



## Social Transmission of a Host Defense Against Cuckoo Parasitism

Nicholas B. Davies, *et al.*  
*Science* **324**, 1318 (2009);  
DOI: 10.1126/science.1172227

**The following resources related to this article are available online at [www.sciencemag.org](http://www.sciencemag.org) (this information is current as of June 5, 2009 ):**

**Updated information and services**, including high-resolution figures, can be found in the online version of this article at:

<http://www.sciencemag.org/cgi/content/full/324/5932/1318>

**Supporting Online Material** can be found at:

<http://www.sciencemag.org/cgi/content/full/324/5932/1318/DC1>

This article **cites 20 articles**, 6 of which can be accessed for free:

<http://www.sciencemag.org/cgi/content/full/324/5932/1318#otherarticles>

This article appears in the following **subject collections**:

Evolution

<http://www.sciencemag.org/cgi/collection/evolution>

Information about obtaining **reprints** of this article or about obtaining **permission to reproduce this article** in whole or in part can be found at:

<http://www.sciencemag.org/about/permissions.dtl>

# Social Transmission of a Host Defense Against Cuckoo Parasitism

Nicholas B. Davies\*† and Justin A. Welbergen

Coevolutionary arms races between brood parasites and hosts involve genetic adaptations and counter-adaptations. However, hosts sometimes acquire defenses too rapidly to reflect genetic change. Our field experiments show that observation of cuckoo (*Cuculus canorus*) mobbing by neighbors on adjacent territories induced reed warblers (*Acrocephalus scirpaceus*) to increase the mobbing of cuckoos but not of parrots (a harmless control) on their own territory. In contrast, observation of neighbors mobbing parrots had no effect on reed warblers' responses to either cuckoos or parrots. These results indicate that social learning provides a mechanism by which hosts rapidly increase their nest defense against brood parasites. Such enemy-specific social transmission enables hosts to track fine-scale spatiotemporal variation in parasitism and may influence the coevolutionary trajectories and population dynamics of brood parasites and hosts.

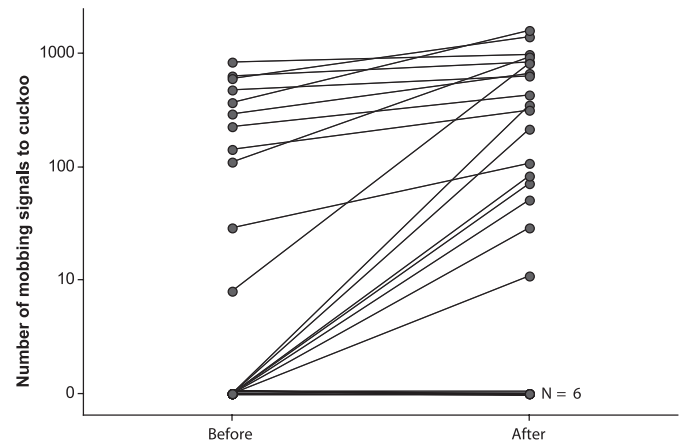
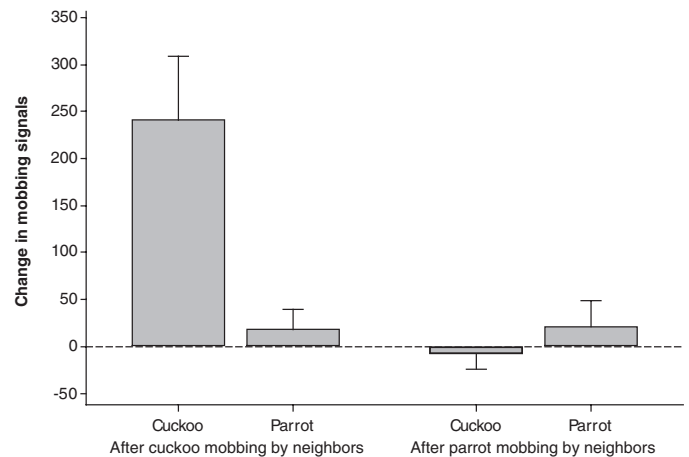
Darwin's concept of the "entangled bank" (1) captures an ecological world in which organisms evolve in response to changes not only in the physical environment but also in their competitors, predators, and parasites. Cuckoo-host interactions involve adaptations and counteradaptations in response to selection from the host and the parasite (2, 3) and are a model for investigating the outcome of biotic changes involving coevolution. However, host defenses can be costly. Attacking and mobbing an adult cuckoo can reduce the chance that the host nest is parasitized, but may attract nest predators or other brood parasites and can put the mobbers themselves at risk (4). Egg rejection may redeem a host's reproductive investment but entails a risk in that the host could reject its own eggs rather than the parasite egg (5). Therefore, defenses are advantageous only above a threshold level of parasitism (6, 7). A host population may experience conditions on either side of this parasitism threshold because of fine-scale spatial and temporal variation in the parasitism rate (4, 8, 9). Therefore, individual hosts would maximize their fitness by adjusting their defenses according to local cuckoo activity. As predicted, the propensity to mob adult cuckoos increases with local parasitism risk (4), and hosts are more likely to reject eggs or desert nests if they see a cuckoo at their nest (5, 10, 11). Phenotypically flexible host behavior is thus likely to explain small-scale geographical variation in host defenses, as well as result in rapid changes in defenses at a site within seasons and between years (4, 9, 12).

