Press Report

March 2016

Alexis M. Vidal


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Fly

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High throughput preparation of fly genomic DNA in 96-well format using a paint-shaker

Michael Lang, Olga Nagy, Claus Lang & Virginie Orgogozo

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TOOLS AND RESOURCES

A genome-wide resource for the analysis of protein localisation in *Drosophila*

Mihail Sarov[1*], Christiane Barz[2], Helena Jambor[1], Marco Y Hein[3], Christopher Schmied[1], Dana Suchold[1], Bettina Stender[2], Stephan Janosch[1], Vinay Vikas KJ[4], RT Krishnan[4], Aishwarya Krishnamoorthy[4], Irene RS Ferreira[2], Radoslaw K Ejsmont[1], Katja Finkl[2], Susanne Hasse[1], Philipp Kämpfer[5], Nicole Plewka[2], Elisabeth Vinis[1], Siegfried Schloissnig[5], Elisabeth Knust[1], Volker Hartenstein[6], Matthias Mann[3], Mani Ramaswami[7], K VijayRaghavan[4], Pavel Tomancak[1*], Frank Schnorrer[2*]

Abstract The *Drosophila* genome contains >13000 protein-coding genes, the majority of which remain poorly investigated. Important reasons include the lack of antibodies or reporter constructs to visualise these proteins. Here, we present a genome-wide fosmid library of 10000 GFP-tagged clones, comprising tagged genes and most of their regulatory information. For 880 tagged proteins, we created transgenic lines, and for a total of 207 lines, we assessed protein expression and localisation in ovaries, embryos, pupae or adults by stainings and live imaging approaches. Importantly, we visualised many proteins at endogenous expression levels and found a large fraction of them localising to subcellular compartments. By applying genetic complementation tests, we estimate that about two-thirds of the tagged proteins are functional. Moreover, these tagged proteins enable interaction proteomics from developing pupae and adult flies. Taken together, this resource will boost systematic analysis of protein expression and localisation in various cellular and developmental contexts.

DOI: 10.7554/eLife.12068.001

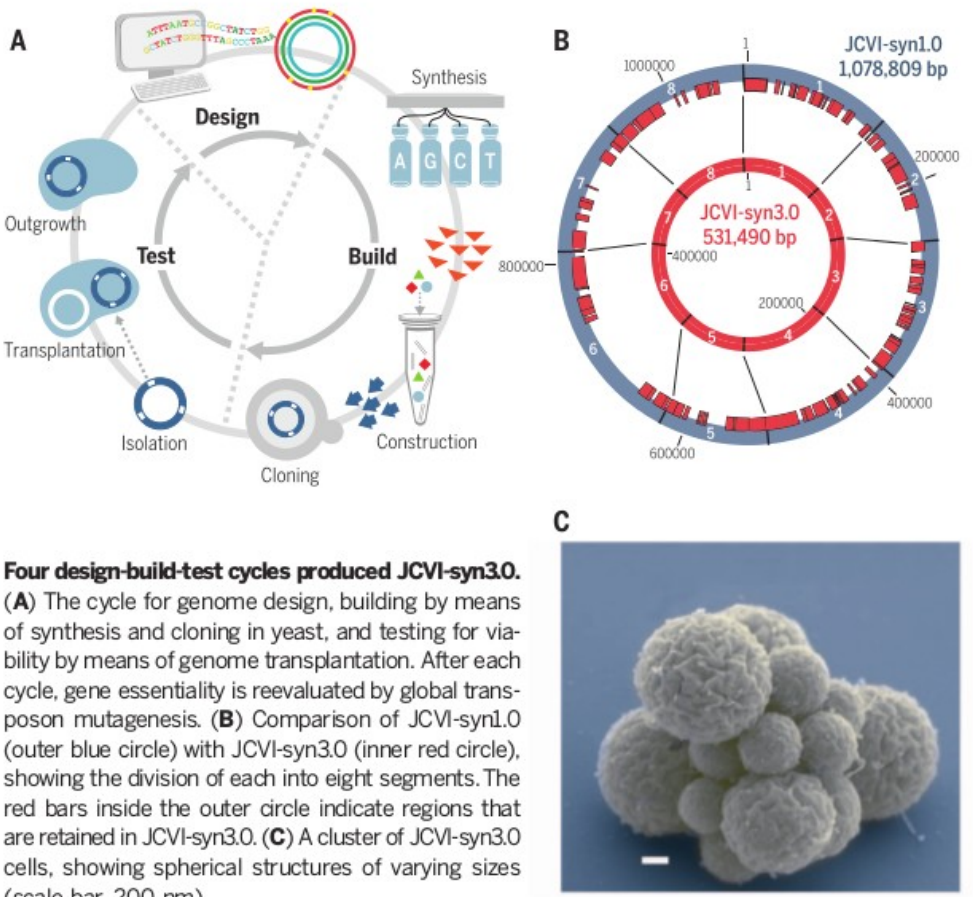
RESEARCH ARTICLE

SYNTHETIC BIOLOGY

Design and synthesis of a minimal bacterial genome

Clyde A. Hutchison III,[1*†] Ray-Yuan Chuang,[1†‡] Vladimir N. Noskov,[1] Nacyra Assad-Garcia,[1] Thomas J. Deerinck,[2] Mark H. Ellisman,[2] John Gill,[3] Krishna Kannan,[3] Bogumil J. Karas,[1] Li Ma,[1] James F. Pelletier,[4§] Zhi-Qing Qi,[3] R. Alexander Richter,[1] Elizabeth A. Strychalski,[4] Lijie Sun,[1||] Yo Suzuki,[1] Billyana Tsvetanova,[3] Kim S. Wise,[1] Hamilton O. Smith,[1,3] John I. Glass,[1] Chuck Merryman,[1] Daniel G. Gibson,[1,3] J. Craig Venter[1,3*]

