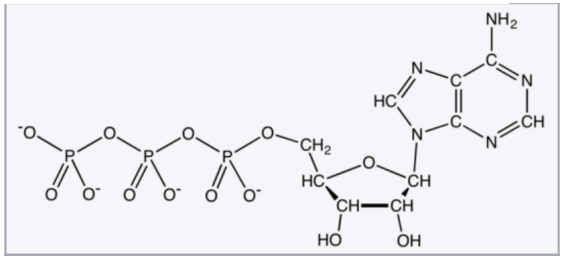
Adenosine triphosphate (**ATP**) is a complex organic chemical that participates in many processes. Found in all forms of life, ATP is often referred to as the "molecular unit of currency" of intracellular energy transfer.[1] When consumed in metabolic processes, it converts to either the di- or monophosphates, respectively ADP and AMP. Other processes regenerate ATP such that the human body recycles its own body weight equivalent in ATP each day.[2] It is also a precursor to DNA and RNA.



$$[RO\text{-}P(O)_2\text{-}O\text{-}P(O)_2\text{-}O\text{-}PO_3]^{4-} + H_2O \rightarrow [RO\text{-}P(O)_2\text{-}O\text{-}PO_3]^{3-} + [PO_4]^{3-} + 2\ H^+$$

$$\Delta G^\circ = -30.5\ \text{kJ/mol} \quad = -12\ k_B T$$

With a typical intracellular concentration of 1–10 mM, ATP is abundant.

ATP can be produced by a number of distinct cellular processes; the three main pathways in eukaryotes are (1) glycolysis, (2) the citric acid cycle/oxidative phosphorylation, and (3) beta-oxidation. The overall process of oxidizing glucose to carbon dioxide, the combination of pathways 1 and 2, is known as cellular respiration, produces about 30 equivalents of ATP from each molecule of glucose.[15]

Mitochondria

ATP recycling [edit]

The total quantity of ATP in the human body is about 0.2 moles. The majority of ATP is recycled from ADP by the aforementioned processes. Thus, at any given time, the total amount of ATP + ADP remains fairly constant.

The energy used by human cells requires the hydrolysis of 100 to 150 moles of ATP daily, which is around 50 to 75 kg. A human will typically use up his or her body weight of ATP over the course of the day. Each equivalent of ATP is recycled 500-750 times during a single day (100 / 0.2 = 500).

Source: Wikipedia

REVIEW

7 APRIL 2000 VOL 288 SCIENCE

The Way Things Move: Looking Under the Hood of Molecular Motor Proteins

Ronald D. Vale[1]* and Ronald A. Milligan[2]

