Dear ISB,

I participated in Neuroscience 2005, Society for Neuroscience 35th annual meeting, arranged in Washington DC, USA 12-16 November 2005. The meeting spanning through five packed days of scientific programs and with over 35 000 participants was probably one of the largest scientific meetings this year. The meeting was filled with presentations, exhibits and networking among a diverse group of attendees representing every corner of the world. I presented my latest study in a poster format and obtained excellent feedback from a large group of visitors. The conference was very rewarding due to the positive feedback given on my work and the great possibility to discuss and exchange ideas with people working with the same systems as myself.

Best regards, Benny Björkblom

Conference homepage: http://apu.sfn.org/am2005

Abstract of study presented at the meeting: Normal functioning of the nervous system requires precise regulation of dendritic shape and synaptic connectivity. Here we report a severe impairment of dendritic structures in the cerebellum and motor cortex of JNK1-deficient mice. Using an unbiased screen for candidate mediators we identify the dendrite-specific high molecular weight microtubule-associated protein 2 (MAP2) as a JNK substrate in brain. We subsequently show that MAP2 is phosphorylated by JNK in intact cells and that MAP2 proline-rich domain phosphorylation is decreased in JNK1-/- brain. We developed compartmenttargeted JNK inhibitors to define whether a functional relationship exists between the physiologically active, cytosolic pool of JNK and dendritic architecture. Using these,we demonstrate that cytosolic but not nuclear JNK determines dendritic length and arbour complexity in cultured neurons. Moreover, we confirm that MAP2-dependent process elongation is enhanced upon activation of JNK. Using JNK1-/- neurons, we reveal a dominant role for JNK1 over ERK in regulating dendritic arborisation, while ERK only regulates dendrite shape under conditions where JNK activity is low (JNK1-/- neurons). These results reveal a novel antagonism between JNK and ERK potentiallyproviding a mechanism for fine tuning the dendritic arbour. Together, these data suggest that JNK phosphorylation of MAP2 plays an important role in defining dendritic architecture in the brain.