



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

Wisotzkey RG, Quijano JC, Stinchfield MJ, Newfeld SJ
Mol Biol Evol. 2014 May 31

Save/Follow Export Get Article

8+1

RECOMMENDATIONS 1 | ABSTRACT | COMMENTS 1

expand all

Recommendations:

Very Good

16 Jul 2014



Manyuan Long

F1000 Genomics & Genetics

University of Chicago, Chicago, IL, USA.

NEW FINDING

DOI: 10.3410/f.718426585.793497317

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.

© The Author 2014. Published by Oxford University Press on behalf of the Society for Molecular Biology and Evolution. All rights reserved. For permissions, please e-mail: journals.permissions@oup.com.

DOI: 10.1093/molbev/msu175

PMID: 24881051



Abstract courtesy of PubMed: A service of the National Library of Medicine and the National Institutes of Health.

Comments:

COMMENTS (1)

[add a comment](#)

21 Jul 2014 | 11:00 PM

[Report](#)

Manyuan Long [Recommending Faculty Member](#)
University of Chicago Libraries

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.

An open access journal that expands on the recommended literature coverage of **F1000Prime** by publishing unique, peer-reviewed reports to provide context on emerging themes in biology and medicine.

[view all](#)

Developmental Biology | Cell Biology | Physiology

Retrotransposons and germ cells: reproduction, death, and diversity

Stefanie Seisenberger, Christian Popp, Wolf Reik
F1000 Biology Reports 2010 2:(44) (16 Jun 2010)

[Full text](#) | [PDF](#) | [Abstract on PubMed](#)

Cell Biology | Molecular Medicine | Physiology | Developmental Biology | Cancer Biology

Plant asymmetric cell division regulators: pinch-hitting for PARs?

Carrie A Metzinger, Dominique C Bergmann
F1000 Biology Reports 2010 2:(25) (12 Apr 2010)

[Full text](#) | [PDF](#) | [Abstract on PubMed](#)

Librarian Resources

Press Office

F1000 Specialists

F1000 Updates

About/Contact

Article Recommendations

F1000Prime Reports

F1000Prime Faculty

Blog

Subscribe

About

FAQs

Contact

Articles

Advisory Panel

Blog

Submit

Author Guidelines

Register

About

Contact

Posters

Upcoming meetings

For Depositors

For Societies

Register

About/Contact

© 2000-2014 Faculty of 1000 Ltd. ISSN 2051-9796 | Legal | Partner of HINARI • CrossRef • ORCID

F1000 is a registered trademark of Faculty of 1000 Limited