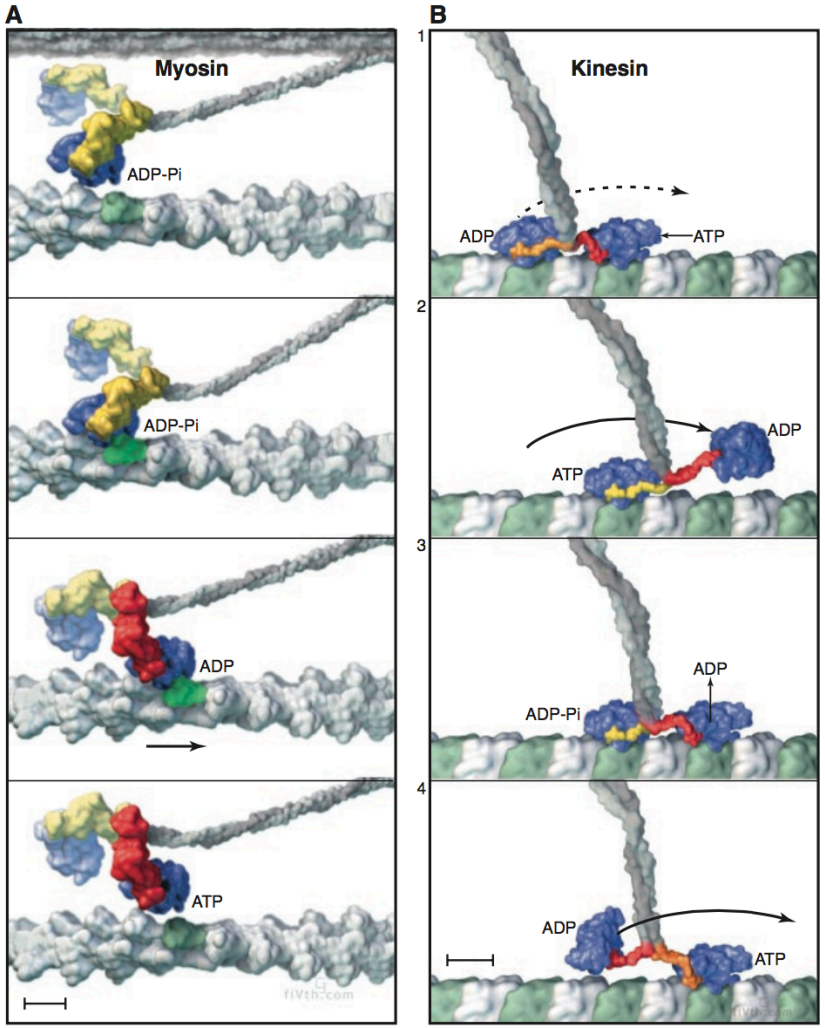


Fig. 1. Models for the motility cycles of muscle myosin and conventional kinesin [see animation (*23*)]. (**A**) Muscle myosin. Frame 1: Muscle myosin is a dimer of two identical motor heads (catalytic cores are blue; lever arms in the prestroke ADP-Pi state are yellow), which are anchored to the thick filament (top) by a coiled coil (gray rod extending to the upper right). In the ADP-Pi–bound state, the catalytic core binds weakly to actin. Frame 2: One head docks properly onto an actin binding site (green). The two myosin heads act independently, and only one attaches to actin at a time. Frame 3: Actin docking causes phosphate release from the active site. The lever arm then swings to the poststroke, ADP-bound state (red), which moves the actin filament by ~100 Å. Frame 4: After completing the stroke, ADP dissociates and ATP binds to the active site, which rapidly reverts the catalytic core to its weak-binding actin state. The lever arm will then recock back to its prestroke state (i.e., back to frame 1). (**B**) Conventional kinesin. Unlike myosin, the two heads of the kinesin dimer work in a coordinated manner to move processively along the track. The coiled coil (gray) extends toward the top and leads up to the kinesin cargo. Frame 1: Each catalytic core (blue) is bound to a tubulin heterodimer (green, β subunit; white, α subunit) along a microtubule protofilament (the cylindrical microtubule is composed of 13 protofilament tracks). To adopt this position, the neck linker points forward on the trailing head (orange; neck linker next to but not tightly docked to the core) and rearward on the leading head (red). ATP binding to the leading head will initiate neck linker docking. Frame 2: Neck linker docking is completed by the leading head (yellow), which throws the partner head forward by 160 Å (arrow) toward the next tubulin binding site. Frame 3: After a random diffusional search, the new leading head docks tightly onto the binding site, which completes the 80 Å motion of the attached cargo. Polymer binding also accelerates ADP release, and during this time, the trailing head hydrolyzes ATP to ADP-Pi. Frame 4: After ADP dissociates, an ATP binds to the leading head and the neck linker begins to zipper onto the core (partially docked neck indicated by the orange color). The trailing head, which has released its Pi and detached its neck linker (red) from the core, is in the process of being thrown forward. The surface features of the motors and filaments were rendered by G. Johnson (fiVth media: www.fiVth.com) using the programs MolView, Strata Studio Pro, and Cinema 4D (also for Figs. 4 and 5). Protein Data Bank (PDB) files used throughout the figures are as follows: ADP-AlF_4^- smooth muscle myosin [prestroke, yellow: 1BR2 (*16*)], nucleotide-free chicken skeletal myosin [poststroke, red: 2MYS (*14*)], human conventional kinesin [prestroke, red: 1BG2 (*6*)], and rat conventional kinesin [poststroke, yellow: 2KIN (*40*)]. Scale bars, 60 Å (A) and 40 Å (B).

