Molecular Neurobiology of Social Bonding

Understanding the neural mechanisms underlying normal social interactions and social attachments may have important implications for understanding psychiatric disorders with impairments in social behavior, including autism spectrum disorders. We have been performing comparative studies in rodent species with divergent social organization to gain insights into the neural circuits and molecular mechanisms that regulate complex social behaviors, including social bonding between mates. Comparative studies in socially monogamous prairie voles and promiscuous montane and meadow voles suggest that the neuropeptides oxytocin and vasopressin play important roles in social bonding bonding. Oxytocin and vasopressin, acting through their respective receptors, facilitate social interactions, are involved in the neural processing of social cues, and are critical for pair bond formation. Species differences in social organization appear to be the result of species differences in oxytocin and vasopressin receptor gene expression patterns in the brain. Highly affiliative and socially monogamous prairie voles have high levels of receptor expression in the nucleus accumbens and ventral pallidum, regions involved in regulating reward and reinforcement, while non-monogamous species do not. Infusion of oxytocin or vasopressin antagonists into these regions blocks pair bond formation. Over expressing the vasopressin receptor in the ventral pallidum of non-monogamous species using viral vector gene transfer results in the expression of pair bonds. Polymorphisms in a repetitive element in the promoter of the vasopressin receptor gene may contribute to both species differences and individual variation in receptor expression patterns and social behavior. Male prairie voles with longer microsatellites express higher levels of paternal care and are more likely to form a pair bond than males with shorter microsatellites. I will discuss studies in humans suggesting that the oxytocin and vasopressin system also modulate social behavior in humans in ways remarkably parallel to our discoveries in voles.
Narrative Biographical Sketch:

Dr. Larry J. Young is a William P. Timmie Professor in the Department of Psychiatry and Behavioral Sciences at the Emory University School of Medicine, and Division Chief for the Center for Behavioral Neuroscience at Yerkes National Primate Research Center. Dr. Young is also Director of Graduate Studies for the Emory Neuroscience Graduate Program. Dr. Young received his undergraduate degree in biochemistry at the University of Georgia and earned his Ph.D. in the laboratory of David Crews in the Department of Zoology at the University of Texas in Austin. Dr. Young received his post-doctoral training with Dr. Thomas Insel in the Department of Psychiatry at Emory University.

The goal of Dr. Young’s research is to understand how genetic, cellular and neurobiological mechanisms regulate complex social behavior, including social cognition and social bonding. His research focuses heavily on the roles of the neuropeptides oxytocin and vasopressin in regulating the neural processing of social signals and social attachment. Dr. Young uses a comparative neuroethological approach to investigate the nature of social bonding in highly affiliative and socially monogamous prairie voles. His work incorporates a comprehensive genetic approach that involved genetic manipulation of both mice and prairie voles. This work has led to the development of neural model of social bonding which shares many features with addiction. In addition, his research provides insights into the evolution of social behavior at a molecular level, as well as into genetic mechanisms underlying individual variation in social behavior. His research demonstrates that variation in the expression of neuropeptide receptor genes in specific brain regions contributes to the diversity in social behavioral phenotypes, both among species and between individuals of a species. This work has important implications for understanding the neurobiology of psychiatric disorders with disruptions in social behavior, such as Autism Spectrum Disorders.

Selected References:

