

the flexible domain in the DNA polymerase structure. The low resolution of the RNA polymerase structure (compared with that of the X-ray structure of DNA polymerase) allows this feature to be observed. The existence in DNA and RNA polymerases of this flexible domain, which might surround the DNA substrate, suggests a means by which these enzymes could act processively in the polymerization of their respective products<sup>10,15</sup>.

The significance of the structural similarity between DNA and RNA polymerases is supported by a small degree of amino-acid sequence conservation<sup>4</sup>. There is a weak homology between residues 666–695 in DNA polymerase I and residues 350–380 of the  $\beta$ -subunit of RNA polymerase<sup>4</sup>. The conserved residues are on the floor of the DNA-binding cleft of DNA polymerase I (ref. 10), part of the region that matches well with the RNA polymerase structure. Moreover, the  $\beta'$ -subunit of RNA polymerase is believed, based on other grounds, to be involved in DNA binding<sup>1</sup>.

The 55 Å length of the putative DNA-binding channel in the RNA polymerase map is sufficient to accommodate ~16 base pairs of double-helical DNA in the B form, which may relate to the 16–18 base pairs that become unwound in the transcription complex<sup>16</sup>. This single-stranded region lies within a longer

stretch of 50–60 base pairs (at least 170-Å long in the B form) associated with the enzyme in the open-promoter complex<sup>17</sup>. There does not seem to be any way in which a 170 Å length of DNA can be bound to the enzyme without significant bending of the DNA. Evidence for such bending has come from electrophoretic-mobility shift experiments<sup>18</sup>. The path followed by the DNA across the surface of the enzyme remains to be seen. Crystallization and imaging of enzyme–DNA complexes by the methods used here should help assess the role of the putative DNA-binding channel and reveal other features of the protein–DNA interaction. □

Received 24 March; accepted 13 July 1989.

1. von Hippel, P. H., Bear, D. G., Morgan, W. D. & McSwiggen, J. A. *Rev. Biochem.* **53**, 389–446 (1984).
2. Chamberlin, M. J. *Enzymes* **15**, 61–86 (1982).
3. Lewis, M. K. & Burgess, R. R. *Enzymes* **15**, 109–153 (1982).
4. Allison, L. A., Moyle, M., Shales, M. & Ingles, C. J. *Cell* **42**, 599–610 (1985).
5. Biggs, J., Searles, L. L. & Greenleaf, A. L. *Cell* **42**, 611–621 (1985).
6. Sweetser, D., Nonet, M. & Young, R. A. *Proc. natn. Acad. Sci. U.S.A.* **84**, 1192–1196 (1987).
7. Ahearn, J. M., Bartolomei, M. S., West, M. L., Cisek, L. J. & Corden, J. L. *J. biol. Chem.* **262**, 10695–10705 (1987).
8. Darst, S. A., Ribi, H. O., Pierce, D. W. & Kornberg, R. D. *J. molec. Biol.* **203**, 269–273 (1988).
9. Amos, L. A., Henderson, R. & Unwin, P. N. T. *Prag. biophys. molec. Biol.* **39**, 183–231 (1982).
10. Ollis, D. L., Brick, P., Hamlin, R., Xuong, N. G. & Steitz, T. A. *Nature* **313**, 762–766 (1985).
11. Strickland, M. S., Thompson, N. E. & Burgess, R. R. *Biochemistry* **27**, 5755–5762 (1988).
12. Henderson, R., Baldwin, J. M., Downing, K. H., Lepault, J. & Zemlin, F. *Ultramicroscopy* **19**, 147–178 (1986).
13. Agard, D. A. *J. molec. Biol.* **167**, 849–852 (1983).
14. Tichelaar, W., Schutter, W. G., Arnberg, A. C., Van Bruggen, E. F. J. & Stender, W. *Eur. J. Biochem.* **135**, 263–269 (1983).
15. Fairfield, F. R., Newport, J. W., Dolejsi, M. K. & von Hippel, P. H. *J. biomolec. Struct. Dyn.* **1**, 715–727 (1983).
16. Gamper, H. B. & Hearst, J. E. *Cell* **29**, 81–90 (1982).
17. Siebenlist, u., Simpson, R. B. & Gilbert, W. *Cell* **20**, 269–281 (1980).
18. Kuhnke, G., Fritz, H. & Ehring, R. *EMBO J.* **6**, 507–513 (1987).
19. Goodsell, D. S., Miam, I. S. & Olson, A. J. *J. molec. Graphics* **7**, 41–47 (1989).