Host responses to adult cuckoos might involve learning, which would permit hosts to fine-tune defenses to the magnitude of the local threat (13). Hosts of the common cuckoo (*Cuculus canorus*) introduced from Britain to New Zealand some 130 years ago, and isolated from that brood parasite ever since, have retained the be-

havior of rejecting foreign eggs but, unlike their ancestral populations in Europe, do not mob a cuckoo mount (14). In general, birds isolated from predators are less responsive to nest enemies but can rapidly learn to increase their response, either from their own experience of predation or by observing others mobbing an enemy (13, 15). This suggests that introduced birds might have lost their response to adult cuckoos through lack of experience with parasit-

**Fig. 1.** Change in mobbing signals (number of rasp calls and mandible snaps per 5 min) given by focal pairs at their own nest to presentations of both a model cuckoo and a model parrot, after they had observed mobbing of either a cuckoo ( $n = 24$  nests) or a parrot ( $n = 17$  nests) at the nest on a neighboring territory. The figure plots mean change in response per 5 min ( $\pm$  SE) from trials before to trials after observation of neighbor mobbing (dashed line indicates no change). There was a significant interaction between the response to cuckoo versus parrot and the mobbing experience on the neighboring territory ( $F_{1,55.1} = 8.84$ ,  $P = 0.004$ ).

**Fig. 2.** Number of mobbing signals (rasp calls and mandible snaps per 5 min; log scale) given by focal pairs ( $n = 24$ ) to a model cuckoo at their own nest, before and after their experience of cuckoo mobbing on the neighboring territory.



Department of Zoology, University of Cambridge, Downing Street, Cambridge CB2 3EJ, UK.

\*To whom correspondence should be addressed. E-mail: n.b.davies@zoo.cam.ac.uk

†The authors contributed equally to this work.

ism rather than genetic change (14). In central Japan, common cuckoos began to parasitize azure-winged magpies (*Cyanopica cyana*) 40 years ago (16). Initially, magpies showed little defense, but aggression toward cuckoos and egg rejection increased more rapidly than can be explained by a change in host genotypes. Instead, it suggests that these birds learned to respond to the cuckoo and to exhibit a preexisting, but phenotypically flexible, egg rejection behavior (16).

Hosts might learn to increase their defenses against adult cuckoos from direct experience. However, opportunities for individual learning are limited because cuckoos are very secretive when surveying prospective hosts, and their laying visits are often extremely short [ $\sim 10$  s (3)]. Furthermore, close inspection by hosts may be dangerous because cuckoos resemble potentially lethal sparrowhawks (*Accipiter nisus*) (17). It is also possible that hosts increase defenses through social learning. When individual learning is costly and environments are variable, models suggest that it is beneficial to use the behavior of others as a source of information about local conditions (18). Such information could be socially transmitted, enabling individuals to modify their behavior by observing others (19). Social learning could lead to rapid cultural transmission of

defenses in local host populations if naïve birds learn to mob a novel enemy by observing a conspecific mobbing it, and then socially transmit the response to other individuals (20).