Kinesin hydrolyses one ATP per 8-nm step

Mark J. Schnitzer*† **& Steven M. Block**†‡

*Departments of * Physics and † Molecular Biology, and ‡ Princeton Materials Institute, Princeton University, Princeton, New Jersey 08544, USA*

NATURE | VOL 388 | 24 JULY 1997

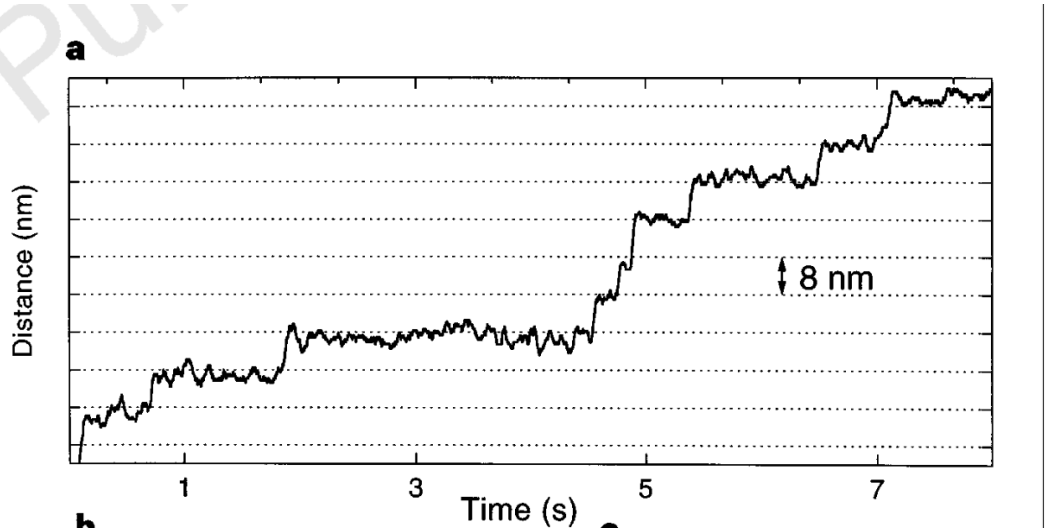


Figure 2 a, Sample record of movement at 2 μM ATP, showing the elementary steps (solid line). Horizontal grid lines (dotted lines) are spaced 8 nm apart. Data were median-filtered with a window width of 60 ms. **b**, Normalized histogram of

Kinesin Walks Hand-Over-Hand

Ahmet Yildiz,[1] Michio Tomishige,[3*] Ronald D. Vale,[3]
Paul R. Selvin[1,2†]

