Supplement 3. Hodgkin-Huxley model and a four-compartment model

Encoding in the Hodgkin-Huxley model

S3-Figure 1 and is analogous to S2-Figure 1 but for the Hodgkin-Huxley model. S3-Figure 1A shows the spike height against spike width for the same conductance stimulus we have used in the rest of the paper. For this model, there is variability mainly in the spike height, in contrast to our experimental data and to the Wilson model of cortical neuron. We have analyzed the results both by height (S3-Fig. 1) and width (S2-Fig. 2), giving similar results. As it is clear from S3-Figures 1B and 1C, there is a poor encoding of conductance stimulus history into the spike waveforms.

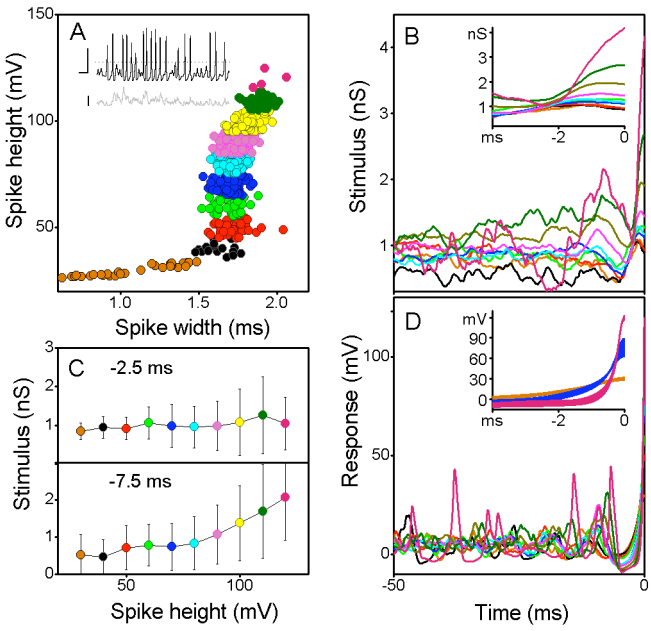

Figure S3-1: Action potential waveforms of the Hodgkin-Huxley model to naturalistic input during a conductance injection experiment. (A) The heights and widths of action potentials occupy a large nearly vertical range. Action potential waveforms (top inset; black trace) vary with synaptic input patterns of a sum of AMPA unitary events. Scale bars: 50 mV and 0.1 seconds. The thin horizontal line indicates the voltage (-43 mV) where the action potential widths were measured. The heights are color-coded into 10 groups (lower inset, 9 equally-spaced with the 10th including the remainder), from short to tall as: orange, black, red, green, cyan, blue, magenta, yellow, purple and olive. (B) Average stimulus (conductance) history preceding the peak of the action potentials for each group (same coloring). The inset shows the mean conductance before firing for each action potential groups. (C) Mean and SD of the conductance histories for each action potential group at 2.5 ms (upper window) and 7.5 ms (lower window) before firing. (D) Average voltage responses preceding the action potential waveforms (same grouping and coloring). The inset shows the mean and SD of the voltage response 4 ms before firing for three action potential groups.

