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Abstracts

# Stem cells and tissue regeneration

## Program/Abstract # 270

### The zebrafish *laf/alk8* mutant as an *in vivo* model for molecular dissection of replacement tooth formation

Pamela C. Yelick [3], Ann Huysseune [1], R. Craig Albertson [2]

[1] *Department of Biology, Ghent University, Ghent, Belgium*

[2] *Department of Biology, Syracuse University, Syracuse, NY 13244, USA*

[3] *Division of Craniofacial and Molecular Genetics, Department of Oral and Maxillofacial Pathology, Tufts University, Boston, MA 02111, USA*

Zebrafish exhibit continuous replacement tooth formation in a manner very similar to adult tooth formation, providing the means to elucidate molecular signaling cascades regulating this process. The zebrafish *alk8* mutant *lost-a-fin (laf/alk8)* is a valuable model for replacement tooth formation. Although homozygous recessive *laf/alk8* mutants exhibit an early lethal phenotype, heterozygous *laf/alk8* mutants are viable and fertile, but do not form replacement teeth. We are currently elucidating *alk8* downstream signaling partners participating in primary and replacement tooth formation. Our studies in zebrafish complement mammalian tooth tissue engineering studies currently being performed in this laboratory. We anticipate that the proposed studies in zebrafish will likely result in the identification of molecular signaling networks mediating replacement tooth formation in the zebrafish, which may facilitate the creation of clinically relevant, molecular based replacement tooth therapies in humans. Supported by NIH/NIDCR grants DE1 2076, DE12024, and RO1 DE018043.

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## Program/Abstract # 271

### The role of $IP_3$ signalling during embryonic wound healing in *Xenopus*

Ximena Soto [1], J. Sivak [2], L. Petersen [2], E. Amaya [1]

[1] *The Healing Foundation Centre, University of Manchester, Manchester M13 9PT, UK*

[2] *The Gurdon Institute, University of Cambridge, Cambridge CB2 1QN, UK*

A major goal in regenerative medicine is to understand and ultimately facilitate our body's ability to repair itself following injury. We have begun to investigate the molecular and cellular basis of embryonic wound healing, given that embryos have the capacity to heal wounds quickly and completely. Tissue repair resembles embryo morphogenesis in several ways, including cell migration, proliferation and differentiation. In a screen for genes involved in morphogenesis in *Xenopus*, we identified an $Ins(1,4,5)P_3$ phosphatase ($IP_3P$), which impairs embryonic wound healing, suggesting that $Ins(1,4,5)P_3$ ($IP_3$) signalling plays a role in this process. This finding has motivated us to investigate the role of $IP_3$ signalling during embryonic wound healing, using *Xenopus* as a model system. Two sets of enzymes regulate this pathway, the $Ins(1,4,5)P_3$ 3-kinases ($IP_3K$) and $IP_3P$. $IP_3P$, such as $IP_3P$-5, promote $Ins(1,4)P_2$ ($IP_2$) generation, which is an inactive product, while $IP_3K$ generate $Ins(1,3,4,5)P_4$ ($IP_4$), which is involved in calcium release modulation. We have cloned $IP_3K$ B and $IP_3P$-5 A from *X. tropicalis* and have begun to investigate their temporal and spatial patterns of expression. In addition, we are misexpressing these genes and modulating the activity of $IP_3K$ during early development in order determine the effect of modulating $IP_3$ levels in embryonic wound healing assays. Finally we are investigating the consequence of these manipulations on the organization of the cytoskeleton. The aim of these studies will be to understand the role of $IP_3$ signalling in embryonic wound healing.

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## Program/Abstract # 272

### Strain and age differences in ear wound healing in mice

Cláudia R. Carvalho [1], Raquel A. Costa [1], Geraldo M. Azevedo-Junior [3], Valéria Ruiz-de-Souza [1], Nelson M. Vaz [2]

[1] *Dept of Morphol, ICB-UFMG, MG, Brazil*

[2] *Dept of Immunol, ICB-UFMG, MG, Brazil*

[3] *Public Hospital, Betim, MG, Brazil*

Small (2 mm) round holes in the ears of MRL and nude mice tend to close with characteristics of regeneration believed not to