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An iconic ionic transporter: function and failure of the Na,K-ATPase

Neuronal signalling depends on the steep gradients of sodium and potassium across the plasma membrane, and the majority of the brain’s energy is spent by the Na,K-ATPase to maintain these gradients. In the past decade, huge advances have been made in our understanding of how the pump functions as a molecular machine, and novel light is being shed on the physiological roles of the different isoforms of the catalytic Na,K-ATPase subunit by the severe pathophysiological effects of mutations in the genes encoding it. Three neurological diseases originally described as phenotypically distinct have been linked to mutations in a single gene, but it remains unknown why the mutations cause phenotypically different diseases. In this presentation, I will discuss the pump’s basic ion transport mechanism and the effects of disease-causing mutations.