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DEPARTMENT OF MEDICAL NEUROSCIENCE

TITLE OF THESIS:	THE INFLUENCE OF NEURAL CELL ADHESION MOLECULE ON AGE-RELATED CHANGES IN VISION
TIME/DATE:	9:30 am, Monday, March 20, 2017
PLACE:	Room 3107, The Mona Campbell Building, 1459 Lemarchant Street

EXAMINING COMMITTEE:
Dr. Adriana Di Polo, Department of Neuroscience, University of Montreal, Southwestern (External Examiner)

Dr. William Currie, Department of Medical Neuroscience, Dalhousie University (Reader)

Dr. William Baldridge, Department of Medical Neuroscience, Dalhousie University (Reader)

Dr. David Clarke, Department of Medical Neuroscience, Dalhousie University (Supervisor)

DEPARTMENTAL REPRESENTATIVE:	Dr. Angelo Iulianella, Department of Medical Neuroscience, Dalhousie University
CHAIR:	Dr. Meinhard Doelle, PhD Defence Panel, Faculty of Graduate Studies

ABSTRACT

The Neural Cell Adhesion Molecule (NCAM) is involved in cell migration, axonal fasciculation, neurite outgrowth, and the formation and stabilization of synapses during development. It also plays an important survival role in the adult nervous system. There has been extensive research focusing on how NCAM affects age-associated cognitive decline; however, little is known concerning the effect of NCAM in the visual system. Using a battery of behavioral, functional, and anatomical assays, I investigated the visual function of young and aged wild type and NCAM deficient (−/−) mice. My results provide evidence that NCAM −/− mice have altered retinal architecture and physiology, impaired pattern discrimination ability, and premature loss of visual acuity during aging.

These observations lead me to further investigate whether NCAM plays a role in protecting retinal neurons following injury in the adult mouse. Using light-induced retinal degeneration, I found that NCAM protects retinas from light-induced injury, and that the protective effect of NCAM is, in part, attributed to its effect on $p75^{NTR}$. To determine whether NCAM is involved in visual system plasticity, I subjected adult mice to long-term monocular deprivation, and demonstrated that PSA-NCAM is required for the reactivation of visual cortical plasticity and recovery of visual function.

Together, I have shown that NCAM plays vital roles in promoting retinal cell survival and in maintaining visual physiology in the nervous system during aging.