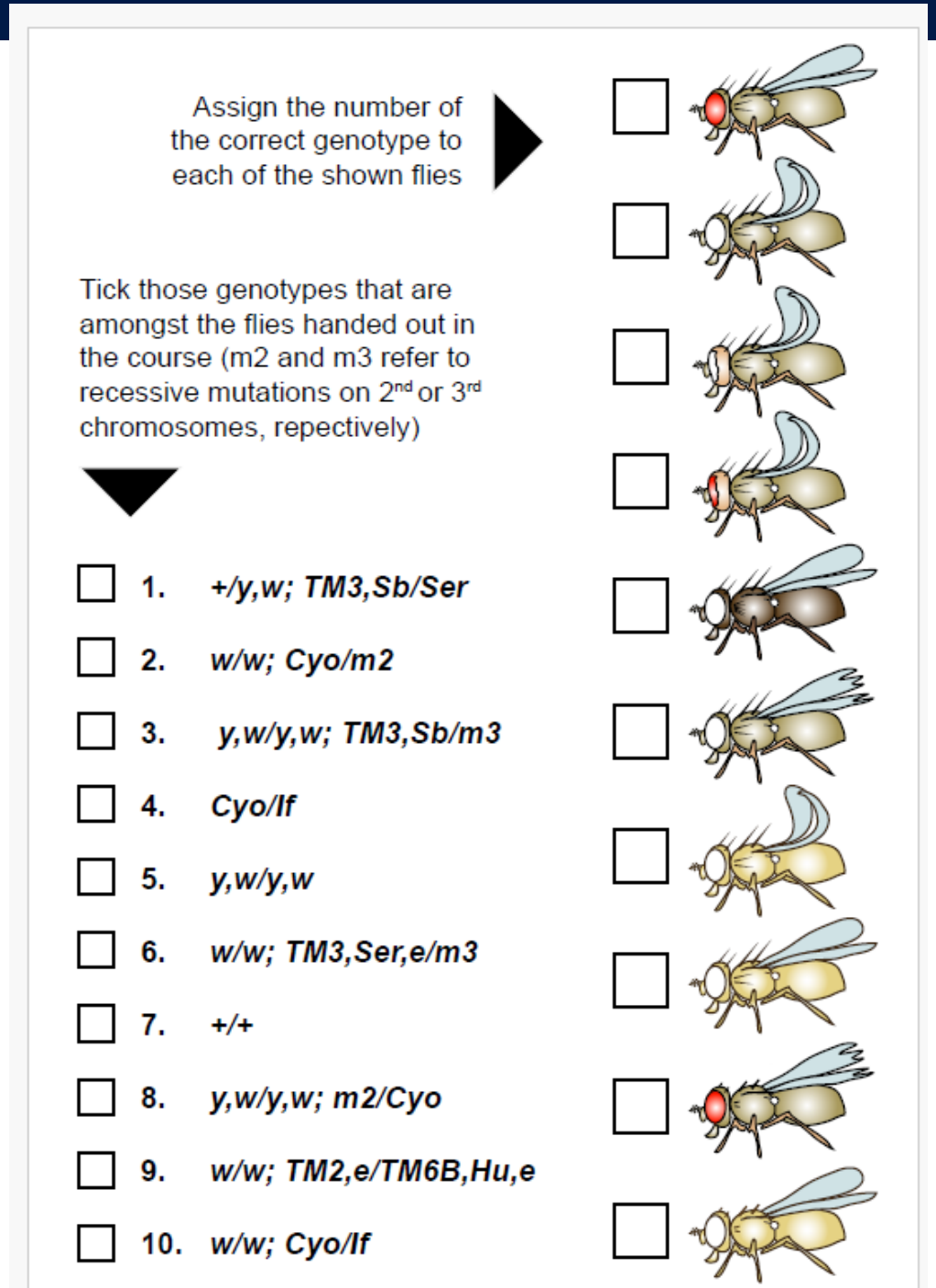
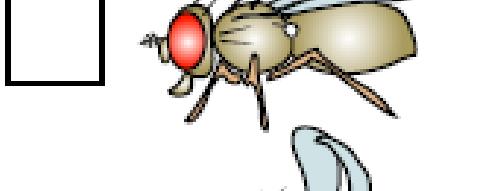
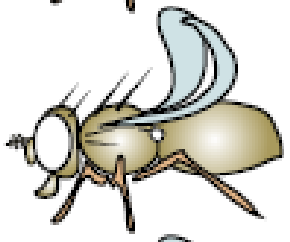
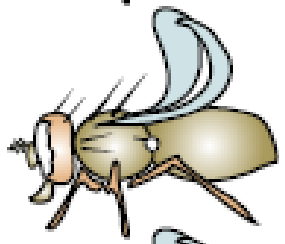
- From last week…
- Visible mutations, Dominant mutations and balancers.
- Read the genotype from the fly.

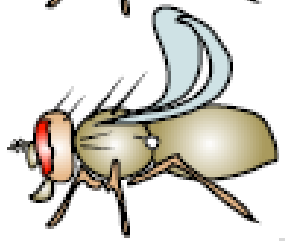


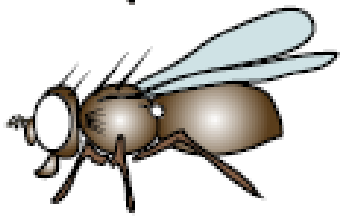
Assign the number of the correct genotype to each of the shown flies

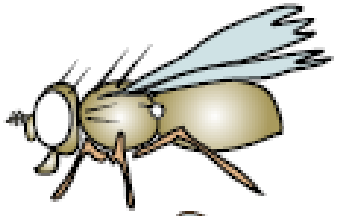












Tick those genotypes that are amongst the flies handed out in the course (m2 and m3 refer to recessive mutations on 2nd or 3rd chromosomes, repectively)

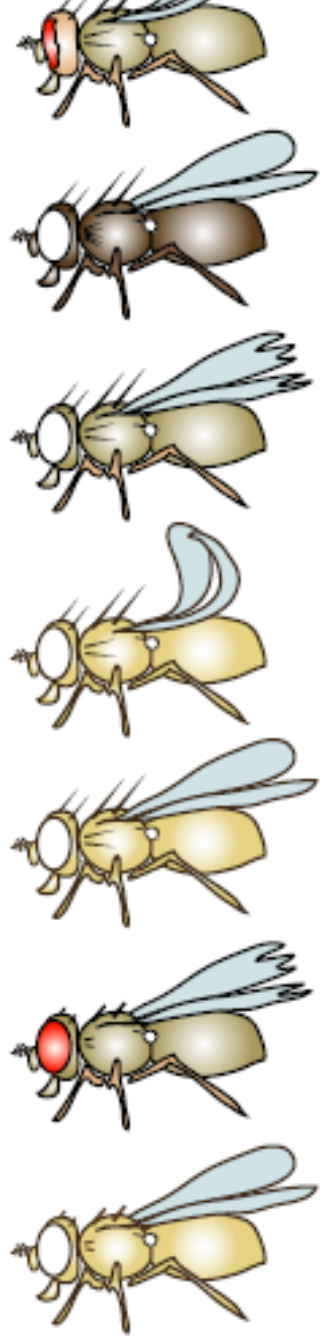
1. ***+/y,w; TM3,Sb/Ser***
2. ***w/w; Cyo/m2***
3. ***y,w/y,w; TM3,Sb/m3***

Assign the number of the correct genotype to each of the shown flies

Tick those genotypes that are amongst the flies handed out ir the course (m2 and m3 refer to recessive mutations on 2nd or 3 chromosomes, repectively)

- ☐ 1. *+/y,w; TM3,Sb/Ser*
- ☐ 2. *w/w; Cyo/m2*
- ☐ 3. *y,w/y,w; TM3,Sb/r*
- ☐ 4. *Cyo/If*
- ☐ 5. *y,w/y,w*
- ☐ 6. *w/w; TM3,Ser,e/m3*
- ☐ 7. *+/+*
- ☐ 8. *y,w/y,w; m2/Cyo*

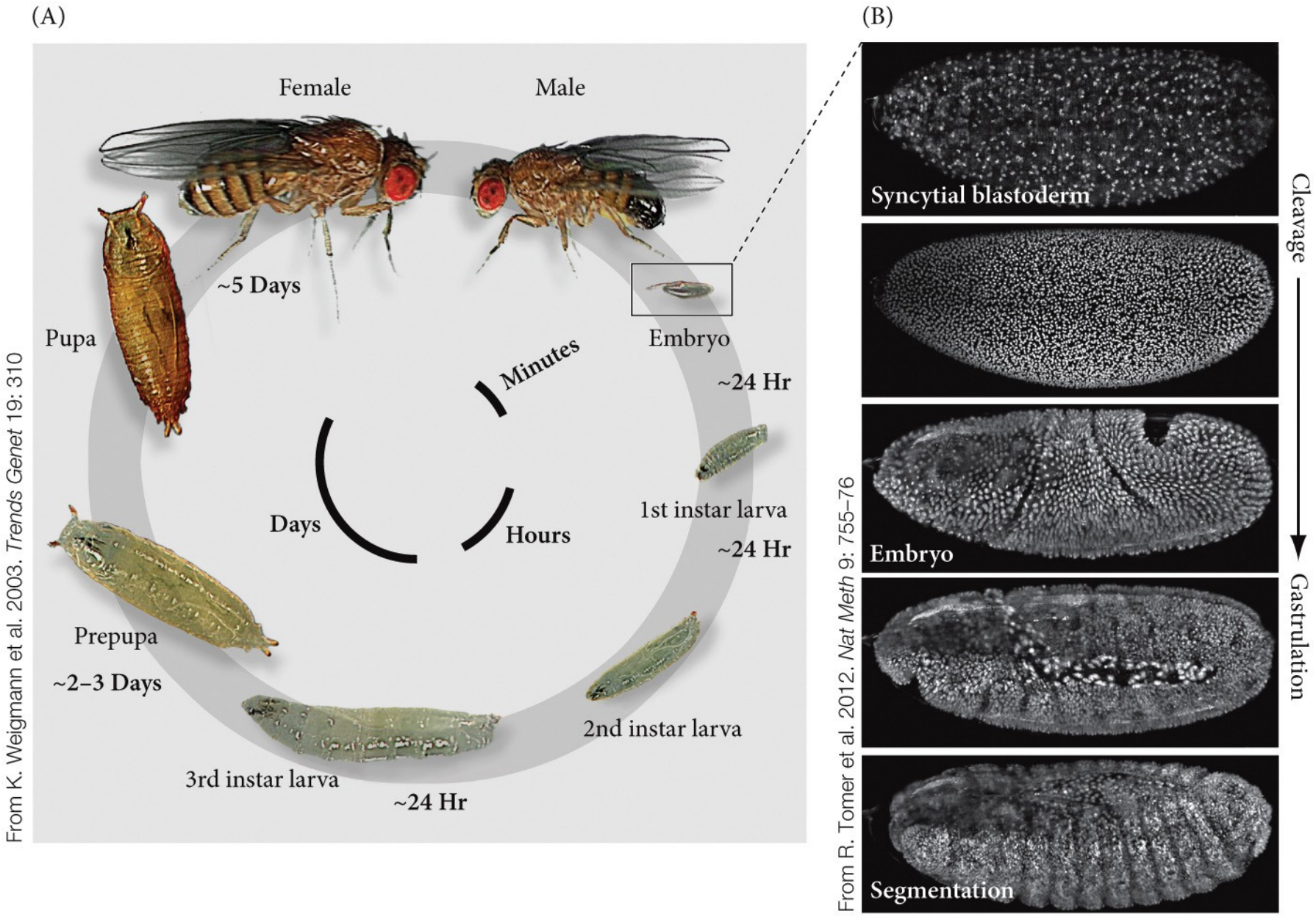
- ☐ 1. *+/y,w; TM3,Sb/Ser*
- ☐ 2. *w/w; Cyo/m2*
- ☐ 3. *y,w/y,w; TM3,Sb/m3*
- ☐ 4. *Cyo/If*
- ☐ 5. *y,w/y,w*
- ☐ 6. *w/w; TM3,Ser,e/m3*
- ☐ 7. *+/+*
- ☐ 8. *y,w/y,w; m2/Cyo*
- ☐ 9. *w/w; TM2,e/TM6B,Hu,e*
- ☐ 10. *w/w; Cyo/If*



- Lecture 2.

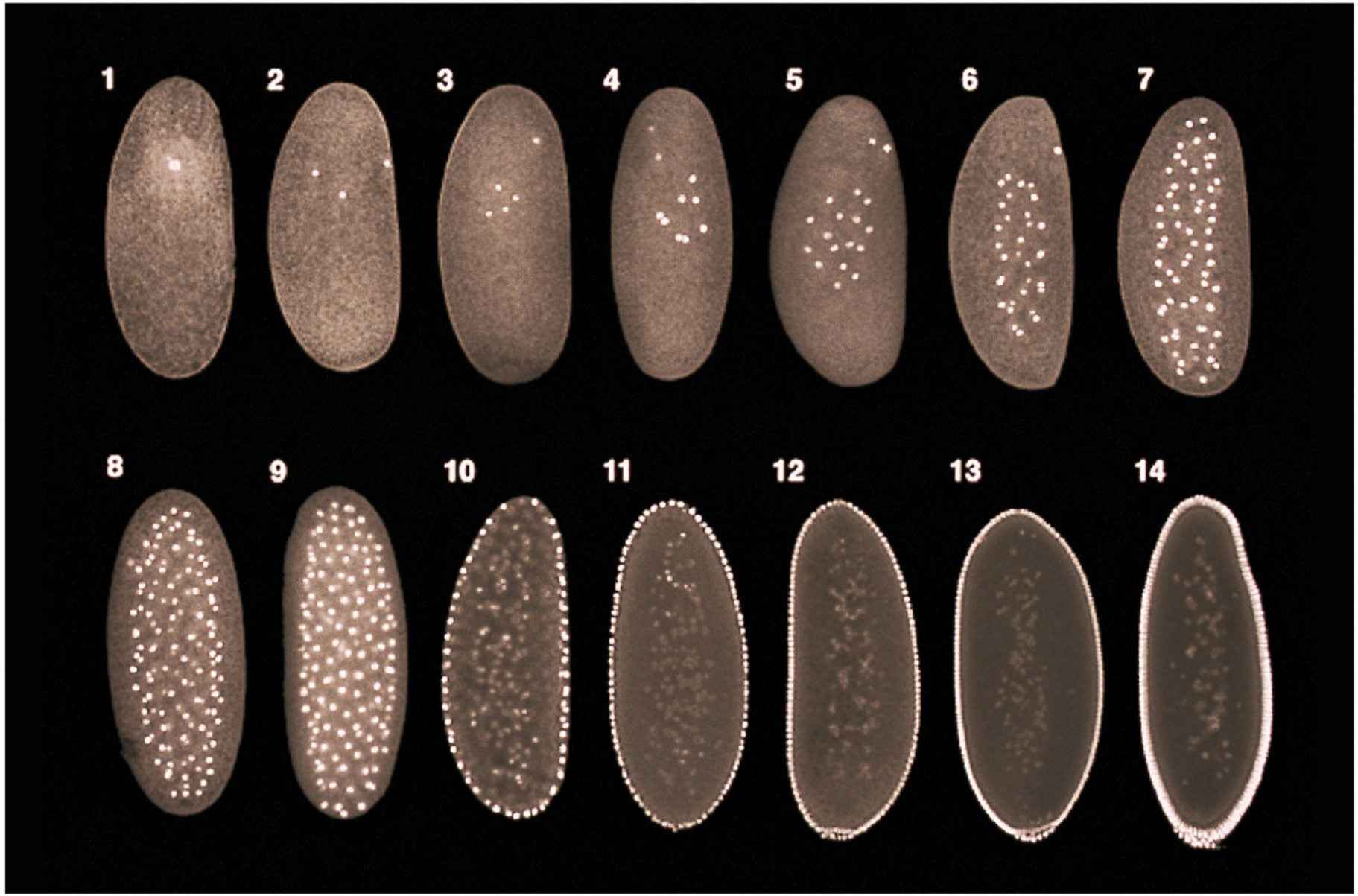
- Genetic screens for *Drosophila* mutants affecting embryonic pattern formation

Figure 10.3 Life cycle and early embryonic development of *Drosophila melanogaster*



DEVELOPMENTAL BIOLOGY 13e, Figure 10.3

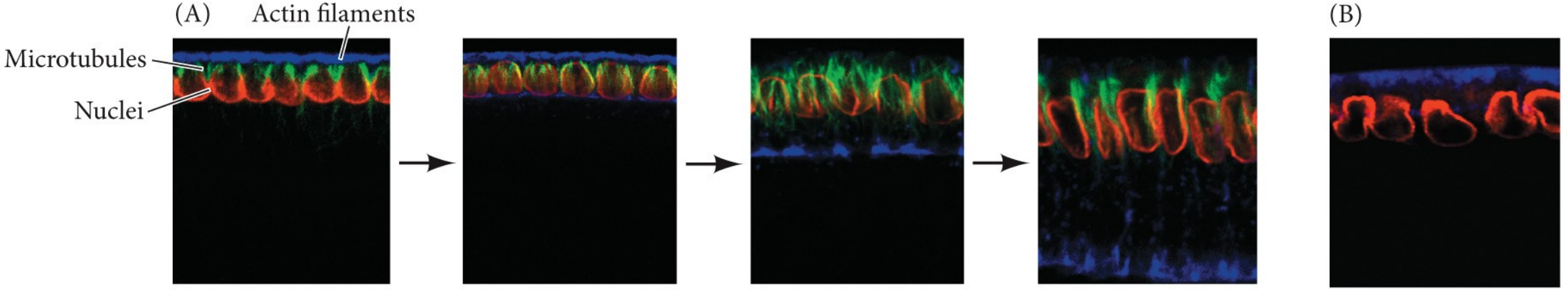
Figure 10.4 Laser confocal micrographs of stained chromatin showing syncytial nuclear divisions and superficial cleavage in a series of *Drosophila* embryos



Courtesy of D. Daily and W. Sullivan

DEVELOPMENTAL BIOLOGY 13e, Figure 10.4

Figure 10.6 Formation of the cellular blastoderm in *Drosophila*



All photographs from A. Brandt et al. 2006. *Curr Biol* 16: 543–552, courtesy of J. Grosshans and A. Brandt

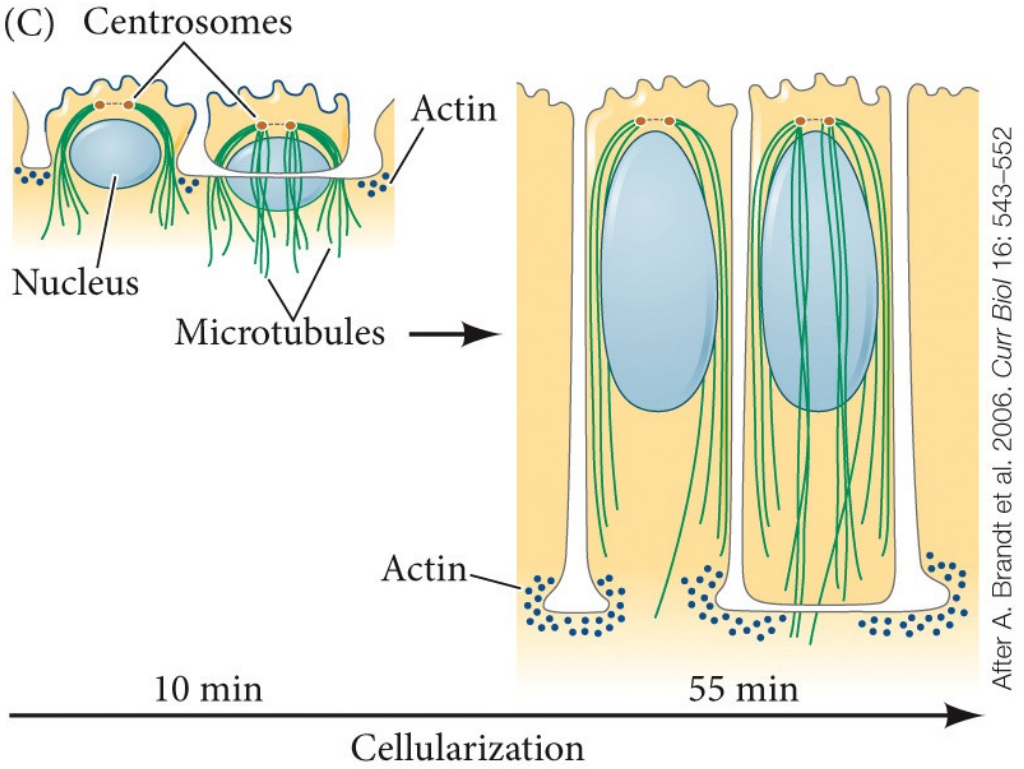
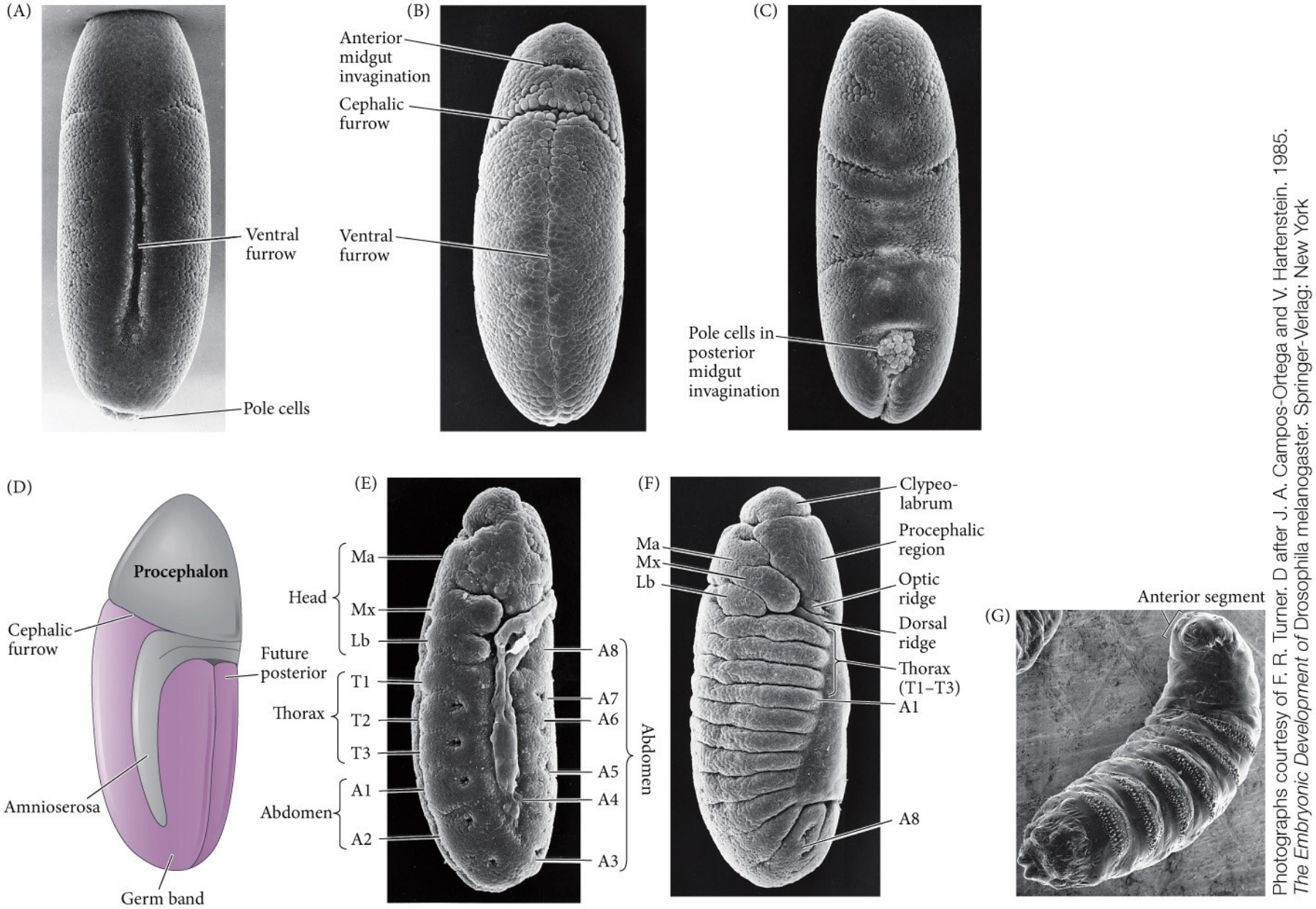


Figure 10.7 Gastrulation in *Drosophila*



Photographs courtesy of F. R. Turner. D after J. A. Campos-Ortega and V. Hartenstein. 1985. *The Embryonic Development of* Drosophila melanogaster. Springer-Verlag: New York

***DEVELOPMENTAL BIOLOGY 13e*, Figure 10.7**

The Heidelberg Screen for Pattern Mutants of *Drosophila*: A Personal Account

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Keywords

pattern formation, *Drosophila*, saturation screens, larval cuticle, embryonic lethal mutations

Abstract

In large-scale mutagenesis screens performed in 1979–1980 at the EMBL in Heidelberg, we isolated mutations affecting the pattern or structure of the larval cuticle in *Drosophila*. The 600 mutants we characterized could be assigned to 120 genes and represent the majority of such genes in the genome. These mutants subsequently provided a rich resource for understanding many fundamental developmental processes, such as the transcriptional hierarchies controlling segmentation, the establishment of cell states by signaling pathways, and the differentiation of epithelial cells. Most of the Heidelberg genes are now molecularly known, and many of them are conserved in other animals, including humans. Although the screens were initially driven entirely by curiosity, the mutants now serve as models for many human diseases. In this review, we describe the rationale of the screening procedures and provide a classification of the genes on the basis of their initial phenotypes and the subsequent molecular analyses.

