Modeling a transient thalamocortical circuit (L5b-L4 loop) in the developing mouse neocortex.

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The brain is a complex network of 10^{11} neurons with 10^{14} synapses. Our understanding of how such networks develop is still limited. Novel genetic manipulations and optical techniques make it possible to create detailed connectivity maps at different times during development. However, it is nearly impossible to study the mechanisms underlying the slow transitions from one developmental stage to the next experimentally. Here we use computer models of developing circuits to study the synaptic plasticity rules that effect the changes in network connectivity. In particular, we focus on an early thalamocortical circuit between thalamic afferents, interneurons in layer 5b and spiny stellate neurons in layer 4 (L5b-L4 loop). Unpublished work from the lab has shown that this circuit is transient in nature and shortly after the L4 critical period for plasticity has ended (~postnatal day 9) synaptic connections between these cells are replaced by connections previously reported for the canonical cortical circuits (Anastasiades et al., in revision; Marques-Smith et al., submitted). The computer model of this developing circuit is composed of approximately 1,000 integrate-and-fire neurons. Each neuron is classified as one of four distinct cell types (PV+, SOM+, 5HT3R+, and PYR) and their firing properties have been matched to experimental recordings. After obtaining static models of the circuit at each of its stages in development, we tested distinct synaptic plasticity rules that may underpin the dynamic formation and destruction of these transient developmental circuits. Ultimately, this type of models may enable a targeted exploration of developing circuits and provide a framework for translational work and a bridge between experimental and theoretical neuroscience research.

References

[1] Anastasiades PG, et al. Developmental phases of inhibitory connectivity in the early postnatal neocortex. *in revision*

[2] Marques-Smith A., et al. The involvement of a transient interneuron circuit in normal thalamocortical integration. *submitted*