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One cell, two bursting mechanisms. *In vivo* conditions change the *in vitro* burst in pyramidal cells of the ElectroLateral Lobe (ELL) of electric fish

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The intrinsic mechanisms underlying burst generation *in vitro* where neurons are in relative isolation are generally well understood [1]. However, how these mechanisms are implemented under *in vivo* conditions where cells receive massive synaptic bombardment is still not clear. Pyramidal cells within the electrosensory lateral line lobe (ELL) of weakly electric fish have a well-defined burst mechanism *in vitro* (Fig. 1A), which is based on a somato-dendritic interaction [2]. Surprisingly, *in vivo* recordings from ELL pyramidal cells (Fig. 1B) do not show any of the characteristics associated with bursting found *in vitro* [3]. The goal of this project is to understand how *in vivo* conditions can give rise to these differences.

One of the striking differences between *in vivo* and *in vitro* conditions is the absence of glutamatergic input to the

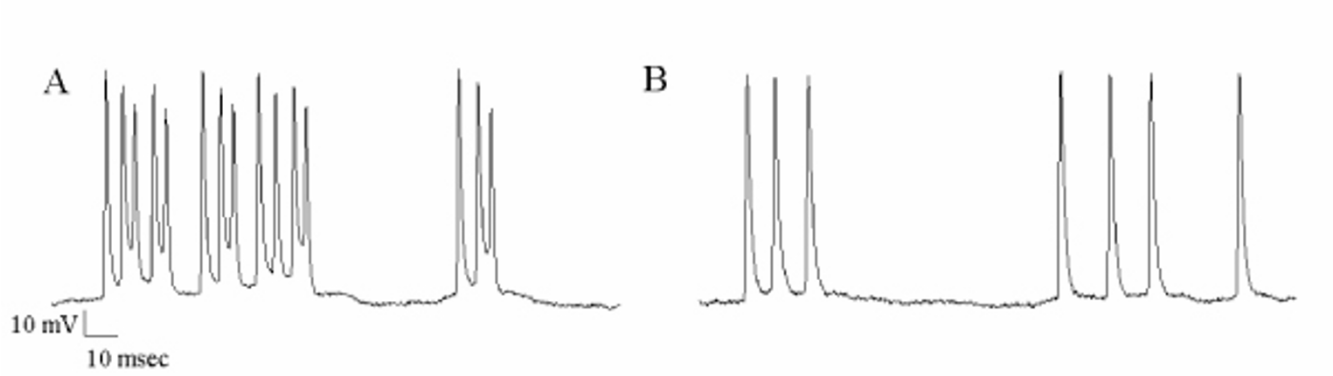

Figure 1
Bursting in the ELL pyramidal cells. (A) Representative trace of the *in vitro* recorded burst. Somatic spikes backpropagate into the dendrites, generating a dendritic spike which will move back into the soma, generating a depolarizing afterpotential (DAP), which will trigger a new somatic action potential The burst terminates with a characteristic doublet, followed by the burst after hyperpolarization (bAHP) (B) Representative trace of the *in vivo* recorded burst. Here there is no significant decrease in the interspike interval during a burst, bursts do not terminate with a doublet and the bAHP is absent. Also, the somatic spike shape recorded *in vivo* differs from the one recorded *in vitro*, with a pronounced hyperpolarization (AHP) following every spike within a burst.

cells *in vitro* which might provide the major source of Ca^{2+} to the cell via NMDA receptors. To test this hypothesis, we injected the calcium chelator, BAPTA, in pyramidal cell *in vivo*. The resulting removal of intracellular Ca^{2+} changed the cell bursting pattern to one characteristic of *in vitro* recordings.

To understand these observations, we have used a computational approach to propose a cellular mechanism for burst generation *in vivo*. In our computational model, which is based on the *in vitro* ghost-burst model, Ca^{2+} enters the cell through NMDA channels in the dendrites. When Ca^{2+} diffuses into the soma, it affects the Ca-activated potassium current. Gradual increases in Ca^{2+} concentration increases this current and eventually terminates the burst. This current also creates a spike shape characteristic to an *in vivo* burst, with a strong hyperpolarization after every spike within a burst.

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