The vertebrate body forms in an anterior to posterior progression, driven by a population of undifferentiated cells at the posterior-most end of the embryo, called the tailbud, that contributes cells to newly formed tissues of the body. This process of posterior growth requires the addition of new cells to multiple germ layers and tissue types. A long-standing debate has existed on whether cell types are specified during gastrulation and maintained as lineage specific progenitor cells, or whether tailbud cells are multipotent stem cells that are specified to form tissue specific lineages by local signaling cues. Canonical Wnt signaling is present in the tailbud of all vertebrates throughout the duration of body formation, yet its role during posterior growth is unknown, hampered by the catastrophic disruption of posterior structures that occurs after traditional loss of function techniques. Using a combination of cell transplantation and heat-shock inducible Wnt inhibitor and activator transgenes in zebrafish, we show that canonical Wnt signaling plays multiple essential cell-autonomous roles in instructing the fate of tailbud stem cells. Wnt signaling is necessary and sufficient to specify mesoderm from a neural/mesodermal precursor, and also functions within the mesodermal lineage to specify paraxial mesoderm instead of posterior vasculature. Our results demonstrate that multipotent stem cells persist within the tailbud throughout the formation of the vertebrate body, and that local Wnt signaling cues specify germ layer contribution and mesodermal tissue type specification to balance the allocation of tissues as the embryonic body extends.

doi:10.1016/j.ydbio.2011.05.019

Control of the differentiation potential of cardiac neural crest and impact on vascular performance
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Neural crest (NC) progenitors have the innate potency to generate a wide array of cell types. However, the factors controlling NC multipotency and self-renewal are poorly understood. Our earlier work demonstrated that Foxd3 is required for maintenance of NC progenitors in the embryo, yet the cardiac NC remained relatively unscathed and outflow tract patterning progressed normally. Both in vivo and clonal in vitro analyses were used to determine that Foxd3 mediates a fate restriction choice for multipotent NC progenitors with a NC-specific mutation of Foxd3 biasing these multipotent cells toward a mesenchymal fate. Normally, the dorsal aorta consists of vascular smooth muscle cells (VSMCs) derived from both the cardiac NC and mesoderm. Most interestingly, these two cell populations are not intermingled; they form a distinct boundary dividing the ascending aorta from the descending aorta at the level of the ductus arteriosus and this border is maintained in adults. Almost nothing is known about molecules regulating this border, although the functional significance of these two distinct VSMC populations is clear because properties of this vessel vary along its length. In the NC-specific deletion of Foxd3, this border is not maintained and NC-derived VSMCs are found ectopically down the length of the aorta. Our recent work has defined molecular differences between these two regions of the aorta, and our goals are to determine a molecular signature for these different VSMC types and link this directly to vascular performance.

doi:10.1016/j.ydbio.2011.05.020