In fenland sites in Cambridgeshire, UK, where reed warblers (*Acrocephalus scirpaceus*) are the main cuckoo host (5), we found a striking dichotomy in reed warbler responses to cuckoo mounts placed next to their nests (4). At 48% of nests observed ( $n = 191$ ), the warblers mobbed the mounts, most of them intensively, with threat postures, swoops, and direct attacks, accompanied by loud rasp calls and bill snaps. However, at 52% of nests, there was no mobbing response, and warblers retreated after a brief inspection of the mount. Part of this variation is related to the degree of parasitism risk. Reed warblers were more likely to risk close inspection and mob if their nest was more vulnerable to parasitism (4). However, even at high-risk sites, many reed warblers did not mob. These were more likely to be younger, inexperienced birds, because earlier breeders both mobbed more (4) and were likely to be older, returning breeders (21). Our previous experiments showed that a resident pair's mobbing calls often attracted birds from neighboring territories (22). It is unlikely that reed warblers produced mobbing calls in order to attract neighbors, because mobbing calls were given irrespective of whether other pairs were nesting nearby, and attracted neighbors were often chased away (22). Nevertheless, neighbors could eavesdrop on residents' mobbing behavior and thus learn about the danger of cuckoos.

In order to test whether reed warblers acquire or enhance their mobbing defense through social learning from experienced individuals, we examined the response to model cuckoos and parrots (parrots were chosen to represent a novel, harmless intruder) (21). At 41 focal nests, we first recorded the baseline response to models as the number of mobbing signals (rasp calls and mandible snaps) given within 5 min after the arrival of the first focal warbler to within 1 m of the model (21). Then, the focal pair was given

an opportunity for social learning by the placement of either a model cuckoo or a model parrot next to the nest on the adjacent, neighboring territory. Therefore, each focal pair experienced neighbor mobbing of only one of the two models, either a cuckoo ( $n = 24$  nests) or a parrot ( $n = 17$  nests). To encourage a mobbing response from the neighbors and to attract the focal pair into the neighboring territory, a loud-speaker broadcast reed warbler mobbing signals near the model for 10 min (21). After this playback period, we recorded the mobbing signals of the neighboring birds for 1 min. Finally, we retested the focal pair with both a cuckoo and a parrot model at their own nest, and then again 3 to 6 days later. We predicted that if social learning was specific for the mobbing stimulus, then the focal pair's mobbing would increase only in response to the model mobbed on the neighboring territory.

Focal pairs were attracted to the models (to a distance of  $<2$  m) at the neighbors' nest in the majority of cases [25 out of 27 (25/27); in 14/41 trials, the vegetation was too dense to record focal bird activity]. They were as likely to be attracted to the neighbors' nest during parrot trials (11/11) as during cuckoo trials (14/16; Fisher's exact test,  $P = 0.499$ ). Reed warblers normally mob model cuckoos much more strongly than model parrots (4), but our data show that the playback stimulated equally strong neighbor mobbing of both models. Neighbors were as likely to mob the parrot (14/17) as the cuckoo model (19/24;  $P = 1.0$ ) and gave as many mobbing signals to the parrot (mean  $\pm 1$  SE,  $57.1 \pm 17.9$ /min) as to the cuckoo ( $66.4 \pm 14.9$ /min;  $t$  test,  $t_{34} = 0.40$ ,  $P = 0.692$ ). Therefore, parrot and cuckoo presentations on neighboring territories provided equal opportunities for social learning by the focal birds. The proportion of neighbors that mobbed the cuckoo after hearing the playbacks was significantly higher than the proportion of focal pairs that mobbed the cuckoo in baseline trials (19/24 versus 18/41;  $\chi^2 = 7.68$ ,  $df = 1$ ,  $P = 0.006$ ). Thus, not only does

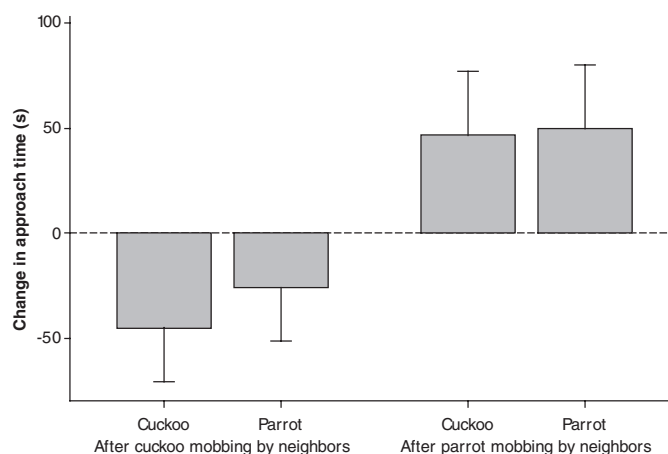
mobbing attract neighboring individuals (22), it can also be induced by mobbing signals [response facilitation (19)] and so is a likely candidate for social transmission.

