

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 microtubules¹⁴. 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 torque¹⁴. The mechanism of torque generation is unknown.

The *claret* motor protein was sensitive to inhibitors at concentrations known to inhibit cytoplasmic dynein activity⁸. 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 superfamily¹⁸ 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 dynein⁸, the rate at which the *claret* motor protein translocates microtubules ($4 \mu\text{m min}^{-1}$) is close to the rate of chromosome movement observed during most of mitosis and meiosis¹⁹. □

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ERRATUM

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

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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.

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