Abstract

The ability to recreate lost or damaged tissues and organs in humans is still a big challenge for scientists. Studies with animal models with extraordinary regenerative properties such as echinoderms, which share a common ancestor with vertebrates and conserve fundamental regulatory mechanisms in the first steps of fate specification of similar tissues, can be translated into viable clinical approaches. Our lab focuses a great part of its studies on the cellular and molecular mechanisms of visceral regeneration using the echinoderm sea cucumber *Holothuria glaberrima* as animal model. Under stressful conditions, *H. glaberrima* has the capacity to eviscerate their internal organs; including the digestive tract by rupturing it from the esophagus and the cloaca, while leaving the torn edges of the mesentery free in the body cavity. From the mesentery, they are able to regenerate a new intestine within a few weeks. Our project focuses on three different cellular events that are involved in the early regenerative process: cell proliferation, cell death, and changes in inflammation/reactive oxygen species. We expect that our findings allow us to know the temporal sequence of these events and to understand their dependence among themselves. We plan to approach this by inhibiting cell proliferation and apoptosis mechanisms, and then measuring their effects upon different cellular processes using histological techniques. One of the main cellular processes that we are going to focus is muscle cell dedifferentiation. Our lab showed that muscle cells dedifferentiate in order to migrate and form part of the connective tissue. During this process, they eject their contractile apparatus condensed in a Spindle-like Structure (SLS), which we are then able to identify with fluorescence by histochemistry. We hypothesize that inhibition of cell proliferation will lead to a compensative increase in the dedifferentiation process, increasing the number of SLSs in the mesentery and a larger dedifferentiated area. On the other hand, inhibition of apoptosis will decrease the dedifferentiation of the mesenterial muscle. The other cellular processes that we will jointly be studying after inhibition include: extracellular matrix (ECM) remodeling, epithelial to mesenchymal transition (EMT), and rudiment growth of the blastema-like structure.