

Supplementary Material

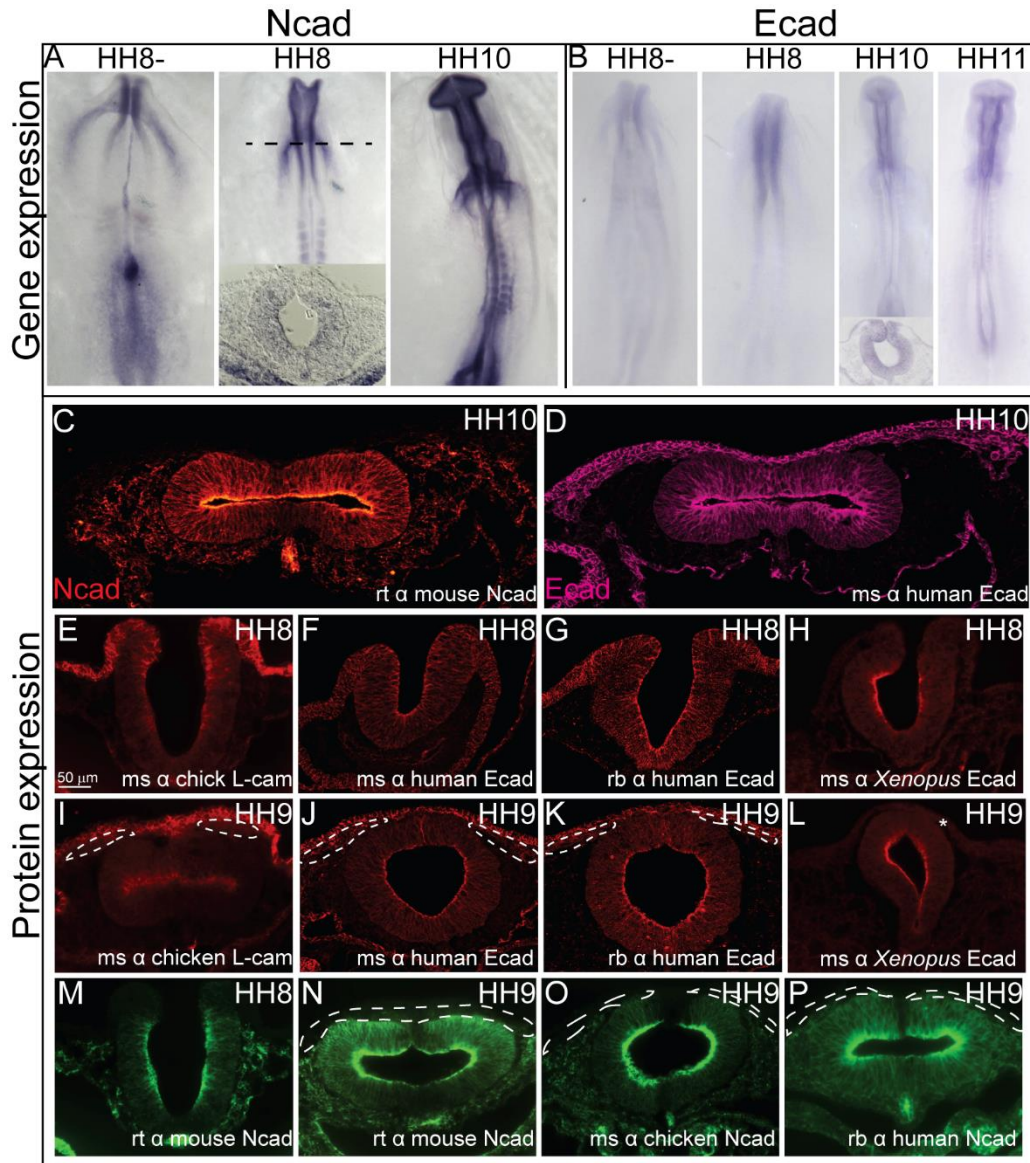


Figure S1. IHC using various antibodies for Ecad and Ncad. (A-B) Whole mount in situ hybridization using antisense probes to (A) Ncad at stages HH8-, HH8 and HH10, and (B) Ecad at stages HH8-, HH8, HH10, and HH11 demonstrates expression of the transcripts in the neural tube of the embryos. Transverse section IHC comparing expression of (C) Ncad and (D) Ecad at HH10. (E-P) To verify that our characterization of the type I cadherin proteins was accurate, we performed IHC on embryos that were fixed together at similar stages using four different Ecad

antibodies and three different Ncad antibodies (see methods and Table 1). (E-L) IHC for Ecad at 3 ss and 9 ss showed that with the anti-chicken anti-L-CAM and two anti-human Ecad antibodies, the protein was localized to the ectoderm and neural tube. All of the experiments from this study used the three Ecad antibodies shown in E-G. (H, L, asterisk) The *Xenopus* Ecad antibody only showed localization