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18th International Conference on

Neurology and Neurological Disorders

August 23-24, 2018 | Paris, France

Chronic stress and moderate exercise prompt widespread common activation and limited differential activation in specific brain regions

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Chronic stress in rodents produces depressive behaviors, whereas moderate exercise counteracts stress-induced depressive behaviors. Stress and exercise appear to produce such opposing effects by changing the neural activity of specific brain regions. However, the detailed mechanisms through which the two different types of stimuli regulate brain function in opposite directions are not clearly understood. In the present study, we attempted to explore the neuroanatomical substrates mediating stress-induced depressive behavioral changes and anti-depressant effects of exercise by examining stimulus-dependent c-Fos induction in the brains of mice that were exposed to repeated stress or exercise in a scheduled manner. Systematic and integrated analyses of c-Fos expression profiles indicated that various brain areas, including the prelimbic cortex (PrL), parietal cortex (PaC), lateral septal nucleus (LS), and paraventricular nuclei of hypothalamus (PVN) were commonly and strongly activated by both stress and exercise, while the habenula (HB) and hippocampus (HP) were identified as being preferentially activated by stress and exercise, respectively. Exercise-dependent c-Fos expression in all regions examined in the brain occurred in both glutamatergic and GABAergic neurons. These results suggest that chronic stress and moderate exercise produce counteractive effects on mood behaviors, along with prompting widespread common activation and limited differential activation in specific brain regions.

Speaker Biography

Tae-Kyung Kim graduated from Korea University in South Korea with a bachelor's degree in biology. During graduate study at Rutgers University (Robert Wood Johnson Medical School), he studied the molecular mechanisms and regulation of eukaryotic gene expression under the supervision of Dr. Danny Reinberg (HHMI, currently at NYU). After obtaining a PhD degree in Biochemistry, he continued his research career in the laboratory of Dr. Michael Greenberg (Harvard Medical School) as a postdoctoral fellow, studying how neuronal activity controls gene expression in neurons to mediate synapse remodeling and plasticity. He joined the faculty in the Department of Neuroscience at UT Southwestern in 2010.

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