Figure 18

Eric Wieschaus and Christiane Nüsslein-Volhard in 1979, at the time of the mutagenesis screen.

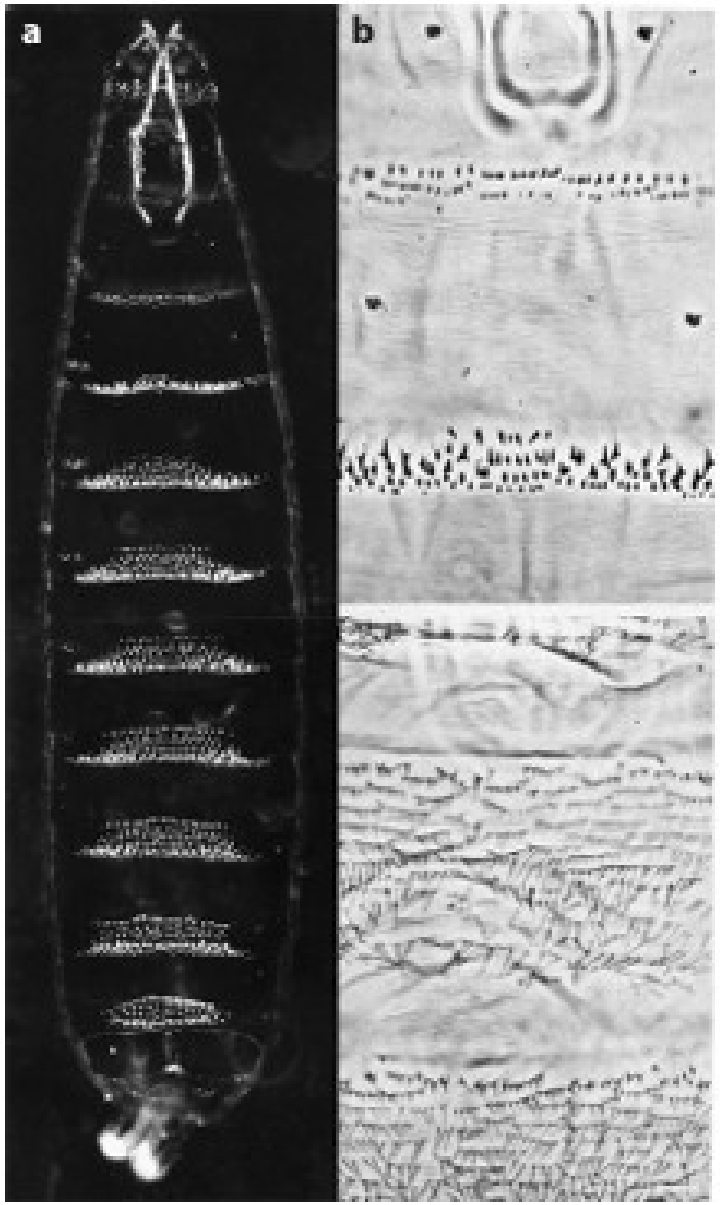


Figure 1

Cuticle preparation of a *Drosophila* first-instar larva. (*a*) Dark-field image of the ventral side. (*b*) Phase-contrast detail of the ventral (*top*) and the dorsal (*bottom*) aspect of the posterior thorax and the first abdominal segment.

(i.e., chromosomes), in contrast, appeared to be distributed equally among cells during cleavage. In a famous experiment, Boveri demonstrated that loss of individual chromosomes caused specific developmental defects in particular differentiation pathways (Boveri 1902; see Wilson 1925). These experiments showed that the abstract Mendelian factors, subsequently referred to as genes, were localized on individual chromosomes, and provided the foundation for the chromosomal theory of inheritance. The central ideas that emerge from Boveri's view of development are that spatial patterns are present as polar distributions of morphogenetic substances from the earliest stages, that these patterns are simple, and that the subsequent activity of genes on chromosomes builds the ultimate functional patterns in the final organism.

What these genes were and how they controlled development were unknown and beyond the reach of the technology available at the time. The strategies pursued by biological researchers after Boveri focused largely on either genetics or development. They aimed to find out more about genes and their organization on chromosomes (Morgan 1933) or to identify substances providing pattern

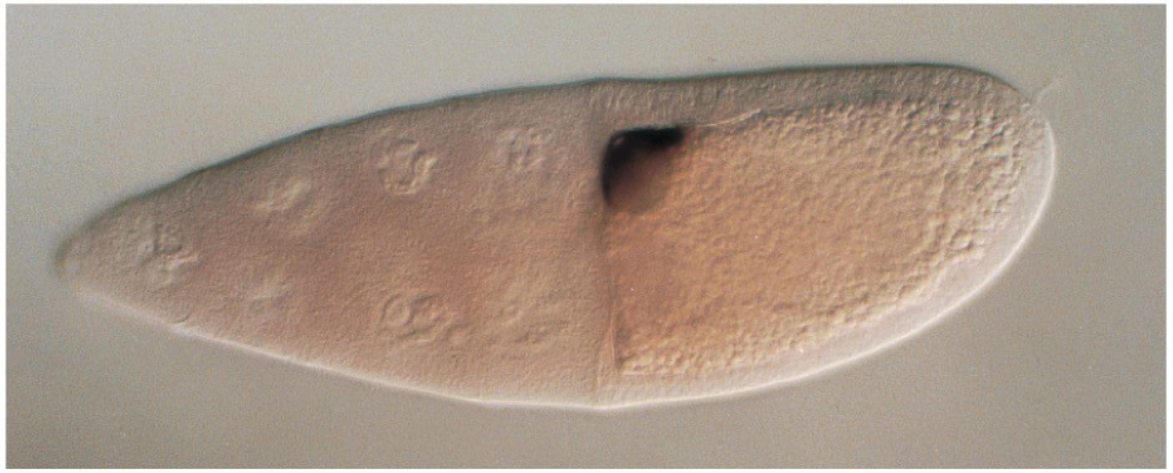
and polarity to the embryo. The discovery of the organizer region in the newt embryo by Boveri's student Spemann provided evidence for sequential induction of cell fate in the amphibian embryo (Spemann 1935). These observations generated great excitement but were followed by years of frustrating attempts to purify factors involved in particular developmental decisions. One major problem was the nature of assays for cell patterning available at the time. These assays generally involved the application of substances or embryonic extracts to fragments of tissue deprived of the intrinsic factor. Even when such experiments "worked," they often gave positive results with compounds that could not possibly have had a biological role. Development seemed infinitely complex and the experiments much too crude and unavoidably accompanied by unwanted side effects. Another problem in developmental biology during this period was that scientists worked on many different organisms, each chosen for a specific experimental advantage or an exciting phenomenon. A classic textbook (Kühn 1965) describes work on algae, frogs, newts, sea urchins, ascidians, daphnids, nematodes, planarians, slime molds, snails, chicken, hydra, crickets, moths, flies, midges, and grasshoppers. The individual communities were small, and the experimental approaches used were very different, such that the results were usually not comparable and did not easily lead to unifying theories of development. In the 1950s, the discovery of the double helix and the excitement of molecular biology of bacteria and bacteriophages pushed research into fields other than developmental biology and led Watson in a famous textbook (Watson 1965) to question whether "we have sufficient background at this time to attack embryology at a molecular level." Nevertheless, theoreticians, on the basis of regeneration experiments with the polyp hydra, proposed the concept of positional information (Wolpert 1969). A theory based on self-enhancement and lateral inhibition could explain the formation of stable patterns from near uniformity with plausible parameters (Gierer & Meinhardt 1972). These models assumed that complexity could arise from morphogen gradients eliciting different responses at different concentrations. But the molecular nature of such morphogens and the response to them remained elusive.

The beginning of the twentieth century also saw the rediscovery of Mendel's laws and the birth of modern genetics (Mendel 1866). Although not appreciated by most developmental biologists, genetic methods would ultimately provide a way to interfere specifically with a developing organism without causing gross disturbances. A mutation allows one to completely deplete a single component in a complex system while leaving everything else intact. Systematic mutant screens had been instrumental in identifying members of biochemical pathways in bacteria and fungi (Beadle & Tatum 1941). In bacteria, the identification of mutations in the *cis*- and *trans*-acting regulatory elements of genes played a crucial role in the biochemical isolation of transcriptional repressors (Jacob & Monod 1961). The identification of key players in the control of the cell cycle by systematic mutant screens in yeast provided another example of the powers of genetic analysis (Hartwell et al. 1970, Nurse 1975). The same strategy might also work for development. If mutations in genes controlling specific activities in the embryo could be found, it might even be possible to identify their protein products and thus gain insights into the biochemical mechanisms controlling developmental decisions.

Figure 10.27 Expression of Gurken between the oocyte nucleus and the dorsal anterior cell membrane

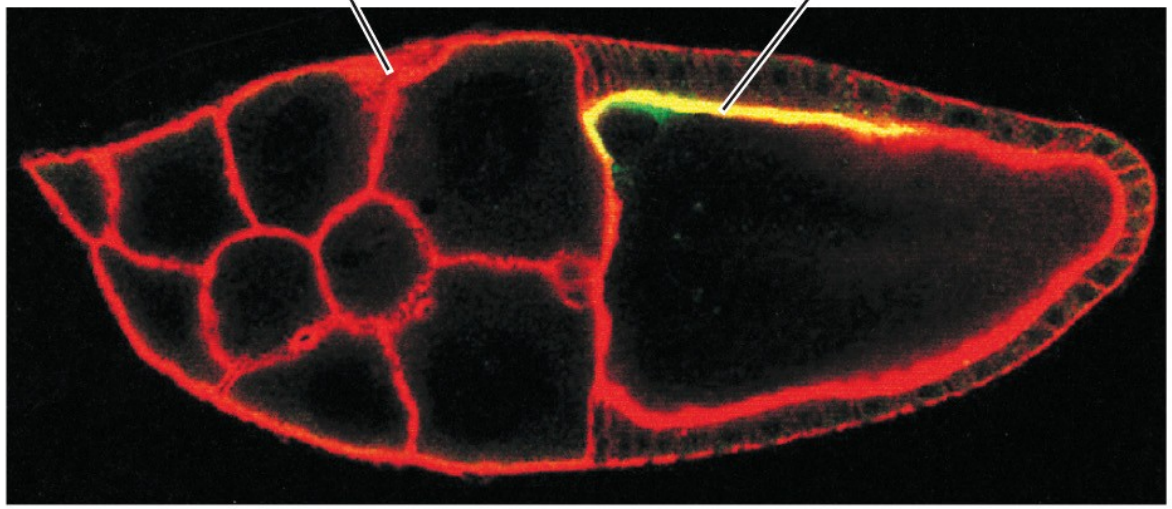
Nurse cells pour proteins and RNAs into the oocyte. Maternal contribution means many zygotic mutants will survive to hatching and die as larvae.

(A)



From R. P. Ray and T. Schüpbach. 1996. *Genes Dev* 10: 1711–1723, courtesy of T. Schüpbach

(B)



Courtesy of C. van Buskirk and T. Schüpbach

DEVELOPMENTAL BIOLOGY 13e, Figure 10.27

Maternal Mutants

Zygotic Mutants

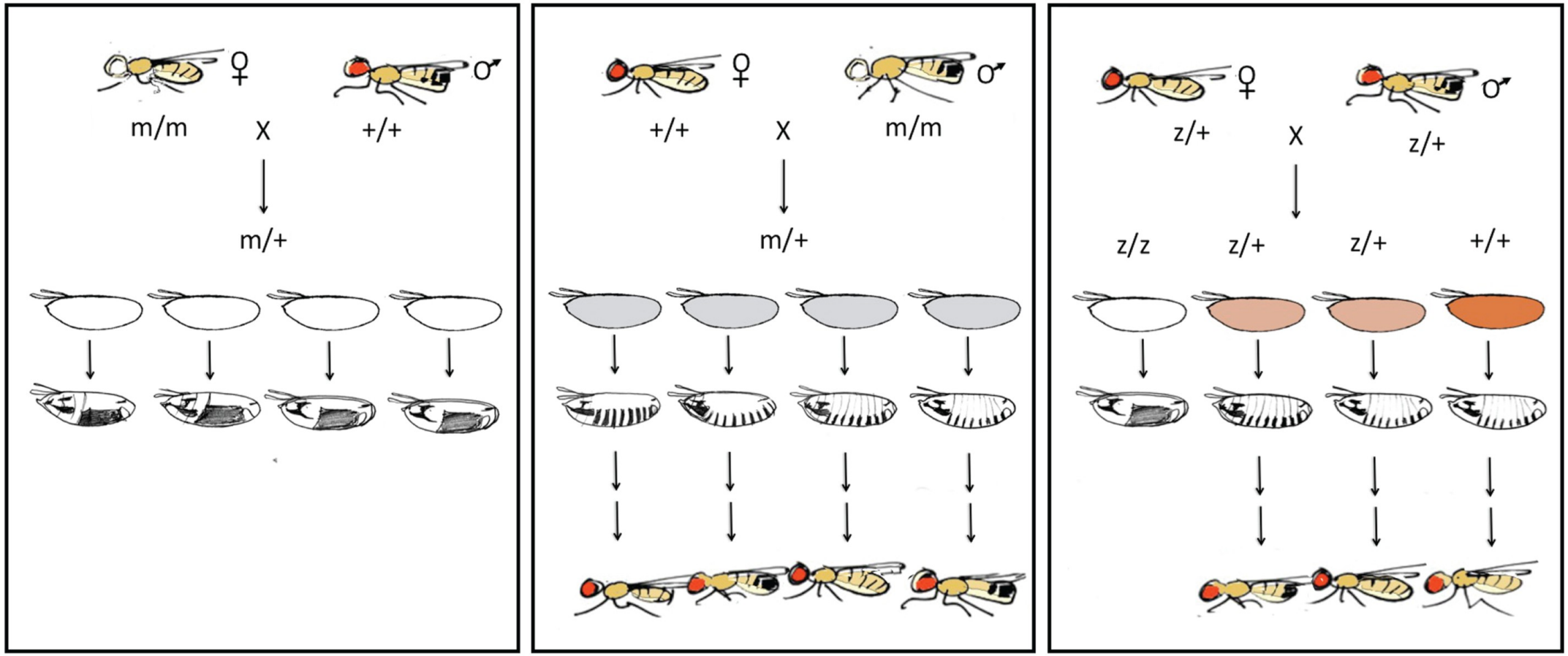


Figure 2

Genetics of embryonic patterning. Maternal and zygotic genes can be distinguished by their genetic behavior. (*Left panel*) All embryos from females that are homozygous mutant for maternally active genes are abnormal, even when crossed with wild-type males. (*Middle panel*) Although the genotype of the resultant embryos is the same as that in the reciprocal cross, in which wild-type females are crossed with mutant males, all embryos are normal. (*Right panel*) For zygotically active genes, only the homozygous one-quarter of the embryos derived from a cross between heterozygotes will be abnormal, even though all embryos

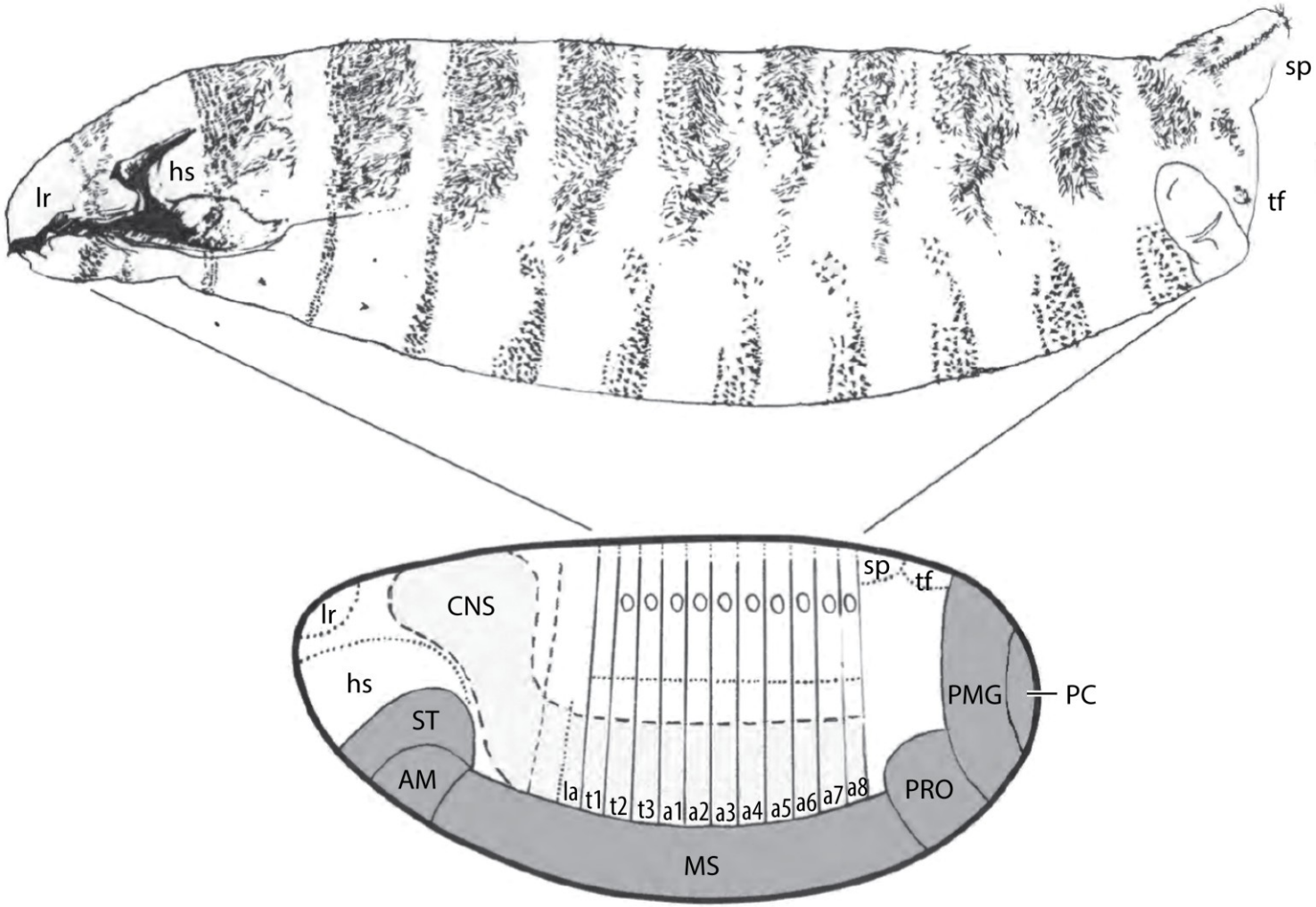
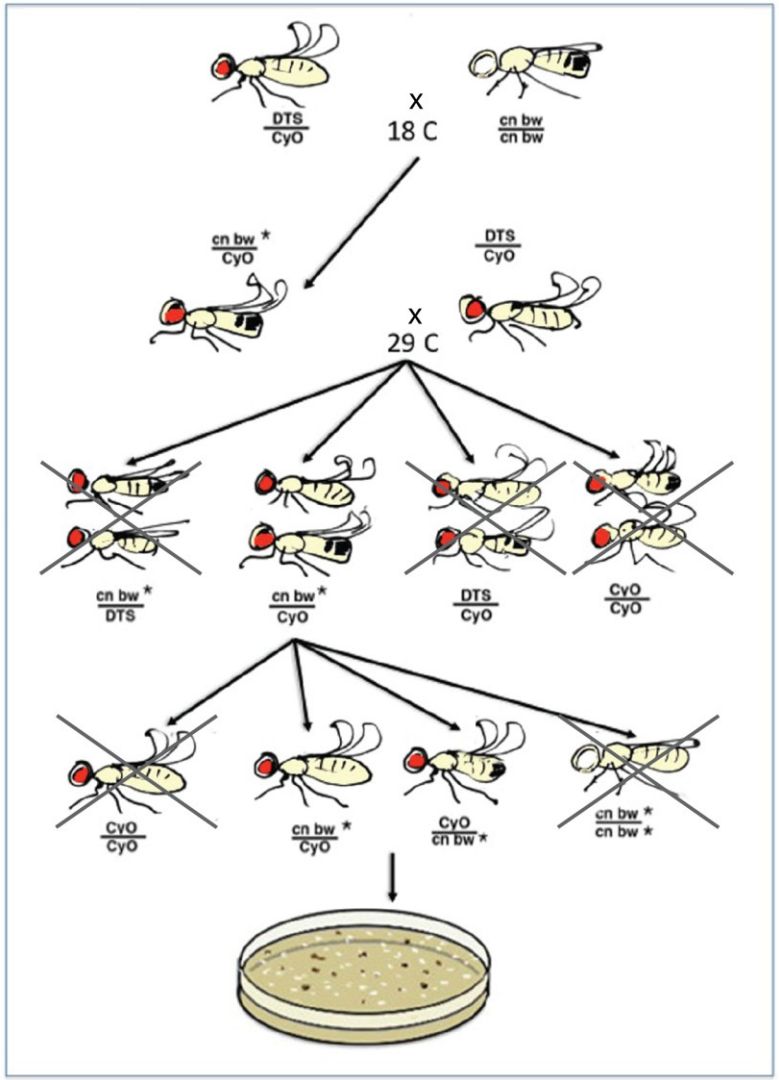
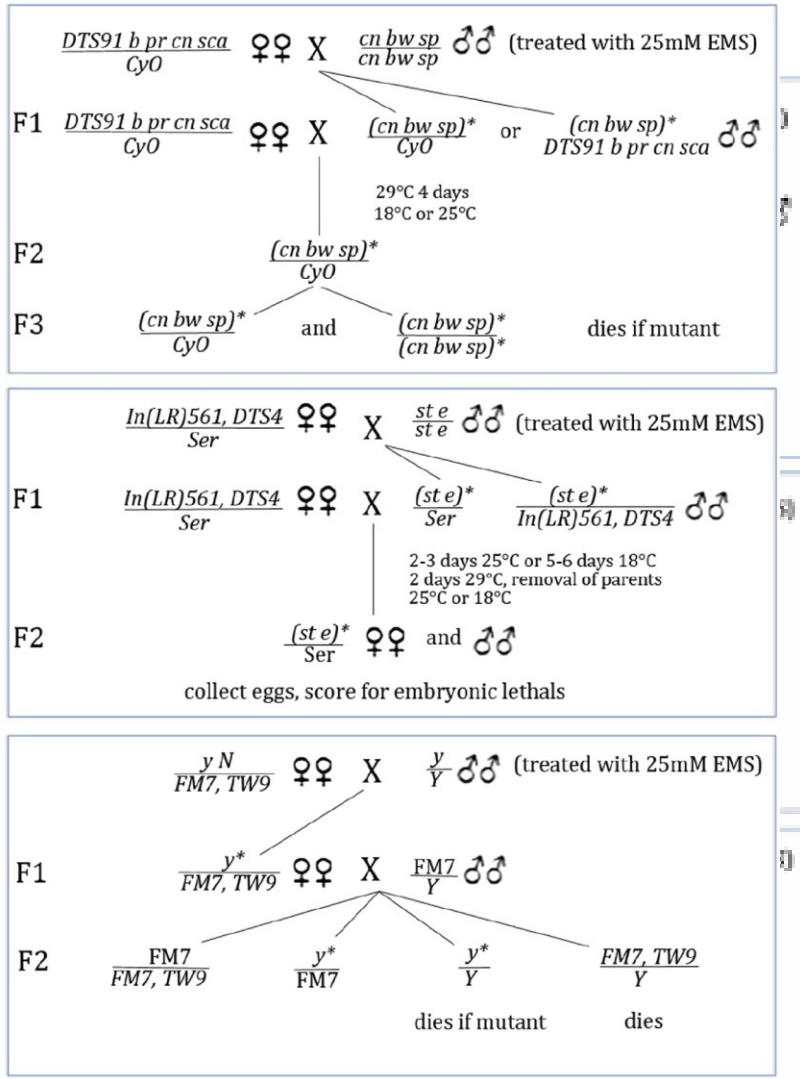