We used whole-genome design and complete chemical synthesis to minimize the 1079–kilobase pair synthetic genome of *Mycoplasma mycoides* JCVI-syn1.0. An initial design, based on collective knowledge of molecular biology combined with limited transposon mutagenesis data, failed to produce a viable cell. Improved transposon mutagenesis methods revealed a class of quasi-essential genes that are needed for robu growth, explaining the failure of our initial design. Three cycles of design, synthesis, and testing, with retention of quasi-essential genes, produced JCVI-syn3.0 (531 kilobase pair 473 genes), which has a genome smaller than that of any autonomously replicating cell found in nature. JCVI-syn3.0 retains almost all genes involved in the synthesis and processing of macromolecules. Unexpectedly, it also contains 149 genes with unknown biological functions. JCVI-syn3.0 is a versatile platform for investigating the core functio of life and for exploring whole-genome design.



Four design-build-test cycles produced JCVI-syn3.0. (**A**) The cycle for genome design, building by means of synthesis and cloning in yeast, and testing for viability by means of genome transplantation. After each cycle, gene essentiality is reevaluated by global transposon mutagenesis. (**B**) Comparison of JCVI-syn1.0 (outer blue circle) with JCVI-syn3.0 (inner red circle), showing the division of each into eight segments. The red bars inside the outer circle indicate regions that are retained in JCVI-syn3.0. (**C**) A cluster of JCVI-syn3.0 cells, showing spherical structures of varying sizes (scale bar, 200 nm).

CellPress

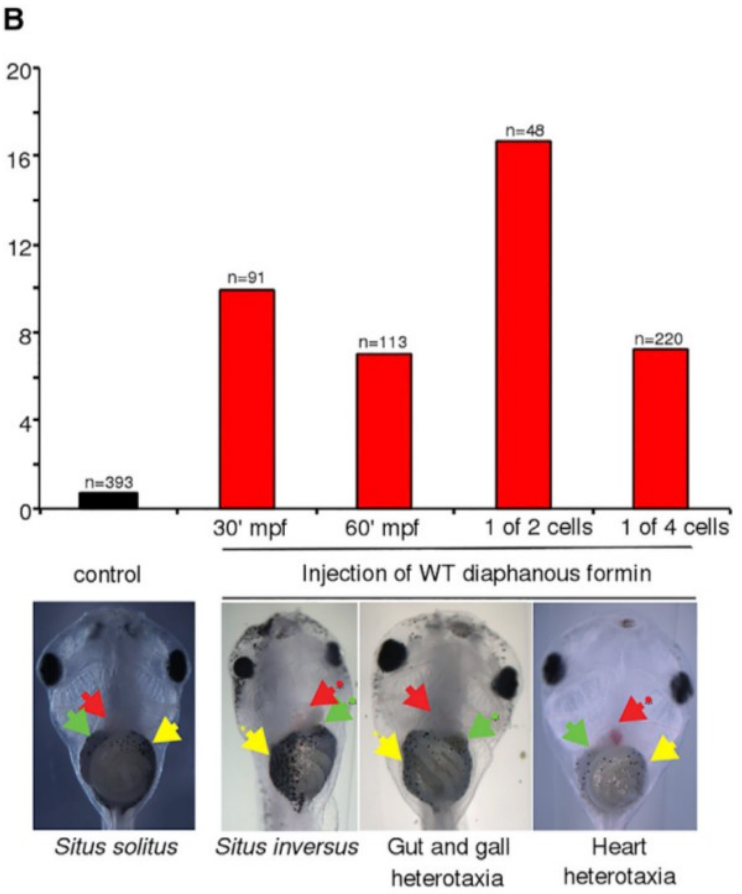
Current Biology
Report

Formin Is Associated with Left-Right Asymmetry in the Pond Snail and the Frog

Angus Davison,[1,*] Gary S. McDowell,[2] Jennifer M. Holden,[1,9] Harriet F. Johnson,[1] Georgios D. Koutsovoulos,[3] M. Maureen Liu,[1] Paco Hulpiau,[4] Frans Van Roy,[4] Christopher M. Wade,[1] Ruby Banerjee,[5] Fengtang Yang,[5] Satoshi Chiba,[6] John W. Davey,[4,10] Daniel J. Jackson,[7] Michael Levin,[2] and Mark L. Blaxter[3,8]
[1]School of Life Sciences, University of Nottingham, Nottingham NG7 2RD, UK

