Inhibition of noggin expression in the dorsal neural tube by somitogenesis: a mechanism for coordinating the timing of neural crest emigration.

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Sela-Donenfeld, D, Kalcheim C. 2000.

Abstract:

We have previously shown that axial-dependent delamination of specified neural crest cells is triggered by BMP4 and negatively regulated by noggin. Increasing activity of BMP4 towards the rostral part of the axis is achieved by graded expression of noggin in the dorsal neural tube, the latter being high opposite unsegmented mesoderm, and progressively downregulated facing epithelial and dissociating somites, coinciding in time and axial level with initial delamination of neural crest cells (Sela-Donenfeld, D. and Kalcheim, C. (1999) Development 126, 4749-4762). Here we report that this gradient-like expression of noggin in the neuroepithelium is controlled by the paraxial mesoderm. Deletion of epithelial somites prevented normal downregulation of noggin in the neural tube. Furthermore, partial ablation of either the dorsal half or only the dorsomedial portion of epithelial somites was sufficient to maintain high noggin expression. In contrast, deletion of the segmental plate had no effect. These data suggest that the dorsomedial region of developing somites produces an inhibitor of noggin transcription in the dorsal neural tube. Consistent with this notion, grafting dissociating somites in the place of the unsegmented mesoderm precociously downregulated the expression of noggin and triggered premature emigration of neural crest progenitors from the caudal neural tube. Thus, opposite the unsegmented mesoderm, where noggin expression is high in the neural tube, BMP4 is inactive and neural crest cells fail to delaminate. Upon somitogenesis and further dissociation, the dorsomedial portion of the somite inhibits noggin transcription. Progressive loss of noggin activity releases BMP4 from inhibition, resulting in crest cell emigration. We propose that this inhibitory crosstalk between paraxial mesoderm and neural primordium controls the timing of neural crest delamination to match the development of a suitable mesodermal substrate for subsequent crest migration.

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