give more useful numbers. For birds and mammals, this line of analysis implies extinction of half the species within roughly 200–300 years, and for palms it suggests 50–100 years.

We re-emphasize that the data in the Red Lists have been compiled opportunistically rather than systematically, so that these projected rates of extinction probably owe more to the vagaries of sampling effort and data entry than to real changes in the status of species. However, we believe that the approach does have potential merit in suggesting a new line of investigation of the dynamics of species extinction. The consequent estimates of extinction rates for the better-known taxa are of the same order of magnitude as those derived from totally unrelated species–area relations, and they suggest that the time available for research into other plant and animal groups may be severely limited.

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FIG. 2 Modification of the molecule in Fig. 1 by DMS. Lanes 1–3, 20 mM Tris-HCl pH 7.5, 1 mM EDTA plus 50 mM NaCl, 100 mM NaCl and 150 mM NaCl, respectively. Lanes 4–6, 20 mM Tris-HCl pH 7.5, 10 mM $MgCl_2$ plus 50 mM NaCl, 100 mM NaCl and 150 mM NaCl, respectively. The duplex was modified with 0.5% DMS for 25 s at 20 °C in the appropriate buffer. Alkylated DNA was cleaved in 10% hot piperidine and the product analysed in denaturing 15% polyacrylamide gel.

Triplex model of chromosome ends

SIR — Sundquist and Klug[1] demonstrated that synthetic models of *Tetrahymena* chromosome telomeric terminus, consisting of the DNA duplex and the single-stranded 'tail' $(T_2G_4)_2$, stably dimerize in monovalent salts owing to quadruplex formation by the two tails. We have found that in physiological buffer containing about 100 mM Na^+ and 10 mM Mg^{2+} the same model of chromosome ends forms an intramolecular pyrimidine–purine–purine triplex: the single-stranded G-rich overhang folds back, lying into the major groove of the terminal duplex forming the CGG base triads (Fig. 1).This new structure of telomeres provides a possible explanation for the stability of the ends of chromosomes *in vivo* against digestion and recombination.

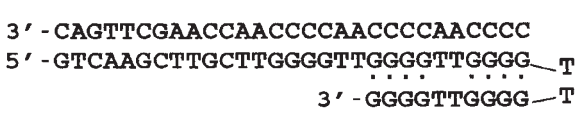

FIG. 1 Synthetic *Tetrahymena* chromosome terminus forming the proposed foldback triplex structure.

Figure 2 presents our data on probing of the structure with dimethyl sulphate (DMS). Without Mg^{2+}, we observe homogeneous modification of guanines along the whole G-rich strand (lanes 1–3). In the presence of Mg^{2+}, a completely different pattern of modification is observed: one can see the clear-cut protection of guanines in two blocks within the duplex adjacent to single-stranded tails (lanes 4–6). Moreover, at least one of two guanine blocks belonging to the tail is also virtually fully protected. Some protection is visible also for the terminal block of the tail.

At 10 °C and in the presence of Mg^{2+}, the molecules move in the gel as a single band with mobility corresponding to the mobility of monomers (data not shown), although we observed formation of dimers described by Sundquist and Klug[1] in experiments without magnesium after long incubation. For the incubation times we used in our experiments in Fig. 2, dimers never form (our unpublished observations and ref. 1). This means that the clear-cut protection effect against methylation in Fig. 2 results from an internal structural rearrangement in the molecule. We therefore assume that in the presence of Mg^{2+} ions the single-stranded tail folds back, forming a pyrimidine–purine–purine triplex consisting of the CGG base triads (Fig. 1).

The model in Fig. 1 explains all major features of the results in Fig. 2 and is fully consistent with the available data on the CGG type of pyrimidine–purine–purine triplexes[2–5]. Formation of the pyrimidine–purine–purine triplex leads to the complete protection of guanines in the duplex against modification by DMS[4,5] and, as we have recently shown, formation of the CGG base triad leads to significant protection of guanines in the third strand[4].

Our foldback triplex model provides a natural explanation for the stabilizing effect of chromosome ends. Occupation of the major groove and concomitant deformation of the minor groove due to triplex formation at the very end of chromosomal DNA should dramatically decrease vulnerability of the ends of chromosomes as targets for enzymatic cleavage. One can also assume that the foldback triplex structure would protect the telomeric ends against digestion by both 5′–3′ and 3′–5′ exonucleases. It is also quite reasonable to assume that the structure in Fig. 1 would prevent the telomeric ends from participating in recombination events.

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