along DNA single-strands[21,22], is also possible. Note that, in this view, the ATP-dependent relaxation of non-origin-containing DNA molecules observed *in vitro* reflects an uncoupled version of a site-specific DNA gyration reaction catalysed by the topoisomerase *in vivo*.

The site-specific gyration model in Fig. 4 postulates that the initial opening of the DNA helix is followed by an RNA-primed DNA chain start, which actually begins DNA synthesis. This first RNA primer could either be synthesised by the same enzymes that prime the Okazaki pieces made on the lagging strand of the T4 replication fork, or be made by a separate mechanism[3,23]. It has been shown recently that the T4 gene *61* protein, in conjunction with the T4 gene *41* protein, can synthesise pentaribonucleotides, and that this synthesis has an absolute requirement for a single-stranded DNA template (ref. 19 and C.-C.L., unpublished results). When additional replication proteins are present, these short ribonucleotides will prime lagging strand DNA synthesis in the T4 *in vitro* system[19]. These two T4 proteins may therefore also have a crucial role in replication fork initiation.

Despite our limited knowledge concerning the mechanism of initiation of DNA replication forks, some similarities can be seen between different systems. As is the case for many other genomes (bacteriophage λ, *E. coli* and eukaryotic chromosomes), replication forks in T4 seem to be able to start out bi-directionally from an initial replication bubble[24]. We therefore expect that a detailed study of the T4 DNA topoisomerase and its interaction with the other T4 replication proteins can provide a model system in which some general properties of fork initiation mechanisms can be elucidated.

Genetic data suggest that genes *O* and *P* of bacteriophage λ may act in a manner analogous to T4 DNA topoisomerase. As for T4 genes *39*, *52* and *60*, the λ *O* and *P* products interact with each other, and when their levels are decreased, the initiation of λ DNA replication is altered[25]. Experiments comparing various lambdoid phages have shown the gene *O* product is highly phage specific, presumably because it recognises origin DNA sequences[26]. Similarly, unlike most T4 genes, gene *39* function cannot be complemented by bacteriophage T2 (W. M. Huang, personal communication); this suggests that gene *39* may have a role in T4 replication which is analogous to the role of gene *O* in λ replication.

Further study of the T4 DNA topoisomerase should also provide valuable knowledge concerning the functions and mechanisms of the important class of enzymes known as DNA topoisomerases. In this respect, we have observed that, at much higher enzyme concentrations (about equal weights of DNA and T4 DNA topoisomerase), a novel ATP-independent DNA topoisomerisation reaction can be detected, in which topologically knotted, double-stranded superhelical DNA molecules are created. Most strikingly, these knots can subsequently be removed by catalytic amounts of the topoisomerase, in an efficient ATP-dependent reaction. As will be described in detail elsewhere, such data reveal that the T4 DNA topoisomerase is working by making transient double-stranded DNA breaks which are rapidly and efficiently resealed in its 'closing' reaction.

This work was supported by USPHS grant GM24020 from the National Institute of General Medical Sciences to B.M.A. and an American Cancer Society postdoctoral fellowship to L.F.L.

Received 21 May; accepted 30 August 1979.

