unusual myosin VI motor has surprises in store in this regard, because it can move processively along actin with forward steps averaging ~30 nm (ref. 14), even though it has an extremely short lever arm with only one light-chain-binding site; some unexpected conformational transformations must occur in myosin VI, which also shows a much broader spectrum of step sizes than does myosin V. In the case of myosin VI, the diffusive component revealed by Veigel *et al.* for myosin V is likely to be a much more significant part of the overall step. As investigators continue to dig deeper into the technological developments that allow single molecules to be analysed in ever increasing detail, many exciting new findings are sure to emerge. □

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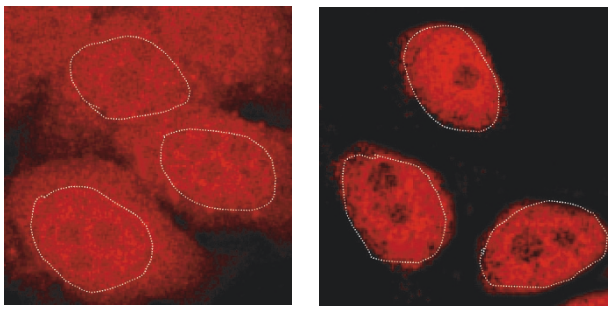
1. Mehta, A. D. *et al. Nature* **400,** 590–593 (1999).
2. Sakamoto, T., Amitani, I., Yokota, E. & Ando, T. *Biochem. Biophys. Res. Commun.* **272,** 586–590 (2000).
3. Rief, M. *et al. Proc. Natl Acad. Sci. USA* **97,** 9482–9486 (2000).
4. Veigel, C., Wang, F., Bartoo, M. L., Sellers, J. R. & Molloy, J. E. *Nature Cell Biol.* **4,** 59–65 (2002).
5. Svoboda, K., Schmidt, C. F., Schnapp, B. J. & Block, S. M. *Nature* **365,** 721–727 (1993).
6. Finer, J. T., Simmons, R. M. & Spudich, J. A. *Nature* **368,** 113–119 (1994).
7. Vale, R. D. *et al. Nature* **380,** 451–453 (1996).
8. Noji, H., Yasuda, R., Yoshida, M. & Kinosita, K., Jr *Nature* **386,** 299–302 (1997).
9. Wells, A. L. *et al.* Myosin VI is an actin-based motor that moves backwards. *Nature* **401,** 505–508 (1999).
10. Spudich, J. A. The myosin swinging cross-bridge model. *Nature Rev. Mol. Cell Biol.* **2,** 387–392 (2001).
11. De La Cruz, E. M., Wells, A. L., Rosenfeld, S. S., Ostap, E. M. & Sweeney, H. L. *Proc. Natl Acad. Sci. USA* **96,** 13726–13731 (1999).
12. Moore, J. R., Krementsova, E. B., Trybus, K. M. & Warshaw, D. M. *J. Cell Biol.* **155,** 625–635 (2001).
13. Rice, S. *et al. Nature* **402,** 778–784 (1999).
14. Rock, R. S. *et al. Proc. Natl Acad. Sci. USA* **98,** 13655–13659 (2001).

Finding the path less followed



IMED-EDDINE GALLOUZI AND JOAN A. STEITZ

Reaching the cytoplasm by transport through the nuclear pore is no trivial matter. To achieve this feat, both proteins and RNAs must first bind to adaptor molecules that, in turn, interact with transport receptors on the pore itself. So far, it has proved more difficult to study the pathways that regulate the transport of mRNAs, owing largely to the lack of tools to address single adaptor–receptor pairs. However, Gallouzi and Steitz (*Science* **294,** 1895–1901 (2001)) have now described the use of cell-permeable peptides that allow the specific inhibition of individual mRNA export pathways in intact mammalian cells.

In their study, they focused on HuR, an adaptor that mediates the export of short-lived mRNAs such as c-*fos*. HuR appears to use two pathways, one of which depends on CRM1. The CRM1-dependent path acts through pp32 and APRIL, both of which contain CRM1-binding NES domains, but the CRM1-independent path acts through the HuR-shuttling domain (HNS) to bind an unknown receptor.

Gallouzi and Steitz set out to test whether HuR does indeed use two pathways for mRNA export and to characterize what this other pathway might be. To do this, they fused different nuclear export sequences to a peptide from the homeodomain of *Drosophila* Antennapedia (AP) that is sufficient for cellular uptake. Intriguingly, they found that, depending on the sequence used, the peptide could inhibit specific mRNA export pathways. Also, whereas AP–NES and AP–HNS only partly inhibited HuR-mediated export of c-*fos* mRNA (shown in red, left panel; the white line indicates the boundary of the nucleus), they strongly inhibited export when present together (right panel), confirming that HuR uses two export pathways.

So what is this other pathway? One candidate was transportin 2 (Trn2), a putative transport receptor with an unassigned function. Gallouzi and Steitz found that Trn2 binds to HuR and, moreover, that treatment of cells with AP–HNS blocked the interaction between HuR and Trn2, indicating that Trn2 is the other transport receptor used by HuR. The question now is why HuR needs to have more than one export pathway. One possibility is that different pathways predominate in different environmental conditions, allowing adaptability—indeed, during heat shock, HuR has been shown to switch to the CRM1-dependent pathway. Consistent with this, Gallouzi and Steitz found that the interaction between HuR and Trn2 is blocked during heat shock.

Importantly, these peptides did not affect the total polyA$^+$ RNA distribution between the cytosol and nucleus, indicating that the all-important Ran–GTP gradient is unaffected by their addition. Given this specificity, it should now be feasible to use this method to unravel the pathways that mediate the export of endogenous mRNAs.

ALISON SCHULDT

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