30 JANUARY 2004 VOL 303 SCIENCE

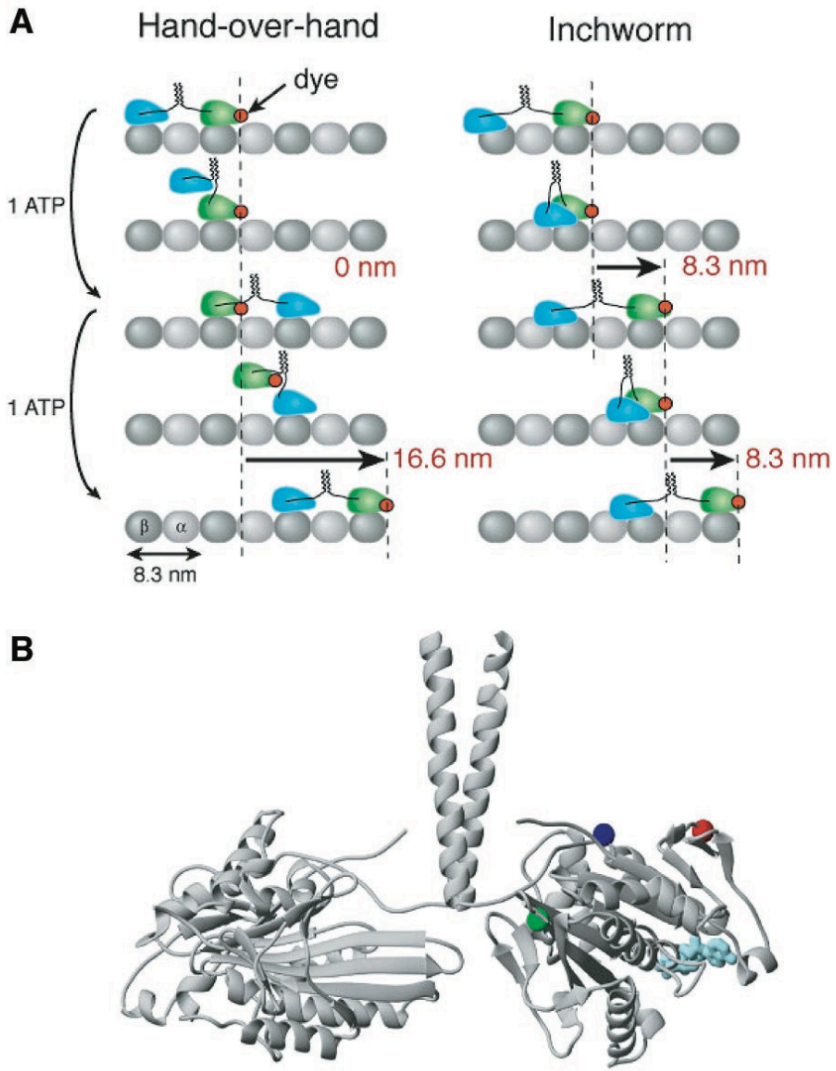


Fig. 1. (**A**) Examples of two alternative classes of mechanisms for processive movement by kinesin. The hand-over-hand model (left) predicts that a dye on the head of kinesin will move alternately 16.6 nm, 0 nm, 16.6 nm, whereas the inchworm mechanism (right) predicts uniform 8.3-nm steps. The inchworm model was adapted with slight modification from (*9*). (**B**) The positions of S43 (red), E215 (green), and T324 (blue) on the rat kinesin crystal structure [from (*6*), Protein Data Base 2KIN]. These residues, whose numbers correspond to conventional human kinesin, were mutated to cysteines for fluorescent dye labeling as described in the text. The bound nucleotide (adenosine diphosphate) is shown as a space-filling model in cyan. This figure was made with MolMol (*22*).

[Movie: Kinesin protein walking on microtubule](https://www.youtube.com/watch?v=tMKlPDBRJ1E)
https://www.youtube.com/watch?v=tMKlPDBRJ1E

Madison Johnson vor 2 Jahren (bearbeitet)

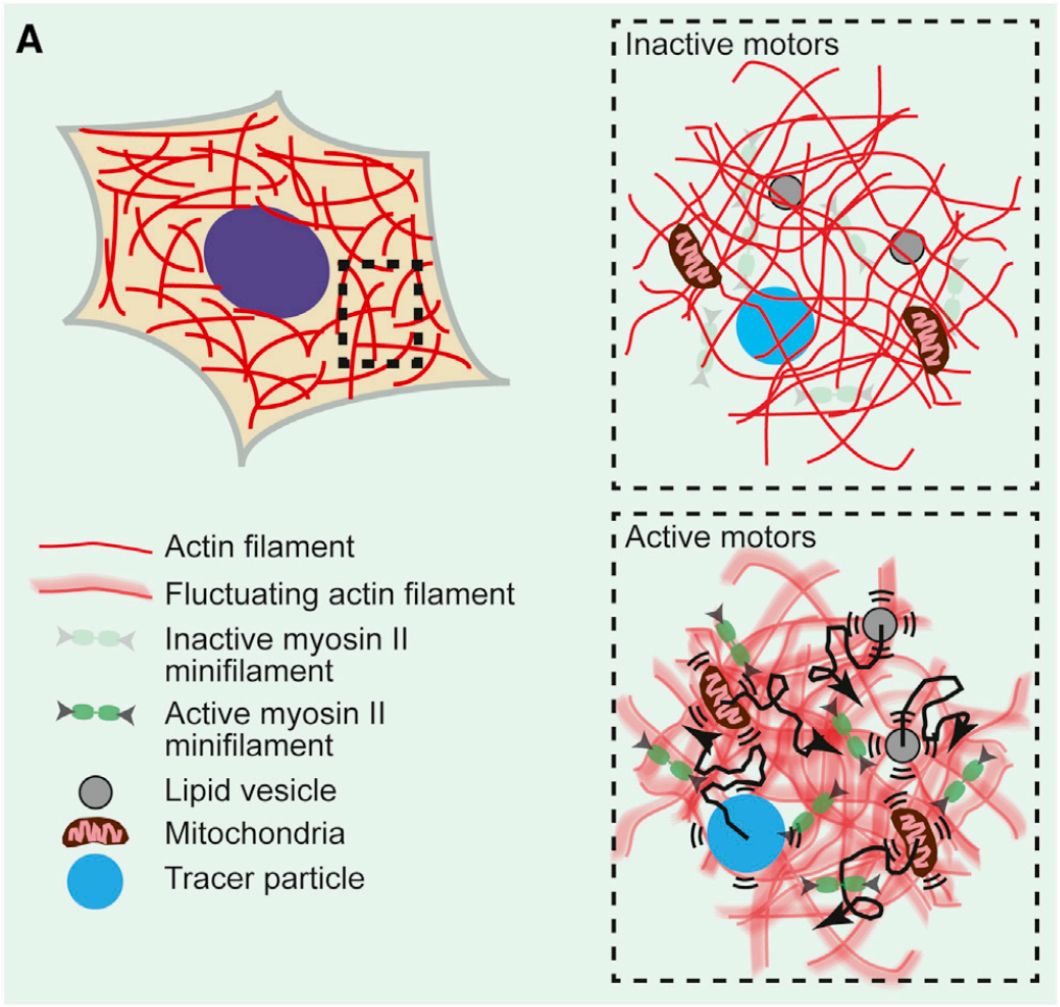
Play this but with Bees Gees "Stayin Alive" in the background.

