Supplementary Figure 1.

Reducing the density of g_{KLVA} and g_H in the axon initial segment did not alter the impact of axonal g_{Na} on somatic EPSPs. *A*, Passive properties measured with g_{KLVA}, g_{leak}, and g_H, but no g_{Na}. Step pulses (5 ms, -0.2 to 0.2 pA in 0.05 pA steps) were injected at the initial segment. The slope of the maximal voltage response plotted vs. current amplitude indicated the input resistance for the high axonal conductance condition (the same condition as shown in Figure 7) was 59 MΩ (V_{rest} = -61 mV). Lowering $g_{K\text{-}LVA}$ g_{leak}, and g_H an order of magnitude in the initial segment increased input resistance to 60.0 MΩ (V_{rest} = -61 mV). The additional increase of somatic input resistance (to 24.0 MΩ) through the reduction of all somatic and initial segment conductances by an order of magnitude increased the input resistance in the initial segment to 70.0 MΩ (V_{rest} = -57 mV). *B,* For the same model cell as in *A* (data also shown in Figure 7), two simulation configurations are indicated using a 2-dimensional morphological representation of the cell. An EPSG-like waveform at the soma evoked responses recorded at the soma (left panels) or at the distal initial segment (right panels). Sodium conductance was inserted in the initial segment (red). The amount of sodium conductance is indicated by line or trace color (S/cm^2). Sample traces show responses to EPSGs in 5 nS steps to threshold, set at a 12 mV EPSP at the soma. A plot of EPSP amplitude vs. EPSG amplitude (0 to 50 nS, 5 nS steps) is shown below. Asterisks indicate when action potentials were generated below a 12 mV depolarization at the soma. Sodium conductance in the initial segment greatly amplified EPSPs in the initial segment, but only prolonged the peak of the somatic EPSPs. *C,* Lowering the density of g_{KLVA}, g_{leak}, and g_H in the initial segment increased the amount of g_{Na}-dependent EPSP amplification in the initial segment, but

axonally-located g_{Na} still did not amplify the peak of somatic EPSPs. *C,* Lowering the density of g_{KLVA}, g_{leak}, and g_H in both the initial segment and soma greatly enhanced EPSP amplification in the axon initial segement. Moderate boosting of the somatic EPSPs occurred in extreme cases, in which near-electrogenic activity was displayed in both compartments. However, the somatic EPSP was highly distorted, which was never recorded in MSO neurons.

Supplementary Figure 2.

Properties of the voltage-dependent conductances used in the computational model. *A,* Voltage dependence of the activation gate for I_h (left panel). The fast (middle panel) and slow (right panel) time constants for the activation gate over the voltage range covered during computational simulations. *B,* Voltage dependence of the activation (black) and inactivation (red) gate for I_{Klva} (left panel). Time constants for the activation gate (middle panel) and inactivation gate (right panel) over the voltage range of the computational simulations. *C,* Voltage dependence of the activation (black) and inactivation (red) gate for I_{Na} (left panel). Time constants for the activation gate (middle panel) and inactivation gate (right panel) over the voltage range of the computational simulations.