



Currie Group

Our group uses the many advantages of zebrafish embryology to dissect molecular mechanisms that act to pattern the vertebrate embryo. In particular, they are interested in how specific muscle cell types are determined within the developing embryo, how they grow and how they regenerate after injury. We are also interested in determining how these different muscle cell types have evolved.

Prof Peter Currie

Professor Currie is the Deputy Director of ARMI. Previously, he was laboratory head of the Developmental Biology Program at the Victor Chang Cardiac Research Institute in Sydney and held a senior program leadership position at the MRC Human Genetics Unit in Edinburgh in the United Kingdom.

Professor Currie has an active research program focusing on zebra fish muscle development, evolution and disease. He has published in the leading journals of developmental biology and regenerative medicine and has a demonstrated capacity for leadership along with a strong desire to mentor young scientists.



Current Projects

- Muscle specification and growth
- Evolution of the muscle of the paired fins
- Zebrafish models of muscle disease
- 3D imaging of zebrafish development

Skeletal muscle of the vertebrate body derives from segmented arrays of mesodermal structures termed somites that form from the paraxial mesoderm in a stereotypic rostral to caudal progression. Each somite is then partitioned into dorsal and ventral compartments that contain progenitors for individual structures of the developing embryo. The ventral portion of the somite undergoes a mesenchymal transition and gives rise to the cells of the sclerotome that migrate and differentiate into components of the axial skeleton. The dorsal segment of the somite remains epithelial and forms the dermomyotome that will give rise to the dermis and skeletal muscle of the trunk, tail and fins/limbs.

Research in the Currie laboratory focuses on attempts to understand how the cells of the vertebrate myotome are specified to become individual muscle cells later in development. We concentrate on two different populations of differentiating muscles, those that form the muscles of the axis and those that generate the appendicular muscles of the fins.

The myotomal muscles of the axis differentiate into three basic cell types. Slow muscle cells, fast muscle cells and muscle pioneer cells. These different cell populations have different embryonic origins. Slow muscle cells arise and differentiate next to the notochord, and following this differentiation the majority of slow muscle undergo a remarkable migration to transverse the entire extent of the forming myotome to produce a superficial subcutaneous layer of differentiated slow twitch muscle cells.

Muscle pioneer cells are the first differentiating slow muscle cells of the zebrafish myotome and express a unique cohort of genes. They are further characterised by the fact that they fail to migrate with latter differentiating slow cells. Fast muscle cells differentiate behind the wave of migrating slow twitch muscle cells and arise from the remainder of the cells of the myotome.

We are using a number of genetic and molecular approaches to dissect the events underlying the specification of these cells. Many of the processes we examine also occur in heart muscle cells. We also study the function of identified genes in the context of cardiogenesis.

In contrast, the appendicular muscles of the zebrafish fin arise via completely different morphogenesis. Fin and limb muscles are derived through migratory myoblasts cells that originate from fin/limb level somites. We study how these cells are specified and controlled in the developing embryo. We are also interested in how this process has evolved throughout vertebrate species, and examine the embryos of numerous extant fishes in an attempt to answer this question.

Finally, we are intrigued by zebrafish mutations that fail to undergo or retain the normal pattern of muscle differentiation within the embryo. We are particularly intrigued by mutations that mirror the onset of human muscular dystrophy and have developed zebrafish models of common muscular dystrophies. We hope this analysis will lead to novel understandings of the cell biological and developmental mechanisms that underlie the pathogenesis of this group of diseases.

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A combination of genetic and embryological amenability has placed zebrafish at the forefront of attempts to understand how genes function to control vertebrate development. The optical transparency of the zebrafish embryo provides the ability to visualise every cell in the forming embryo by simple optical inspection, as well as enabling the use of a host of cell labeling and transgenic approaches to dissect embryonic development. The large-scale mutagenesis of the zebrafish genome has also produced different classes of mutations that disrupt gene function.