ANTWORTEN 36

Probing the Stochastic, Motor-Driven Properties of the Cytoplasm Using Force Spectrum Microscopy

Cell *158*, 822–832, August 14, 2014

Ming Guo,[1] Allen J. Ehrlicher,[1,2,8] Mikkel H. Jensen,[1,3] Malte Renz,[4] Jeffrey R. Moore,[3] Robert D. Goldman,[5] Jennifer Lippincott-Schwartz,[4] Frederick C. Mackintosh,[6] and David A. Weitz[1,7,*]



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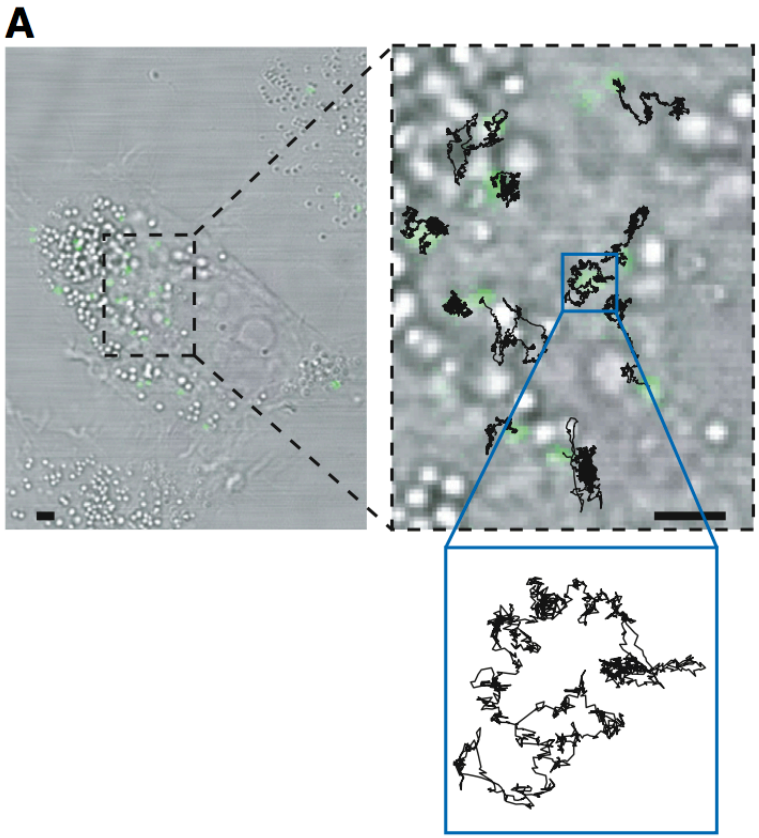
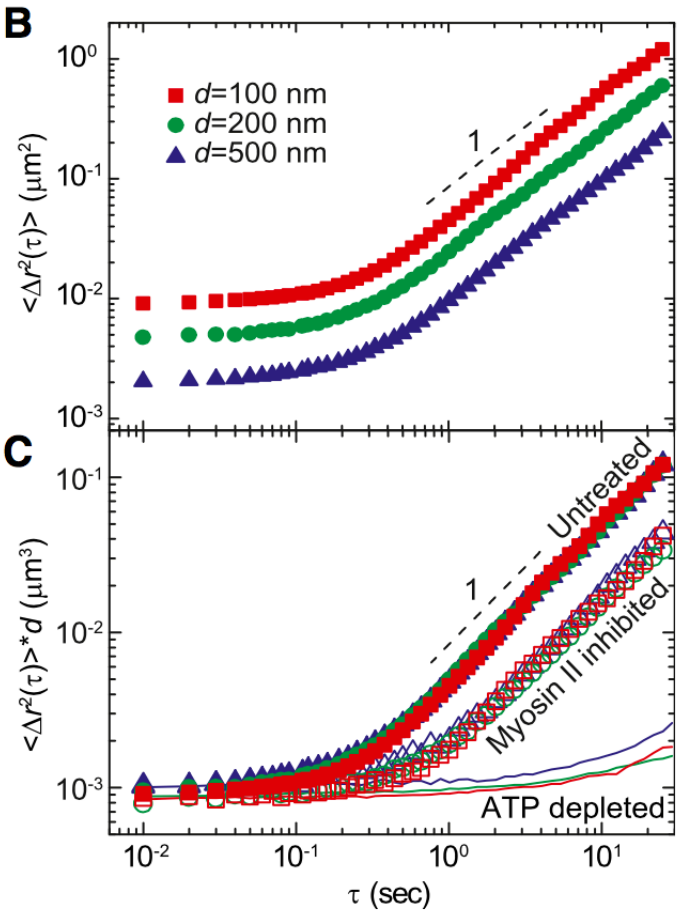
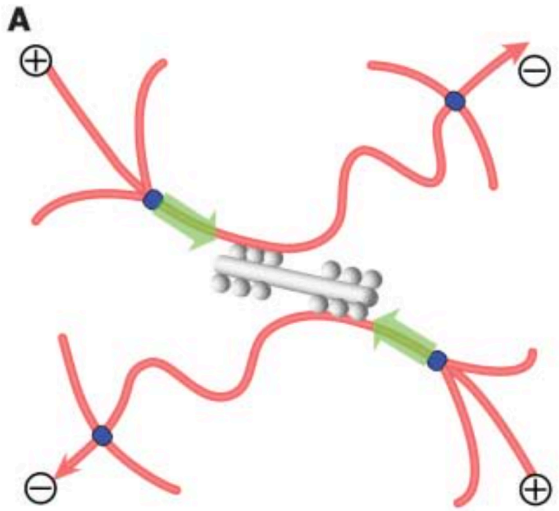