SUMMARY

While components of the pathway that establishes left-right asymmetry have been identified in diverse animals, from vertebrates to flies, it is striking that the genes involved in the first symmetry-breaking step remain wholly unknown in the most obviously chiral animals, the gastropod snails. Previously, research on snails was used to show that left-right signaling of Nodal, downstream of symmetry breaking, may be an ancestral feature of the Bilateria [1, 2]. Here, we report that a disabling mutation in one copy of a tandemly duplicated, diaphanous-related formin is perfectly associated with symmetry breaking in the pond snail. This is supported by the observation that an anti-formin drug treatment converts dextral snail embryos to a sinistral phenocopy, and in frogs, drug inhibition or overexpression by microinjection of formin has a chirality-randomizing effect in early (pre-cilia) embryos. Contrary to expectations based on existing models [3–5], we discovered asymmetric gene expression in 2- and 4-cell snail embryos, preceding morphological asymmetry. As the formin-actin filament has been shown to be part of an asymmetry-breaking switch in vitro [6, 7], together these results are consistent with the view that animals with diverse body plans may derive their asymmetries from the same intracellular chiral elements [8].



nature COMMUNICATIONS

ARTICLE

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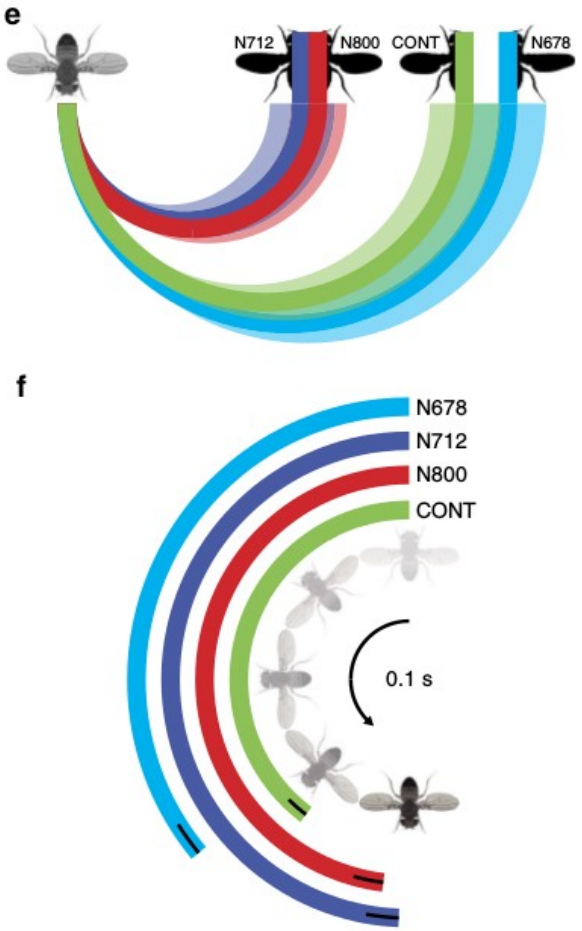
Enhanced flight performance by genetic manipulation of wing shape in *Drosophila*

Robert P. Ray[1,†], Toshiyuki Nakata[2], Per Henningsson[3] & Richard J. Bomphrey[2]



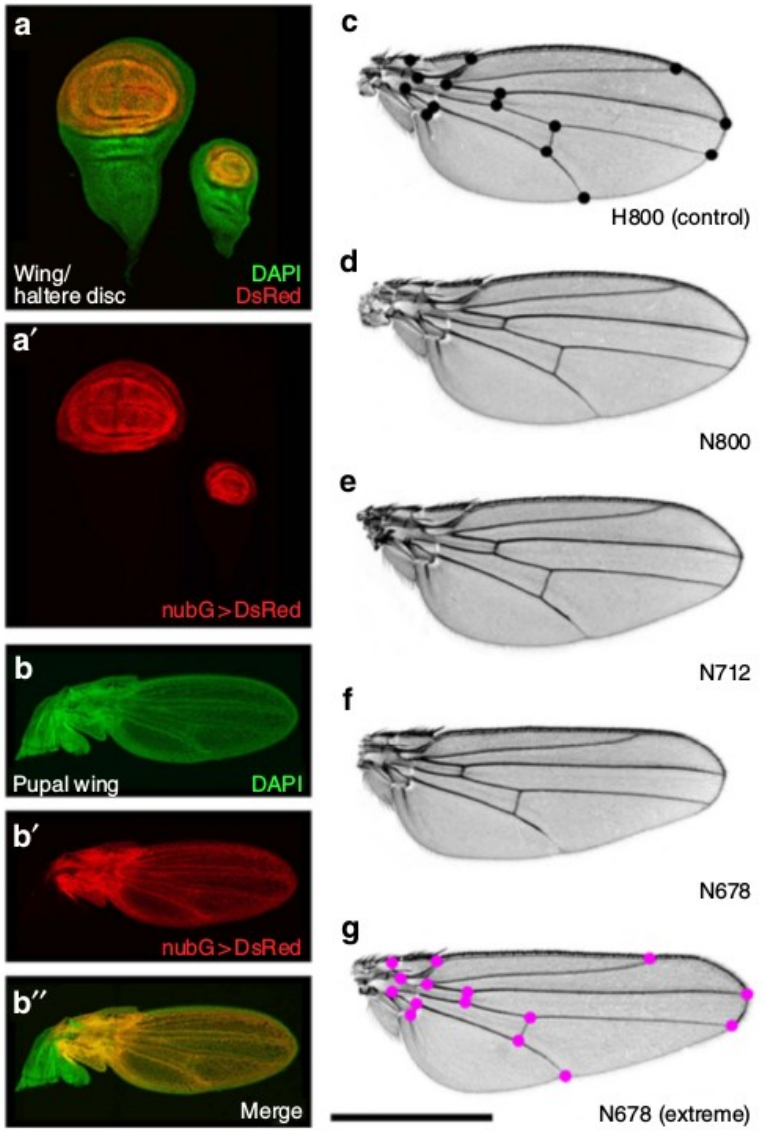


Figure 1 | Changing wing shape with *nub-Gal4* and *nw-RNAi*. (**a**) Larval

PLOS | GENETICS

RESEARCH ARTICLE

Hybrid Dysgenesis in *Drosophila simulans* Associated with a Rapid Invasion of the *P*-Element