Figure 4

The relationship between the cuticle pattern of the hatching embryo and the fate map at the blastoderm stage. The primordium for the segmented epidermis represents a substantial fraction of the blastoderm and gives rise to the labial segment (la), three thoracic segments (t1 through t3), and eight abdominal segments (a1 through a8). Its pattern in cuticle preparations provides a simple assay for patterning in the earlier stages of development. Internalized structures such as the labrum (lr), head skeleton (hs), spiracles (sp), and tuft (tf) also provide useful markers for correct patterning. Mutants affecting regions of the blastoderm that give rise to soft internal tissues such as the stomodeum (ST), anterior midgut (AM), mesoderm (MS), proctodeum (PRO), posterior midgut (PMG), and pole cells (PC) can be scored only if those abnormalities have secondary consequences on the morphology of the epidermis.





Wieschaus E, Nüsslein-Volhard C. 2016.
Annu. Rev. Cell Dev. Biol. 32:1–46

Figure 5

Crossing schemes to produce inbred lines to be tested for homozygous mutant embryos with altered patterns. The left panel provides a general schematic of the crosses, and the right panels give genetic details of the second chromosome (*top*), third chromosome (*middle*), and X chromosome (*bottom*) crosses. EMS denotes ethyl methane sulfonate.

Figure 3

Replica plating egg collections from multiple mutant stocks. Flies from different mutagenized lines are transferred to tubes glued together in a block formation. Females lay eggs in defined position on yeasted apple juice agar plates. After 24 h, the normal embryos have hatched, and the unhatched mutant embryos can be collected for microscopic examination.

Genes on the same chromosome will recombine

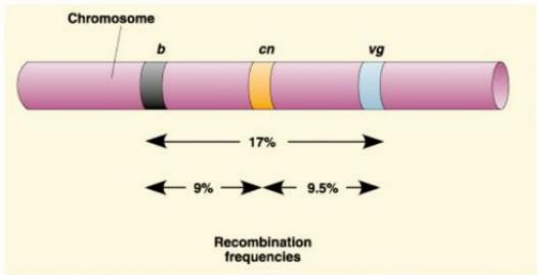
New pattern-forming mutants were mapped by recombination with visible markers

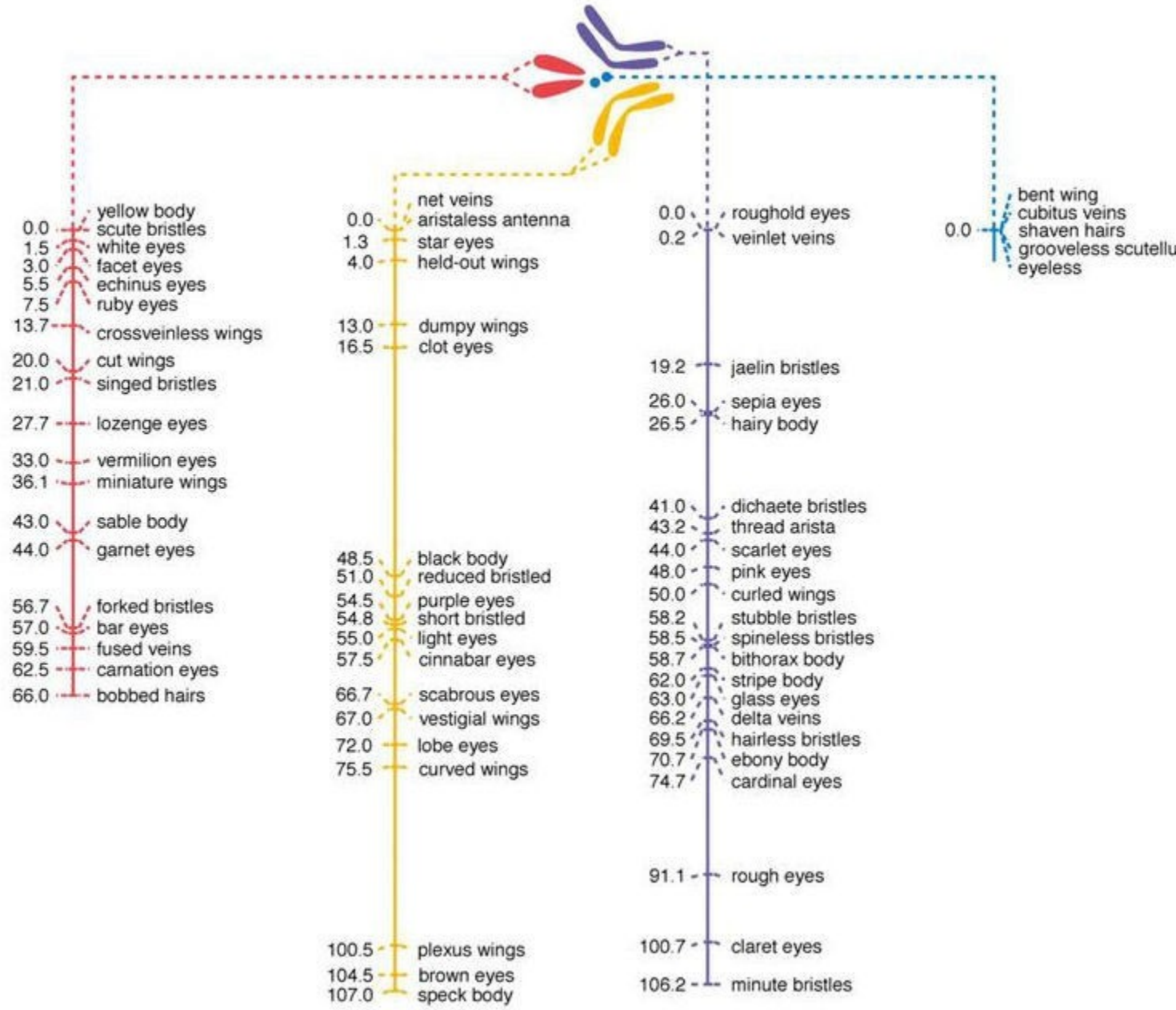
Sturtevant's linkage map of 3 genes in *Drosophila*

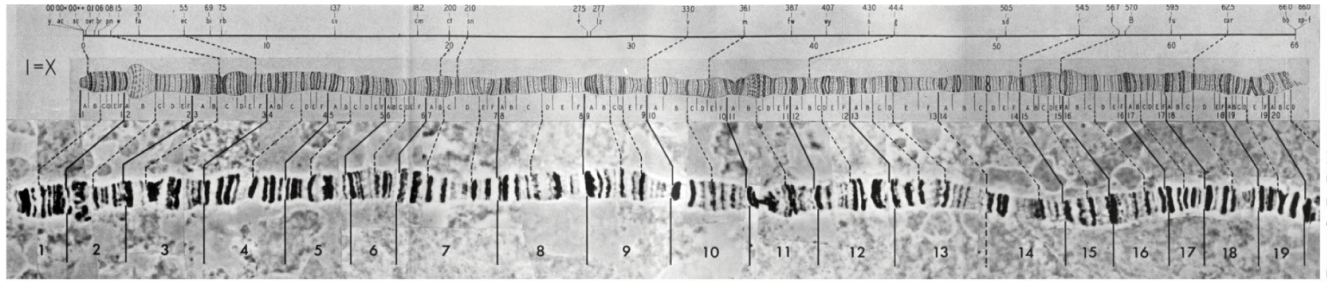
- 3 genes: body-color (b), wing-size (vg), and cinnebar (cn) –one of many genes affecting eye color
- Observed recombination frequencies:
  - cn and b = 9%
  - cn and vg = 9.5%
  - b and vg = 17%

*Crossing over would occur most frequently between genes b and vg

- He decided to "map" these out on a chromosome
  - 1 map unit is = to 1% recombination frequency

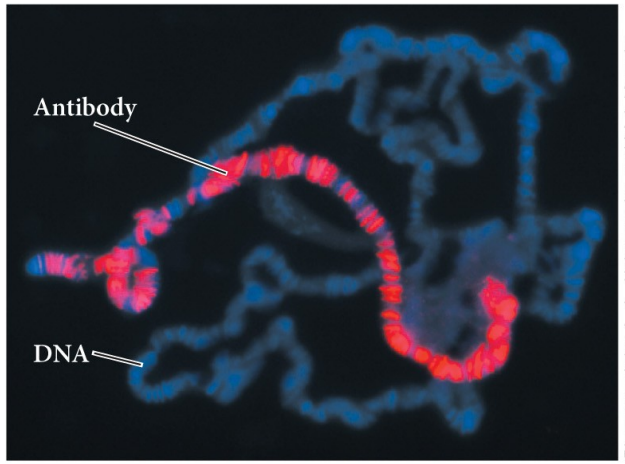






From C. B. Bridges. 1938. *J Hered* 29: 11–13

(B)



Photograph by A. A. Alekseyenko and M. I. Kuroda

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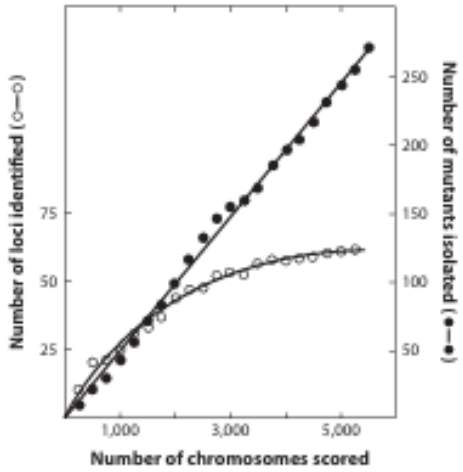


Figure 7

Saturation curve. The plot shows the number of pattern mutants isolated over the numbers of lines (chromosomes) scored (*closed circles*) as well as the numbers of new loci identified (*open circles*). From Nüsslein-Volhard et al. (1984).

Expected 5000 lethal genes in flies, 1000/chromome arm and 1,000 on Chr 2 ; similar to number of bands in interphase polytene chromosomes. In fact there are about 13,700 genes but only 5,000 mutate to give lethals.

Most lethals survive embryogenesis and die as larvae. 61 mutant genes affecting cuticle pattern were found on Chr 2.

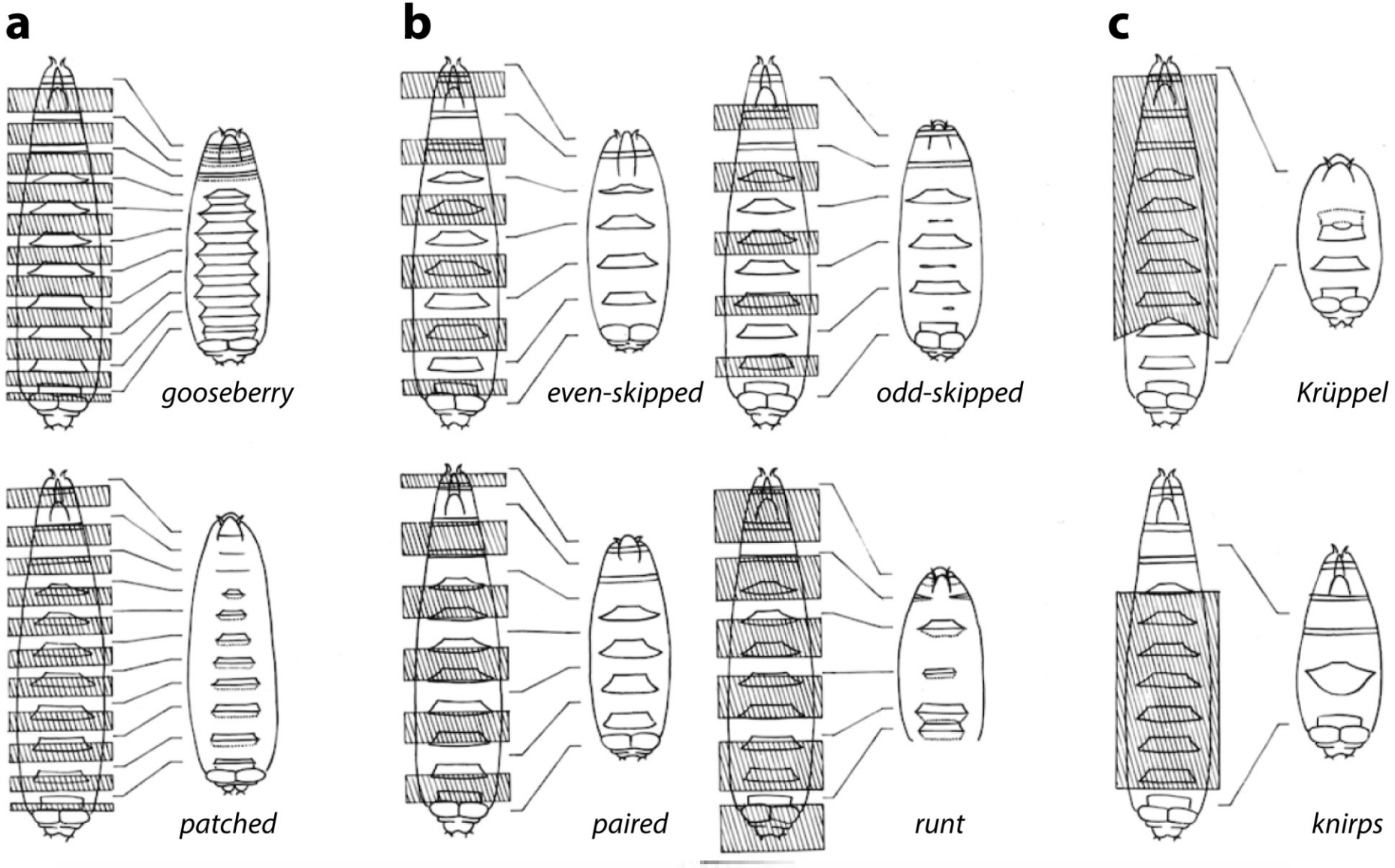


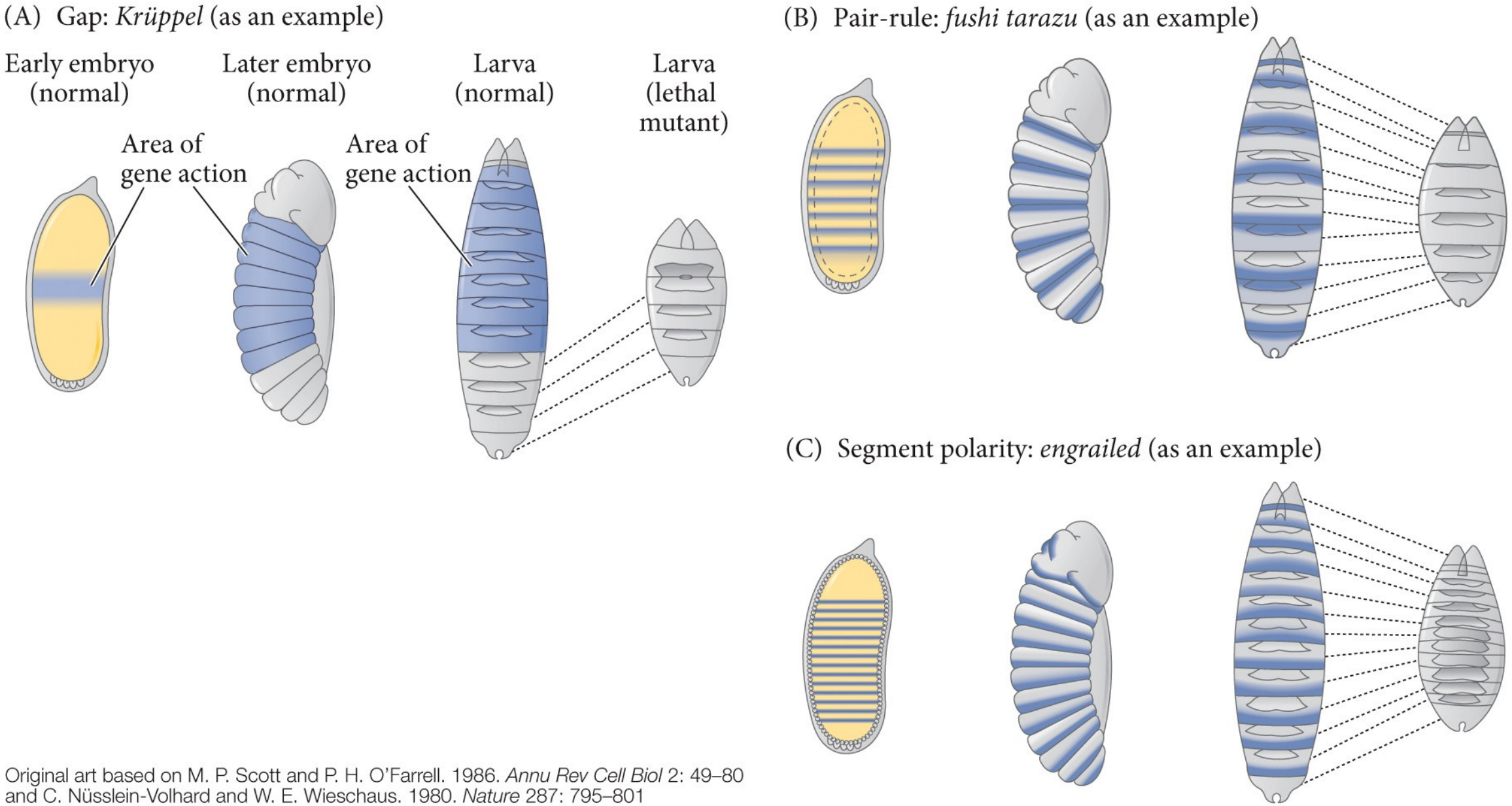
Figure 10

Pattern deletions in an embryo homozygous mutant for genes (*a*) in the segment polarity class (*gooseberry* and *patched*), (*b*) in the pair rule class (*even-skipped*, *odd-skipped*, *paired*, and *runt*), and (*c*) in the gap gene class (*Krüppel* and *knirps*). Modified from Nusslein-Volhard & Wieschaus (1980).

Figure 6

The altered segmentation patterns of embryos homozygous for *paired* and for *knirps*, show wild-type pattern in the cover illustration of the *Nature* paper describing the first mutants mutagenesis screen (Nusslein-Volhard & Wieschaus 1980).

