

which argues that the effect was not an artefact of apparatus or method. This observation implies that processing of information from the dominant (right) visual field is more extensive than that of information from the non-dominant (left) visual field, irrespective of the hemisphere or region within which this processing is realized. □

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## Homeotic genes and the regulation and evolution of insect wing number

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THE evolution of wings catalysed the radiation of insects which make up some 75 per cent of known animals. Fossil evidence suggests that wings evolved from a segment of the leg<sup>1</sup> and that early pterygotes bore wings on all thoracic and abdominal segments<sup>2</sup>. The pterygote body plan subsequently diverged producing orders bearing three, two or just one pair of thoracic wings. We have investigated the role of homeotic genes in pterygote evolution by examining their function in *Drosophila* wing development and their

expression in a primitive apterygote. Wing formation is not promoted by any homeotic gene, but is repressed in different segments by different homeotic genes. We suggest here that wings first arose without any homeotic gene involvement in an ancestor with a homeotic 'groundplan' similar to modern winged insects and that wing formation subsequently fell under the negative control of individual homeotic genes at different stages of pterygote evolution.

The *Drosophila* wing and haltere primordia can first be visualized as discrete clusters of *vestigial*-expressing (Fig. 1a) and *snail*-expressing<sup>3</sup> (Fig. 1b) cells in the second and third thoracic segments of the stage 12 embryo. Antibodies specific for the *vestigial* and *snail* proteins serve as interchangeable markers for the flight appendage primordia because they label the same cells at different embryonic stages (Fig. 1c–h). Both genes have a role in formation of the flight appendages because *vestigial* mutants lack wings and halteres<sup>4</sup> and weak *snail* mutants that survive to adulthood exhibit a hemithorax or missing haltere phenotype<sup>5</sup>.

The restriction of the flight appendage primordia to the two posterior thoracic segments of the *Drosophila* embryo suggests a role for homeotic genes in specifying which segments will bear wings. Because the *Antennapedia* (*Antp*) gene is expressed in the second and third thoracic segments during embryogenesis<sup>6,7</sup>, this gene might regulate the formation of the wing and haltere primordia. Surprisingly, there is no requirement for *Antp* gene function during the formation of these primordia (Fig. 1i). This suggests that wing primordium formation as it occurs in the mesothoracic (T2) segment represents a 'ground state' of embryonic development without the input of the homeotic genes. Furthermore, this indicates that ectopic wing primordia found in other homeotic mutants (see below) result from derepression of the wing developmental programme, rather than the derepression of *Antp*.

It appears that, rather than *Antp* positively regulating wing and haltere formation in T2 and T3, other homeotic genes repress the wing primordia in different body segments. For example, the *Sex combs reduced* (*Scr*) gene is expressed in the labial and prothoracic (T1) segment<sup>8,9</sup>. In *Scr* mutant embryos ectopic flight appendage primordia arise in the T1 segment, demonstrating that *Scr* normally represses wing formation there (Fig. 1j). Similarly, in mutants for the *Ultrabithorax* (*Ubx*) gene, which normally functions in both the metathoracic (T3) and first abdominal segment (A1)<sup>10</sup>, the haltere primordia in T3 expands to the size of the wing primordia (data not shown) and an ectopic primordium forms in the ventral region of A1 (Fig. 1k). The potential for wing formation exists in most or all trunk segments as illustrated by embryos lacking the *abd-A* gene (Fig. 1l) or the *Ubx* and *abdominal-A* (*abd-A*) genes (Fig. 1m) (see also ref. 11).

The experiments above address only the formation of the embryonic wing primordia and not the growth and development of adult wings from these primordia. Cells may respond differently to the expression of a particular homeotic gene at different stages of development<sup>12</sup>; thus *Antp* could function during later stages of wing formation. There are no genetic requirements for *Antp*, *Scr* and *Ubx* in the formation of adult wings (*Antp* is required in the mesonotum)<sup>13</sup>. Furthermore, we find that *Antp* protein expression is largely absent from the region of the growing third instar imaginal disc that will give rise to the wing itself (Fig. 2a, b), again illustrating that this appendage forms without homeotic input.