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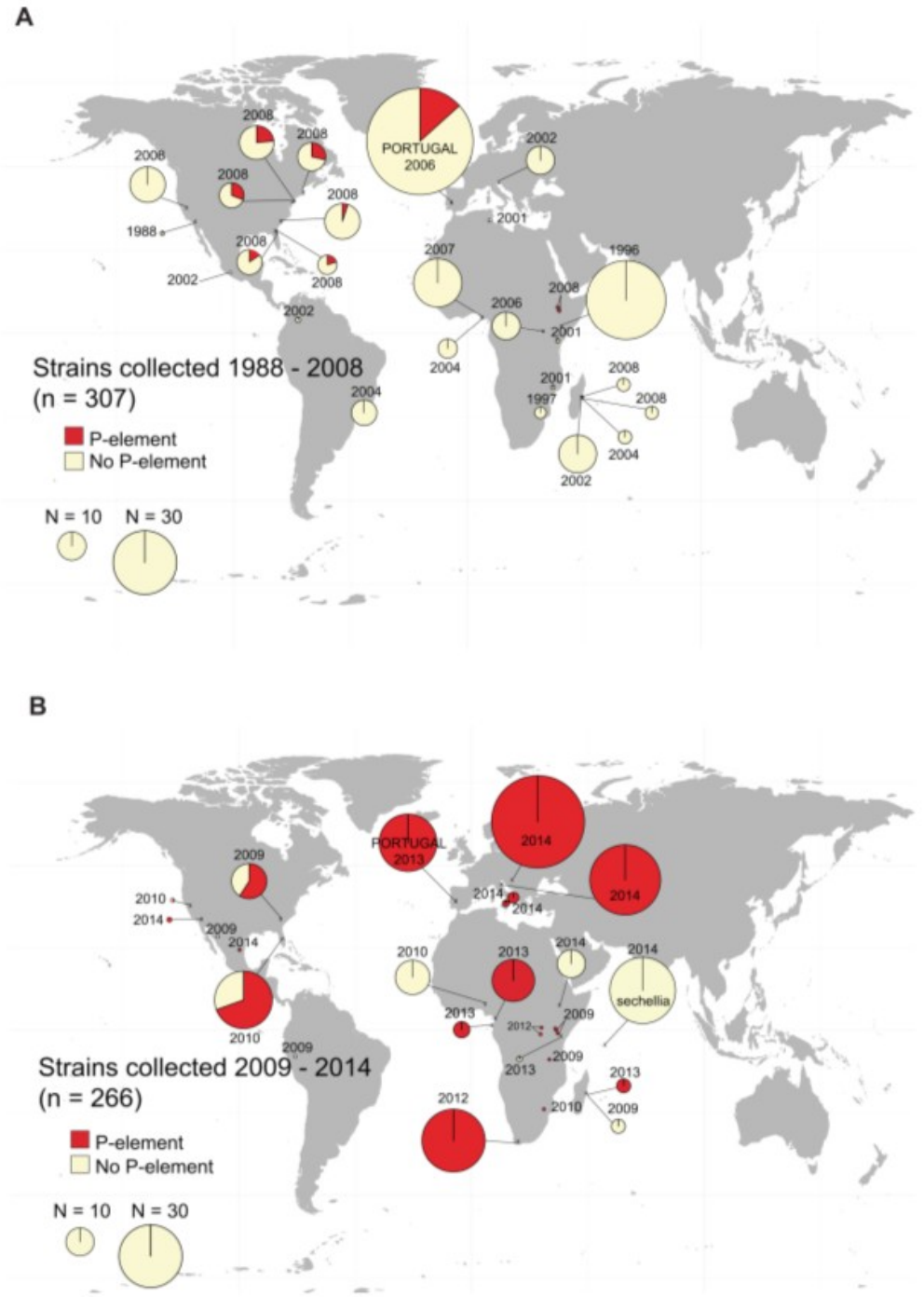
Abstract

In a classic example of the invasion of a species by a selfish genetic element, the *P*-elemer was horizontally transferred from a distantly related species into *Drosophila melanogaster*. Despite causing 'hybrid dysgenesis', a syndrome of abnormal phenotypes that include sterility, the *P*-element spread globally in the course of a few decades in *D. melanogaster*. Unti recently, its sister species, including *D. simulans*, remained *P*-element free. Here, we find a hybrid dysgenesis-like phenotype in the offspring of crosses between *D. simulans* strains collected in different years; a survey of 181 strains shows that around 20% of strains induce hybrid dysgenesis. Using genomic and transcriptomic data, we show that this dysgenesis-inducing phenotype is associated with the invasion of the *P*-element. To characterize this invasion temporally and geographically, we survey 631 *D. simulans* strains collected on three continents and over 27 years for the presence of the *P*-element. We find that the *D. simulans* *P*-element invasion occurred rapidly and nearly simultaneously in the regions surveyed, with strains containing *P*-elements being rare in 2006 and common by 2014. Importantly, as evidenced by their resistance to the hybrid dysgenesis phenotype, strains collected from the latter phase of this invasion have adapted to suppress the worst effects of the *P*-element.