ACKNOWLEDGEMENTS. We thank Dr M. J. Chamberlin and the members of his laboratory for providing pure RNA polymerase and for suggestions and encouragement. We also thank Drs C. W. Akey, H. O. Ribi and P. N. T. Unwin for discussion. S.A.D. was a recipient of an American Cancer Society Postdoctoral Fellowship. This work was supported by the NIH.

## ERRATA

### New light on the Lysenko era

Valery N. Soyfer

*Nature* **339**, 415–420 (1989)

IN this Commentary, the author's affiliation (page 420) should read: Department of Molecular Genetics and Biotechnology Center, The Ohio State University, Columbus, Ohio 43210, USA. And the sentence running from page 417 to page 418 should read: "Lysenko had also harmed his own cause by his resolute repudiation of plant hormones, and his reputation was undermined when the Dutchman Went and the Soviet psychologist N. G. Kholodony were honoured for their discovery." □

### Substrate specificity and affinity of a protein modulated by bound water molecules

F. A. Quiocho, D. K. Wilson & N. K. Vyas

*Nature* **340**, 404–407 (1989)

THE 'Acknowledgements' section was omitted from the above letter during the production process. It should read: This work was supported by the Howard Hughes Medical Institute and grants from NIH and the Welch Foundation. □

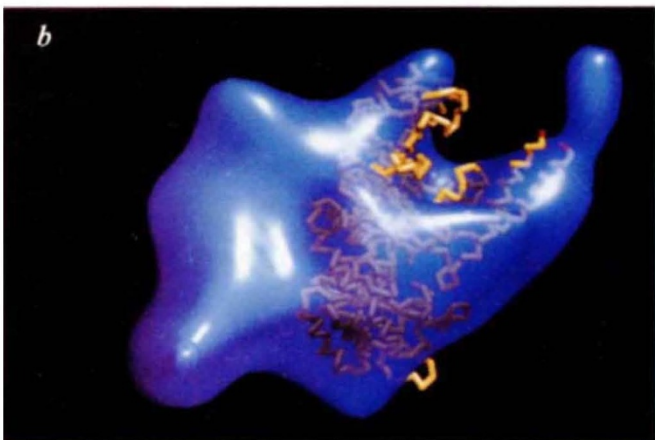
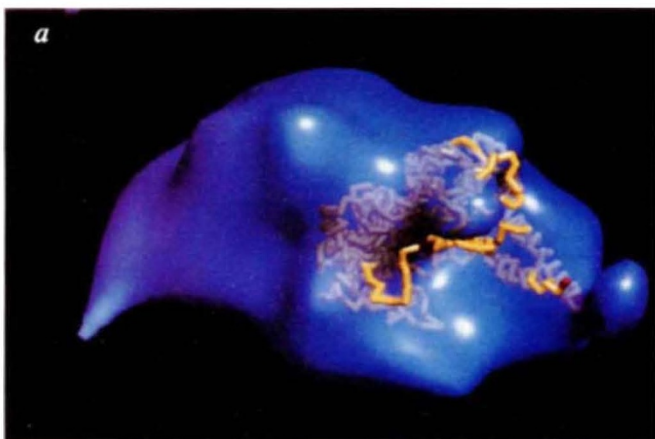


FIG. 3 Computer-generated models of a single *E. coli* RNA polymerase holoenzyme molecule. Matched to the RNA polymerase map is the  $\alpha$ -carbon backbone of the DNA polymerase I Klenow fragment<sup>10</sup> (shown in grey, except for parts protruding from the RNA polymerase map, which are shown in yellow). The  $\alpha$ -carbon atoms on each side of the unobserved gap in the DNA polymerase chain<sup>10</sup> are shown in red. The contour represents negative stain-excluding region. *a*, The molecule viewed in the plane of the two-dimensional array and perpendicular to the crystallographic *a*-axis. The lipid layer to which the molecule is adsorbed would lie underneath. *b*, The molecule viewed from the bottom (as if looking up through the lipid layer) along the axis of the cleft. Images courtesy of D. S. Goodsell and A. J. Olson<sup>19</sup> (magnification,  $\times 4.7 \times 10^6$ ).