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### Studies of Immunoglobulin G in the Aetiology of Myasthenia Gravis.

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An impairment in neuromuscular transmission is a characteristic feature of patients with myasthenia gravis, an autoimmune disease (Simpson, 1960). Attempts to isolate the 'active' factor from the thymus gland, muscle and serum of myasthenic patients have all proved unsuccessful. Toyka and coworkers (1976) have postulated that immunoglobulin G (IgG) is the direct, acting antibody against the mammalian acetylcholine receptor (ACh-R) following their successful 'passive' transfer of myasthenia gravis from man to mouse.

Daily injections (i.p.) of IgG (10-13 mg/ml) from either individual or 'pooled' batches of myasthenic sera from patients with Group I to V symptomology for 3 to 15 days produced no impairment in neuromuscular transmission in the soleus, diaphragm or intercostal muscles of LACA, B10D2/f<sub>1</sub> or B6D2/f<sub>1</sub> mice. Furthermore, exposure of neonatal and adult mouse isolated diaphragm and intercostal neuromuscular preparations to myasthenic IgG at concentration of 1-1000µg/ml produced no impairment in neuromuscular efficacy after 30 hours of continuous perfusion. These results will be discussed with regard to the titres of ACh-R antibodies detected in the sera of the myasthenic patients studied. Interestingly, B6D2/f<sub>1</sub> mice given only one injection (i.p.) of sheep ACh-R antibody showed both impaired neurotransmitter release and fragmentation of the neuromuscular junction.

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