Figure 10.16 Three types of segmentation gene mutations



Original art based on M. P. Scott and P. H. O'Farrell. 1986. *Annu Rev Cell Biol* 2: 49–80 and C. Nüsslein-Volhard and W. E. Wieschaus. 1980. *Nature* 287: 795–801

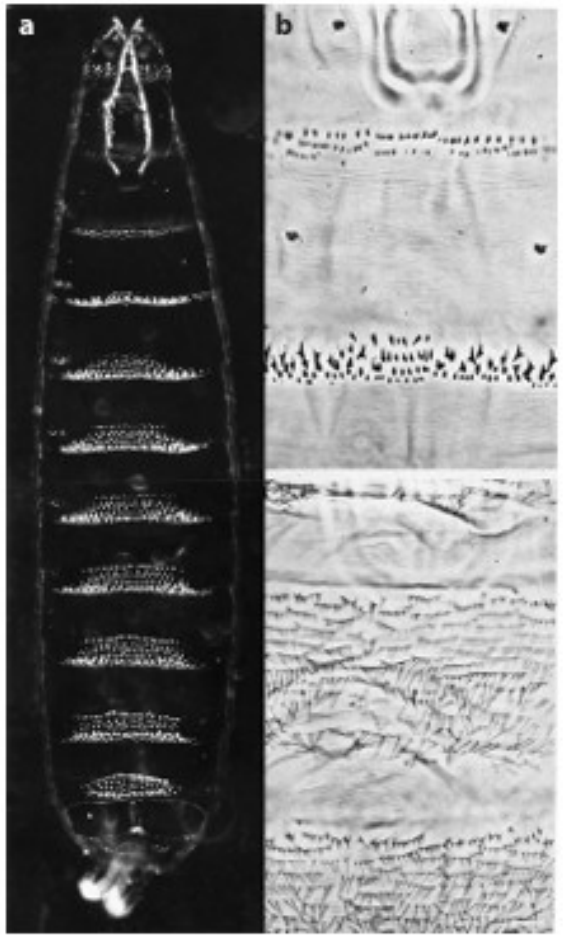


Figure 1

Cuticle preparation of a *Drosophila* first-instar larva. (*a*) Dark-field image of the ventral side. (*b*) Phase-contrast detail of the ventral (*top*) and the dorsal (*bottom*) aspect of the posterior thorax and the first abdominal segment.

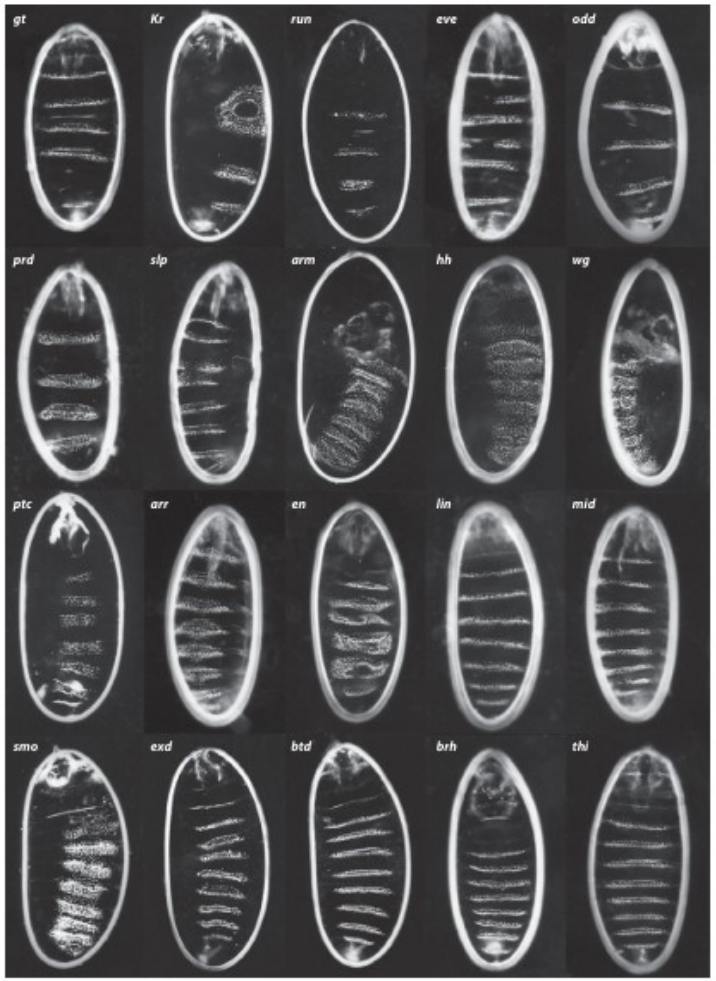


Figure 8

Mutants affected in AP patterning: 20 mutants of genes listed in **Table 1** represent the following classes: gap genes [*giant* (*gt*) and *Krüppel* (*Kr*)], pair rule genes [*runt* (*run*), *even-skipped* (*eve*), *odd-skipped* (*odd*), *paired* (*prd*), and *sloppy-paired* (*slp*)], segment polarity genes [*armadillo* (*arm*), *hedgehog* (*hh*), *wingless* (*wg*), and *patched* (*ptc*)], segment pattern genes [*arrow* (*arr*), *engrailed* (*en*), *lines* (*lin*), *midline* (*mid*), and *smoothened* (*smo*)], homeotic genes [*extradenticle* (*exd*)], and head genes [*buttonhead* (*btd*), *brown head* (*brh*), and *thick head* (*thi*)]. From Jürgens et al. (1984), Nüsslein-Volhard et al. (1984), and Wieschaus et al. (1984a).

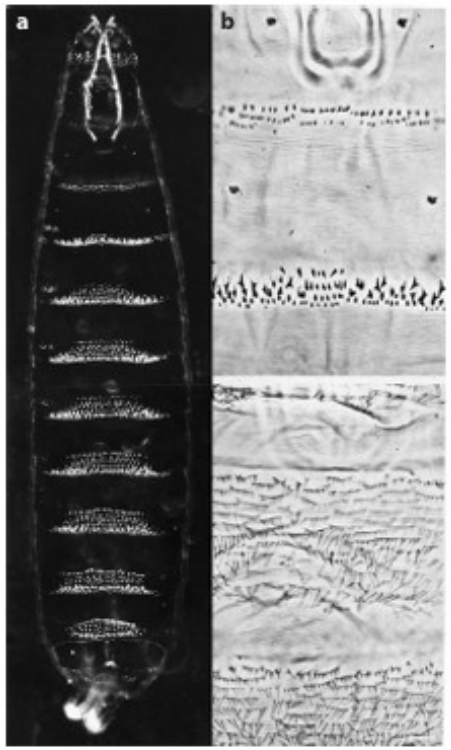


Figure 1

Cuticle preparation of a *Drosophila* first-instar larva. (*a*) Dark-field image of the ventral side. (*b*) Phase-contrast detail of the ventral (*top*) and the dorsal (*bottom*) aspect of the posterior thorax and the first abdominal segment.

Figure 9

Gap and pair rule mutants: gap mutants *Kruppel* (*Kr*) (strong phenotype), *hunchback* (*hb*) (weak phenotype), *odd-skipped* (*odd*) (strong phenotype), and *even-skipped* (*eve*) [weak (eve^{ID}) and strong (eve^{KR13}) phenotypes]. The ventral aspects of mutant larvae were dissected out of the vitelline membrane.

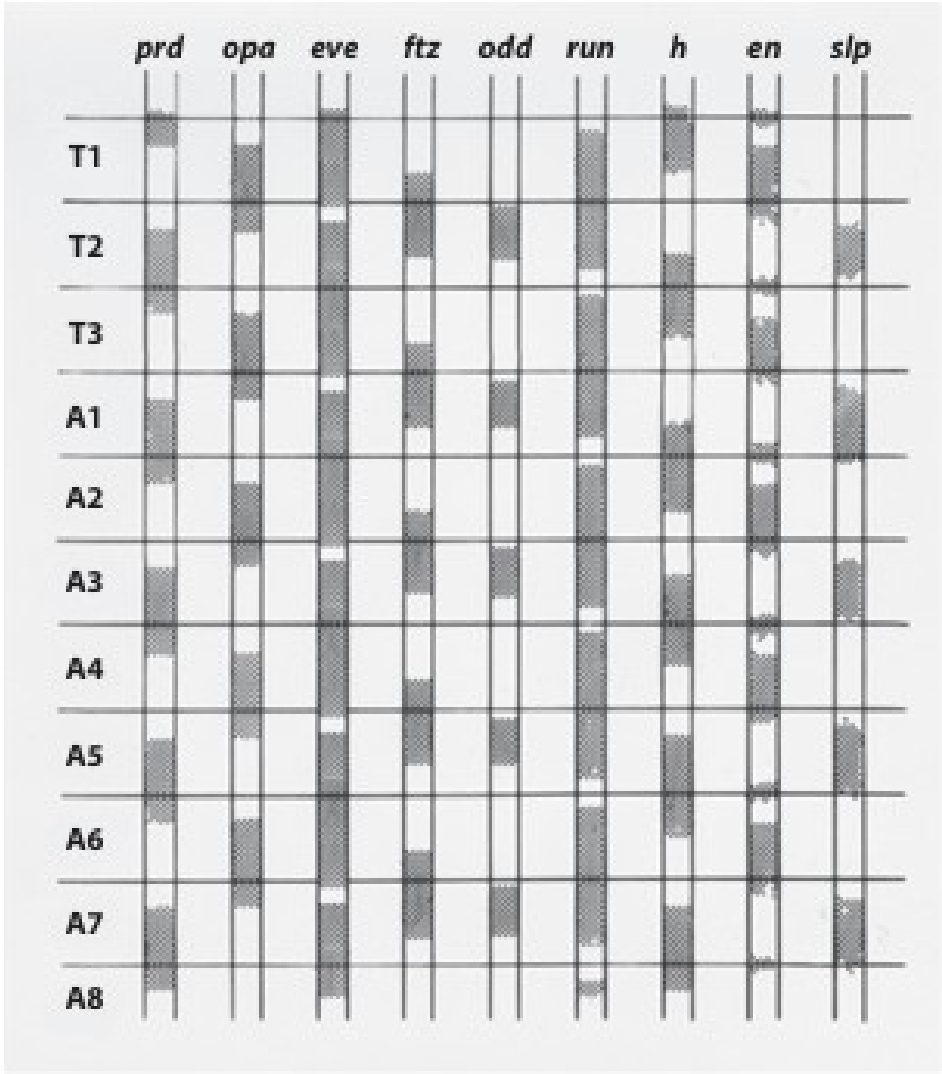


Figure 11

Schematic representation of deletion patterns in pair rule mutants. The shaded areas indicate the regions lost in the mutant patterns of strong alleles. Gene abbreviations (from *top left* to *top right*): *prd*, *paired*; *opa*, *odd-paired*; *eve*, *even-skipped*; *ftz*, *fushi tarazu*; *odd*, *odd-skipped*; *run*, *runt*; *h*, *hairy*; *en*, *engrailed*; *slp*, *sloppy-paired*.

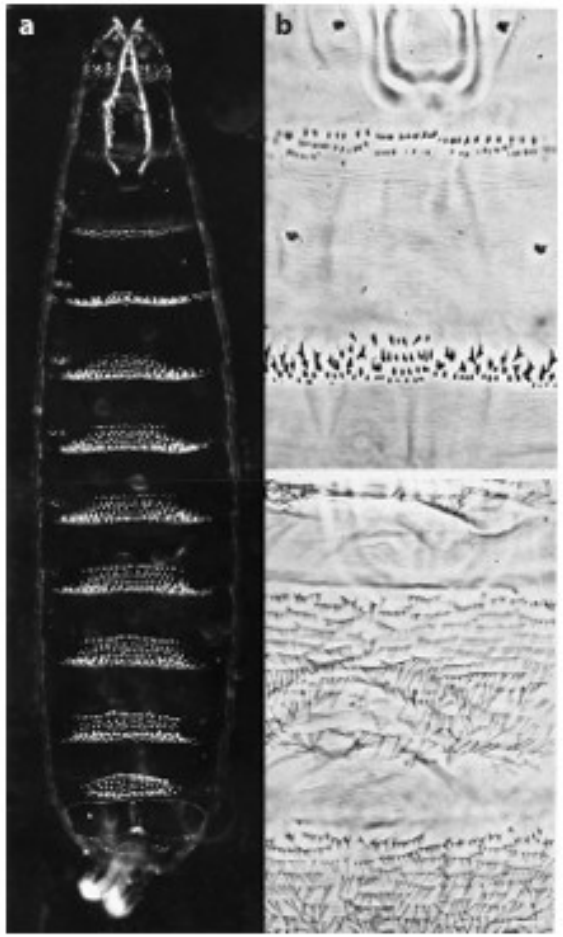


Figure 1

Cuticle preparation of a *Drosophila* first-instar larva. (*a*) Dark-field image of the ventral side. (*b*) Phase-contrast detail of the ventral (*top*) and the dorsal (*bottom*) aspect of the posterior thorax and the first abdominal segment.

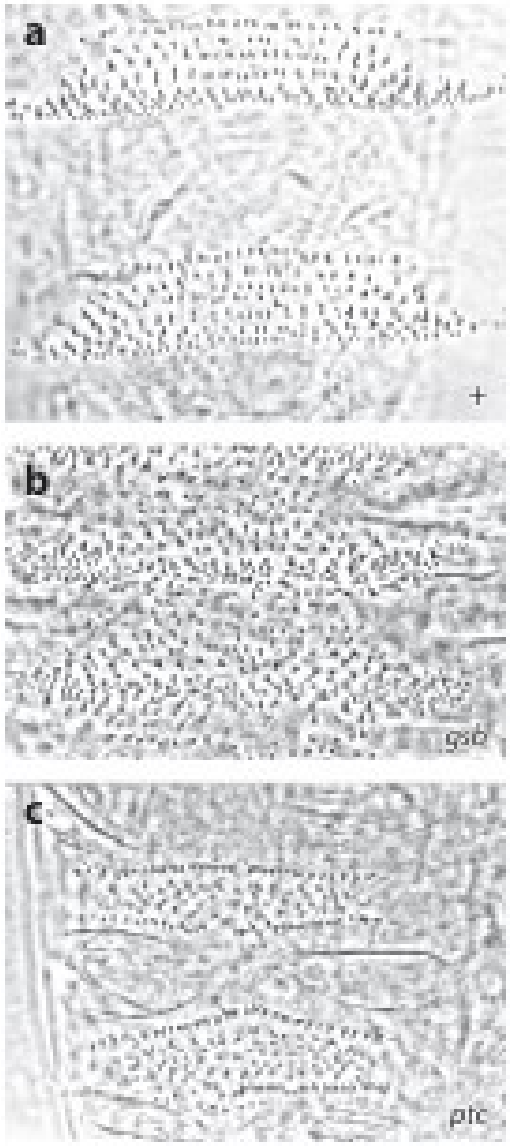


Figure 12

Segment polarity mutants: details from the ventral anterior abdomen of (*a*) wild-type (+), (*b*) *gooseberry* (*gsb*), and (*c*) *patched* (*ptc*) larvae (phase contrast).

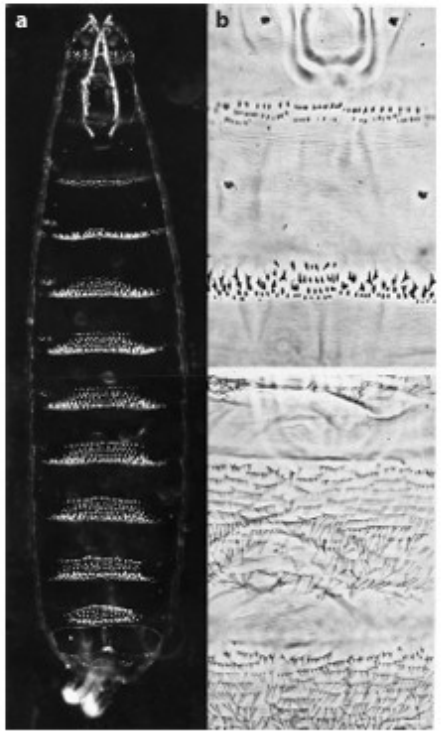


Figure 1

Cuticle preparation of a *Drosophila* first-instar larva. (*a*) Dark-field image of the ventral side. (*b*) Phase-contrast detail of the ventral (*top*) and the dorsal (*bottom*) aspect of the posterior thorax and the first abdominal segment.

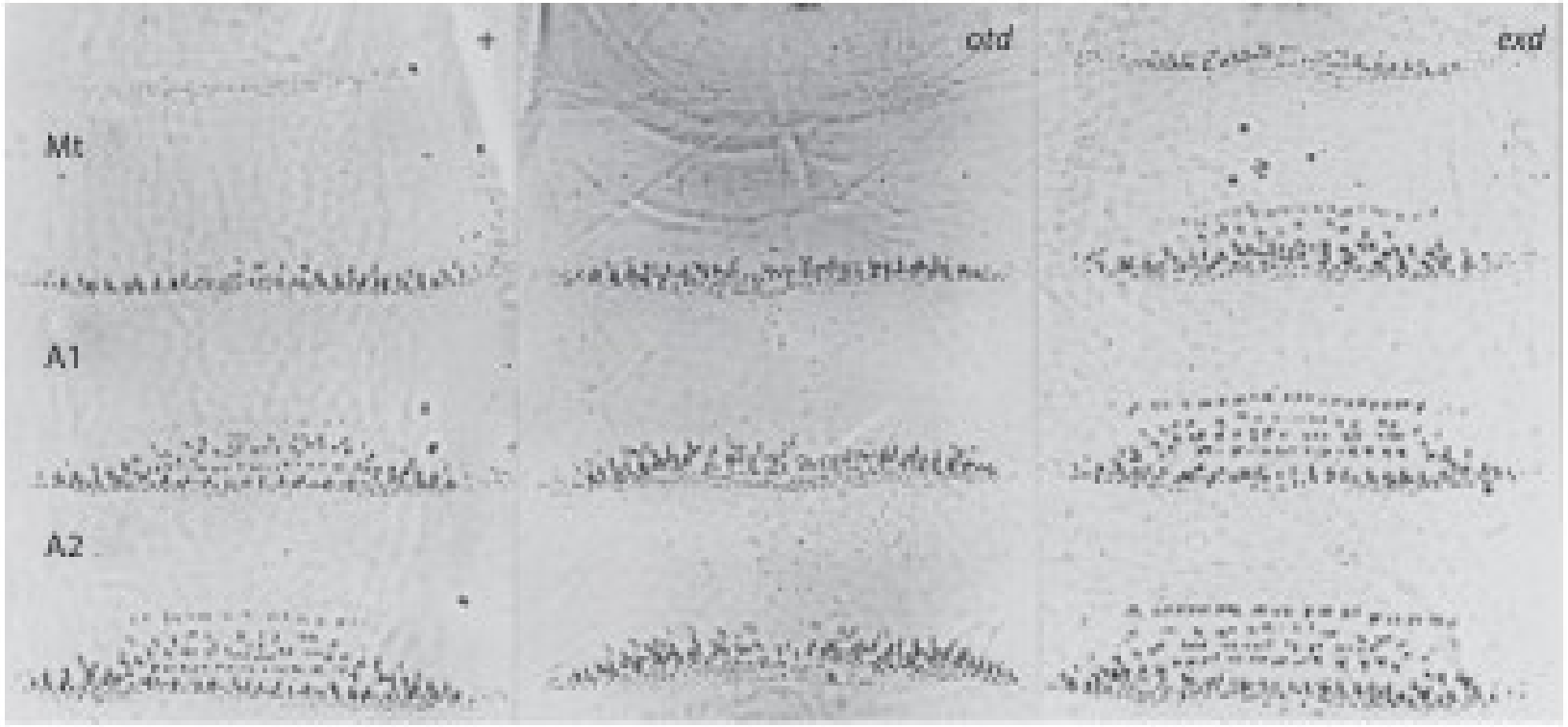
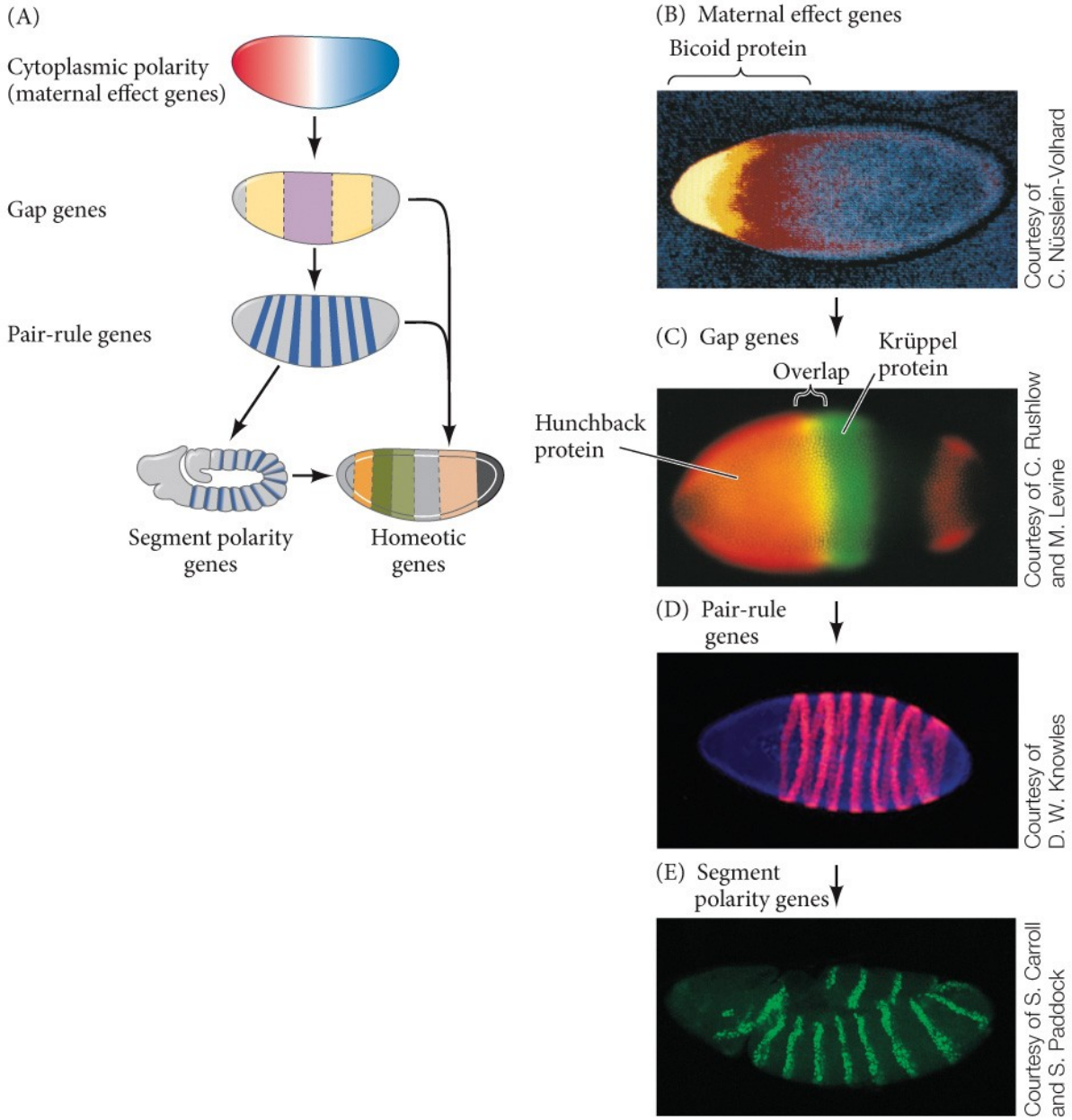


