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THE PHYSIOPATHOLOGY OF LAV/HTVL-III: A COMPLEX PATHWAY OF HOST-VIRUS INTERACTION

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Once established evolution of LAV infection depends mainly on three factors: (1) The virus tropism will determine its target cells and organs. Among T lymphocytes, CD8 cells are completely resistant to infection while CD4⁺ cells are highly susceptible because the crucial role of the CD4 molecule in controlling virus binding and its subsequent penetration into CD4⁺ cells. However many other cell types are now known to be more or less susceptible to LAV. Nevertheless the immune system as a whole, via CD4⁺ T lymphocytes and monocytes, but also via B lymphocytes or even hematopoietic stem cells is the most visibly affected by LAV infection, though the brain and probably the gut and the lungs are directly infected by LAV. (2) The persistence and spread of the virus as well as its effect on infected cells will depend on LAV biological properties. LAV cDNA is latently integrated into LAV or express viral antigens till their activation, which will trigger intense replication depending on very powerful viral *cis* and *trans*-activators. This will result in a cytolytic effect and/or modification of the cell normal function, according to the nature of the infected cell. Also LAV proteins may have direct inhibitory activity on the immune system. (3) The immune response of the host will also influence evolution of the infection. The depressed immune system might help the establishment of LAV primary infection. The perverse effect of LAV tropism will affect both the possibility of mounting a LAV-specific response (killing specific cells) and the whole immune system due to the central role of CD4⁺ cells. Host's genetic factors might tilt the balance one way or another, depending on whether they control the immune response to LAV or the development of autoimmune process. Finally, combination of the basic characteristics of the virus and of the host will determine what probably controls disease evolution, the extent of virus replication. The more the virus will replicate and spread the more cells and organs will be subsequently affected. In this respect cofactors (antigenic stimulations, concurrent viral infections, etc.) may be determinant in modifying the complex equilibrium between LAV and its host.

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