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**BIOSENSORS AND BIOELECTRONICS** 

September 27-28, 2017 Chicago, USA

## Optimal control model for immune effectors response and multiple chemotherapy treatment (MCT) of dual delayed HIV pathogen infections

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In tackling the persistent menace of the deadly human immunodeficiency virus (HIV) and its accompanying acquired In tackling the persistent menace of the dealing human human denoted, the formulated. In this present study, a limmunodeficiency syndrome (AIDS), some notably mathematical models have been formulated. In this present study, a number of compatible models were studied. The result of which led to the formulation of a classical 5-Dimensional delaydifferential dynamic equations, principally primed with the investigation of the methodological application of multiple chemotherapy treatment (MCT) in the presence of delay intracellular and cell-mediated immune effectors response on the interplay of dual delayed HIV-pathogen infections and the T-lymphocytes cells. The model was presented as an optimal control problem and analyses conducted using classical numerical methods - Pontryagin's minimum principle. The method demanded for the verification of positivity of state variables and boundedness of solution; as well as the establishment of model existence of optimal control pair for MCT and the system dynamic optimality solution. Using in-built Runge-Kutter of order of precision four in a Mathcad platform, the resulting analyses were subjected to numerical verification. Numerical simulations indicated that maximization of uninfected T-lymphocytes cells is dynamic under drug validity period. Importantly, the model established the fact that upper bounds on treatment optimal weight factors and presence of delay intracellular are crucial to the maximization of healthy CD4+ T cells, significant reduction of virions and suppression of infected CD4+ T cells. Furthermore, the rapid response of virions and infected cells to multiple chemotherapy treatment is emphatically attributed to the enormous role of boosted immune effectors response. The study therefore, advocate for a more accurate model that extensively define the role of the immune effectors response.

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