slow as molasses: a new gene in *Drosophila*

The stately progression of cell division in an early embryo is familiar to us from video footage used in countless biology classes. But this image does not portray the importance of cell location and movement. The embryo could divide indefinitely, but without spatial information it would remain an undifferentiated blob. Something must break the symmetry to enable development of a front end, a back end, and everything in between. Some species use an environmental cue (gravity), some use a physical cue (sperm entry point) but, more commonly, they use a chemical cue to initiate asymmetry and form axes of differentiation.

Spatial information is particularly important for cells that migrate during embryo development. In *Drosophila* eggs, the nuclei divide, but no cell walls form, producing a syncitium (polynucleate cell) that persists for the first few hours of embryogenesis. The nuclei migrate to the periphery of the embryo, where cell walls grow around them. How do the cells know where to go? Chemical cues have been identified that guide cells to their destination (and indeed can attract cells in the wrong direction if mis-expressed). A new gene has been identified in *Drosophila* that produces a germ cell guidance signal, active in the earliest stages of embryogenesis [1]. When this new gene is missing, germ cells fail to traverse the embryo to reach the developing gonad in time, and are left in the midgut (so, although the unfortunate mutants survive to wriggle around as larvae, they presumably never have offspring of their own). This is not because the germ cells themselves are deficient, but because of the lack of a guidance system that tells them where to go. Similar to other early embryonic signalling genes, this gene is expressed both maternally and zygotically. It is also responsible for cellularization, which turns the syncytial bag of nuclei into a multicellular embryo.

Counter to common sense, new genes are not named for what they do, but for what happens when they are not expressed. Because of the slow progress of germ cells in these mutants, this gene has been named *slow as molasses* (*slam*). Development has been the missing link in evolutionary biology, the 'black box' between genotype and phenotype, but new techniques are uncovering developmental processes at such an astounding rate that it is almost impossible to keep up with new discoveries. The penchant of developmental biologists to give novel genes slightly silly names might simply reflect a field where discovering a new gene has become an almost daily activity. At least if you call a gene *makes caterpillars floppy* (*mcf*), there is no doubt about what it does [2].

1 Stein, J.A. *et al.* (2002) Slow as molasses is required for polarized membrane growth and germ cell migration in *Drosophila*. *Development* 129, 3925–3934
2 Daborn, P.J. *et al.* (2002) A single *Photorhabdus* gene, *makes caterpillars floppy* (*mcf*), allows *Escherichia coli* to persist within and kill insects. *Proc. Natl. Acad. Sci. U. S. A.* 10.1073/pnas.102068099

Lindell Bromham
L.D.Bromham@sussex.ac.uk

Ups and downs of wildlife population regulation by macroparasites

Whilst many empirical studies have quantified the negative impact of parasites on host vital rates (survival, fecundity, etc.), few have come close to demonstrating that macroparasites are capable of regulating wildlife host populations. Arguably the only convincing demonstration of the regulatory effect of macroparasites on a wildlife host comes from the red grouse–*Trichostrongylus tenuis* system in northern England, where the impact of macroparasites was demonstrated at both the individual and population levels. However, even this study is not without its critics. Now, a new study adds additional weight to the idea that macroparasites can be important regulating agents [1].

Albon *et al.* [1] describe new analyses that suggest that gastrointestinal nematodes are the main regulatory influence on the population dynamics of Svalbard reindeer *Rangifer tarandus plathyrynchus*. They used a combination of long-term monitoring over a six-year period, experimental manipulations and sophisticated statistical analyses to show that, although parasites do not appear to have a major effect on host survival, they do have a significant negative impact on the probability of female reindeer becoming pregnant. As parasitism (and hence parasite-mediated fecundity reduction) is host density-dependent, the parasites have the potential to regulate the population dynamics of their hosts. To test this idea further, Albon and colleagues constructed a Leslie matrix model parameterized with field data. Simulations of this model predicted that, in the presence of the parasites, the reindeer population would exhibit stable population dynamics, whereas in their absence, the population would grow unchecked. These results are consistent with the idea that the nematode parasites are regulating the population dynamics of their hosts. Interestingly, this analysis suggests that population regulation is achieved via a relatively small density-dependent reduction in reindeer fecundity; removing gut parasites with anthelmintics increased calf production by just 5–14%. Albon *et al.* argue that this effect is possible only because of the inherently low population growth rate of the reindeer (1–5%), which is constrained by the negative impact of winter precipitation on the production of calves.

This study has made a significant contribution to the debate concerning the role of macroparasites in regulating natural populations of wildlife hosts. Albon and colleagues have shown that the nematodes impact on their hosts at the individual level and this, in theory, is sufficient to regulate the host population dynamics. However, they recognize that they still have some way to go before they can demonstrate conclusively that macroparasites are necessary and sufficient for population regulation. Ultimately, of course, population-level experiments are required, but these are inherently difficult and expensive. However, it is only via long-term, large-scale studies that population-level questions such as these can be addressed adequately.

1 Albon, S.D. *et al.* (2002) The role of parasites in the dynamics of a reindeer population. *Proc. R. Soc. Lond. Ser. B.* 269, 1625–1632

Ken Wilson
ken.wilson@stirling.ac.uk