1. Alberts, B. M. & Sternglanz, R. *Nature* **269,** 655–661 (1977).
2. *Cold Spring Harb. Symp. quant. Biol.* **43** (1979).
3. Tomizawa, J. & Selzer, G. *A. Rev. Biochem.* **48** 999–1034 (1979).
4. Mufti, S. & Berstein, H. *J. Virol.* **14,** 860–871 (1974).
5. Yegian, C. D., Mueller, M., Selzer, G., Russo, V. & Stahl, F. W. *Virology* **46,** 900–919 (1971).
6. McCarthy, D., Minner, C., Bernstein, H. & Bernstein, C. *J. molec. Biol.* **106,** 963–981 (1976).
7. Wang, J. C. & Liu, L. F. in *Molecular Genetics* Part III (ed. Taylor, J. H.) 65–88 (Academic, New York, 1979).
8. Stetler, G. L., King, G. J. & Huang, W. M. *Proc. natn. Acad. Sci. U.S.A.* **76** (in the press).
9. Vinograd, J. & Lebowitz, J. *J. gen. Physiol.* **49,** Suppl., 103–125 (1966).
10. Epstein, R. H. *et al. Cold Spring Harb. Symp. quant. Biol.* **28,** 375–394 (1963).
11. McCarthy, D. *J. molec. Biol.* **127,** 265–283 (1979).
12. Gellert, M., O'Dea, M. H., Itoh, T. & Tomizawa, J. *Proc. natn. Acad. Sci. U.S.A.* **73,** 4474–4478 (1976).
13. O'Farrell, P. Z. & Gold, L. M. *J. biol. Chem.* **248,** 5499–5501 (1973).
14. Sugino, A., Peebles, C. L., Kreuzer, K. N. & Cozzarelli, N. R. *Proc. natn. Acad. Sci. U.S.A.* **74,** 4767–4771 (1977).
15. Gellert, M., Mizuuchi, K., O'Dea, M. H., Itoh, T. & Tomizawa, J. *Proc. natn. Acad. Sci. U.S.A.* **74,** 4772–4776 (1977).
16. Sugino, A., Higgins, N. P., Brown, P. O., Peebles, C. L. & Cozzarelli, N. R. *Proc. natn. Acad. Sci. U.S.A.* **75,** 4838–4842 (1978).
17. Wang, J. C. *J. molec. Biol.* **55,** 523–533 (1971).
18. Peebles, C. L. *et al. Cold Spring Harb. Symp. quant. Biol.* **43** 41–52 (1979).
19. Liu, C.-C., Burke, R. L., Hibner, U., Barry, J. & Alberts, B. M. *Cold Spring Harb. Symp. quant. Biol.* **43,** 469–487 (1979).
20. Mosig, G. *J. molec. Biol.* **53,** 503–514 (1970).
21. Abdel-Monem, M. & Hoffmann-Berling, H. *Eur. J. Biochem.* **65,** 441–449 (1976).
22. Scott, J. F., Eisenberg, S., Bertsch, L. L. & Kornberg, A. *Proc. natn. Acad. Sci. U.S.A.* **74,** 193–197 (1977).
23. Backman, K., Betlach, M., Boyer, H. W. & Yanofsky, S. *Cold Spring Harb. Symp. quant. Biol.* **43** (in the press).
24. Delius, H., Howe, C. & Kozinski, A. W. *Proc. natn. Acad. Sci. U.S.A.* **68,** 3049–3053 (1971).
25. Ogawa, T. & Tomizawa, J. *J. molec. Biol.* **38,** 217–225 (1968).
26. Furth, M. E. & Blattner, F. R. *Science* **198,** 1051–1055 (1977).
27. Zimmerman, S. B. & Kornberg, A. *J. biol. Chem.* **236,** 1480–1486 (1961).
28. Hershfield, M. S. & Nossal, N. G. *J. biol. Chem.* **247,** 3393–3404 (1972).
29. Morris, C. F., Hama-Inaba, H., Mace, D., Sinha, N. K. & Alberts, B. *J. biol. Chem.* **254,** 6787–6796 (1979).
30. Laemmli, U. K. *Nature* **227,** 680–685 (1970).
31. Cousens, L. S., Gallwitz, D. & Alberts, B. M. *J. biol. Chem.* **254,** 1716–1723 (1979).
32. Morris, C. F., Moran, L. & Alberts, B. *J. biol. Chem.* **254,** 6797–6802 (1979).
33. Wang, J. C. *J. molec. Biol.* **89,** 783–801 (1974).
34. Huang, W. M. *Virology* **66,** 508–521 (1975).
35. Hamilton, S. & Pettijohn, D. E. *J. Virol.* **19,** 1012–1027 (1976).
36. Gellert, M., Mizuuchi, K., O'Dea, M. H. & Nash, H. A. *Proc. natn. Acad. Sci. U.S.A.* **73,** 3872–3876 (1976).

Erratum

In the letter 'Basaltic pillars in collapsed lava-pool on the deep ocean floor' by J. Francheteau *et al.*, *Nature* **281,** 209–211, the third author's name was mis-spelt; it should read C. Rangin. Figures 1 and 2 were incorrectly reproduced. They are shown correctly below.

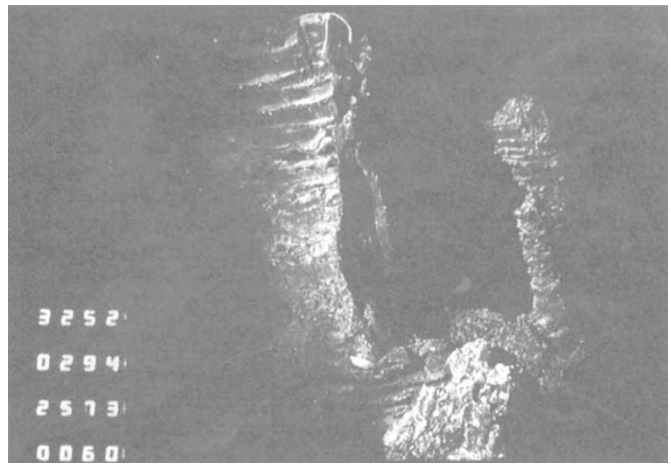

Fig. 1 General landscape: pillars with collapsed lava pond.

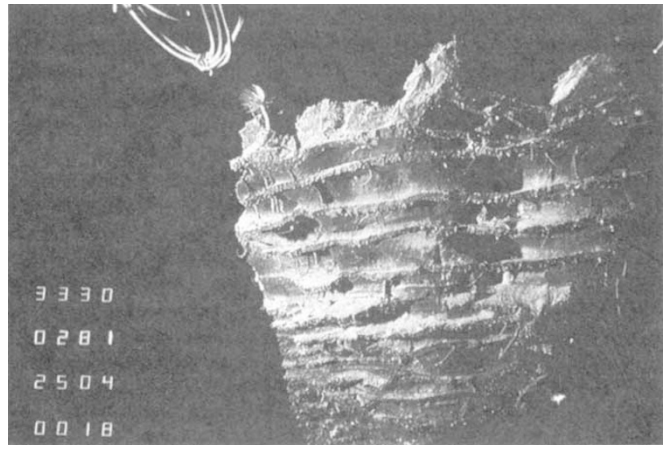

Fig. 2 Detail of a pillar showing the centimetric pseudo layering with thin and darker salient glass layers projecting from the basaltic surface of the pillar.