A key feature of the central nervous system is its ability to process multimodal sensory information. The ensembles of neurons that permit the performance of such a challenging task are established during development. In the somatosensory cortex, GABAergic interneurons born in the ventral telencephalon undergo protracted migration to reach their final position in the cortex and acquire stereotypic morphologies before contacting their targets, the pyramidal cells and other interneurons. However, the cellular and molecular mechanisms that regulate interneuron maturation are poorly understood. In my talk, I will discuss the mechanisms by which gene expression and neuronal activity regulate the establishment of cortical interneuron subtypes, as well as their requisite laminar targeting and morphology. These questions will be examined through a combination of mouse genetics and physiology. The development of cortical interneuron diversity appears to arise through two sequential steps. We hypothesize that the initial specification of interneurons occurs at the progenitor stage and utilizes intrinsic genetic programs to divide them into approximately seven “cardinal” groups. Upon becoming postmitotic, immature interneurons migrate and integrate within distinct cortical regions. As they integrate into cortical plate, we have recently demonstrated that activity is selectively required for their selection of cortical laminae and morphological maturation. We suspect they are also receiving further signals that aid in their appropriate regional differentiation. Our hope is that through these approaches the cellular logic underlying circuit maturation can be understood and provide a systematic approach for the visualization and targeted manipulation of interneuron excitability. As we believe that dysfunction of cortical interneurons underlies a variety of affective neurological disorders, our approach provides the means to explore the etiology of brain pathology.