A novel gene controlling the timing of courtship initiation in *Drosophila melanogaster*

Peter Luu*, Sadaf A. Zaki*, David H. Tran†,*, and Rachael L. French*

ABSTRACT

Over the past 35 years, developmental geneticists have made impressive progress towards an understanding of how genes specify morphology and function, particularly as relates to the specification of each physical component of an organism. In the last 20 years, male courtship behavior in *Drosophila melanogaster* has emerged as a robust model system for the study of genetic specification of behavior. Courtship behavior is both complex and innate, and a single gene, *fruitless (fru)*, is both necessary and sufficient for all aspects of the courtship ritual. Typically, loss of male-specific Fruitless proteins function results in male flies that perform the courtship ritual incorrectly, slowly, or not at all. Here we describe a novel requirement for *fru*: we have identified a group of cells in which male Fru proteins are required to reduce the speed of courtship initiation. In addition, we have identified a gene, *Trapped in endoderm 1* (*Tre1*), which is required in these cells for normal courtship and mating behavior. *Tre1* encodes a G-protein-coupled receptor required for establishment of cell polarity and cell migration, and has previously not been shown to be involved in courtship behavior. We describe the results of feminization of the *Tre1*-expressing neurons, as well as the effects on courtship behavior of mutation of *Tre1*. In addition, we show that *Tre1* is expressed in a sexually dimorphic pattern in the central and peripheral nervous systems, and investigate the role of the *Tre1* cells in mate identification.

doi:10.1111/evo.12870

The search for causal traits of speciation: Divergent female mate preferences target male courtship song, not pheromones, in *Drosophila athabasca* species complex

Roman Yukilevich,[1,2] Taylor Harvey,[1] Son Nguyen,[1] Joanne Kehlbeck,[1] and Agnes Park[1]

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Understanding speciation requires the identification of traits that cause reproductive isolation. This remains a major challenge since it is difficult to determine which of the many divergent traits actually caused speciation. To overcome this difficulty, we studied the sexual cue traits and behaviors associated with rapid speciation between EA and WN sympatric behavioral races of *Drosophila athabasca* that diverged only 16,000–20,000 years ago. First, we found that sexual isolation was essentially complete and driven primarily by divergent female mating preferences. To determine the target of female mate choice, we found that, unlike cuticular hydrocarbons (CHCs), male courtship song is highly divergent between EA and WN in both allopatry and sympatry and is not affected by latitudinal variation. We then used pheromone rub-off experiments to show no effect of CHCs on divergent female mate choice. In contrast, both male song differences and male mating success in hybrids exhibited a large X-effect and playback song experiments confirmed that male courtship song is indeed the target of sexual isolation. These results show that a single secondary sexual trait is a major driver of speciation and suggest that we may be overestimating the number of traits involved in speciation when we study older taxa.

KEY WORDS: **Cuticular hydrocarbons, ecological speciation, female mating preference, large X-effect, magic traits, male courtship song, sexual isolation, sexual selection, speciation phenotypes.**

PLOS | COMPUTATIONAL BIOLOGY

RESEARCH ARTICLE

Genetic Correlations Greatly Increase Mutational Robustness and Can Both Reduce and Enhance Evolvability

Sam F. Greenbury[1]*, Steffen Schaper[2¤], Sebastian E. Ahnert[1], Ard A. Louis[2]

Abstract

Mutational neighbourhoods in genotype-phenotype (GP) maps are widely believed to be more likely to share characteristics than expected from random chance. Such *genetic correlations* should strongly influence evolutionary dynamics. We explore and quantify these intuitions by comparing three GP maps—a model for RNA secondary structure, the HP model for protein tertiary structure, and the Polyomino model for protein quaternary structure—to a simple random null model that maintains the number of genotypes mapping to each phenotype, but assigns genotypes randomly. The mutational neighbourhood of a genotype in these GP maps is much more likely to contain genotypes mapping to the same phenotype than in the random null model. Such *neutral correlations* can be quantified by the robustness to mutations, which can be many orders of magnitude larger than that of the null model, and crucially, above the critical threshold for the formation of large neutral networks of mutationally connected genotypes which enhance the capacity for the exploration of phenotypic novelty. Thus neutral correlations increase evolvability. We also study *non-neutral correlations*: Compared to the null model, i) If a particular (non-neutral) phenotype is found once in the 1-mutation neighbourhood of a genotype, then the chance of finding that phenotype multiple times in this neighbourhood is larger than expected; ii) If two genotypes are connected by a single neutral mutation, then their respective non-neutral 1-mutation neighbourhoods are more likely to be similar; iii) If a genotype maps to a folding or self-assembling phenotype, then its non-neutral neighbours are less likely to be a potentially deleterious non-folding or non-assembling phenotype. Non-neutral correlations of type i) and ii) reduce the rate at which new phenotypes can be found by neutral exploration, and so may diminish evolvability, while non-neutral correlations of type iii) may instead facilitate evolutionary exploration and so increase evolvability.

