

Thalamic inhibition is essential for critical period plasticity in the visual cortex

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During critical periods of development, experience shapes cortical circuits resulting in the acquisition of functions used throughout life. The classic example of critical period plasticity is ocular dominance (OD) plasticity which optimizes binocular vision but can reduce responsiveness of the primary visual cortex (V1) to an eye providing low-grade visual input. The onset of its critical period is thought to involve maturation of inhibitory synapses within V1, specifically those containing the GABA_A receptor alpha1 subunit. Surprisingly, we find that removing alpha1 from mouse thalamus, but not from cortex, disrupts OD plasticity in V1. Furthermore, we show that thalamic relay neurons in the dorsolateral geniculate nucleus undergo OD plasticity, which requires thalamic inhibition. Our findings demonstrate that in critical period regulation, thalamic inhibitory circuits are central. This has far-reaching consequences for the interpretation of studies investigating the molecular and cellular mechanisms regulating critical periods of brain development.