Figure 13

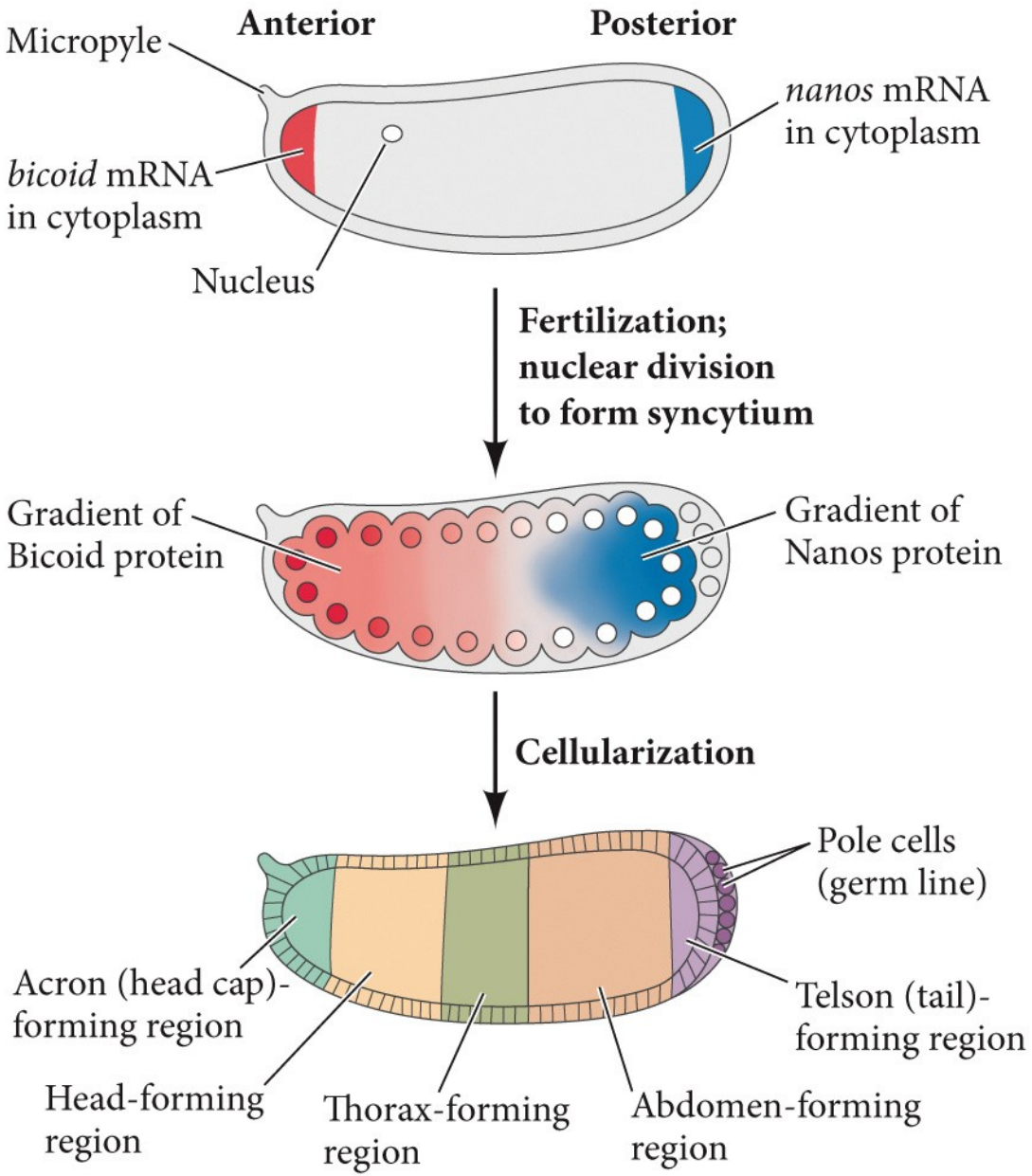
Segment pattern and homeotic mutants: posterior thorax and anterior abdomen of *orthodenticle* (*otd*) (abdominal denticle belts reduced) and *extradenticle* (*exd*) (posterior transformations). Other abbreviations: A, abdominal segment; Mt, metathorax. From Wieschaus et al. (1984a).

Figure 10.10 Generalized model of *Drosophila* anterior-posterior pattern formation



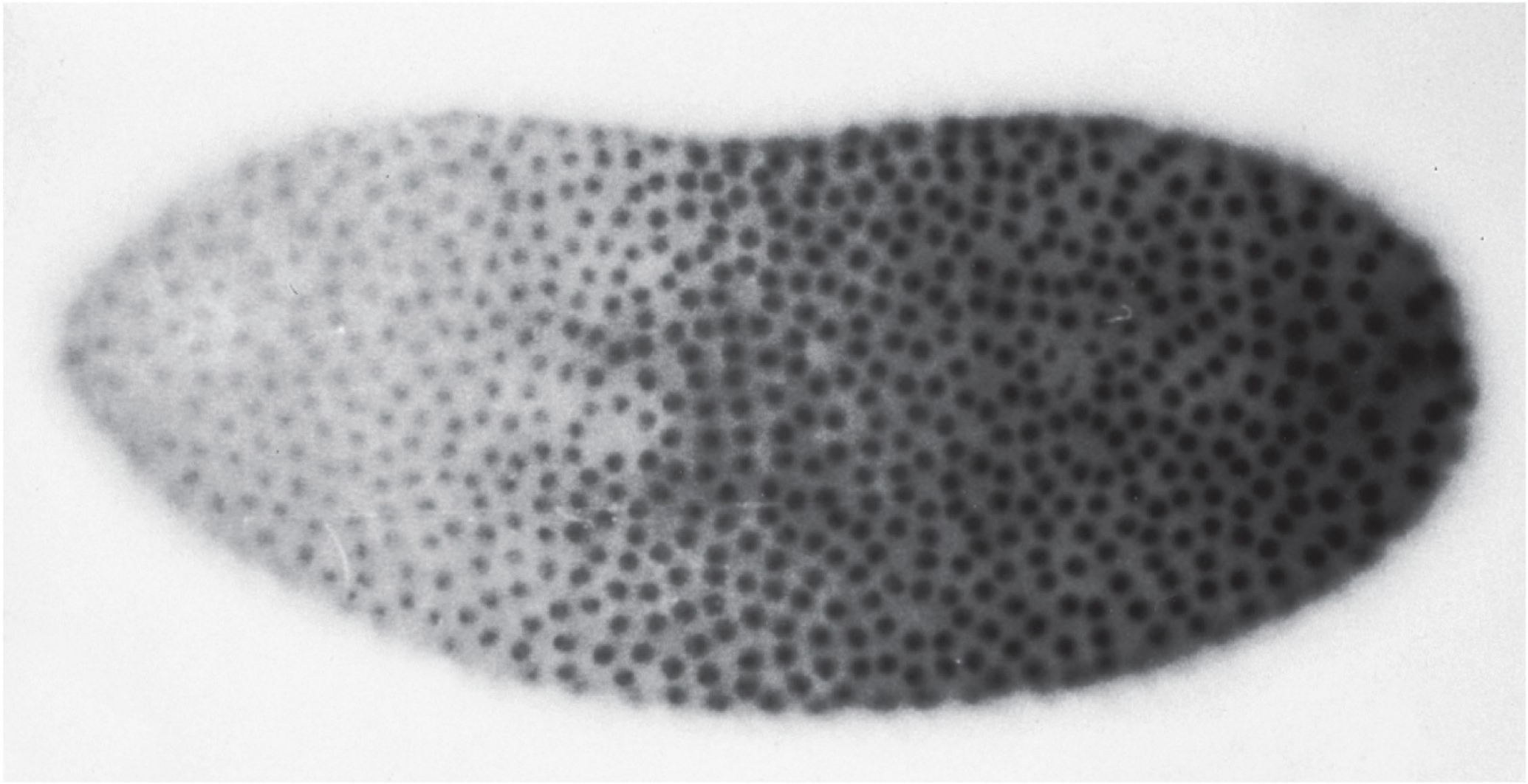
DEVELOPMENTAL BIOLOGY 13e, Figure 10.10

Figure **10.11** Anterior-posterior specification in *Drosophila* originates with morphogen gradients



DEVELOPMENTAL BIOLOGY 13e, Figure 10.11

Figure 10.13 Caudal protein gradient of a wild-type *Drosophila* embryo at the syncytial blastoderm stage



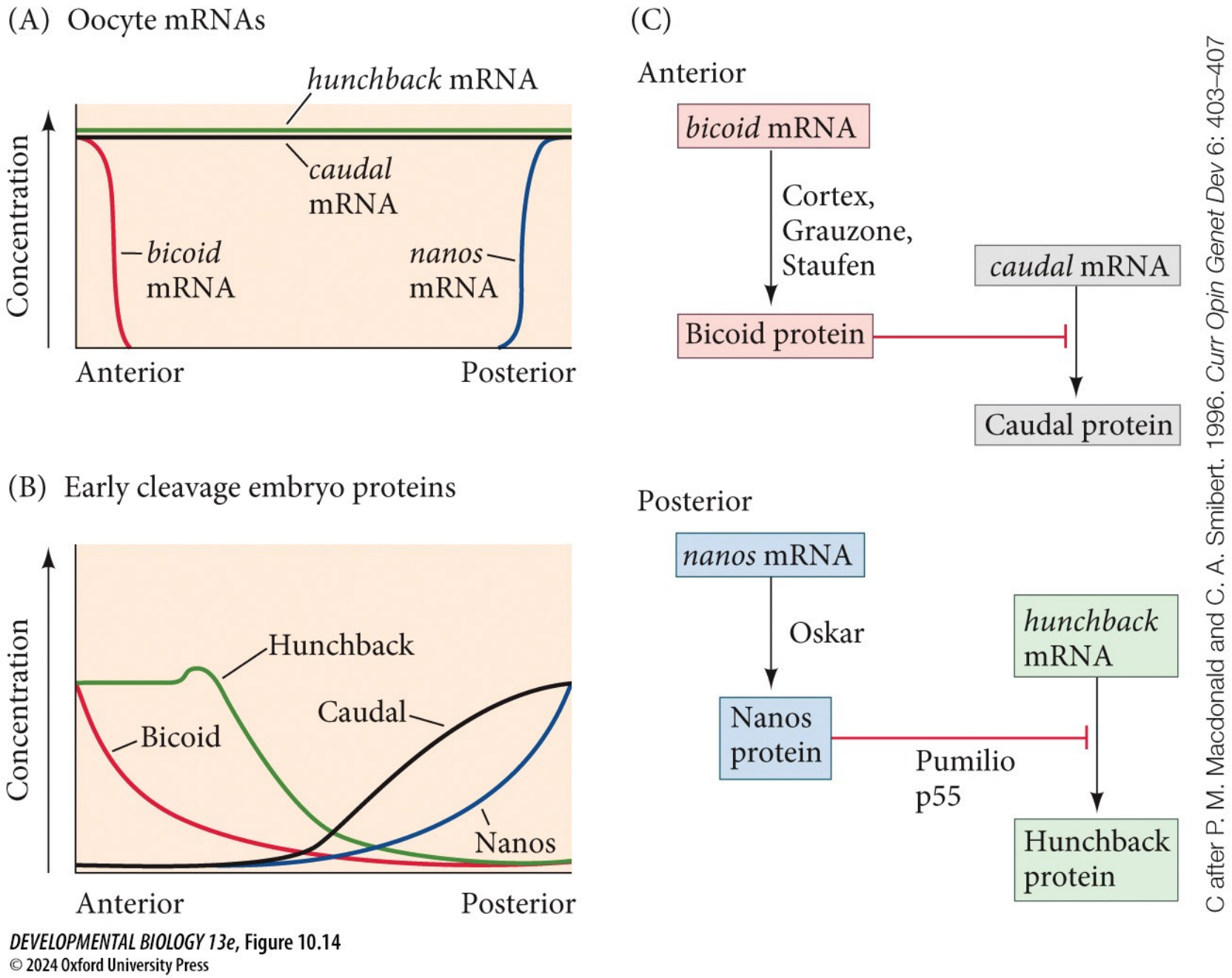
From P. M. Macdonald and G. Struhl. 1986. *Nature* 324: 537–545, courtesy of G. Struhl

Anterior

Posterior

DEVELOPMENTAL BIOLOGY 13e, Figure 10.13

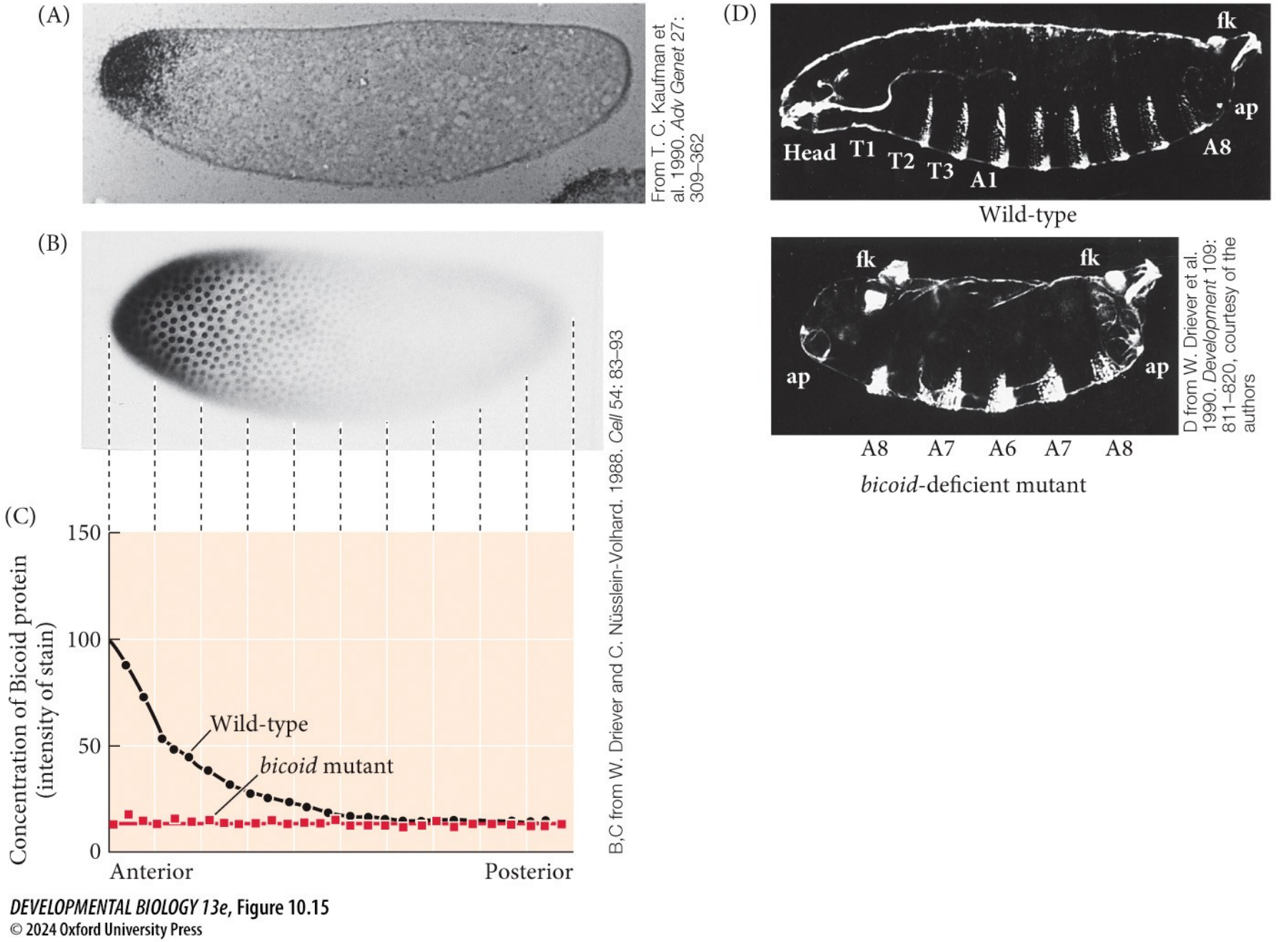
Figure 10.14 Model of anterior-posterior pattern generation by *Drosophila* maternal effect genes



C after P. M. Macdonald and C. A. Smibert. 1996. *Curr Opin Genet Dev* 6: 403–407

DEVELOPMENTAL BIOLOGY 13e, Figure 10.14

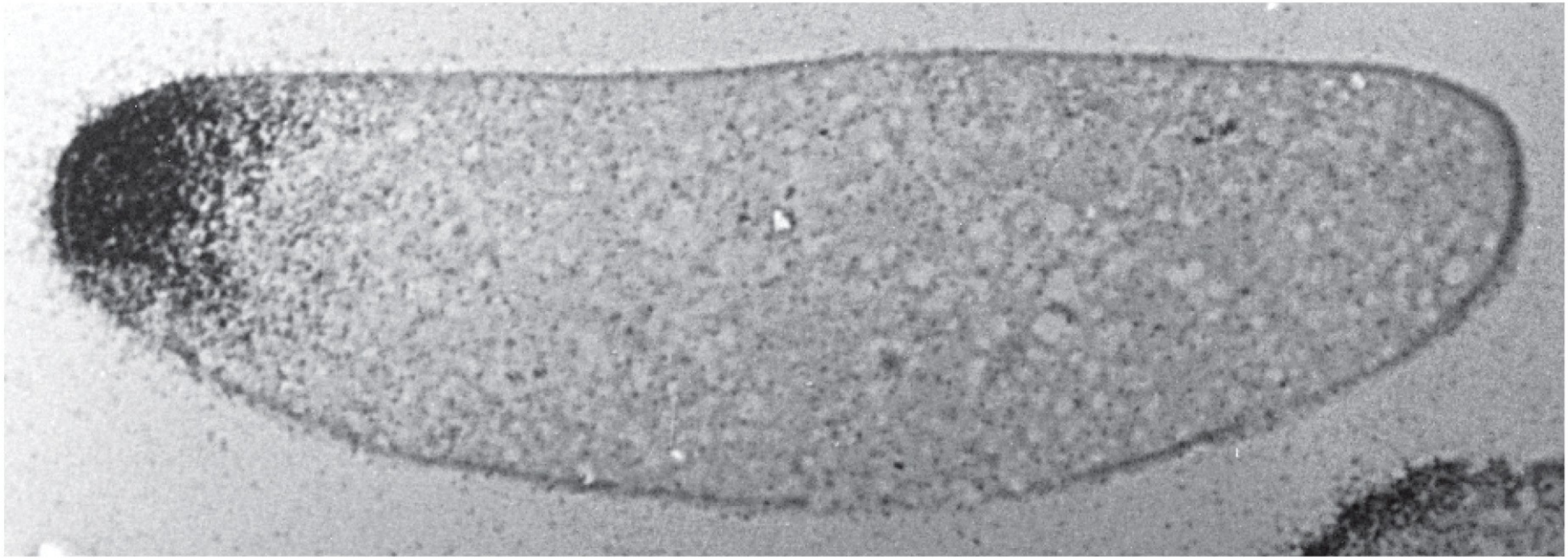
Figure 10.15 Bicoid protein gradient in the early *Drosophila* embryo



DEVELOPMENTAL BIOLOGY 13e, Figure 10.15

Figure 10.15 Bicoid protein gradient in the early *Drosophila* embryo (Part 1)

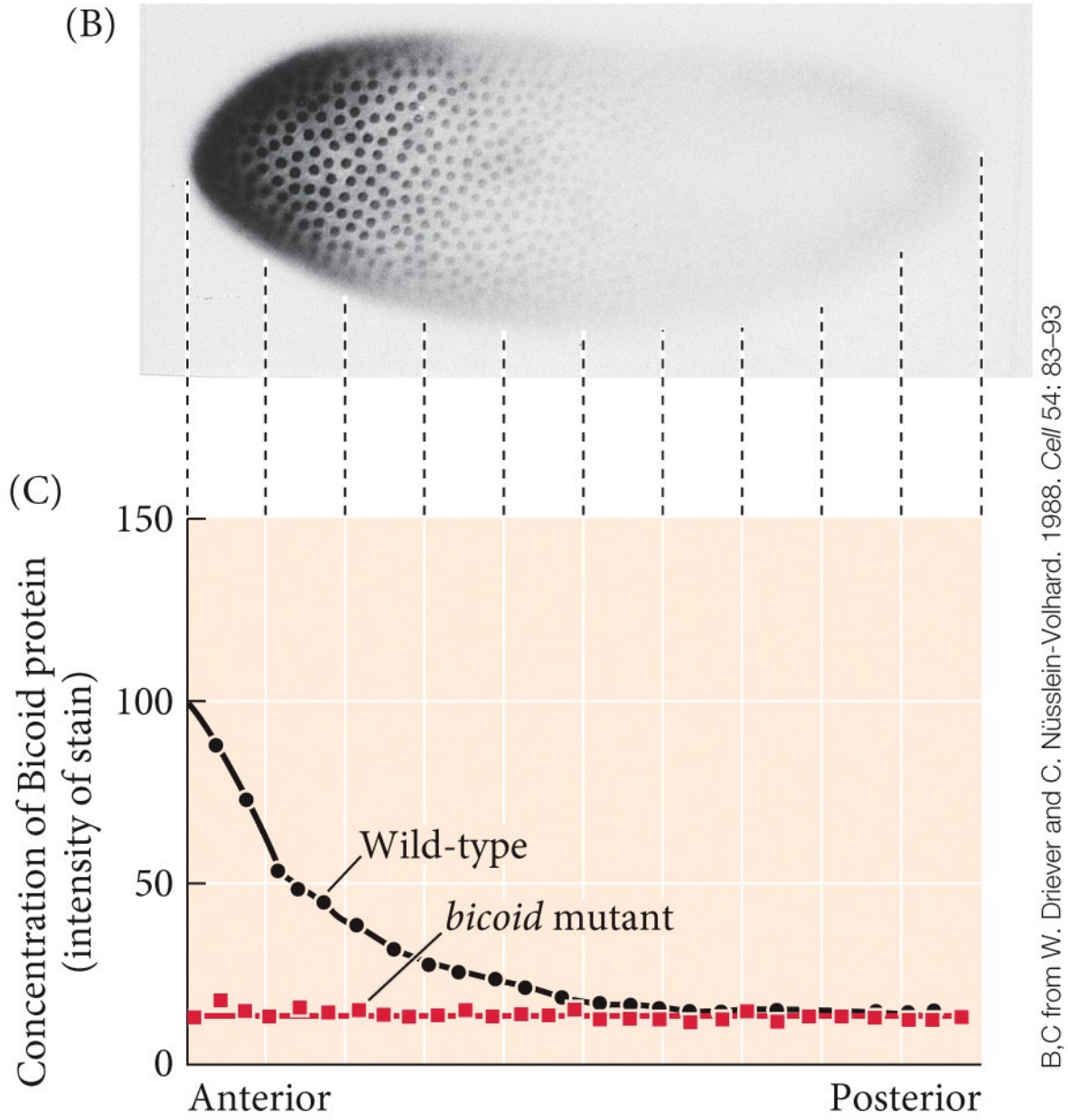
(A)