To test whether the absence of *Antp* was important for wing formation, we examined wing discs in which *Antp* was ectopically expressed using the GAL4-UAS system<sup>14</sup>. Ectopic *Antp* expression had no effect on wing disc morphology or *vestigial* expression (not shown). Similarly, we examined the impact of *Scr* expression, which is normally absent from the wing disc, upon wing development. We find that wing development is sensitive to *Scr* repression in the embryo, but not at later stages. For instance, when *Scr* expression is driven ectopically in the growing wing disc, there is no apparent effect on wing formation as indi-

cated by disc morphology or *vestigial* gene expression (Fig. 2c). By contrast, ectopic *Scr* expression in the early embryo represses *vestigial* gene expression in the wing and haltere primordia (Fig. 2d, e).

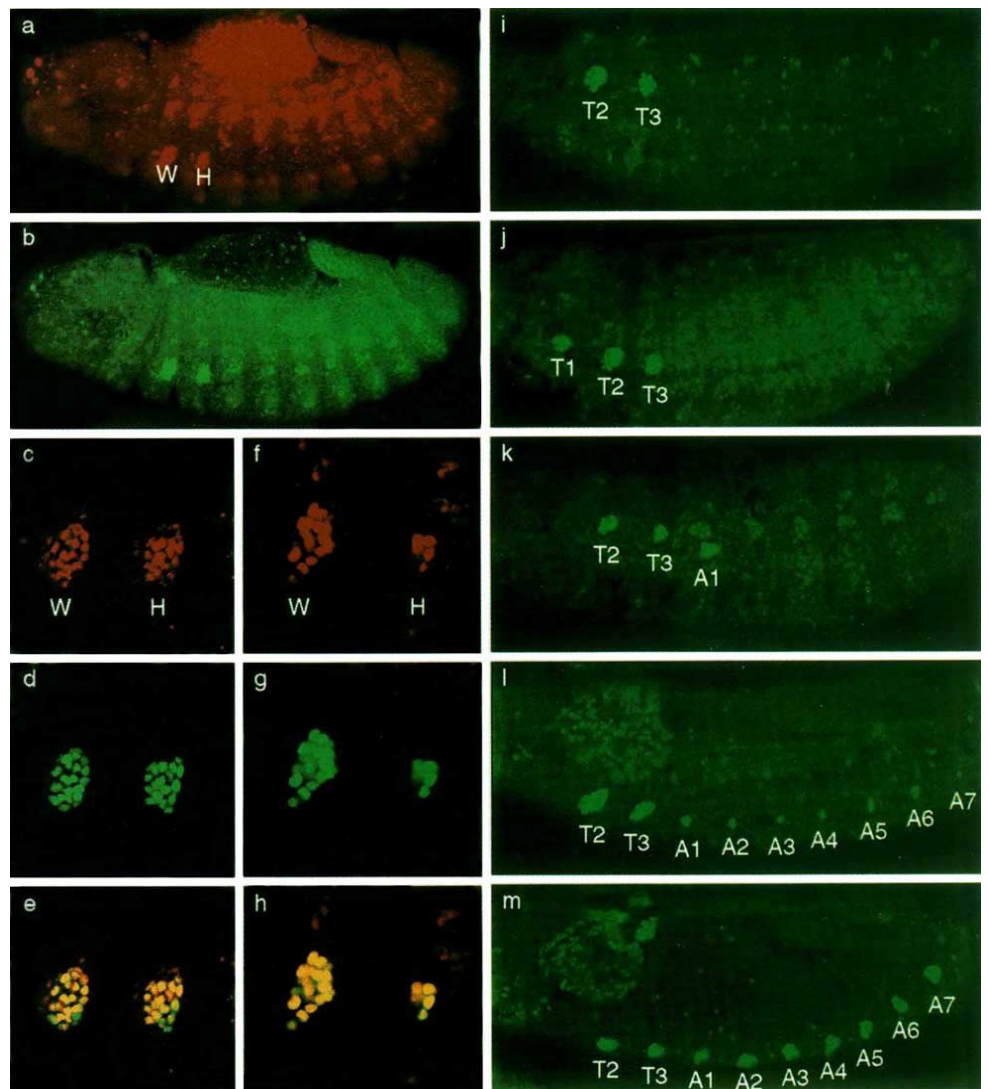
Fossil evidence indicates that wings or wing-like structures first appeared on all body segments of primitive pterygotes<sup>15,16</sup> probably at first in the immature stages. This raises the question of how *Scr*, *Ubx* and *abd-A* came to repress wing formation in the course of pterygote and dipteran evolution. Two possible explanations are that these genes came into existence during pterygote evolution or that the homeotic genes diversified before pterygote evolution and homeotic regulation of wing number and type evolved subsequently. The presence of distinct *Scr*, *Ubx* and *abd-A* genes in crustacea<sup>17</sup> suggests that the homeotic genes diversified before the insect/crustacean divergence and would each be present in a pterygote ancestor.

