Oligodendrocytes are the myelin-forming cells of the central nervous system (CNS). Neurofibromatosis Type 1 (NF1) is an autosomal dominant disease affecting around 1:3500 people worldwide. In NF1 patients, white matter (myelin) tracts are enlarged, oligodendrocyte proteins are expressed in pilocytic astrocytoma tumors, and myelination defects have been proposed to account for the existence of hyper-intense spots on brain MRI. The Ratner lab previously showed that oligodendrocyte progenitor numbers are amplified when Nf1 is mutant in vitro and in vivo (Bennett et al., J. Neurosci., 2003). Now, they have several tamoxifen-inducible mouse model systems that allow specific loss of Nf1 in vivo within either progenitor or mature oligodendrocytes in order to examine whether cellular or signaling defects result in aberrant increase of these cells within the brain. With these models, I studied the effect(s) Nf1 loss has on oligodendrocyte growth and differentiation, and tumor formation, using histology and MRI imaging.