

Hiv actively causes cell destruction biology essay



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The HIV-AIDS hypothesis has come under onslaught by a few dissenters from the chief watercourse scientific community. Peter Deusberg is one who has denounced the thought that HIV infection causes AIDS. He suggests a chemical footing for the acquisition of AIDS that is independent of HIV. As of yet, there is no clear reply to this argument, but there is merely grounds that can back up one side or the other.

One of Deusberg ' s allegations against the HIV-AIDS hypothesis is that HIV is said to do AIDS by killing more CD4 T-cells than the organic structure can replace ; nevertheless others have shown that less than 1 in 500 T-cells that is killed is infected with HIV, even in terrible AIDS patients (Duesberg et al. , 2003) . From this he concludes that HIV must be a latent rider virus.

The information, nevertheless, seem to be somewhat misdirecting in that they do non straight suggest that HIV is non doing injury, as he claims, because they do non account for any effects that HIV has on cells that it is non straight infecting. If it can be shown that the class of HIV infection induces greater cell decease than simply the cells it is straight associated with, so it would look that HIV is actively causing harm to the host and is non a rider virus. This would do Deusberg ' s claim shut-in because HIV would finally be responsible for much greater cell decease than he has implied. Such immune suppression would besides be ideal for the patterned advance of many timeserving infections that are characteristic in AIDS patients (Deusberg et al.

, 2003) . A survey by Mohri et Al. (2001) compares the proliferation and decease of T cells between healthy controls and HIV-positive, untreated

patients. Their consequences showed an overall addition of cell proliferation in CD4 T cells of 6.

3 times higher than that in healthy controls and a cell decrease rate of 3 times greater. Therefore in HIV-positive, untreated patients the turnover of CD4 T cells is significantly higher than their healthy controls. These high turnover rates in patients were greatly decreased when put on antiretroviral medicine and became about normal over clip (Mohri, et al. , 2001) .

A possible mechanism for this immune hyper activation is described by Ott et Al. (1997) . They have determined that the HIV protein Tat is responsible for increased release of IL-2, a proliferation and activation signal released by T cells. They have used this in combination with a survey by Zack et Al. (1990) who have shown that HIV does non infect inactive cells to demo that this is a possible mechanism that allows HIV to proliferate into many antecedently unaccessible cells. It would look that the hyper activation of immune system should take to increased Numberss of T cells in blood instead than a lower count that is declarative of an HIV infection (Mohri, et al.

, 2001) . However, Correa and Munoz-Fernandez (2001) have shown that even though rates of division of T-cells are high, the Thymus is shown to hold lower than normal degrees of naA? ve T-cell end product in HIV positive patients. Thus, even though mature T-cells may be splitting quickly to bring forth ringers, over clip they will decease off and are non replaced by new cells. This is suggested to take to the low T-cell count phenotype seen in untreated HIV patients (Correa & A ; Munoz-Fernandez, 2001) .

In this manner, it can be seen how HIV, while present in one cell, can let go of proteins to increase cell proliferation, and at the same clip diminish the coevals of new cells. In this state of affairs there would be a big figure of shorter lived mature T-cells, but over clip the regenerative abilities of the Thymus are unequal at replacing lost cells taking to T-cell depletion and immune suppression. This described mechanism outlines how immune suppression can develop by loss of regenerative ability instead than increased loss of septic cells. Next, a survey by Banda et Al.

(1992) describes how CD4 T cells stimulated with gp120 show rates of programmed cell death four times greater than that of normal cells in vitro. Even though their method involves in vitro testing, they suggest that it is comparable to in vivo state of affairss as it has been found that gp120 is released from septic cells and can be found free in solution around septic cells (Schneider et al. 1986) .

This mechanism for increased programmed cell death in CD4 T cells is expanded upon by Finkel et Al. (1995) who have found that cells deceasing from programmed cell death are non cells that are infected with HIV, but are alternatively cells in close propinquity to HIV septic cells. Of the 1000 apoptotic cells and 700 HIV infected cells that they counted utilizing microscopy, none were both apoptotic and HIV infected. This adds extra grounds against the claim made by Deusberg et Al. (2003) . HIV does non needfully move by killing the cells that it infects, but alternatively additions cell decease rates in all of the T cells around it.

In this manner one would anticipate to happen HIV nowadays in merely a little per centum of cells that die because it is increasing the cell decrease rate of clean cells, but non septic cells. I have described above two methods that suggest that HIV does non move as a rider virus. HIV is accountable for lower regenerative capacity of the immune system to replace lost T cells and the gp120 glycoprotein from HIV is responsible for programmed cell death in cells in propinquity to an infected cell, but non the infected cell itself.

Therefore, HIV kills T cells and leaves the organic structure incapable of replacing them. Not merely does this suggest that HIV is non a latent rider virus, but it besides offers a possible mechanism for immune suppression in the host. This could take to increased susceptibleness to timeserving infections that are declarative of an AIDS diagnosing.

Even though HIV infected cells constitute merely a little per centum of the entire T-cell population, they cause harm that is far more important.