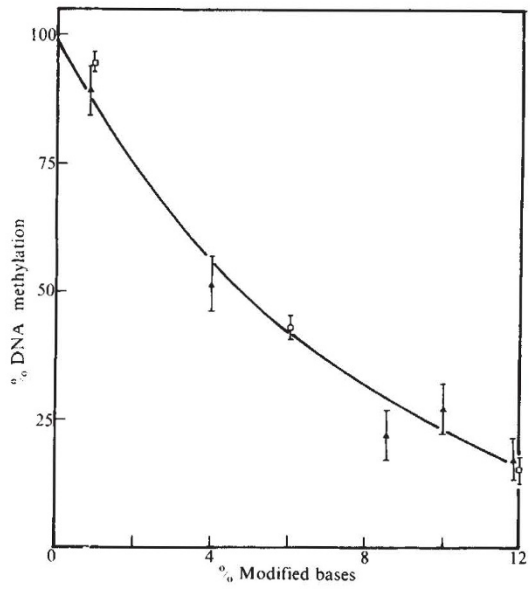

Fig. 2 The methylation of *in vitro* modified DNA-AAF: *N*-AcO-AAF was synthesised by a modification of the original procedure[29] consisting of direct hydrogenation and acetylation of 2-nitrofluorene[18]. The purity of the *N*-AcO-AAF obtained was checked by thin-layer chromatography and the final product was stored at −20°C as alcoholic solution. DNA bound to the carcinogen was prepared as described elsewhere[14]. The *in vitro* methylation reaction of carcinogen modified chicken erythrocyte or *Micrococcus lysodeikticus* DNAs were done as described in the legends to Fig. 1. The results are expressed as percentage relative to control (100%) determined with non-modified DNA. Blank values were subtracted in each case. ▲, Chicken erythrocyte DNA; □, *Micrococcus lysodeikticus* DNA. Averages with s.d. are from four experiments made in triplicate except for the experiments with 11, 3 and 13% modified bases, which are in duplicate.

Arylamidation of the C8 of guanine by some aromatic amines induces a rotation of the bases involved in the double helix, altering its normal conformation and carrying a local destabilisation of the DNA[13,19]. As shown previously, this local denaturation may change some biological properties of nucleic acids by inducing cell transformation[10], premature termination of transcription at (or near) the site of DNA modification[20] and more recently *in vitro* inhibition of DNA synthesis[21]. Our results show that modification of guanine residues in position 8 impairs another biological property of the double helix.

The carcinogen mediated effect on DNA methylation can be explained as follows: binding of methyltransferase near or at the position of methylation in DNA may be hampered by the presence of carcinogen-modified guanines paired to cytosine residues susceptible to methylation. Consequently, DNA methylation is now restricted to cytosine residues paired to guanines which are recognised by the enzyme but which are not modified. Alternatively, it can be proposed that binding of the enzyme to DNA is not affected by the presence of carcinogen, but, subsequent walking of the enzyme on the DNA helix is interrupted by the carcinogen. The last alternative agrees with the proposed mechanism of action described for DNA (cytosine-5) methyltransferase[22–24] as well as with the model explaining the premature termination of DNA transcription in DNA-AAF[20].

Further work is required to establish whether DNA undermethylation caused by aromatic amides *in vitro* can also be obtained *in vivo*, and to demonstrate the effect of DNA hypomethylation on cell differentiation and/or carcinogenesis.

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Corrigenda

In the letter 'Speciation of dissolved iodine in estuarine waters' by J. D. Smith and E. C. V. Butler, *Nature* **277,** 468–469, the values for iodine in the Yarra River estuary in the text and Fig. 1 should all be multiplied by 0.73.

In the letter 'Opposing effects of tumour promoters on erythroid differentiation' by R. M. Miao *et al.*, *Nature* **274,** 271 (1978), all concentrations of 12-*O*-tetradecanoyl-13-acetate (TPA) should be 1,000-fold lower than stated; for example, 10^{-7} M should read 10^{-10} M.

Errata

In the letter 'Immunological properties of the surface of parasitic nematodes' by C. D. Mackenzie, P. M. Preston and B. M. Ogilvie, *Nature* **276,** 826–828 (1978), the following acknowledgement was omitted: 'P. M. Preston was financed by a grant from the World Bank/UNDP/WHO Special Programme for Research and Training in Tropical Diseases.'

In the letter 'Measurement of carbon tetrafluoride in the atmosphere' by R. A. Rasmussen *et al. Nature* **277,** 549–551, for ‰ read p.p.t. (parts per 10^{12}), and in paragraph 8, line 8, for p.p.b. read p.p.t.