

CV of Invited Faculty



Yi-Cheng CHANG

Position	<ol style="list-style-type: none"> 1. Attending physician 2. Associate professor 3. Joint appointment Associate fellow
Department, Affiliation	<ol style="list-style-type: none"> 1. Division of Endocrinology and Metabolism, National Taiwan University Hospital, Taiwan 2. Graduate Institute of Medical Genomics and Proteomics, National Taiwan University, Taiwan 3. Institute of Biomedical Sciences, Academia Sinica, Taiwan
Major Field	Obesity, Diabetes, Ageing
Professional Activities: (Career or membership)	<p>Member, Asian Association for Diabetic Study</p> <p>Member, the Endocrine Society and Diabetes Association of the Republic of China (Taiwan)</p> <p>Member, Human Genome Organization</p> <p>Member, the Obesity Society (TOS)</p> <p>Member, American Association of Advancement of Science</p> <p>Founding Member, Taiwan Alcohol Intolerance Education Society</p> <p>Founding Member and Control Board, Taiwan Circulating Research Society</p>
Short Bio (in 150 words):	<p>Our team focus on translation research and preclinical drug development of metabolic disease including diabetes, obesity, sarcopenia, chronic kidney disease, and metabolic dysfunction-associated steatotic liver disease. We have published 149 scientific papers with 5,214 citations. We developed ALDH2 (acetaldehyde dehydrogenase 2) activators that reduce toxic aldehydes. These drugs effectively improve glucose homeostasis, prevent diet-induced obesity and fatty liver, alleviate chronic kidney disease and acute kidney injury, and diastolic and systolic cardiac dysfunction. We also focused on the identification of endogenous PPARγ</p>

Short Bio (in 150 words):

ligands, mostly are bioactive fatty acid, which can dramatically improve glucose homeostasis, prevent diet-induced obesity, reduce steatohepatitis, and prevent sarcopenia. We developed small-molecular drugs that can increase the level of endogenous PPAR γ ligand, which improve insulin sensitivity, reduce glucose intolerance, and prevent diet-induced obesity and fatty liver in mice (patented). We identified a new insulin signaling pathway to regulate GLUT4 endocytosis through GSK3 α and proof the efficacy of GSK3 α inhibitors in reducing insulin resistance in mice.

***The information will be shown on the website and conference materials only.**