Repression of GAP-43 expression by cyclic AMP in the neuronal cell line RN46A.<u>D.J. Schreyer</u>¹, P.L. Andersen¹ and S.R. Whittmore². ¹Dept of Anatomy and Cell Biology, Univ of Saskatchewan, Saskatoon, Canada and ²Dept of Neurological Surgery, Univ of Miami, USA.

Neurons of the adult mammalian central nervous system (CNS) display poor regenerative growth following axon injury. Regenerative failure has been attributed to growth inhibitors associated with CNS myelin. It is not known whether CNS myelin growth inhibitors act only at the level of the growth cone, or additionally influence neuronal gene expression which supports growth. Using a cell-ELISA assay, we show that expression of the growth associated protein GAP-43 is normally detectable in RN46A cells, a cell line derived from E13 raphe nucleus neurons. RN46A cell expression of GAP-43 is markedly suppressed by chronic exposure to cholera toxin, forskolin, cAMP analogues and phosphdiesterase inhibitors. DNA assays performed on sister cultures indicate that the changes in GAP-43 are not due to changes in cell survival. These findings suggest that extracellular influences may inhibit GAP-43 expression in CNS neurons via a G-protein linked receptor and adenyl cyclase.

Supported by MRC Canada and NS26887.