in the neural tube and was absent from the ectoderm and NC cells and was therefore excluded from the study. (M-P) All three antibodies against Ncad were consistent with expression in the neural tube, cranial mesenchyme and notochord, with absence of Ncad in the dorsal neural tube and early migratory NC cells, and two antibodies (Rat and Rabbit) were used in the study. Dashed lines demonstrate migratory NC cells. Scale bar is as marked.

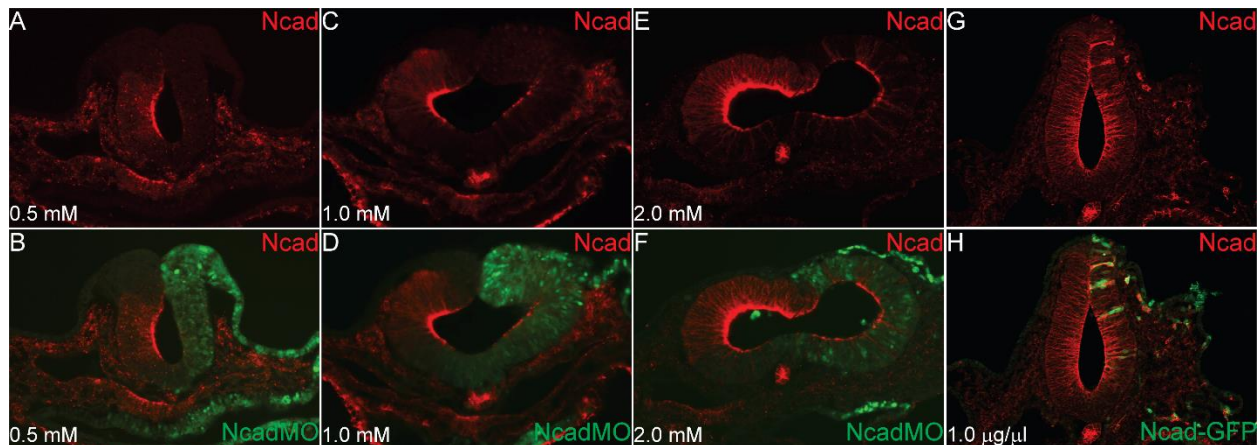


Figure S2. Titration of Ncad translation-blocking morpholino and Ncad-GFP. IHC for Ncad in embryos electroporated with (A, B) 0.5 mM, (C, D) 1.0 mM, (E, F) 2.0 mM NcadMO, or (G, H) 1.0 $\mu\text{g}/\mu\text{l}$ Ncad-GFP demonstrates that Ncad translation is blocked efficiently at all concentrations and that Ncad-GFP induces ectopic Ncad expression. The middle concentration of morpholino 1.0 mM was used in all loss of function studies in the paper, and 1.0 $\mu\text{g}/\mu\text{l}$ of DNA was used in all gain of function studies.