Nevertheless, when a parrot was mobbed on the neighboring territory it had no effect on the mobbing of either parrots or cuckoos by focal pairs at their own nests. In contrast, neighbors mobbing a cuckoo led to a marked increase in focal pair mobbing, but only of cuckoos, not of parrots (Fig. 1). Therefore only cuckoo mobbing led to a response, and furthermore, observational conditioning (19) was specific to cuckoos. This result is not confounded by the fact that focal individuals that experienced cuckoo mobbing on a neighboring territory encountered a cuckoo three times during our experimental trials, whereas those that experienced parrot mobbing encountered cuckoos just twice (21).

After experiencing neighbor mobbing of a cuckoo, all 11 focal pairs that mobbed the cuckoo in their first, baseline trial increased their cuckoo mobbing, and 7/13 focal pairs that did not mob the cuckoo on the first trial now did so (Fig. 2). We were unable to explain why six of the pairs retained a nonmobbing response; the increase in mobbing by the focal pair was not related to whether we saw them attracted to the neighbors' nest ( $F_{1,3,5} = 0.35$ ,  $P = 0.581$ ), nor to the magnitude of the neighbors' mobbing response ( $F_{1,17,8} = 0.08$ ,  $P = 0.776$ ). Fourteen of the 24 focal pairs that experienced neighbor mobbing of cuckoos were retested with the cuckoo model 3 to 6 days later (23); their mobbing response was still significantly higher than on the first, baseline trial (paired  $t$  test,  $t_{13} = 2.55$ ,  $P = 0.024$ ) and as strong as on the previous post-social learning trial ( $t_{13} = -1.19$ ,  $P = 0.255$ ), indicating that any increased mobbing response was retained.

Cuckoo mobbing by neighbors alerted the focal pair, resulting in a relatively quicker inspection of both cuckoo and parrot mounts as compared to parrot mobbing (Fig. 3). This suggests that focal pair attention is directed at the neighbor responses toward specific enemies, not just at any intruder. This response to cuckoos is most likely adaptive because female cuckoos have laying territories (5), and cuckoo activity on a neighbor's territory signals an increased risk of parasitism (4). More rapid inspection of potential nest enemies is typical of reed warblers nesting in sites with a high risk of parasitism (21). The specific increase in cuckoo mobbing (Fig. 1) suggests that reed warblers may be predisposed to learn to target cuckoos. However, it is also possible that response intensity increased toward the cuckoo mounts because our procedures added to their stimulus salience because of prior experience with cuckoos, which could not have been the case for parrots. Predispositions to learn to respond to stimuli relevant to survival and reproduction have been demonstrated in rhesus macaques (*Macaca mulatta*), in which social learning readily leads to fear of snakes but not of harmless stimuli, such as flowers and rabbits (24).

**Fig. 3.** Change in a focal pair's time to first approach to cuckoo and parrot models presented at their own nest, after experiencing mobbing of either a cuckoo ( $n = 24$  nests) or a parrot ( $n = 17$  nests) at a nest on a neighboring territory. The figure plots mean  $\pm$  SE change in approach time from trials before to trials after observation of neighbor mobbing. There was an effect of mobbing experience on the neighboring territory ( $F_{1,78,0} = 7.23$ ,  $P = 0.009$ ) but no effect of model type ( $F_{1,78,0} = 0.11$ ,  $P = 0.738$ ) and no interaction between mobbing experience and model type ( $F_{1,78,0} = 0.00$ ,  $P = 0.966$ ).



Reed warblers distinguish cuckoos from other nest enemies (22) and specifically adjust cuckoo mobbing to local parasitism risk (4). The specificity of social learning observed here provides evidence that mobbing is a phenotypically plastic trait, adaptive in the context of brood parasitism. We suggest that naïve individuals may learn from bolder birds or from those who, by chance, observed a cuckoo depredate or parasitize their nest. Further experiments are needed to test whether social learning leads only to a change in the perception of parasitism risk or also may involve the refining of a template for cuckoo recognition, akin to the genetic predispositions that guide learning in other contexts (13).

