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A critical period for the itch spinal cord neural circuit?

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Itch is a widespread symptom associated with a diverse array of diseases. Despite its prevalence in the world population, our understanding of the development, maturation and mechanisms associated with itch neural circuits is lacking behind that of other somatosensory modalities. We have previously characterized the importance of spinal Ptf1a-derived inhibitory neurons in controlling the entry of innocuous mechanosensory information into the spinal cord dorsal horns. Loss of Ptf1a-derived adult neurons leads to the development of an intense chronic itch phenotype and increased hairy skin sensitivity. Here, we study the consequences of ablating Ptf1a-derived neurons both in early embryonic development, well before somatosensory input becomes active, and in newborns, when somatosensory circuits are being refined with the arrival of extrinsic stimuli. Our results show that postnatal loss of Ptf1a-derived neurons cannot be compensated, even as early as postnatal day 0, resulting in the development of chronic itch. However, elimination of these neurons during embryonic development leads to apparently normal itch sensation. The potential mechanisms explaining these observations point to the possible existence of a critical period for the establishment of functional itch circuits in the spinal cord. Moreover, these results suggest that loss of specific populations of inhibitory neurons in the dorsal horns activates endogenous compensatory mechanisms that yields a functionally normal mature spinal itch circuit.