

ENDOCRINOLOGY: Role of GPCRs and beta-arrestins in diabetes and obesity

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A postdoctoral position is available to study the role of G protein-coupled receptor (GPCR)- and beta-arrestin-dependent signaling pathways in type 2 diabetes and obesity. The project involves the generation and analysis of new mouse models which express novel classes of designer GPCRs in a cell-type specific fashion (Wang L, et al. *Annu Rev Pharmacol Toxicol* 61:421-440, 2021). The use of these designer GPCRs makes it possible to activate distinct G protein- or beta-arrestin-dependent signaling pathways in a conditional and reversible fashion. Additional novel mouse models that lack distinct GPCRs or downstream signaling molecules including beta-arrestin-1 and -2 in a cell-type specific fashion will also be generated and analyzed (Pydi SP, et al. *Trends Endocrinol Metab* S1043-2760(20)30232-0. doi: 10.1016/j.tem.2020.11.008 (2020)).

The primary focus of the work will be on how GPCRs and beta-arrestins regulate the function of fat, liver, pancreatic alpha/beta cells, and certain types of neurons to maintain proper glucose and energy homeostasis. Mice will be analyzed by physiological (metabolic), pharmacological, molecular, and biochemical techniques. The ultimate goal is to identify molecular pathways through which GPCRs and GPCR-associated proteins contribute to the pathophysiology of type 2 diabetes and obesity.

Background in mouse physiology/pharmacology and experience with genetically modified mice is required. Basic molecular biological and biochemical skills are also essential.

Tentative starting date: flexible

The salary and benefits are as per NIH regulations commensurate with experience.

Stipend for 1st year postdoc: ~USD 60,000/year

To apply, please send a cover letter with CV, bibliography, and names of three references (including e-mail addresses and telephone numbers) to:

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For more information, contact:

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