
3. HIV-1 VIF protein has the tridimensionnal structure and active site residues (His, Ser) of a phospholipase A.

Conclusion:

These striking homologies between virus and cholesterol receptors, and between HIV-1 VIF and phospholipase A may explain the abnormal occurrence of severe atherosclerosis in such a young population as AIDS. Treatment must focus on anti-herpes virus drugs (and bee propolis, an antiviral natural product) as well as on VIF for a component of AIDS vaccine and anti-phospholipase drug.

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Immune Reconstitution by DHEA - A significant Clinical Case

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Background:

DHEA prevents TH1 and TH2 imbalance, known to contribute to the progression of HIV. Moreover, HIV+ patients show generally a decline of DHEA blood level.

Method:

Blood DHEA sulphate(DHEAS), cortisol, CD4 and CD8 counts were measured for an HIV+ patient presenting initially a low DHEAS/cortisol level, during supplementation by 7-keto DHEA for 16 months.

Results:

A DHEA supplementation of a 60 year old osteoporotic patient HIV+ since 20 years, with a stable biological and clinical state during the preceeding two years (CD4 =325±20/mm3; viral load After 3 months, blood DHEAS level increases to 1433ng/ml. At 6 and 9 months, DHEAS level increases to 2700ng/ml and then decreases to 1200ng/ml at 16 months. CD4 increases progressively, 407 (+24%) at 3 months, 520(+58%) at 6 months and then remains stable 515+/-22. This increase is highly significant (p