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Research Article

IN SILICO STRUCTURAL AND MOLECULAR INTERACTIONS STUDY BETWEEN HIVGP120 & HUMAN CD4+

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ABSTRACT

The immune system is made up of special cells involved in protecting the body by HIV are the CD4+ lymphocytes, which help direct immune function in the body. CD4+ lymphocytes are required for proper immune system function, when enough CD4+ cells have been destroyed by HIV. Many of the troubles accomplished by people infected with HIV result from a failure of the immune system, to form possible structure of the HIV gp120 which is cause of T cell infection and a structure of the CD4+. The present study conducting of on binding pattern of the gp120 and CD4+ through the docking process.

Keywords: gp120, CD4+, Immune system, docking.

INTRODUCTION

The interaction between the HIV gp120 and its receptor CD4, on the target cell, provokes conformational changes in gp120 and allows access of the V3 loop of gp120 to the coreceptor (a chemokine receptor on the target cell) [1]. A fusion peptide within gp41 of HIV gp120 causes the fusion of the viral envelope and the host-cell envelope, making the capsid protein to enter into the target cell. The mechanism by which gp41 causes the fusion in the target cell is still unknown [2].

The production of the virus is regulated by regulatory proteins Tat and Rev. As Rev accumulates, viral DNA starts to inhibit mRNA splicing [3]. The integration of the proviral DNA into the host genome is carried out by another viral enzyme called integrase and is called the latent stage of HIV infection [4].

The Env polyprotein (gp160) diffuses through the endoplasmic reticulum and is transported to the Golgi complex where it is cleaved by protease enzyme and processed into the two HIV envelope glycoproteins gp41 and gp120. These are again transported to the plasma membrane of the host cell where gp41 anchors the gp120 to the membrane of the infected cell. The Gag (p55) and Gag-Pol (p160) polyproteins also associated with the inner surface of the plasma membrane along with the HIV genomic RNA as the potential virion begins to bud from the host cell [5]. This process can be inhibited by antiviral drugs.

Most AIDS researchers thought Duesberg was exploiting qualms about the specific mechanism of disease causation to reduction a mountain of gripping epidemiologic, laboratory, and animal data supporting the conclusion that HIV causes AIDS [6]. The HIV envelope glycoprotein gp160 is synthesized as an inactive precursor, gp120/gp41 by host cell proteinases during its intracellular trafficking [7]. Fusion regulatory protein-1 regulates virus-mediated cell fusion and monocytes fusion of the virion[8].

MATERIALS AND METHODS:

Swiss Model Server

SWISS-MODEL is a fully automated protein structure homology-modeling server, to make protein modeling accessible to all biochemists and molecular biologists world wide. The SWISS-MODEL is a simple and popular homology-modeling program, uses the “building by fragments” method to construct the model on the template structures.

Swiss PDB viewer for Homology modeling:

Swiss-PDB viewer is and application used to analyze several proteins and can be superimposed in order to deduce structural alignments and compare their active sites.

Hex 5.1model for docking:

Hex5.1 is an interactive protein docking and molecular superposition program, for calculating and displaying feasible docking modes of pairs of protein and DNA molecules. *Hex5.1* can also used to calculate protein-ligand docking, assuming that the ligand is rigid.

RESULTS AND DISCUSSION:

In the homology modeling structure prediction method, we have predicted the structure of HIV gp120 and Human CD4+. Percentage identity shows the similarity in functions. As per the Percentage identity, the SPDBV predicted more than 60%, and made with the conclusion that the sequence of interest has exact same function as that of the template.

The ETotal for the HIV gp120 and human CD4+ should be less than Estart and should lie in between Emin and Emax. As per the docking results, Hex predicated Estart as 20.55, and Etotal is -297.54kcal. So it is less than Estart and it's more lying towards Emin. Hence a stable complex as per the docking result has been predicated. So this docking complex is valid one complex and these two molecules gp120 and CD4+ are having binding affinity which actually meant for the HIV infection.

CONCLUSIONS:

Various mathematical models are present and can be used to elucidate the principles which govern HIV and immune system dynamics in relation to antiviral drug therapy. The immune system continuing health of an animal depends upon its ability to recognize and repel disease such as HIV.

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References:

1, Chan. DC, Kim. PS, 1998, Cell**,** Vol 93, pp681-684.

2. Wyatt. R, Sodroski. J, 1998, Science, Vol 280, pp1884-1888.

3. Pollard. VW, Malim. MH, 1998, Annu Rev Microbiol., Vol 52, pp491-532.

4. Zheng. YH, Lovsin. N, Peterlin. BM, 2005, Immunol Lett., Vol 97, pp225-234.

5. Gelderblom. HR, 1997, Los Alamos National Laboratory (ed.) HIV Sequence Compendium, Los Alamos, New Mexico: Los Alamos National Laboratory, pp31-44.

6. Cohen. J, 1994, Science, Vol 266, pp1642-1644.

7. Etienne. D, Sandrine. W, Carlo. DB, Claude. L, Jean-Marie. R, Nabil. GS, 1996, The Journal of Biological Chemistry, Vol 271, pp30442-30450.

8. Ohgimoto. S, Tabata. N, Suga. S, Nishio. M, Ohta. H, Tsurudome. M, Komada. H, Kawano. M, Watanabe. N, Ito. Y, 1995, The Journal of Immunology, Vol 155, Issue 7, pp3585-3592.

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