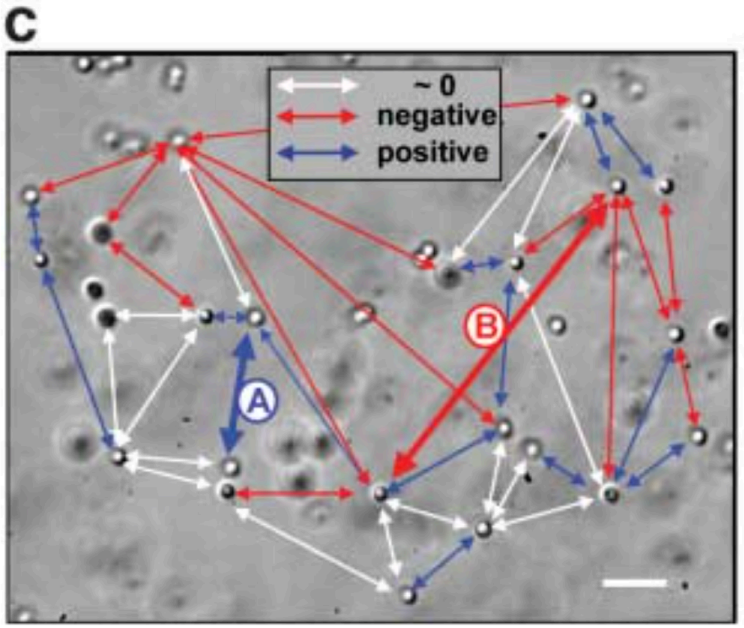


Nonequilibrium Mechanics of Active Cytoskeletal Networks

19 JANUARY 2007 VOL 315 SCIENCE

Daisuke Mizuno,[1] Catherine Tardin,[1] C. F. Schmidt,[1,2]* F. C. MacKintosh[1]*





