IN THE NEWS

UK public reject GM crops
The vast majority of the United Kingdom public are frightened of genetically modified (GM) crops, according to the 'GM Nation?' report presented to the government on 24 September 2003.

Chair of the GM debate, Malcolm Grant, said the overwhelming response to GM was one of "concern and scepticism" (*BBC News*). "The GM debate reflected a weakening in the faith in the ability or even the will of any government to defend the interest of the general public" he said (*Sydney Morning Herald*).

Strangely enough, on the same day that GM crops were taking a battering in the United Kingdom media, there was some good news for GM advocates in Brazil, where Vice President Jose Alencar announced that the ban on transgenic crops was lifted (*The New York Times*).

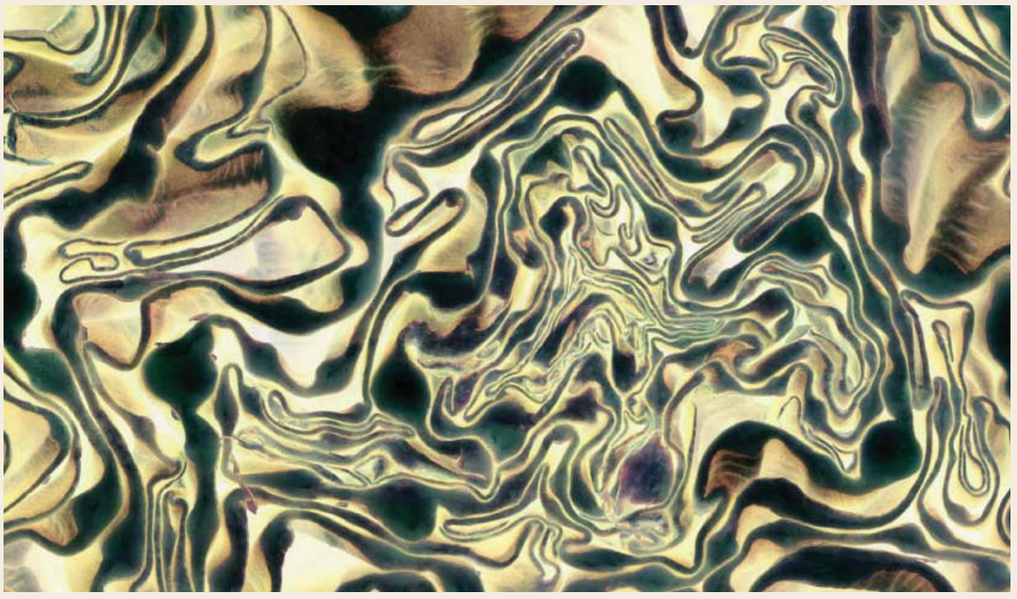
However, the United Kingdom news just seemed to get worse for supporters of GM crops. Buoyed by the results of the GM debate, an environmental group claimed to have signed up hundreds of people online to a pledge to pull up any future commercial plantings of GM crops (*The Guardian*).

Almost at the same time, Bayer announced their decision to halt United Kingdom trials of GM plants until conditions were "more favourable" (*The Observer*).

So, things are not looking comfortable for the United Kingdom Government and for GM's strongest political advocate, Prime Minister Tony Blair. The 'GM Nation?' report was a blow to his five-year mission to introduce GM agriculture (*The Independent*).

Now that the field trials that originally delayed the GM decision look like providing plenty of ammunition for GM opponents (*The Guardian*), it is hard to see the government pushing through GM crops in the face of an unreceptive public.

Nick Campbell



DEVELOPMENTAL GENETICS

Interfering with development

Adding to the growing evidence for the biological significance of RNA interference (RNAi), two recent reports in *Nature Genetics* show that Dicer, an enzyme that cleaves longer RNAs to produce microRNAs and small interfering RNAs (siRNAs), has an essential function in development. Ronald Plasterk and colleagues, and Gregory Hannon and colleagues, show that knocking out Dicer in zebrafish and mice results in early embryonic arrest.

When RNAi was first discovered, most thought that its main purpose was to act as a defence against viruses and transposition. However, increasing numbers of studies are showing that components of the RNAi machinery are involved in essential cell functions.

Using an *in vivo* recombination strategy, Hannon and colleagues targeted Dicer's first RNase III catalytic domain to render the enzyme inactive in mice. Indeed, the authors found that an equivalent mutant form of human DICER1 was unable to cleave siRNA precursors in tissue culture. The development of *Dicer1*$^{-/-}$ mice was arrested by the E7.5 stage. The absence of brachyury expression — a marker of the primitive streak — in *Dicer1*$^{-/-}$ embryos indicates that the mutants arrest before the body plan is laid down during gastrulation.

Components of the RNAi machinery have been implicated in the maintenance of pluripotency in plant and invertebrate development. The fact that, despite many attempts, these authors failed to isolate *Dicer1*$^{-/-}$ embryonic stem (ES) cells indicated that Dicer might be essential for stem cell maintenance. They used Oct4 — a regulator of ES cell maintenance and proliferation — as a marker to look for stem cells in *Dicer1*$^{-/-}$ mice. So, the drastic reduction in *Oct4* expression in these embryos indicated that they had lost stem cells, and points to a potential cause of their lethality owing to stem cell depletion. Whether Oct4 is a direct target of Dicer1 remains to be seen.

Developmental effects of Dicer might be mediated by microRNAs (miRNA) — it has recently been reported that there might be stem cell-specific microRNAs in the mouse. Technical difficulties prevented the authors from testing whether miRNA levels declined in *Dicer1*$^{-/-}$ embryos. Nonetheless, that is exactly what Plasterk and colleagues observed in zebrafish in which *dicer1* had been knocked out.

Mutant *dicer1* zebrafish embryos become sluggish and growth begins to arrest at 8 days post fertilization (dpf), and by 15 dpf all embryos die. The effects are seen earlier in development when *dicer1* is knocked down using morpholinos, probably because the maternal contribution is also knocked down in this case. Using Northern blot analysis, the authors showed that miRNAs build up during the first days of development, and although they persist in wild type and heterozygous fish, they decline in the homozygous mutants.

The role of Dicer in development has now been established beyond any doubt, and it is probably mediated through miRNAs. The challenge will be to identify the relevant miRNA species and ascribe individual functions to them.

Magdalena Skipper

References and links

ORIGINAL RESEARCH PAPERS Wienholds, E. *et al.* The microRNA-producing enzyme Dicer1 is essential for zebrafish development. *Nature Genet.* 5 October 2003 (10.1038/ng1253) | Bernsten, E. *et al.* Dicer is essential for mouse development. *Nature Genet.* 5 October 2003 (10.1038/ng1251)

FURTHER READING Hannon, G. J. RNA interference. *Nature* **418**, 244–251 (2002) | Houbaviy, H. B. *et al.* Embryonic stem cell-specific microRNAs. *Dev. Cell* **5**, 351–358 (2003)