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Functional cure for HIV

While current antiretroviral treatment regimens have been extremely effective, issues of associated toxicity, cost and resistance remain. There is a need for novel approaches and antiretroviral compounds to complement the existing therapy and for developing new strategies for inhibition of HIV infection. Transcription from the HIV promoter in the viral long terminal repeat is regulated by the combined activity of the host transcription machinery and the viral transactivator Tat protein. Recently, the Tat inhibitor didehydro-cortistatin (dCA) has been reported to prevent viral reactivation from latent reservoirs, resulting in a permanent state of latency or block-and-lock that could eventually culminate in a functional cure of HIV (Mousseau et al., 2015). When introduced to humanized mice models of HIV-1 infection, dCA stops the virus's production, activation, and the replenishment of other infected cells that would contribute to the spread and growth of HIV (Kessing et al., 2017), this work demonstrated the potential of block-and-lock cure strategies. We have discovered that spironolactone (SP) - an antagonist of aldosterone - specifically inhibits HIV infection of permissive T cells and reactivation of latently infected T cells (Lacombe et al., 2016). SP-induced inhibition is mediated by blocking the Tat-dependent transcription of HIV-1 and HIV-2 and the reactivation of transcription from latent HIV-1 promoters in latently infected T cells. SP offers the advantages of being cheap and acting both in permissive cells as well as in reservoirs where the virus persists despite antiretroviral therapy. Finally, only HIV transcriptional inhibitors like dCA and SP that prevent infected cells from making viruses, stop the side effects of low-level virus production.

Biography

Cecilia Ramirez received her master's degree at the University of Maryland (College Park, USA) and her Ph.D. degree at the Institut Jacques Monod (Paris) from the Université Paris Diderot. She completed her training at Rutgers University (NJ, USA) as a Post-doctoral fellow and joined the French "Centre National de la Recherché Scientifique" (CNRS) in 1996. She is project leader at the Institut Cochin in Paris. She has focused her research work on the study of viruses-host interactions to understand the molecular mechanisms used by viruses to efficiently multiply by borrowing host's cell machineries and metabolic pathways. Her team recently showed that spironolactone (SP) is a specific inhibitor of HIV Tat-dependent transcription. They continue to evaluate the transcription block by SP in several latent reservoir models to determine the possibility to use of SP to ''block and lock'' latent reservoirs leading to a functional cure of HIV as a new strategy to control HIV infection.

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