("The Biology of DNA", 26 February–2 March, Cold Spring Harbor Laboratory).

The European scientific celebrations will be launched with "Nobel Day" at the World Life Sciences Forum (8 April, Lyons, France), which boasts no fewer than 11 Nobel prize winners, including Watson, and covers the entire breadth of the discovery's impact. Watson also finds time to attend a genome-focused symposium ("From Double Helix to Human Sequence — and Beyond" 14–15 April, Bethesda, USA), a Royal Society discussion meeting ("Replicating and Reshaping DNA", 23–24 April, The Royal Society, London, UK) and a conference held on the exact anniversary of the famous publication ("DNA: 50 years of the Double Helix", 25 April, Cambridge, UK).

Following the frenzy of double-helix related activity in April, the programme of scientific events slows down, but continues for the rest of the year. The annual Cold Spring Harbor Symposium this year celebrates both the anniversary of the double helix and the impending completion of the human genome ("The Genome of *Homo sapiens*", 27 May–3 June). The International Congress of Genetics — a flagship event for the community that is only held once every five years — also has a genomic flavour ("Genomes — The Linkage to Life", 6–12 July, Melbourne, Australia).

Meetings in many other disciplines on which genetics has had an impact on in the past 50 years will be hosting symposia or discussions for the anniversary; for example, the symposium on "Exploiting Genomes: Bases to Megabases in 50 years" at the Society of General Microbiology's meeting (8–9 September, Manchester, UK).

Biotechnology is another area that owes a debt to the double helix, and later in the year a symposium at UC Berkeley (10–11 October, San Francisco, USA) will explore its impact over the past 50 years (the ubiquitous Watson will be in attendance!).

So, it will be a busy year for geneticists world-wide, but, as I'm sure most attendees at these meetings will agree, while it must have been great for Watson and Crick to lay the foundation stone, it is also good to be a humble bricklayer on a construction as exciting as this one.

Nick Campbell

FlavrSavr tomato — reached the supermarkets in 1994; and 'Dolly the sheep' — the first cloned mammal — was born in 1996.

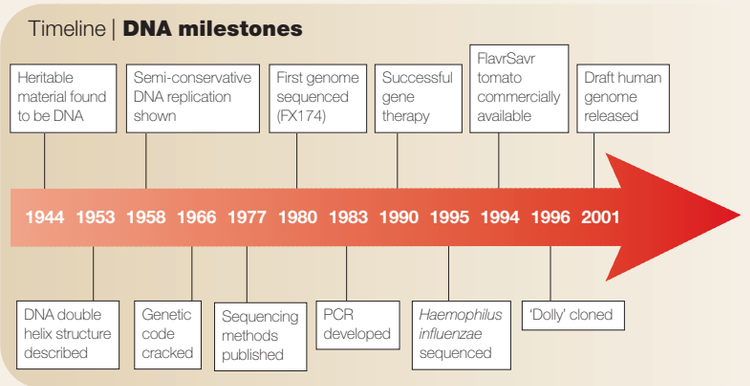
Combining molecular biology techniques with the ever-expanding volume of genomic, proteomic and phenotypic data should enable geneticists to make further exciting developments over the next 50 years. There is little doubt that genetics will continue to benefit society, in particular through improvements in healthcare and agriculture.

Catherine Baxter

References and links

FURTHER READING

The double helix – 50 years. *Nature* **491**, 396–453 (2003)



IN BRIEF

GM ORGANISMS

Impact of genetic manipulation on the fitness of *Anopheles stephensi* mosquitoes.

Catteruccia, F. *et al. Science* **299**, 1225–1227 (2003)

It has been argued that malaria could be controlled by introducing into natural populations transgenic mosquitoes that express genes that impair parasite transmission. For this strategy to be successful, the transgenic mosquitoes must be able to survive and reproduce competitively in the wild . However, this study shows that transgene expression, mutations introduced by transgene insertion, and inbreeding can result in a lower fitness of transgenic mosquitoes relative to wild type.

MOUSE MODELS

Modification of ocular defects in mouse developmental glaucoma models by tyrosinase.

Libby, R. T. *et al. Science* **299**, 1578–1581 (2003)

Human primary congenital glaucoma (PCG) is often caused by mutations in the cytochrome P450 family member *CYP1B1*, and is associated with abnormal ocular drainage structures. This paper shows that *Cyp1b1*-deficient mice provide a good model for this type of glaucoma. Libby *et al.* used these knockout mice to show that tyrosinase gene deficiency increases the severity of the disease phenotype and that this is alleviated by applying dihydoxyphenylalanine (L-dopa). This raises the possibility of new glaucoma therapies.

TECHNOLOGY

RNA interference targeting *Fas* protects mice from fulminant hepatitis.

Song, E. *et al. Nature Med.* **19**, 347–351 (2003)

RNAi can target and silence mammalian genes but can it prevent disease? Song *et al.* show that, in mice, RNAi can silence the gene *Fas* that codes for an important mediator of hepatocyte apoptosis. This indicates that RNAi could be used to prevent the adverse effects of hepatitis that are linked to cell death. The authors test this hypothesis in two models of *Fas*-mediated liver damage, and show for the first time that siRNA can prevent disease *in vivo*.

FUNCTIONAL GENOMICS

Scanning the human genome with combinatorial transcription factor libraries.

Blancafort, P. *et al. Nature Biotech.* **21**, 269–274 (2003)

Blancafort *et al.* report a new technology that could be useful for studying and modulating gene function. They have constructed large libraries of artificial transcription factors that contain between three and six zinc-finger domains (TF_{ZF}s) that can either activate or repress gene expression. TF_{ZF}s can be applied to a cell line that is then screened for a desired phenotype. In this example, TF_{ZF}s were identified that were able to induce expression of the endothelial marker VE-cadherin in non-endothelial cell lines and to repress its expression when combined with a repression domain.