R. Cassidy[1], Y. Xu[2], Y. Lu[2], L. Mangieri[2], B. Arenkiel[3], Q. Tong[2]

Brain states and the hypothalamus: Generalized manifestations of hunger and anxiety produced by activation of lateral & paraventricular hypothalamic projections to the basal forebrain

[1]Department of Psychiatry and Behavioral Sciences, VUMC
[2]The University of Texas Health Science Center at Houston
[3]Baylor College of Medicine

"Behavior", narrowly defined, is the multi-muscle motor output produced by activating a neural circuit - the most famous example being that of the gill-siphon retraction reflex in Aplysia californica. Such circuits may have their activation threshold altered to adapt to the environment. In this case, intracellular protein modifications occur after repeated activation to raise the threshold. Vertebrate brains abstract this principle; activation of certain diffusely projecting neurocircuits raise or lower the threshold of most other neurocircuits, collectively producing a "brain state". For example, "asleep" and "awake" are generated by the antagonistic action of orexin- and melanin-concentrating hormone-expressing hypothalamic neurons. My PhD research, presented in brief, explores this principle in mice. Our laboratory discovered two hypothalamic neurocircuits which produce "hunger" and "anxiety" brain states via their action on the attention circuits of the basal forebrain. The circuits are the lateral hypothalamus (GABA) --> medial septum/diagonal band of Broca (GABA) & the paraventricular hypothalamus (glutamate) --> lateral septum (GABA). This contrasts well with research demonstrating that specific basal forebrain projections only alter context-specific eating. Understanding the origins of hunger and anxiety brain states will improve the care of disordered eating, both obesogenic and leptogenic.

Acknowledgments: This work was funded by NIDA F30 DA047030-01A1, CCTS TL1 4TL1R000369-10. Many thanks to all members of my PhD laboratory and my advisory committee in Houston.