Neuronal ceroid lipofuscinosis type 2 (CLN2) is an autosomal recessive, neurodegenerative lysosomal storage disorder due to a deficit of the metabolic enzyme tripeptidyl peptidase (TPP-1). Due to the lack of TPP-1, an aggregation of lysosomal waste leads to neurological complications and early death. Currently, a clinical trial of the experimental treatment Brineura® (cerilponase alfa) is the only approved treatment for CLN2. Cerilponase alfa is recombinant TPP-1, which is delivered directly to the brain via intracerebroventricular infusion. Diffusion models suggest TPP-1 will spread along a concentration gradient through the brain. We are examining proliferation, cell cycle kinetics, differentiation, and cell death in human neural stem/progenitor cells (hNPCs) following overexpression or exposure to human recombinant TPP-1. These results may have indications in clinical trials using intracerebroventricular infusion for enzyme replacement therapies and dosing.