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Abstract

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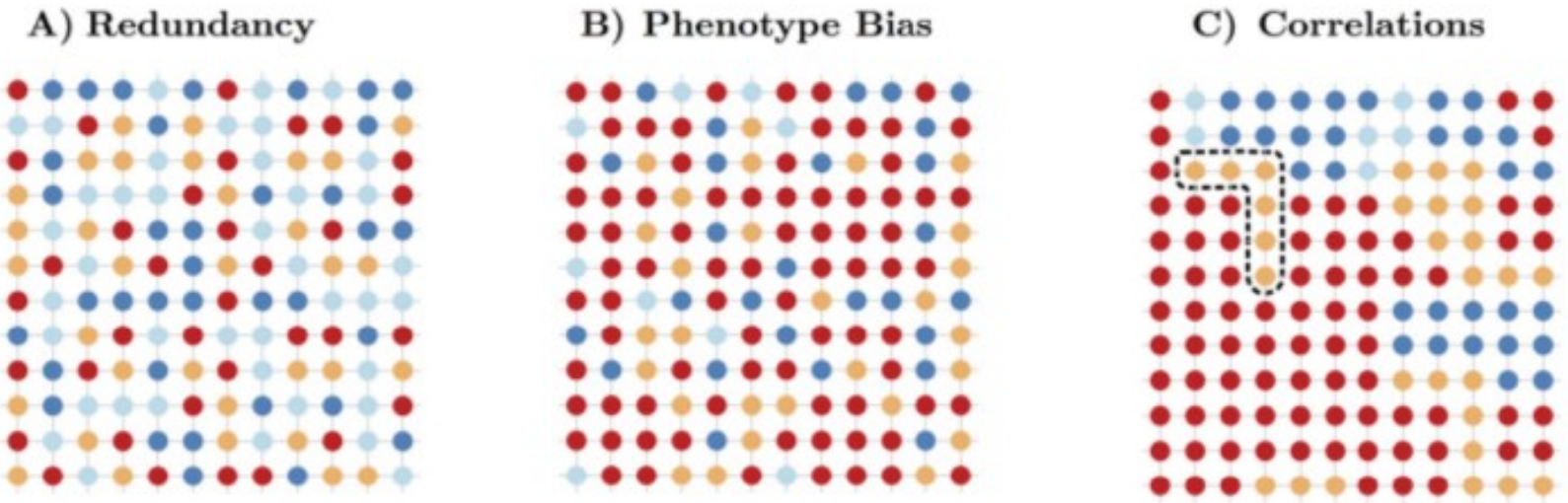

Fig 1. Schematic depiction of the GP map properties of redundancy, phenotype bias and neutral correlations. Phenotypes are represented by colours, genotypes as nodes and mutations as edges. A) Each colour appears multiple times with uniform redundancy. B) Some colours appear more often than others, demonstrating a phenotype bias. C) A rearrangement of the colours from the middle plot illustrates positive neutral correlations where the same colours are more likely to appear near each other than would be expected by random chance arrangement. The black box surrounding the six orange genotypes depicts a single component (a set of genotypes connected by neutral point mutations, also called a neutral network) of the orange phenotype. Such positive neutral correlations enhance the probability that such neutral networks occur.

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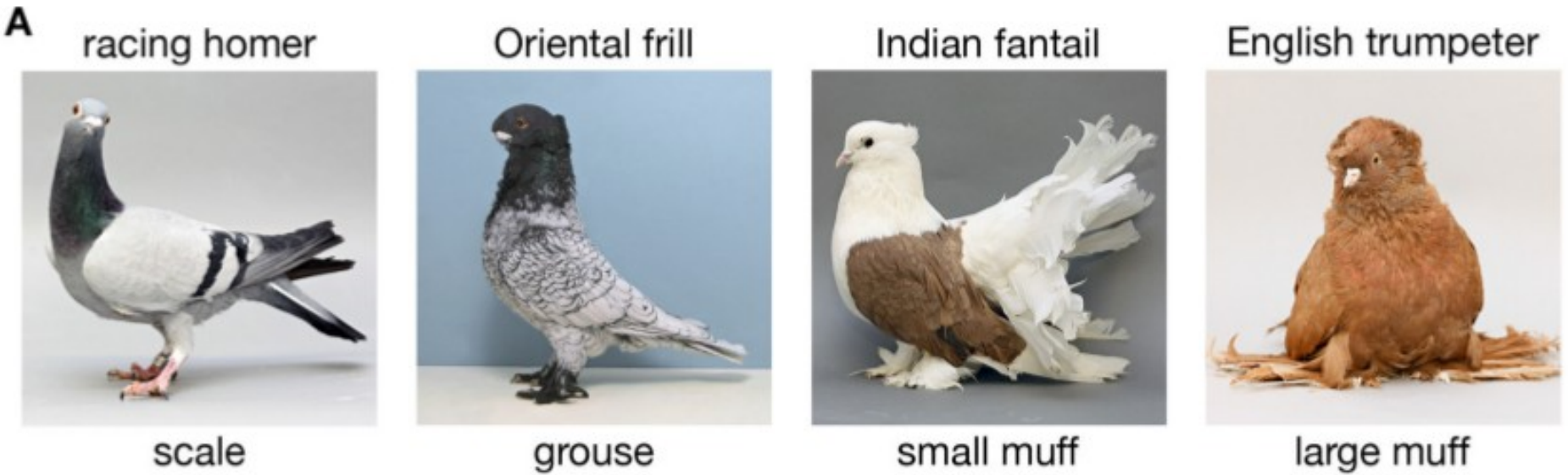
RESEARCH ARTICLE