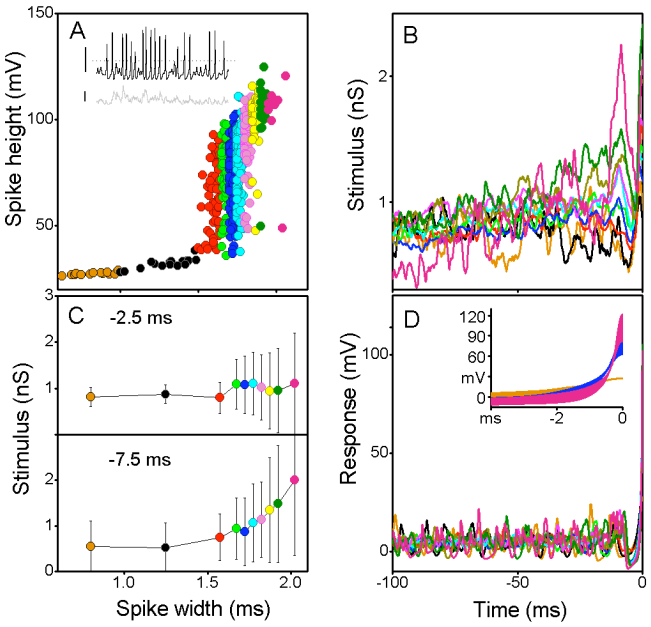

Figure S3-2: Action potential waveforms of the Hodgkin-Huxley model to naturalistic input during a conductance injection experiment. (A) The heights and widths of action potentials occupy a large nearly vertical range. Action potential waveforms (top inset; black trace) vary with synaptic input patterns of a sum of AMPA unitary events. Scale bars: 50 mV and 0.1 seconds. The thin horizontal line indicates the voltage (-42 mV) where the action potential widths were measured. The widths are color-coded into 10 groups (lower inset, 9 equally-spaced with the 10th including the remainder), from narrow to wide as: orange, black, red, green, cyan, blue, magenta, yellow, purple and olive. (B) Average stimulus (conductance) history preceding the peak of the action potentials for each group (same coloring). (C) Mean and SD of the conductance histories for each action potential group at 2.5 ms (upper window) and 7.5 ms (lower window) before firing. (D) Average voltage responses preceding the action potential waveforms (same grouping and coloring). The inset shows the mean and SD of the voltage response 4 ms before firing for three action potential groups.

Effect of dendritic sodium channels on spike waveforms

Modeling in the main text used only a single compartment. We have also considered models with several compartments to assess the effect of dendritic currents on spike waveforms. In a recent experimental and computational study it has been shown that the action potential waveform depends on the participation of dendritic sodium currents (Paré et al., 1998). These authors also showed that proximal inhibitory postsynaptic potentials could then prevent this dendritic participation and therefore reduce the amplitude of the spike at the soma. To investigate the effect of spatial compartmentalization of spike generation, we examined a simple model with four compartments, as illustrated in S3-Fig.3A. Our model has the same sodium and potassium conductances as the Wilson model in the main text for compartment C1, which represents the axon. Compartment C2, representing the soma, has 30% of the concentration of these channels. Compartments C3 and C4 represent the dendrite and 0% (B), 5% and 0%, respectively (C), or both 5% (D). Figures S3–Fig. 3 (B-D) give the spike height versus spike width for this model when stimulated by an AMPA conductance input in compartment C2 analogous to the one used in our experiments. It is clear from these figures that the dendritic sodium currents increase the spike height. However, the differences among (B)-(D) are smaller than the variability present in each of them. In this model, the effect of dendritic sodium currents on the spike waveform is therefore less than that of the input history, but not negligible. The details of the model are given in the Materials and Methods**.**

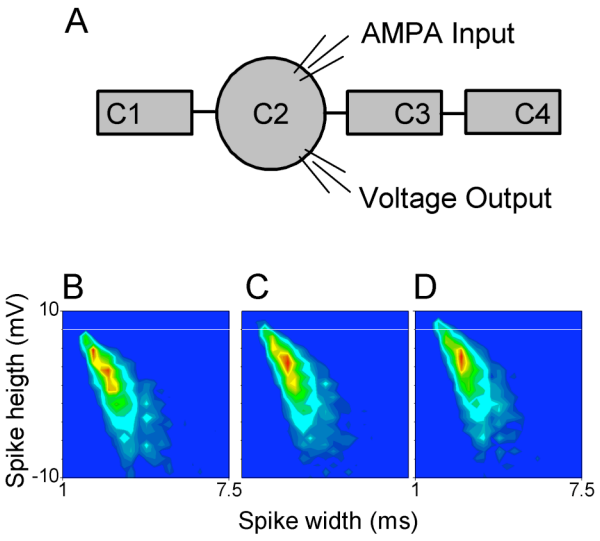

Figure S3-3: Test of the effect of dendritic sodium currents using naturalistic input in dynamic-clamp. (A) Four-compartment model used for the test. Compartment C1 has the same Na^+ and K^+ currents as the Wilson model in the main text. C2 has 30% of the Na^+ and K^+ currents. Compartments C3 and C4 represent the dendrite, modelled as having either 0% or 5% of the voltage-dependent currents of compartment C1. (B) Spike height versus spike width for the action potentials resulting from the model in a dynamic-clamp situation using naturalistic input. Compartments C3 and C4 have 0% of the currents in C1. (C) Same as (B) but C3 and C4 have 5% and 0% of the voltage-dependent currents in C1, respectively. (D) Same as (B) but C3 and C4 both with 5% of the Na^+ current in C1

References

Paré D, Lang EJ, Detexhe A (1998) Inhibitory control of somatodendritic interactions underlying action potentials in neocortical pyramidal neurons in vivo: Intracellular and computational study. Neuroscience 84: 377-402.