Motoneurons, like other neurons, transform synaptic inputs into frequency-coded outputs. The rate of discharge is determined by the total amount of current reaching the soma (and the adjacent axon, where spikes are initiated). The amount of synaptic current transferred to the soma during increasing activation of excitatory synapses is limited by a progressive decrease in driving force and a progressive increase in membrane conductance [5]. Recent simulations of synaptic current transfer in motoneuron dendrites suggest that passive dendrites are incapable of delivering sufficient current to maximally activate the cell [8]. However, there is now a considerable body of experimental evidence that active conductances in motoneuron dendrites supply a persistent inward current that acts to amplify the synaptic current transferred to the soma [7]. The present report describes results from both experimental and simulation work that suggest potential mechanisms for this amplification.

In both turtle and mouse spinal motoneurons, the persistent inward current is mediated primarily by a calcium current flowing through L-type channels with a low voltage threshold for activation [3,4]. One sign of the dendritic location of the channels carrying the inward current is a clockwise hysteresis in the whole-cell current recorded in response to ascending then descending voltage ramp commands, i.e., the deactivation of the inward current on the descending ramp occurs at a lower voltage than activation on the ascending ramp [3,6,9]. This phenomenon is thought to arise from the fact that the membrane supplying the inward current is electrically distant from the soma and not under voltage clamp control. As a result, the distal L-channels are only activated at relatively high somatic depolarizations during the ascending voltage ramp command, but then continue to supply current to the soma during the descending ramp command [1, 3,6].

We characterized inward currents in rat hypoglossal motoneurons following following block of potassium currents with internal cesium and external 4-AP and TEA. Part of the current was due to a persistent sodium current blocked by TTX, while the remainder was mediated by calcium channels. We found roughly the same proportion of low-voltage-activated and P, N and L-type calcium currents that had previously been found in neonatal rat HG motoneurons [10]. Clockwise hysteresis in the I-V relation and long tail currents following voltage clamp steps were observed in about one third of the cells, consistent with a prominent contribution from dendritic channels. However, in contrast to previous reports on turtle and mouse motoneurons, these currents appear to be mediated largely by N- and P-type channels rather than L-type channels.

We conducted computer simulations of synaptic current transfer in complex dendritic trees whose structure was based on anatomical reconstructions of cat spinal motoneurons. The effects of asynchronous activation of excitatory synapses at different mean frequencies were approximated by varying the amplitude of a steady synaptic conductance applied throughout the dendritic tree. The current transferred to the soma was measured during somatic voltage clamp. Insertion of calcium channels with properties similar to those of L-channels amplified the current transferred to the soma during low frequency activation of excitatory synapses. However, at higher levels of activation, the production of dendritic plateaus shunted current flow from more distal areas and limited the amount of current transferred to the soma. Insertion of high threshold (N-type) calcium channels increased the operating range over which amplification occurred.

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