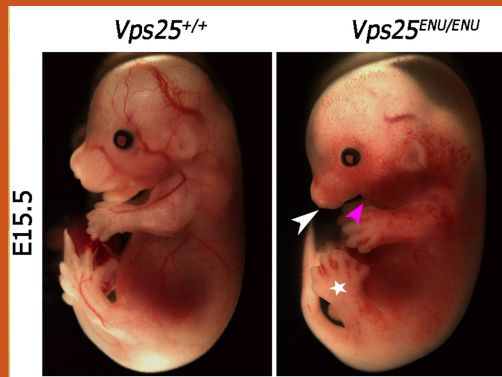


Mutation of VPS25, an ESCRT component, during early embryonic development leads to craniofacial malformations in mice, including hypoplastic jaws, stunted snouts, and cleft palates



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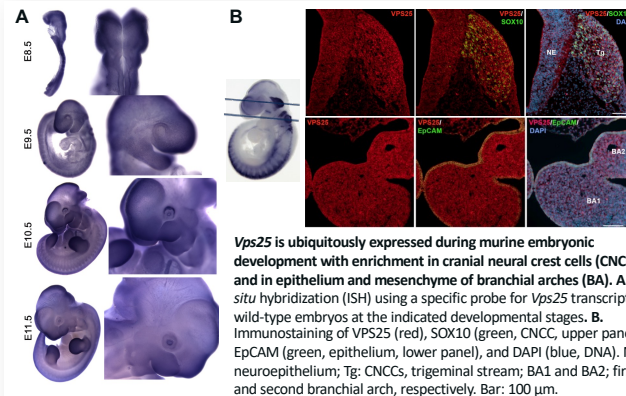
Tissue-specific roles of ESCRT machinery component VPS25 in craniofacial morphogenesis

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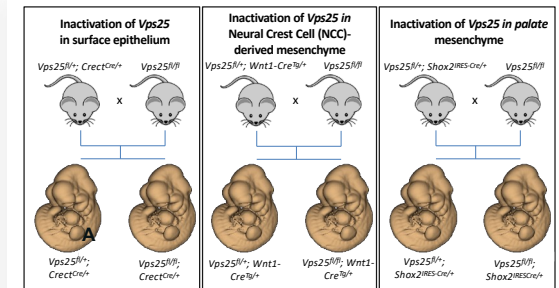


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Vps25 expression during murine embryonic development



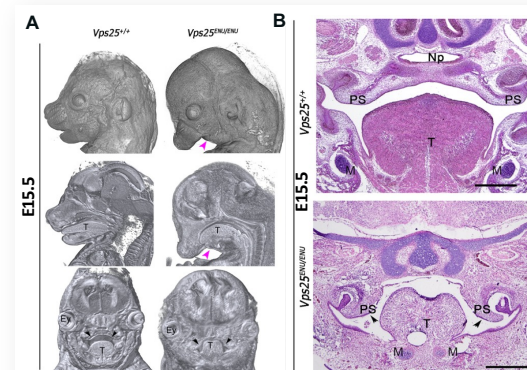
Generation of tissue specific Vps25 mutants



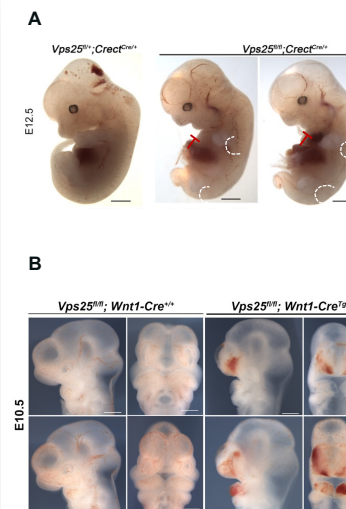
Vps25 tissue-specific roles were established by conditional mutagenesis of murine Vps25 using different Cre deleter lines.

Tissue specific deletion of Vps25 leads to various phenotypic abnormalities

Phenotypic abnormalities in Vps25 mutants



Vps25^{ENU/ENU} hypomorphic mutant embryos exhibit craniofacial defects. Side Panel. Gross morphology of mutant embryos at 15.5 days of gestation (E15.5) show edema, stunted snout (arrowhead), and polydactyly (star) compared to littermate controls. **A.** μ CT analyses reveal hypoplastic jaw (black arrowhead) and cleft of the secondary palate (red arrowheads) in Vps25^{ENU/ENU} embryos. **B.** H&E staining confirms cleft palate. Np: nasopharynx; T: tongue; PS: palatal shelf; M: Meckel's cartilage. Bar: 500 μ m.



A. Epithelial-specific deletion of Vps25 leads to craniofacial and limb abnormalities. Gross morphology of control and mutant embryos with epithelial inactivation of Vps25 at 12.5 days of gestation is shown. Bar: 1 mm. **B. Inactivation of Vps25 in CNCC-derived mesenchyme results in craniofacial malformations and early lethality.** Mutant embryos are hemorrhagic and exhibit hypoplastic first branchial arches compared to controls. Bar: 500 μ m. **C. Palate mesenchyme-specific deletion of Vps25 results in facial cleft.** Gross morphology of E14.5 control and mutant embryos with specific inactivation of Vps25 in palate mesenchyme. Mutant embryos display fully penetrant facial cleft (red arrow). Bar: 1 mm.

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