One way to address the issue of homeotic gene expression in pterygote ancestors is to examine their deployment in a primitive apterygote which would represent the condition before the 'invention' of wings. The Thysanura are the sister group of

pterygotes<sup>16</sup> which have retained many primitive features. The *Ubx* and *abd-A* proteins<sup>18</sup> are expressed in the posterior thoracic and anterior abdominal segments of the apterygote thysanuran *Thermobia domestica* (Fig. 3), as they are in pterygotes<sup>18</sup>. The similarity between apterygote and modern winged insects' BX-C protein patterns indicates that the spatial domains of these homeotic genes were probably established before the origin of wings and the diversification of the pterygotes. In primitive pterygotes that bore wings on abdominal segments, BX-C genes, although present and expressed in the abdomen, apparently did not regulate wing formation as they do in modern pterygotes.

We propose a plausible scenario for the role of homeotic genes in pterygote evolution that is based on the above developmental genetic studies of *Drosophila*, comparative analysis of homeotic gene expression and the fossil record (Fig. 4). When wings or their progenitors first arose and were found on each body segment, there was no homeotic gene input into their number or design (compare Fig. 4a and b). This is reflected by the absence of any requirement for *Antp* function in *Drosophila* embryos (Fig. 1i) or imaginal discs<sup>13</sup> for the establishment of the wing

FIG. 1 Homeotic genes repress but do not promote formation of the flight appendage primordia in *Drosophila*. a, c, Stage-12 *Drosophila* embryo, about 20 *vestigial*-expressing cells mark the wing (W) and haltere (H) primordia, the rest of the pattern is largely in the muscle precursors; b, d, stage-12 *Drosophila* embryo, about 20 *snail*-expressing cells mark the wing and haltere primordia, the other labelled cells are in proneural clusters of the peripheral nervous system; e, stage-12 embryo double-labelled for *vg* and *sna* proteins, the patterns within the wing and haltere primordia are nearly or completely coincident; f, g, h, stage-17 embryo double-labelled for *vg*, (f), *sna*, (g), the patterns are coincident (h). The difference in the number of cells stained at the two stages is due to the restriction of gene expression to a subset of the cells within the developing disc. i–m, Wing and haltere primordia in stage-15 embryos homozygous for either the *Antp*<sup>W10</sup>, *Scr*<sup>W17</sup>, *Ubx*<sup>6,28</sup>, *Ubx*<sup>109</sup> or *abd-A*<sup>Mx1</sup> mutations detected by *snail* expression. i, Wing and haltere primordia are normal in an *Antp* mutant embryo; j, an additional primordium forms in the T1 segment of a *Scr* mutant embryo; k, the haltere primordia are enlarged and an ectopic primordium forms in a *Ubx* mutant embryo; l, a few *snail*-expressing cells arise in the posterior of segments A1–A7 in an *abd-A*<sup>-</sup> embryo, these cells stain because the wing primordia cross the parasegmental boundary and *Ubx* (which represses wing formation in the anterior compartment of A1–A7 in an *abd-A* mutant see (m)) is still repressed in the posterior compartment of segments A1–A7 in an *abd-A* mutant embryo by the *en* protein<sup>21</sup>; and m, wing primordia arise in segments T2–A7 in an *Ubx*<sup>109</sup> (*Ubx*<sup>-</sup>, *abd-A*<sup>-</sup>) embryo. All embryos are magnified ×540 except c–h which are magnified ×1,600. METHODS. *Drosophila* embryos (OregonR) were stained with a rabbit anti-*vestigial* antibody and/or a monoclonal antibody to the *snail* protein<sup>26</sup> (gift from A. Alberga), an unconjugated goat anti-rabbit and/or a biotinylated donkey anti-mouse secondary antibody was added,



and either indodicarbocyanine (Cy5)-conjugated streptavidin and/or a fluorescein-conjugated donkey anti-goat antibody were used as tertiary reagents. All embryos were visualized by confocal microscopy and mutant genotypes were confirmed by staining with antibodies against *Scr*, *Antp*, *Ubx*, or *Ubx/abd-A*.

