Name: SHAMSUN NAHAR KHAN, Ph.D

E-mail: nahar305@yahoo.com

Name of the Host Institute:

Department of molecular and Cellular Biology, Harvard University, Cambridge, MA-02138, USA

Name of the Home Institute:

Department of Pharmacy, East West University, Dhaka, Bangladesh

Abstract of my Research Work is as follows:

Title: Structure and Function of Human 7SK snRNA and HIV1 Tat Interaction

Human immunodeficiency virus (HIV) exploits host's cellular proteins during its replicative cycle and latent infection. The positive transcription elongation factor b (P-TEFb) is a key cellular transcription factor critical for these viral processes.

7SK is an abundant, 331-nucleotide small nuclear RNA (snRNA) that functions as a transcriptional regulator during the elongation phase. 7SK sequesters P-TEFb as 7SK/HEXIM1/P-TEFb ribonucleoprotein complex. 7SK RNA binds to HEXIM1 regulatory domain and promotes the binding of the HEXIM C-terminal domain to cyclin T1/T2 of P-TEFb. P-TEFb shows little CTD kinase activity which indicates that 7SK snRNA in collaboration with HEXIM1 function as an inhibitory factor of P-TEFb.

During viral replication, P-TEFb is recruited via interactions of its cyclin T1 subunit with the HIV Tat (transactivator of transcription) protein and TAR (transactivation response) element. Currently 7SK snRNA structure is understood and Tat contains an arginine-rich motif (ARM) in which a single arginine residue has been shown to confer specific binding to the TAR bulge region.

In our poster we will be presenting the strong interaction of 7SK snRNA and Tat through 2D NMR spectroscopic studies and ITC (isothermal calorimetric analysis). In our initial study we have evidence for preformed arginine binding motifs in 7SK snRNA with pseudo triple platform which is responsible for its strong interaction with Tat peptide. ITC data also supports this strong binding interaction with preliminary data indicates a high binding affinity of TAT peptide to 7SK-SL1 (Kd = ~50 nM, n=~1.36).