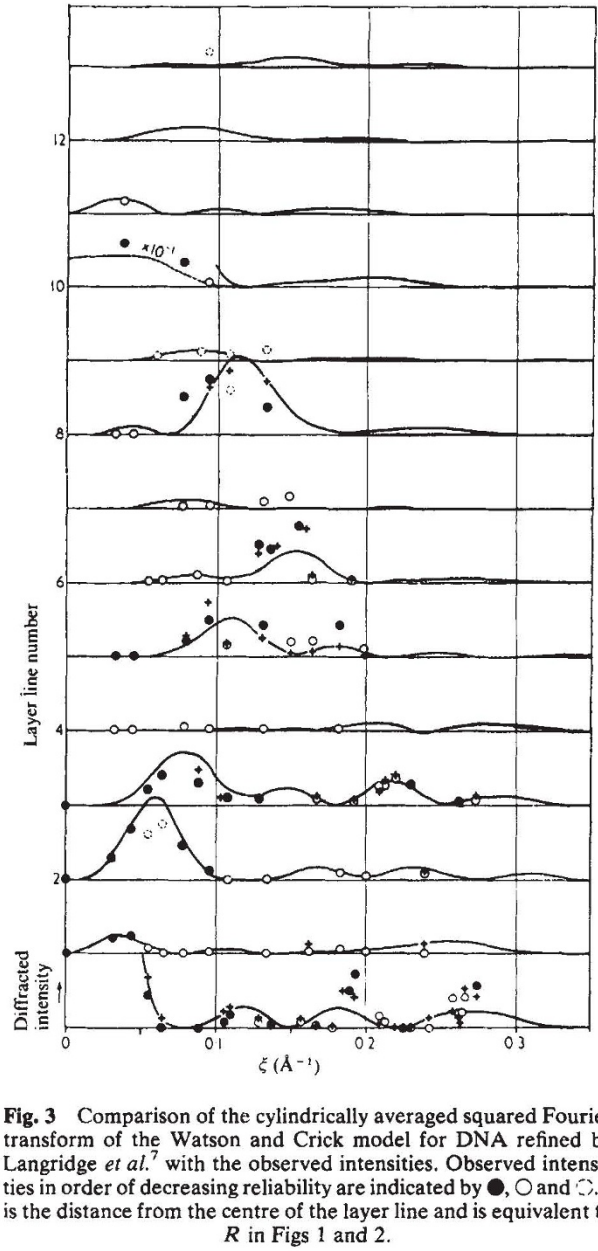

Fig. 3 Comparison of the cylindrically averaged squared Fourier transform of the Watson and Crick model for DNA refined by Langridge *et al.*[7] with the observed intensities. Observed intensities in order of decreasing reliability are indicated by ●, ○ and ◌. ξ is the distance from the centre of the layer line and is equivalent to R in Figs 1 and 2.

Watson and Crick models, the differences are not large and for both models the calculated diffraction is similar to that observed. Even the most superficial examination of Figs 1–3 reveals that the diffraction calculated for the two SBS models differs markedly from that observed.

A particularly serious deficiency of the original SBS model is the occurrence of diffraction on layer planes which are neither observed nor predicted by the Watson–Crick models, that is on $l = 8$, 12, 18, 22, 26, 78 and 88 in Fig. 1. There is no doubt that the quality of the X-ray diffraction patterns from crystalline fibres of LiDNA is sufficient for the original SBS model to be eliminated on the basis of the non-observation of these layer planes. The SBS model also predicts substantial meridional diffraction on layer planes $l = 60$, 70 and 90 (corresponding to $l = 6$, 7 and 9 in the normal B-DNA nomenclature). This is also at variance with the observed diffraction. In addition to predicting diffraction which is not observed, the SBS model fails to predict the strong diffraction which is observed on layer planes $l = 10$, 20, 30, 50, 60 and 80 ($l = 1$, 2, 3, 5, 6 and 8 in the normal nomenclature).

It can be seen from Figs 2 and 3 that while the distorted SBS model only predicts intensity on those layer planes for which diffraction is actually observed, the magnitudes of these predictions are seriously in error. Specifically, substantial meridional diffraction is predicted but not observed on layer planes $l = 6$, 7 and 9. Furthermore, the distorted model is seriously in error in its prediction of the relative intensity of diffraction on the various layer planes. These discrepancies are particularly serious on $l = 2$, 3 and 8 where the overall calculated intensity is much less than is observed and on $l = 4$ for which substantial diffraction is predicted where the observed intensity is essentially zero.

We may conclude that there are major and quite unacceptable discrepancies between the observed diffraction from the B form of DNA and that calculated for the SBS model proposed by Rodley *et al.*[2]. Although some of the more serious discrepancies can be removed by the simple distortion of the original model described here, the degree of agreement between observed and calculated diffraction is still very poor and very much inferior to that reported for the best models of the Watson–Crick type. It should also be emphasised that DNA can change reversibly within a fibre from a conformation that gives the semicrystalline B pattern to one that gives a fully crystalline A pattern[11]. This change is easily explained in terms of the Watson–Crick model by an increase in the number of nucleotide pairs per pitch from 10 to 11. Even if an SBS model could be found to account for the B diffraction data, it would also need to be capable of undergoing a stereochemically plausible transition to account for the A diffraction pattern.

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Errata

The authorship of the article 'Crust of oceanic affinity in Iceland', *Nature* **281**, 347 (4 October) emerged incorrectly as a result of a misunderstanding between us and our typesetters on the telephone. It should have read:

Scientific Party, Iceland Research Drilling Project (I. L. Gibson, editor).

We regret this error.

In the letter 'Chlorophyll and nitrate fine structure in the southeastern Bering Sea shelf break front' by R. L. Iverson *et al.*, *Nature* **281**, 664–666, the units mentioned in the legends to Figs 3 and 5 should read: (μg atoms NO_3–N l^{-1}).

In the obituary to Dr Scott Mazzur, *Nature* **280**, 708, the new antigenic marker mentioned in the penultimate paragraph is the 'l' antigen, not the 'e' antigen as shown.