Molecular shifts in limb identity underlie development of feathered feet in two domestic avian species

Eric T Domyan[1†], Zev Kronenberg[2‡], Carlos R Infante[3§], Anna I Vickrey[1], Sydney A Stringham[1], Rebecca Bruders[1], Michael W Guernsey[1¶], Sungdae Park[3], Jason Payne[4], Robert B Beckstead[4], Gabrielle Kardon[2], Douglas B Menke[3], Mark Yandell[2,5], Michael D Shapiro[1*]

Abstract Birds display remarkable diversity in the distribution and morphology of scales and feathers on their feet, yet the genetic and developmental mechanisms governing this diversity remain unknown. Domestic pigeons have striking variation in foot feathering within a single species, providing a tractable model to investigate the molecular basis of skin appendage differences. We found that feathered feet in pigeons result from a partial transformation from hindlimb to forelimb identity mediated by *cis*-regulatory changes in the genes encoding the hindlimb-specific transcription factor Pitx1 and forelimb-specific transcription factor Tbx5. We also found that ectopic expression of *Tbx5* is associated with foot feathers in chickens, suggesting similar molecular pathways underlie phenotypic convergence between these two species. These results show how changes in expression of regional patterning genes can generate localized changes in organ fate and morphology, and provide viable molecular mechanisms for diversity in hindlimb scale and feather distribution.

