INDRAPRASTHA INSTITUTE *of*
INFORMATION TECHNOLOGY **DELHI**

Invitation to M.Tech. Thesis Defense of Kajal Singla : August 20, 2021 (Friday): 15.00-16.00 IST

In Partial Fulfilment of the Requirements for the Degree of

M.Tech. CB

Kajal Singla (MT19214)

Will defend her thesis

Title: "**A Computational Framework to Study Levodopa Induced Dyskinesia Effect in Parkinson's Disease**"

IIIT-D Faculty and Students are invited

Date: August 20, 2021 (Friday)
Time: 15.00-16.00 IST
Online over Google meet (https://meet.google.com/wex-drcf-tor)

Examiner:	**Internal:**	**Ganesh Bagler**
	External/~~Internal~~:	**S. Ramachandran, IGIB**
	Advisor:	**K. Sriram**

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Abstract

"Parkinson disease (PD) is the second most common neurodegenerative disorder and results from the death of dopaminergic neurons of Substantia nigra which affects the movement as well as cognition. At a molecular level, the loss of dopaminergic neurons results in loss of dopamine levels that results in tremor. Presently, Dopamine replacement therapy, carried out using the drug levodopa, is found to be the most effective drug for improving movement in Parkinson patients. Basal Ganglia (BG) is the site of action where neurotransmitter Dopamine plays an important role in Parkinson' s disease. The striatal neurons in BG harbours two types of receptors, namely D1 and D2 to which dopamine, or the substituent of dopamine, namely the drug levodopa binds to the receptors and ameliorates the tremor. However, long-term use of levodopa increases complications by causing dyskinesia or what is famously called as "Levodopa-induced dyskinesia" or LID for short. LID changes synaptic plasticity (in terms of LTP and LTD) between the Cortex and Striatum neurons (connected with glutamatergic synapses). In order to counteract this dyskinesia, at an advanced stage of PD, levodopa is supplemented with the drug Amantadine to treat levodopa-induced dyskinesia which changes the synaptic plasticity and reduces dyskinesia. In the present thesis work, we model the effect of the drug Amantadine reduced LID by taking into consideration the role of various channels that play an important role in these processes. This extensive mathematical model captures the main features of LID in the presence of both the drugs levodopa and amantadine and indicates the reduction in dyskinesia is due to the role played by the striatal potassium rectifier channel. The simulation results are validated with the experimental data extensively and we hope that this modeling study will help to support the existing theories in LID."