FIG. 2 *Antp* and *Scr* do not influence imaginal wing outgrowth. **a**, Third instar imaginal disc stained with a monoclonal antibody to *Antp* which is expressed in a minor subset of anterior cells (purple), primarily in the future dorsal notum. **b**, Only a few cells of the thousands that will make up the future wing pouch and wing hinge (depicted here in green by *vestigial* expression) express the *Antp* protein (purple). **c**, UAS-*Scr* expression (red) driven along the A/P boundary by a *ptc*-GAL4 driver does not alter the level or pattern of *vestigial* expression (green) or the morphology of the growing third instar disc. **d**, *Scr* expression (purple) and *vestigial* expression (green) in a wild-type embryo; the wing and haltere primordia (are indicated by yellow arrows). **e**, The pattern of *Scr* expression in *Antp* P1GAL4-UAS; *Scr* embryo is shown is purple; *vestigial* expression (green) has been repressed where the wing and haltere primordia would normally arise (yellow arrows).

**METHODS.** Imaginal discs were stained as described in refs 27 and 28 and embryos as described in Fig. 1. UAS-*Scr*<sup>29</sup> expression in imaginal discs was driven with a *ptc*-GAL4 driver<sup>30</sup> and in embryos with an *Antp* P1-GAL4 driver<sup>29</sup>.

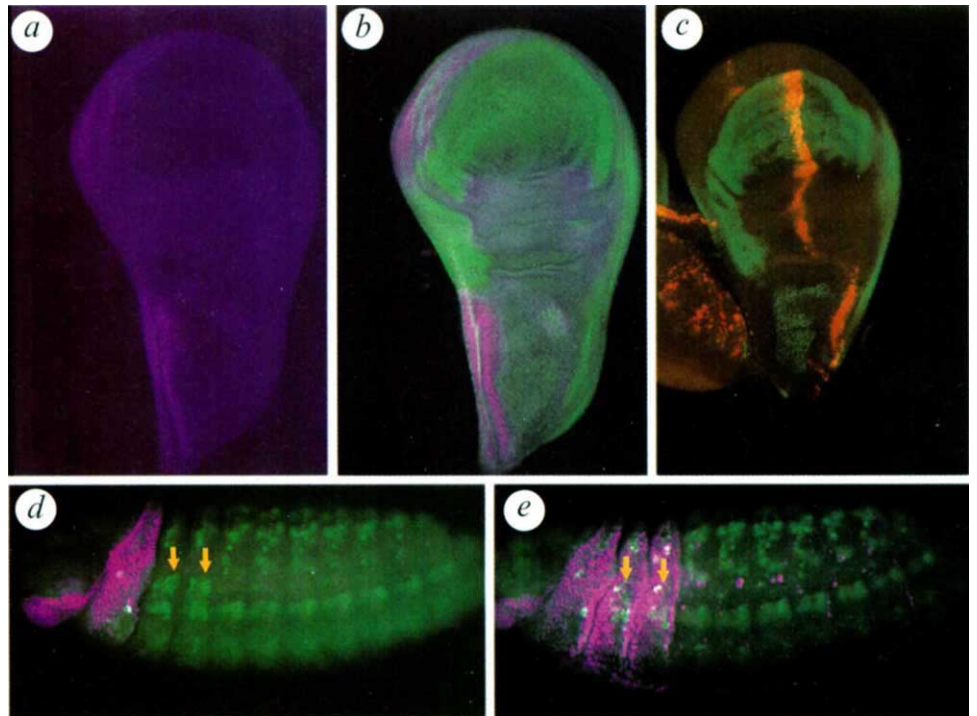
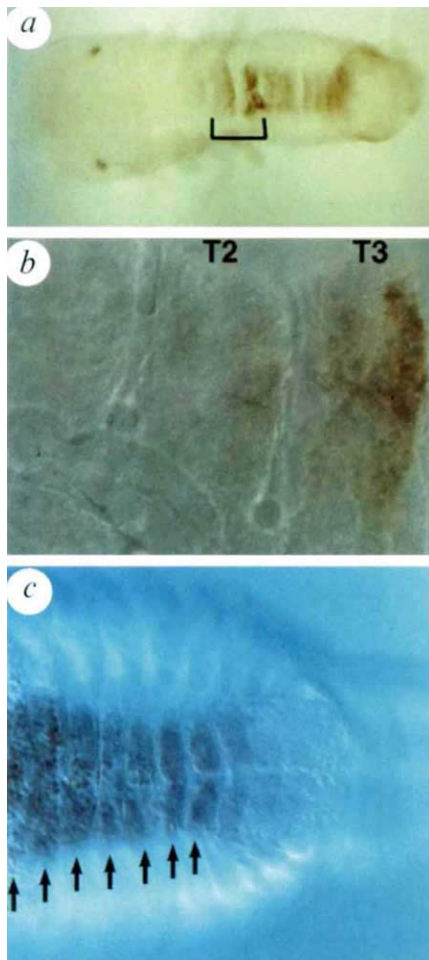


