For most brain malignancies, current treatment options have limited effectiveness, resulting in poor prognosis. To better understand how diffuse gliomas develop, Dr. Fortin and colleagues generated and analyzed mouse and cellular models of specific disease subtypes. In pediatric diffuse midline gliomas (DMGs), the team found that mutations in \textit{ACVR1} are sufficient to arrest cell differentiation, can initiate tumorigenesis alongside cooperating genetic lesions, and can be therapeutically targeted. In ongoing studies, the team is employing functional genomics tools to identify new treatment avenues for DMGs. Furthermore, they are using a similar integrative strategy to investigate how \textit{IDH1}-mutated diffuse gliomas emerge, progress, and respond to therapy.