exception of two, one of which gives rise to a very unstable message, and to a product that does not get into collagen molecules and cannot therefore exert a disruptive influence; the resulting phenotype is mild. In $\alpha 2$, only two lethal skip mutations have been found. The boundary changes are usually straightforward single-base substitutions, but sometimes the splice sites are removed as part of a longer deletion (A. Nicholls, Clinical Research Centre, London; A. Superti-Furga, University of Zurich).

As if to reassure traditionalists, two splicing mutants were described that did behave like globin. A mutation in the donor site of *COL1A1* intron 24 leads not to skipping of the upstream exon but to retention of the mutated intron in the processed RNA (D. Rowe). The rationale was that intron 24 is short enough for the donor site beyond exon 25 to be within reach, so that the whole stretch (exon 24, intron 24 and exon 25) is defined as a single exon and not spliced out. The RNA is unstable and is retained in the nucleus, so the phenotype is mild. For $\alpha 2$, an alternative donor site is close enough to be activated when the normal site is mutated, leading to an 18-bp insertion in the mRNA. This time the message is stable and cells synthesize an $\alpha 2$ chain with six non-helical residues between positions 585–586 (R. Wenstrup).

There were several nice touches outside the 'big two' mechanisms of glycine substitution and exon skipping. In one severe non-lethal case a 3-bp deletion removed the codon for $\alpha 1$ valine 255 (K. Molyneux, University of Leicester). Downstream folding of the helix should be out of phase in this mutant. Another deletion removed a single Gly-Ala-Pro triplet from $\alpha 1$ exon 43, presumably allowing in-phase helix folding to continue to the N terminus but nonetheless resulting in a lethal phenotype (G. Wallis, University of Washington, Seattle).

Although these examples show convincingly that there is enough potential within the structure of the two collagen genes for mutations to express the entire range of phenotype, the most interesting mechanism for introducing variation, and one with far-reaching implications for genetics in general, came from the study of sibling recurrences in the lethal form of OI. Lethal OI was originally thought to be an autosomal recessive disease because of the occurrence of multiply affected siblings among the offspring of unaffected parents. Over the years a few pedigrees came to light in which there were affected half-sibs (that is, siblings with only one parent in common). The isolation of the causal mutation showed that they were due to the same sort of dominant mutations as the isolated cases and that the common parent was a germ-line mosaic. It now turns out that, for each of the eight cases of recurrences in sibs and half-sibs that have been studied, one parent is not only a germ-line but also a somatic mosaic (D. Cohn, Cedars-Sinai, Los Angeles) with the mutant allele present in up to 80 per cent of lymphocytes and 100 per cent of cultured skin fibroblasts. Because the probability of recurrence is a function only of the extent of germ-line mosaicism, it is not surprising that a parent of a sporadic case could also be mosaic and in one case ($\alpha 1$ Gly–Cys 904) the mother had the features of mild OI but, or so it appeared, only on her left side (C. Constantinou, Jefferson Medical College, Philadelphia). Nor is the phenomenon restricted to the lethal form. Five recurrences due to parental mosaicism were reported in severe or mild phenotypes (G. Wallis).

The conclusion has to be that the mutations occurred not in the maturation of the germ line but very early on in the embryonic development of the mosiac parent, before the segregation of soma and germ line. This is almost bound to be a general phenomenon because there is nothing special about collagen mutations except that they are frequent and produce an easily ascertainable dominant phenotype. If the study of osteogenesis imperfecta has pushed back the origin of mutation in general to the earliest cell divisions, then perhaps marrying the girl next door was a good idea all along. □

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2. Alo-Kokko, L. *et al. Proc. natn. Acad. Sci. U.S.A.* **87**, 6565–6568 (1990).
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Errata

■ In "The changing shape of actin" by David J. DeRosier (*Nature* **347**, 21–22; 1990) the first full sentence on the second page was meant to read: ". . . the authors were able to . . . locate the myosin binding site on the *small* domain of the actin monomer".

■ David Blow and Peter Brick of Imperial College London should have received full acknowledgement for kindly providing the picture of the structure of tyrosyl tRNA synthetase (**347**, 231).

■ In C. H. Llewellyn Smith's obituary of J. S. Bell (**347**, 514), the opening sentence should have stated that Bell "also made many major contributions to accelerator physics, many-body theory — especially as applied to nuclei — quantum field theory and particle physics".

■ The final sentence of the penultimate paragraph of Fady Malik and Ron Vale's article "A new direction for kinesin" (**347**, 713–714) should have run: "Although thought to operate by different mechanisms, perhaps there are similarities between these prokaryotic and eukaryotic motors that we have yet to appreciate". □

DAEDALUS

Damming the blast

We frequently read nowadays that some luckless car or briefcase has been destroyed by the police in a 'controlled explosion'. There is, of course, no such thing. Once you light the blue touch-paper, events run entirely out of control. Daedalus is now developing a genuine technology of controlled explosions.

In a successful explosion, he points out, each small section of exploding material transfers enough energy to the adjacent unexploded material to set it off as well. Almost all combustion generates charged molecular fragments, so some of this energy will be in the form of hot, fast-moving ions. A high electric field across an explosive might greatly increase its sensitivity and propagation rate, by driving these ions forcibly into the adjacent unreacted material. Conversely, a reversed field would drive them the wrong way, weakening the propagation of the blast. So Daedalus is wiring up sticks of dynamite to a Wimshurst machine, and studying the power and completeness of the subsequent blast as a function of the size and polarity of the applied field. Dynamite may not be ideal for this job. DREADCO's chemists are devising explosives based on caesium perchlorate, which should explode in a more ion-rich, field-sensitive manner.

This exciting research should lead to a truly controllable, field-sensitive explosive. Set off in the normal way, it will be quenched instantly by cutting off or reversing the electric field applied to it. Piezoelectric sensors attached to the object to be blown up, will integrate the acceleration or displacement caused by the blast. When exactly the right amount of force has been applied, electronic controls will reverse the field across the explosive, and just switch it off. They may even switch it on again a microsecond or so later, or modulate it into a programmed series of timed pulses. The relatively slow explosion will be completely controlled by the ultrafast electronics.

Security officers will welcome the new technique. They will be able to give a suspect package just enough shock to set off a possible hidden detonator, with no excessive damage. Tricky engineering operations like righting overturned lorries, lifting locomotives back on the rails, easing buildings apart to widen an urban bottleneck, nudging inconvenient statues of Lenin into side alleys, even blowing a safe without needless violence, all will be possible. But at this time of year, Daedalus is using the principle to invent electronically controlled fireworks. The DREADCO dot-matrix rocket, its nine parallel thrusters switching on and off rapidly under program control, will write a message in the sky, while the circular-scan catherine wheel will present a little video drama in fast-modulated fire.

David Jones