Successful social interactions with others are crucial for healthy relationships in social species, including humans. They allow for effective cooperation and learning from the experiences of others, forming the basis for proper functioning in society. However, individuals differ in the level of sociability, i.e., how much time they voluntarily spend with others, and the observed individual differences in sociability are stable. Decreased or increased sociability is a symptom of many disorders. At the lower end of the continuum, there are social withdrawal, indifference, and anhedonia; at the higher end, there is indiscriminate attachment. The symptoms are caused by brain disorders diagnosed as depression, schizophrenia, or autism spectrum disorders. We know little, however, about **how individual variation in sociability in health and disease is represented and maintained in the brain**. **Which neuronal circuits mediate this variability, and what is the physiological range of their activity? What are levels of their activation that are already pathological?**

Testing causal relationships between neural circuit function and behavior is a technical and ethical challenge in humans. Fortunately, these problems can be avoided by studying the mechanisms of social behavior in mouse models. As mice display a variety of social behaviors, they are widely used to study the mechanisms of such behavior. Recently, in my research group, we have developed methods for recording and analyzing the social behavior of individual mice living in a group, allowing for identifying stable individual traits. Our observations of the spontaneous social behavior of mice indicate that individuals differ in the level of social motivation, i.e., the willingness to establish social relationships. The development of such a tool allows the study of the behavioral and neural mechanisms underlying the natural variability of social behavior. We also identified neural circuits involving several brain structures, including the central amygdala (CeA), the ventral tegmental area (VTA), the anterior cingulate cortex (ACC), and the orbitofrontal cortex (OFC) involved in initiating and maintaining social interactions. **We propose to use our behavioral research methods and the knowledge we have gained about the neural circuits that control social behavior to explain the brain mechanisms underlying differences in sociability.**

In the project, we plan to characterize the dynamics and the physiological range of activity of these neural circuits, which will open the way to developing therapeutic strategies for disturbed social behavior. We plan to establish a therapeutic strategy based on manipulating the activity of characterized neural circuits, which we will test in mouse models. In the longer term, with the rapid advances in brain stimulation methods, it is conceivable that the knowledge accumulated in this project will also be applicable in treating social interaction disorders in humans.