Social learning could trigger a marked increase in host defenses; by focusing on neighbors' responses to adult cuckoos, focal pairs not only increase cuckoo mobbing as a front line of defense (4) but are also alerted to increased vigilance (11) and egg rejection (5, 10). Therefore, our results support the hypothesis that rapid changes in host defenses (14, 16) may reflect social transmission of responses to adult cuckoos as nest enemies. Social learning has implications for the coevolutionary trajectories of brood para-

sites and hosts because it promotes phenotypic plasticity that can drive or impede genetic evolution (25). Furthermore, by influencing how rapidly hosts lose or gain defenses, social learning may affect the population dynamics of both brood parasites and hosts (26).

#### References and Notes

1. C. Darwin, *On the Origin of Species* (Murray, London, 1859).
2. S. I. Rothstein, S. K. Robinson, Eds., *Parasitic Birds and Their Hosts: Studies in Coevolution* (Oxford Univ. Press, Oxford, 1998).
3. N. B. Davies, *Cuckoos, Cowbirds and Other Cheats* (Poysner, London, 2000).
4. J. A. Welbergen, N. B. Davies, *Curr. Biol.* **19**, 235 (2009).
5. N. B. Davies, M. de L. Brooke, *Anim. Behav.* **36**, 262 (1988).
6. A. Lotem *et al.*, *Anim. Behav.* **49**, 1185 (1995).
7. N. B. Davies *et al.*, *Proc. R. Soc. London Ser. B* **263**, 925 (1996).
8. I. J. Øien *et al.*, *J. Anim. Ecol.* **65**, 147 (1996).
9. A. K. Lindholm, *J. Anim. Ecol.* **68**, 293 (1999).
10. A. Moksnes *et al.*, *Ibis* **142**, 247 (2000).
11. N. B. Davies *et al.*, *Anim. Behav.* **65**, 285 (2003).
12. M. de L. Brooke *et al.*, *Proc. R. Soc. London Ser. B* **265**, 1277 (1998).
13. A. S. Griffin, *Learn. Behav.* **32**, 131 (2004).
14. K. Hale, J. V. Briskie, *J. Avian Biol.* **38**, 198 (2007).
15. R. F. Maloney, I. G. MacLean, *Anim. Behav.* **50**, 1193 (1995).
16. H. Nakamura *et al.*, in *Parasitic Birds and Their Hosts: Studies in Coevolution*, S. I. Rothstein,

17. S. K. Robinson, Eds. (Oxford Univ. Press, Oxford, 1998), pp. 94–112.
18. N. B. Davies, J. A. Welbergen, *Proc. R. Soc. London Ser. B* **275**, 1817 (2008).
19. R. Boyd, P. J. Richerson, *Lect. Math. Life Sci.* **20**, 1 (1989).
20. W. Hoppitt, K. N. Laland, *Adv. Stud. Behav.* **38**, 105 (2008).
21. E. Curio *et al.*, *Science* **202**, 899 (1978).
22. See supporting material on Science Online for methods and additional text and data.
23. J. A. Welbergen, N. B. Davies, *Anim. Behav.* **76**, 811 (2008).
24. These tests included 5/11 that mobbed during the baseline trial, 5/7 that did not mob during the baseline trial but did so after social learning, and 4/6 that retained a nonmobbing response throughout.
25. M. Cook, S. Mineka, *J. Abnorm. Psychol.* **98**, 448 (1989).
26. T. D. Price *et al.*, *Proc. R. Soc. London Ser. B* **270**, 1433 (2003).
27. F. Takasu *et al.*, *Am. Nat.* **142**, 819 (1993).
28. We thank the Natural Environment Research Council; the National Trust, C. Thorne, and the Wicken Fen Group; English Nature; and M. Brooke, J. Davies, R. Kilner, O. Krüger, and the Behavioural Ecology Group at Cambridge.

#### Supporting Online Material

www.sciencemag.org/cgi/content/full/324/5932/1318/DC1  
Materials and Methods  
SOM Text  
Fig. S1  
References

12 February 2009; accepted 20 April 2009  
10.1126/science.1172227

## Epigenetic Temporal Control of Mouse *Hox* Genes in Vivo

Natalia Soshnikova<sup>1</sup> and Denis Duboule<sup>1,2\*</sup>

During vertebrate development, the temporal control of *Hox* gene transcriptional activation follows the genomic order of the genes within the *Hox* clusters. Although it is recognized that this “*Hox* clock” serves to coordinate body patterning, the underlying mechanism remains elusive. We have shown that successive *Hox* gene activation in the mouse embryo is closely associated with a directional transition in chromatin status, as judged by the dynamic progression of transcription-competent modifications: Increases in activation marks correspond to decreases in repressive marks. Furthermore, using a mouse in which a *Hox* cluster was split into two pieces, we document the necessity to maintain a clustered organization to properly implement this process. These results suggest that chromatin modifications are important parameters in the temporal regulation of this gene family.

