Diseases of the Nervous System Associated with Calcium Channelopathies
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Propositions

1. The auxiliary subunits $\alpha_2\delta$-2, $\beta_4$, and $\gamma_2$ of CaV2.1 channels are dispensable for neurotransmitter release at the NMJ synapse.  
   \textit{(This thesis)}

2. \textit{Cacna1a} conditional knockout mice will be instrumental in dissecting the cell-specific role of CaV2.1 channels in motor behaviour.  
   \textit{(This thesis)}

3. Dysfunction of Purkinje cells, rather than cerebellar granule cell neurons, is the cause of the abnormal cerebellar morphology and ataxia seen in CaV2.1 $\alpha_1$ knockout mice.  
   \textit{(This thesis)}

4. Increased, not decreased, irregularity in Purkinje cell simple spiking results in motor deficits and cerebellar ataxia.  
   \textit{(This thesis)}

5. The fact that the FHM1 S218L gain-of-function mutation causes cerebellar ataxia compromises the hypothesis that only loss-of-function mutations in \textit{CACNA1A} cause this phenotype.  

6. Drugs such as aminopyridines and EBIO that restore normal Purkinje cell function in experimental animal models, deserve to be tested in prospective clinical trials of patients with cerebellar ataxia  

7. Modulation of calcium channel function by G-proteins and auxiliary subunit interaction is perhaps more relevant to explain disease pathology than direct biophysical consequences of \textit{CACNA1A} mutations on these channels.  

8. “Neurological disorders are among the greatest threats to public health.”  
   \textit{(Neurological Disorders: Public Health Challenges. World Health Organisation, 2006)}

9. “Translational research means different things to different people, but it seems important to almost everyone.”  
   \textit{(Woolf SH, JAMA 2008; 299:211-213)}

10. “The real act of discovery consists not in finding new lands, but in seeing with new eyes.”  
    \textit{(Mascel Proust)}