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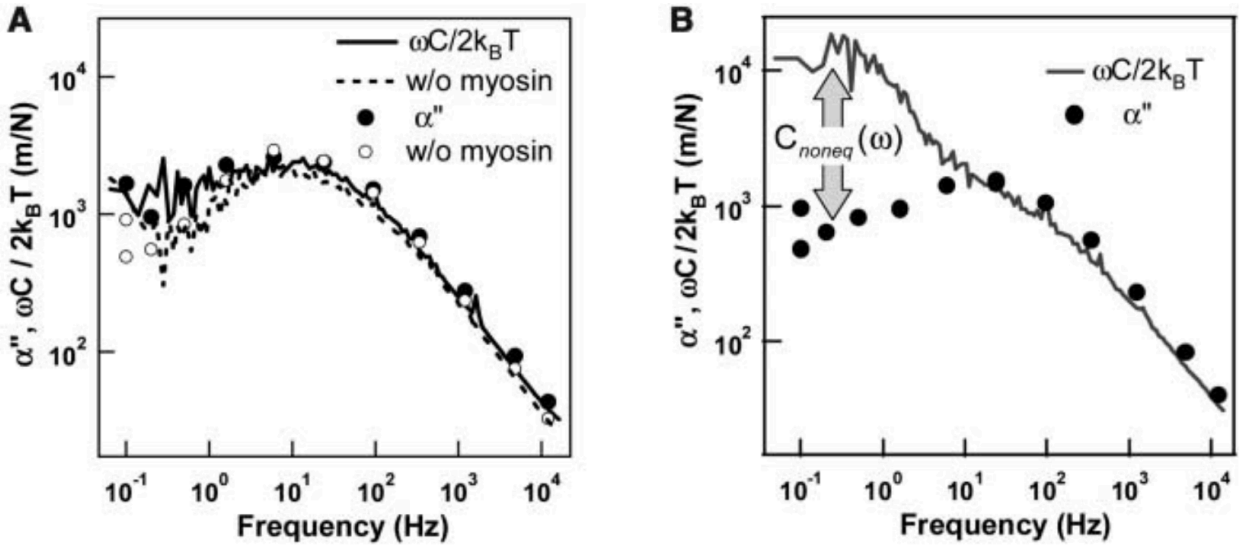


Fig. 2. Mechanical response of cross-linked nonactive and active gels (actin and myosin concentrations as in Fig. 1). (**A**) The imaginary part of the response function α'' measured by AMR (circles) and the normalized power spectrum $\omega C(\omega)/2k_BT$ measured by PMR (lines). Open circles and the dashed line denote cross-linked actin without myosin; solid circles and the solid line denote networks with myosin 2.5 hours after sample preparation. For up to 5 hours, α'' and $\omega C(\omega)/2k_BT$ with and without myosin show good agreement, indicating that myosin activity did not yet produce observable nonequilibrium fluctuations. (**B**) The same as (A) but 6.8 hours after sample preparation (with myosin). Below 10 Hz, nonequilibrium fluctuations are observable as an enhancement of $\omega C(\omega)/2k_BT$ relative to α''.

