Patients undergoing chemotherapy often report disturbances in their ability to taste. These deficits can lead to decreased appetite, malnutrition, and ultimately poorer prognoses. Previous work in this lab has examined the effects of the chemotherapeutic cyclophosphamide (CYP) on the taste system of mice. Initial analyses have shown biphasic deficits in taste sensitivity and acuity through behavioral measures. Immunohistochemical assays have shown a similarly biphasic decrease in markers for taste bud cell types when examining dates corresponding to behavioral losses (Days 0, 4, 8, 10, 12, 16, and 21). Additionally, increased cell death and decreased proliferation are seen following CYP administration. It is our aim to expand upon these initial findings to more fully understand the extent to which the taste system is compromised and the mechanisms involved in the recovery of function. Subsequent analyses will take a closer look at the taste bud recovery by performing IHC for markers of cell type, cell death, and proliferation on tissue taken from each day following CYP administration. To attempt to understand how this regeneration is taking place, we will also determine the role of Sox2, an important developmental transcription factor involved in the embryogenesis of the taste system, in this adulthood recovery process. To do this, we propose viral modulation of Sox2 expression preceding CYP administration using adeno-associated and lentiviral vectors. Following CYP injury, differences in the recovery process and taste bud cell type distribution between Sox2 overexpression, Sox2 knockout, and normal Sox2 expression will help determine the functions for which it is sufficient and necessary.