From T. C. Kaufman et al. 1990. *Adv Genet* 27: 309–362

DEVELOPMENTAL BIOLOGY 13e, Figure 10.15 (Part 1)

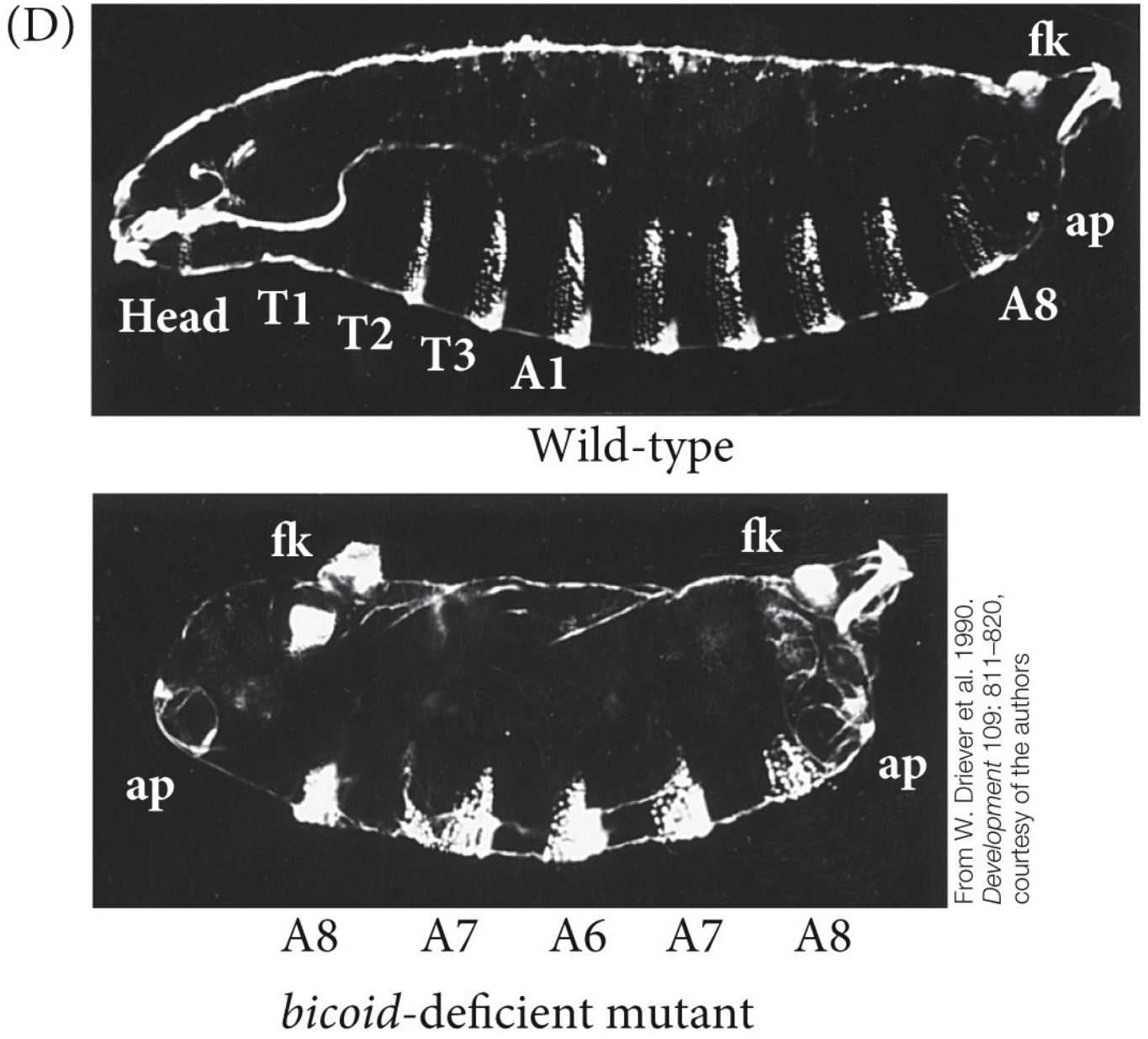
Figure 10.15 Bicoid protein gradient in the early *Drosophila* embryo (Part 2)



B,C from W. Driever and C. Nüsslein-Volhard. 1988. *Cell* 54: 83–93

DEVELOPMENTAL BIOLOGY 13e, Figure 10.15 (Part 2)

Figure 10.15 Bicoid protein gradient in the early *Drosophila* embryo (Part 3)



From W. Driever et al. 1990. *Development* 109: 811–820, courtesy of the authors

DEVELOPMENTAL BIOLOGY 13e, Figure 10.15 (Part 3)

Figure 10.19 Messenger RNA expression patterns of two pair-rule genes, *even-skipped* (red) and *fushi tarazu* (black), in the *Drosophila* blastoderm

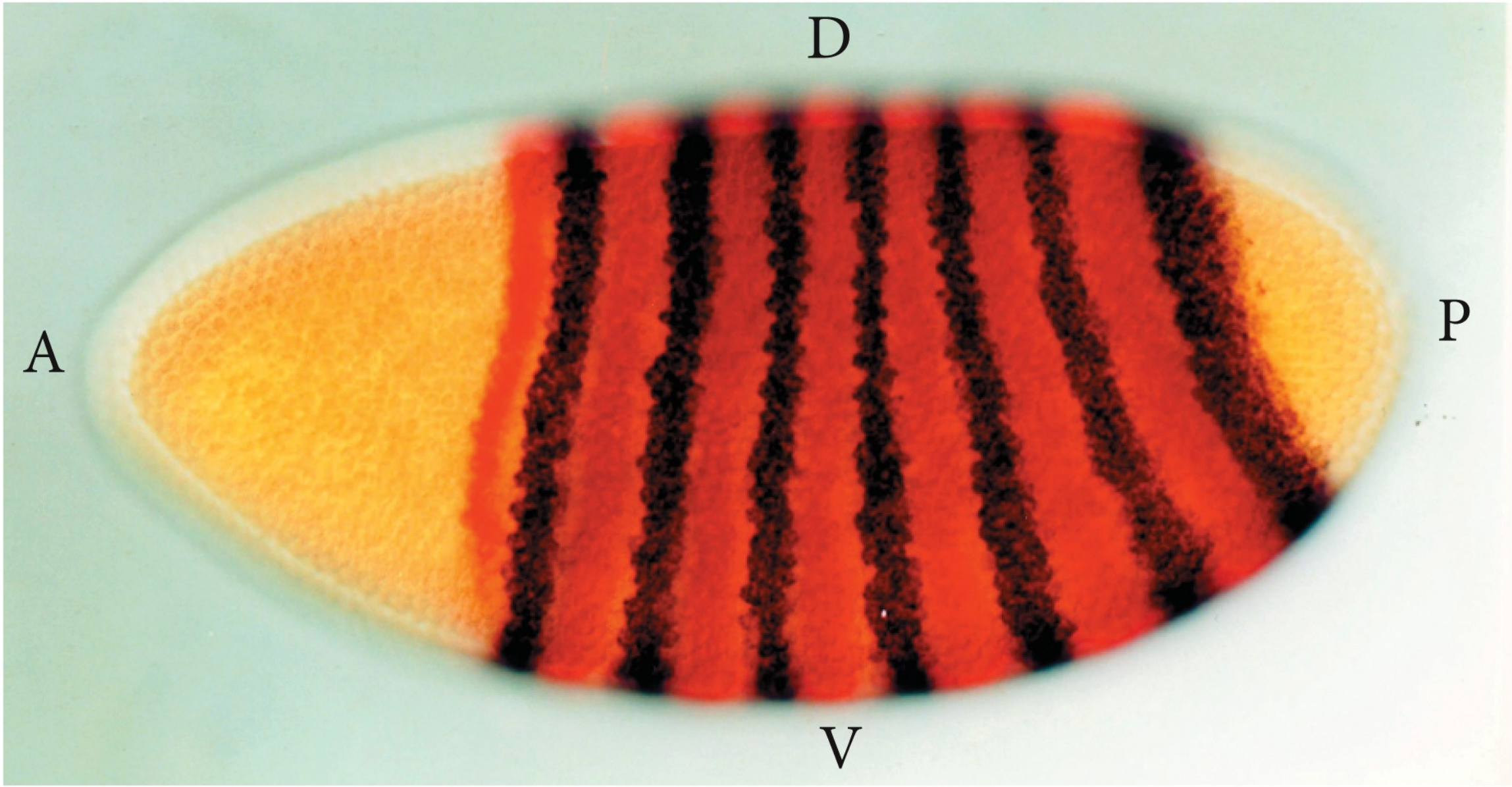
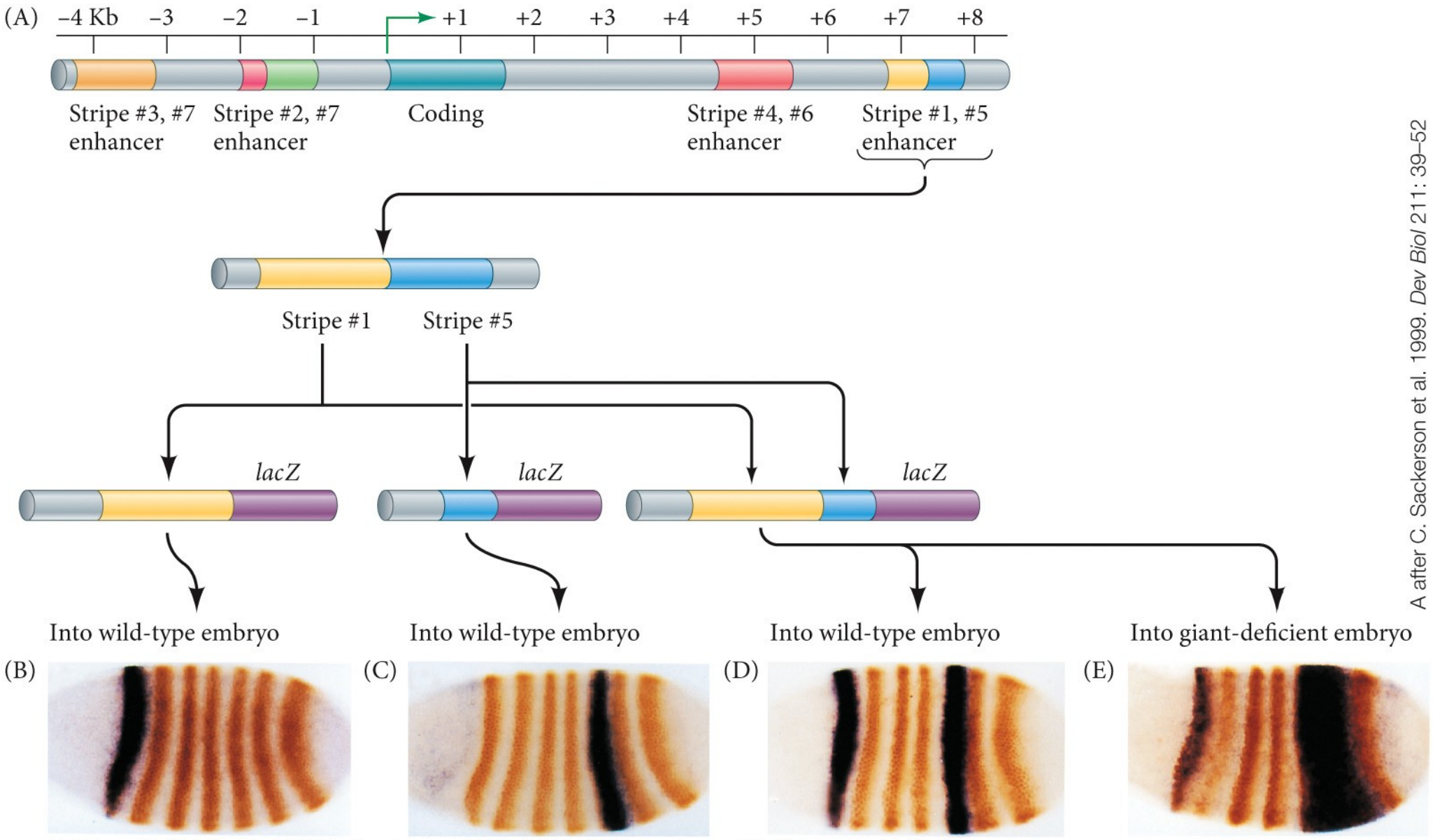


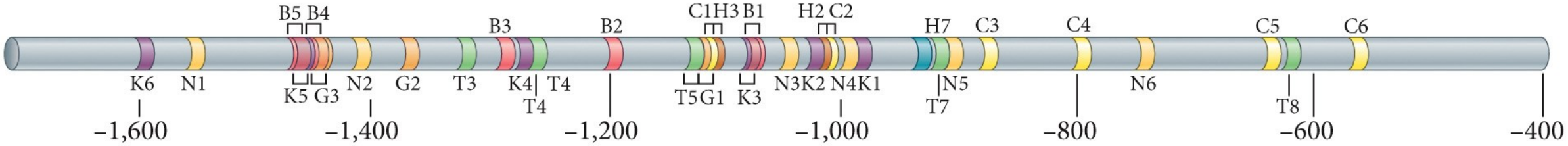
Figure 10.20 Specific promoter regions of the *even-skipped* (*eve*) gene control specific transcription bands in the embryo



A after C. Sackerson et al. 1999. *Dev Biol* 211: 39–52

B–E from M. Fujioka et al. 1999. *Development* 126: 2527–2538, courtesy of M. Fujioka and J. B. Jaynes

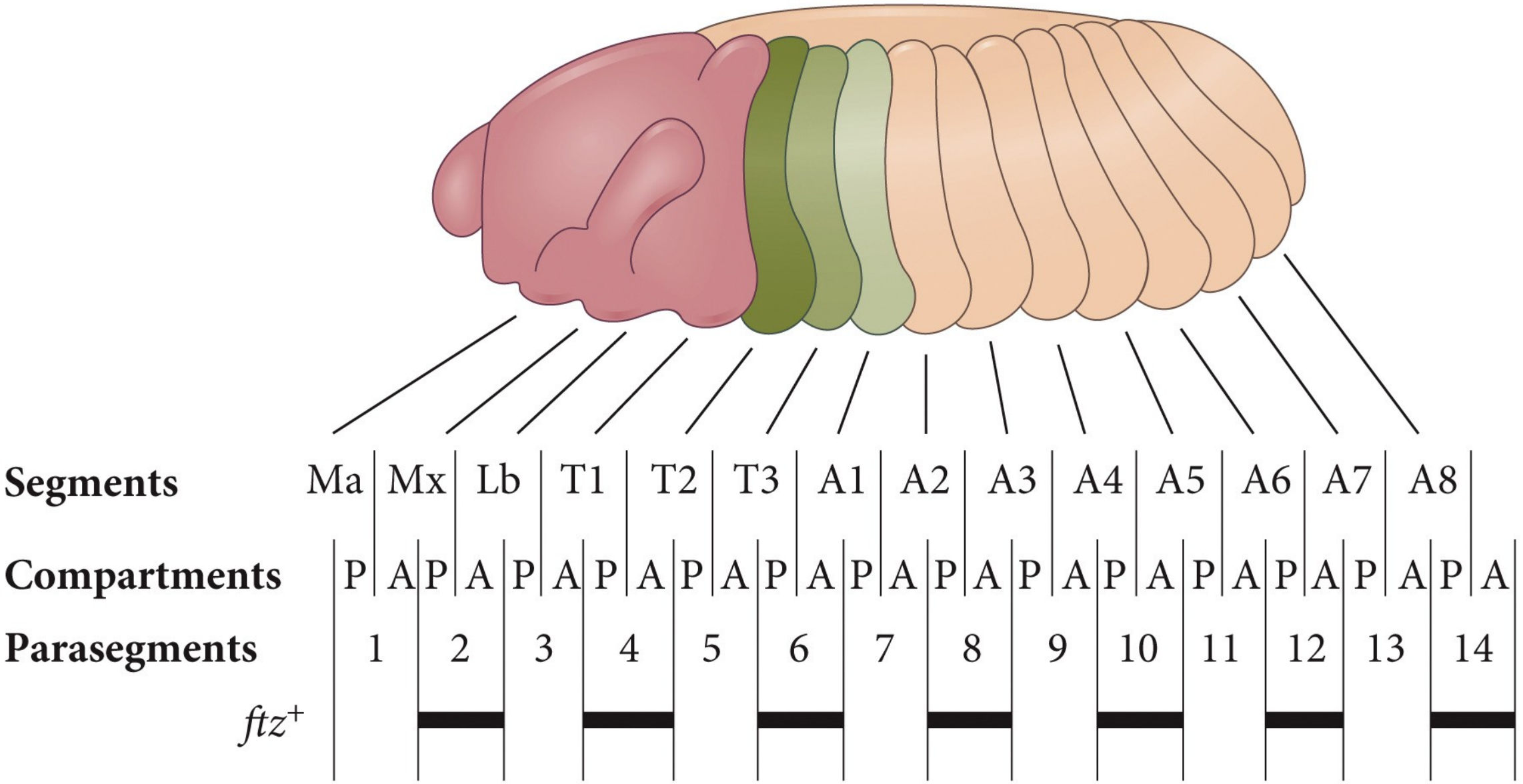
Figure 10.21 Model for formation of the second stripe of transcription from the *even-skipped* gene



After H. Janssens et al. 2006. *Nat Genet* 38: 1159–1165

***DEVELOPMENTAL BIOLOGY 13e*, Figure 10.21**

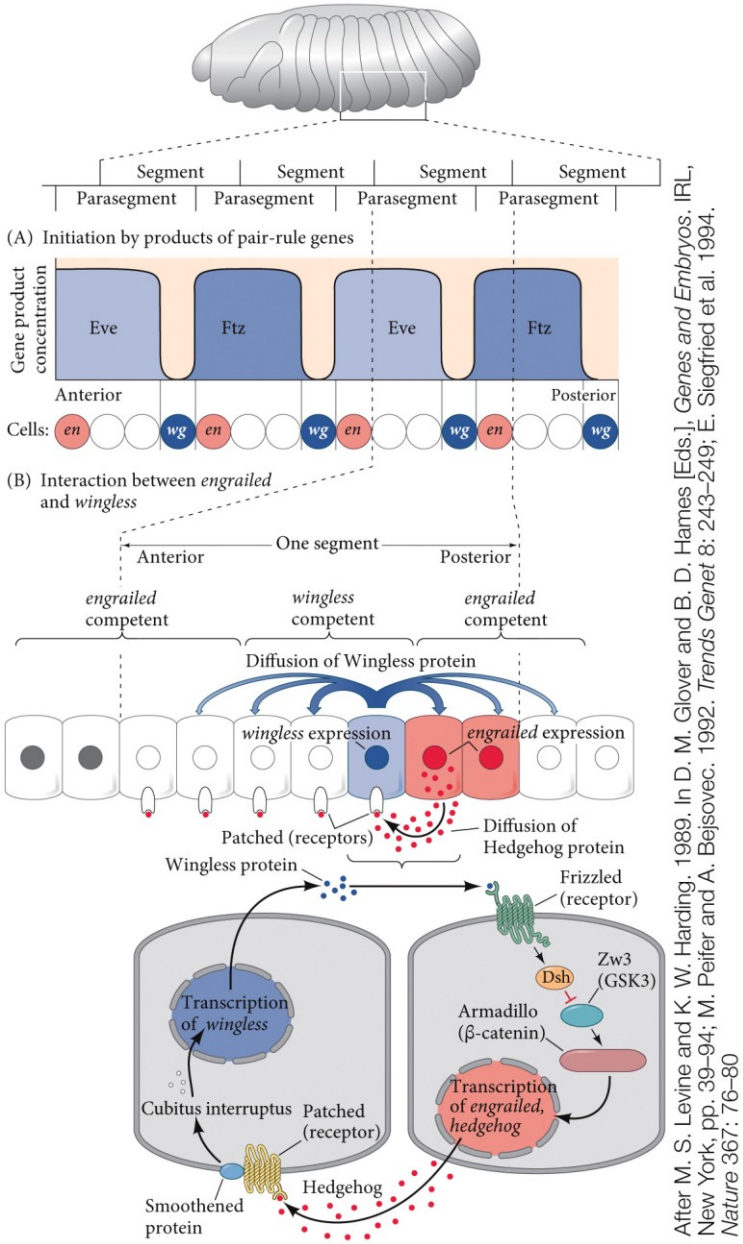
Figure 10.17 Parasegments in the *Drosophila* embryo are shifted one compartment forward in relation to the segments



