Short Communication
Volume 10:S2, 2021

Molecular Biology

ISSN: 2168-9547 **Open Access**

Life Cycle, Replication and Regulation of Gene Expression

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Abstract

The expression of core histone tiny chemical assembly instructions inside of living things is cell cycle controlled. Large amounts of histones are needed to restore copyd chromatin during S phase when DNA answer/copy happens. Over-expression and excess collection over time of histones outside S phase are poisonous to cells and therefore cells need to restrict histone expression to S phase. Misregulation of histone tiny chemical assembly instruction inside of living things expression leads to defects in cell cycle development or increase over time of events or things, total set of tiny chemical assembly instructions of a living thing firm and steady nature/lasting nature, DNA damage response and written version of spoken words al regulation. Here, we discussed the factors involved in histone tiny chemical assembly instruction inside of living things regulation as well as the hidden under machine. Understanding the histone regulation machine/method/way will shed lights on explaining the side effects of certain cancer chemotherapeutic drugs and developing possible biomarkers for tumor cells. The whole total set of tiny chemical assembly instructions of a living thing sequences of many living things have suggested that all the tiny chemical assembly instructions inside of living things present in any oraganism are not active at the same time. It seems that there are inbuilt internal machines that usually in a common and regular way guide that which tiny chemical assembly instruction inside of living things will be active and which active tiny chemical assembly instruction inside of living things will be shut down. The machines/methods/ways involved in the expression and regulation of tiny chemical assembly instructions inside of living things are controlled by many factors such as methylation, acetylation, phosphorylation, role of silencers, different upstream or RNA polymerase binding copying DNA segments into RNA factors and natural interfering RNA.

Keywords: Life Cycle, Gene Expression, DNA, RNA, HIV.

The goal of this chapter is to provide a brief summary on the "life" cycle of HIV, which should maybe be better referred to as the viral "answer/copy" cycle, since viruses do not have their own chemically processing and using food and are this way usually not thought about/believed living things. Viruses can be thought about/believed as within cell things that feed off of, and weaken, other things that are strictly dependent on living host cells for reproduction. Understanding how HIV interacts with its target cells in order to copy is of great interest because it may provide important clues for the generation of improved HIV/AIDS-fighting drugs and the development of new success plans/ways of reaching goals to control or even eliminate the virus. The main targets of HIV are CD4+ helper T cells, which are key devices that control things/groups of people that ensure rules are followed of the humoral and cellular unable to be harmed responses. So, their destruction and using everything up completely by machines/methods/ways that are not fully understood make the body unable to defend itself against grabs at any easy opportunity things that cause disease. When HIV infects an activated CD4+ T cell, it hijacks and controls/moves around/misleads its written version of spoken words al and translational machinery to reproduce itself. As briefly organized and listed below and specified in the following chapters, HIV has to use a large number of cellular factors and to oppose/to go againstact the HIV/AIDS-fighting activity of others in order to complete its answer/copy cycle.

Probably but not definitely, each interaction with cellular factors that are extremely important for virus answer/copy termed "virus-dependency" factors or strengthening of HIV/AIDS-fighting or "host restriction" factors. As described below, CD4 is the first or most important receptor, and the chemokine receptors CCR5 and CXCR4 are the main co-receptors of HIV entry Fusion. These receptors are good enough to make/give cells easily able to be harmed or influenced by HIV entry and so decide/figure out the viral cell tropism. However, the densities of the Env trimers on the virions and of the CD4 receptor on the target cells are often low. So, viral attachment is often inefficient and a limiting step for HIV infection. What's more, they may trap viral particles at the cell surface to make steady/make firm and strong them and to help settle an argument Tran's infection of able to be harmed or influenced T cells. For example, some say that dendritic cells DCs bind HIV virions at the site of related to sex organs exposure and transport them to the small areas in the body that fight disease where they help settle an argument both trans infection and stimulation of T cells that results in huge virus production.

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Received 03 February, 2021; **Accepted** 17 February, 2021; **Published** 24 February, 2021

How to cite this article: Sakti Prakash Patra. Life Cycle, Replication and Regulation of Gene Expression. Mol Biol 10.S2 (2021):267.