microtubule moves forward (Fig. 4). Rotation of the microtubules was most obvious for microtubules grown from fragments of outer doublets that had dissociated from the axonemes. The intrinsic curvature of these fragments provided an asymmetrical marker for the radial orientation of the microtubules[14]. The microtubules rotated in a clockwise direction as viewed from their minus ends. A complete rotation occurred every 6.1 ± 2.6 s ($n = 29$) of forward movement, and there were 3.3 ± 1.3 rotations ($n = 29$) for every 1 μm of forward movement. Forward movement could continue even if rotation of the axoneme fragment was obstructed by the glass surface. These rotational features of the *claret* motor protein are strikingly similar to the rotation produced by 14S axonemal dynein, the only other microtubule motor protein reported to generate torque[14]. The mechanism of torque generation is unknown.

The *claret* motor protein was sensitive to inhibitors at concentrations known to inhibit cytoplasmic dynein activity[8]. Microtubule gliding, but not attachment, was blocked by 10 μM vanadate, 100 μM NEM (10 min treatment) or 2.5 mM of the non-hydrolysable ATP analogue AMP-PNP, but was unaffected by 1 mM AMP-PNP.

Why does a protein with 40–45% sequence identity to the kinesin motor domain have the directionality of a dynein ATPase? One possible explanation is that the *claret* motor can move bidirectionally and the bacterially expressed protein moves in only the minus-end direction. But its sensitivity to inhibitors and its ability to generate torque are more typical of dynein than of kinesin. Therefore, the most likely explanation is that the motor domains of kinesin and the dyneins are similar enough in sequence to allow molecular probes to the kinesin motor domain to identify both kinesin- and dynein-related motors. Like the *claret* motor, other members of the so-called kinesin superfamily[18] of motors, which have been identified by motor domain sequence similarity, may be minus-end directed motors.

The finding that the *claret* motor protein is a minus-end translocator suggests that it could be localized to the kinetochores on chromosomes and act to pull chromosomes polewards (toward the minus ends of microtubules) during prometaphase and/or anaphase. Unlike the rates reported for kinesin and cytoplasmic dynein[8], the rate at which the *claret* motor protein translocates microtubules ($4\ \mu m\ min^{-1}$) is close to the rate of chromosome movement observed during most of mitosis and meiosis[19]. □

Received 19 September; accepted 10 October 1990.

1. Sturtevant, A. H. Z. *Wiss. Zool.* **135,** 323–356 (1929).
2. Lewis, E. B. & Gencarella, W. *Genetics* **37,** 600–601 (1952).
3. Endow, S. A., Henikoff, S. & Niedziela, L. S. *Nature* **345,** 81–83 (1990).
4. Yamamoto, A. H., Komma, D. J., Shaffer, C. D., Pirrotta, V. & Endow, S. A. *EMBO J.* **8,** 3543–3552 (1989).
5. McDonald, H. B. & Goldstein, L. S. B. *Cell* **61,** 991–1000 (1990).
6. Walker, R. A., Gliksman, N. R. & Salmon, E. D. in *Optical Microscopy for Biology* (eds Herman, B. & Jacobsen, K.) 395–407 (Wiley-Liss, New York, 1990).
7. Yang, J. T., Saxton, W. M., Stewart, R. J., Raff, E. G. & Goldstein, L. S. B. *Science* **249,** 42–47 (1990).
8. McIntosh, J. R. & Porter, M. E. *J. biol. Chem.* **264,** 6001–6004 (1989).
9. Paschal, B. M. & Vallee, R. B. *Nature* **330,** 181–183 (1987).
10. Cohn, S. A., Ingold, A. L. & Scholey, J. M. *J. biol. Chem.* **264,** 4290–4297 (1989).
11. Vale, R. D., Schnapp, B. J., Reese, T. S. & Sheetz, M. P. *Cell* **40,** 559–569 (1985).
12. Paschal, B. M., Shpetner, H. S. & Vallee, R. B. *J. Cell Biol.* **105,** 1273–1282 (1987).
13. Paschal, B. M. *et al. Nature* **330,** 672–674 (1987).
14. Vale, R. D. & Toyoshima, Y. Y. *Cell* **52,** 459–469 (1988).
15. Belmont, L. D., Hyman, A. A., Sawin, K. E. & Mitchison, T. J. *Cell* **62,** 579–589 (1990).
16. Hyman, A. A. *et al. Meth. Enzym.* (in the press).
17. Vale, R. D. *et al. Cell* **43,** 623–632 (1985).
18. Vale, R. D. & Goldstein, L. S. B. *Cell* **60,** 883–885 (1990).
19. Salmon, E. D. in *Mitosis* (eds Hyams, J. S. & Brinkley, B. R.) 119–181 (Academic, San Diego, 1989).
20. Studier, F. W., Rosenberg, A. H., Dunn, J. J. & Dubendorff, J. W. *Meth. Enzym.* **185,** 60–89 (1990).
21. Walker, R. A. *et al. J. Cell Biol.* **107,** 1437–1448 (1988).
22. Koshland, D. E., Mitchison, T. J. & Kirschner, M. W. *Nature* **331,** 499–504 (1988).
23. Sale, W. S. & Fox, L. A. *J. Cell Biol.* **107,** 1793–1797 (1988).
24. Kuznetsov, S. A. *et al. EMBO J.* **7,** 353–358 (1988).

ACKNOWLEDGEMENTS. We thank A. Horne and V. Petrie for technical assistance, N. Gliksman for discussions, W. Sale for axonemal dynein and G. Langford for squid kinesin. Taxol was supplied by the Drug Synthesis and Chemistry Branch, Division of Cancer Treatment, National Cancer Institute. Supported by the NIH (S.A.E. and E.D.S.). S.A.E. is a member of the Duke University Comprehensive Cancer Center.

ERRATUM

Identification of the 64K autoantigen in insulin-dependent diabetes as the GABA-synthesizing enzyme glutamic acid decarboxylase

Steinunn Baekkeskov, Henk-Jan Aanstoot, Stephan Christgau, Annette Reetz, Michele Solimena, Marilia Cascalho, Franco Folli, Hanne Richter-Oleson & Pietro De Camilli

Nature **347,** 151–156 (1990).

IN the published version of this article, the name of one of the authors (P. De C.) was incorrectly hyphenated. Indexing services are requested to use the correct form above.