Robert C. Griggs, M.D.

Dr. Griggs is Professor of Neurology, Medicine, Pathology and Laboratory Medicine and Pediatrics at the University of Rochester School of Medicine and Dentistry. He was Chair of the Department of Neurology and Neurologist-in-Chief at Strong Memorial Hospital (1986-2008). He received his BA from the University of Delaware and his M.D. from the University of Pennsylvania. He received training in Internal Medicine at Case Western Reserve University and the University of Rochester where he was Chief Resident in Medicine and Fellow in Immunology. He trained in Neurology at the National Institute of Neurological Disorders and Stroke (NINDS) and the University of Rochester where he was Chief Resident in Neurology.

Dr. Griggs is an internist/neurologist specializing in neuromuscular diseases with a focus on experimental therapeutics. He has directed an NIH-funded training program in the Experimental Therapeutics of Neurological Disease since 1989. This program has trained over 50 clinical neuroscientists who are in positions around the world. He has published over 300 scientific papers and 19 texts which span the fields of medicine and neurology. He served as President of the Association of University Professor of Neurology (1994-1996). He has served on the Council of the American Neurological Association. He served as Editor-in-Chief of Neurology (1997-2007). He is Neurology Editor of Cecil Textbook of Medicine and an Editor of Cecil Essentials of Medicine. In 1998 he received the Robert Wartenberg Award and Lectureship of the AAN. In 2004 he delivered the Soriano Lectureship of the American Neurological Association. He delivered the inaugural Victor Dubowitz Lecture at the National Hospital (Queen Square) in 2008. He was elected to the Institute of Medicine of the National Academy of Sciences in 1998. He is currently President (2009-2011) of the American Academy of Neurology.

Since 1998, Dr. Griggs has chaired the Executive Committee of the Muscle Study Group (MSG), an international consortium of investigators focused on developing new treatments for neuromuscular disease. He is the Principal Investigator of the Consortium for the Investigation of Neurological Channelopathies (CINCH) in the Rare Disease Network. CINCH is focused on developing new and better treatments for the channelopathies: Andersen-Tawil syndrome (a form of periodic paralysis), other periodic paralyses, the episodic ataxias and the non-dystrophic myotonias. CINCH has also trained 22 fellows at Rochester and in other channelopathy centers around the world: London(UK), Harvard, NINDS, UCLA, UCSF, UT (Dallas), and the U of Kansas.
The primary periodic paralyses: diagnosis, pathogenesis and treatment.


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Periodic paralyses (PPs) are rare inherited channelopathies that manifest as abnormal, often potassium (K)-sensitive, muscle membrane excitability leading to episodic flaccid paralysis. Hypokalaemic (HypoPP) and hyperkalaemic PP and Andersen-Tawil syndrome are genetically heterogeneous. Over the past decade mutations in genes encoding three ion channels, CACN1AS, SCN4A and KCNJ2, have been identified and account for at least 70% of the identified cases of PP and several allelic disorders. No prospective clinical studies have followed sufficiently large cohorts with characterized molecular lesions to draw precise conclusions. We summarize current knowledge of the clinical diagnosis, molecular genetics, genotype-phenotype correlations, pathophysiology and treatment in the PPs. We focus on unresolved issues including (i) Are there additional ion channel defects in cases without defined mutations? (ii) What is the mechanism for depolarization-induced weakness in Hypo PP? and finally (iii) Will detailed electrophysiological studies be able to correctly identify specific channel mutations? Understanding the pathophysiology of the potassium-sensitive PPs ought to reduce genetic complexity, allow subjects to be stratified during future clinical trials and increase the likelihood of observing true clinical effects. Ideally, therapy for the PPs will prevent attacks, avoid permanent weakness and improve quality of life. Moreover, understanding the skeletal muscle channelopathies will hopefully lead to insights into the more common central nervous system channel diseases such as migraine and epilepsy.

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