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extensively in order to provide innervation to all the muscle fibres they control. The motor neurone and the muscle fibres it supplies are called a ‘motor unit’. Motor units form in development when axons grow into their target muscles. Once a motor unit has reached its mature configuration, shortly after birth, it normally retains all its connections and they keep working for us for the rest of our adult lives. However, motor nerve endings are among the first components of motor neurones to degenerate in diseases like motor neurone disease, (MND, also called amyotrophic lateral sclerosis or ALS), a devastating progressive and fatal paralysing illness. We do not know why or how this neuromuscular synaptic degeneration occurs and there is no effective treatment or cure for MND/ALS.

Our working hypothesis is that different processes control the survival of motor nerve cell bodies and their dendrites and the mechanisms responsible are different from those that maintain axons or neuromuscular junctions. We are seeking evidence for or against this ‘compartmental neurodegeneration’ hypothesis by studying the development, function and regeneration of motor nerve endings in animal models of nerve degeneration and motor neurone disease. We mainly study a mouse strain called ‘WldS’ in which degeneration of axons and motor nerve endings occurs much more slowly than normal; and in the transgenic mouse strain ‘hSOD1(G93A)’ which carries the same aberrant gene as some patients with MND and in which motor nerve terminals spontaneously and rapidly degenerate as the disease progresses. One of our aims is to use these animal models to find novel ways of studying MND; another is to find ways of slowing down or arresting the degeneration of nerve endings in the disease.

We use a combination of physiological recording using microelectrodes and microscopical, live-imaging techniques for this research. We also collaborate with molecular biologists in various tests designed to understand how the WldS gene slows down axon degeneration; why it doesn’t work quite so well to protect motor nerve endings; and what other genes or gene alterations may be found that may slow down degeneration of motor nerve endings in MND. Ultimately, we are working towards human applications of our methods, and translating these into ways of monitoring new treatments for neuromuscular diseases such as MND/ALS.

D diagram showing the relationship of four different cell types at the mouse NMJ. Insets: ‘EPP’ showing typical endplate potentials recorded with an intracellular microelectrode from a mouse NMJ; plus two monochrome images of NMJ’s visualised four days apart by live-imaging, using a fibre-optic confocal microendoscope, in the SOD1 mouse model of MND.

C. Immunostained neuromuscular junction from a mouse showing axon (red), acetylcholine receptors (blue) and a recently-described capping-cell named a kranocyte (green).

Selected References


