DEVELOPMENT

Jelly belly

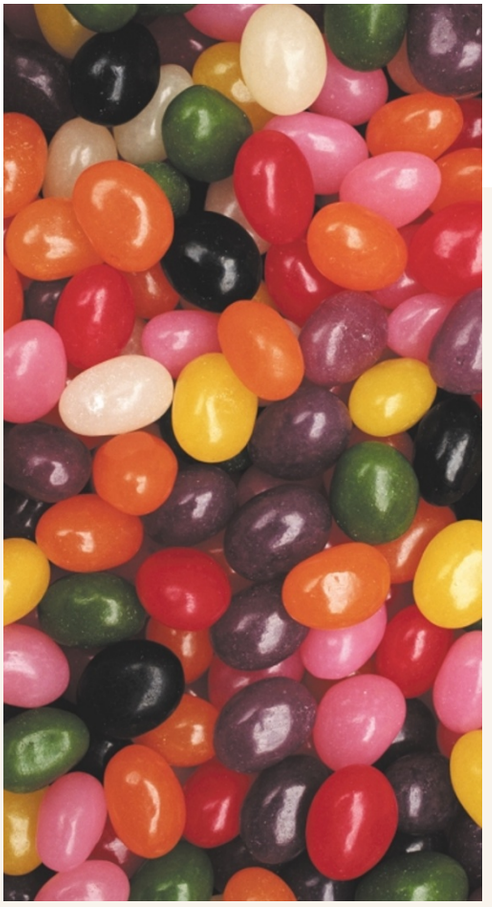
Much is known about early patterning in *Drosophila melanogaster* development, but the subsequent events are still a mystery. For example, which genes control the migration and differentiation processes required to generate specialized tissues? Weiss and colleagues recently addressed this question, and, reporting in *Cell*, they describe the discovery of a novel *Drosophila* gene called *jelly belly* (*jeb*). They propose that the Jeb protein acts as a positive extracellular signalling molecule and that it is essential for the development of midgut muscles (visceral mesoderm).

Visceral mesoderm precursors are formed when the products of the genes decapentaplegic (*dpp*) and hedgehog (*hh*) act in combination to activate bagpipe (*bap*), the product of which is an NK class homeodomain protein.

Weiss and collegues did a screen to identify *Drosophila* genes that are transcriptionally controlled by Tinman (Tin), another member of the NK class of homeodomain proteins and an essential regulator of visceral mesoderm development. One of the DNA fragments isolated in this screen lay adjacent to a novel gene, *jeb*, which is expressed in somatic muscle precursors. The authors showed that, although sufficient, Tin is not necessary for *jeb* expression in these precursor cells. The *jeb* gene is expressed in somatic muscle precursors from stages 8–12 of *Drosophila* embryo development, but Jeb is not required for somatic muscle development.

To investigate Jeb function, Weiss *et al.* looked at *jeb* mutants and found that no differentiated visceral mesoderm could be detected, although other muscular components of the mesoderm developed normally. Visceral mesoderm precursors were specified in the mutants, as indicated by normal staining for Bap, but they failed to migrate and did not differentiate to form the visceral mesoderm. The authors propose that, in *jeb* mutants, these precursors default to a somatic mesoderm cell fate, as they found no evidence of apoptosis and *jeb*-mutant embryos showed an increase in the number of nuclei in the positions of somatic muscle precursors.

The somatic muscle precursors in which Jeb is produced lie next to the visceral mesoderm precursors that depend on Jeb function. Jeb contains a secretory signal sequence and a type A LDL receptor repeat. Based on these features, the authors proposed that Jeb is secreted from somatic precursor cells and that it acts in the extracellular compartment. They confirmed this by showing that Jeb is secreted from *Drosophila* tissue culture cells and that *bap*-expressing visceral muscle precursors, which do not express *jeb* but depend on *jeb* function, contain Jeb protein. The authors showed that visceral mesoderm precursors accumulate Jeb by receptor-mediated endocytosis. This endocytosis requires the type A LDL receptor repeat in Jeb; a dynamin-related GTPase, Shibire, that is required for microtubule-mediated endocytosis; and, probably, a Jeb-specific receptor.



To rule out the possibility that Jeb acts in somatic muscle precursors to produce a signal that is not Jeb, the authors expressed Jeb in the visceral muscle precursors of *jeb*-mutant embryos. They showed that this expression rescued differentiation of these precursor cells, but that migration of the precursors remained defective. Weiss and co-workers therefore propose that Jeb acts as a signal, and that, by being secreted from somatic muscle cell precursors, it conveys positional information to visceral muscle precursors.

In conclusion, Jeb is required for *Drosophila* midgut muscle development. It acts as a positive migratory or differentiation signal for visceral mesoderm precursors, and it is possible that this new signalling system has been conserved in evolution. It seems that Jeb might act as a developmental signal in many contexts, as Weiss *et al.* have shown that *jeb* messenger RNA is expressed in the embryonic central nervous system and that Jeb can be transported along axons. The authors are now investigating Sco-spondin in mice, a secreted protein that is highly similar to Jeb in the functionally important LDL receptor repeat region. This study, combined with the work presented in *Cell*, should further unravel the complexities and evolutionary origins of specialized tissue development.

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References and links

ORIGINAL RESEARCH PAPER Weiss, J. B. *et al.* Jelly belly: a *Drosophila* LDL receptor repeat-containing signal required for mesoderm migration and differentiation. *Cell* **107**, 387–398 (2001)

FURTHER READING Campos-Ortega, J. A. & Hartenstein, V. in *The Embryonic Development of* Drosophila melanogaster. 2nd edn (Springer, Berlin, 1997)

IN THE NEWS

BSE blunder

The reputation of British science took a massive knock from the revelation that a five-year study investigating whether BSE could jump the species barrier to the UK's sheep flock has had to be scrapped as scientists had actually been testing samples from cow's brains.

The error was revealed by genetic tests carried out on the material as a precaution before the results of the study were released. The UK Government had stated that the UK's entire sheep flock would be slaughtered if the tests proved positive.

"Scientists in France and Germany are thinking, what is British science up to? They cannot tell cattle from sheep", Professor David King, the UK government's chief scientific advisor, who recommended carrying out the genetic tests, told *Reuters* (18 October 2001).

The reason for the mix-up may be as simple "as missing out a 'b' on a container — so that 'bovine' became 'ovine' ", revealed *The Guardian* (20 October 2001).

The Environment, Food and Rural Affairs Secretary, Margaret Beckett — who was accused of trying to suppress the mistake by disclosing the error in a low-key statement — admitted, "The finding that there was no sheep material in the samples sent to the DNA laboratory was a totally unforeseen development" (*The Daily Telegraph*, 23 October 2001).

But Beckett insisted that research work would continue and the public would be told the full facts. A 48-hour test for BSE in sheep will now be used to establish conclusively whether the disease is in the UK's sheep flock.

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