response function

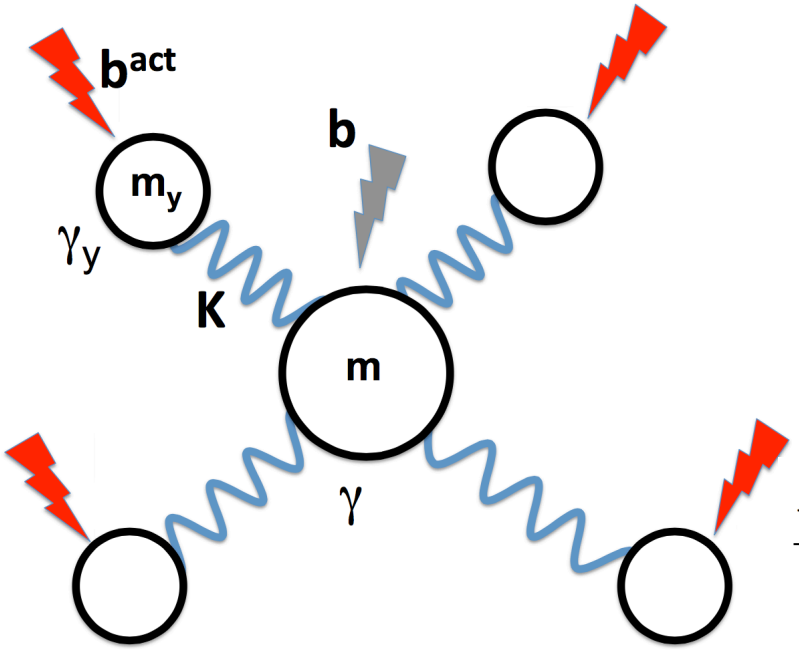
$$\alpha(\omega) = u(\omega)/F$$

autocorrelation

$$C(\omega) = \int \langle u(t)u(0) \rangle \exp(i\omega t)\, dt$$

fluctuation dissipation theorem

$$\alpha''(\omega) = \frac{\omega}{2k_BT} C_{eq}(\omega) \text{ (equilibrium only)}$$



non-equilibrium many-particle model for tracer particle that is elastically coupled to n active particles

generalized non-eq. Fluct.-diss. Theorem

$$\frac{-\omega\tilde{C}_{\rm xx}(\omega)/(2k_{\rm B}T)}{\tilde{\chi}^{I}(\omega)} = \frac{\tilde{C}_{\rm FF}(\omega)/(2k_{\rm B}T)}{\tilde{\Gamma}^{R}(\omega)} = 1+\Xi(\omega)$$

$$\Xi(\omega) = \frac{\alpha n\gamma_y}{n\gamma_y+\gamma+\gamma\gamma_y^2\omega^2/K^2} \simeq \frac{\alpha}{1+\tau^2\omega^2}$$

comparison experiment / theory

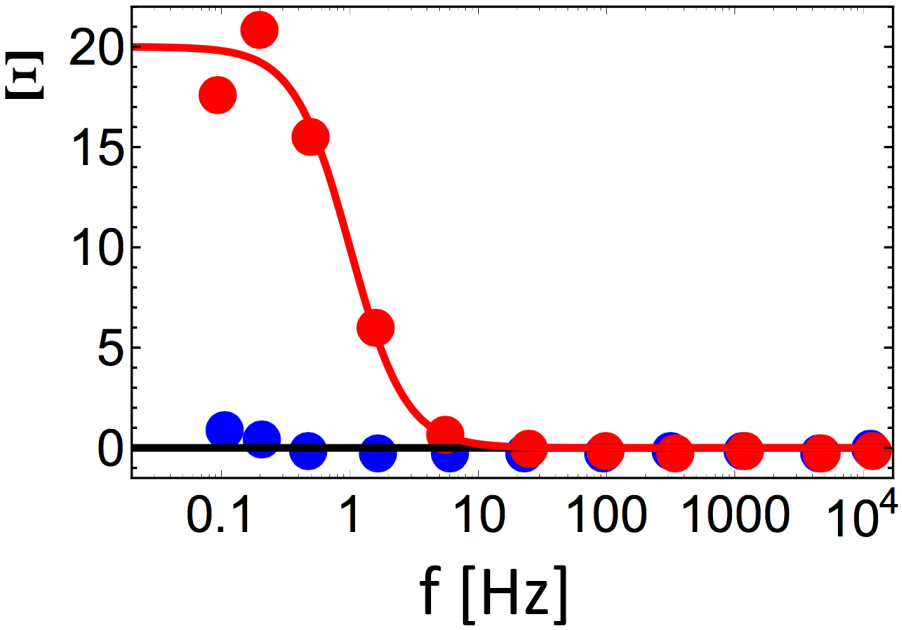


FIG. 2. The spectral function $\Xi(\omega)$ plotted here as a function of frequency $f = \omega/(2\pi)$ characterizes deviations from the equilibrium fluctuation-dissipation theorem and is defined in Eq. 26. Experimental data from motor-protein driven actin networks in the presence of ATP (red circles) [36] are compared with the prediction Eq. 29 (red line), the extracted non-equilibrium parameter is $\alpha = 20$ and the time scale is $\tau = 1s$. Blue circles denote experimental results in the absence of ATP [36] and agree with the expected equilibrium limit $\Xi(\omega) = 0$ (black horizontal line).