2 New Gene Evolution in the Bonus-TIF1-γ/TRIM33 Family Impacted the Architecture of the Vertebrate Dorsal-Ventral Patterning Network.

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Recommendations:

Very Good 16 Jul 2014

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NEW FINDING

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How new gene duplicates are integrated into and help shape an evolving genetic pathway is an important yet little-understood problem. In this experimental comparative analysis of a homologous pathway underlying the early development in Drosophila and vertebrates, these authors found that the relevant genetic components evolved distinct pathways with duplications in the TRIM family involved in vertebrates and homologous Bonus in Drosophila. Their experiments detected Bonus as an agonist of DPP and BMP networks that control dorsal-ventral axis, which is also known in biology, the reverse of dorsal-ventral polarity between their embryos, while the vertebrate TRIM33 duplicate evolved as an antagonist for the network underlying dorsal-ventral axis formation.

Further analysis detected a previously unknown pattern of gene evolution: the TRIM33 replaced a previous gene, nedd4, in the function, so functionally it inherited an old function. However, genetically, TRIM33 obtained a function that was not the one of the TRIM system, so the function of nedd4 was passed to TRIM33 and a gene acquired a function its paralogs did not have: TRIM33 acquired a new function for itself. However, more intriguingly, nedd4 in vertebrates evolved a new function – as a u-ligase of vertebrate Smad evolving from a previous u-ligase of Medea.

Thus, the study provides a novel insight into the understanding of how a new gene duplicate evolves new functions by integrating into and changing ancestral gene networks.

Disclosures
None declared

Add a comment

Abstract:

ABSTRACT

Uncovering how a new gene acquires its function and understanding how the function of a new gene influences existing genetic networks are important topics in evolutionary biology. Here, we demonstrate nonconservation for the embryonic functions of Drosophila Bonus and its newest vertebrate relative TIF1-γ/TRIM33. We showed previously that TIF1-γ/TRIM33 functions as an ubiquitin ligase for the Smad4 signal transducer and antagonizes the Bone Morphogenetic Protein (BMP) signaling network underlying vertebrate dorsal-ventral axis formation. Here,... more »
we show that Bonus functions as an agonist of the Decapentaplegic (Dpp) signaling network underlying dorsal-ventral axis formation in flies. The absence of conservation for the roles of Bonus and TIF1-γ/TRIM33 reveals a shift in the dorsal-ventral patterning networks of flies and mice, systems that were previously considered wholly conserved. The shift occurred when the new gene TIF1-γ/TRIM33 replaced the function of the ubiquitin ligase Nedd4L in the lineage leading to vertebrates. Evidence of this replacement is our demonstration that Nedd4 performs the function of TIF1-γ/TRIM33 in flies during dorsal-ventral axis formation. The replacement allowed vertebrate Nedd4L to acquire novel functions as a ubiquitin ligase of vertebrate-specific Smad proteins. Overall our data reveal that the architecture of the Dpp/BMP dorsal-ventral patterning network continued to evolve in the vertebrate lineage, after separation from flies, via the incorporation of new genes.

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Comments:

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For the convenience of the readers, I would like to indicate that the u-ligase is an abbreviation for ubiquitin ligase. I also want to point out that the chain of evolutionary events

revealed by the authors is not predicted by the classic model of gene duplication but may be more consistent with the evolutionary model of 'tinkering' proposed by Francois Jacob in 1977.

DISCLOSURES None declared.