Ubiquitination and SUMOylation in HIV Infection: Friends and Foes

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Abstract:(600words)

As intracellular parasites, viruses hijack the cellular machinery to facilitate their replication and spread. This includes favouring the expression of their viral genes over host genes, appropriation of cellular molecules, and manipulation of signalling pathways, including the post-translational machinery. HIV, the causative agent of AIDS, is notorious for using post-translational modifications to generate infectious particles. Here, we discuss the mechanisms by which HIV usurps the ubiquitin and SUMO pathways to modify both viral and host factors to achieve a productive infection, and also how the host innate sensing system uses these post-translational modifications to hinder HIV replication.Acquired Immunodeficiency Syndrome (AIDS) was first acknowledged as an infectious disease in the early 1980s, when a large number of young, previously healthy, homosexual men suffered from lymphadenopathy, contracted unusual opportunistic infections and/or experienced other malignancies, causing their imminent death (CDC, 1981a,b). The fact that this disease was initially manifested among homosexual males led the press and some authorities to mistakenly name it GRID (gay-related immunodeficiency, often referred to as gay cancer or gay plague), which further stigmatized an already marginalized gay community. Owing to collaborative efforts between American and French scientists, the causative agent for this disease was successfully identified in 1983 (Barré-Sinoussi et al., 1983; Gallo et al., 1984). The agent, a human retrovirus that substantially differed from the Human T-lymphotropic virus (HTLV) characterized by the Gallo lab, was initially named lymphadenopathy-associated virus (LAV), later known as Human Immunodeficiency Virus or HIV. Remarkably, in 1986, another human retrovirus was found to cause AIDS in West Africa, although with less severity than the virus isolated in the US and Europe. Phylogenetic and immunological analyses revealed that this African virus was related but distinct from the original LAV (Clavel et al., 1986). In consequence, LAV was termed as HIV-1 and the African retrovirus as HIV-2. In this review, we use the broad term HIV to cover general aspects of the biology of both HIV-1 and HIV-2. However, for some particular characteristics we will use their specific denomination.

Biography:(200words)

Marta Colomer-Lluch was working in IrsiCaixa AIDS Research Institute, Hospital Germans Trias i Pujol, Badalona, Spain.Department of Biological Sciences, College of Arts and Sciences, Texas Tech University, Lubbock, TX,USA. He published many articles and very interested in many researches.

About Research Topic: (200 words)

Although HIV primarily infects T helper lymphocytes, the early loss of a large fraction of these cells in the gut-associated lymphoid tissue (GALT) does not lead to the immunodeficiency syndrome, since AIDS is caused by a massive depletion of these cells, mainly in blood, which is only observed in the last stage of the disease. In fact, the ultimate destruction of the host immune system seems to be the result of various factors. On one hand, the reduction in CD4+ T cells in the GALT makes the gastrointestinal mucosa more permeable, which, over time, facilitates the translocation of microbial products to the bloodstream, a fact that causes the characteristic chronic immune activation observed in untreated and AIDS-progressing HIV+ individuals

About Institution :(200 words)

The IrsiCaixa AIDS Research Institute is an international landmark and leading centre for research into the eradication of HIV/AIDS and related diseases. IrsiCaixa researchers also tackle other biomedical challenges, such as those associated with the microbiome and emerging infectious diseases. The IrsiCaixa AIDS Research Institute was created as a private non-profit foundation in 1995 with the support of “la Caixa” and the Ministry of Health of the Government of Catalonia.

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