Descamps, E., H. Tian, E. Sanders, J. Van Hengel, F. Van Roy & D. Adriaens (2010) - Perturbations in the E-cadherin/catenin junctional complex in mouse embryos resulting in exencephaly. Annual Meeting of the Society of Integrative and Comparative Biology (Seattle, USA). (oral presentation)

ABSTRACT:
The essential role of cell-cell adhesion molecules in morphogenetic processes, particularly for the members of the cadherin protein family, has been acknowledged for years. The best-known adhesion and signaling system is the E-cadherin/catenin complex. The catenins are associated with the cytoplasmic domain of cadherin. We want to investigate the role of defects in cell adhesion molecules and their associated signaling molecules in craniofacial malformations in the mouse. To this end, we are generating tissue-specific knock-out mice for selected members of the cadherin/catenin junctional complex. We aim to find out what modifications are linked to a defect in the E-cadherin/catenin complex. For that, Wnt1Cre;p120ctnfl/fl KO mice were generated, where the floxed p120ctn alleles are eliminated in the Wnt1 expressing cells, mainly neural crest cells. This perturbation often resulted in exencephaly phenotypes. The present study provides a 3D-anatomical characterization of the craniofacial complex of this phenotype. By performing a contour-based shape analysis of the head of early mouse embryos (11.5dpc), a phenotypic characterization is done to determine the regions that are affected. For the description of the internal anatomy, 3D reconstructions of mice at 16.5dpc were made based on images of micro CT-scanning. The first results displayed absence of skeletal parts covering the extruded brain and reduced or displaced skeletal parts in the skull. Supported by the Concerted Research Actions (GOA) of Ghent University.