Table 1 Preliminary restrained least squares refinement data for the porcine pancreatic elastase complex with I

	Target standard deviation	Initial model	Final refined model
R-factor*		33.2%	22.8%
Deviations from ideality†			
Bond distance (Å)	0.020	0.016	0.026
Angle distance (Å)	0.030	0.036	0.045
Planarity (Å)	0.040	0.051	0.055
Resolution range (Å)		10.0–2.5	10.0–1.84
No. of reflections		6,887	14,135
No. of atoms		1,841	1,913

The Hendrikson–Konnert program[16,17] was used, as modified by E. Fluder for the IBM 3090.

* The *R*-factor is defined by the expression:

$$\Sigma||F_o|-|F_c||/\Sigma|F_c|$$

where $|F_o|$ are the observed structure factors and $|F_c|$ are the corresponding calculated structure factors.

† Root mean square deviations from ideal values. These refer to a dictionary of standard groups as specified in Table 2 of Sielecki *et al.*[17].

‡ Recommended[17] estimates of standard deviations which are used to determine the relative weights of restraints.

sporins react differently with their corresponding β-lactamases[5], and this behaviour seems to carry over to the cephalosporin sulphone elastase inhibitors such as I. With a stable substituent at the 7-position instead of a relatively good leaving group, however, ring opening might yet be favoured, leading to novel rearrangements and reactions. The 7′-β substituents in the cephalosporin antibiotics are thought to form part of a mimic of the backbone of the D-alanyl-D-alanine-containing substrates of the enzymes of bacterial cell wall synthesis[15]. As such, these substituents can be quite long. In the elastase inhibitors, 7′-α substituents must be short, because they correspond in turn to the side chain of L-amino acid substrates, and must fit the shallow S1 specificity pocket of the enzyme. Given the structure of the complex, 7′-β substituents would not point into the pocket, and as expected, such isomers were weak or inactive when compared to their 7′-α analogues[1].

In conclusion, our elastase inhibition programme has produced a series of selective β-lactam inhibitors which provide a model system for the observation of β-lactam chemistry in action. This system has both significant similarities and differences with the more familiar antibiotic targets. Not surprisingly, conventional β-lactam antibiotics and their analogues do not inhibit elastase and conversely, the β-lactam elastase inhibitors do not act as antibiotics[1]. We hope that by examination of a series of compounds in this class, we will be able to clarify and improve upon the beneficial effects of the β-lactams in therapeutic environments.

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Corrigendum

Evidence for carbonate in the mantle

G. W. Berg

Nature **324,** 50–51 (1986)

IN this letter reference should have been made to three examples[1–3] rather than just a single instance[1] of minute inclusions of dolomite in samples of rock from the mantle ever having been reported despite 25 years of intensive study of these rocks. The author thanks Drs J. V. Smith and D. Smith for pointing out these omissions.

Erratum

A genetic pathway for the specification of the vulval cell lineages of *Caenorhabditis elegans*

Edwin L. Ferguson, Paul W. Sternberg & Robert Horvitz

Nature **326,** 259–267 (1987).

IN Table 2 of this article, the lines referring to mutants *lin-17* and *lin-18* should appear beneath *lin-11* in the 'Expression mutants' section, not under 'Determination mutants'. Also in Table 2, a line referring to 'Cell fates' for the wild type was omitted, and it should read:

	Cell fates							
	P3.p	P4.p	P5.p	P6.p	P7.p	P8.p	P(1, 2, 9–11).p	No.
Wild type	3°/S	3°	2°	1°	2°	3°	S	—

Finally, in Table 2 in the line referring to mutant *lin-12(0)*, the alleles used were *n137* and *n720* (the solidus, /, is misleading in the printed version).

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