FIG. 3 BX-C proteins are deployed in the abdominal segments of a primitive apterygote insect (*Thermobia*). **a**, The *Ubx*/*abd-A* proteins are expressed in the posterior thorax and most of the abdominal segments, with the highest level of staining in T3 and A1 (bracket). Some of the gaps in staining, for example between T3 and A1 in the middle of the bracketed region, are due to curvature and buckling of the embryo, not the lack of antigen expression. **b**, A high-magnification view of the thorax reveals high levels of staining in the posterior of T3 and faint levels of staining in posterior T2. **c**, A high-magnification view of the abdomen reveals a contiguous pattern of expression from A1 to A7 (seven arrows). **METHODS.** *Thermobia domestica* embryos were incubated with an antibody that recognizes the *Ubx* and *abd-A* proteins<sup>13</sup> of flies, locusts and butterflies, and horseradish peroxidase-conjugated goat anti-mouse secondary antibody and stained with diaminobenzidine.



primordia or morphogenesis of the wing. Subsequently, certain genes involved in wing formation must have evolved or acquired regulatory elements which responded to homeotic proteins. The ensuing modifications of the original pterygote wing or pro-wing pattern could have been achieved by the evolution of *Scr*-, *abd-A*- and *Ubx*-responsive *cis*-regulatory elements in genes involved in wing formation and/or through subtle changes in the expression domains of these homeotic repressors (for example, expression of *Scr* in T1 or modification of the *Ubx* pattern in T3). Assuming that the domains of homeotic gene expression have been conserved among insects (as suggested in part by Fig. 3), the evolution of *Scr*-responsive elements led to the modification (Fig. 4d) or elimination (Fig. 4c) of prothoracic wings and the evolution of *abd-A*- and *Ubx*-responsive elements led to the elimination of abdominal wings (Fig. 4d) and, in the Diptera, the reduction of metathoracic wings (Fig. 4f).

The diversification of the pterygotes provides an example of how a new structure, once invented independently of the homeotic genes, may be modified by homeotic genes in the course of evolution. It is possible that the evolution of other body plans has followed a similar sequence of invention followed by homeotic modification or reduction of a character. The formation of trunk limb primordia in *Drosophila* is also under the negative

