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### **Activity-dependent long-term intrinsic plasticity in dentate gyrus granule cells**

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#### **Abstract:**

The concomitant roles of synaptic plasticity and neuron-specific intrinsic plasticity as cellular substrates of learning and memory are well established. The dentate gyrus (DG) has been implicated in spatial navigation, learning and memory. The mechanisms behind and implications for synaptic plasticity in the DG have been thoroughly investigated. In contrast, the assessment of the protocols, mechanisms and implications associated with DG intrinsic plasticity has been surprisingly limited. In this study, motivated by theta-modulated burst firing in DG granule cells, we investigated the ability of theta burst firing (TBF) in inducing activity-dependent intrinsic plasticity. We performed somatic whole-cell current-clamp recordings from DG granule cells in 6–8 weeks old male Sprague-Dawley rats. We assessed changes in several intrinsic properties by recording associated physiological measurements before and 40 minutes after the induction of TBF in DG granule cells ( $n=28$ ). In response to TBF, we observed a significant 16% reduction in input resistance ( $R_{in}$ ) accompanied by a contrasting increase in firing rate, measured as a significant leftward shift in the  $f-I$  curve ( $\sim 7$  Hz increase in response to a 250 pA current). Among other significant TBF-induced changes, we noted a depolarizing shift ( $\sim 4$  mV) in the resting membrane potential, a reduction in the maximal impedance amplitude ( $\sim 15\%$ ), a reduction in temporal summation measured in response to alpha excitatory current injections ( $\sim 5\%$ ), a hyperpolarizing shift in the action potential threshold ( $\sim 3$  mV) and the emergence of spike doublets ( $\sim 38\%$  reduction in the first inter-spike interval). Importantly, although opposing changes in sub- and supra-threshold excitability measurements point to changes in multiple channel properties, we found significant correlations across changes observed in these measurements. For instance, we found significant and strong correlation between the reduction in  $R_{in}$  and the hyperpolarizing shift in action potential threshold, indicating correlated changes in putatively distinct mechanisms that resulted in opposing effects on neuronal excitability. We are currently performing experiments to delineate ion channels that mediate this form of plasticity, apart from exploring potential correlations among molecular mechanisms that underlie the concurrent, yet contrasting, TBF-induced changes in sub- and supra-threshold intrinsic excitability. Finally, we are also addressing the question on whether heterogeneities in baseline neuronal excitability could play a dominant role in the recruitment of

neurons as engram cells through induction of intrinsic plasticity.

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