After A. Martinez-Arias and P. A. Lawrence. 1985. *Nature* 313: 639–642

DEVELOPMENTAL BIOLOGY 13e, Figure 10.17

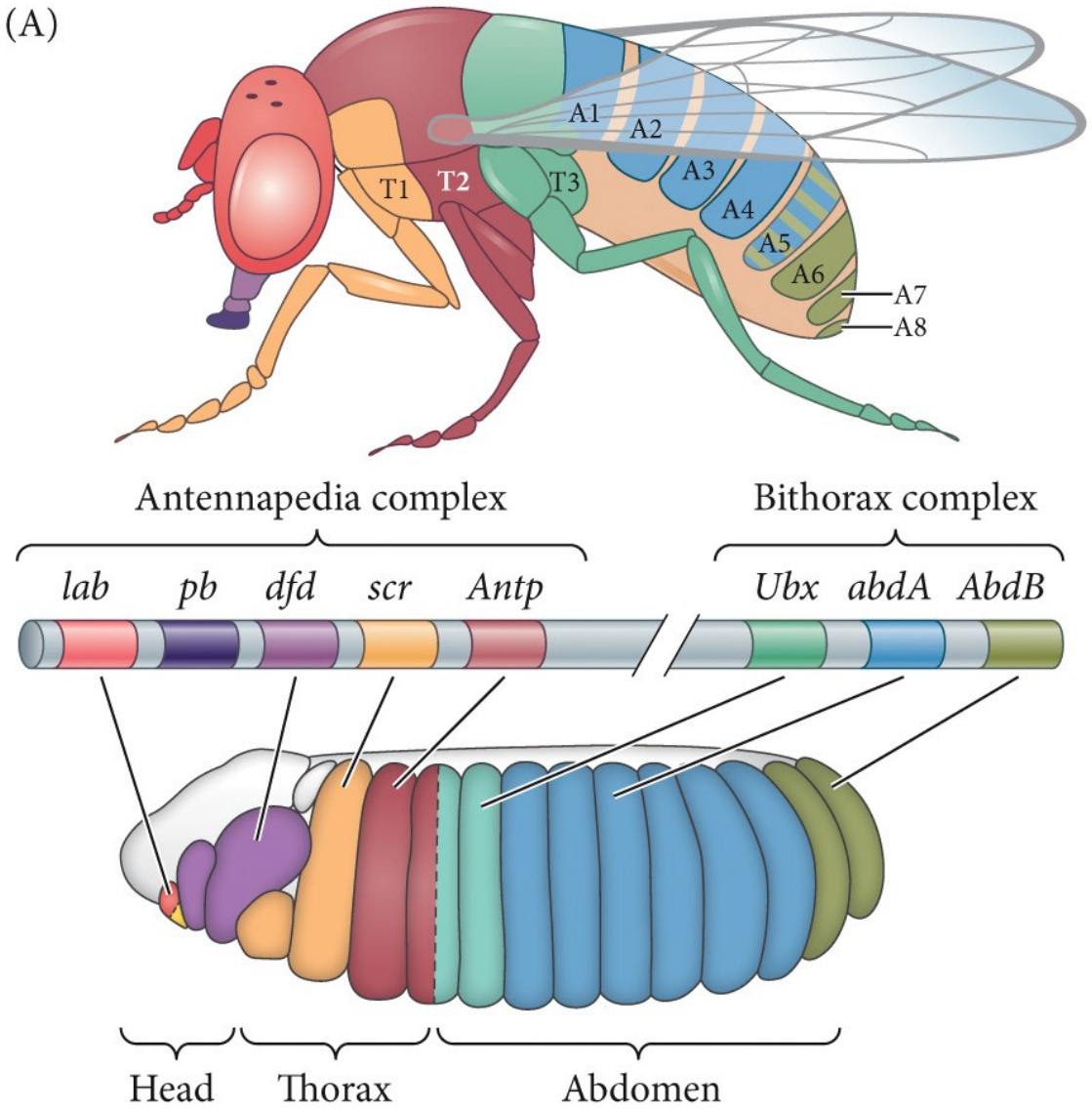
Figure 10.22 Model for transcription of the segment polarity genes *engrailed* (*en*) and *wingless* (*wg*)



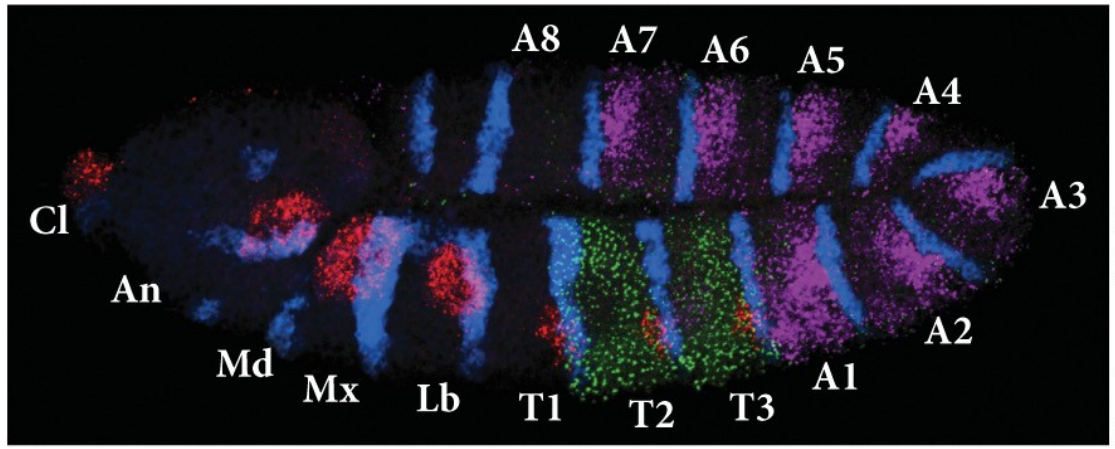
After M. S. Levine and K. W. Harding, 1989. In D. M. Glover and B. D. Hames [Eds.], *Genes and Embryos*. IRL, New York, pp. 39–94; M. Peifer and A. Bejsovec. 1992. *Trends Genet* 8: 243–249; E. Siegfried et al. 1994. *Nature* 367: 76–80

***DEVELOPMENTAL BIOLOGY 13e*, Figure 10.22**

Figure 10.23 Homeotic gene expression in *Drosophila*



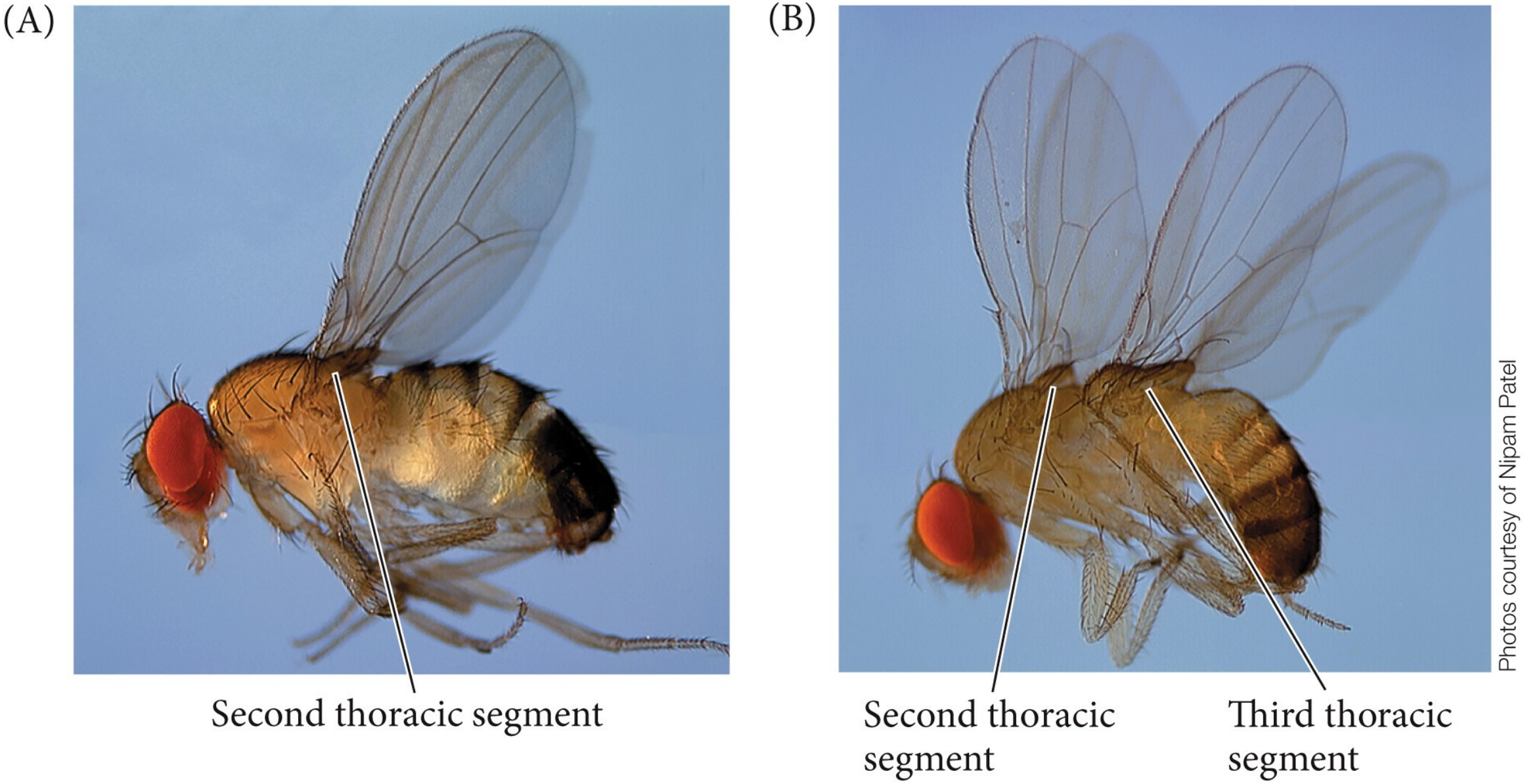
A after T. C. Kaufman et al. 1990. *Adv Genet* 27: 309–362; S. Dessain et al. 1992. *EMBO J* 11: 991–1002



Courtesy of D. Kosman

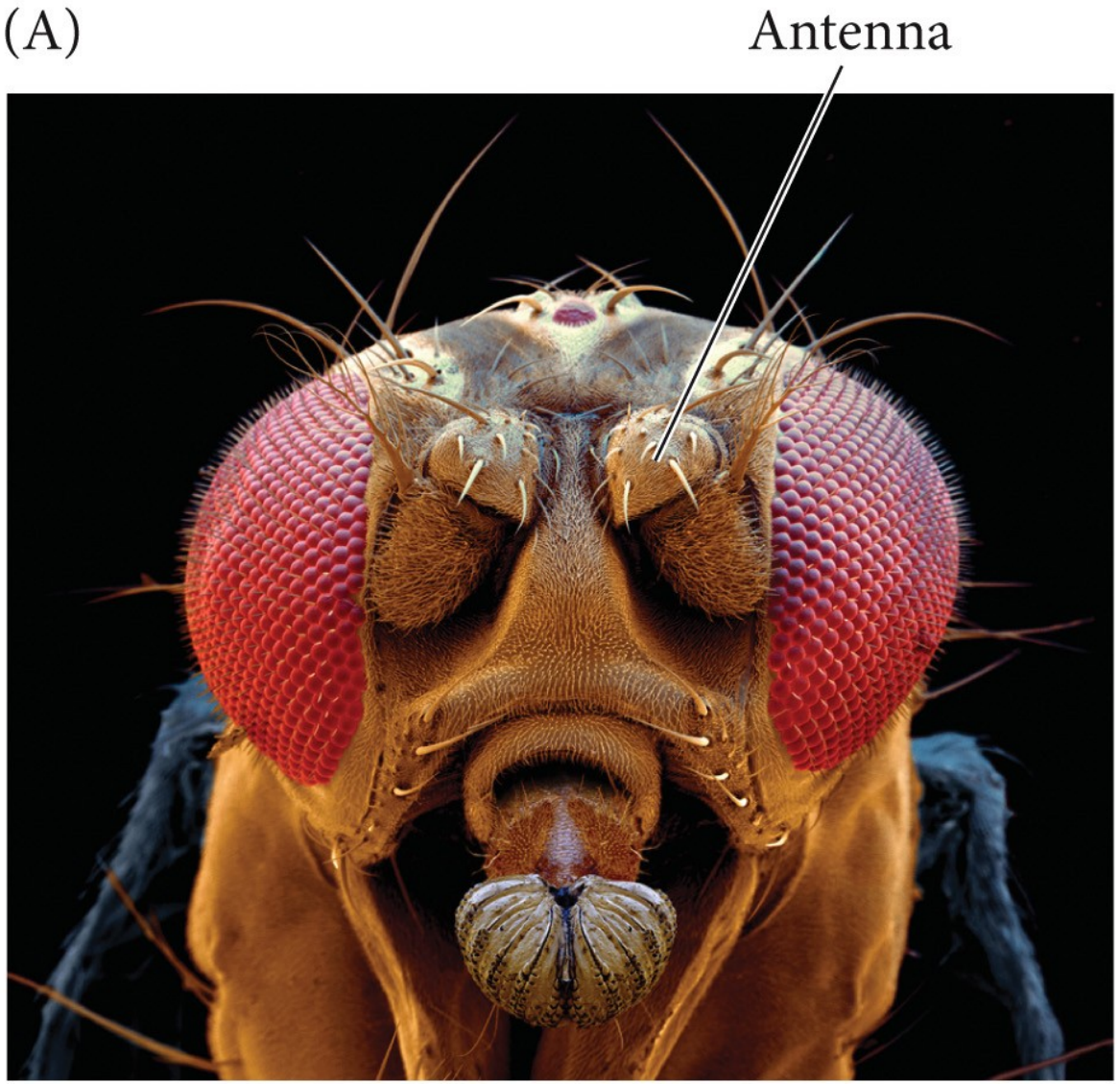
DEVELOPMENTAL BIOLOGY 13e, Figure 10.23

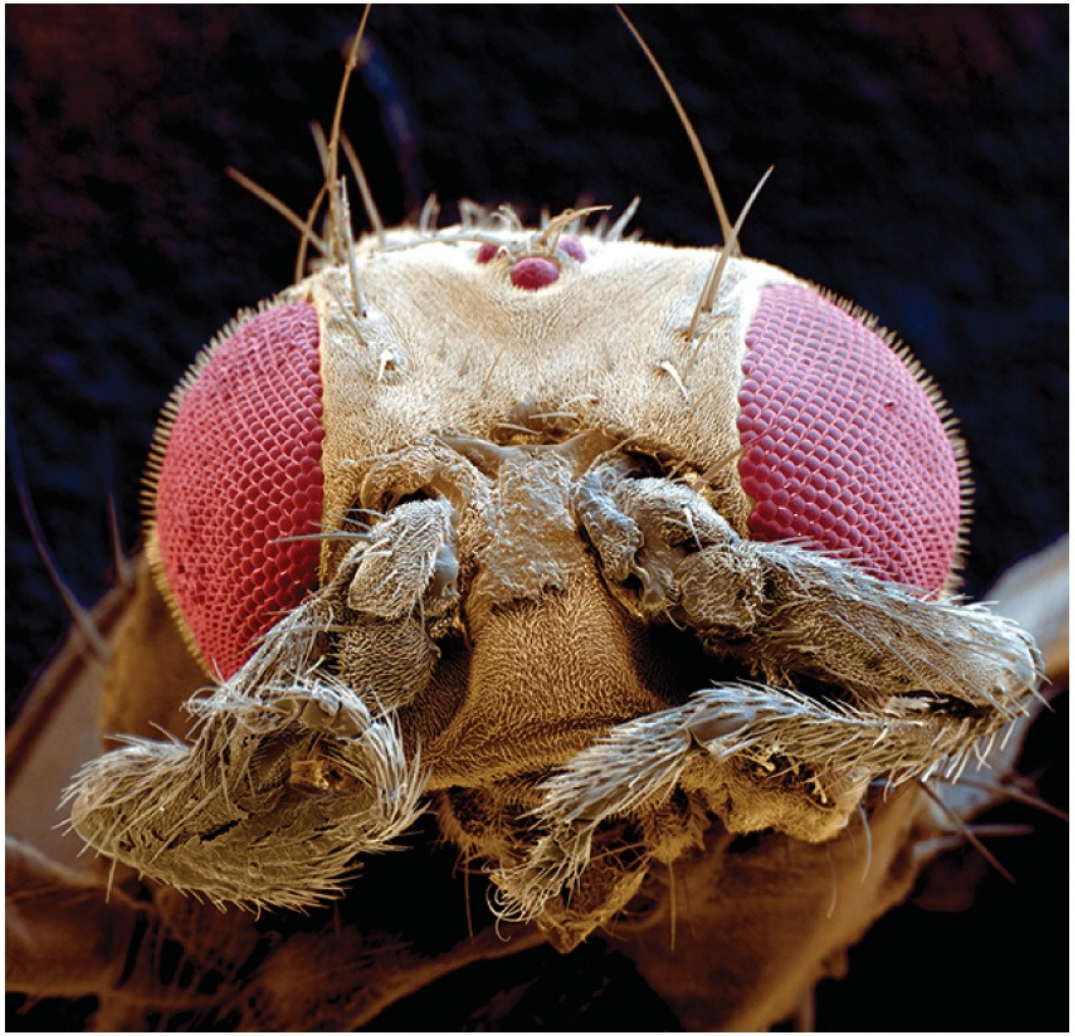
Figure 10.24 (A) Wings of the wild-type fruit fly emerge from the second thoracic segment and (B) a four-winged fruit fly constructed by putting together three mutations in *cis*-regulators of the *Ultrabithorax* gene



DEVELOPMENTAL BIOLOGY 13e, Figure 10.24

Figure 10.25 (A) Head of a wild-type fruit fly. (B) Head of a fly with the *Antennapedia* mutation that converts antennae into legs





A,B © Eye of Science/Science Source

DEVELOPMENTAL BIOLOGY 13e, Figure 10.25

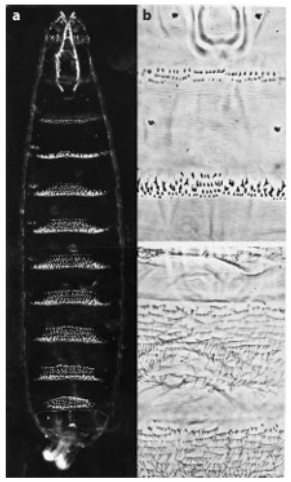
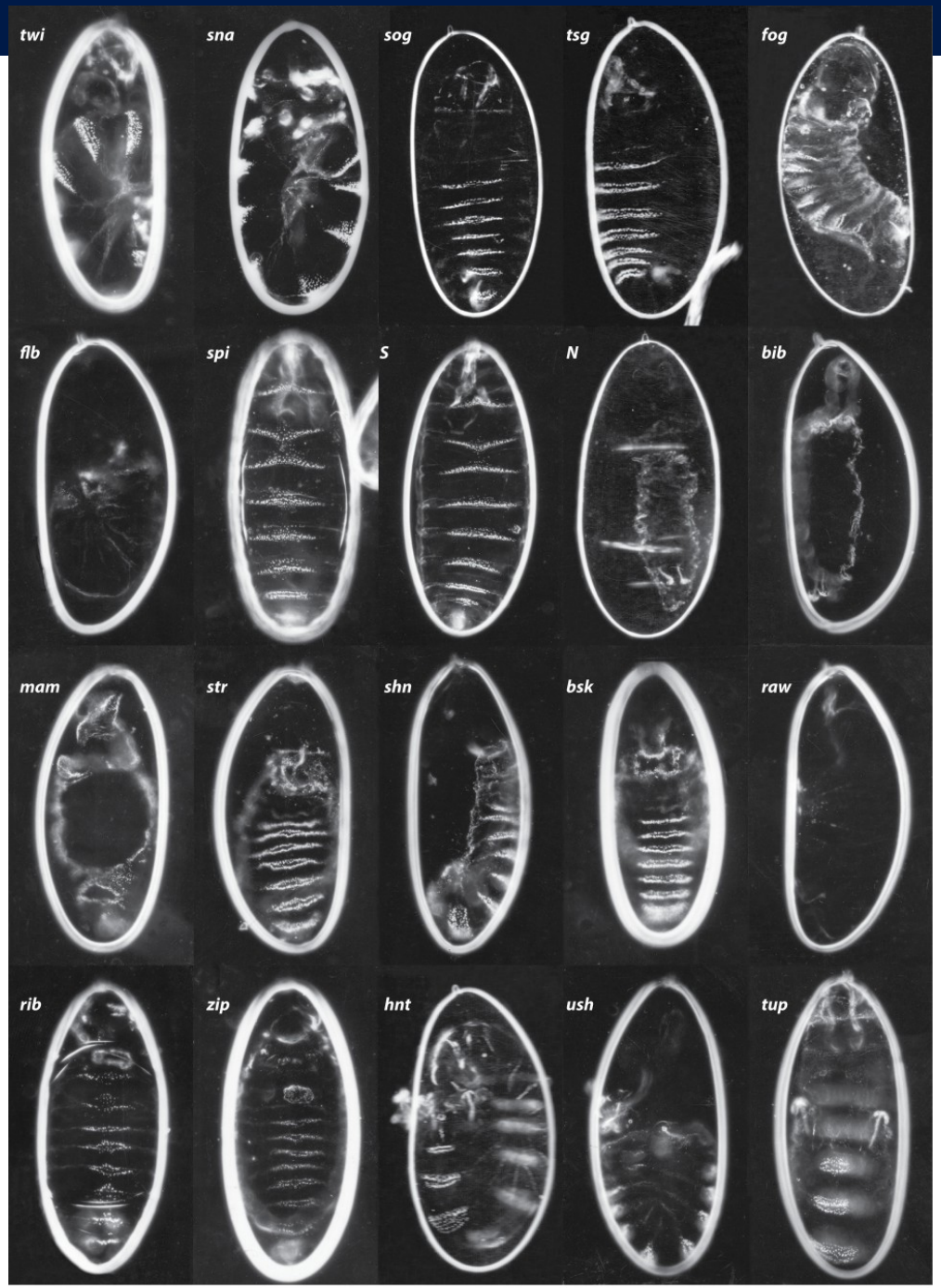


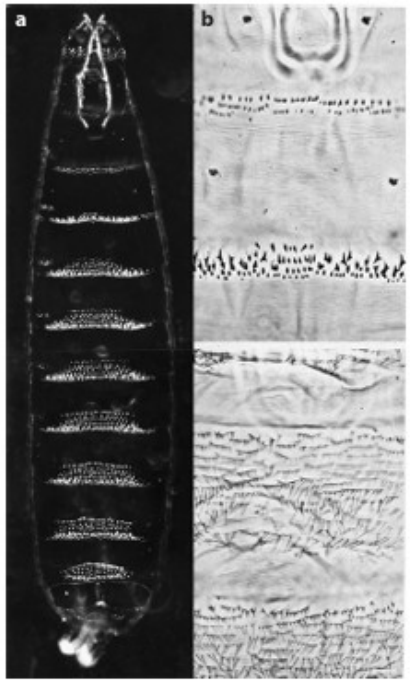
Figure 1
Cuticle preparation of a *Drosophila* first-instar larva. (*a*) Dark-field image of the ventral side. (*b*) Phase-contrast detail of the ventral (*top*) and the dorsal (*bottom*) aspect of the posterior thorax and the first abdominal segment.

Figure 14

Mutants affected in dorsal-ventral patterning: 20 mutants of genes listed in **Table 2** represent the following classes: gastrulation dorsalized group [*twist* (*twi*) and *snail* (*sna*)], gastrulation *decapentaplegic* group [*short gastrulation* (*sog*), *twisted gastrulation* (*tsg*), and *folded gastrulation* (*fog*)], *spitz* group [*faint little ball* (*flb*), *spitz* (*spi*), and *Star* (*S*)], neuralized mutants [*Notch* (*N*), *big brain* (*bib*), and *mastermind* (*mam*)], dorsal pattern mutants [*slater* (*str*)/*thickvein*, *schnurri* (*shn*), and *basket* (*bsk*)], dorsal closure group [*raw*, *ribbon* (*rib*), and *zipper* (*zip*)], and *u-shaped* group [*hindsight* (*hnt*), *u-shaped* (*ush*), and *tail up* (*tup*)]. From **Nüsslein-Volhard et al. (1984)** and **Wieschaus et al. (1984a)**.



