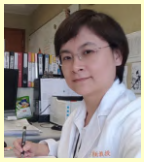
Cross-talk between environment and central nervous system



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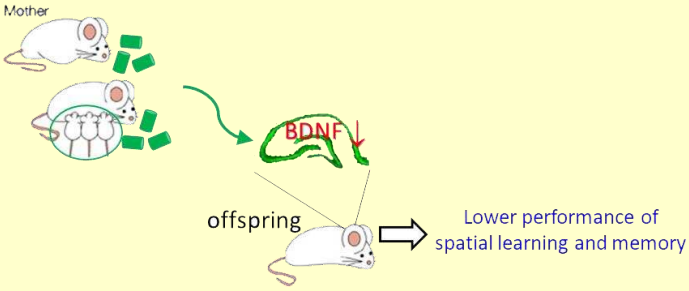
In response to environmental disturbances, the central nervous system (CNS) integrates the information obtained from the vital organs of the individual to maintain physiological functions. In my Lab, we are interested in how environmental stimulation alters the CNS function and progresses diseases. Mitochondria are the core of cellular energy support and play key roles in inflammation and cell death. We focus on revealing the roles of mitochondria in the initiation and progression of diseases and identifying possible interventions. Diet is the major energy source to maintain individual survival. We investigate the direct and transgenerational effects of high-fructose diet (HFD) as a long-term stimulation on the glia-neuron interaction and cognition. We dissect the underlying mechanisms of impaired learning and memory and seek the appropriate time windows for possible preventions and interventions. By working with a neurologist, we dissect the hyperglycemia-induced motor disorder by using the HFD model. For the short-term stress on the CNS, we collaborate with clinicians to explore the molecular mechanisms by using acute heart stroke animal models aiming to seek novel therapeutic strategies.

Research Project I

Fructose-programmed brain dysfunction

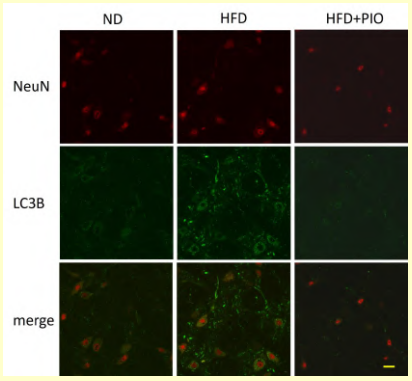
We investigate the underlying mechanisms of the fructose-programmed energy metabolism of the offspring by using the rodent model of maternal high fructose ingestion. Based on the exploring evidence, the candidate signals will be targeted for the prevention and intervention of the cognitive deficiency.



Research Project II

The molecular mechanisms of high fructose-associated autonomic neuropathy

By using a rodent model of high fructose diet (HFD) ingestion, we find out that central autonomic dysregulation contributes to HFD-induced early mortality. Disturbed insulin and energy sensing signals deteriorated autophagy dysfunction. The central autonomic dysregulation-associated early mortality was reversed by pioglitazone.

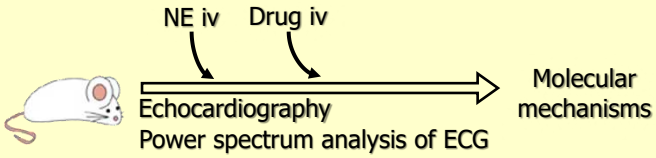


HFD induced the expressions of autophagy proteins in RVLM of adult male WKY at 18 weeks old. The representative images of LC3B accumulation (green) in RVLM neurons (NeuN, red) of ND, HFD, and HFD+PIO. Scale: 200 μm.

Collaboratory Research Project I

Therapeutic strategies in a murine model of catecholamine-Induced cardiomyopathy

We are cooperating with the director of the Children's Internal Medicine Prof. Lin IChun to study the pathophysiology of cardiomyopathy and neuropathy raised by norepinephrine. Based on the accumulating evidence, we aim to develop simple and direct diagnostic methods and more effective clinical medications for clinical therapy.



Collaboratory Research Project II

Elucidating the role of hyperglycemia in motor disorder

We are cooperating with Dr. Fu Mu-Hui, a neurologist, to explore the molecular mechanisms and the temporal profile of neuropathy of glycemic-associated motor disorder by using the maternal HFD rodent model. We uncovered different time windows of the treatments in various cell types in the brain. Based on the accumulating pieces of evidence, we aim to develop more effective therapeutic strategies for clinical therapy.