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The *hox* gene complement of a basal teleost, *Pantodon bucholzi* (Osteoglossomorpha)

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Gene and whole genome duplications have profoundly shaped the structure and function of the vertebrate genome. Teleost fish, which comprise approximately 50% of all known vertebrate species, have undergone a third round of whole genome duplication (3R) above and beyond the two rounds of whole genome duplication shared by all vertebrates (2R). Most non-teleost vertebrates including tetrapods have 4 *Hox* gene clusters: *HoxA*, *HoxB*, *HoxC* and *HoxD*. Teleost models including zebrafish, medaka and pufferfish have been shown to possess 7 clusters including *hoxaa*, *hoxab*,*hoxba*,*hoxbb*, *hoxca*, *hoxda* and either *hoxcb* (medaka and pufferfish) or *hoxdb* (zebrafish),as a consequence of whole genome duplication followed by lineage specific loss of an entire *hox* cluster. This variability in whole *hox* cluster content between teleost clades is mirrored by equally variable *hox* gene content across orthologous clusters in different species, highlighting the putatively plastic nature of the teleost genome. The timing, mechanism, and developmental consequences of the duplication and subsequent loss of individual *hox* genes or whole clusters are currently under investigation. We present data pertaining to the structure, coding and non-coding element content of *hox*gene clusters in the African Freshwater Butterflyfish, *Pantodon bucholzi*, a member of the most basal lineage of teleost fish, the Osteoglossomorpha. Comparative genomic approaches using *Pantodon* and other model teleost fish are used to reconstruct the ancestral post-3R *hox*complement and to probe the basis for and developmental consequences of the variable *hox* gene content in diverse teleost genomes.

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Elucidating the genetic basis of scale loss in fish

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The genetic basis of morphological variation has been a long lasting question in biology. Teleost fish are by far the most diverse and numerous vertebrate class. One prominent characteristic of fish are its scales which cover the whole body. However there are also a lot of examples of scale loss and aberrant scale pattern in nature. Here we describe a dominant zebrafish mutant, *Feigenblatt* (*Fbl*) that exhibits deficient scale formation. The mutation is dose sensitive as homozygotes scales are absent along the flank whereas in heterozygous conditions this mutant displays partial scale loss.

Interestingly, this mutant interacts with another mutant defective in *fgfr1a* (*spiegeldanio*), which shows partial scale loss and aberrant scale pattern. Intercrosses enhance the scale loss phenotype of the *spiegeldanio* mutants, suggesting the use of similar pathways.

We mapped *Fbl* down to a small region on LG6, in which none of the genes in the interval showed any coding mutations, However quantitative real time PCR showed a 3.3-fold down regulation of *receptor tyrosine kinase orphan receptor 1* (*ror1*) in cDNA of adult skin, suggesting that regulation of this gene may be affected in the mutant.

We are currently screening for more alleles of *ror1* in an F1 allele screen. Additionally we are investigating ROR1 expression in natural population of fish showing acquired scale loss as a defining trait for the genus.

We think that this mutant is a great model to begin to understand the genetic and evolutionary basis of scale loss in teleosts.

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