fragments and its formation in the absence of homologous fragments was even greater, the former being 30-fold larger (M.I. and T.S., unpublished observation). However, the formation of form X DNA does occur at a very low rate in the absence of single-stranded DNA and is slightly stimulated by the addition of heterologous single-stranded fragments, but only if double-stranded DNA substrate is form I DNA (Fig. 2*b* and Fig. 3, lanes 15, 16). When double-stranded DNA substrate is relaxed DNA (form IV DNA), the formation of form X DNA seems to require homologous single-stranded fragments (T.S. M.I. and T.O., unpublished observation, and A. M. Wu, and C. M. Radding, personal communication). These results suggest that the unwinding of the double helix by RecA protein might be initiated at all D-loop sites and also initiated at a very low rate at the transient denatured sites in superhelical DNA[31–33]. The initiation at the latter sites is stimulated by any single-stranded DNA (data not shown) as the unwinding in the presence of ATPγS (ref. 23).

The enzymes which catalyse unwinding of the double helix in the presence of a high-energy cofactor such as ATP are called DNA helicases, and include DNA helicase I, II (refs 34–36) and III (refs 37, 38) from *E. coli*, *E. coli* Rep protein[39–42] and ATP-dependent DNases from *E. coli*[43–45] and *Haemophilus influenzae*[46]. As a DNA helicase, RecA protein is unique; it unwinds closed-circular DNA without strand termini, whereas all other DNA helicases require a single-stranded tail, gap or free end in the DNA to initiate the unwinding of the double helix. Moreover, RecA protein has not been found to initiate unwinding from a single-stranded tail or gap (ref. 47 and R. P. Cunningham, unpublished observation). DNA helicase activity of RecA protein seems to have a role in the elongation of heteroduplex joints by RecA protein *in vitro*[10–12] and in branch migration during recombination *in vivo*[15].

The present study also indicates that closed-circular double-stranded DNA with an extraordinarily large number of negative-superhelical turns can be formed in physiological conditions by the two proteins, RecA protein and topoisomerase I. In eukaryotes in which gyrase activity has not been found, RecA-like protein[48] and topoisomerase I might promote a high degree of supercoiling.

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Errata

In the letter 'Curare can open and block ionic channels associated with cholinergic receptors' by A. Trautmann *Nature* **298,** 272–275 (1982), the symbols in Table 1 representing the different types of transitions observed should be rotated through 180°.

In the letter 'Electrical stimulation of hindlimb increases neuronal cell death in chick embryo' by R. W. Oppenheim & R. Nunez *Nature* **295,** 57–59 (1982), in Fig. 2 the bars representing pooled data should be labelled CE & SE (left-hand bar) and CU & SU (right).

Corrigenda

In the letter 'Differences in the kinetics of rod and cone synaptic transmission' by J. L. Schnapf & D. R. Copenhagen, *Nature* **296,** 862–864 (1982), the plots of the equation shown in Fig. 1 legend (that is, in Figs 1*c*, 2*c* and 3) do not have large enough undershoots. This in turn leads to an overestimate of the decay time of the impulse response. The impulse response actually falls to 1/e its peak value in 80 ms for the rod synapse and 10 ms for the cone synapse (not 131 ms and 16 ms as stated in the second-to-last paragraph). The major conclusions of the paper remain unchanged: the synapses display high and low pass filter characteristics and the rod synapse is approximately 10 times slower than the cone synapse.

In the letter 'Directed effector cells selectively lyse human tumour cells' by C. B. Simone, *Nature* **297,** 234–236 (1982), line 16 on page 236 states that "direct effector cells . . . cause no adverse reactions *in vivo* (mice and one human)." Instead of "no adverse reactions", the author should have stated that the patient did have two fevers during the five day treatment which responded to acetoaminophen. However, no other toxicities were noted. Obviously, a full trial needs to be done to explore the efficacy of directed effector cell treatment.