Wieschaus E, Nüsslein-Volhard C, 2016



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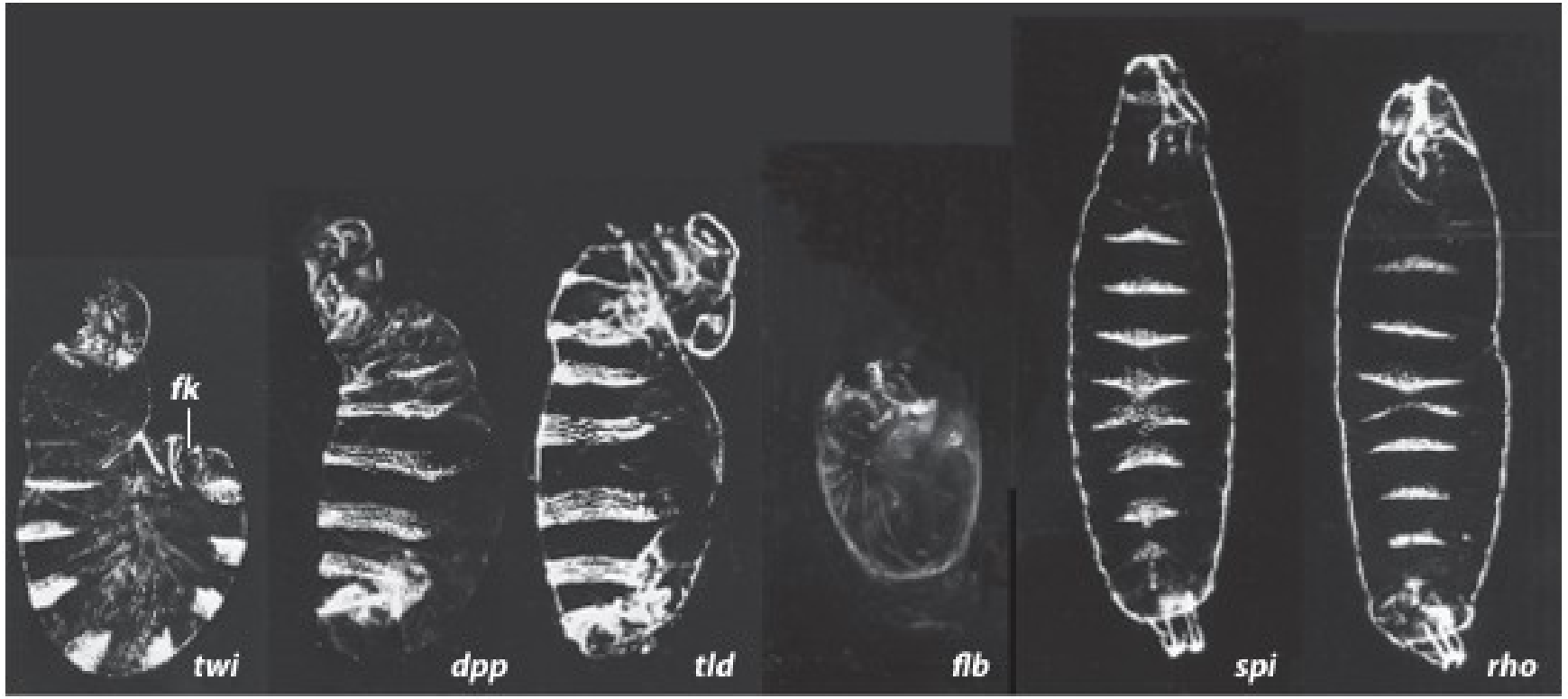


Figure 15

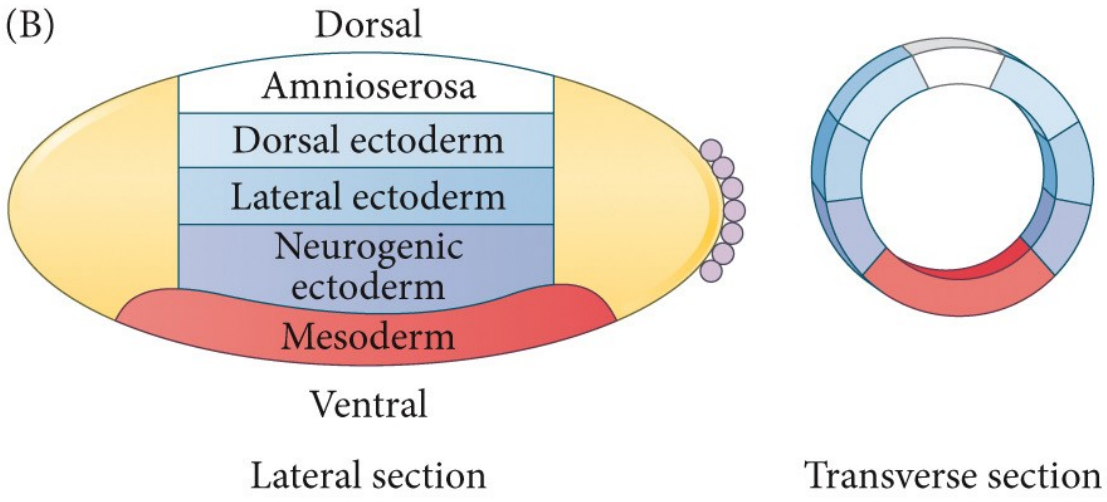
Gastrulation and ventral pattern mutants: gastrulation dorsalized *twist* (*twi*) and gastrulation ventralized *decapentaplegic* (*dpp*) (weak allele) and *tolloid* (*tld*); *spitz* group *faint little ball* (*flb*), *spitz* (*spi*), and *rhomboid* (*rho*). The ventral aspects of mutant larvae dissected out of the vitelline membrane. The vitelline membrane of the *flb* embryo was removed by Photoshop. From Arora & Nüsslein-Volhard (1992) and Mayer & Nüsslein-Volhard (1988).

Figure 10.28 Specification of cell fate by the Dorsal protein

(A)

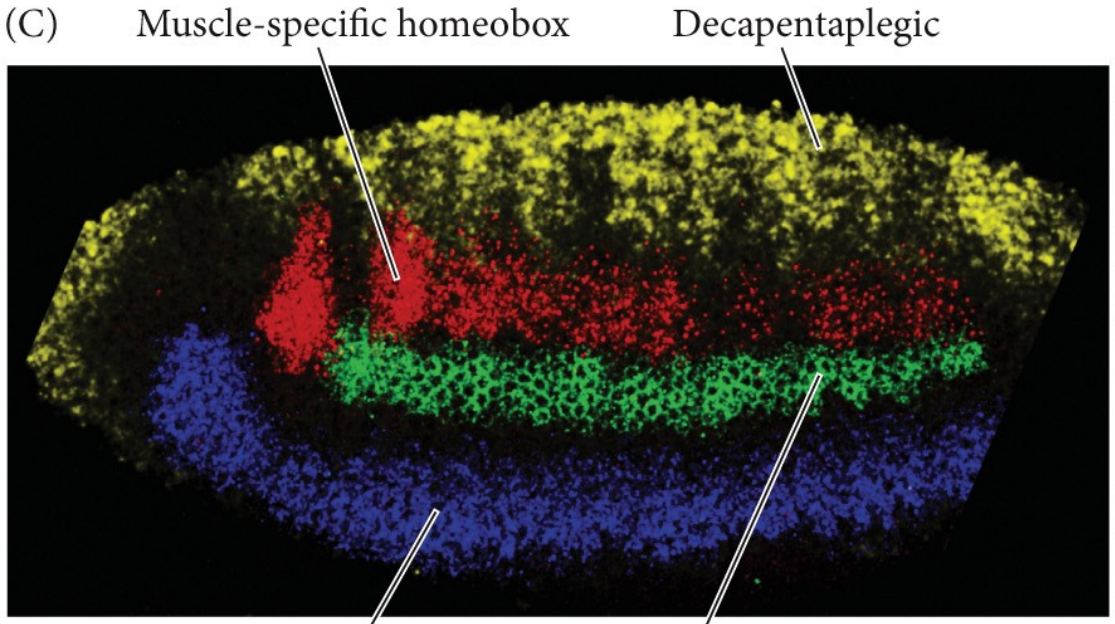
From S. Roth et al. 1989. *Cell* 59: 1189–1202, courtesy of the authors

(B)



After C. A. Rushlow et al. 1989. *Cell* 59: 1165–1177

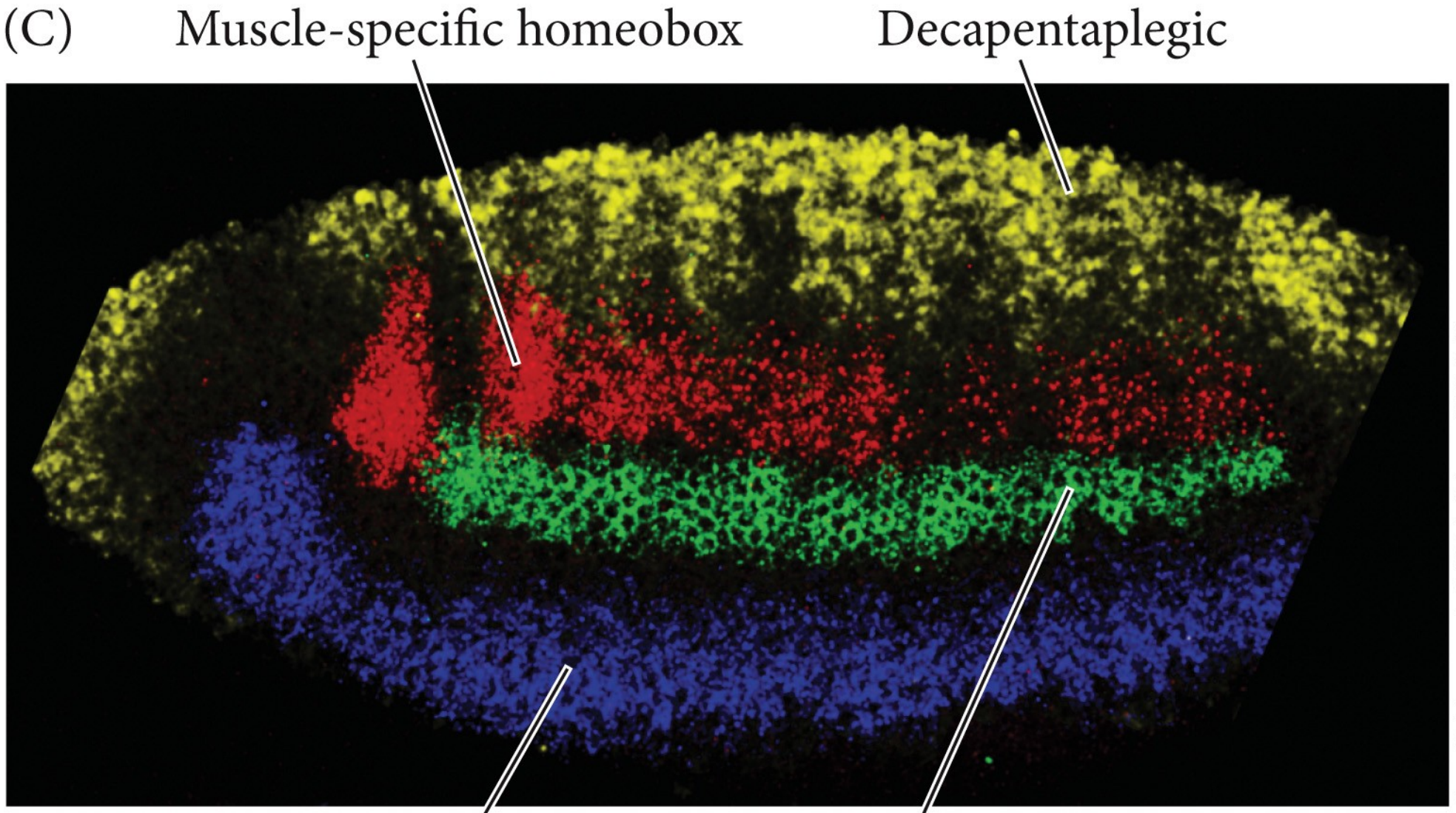
(C)



From D. Kosman et al. 2004. *Science* 305: 846, courtesy of D. Kosman and E. Bier

***DEVELOPMENTAL BIOLOGY 13e*, Figure 10.28**

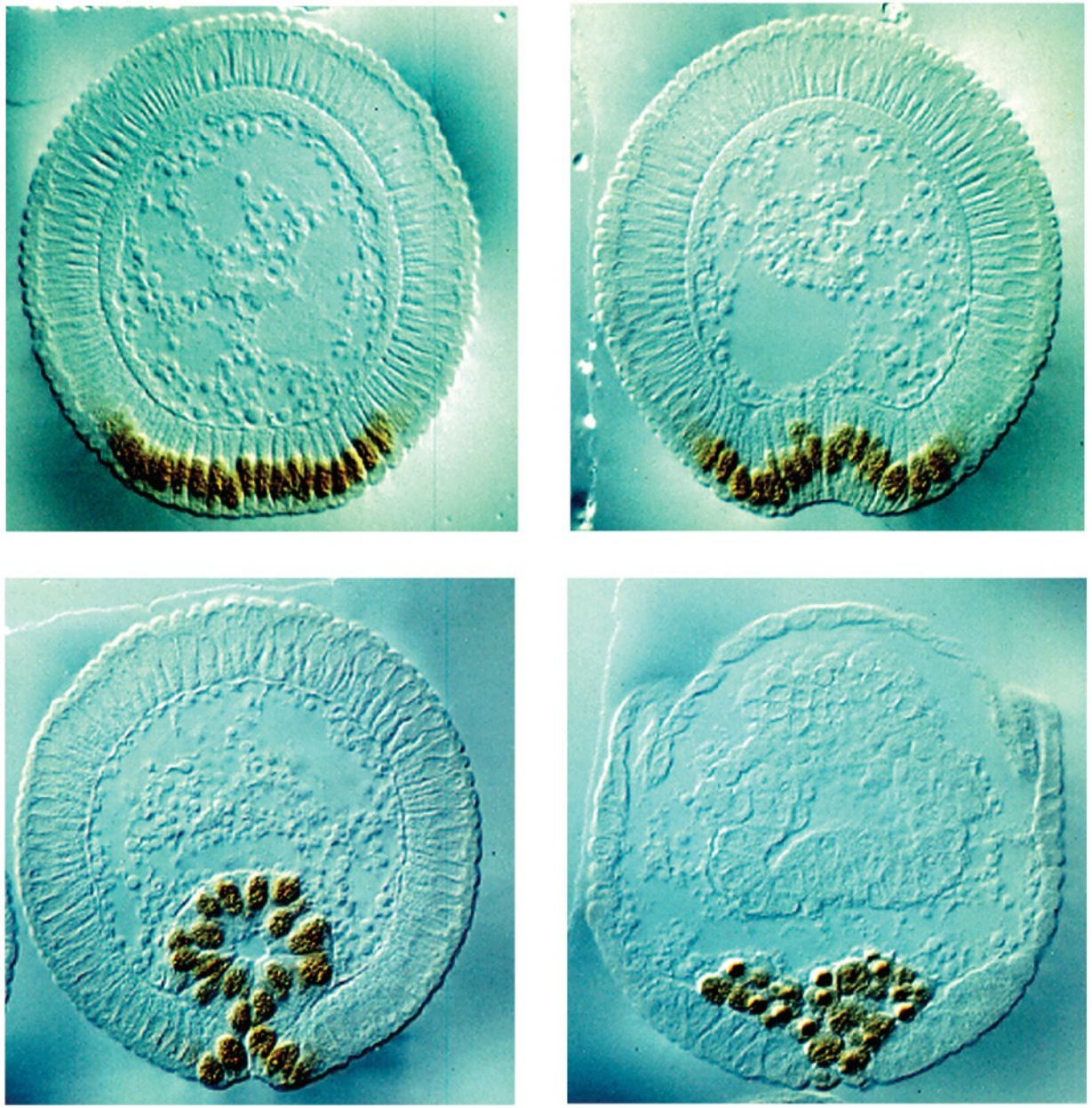
Figure 10.28 Specification of cell fate by the Dorsal protein (Part 3)



From D. Kosman et al. 2004. *Science* 305: 846, courtesy of D. Kosman and E. Bier

DEVELOPMENTAL BIOLOGY 13e, Figure 10.28 (Part 3)

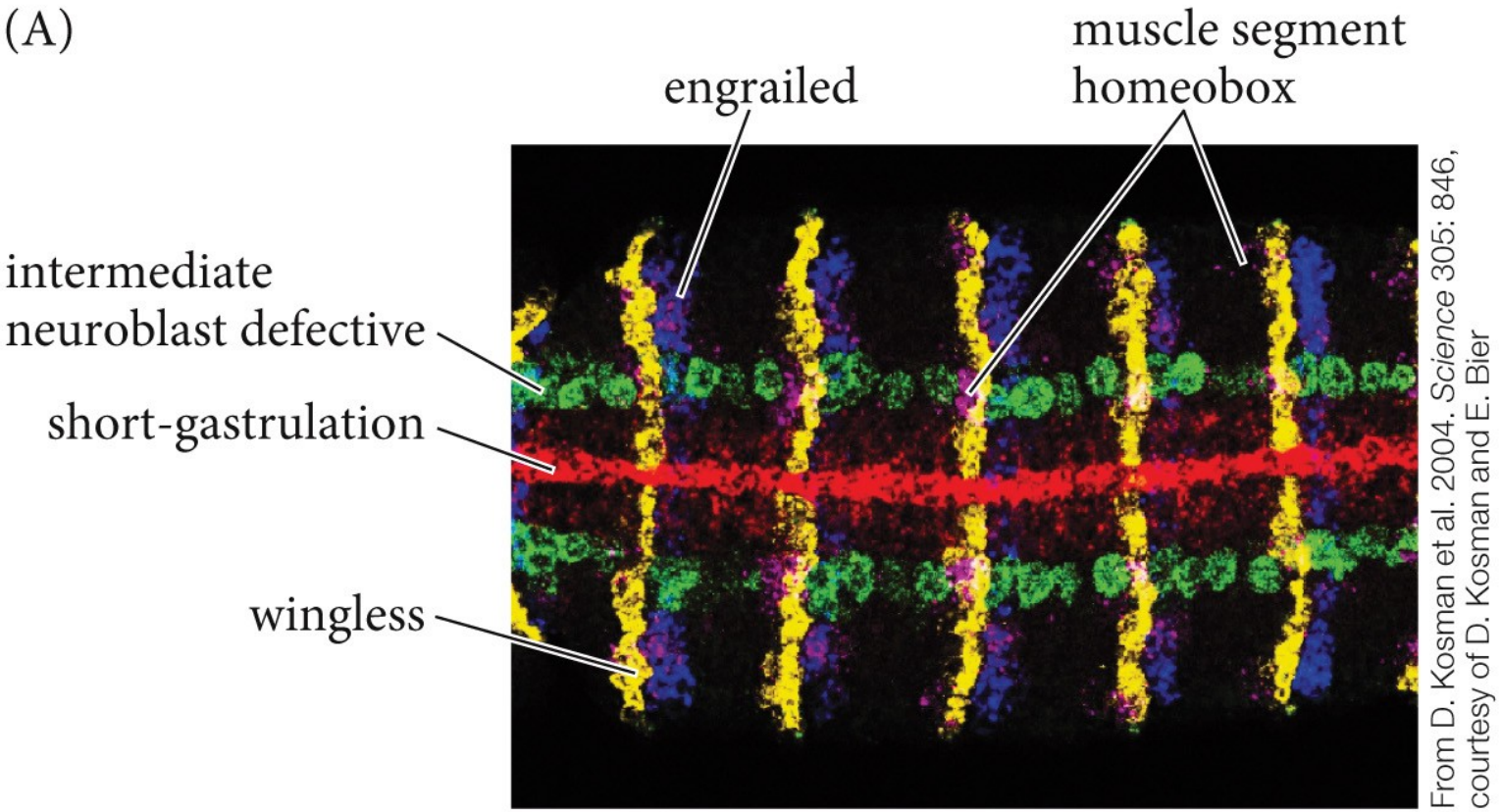
Figure 10.29 Gastrulation in *Drosophila*

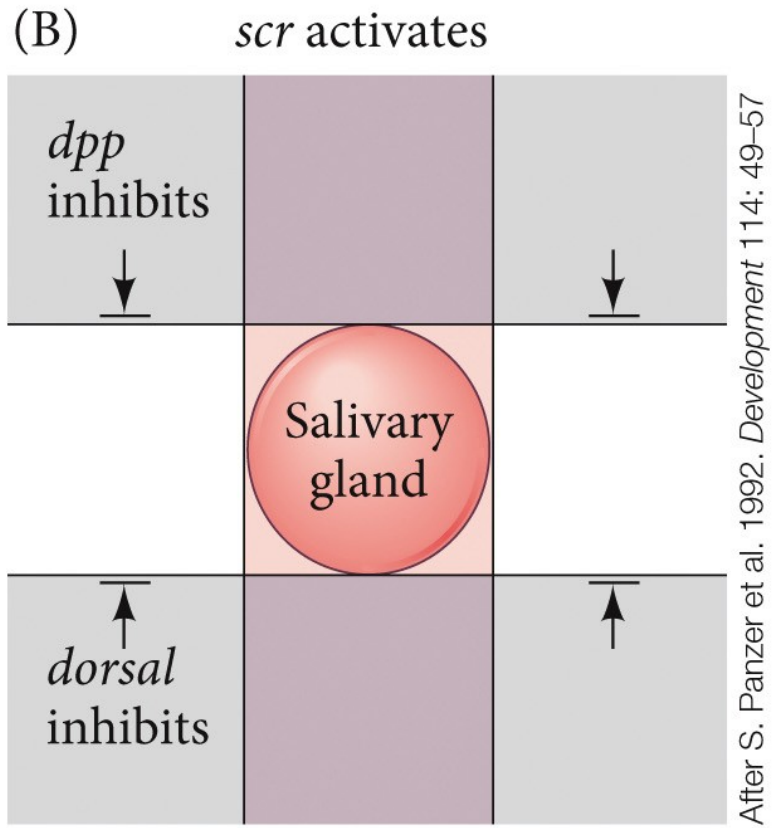


From M. Leptin. 1991. In *Gastrulation: Movements, Patterns, and Molecules*, R. Keller et al. (Eds.), pp. 199–212. Plenum: New York, courtesy of M. Leptin

DEVELOPMENTAL BIOLOGY 13e, Figure 10.29

Figure 10.30 Cartesian coordinate system mapped out by gene expression patterns





DEVELOPMENTAL BIOLOGY 13e, Figure 10.30

- Reading
- Heidelberg screen, Wieschaus and Nusslein Vollhard
- Scot Gilbert textbook chapter of Drosophila