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My interest in Neuroscience developed while carrying out electron microscopical studies comprising my M.Sc. studies at the University of Western Ontario. Subsequently during Ph.D. research at York University in Toronto, I utilized electrical stimulation of nervous tissue to increase neurohormone release as studied ultrastructurally and biochemically. From these studies I decided to learn more about neurophysiological techniques as research tools. As a post-doc at the Tulane Medical School with Dr. Ed Dudek, I studied the electrophysiology of mammalian neuroendocrine cells and used live hippocampal slices to examine electronic coupling among neurons.

This approach continued in my own lab, evolving into neurophysiological studies of neuronal swelling caused when neurons are osmotically challenged, become hyperactive (as during epilepsy) or are metabolically challenged (as during stroke). Recording intracellularly and extracellularly from neurons, glia and neuronal populations is combined with simultaneous imaging techniques measuring, for example, light transmittance through live brain slices to monitor tissue swelling and injury. Most recently I have been collaborating with Dr. Sergei Kirov using 2-photon scanning laser confocal microscopy that reveals volume changes and damage to single neurons and glial in real time. Individual brain cells can be imaged in transgenic rodents engineered so that a few cells are dramatically fluorescent on a dark background in the live state. We are particularly interested in how neurons and glia respond during the early period of simulated stroke or head trauma. We can then assess potentially therapeutic drugs that reduce or prevent acute neuronal injury.

One particular research interest is a phenomenon termed spreading depression (SD), a migrating depolarization of brain cells that can be imaged in neocortical and hippocampal brain slices. Spreading depression underlies the aura (e.g. flashing lights or numbness) preceding migraine headache and may actually be a cause of migraine pain, rather than just a symptom.

A second interest is a process similar to spreading depression termed anoxic depolarization (AD). As soon as

cortical tissue experiences severe metabolic stress (as during stroke or head trauma), AD generation contributes even more stress, damaging neurons wherever it propagates. AD-like events (peri-infarct depolarizations, PIDs) recur and expand brain damage in the hours following stroke or brain trauma. We are currently testing a family of drugs that dramatically reduce such damage in brain slices by blocking the AD. They also inhibit SD. I consider AD and PID inhibition to be the key targets for improving patient outcome following stroke or traumatic brain injury rather than the textbook explanation that blames excessive glutamate release ('excitotoxicity').



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