DOI: 10.7554/eLife.12115.001



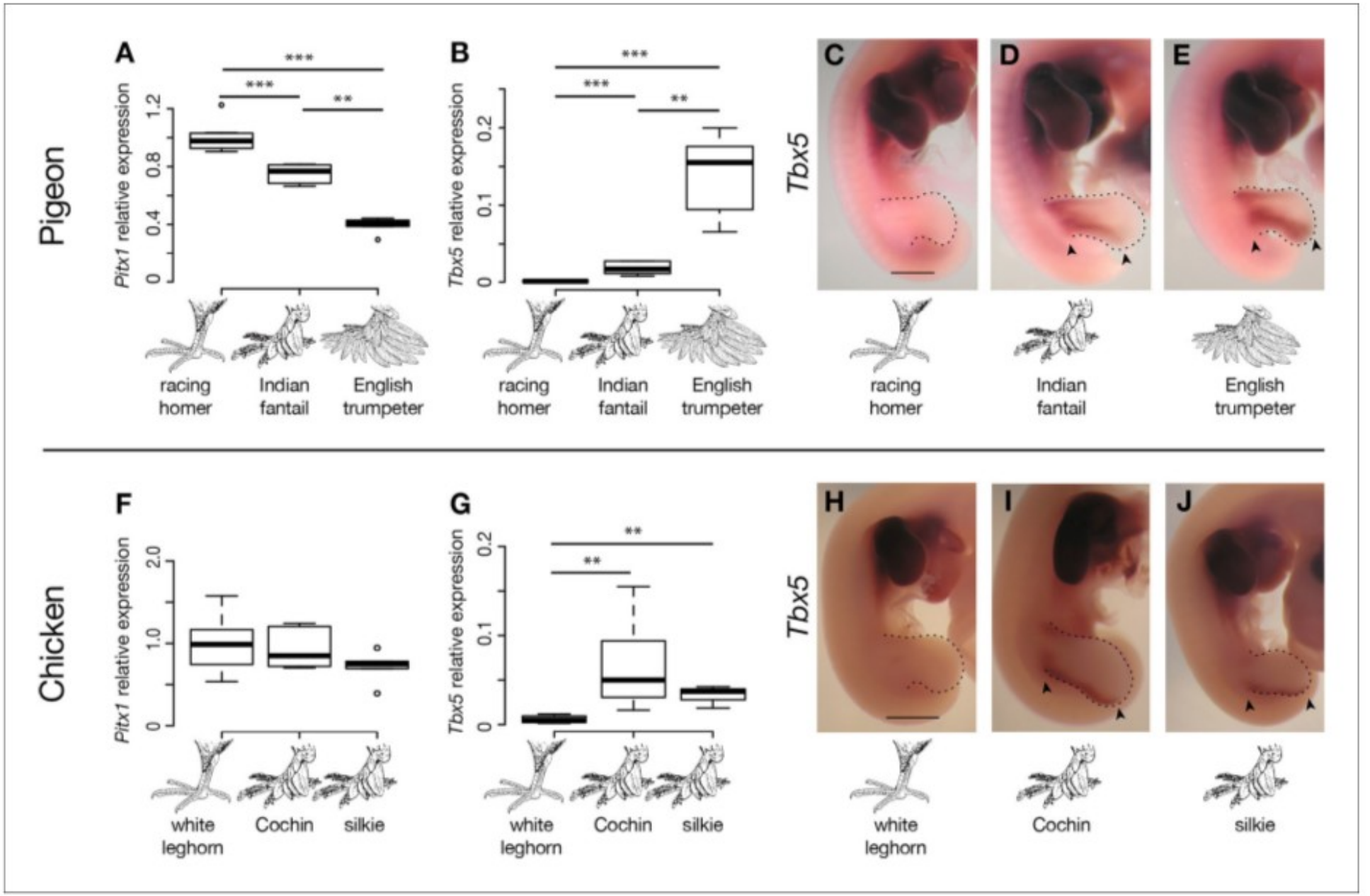


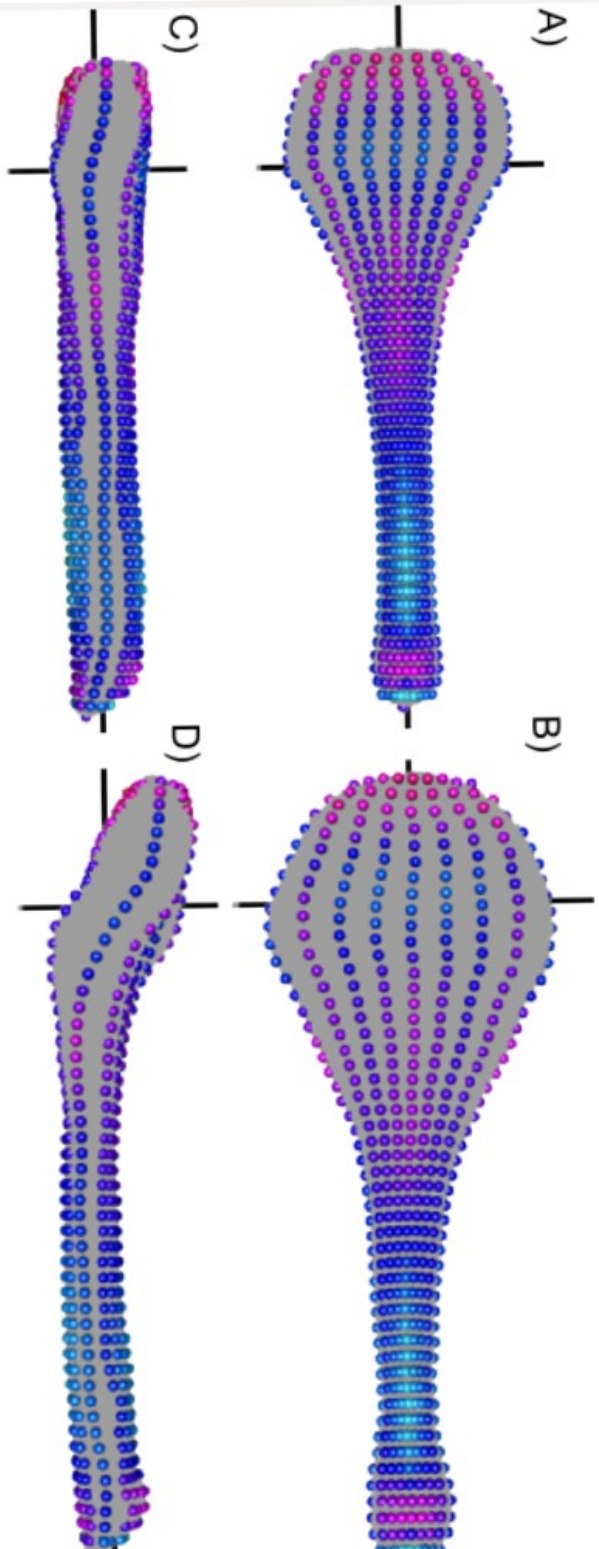
Figure 3. Limb-type gene expression varies among feathered and scaled pigeons and chickens. (**A**,**B**,**F**,**G**) qRT-PCR analyses of *Pitx1* and *Tbx*

The Genetic Basis of Baculum Size and Shape Variation in Mice

Nicholas G. Schultz[1], Jesse Ingels[2], Andrew Hillhouse[3], Keegan Wardwell[4], Peter L. Chang[1], James M. Cheverud[5], Cathleen Lutz[4], Lu Lu[2], Robert W. Williams[2] and Matthew D. Dean[1,*]

+ Author Affiliations

*To whom correspondence should be addressed. E-mail: matthew.dean@usc.edu



Abstract

The rapid divergence of male genitalia is a pre-eminent evolutionary pattern. This rapid divergence is especially striking in the baculum, a bone that occurs in the penis of many mammalian species. Closely related species often display diverse baculum morphology where no other morphological differences can be discerned. While this fundamental pattern of evolution has been appreciated at the level of gross morphology, nearly nothing is known of the genetic basis of size and shape divergence. Quantifying the genetic basis of baculum size and shape variation has been difficult because these structures generally lack obvious landmarks, so comparing them in three dimensions is not straightforward. Here we develop a novel morphometric approach to quantify size and shape variation from three-dimensional micro-CT scans taken from 369 bacula, representing 75 distinct strains of the BXD family of mice. We identify two quantitative trait loci (QTL) that explain ~50% of the variance in baculum size, and a QTL that explains more than 20% of the variance in shape. Together, our study demonstrates that baculum morphology may diverge relatively easily, with mutations at a few loci of large effect that independently modulate size and shape. Based on a combination of bioinformatic investigations and new data on RNA expression, we prioritized these QTL to 16 candidate genes which have hypothesized roles in bone morphogenesis, and may enable future genetic manipulation of baculum morphology.