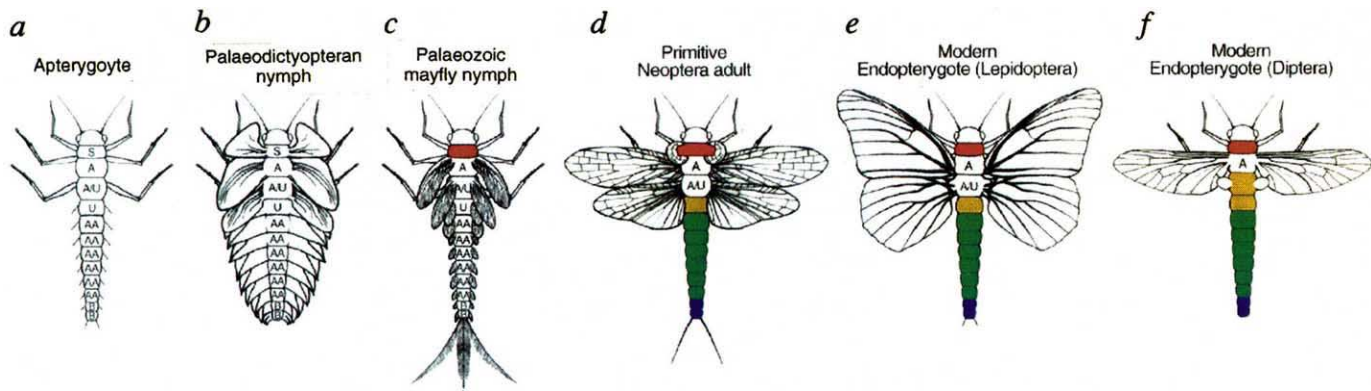


FIG. 4 Homeotic genes and pterygote evolution. A highly stylized view of pterygote evolution from an apterygote ancestor (a) to various extinct orders (b–d) to modern Lepidoptera (e) and Diptera (f). The fossil evidence upon which a–d are based is from Kukalova-Peck<sup>1,2</sup> and specimens described by Wootton<sup>15</sup>. The segmental body plan of each animal is represented as identical body designs and the homeotic genes expressed in different segments are represented by letters (S, Scr, A, Antp; U, Ubx; AA, abd-A; B, abd-B). The segments in which individual

homeotic genes are postulated to regulate wing formation are indicated by colouring in the respective segments at the appropriate stage of pterygote evolution (Scr, red; Ubx, yellow; abd-A, green). Note that *Antp* is never depicted as regulating wing formation. In the palaeodictyopteran nymph (b), the homeotic 'groundplan' was fully diversified as in modern insects but wings formed on all segments. In four-winged insects (e) Ubx represses wing formation in A1 and on two-winged insects (f) in T3 as well.

control of homeotic genes<sup>19,20</sup>, and as in wing formation, *Antp* is not required for limb formation<sup>21</sup>. It is possible that the evolution of insects from a myriapod-like ancestor<sup>22</sup> involved the evolution of homeotic repression of limb formation, rather than the evolution of new homeotic genes, as has been postulated<sup>23–25</sup>. The evolution of regulatory interactions between homeotic proteins and genes involved in appendage formation and of changes in homeotic gene regulation may then be the key steps in the diversification of arthropod body plans and appendages. □

## Defective axonal transport in a transgenic mouse model of amyotrophic lateral sclerosis

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AMYOTROPHIC lateral sclerosis (ALS) is a degenerative disease of motor neurons, characterized by depositions of neurofilaments in the perikarya and proximal axons. The pathogenesis of ALS remains poorly understood, but two lines of evidence suggest that neurofilament accumulation may play a causal role. First, transgenic mice that overexpress neurofilament proteins show motor neuron degeneration<sup>1–3</sup> and, second, variant alleles of the neurofilament heavy-subunit gene (*NF-H*) have been found in some human ALS patients<sup>4</sup>. To investigate how disorganized neurofilaments might cause neurodegeneration, we examined axonal transport of newly synthesized proteins in mice that overexpress the human *NF-H* gene<sup>1</sup>. We observed dramatic defects of axonal transport, not only of neurofilament proteins but also of other proteins, including tubulin and actin. Ultrastructural analysis revealed a paucity of cytoskeletal elements, smooth endoplasmic reticulum and especially mitochondria in the degenerating axons. We therefore propose that the neurofilament accumulations observed in these mice cause axonal degeneration by impeding the transport of components required for axonal maintenance, and that a similar mechanism may account for the pathogenesis of ALS in human patients.

The motor dysfunction in mice with a modest overexpression of the human *NF-H* transgene (line 200) (ref. 1) progresses during ageing by the atrophy and subsequent degeneration of axons distal to the neurofilament swellings. Light microscopy examination of the L5 ventral roots from a 2-year-old *NF-H* transgenic mouse revealed a massive degeneration of large axons derived from spinal motor neurons (Fig. 1).

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