**H***ox* genes, which are generally arranged in clusters at genomic loci, are essential for patterning the anterior to posterior animal body axis (1–3). In vertebrates, these genes are activated in a time sequence that follows their physical order within the cluster, a process referred to as temporal collinearity (4). This property is observed in animals developing their trunk via a rostral to caudal time sequence, yet the underlying molecular mechanism is elusive (5, 6). A progressive transition in chromatin state was hypothesized (7, 8), whereby an initially repressed configuration becomes open for

transcription. The subsequent observation of chromatin decondensation at these loci when transcription is induced supported this hypothesis (9).

*Hox* genes are repressed by Polycomb group (PcG) proteins (10). Mutation of *PcG* genes induces ectopic *Hox* expression and results in posterior homeotic transformations (11, 12). PcG proteins form large complexes with histone-modifying activities; for example, Polycomb Repressive Complex 2 (PRC2) trimethylates histone H3 at lysine 27 (H3K27me3) (13–16), an essential modification for long-term repression of target genes. In contrast, *Trithorax* group (TrxG) proteins antagonize *PcG* proteins and activate target gene expression (10). TRX complexes trimethylate histone H3 at lysine 4 (H3K4me3), a mark generally associated with active transcription (17). Genome-wide studies of both H3K27me3 and H3K4me3 modifications in embryonic stem cells (ESC) and other cultured cells have revealed specific profiles during the maintenance phase of *Hox* gene expression in vitro

(18–21). We looked at the in vivo dynamics of chromatin marks during the sequential activation of *Hoxd* genes in developing murine tail buds.

We dissected out mouse tail buds during late somitogenesis when the last *Hox* genes become transcribed (22) and performed expression profiling at E8.5 (embryonic day 8.5), E9.0, and E9.5 (Fig. 1A) using tiling arrays covering 2 Mb of DNA containing the *HoxD* cluster. This highly syntenic region (23) also contains four ubiquitously expressed genes, *Atp5g3*, *Lnp*, *Mx2*, and *Hmrpa3*, and two gene deserts (fig. S1). Transcription of *Hoxd1* to *Hoxd9* was active at all three time points (Fig. 1B), reflecting the onset of *Hox* gene transcription during early gastrulation. However, transcriptional progression was observed for more posterior genes, with *Hoxd10* and *Hoxd11* transcribed at E9.0 (Fig. 1B), whereas by E9.5 transcriptional activity had spread over *Hoxd12*, *Hoxd13*, and the nearby neighbor gene *Evx2* (Fig. 1B). Low transcript levels were detected for *Hoxd13* before activation of *Hoxd10* (Fig. 1B, arrow).

We mapped the sites occupied by RNA polymerase II using chromatin immunoprecipitation combined with hybridization on tiling array (ChIP-chip) (Fig. 1B and fig. S1). The Pol II profile corresponded to transcribed regions; whereas virtually no Pol II was scored centromeric to *Hoxd10* at E8.5, signals were detected for both *Hoxd10* and *Hoxd11* at E9.0. At E9.5, the whole centromeric part of the cluster was fully occupied by Pol II (Fig. 1B), indicating that it was recruited in a collinear manner too. In agreement with transcript profiling, a weak Pol II binding was scored at the *Hoxd13* locus at E8.5. Similarly, high levels of H3K9/K14 acetylation (AcH3) were found in

<sup>1</sup>National Research Centre Frontiers in Genetics, Department of Zoology and Animal Biology, University of Geneva, Sciences III, Quai Ernest-Ansermet 30, 1211 Geneva 4, Switzerland. <sup>2</sup>National Research Centre Frontiers in Genetics, School of Life Sciences, Federal Institute of Technology (EPFL), Lausanne, Switzerland.

\*To whom correspondence should be addressed. E-mail: Denis.Duboule@unige.ch; Denis.Duboule@epfl.ch