Fei Liu

Current Position: Research Scientist at New York State Institute for Basic Research in Developmental Disabilities; and Adjunct Professor of Biochemistry at Nantong University in the People's Republic of China

Education: Ph.D. in Biochemistry and Molecular Biology (1999) from Shanghai Medical College, Fudan University in the People's Republic of China

Non-scientific Interests: Knitting and cooking

I have been working at New York State Institute for Basic Research since 1999 where as a postdoctoral fellow I studied post-translational modifications of proteins involved in the pathogenesis of Alzheimer disease. During this time I became very much interested in the physiological and pathological functions of the microtubule associated protein tau, which is hyperphosphorylated and aggregated in the Alzheimer disease brain as neurofibrillary tangles.

Tau exon 10 is an alternatively spliced exon. In the normal adult human brain, the ratio of exon 10 excluded (3 Repeat tau) and included (4 Repeat tau) is ~1:1. Dysregulation of exon 10 splicing is associated with several age-related neurodegenerative disorders called tauopathies. Individuals with Down syndrome caused by trisomy 21 develop Alzheimer disease histopathology as early as the fourth decade of the life. The extra gene dosage of amyloid precursor protein (APP) could explain the early deposition of β-amyloid plaques. However, the occurrence of neurofibrillary tangles in Down syndrome brain is not understood—although neurofibrillary tangles are a hallmark of Alzheimer histopathology.

Dyrk1A lies in the Down syndrome critical region of chromosome 21 and contributes to several phenotypes of Down syndrome in transgenic mice. This prompted me to investigate the effect of overexpression of Dyrk1A on the alternative splicing of tau and the molecular sequence of events through which it contributes to early onset tau pathology in Down syndrome brain.

Read Dr. Liu's article entitled: Increased Dosage of Dyrk1A Alters Alternative Splicing Factor (ASF)-regulated Alternative Splicing of Tau in Down Syndrome

http://www.jbc.org/cgi/content/full/283/42/28660