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Editorial

Immunology of HIV

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INTRODUCTION

Retroviruses are genome invaders that have shared an extended history of coevolution with vertebrates and their system. Found endogenously in genomes as traces of past invasions, retroviruses also are considerable threats to human health once they exist as exogenous viruses like HIV. The immune reaction to retroviruses is engaged by germline-encoded sensors of natural immunity that recognize viral components and damage induced by the infection. This response develops with the induction of antiviral effectors and launching of the clonal adaptive immune reaction, which may contribute to protective immunity. However, retroviruses efficiently evade the immune reaction, due to their rapid evolution. The failure of specialised immune cells to reply, a sort of neglect, can also contribute to inadequate antiretroviral immune responses. Here, we discuss the mechanisms by which immune responses to retroviruses are mounted at the molecular, cellular, and organismal levels.

IMMUNOLOGICAL ASPECTS OF HIV

Recent advances within the understanding of the pathogenesis of infection with human immunodeficiency virus (HIV) stems from the demonstration that the membrane glycoprotein, CD4, is that the cellular receptor for HIV. This glycoprotein is found mainly on the surface of a serious subpopulation of T lymphocytes and also on macrophages, natural killer cells, some B lymphocytes, and neuronal cells [1]. Cells infected with HIV could also be destroyed or have their normal function impaired. Host immune responses to HIV are poor and aren't sustained. Neutralizing antibody often isn't produced, or HIV may shake normal immunosuppressive mechanisms through the method of rapid antigenic variation. Factors and markers which will be important within the outcome or which will predict progression of HIV infection are genetic (Gc type), environmental (nutritional status or intercurrent sexually transmitted diseases sustained by the host), and immunologic (rate of decline in number and impairment of function of CD4 lymphocytes and of decline in antibody titers to HIV core protein, p24). A recombinant vaccine will probably be developed for testing in future clinical trials.

The cellular system is that the main component of adaptive immune responses that focus on virally infected cells and is preferentially destroyed in HIV disease [2]. This section will highlight aspects of adaptive and natural immunity including T-cell development, antigen presentation and recognition, T-cell memory, chemokine biology, lymph gland architecture and B cell development, relevant to HIV disease pathogenesis.

Since the identification of HIV and HCV much progress has been made within the understanding of their life cycle and interaction with the host system. Despite these viruses markedly differ in their virological properties and in their pathogenesis, they share many common features in their immune escape and survival strategy. Both viruses have developed sophisticated ways to subvert and antagonize host innate and adaptive immune responses [3]. Within the last years, much effort has been wiped out the study of the AIDS pathogenesis and within the development of efficient treatment strategies, and a fatal infection has been transformed during a potentially chronic pathology. Much of this data is now being transferred within the HCV research field, especially within the development of latest drugs, although an enormous difference still remains between the result of the 2 infections, being HCV eradicable after treatment, whereas HIV eradication remains at the present unachievable thanks to the establishment of reservoirs. During this review, we present current knowledge on innate and adaptive immune recognition and activation during HIV and HCV mono-infections and evasion strategies. We also discuss the genetic associations between components of the system, the course of infection, and therefore the outcome of the therapies.

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