New and Notable

Don't Blink: Observing the Ultra-Fast Contraction of Spasmonemes

Wallace F. Marshall

Department of Biochemistry & Biophysics Integrative Program in Quantitative Biology, University of California at San Francisco, San Francisco, California 94158

Try to imagine a fast biological movement. Perhaps you visualize the twitch of an eye or the flicker of a boxer's jab. These movements may seem fast, but in this issue of Biophysical Journal, Upadhyaya and colleagues take biological speed to a whole new level by analyzing the contractions of Vorticella, a wineglass shaped ciliated protist (1). When a Vorticella cell is frightened, it can contract its tail, which contains a striated fiber called the spasmoneme, at a rate of 10 cm/s. Expressed in units of lengths per second (Ls⁻¹), the standard way that muscle contraction speed is measured, this works out to around 200 Ls⁻¹. This speed is an order of magnitude faster than the fastest muscles, which contract at around 20 Ls⁻¹. Here's the best part: spasmoneme contraction doesn't even require ATP hydrolysis! Instead, contraction of isolated spasmonemes can be driven simply by increasing the calcium concentration from 10^{-8} M to 10^{-6} M.

The fact that the spasmoneme can perform this huge rapid contraction without ATP hydrolysis doesn't mean that Vorticella has invented a perpetual motion machine. To perform multiple cycles of contraction and extension, calcium concentration would have to switch back and forth between different levels, whichof course consumes energy. In fact, the example of the

Submitted August 2, 2007, and accepted for publication August 6, 2007.

Address reprint requests to Wallace F. Marshall, Dept. of Biochemistry and Biophysics, University of California at San Francisco, San Francisco, CA 94143. Tel.: 415-514-4304; E-mail: wmarshall@biochem.ucsf.edu.

Editor: Alexander Mogilner.
© 2008 by the Biophysical Society 0006-3495/08/01/4/02 \$2.00

spasmoneme provides a particularly dramatic illustration of the basic principle that ATP hydrolysis is often not directly coupled with the power stroke of a motor protein but only plays a role in resetting the motor for the next cycle.

The two main questions about spasmoneme contraction are first, what molecular mechanisms drive the contraction, and second, how is the contraction coordinated along the length of the entire structure. The paper by Upadhyaya and coworkers addresses both questions using high speed video microscopy. First, they measure the rate of contraction as a function of the viscosity of the surrounding media. From the scaling relation between maximum speed and viscosity, they conclude that the speed is limited by the power dissipated by dragging the top of the Vorticella through the surrounding viscous media and not, for example, by some rate-limiting conformational rearrangement within the spasmoneme itself. This is an important result that puts constraints on possible models for how the system works.

The high speed of spasmoneme contraction also poses a challenge at the level of control. What mechanism could transmit the contraction-triggering signal over the whole length of the spasmoneme, given that the contraction only takes a few milliseconds? To provide more physical insight into the control of contraction, the authors tracked the motion of beads stuck onto the Vorticella stalk to show that contraction initiates near the body of the Vorticella and propagates like a wave down the stalk. This strongly suggests that some stimulus emanates from the body down the stalk, although the observation itself doesn't identify the nature of the stimulus. Given that contraction is driven by calcium binding, the obvious model would be a calcium wave mediated by calcium-triggered calcium release from the endoplasmic reticulum. However, such calcium waves move much too slowly (2) to account for the rapid propagation of the contractile signal,

which Upadhyaya and coworkers have clocked at around 10 cm/s. The authors speculate that an electrical signal may be responsible for the propagation, but it also seems formally possible that the stimulus could be carried by a propagating wave of protein conformational change within the spasmoneme. Computational models for propagating conformational waves predict extremely high speeds with theoretical estimates exceeding 100 m/s (3). The rate at which a conformational change could propagate would ultimately be limited by the speed of sound through the protein lattice of the spasmoneme. The speed of sound through protein crystals is on the order of 1 km/s (4), which would be more than fast enough to account for the transmission speeds observed. In any case, further study of how the contraction wave is generated and propagated in spasmonemes may hold important lessons for long-range rapid information transmission through cells.

Although the spasmoneme is unique to protists, its main protein constituent spasmin is closely related to the centrin family of calcium-binding protein found associated with centrioles and basal bodies in many eukaryotes including humans. Centrin assembles into fibers that can contract when calcium is added, and in some organisms the contraction of centrin fibers is used to steer cell motility by changing the angle at which cilia emerge from the cell surface (5). Presumably the spasmoneme evolved from such structures under selective pressure to contract at high speeds. This suggests that detailed molecular comparisons of centrin and spasmin, together with the behaviors of their corresponding fibers, may shed light on the adaptations that allow spasmin to contract so fast.

Next time you stop to feed the ducks, you can take a moment to reflect on the biophysical mystery posed by the tiny Vorticella contracting beneath the scum.

New and Notable 5

REFERENCES

- Upadhyaya, A., M. Baraban, J. Wong, P. Matsudaira, A. van Oudenaarden, and L. Mahadevan. 2007. Power-limited contraction dynamics of *Vorticella convallaria*: an ultrafast biological spring. *Biophys. J.* 94:265–272.
- 2. Jaffe, L. F., and R. Créton. 1998. On the
- conservation of calcium wave speeds. *Cell Calcium*. 24:1–8.
- 3. Ciblis, P., and I. Cosic. 1997. The possibility of soliton/exciton transfer in proteins. *J. Theor. Biol.* 184:331–338.
- Edwards, C., S. B. Palmer, P. Emsley, J. R. Helliwell, I. D. Glover, G. W. Harris, and D. S. Moss. 1990. Thermal motion in protein
- crystals estimated using laser-generated ultrasound and Young's modulus measurements. *Acta Crystallogr. A.* 46:315–320.
- McFadden, G. I., D. Schulze, B. Surek, J. L. Salisbury, and M. Melkonian. 1987. Basal body reorientation mediated by a Ca²⁺-modulated contractile protein. *J. Cell Biol*. 105:903–912.