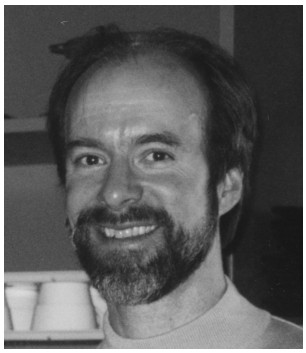
Paul F. Cranefield Award to David D. Friel



Paul F. Cranefield, M.D., Ph.D. was the Editor of *The Journal of General Physiology* for 30 years, from 1966 to 1995. During this time, he worked incessantly to further the mission of the journal: to promote and publish studies at the interface between biology, chemistry, and physics to obtain insights into fundamental mechanisms that underlie biological function at all levels.

When Paul stepped down as Editor, the Council of the Society of General Physiologists discussed how best to acknowledge his numerous contributions to *The Journal* and, thus, to the Society. In the end, it was decided to institute a Paul F. Cranefield Award, which should go to a young investigator who in the preceding year had published an article of exceptional quality in *The Journal*. The award would be given at the Annual Meeting and Symposium of the Society, which takes place in Woods Hole, Massachusetts. It also was decided that the criteria for selecting the awardee should be so stringent that the award might not be given every year.

I am pleased to say that despite these high standards, there were many outstanding candidates. Nevertheless, one stood out, namely David D. Friel, from the Department of Neuroscience at Case Western Reserve University School of Medicine, who has made many important contributions toward understanding cellular Ca^{2+} dynamics. Dr. Friel accepted the Award at the September 2001 meeting of the Society.

Dr. Friel received his Ph.D. from the University of Chicago, in the laboratory of Richard J. Miller, where he worked on the effects on neuropeptide Y on intestinal epithelial transport. He received his postdoctoral training with Bruce P. Bean, where he worked on ATP-activated currents (including what now is known as P2X currents), and with Richard W. Tsien, where he first provided conclusive evidence for anomalous mole fraction effects in single voltage-dependent calcium channels. Then, he began the work that has occupied him since then, namely, cellular Ca^{2+} dynamics and the interplay between plasma membrane and organellar Ca^{2+} movements. Cytoplasmic $[Ca^{2+}]$ in the presynaptic nerve terminal is an important regulator of synaptic transmission, and cytoplasmic Ca^{2+} oscillations in the postsynaptic neuron are important for long-term changes in synaptic transmission. The dynamics of the cytoplasmic Ca^{2+} oscillations are determined by both plasma membrane and organellar transport events, and Dr. Friel has pursued this important problem through technically demanding experiments and detailed modeling. Dr. Friel and his coworkers have, in a series of what will become classic articles on the Ca^{2+} dynamics in intact neurons, demonstrated how the mitochondria and the ER cooperate in the regulation of cytoplasmic $[Ca^{2+}]$. In the March 2000 issue of *The Journal* Dr. Friel and his colleagues (S.L. Colegrove and M.A. Albrecht) showed how the complex time course of the cytoplasmic Ca^{2+} transient after a depolarization-induced Ca^{2+} influx was influenced by two mitochondrial Ca^{2+} transporters: an early catalyzed diffusion into the mitochondrial matrix, mediated by a Ca^{2+} uniporter; and a subsequent active extrusion, mediated by a Na^+/Ca^{2+} exchanger. The plasma membrane Ca^{2+} influx dynamics was "simplified," by the use of controlled, steady depolarization pulses imposed by the permeabilized patch method, which made it possible to derive a relatively simple model of the combined action of the mitochondrial uniporter and exchanger. One of the unexpected conclusions of this work was that small depolarizations could alter Ca^{2+} in the mitochondrial matrix, with little effect on the cytoplasmic $[Ca^{2+}]$, which provides for a novel mechanism for coupling membrane activity to intracellular metabolism. We were most pleased to publish this work.

But that is not all, in two articles published in the July 2001 issue, M.A. Albrecht, S.B. Andrews, S.L. Colegrove, D.D. Friel, J. Hongpaisan, R.D. Leapman, and N.B. Pivarova explored the interrelationship between the ER and mitochondrial Ca^{2+} handling, in which they provided a comprehensive analysis of the contributions of the ER to cellular Ca^{2+} dynamics. One of the surprising results of this work was the demonstration of intracellular gradients in the Ca^{2+} loading by the ER. Moreover, the total Ca^{2+} load in the ER is much larger than in cytoplasm, whereas there is virtually no Ca^{2+} in the mitochondrial matrix under resting conditions. The ER and the mitochondria play important, but complementary, roles in cellular Ca^{2+} metabolism. The relative contributions of these, and other, components in the overall Ca^{2+} dynamics will occupy many investigators for years to come; but Dr. Friel has already demonstrated his position in the field.

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