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## The "Combined" 192 IGG Saporin Lesion Approach as an Animal Model of Alzheimer's Disease

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Poster Presentation 1

THE "COMBINED" 192 IGG SAPORIN LESION APPROACH AS AN ANIMAL MODEL OF ALZHEIMER'S DISEASE

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While significant progress has been made toward an understanding of the etiology of AD, presently no reliable animal model exists that mimics the profound pathological and behavioral changes that characterize the disease. Accumulating evidence from a large number of studies conducted in the rat reveal that disruption of the functional integrity of the cholinergic basal forebrain projection to the hippocampus and cortex using cholinergic antagonists or specific lesions of the medial septal area (MSA) and nucleus basalis magnocellularis (NBM) induce marked impairments on a variety of behavioral tasks, particularly those that involve spatial learning (e.g. Morris water maze, and radial arm maze). Recently, 192 IgG saporin has been reported to be a selective cholinergic neurotoxin. Unilateral injections of 192 IgG saporin into the lateral ventricles induce a 80–90% reduction of acetylcholine levels in the cortex and hippocampus. Although several studies have reported an impairment of spatial learning following intraventricular injections of saporin, all have noted that the effects observed may be due to loss of cerebellar Purkinje cells damaged following intraventricular (i.c.v.) injections of 192 IgG saporin. Indeed, in a recent article published by Walsh, Kelly, Dougherty, Stackman, Wiley, and Kutscher in the journal *Brain Research* (702: 233–245, 1995) the authors conclude that although 192 IgG saporin is a highly selective cholinergic toxin, the secondary effects induced by i.c.v. injections of 192 IgG saporin "makes the i.c.v. model of 192 IgG-saporin problematic for studying the role of the cholinergic basal forebrain (CBF) in normative behavior and in disease states". The authors further suggest that site-specific injections of 192 IgG saporin would provide a viable approach to model Alzheimer's disease. In order to circumvent the problem of cerebellar Purkinje cell damage following i.c.v. injection of 192 IgG saporin, while at the same time producing a cholinergic lesion that essentially destroys 80–90% of the cholinergic input to the hippocampus and cortex in the rat, we have employed a "combined lesion" technique where animals receive three injections of 192 IgG saporin into the medial septal area, and two (bilateral) injections into the nucleus basalis magnocellularis. In a series of studies that will be presented at this conference, the effects of this "combined" lesion approach on spatial learning in the rat will be reported. We hope that the "combined lesion model" using 192 IgG-saporin by circumventing the inherent problems associated with i.c.v. injections of saporin may provide an avenue to test novel therapeutic drugs to be used as a palliative treatment for Alzheimer's disease.