



**Research**

**Techniques**

**Publications**

**Opportunities**

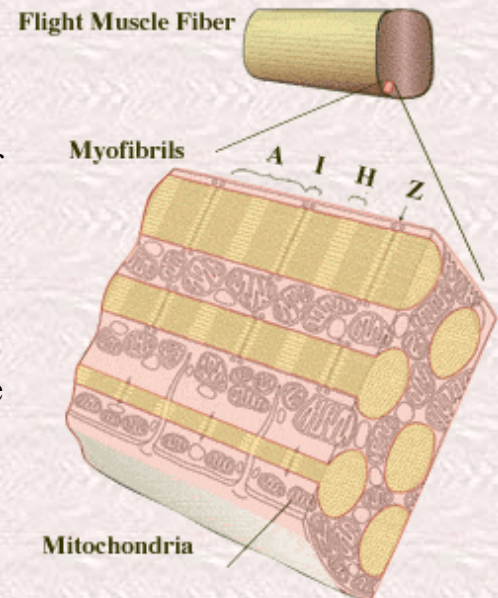
**Other Sites**

**Contact Us**

**Home**

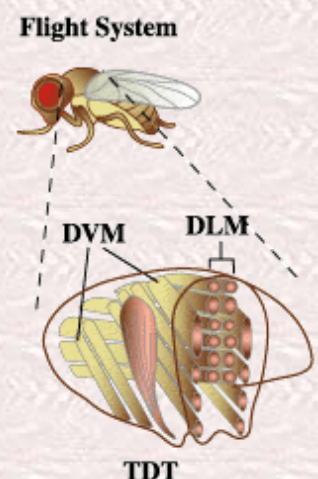
### Unique Muscle Structure

Typically there is a trade of between muscle force and speed. Fast muscles have narrow myofibrils (that allow rapid calcium diffusion into and out of the myofibrils) along with extensive sarcoplasmic reticulum (for rapid calcium release and reuptake). Slower muscles can pack in thicker myofibrils with less sarcoplasmic reticulum at the expense of contraction speed. Insect flight muscle fibers have a unique structure that seems to break these rules. Myofibrils are cylindrical shaped with a large diameter of ~1.5 um. Within the myofibrils the characteristic banding pattern of striated muscle can be seen with sarcomeres that range from 3.0-3.4 um. Although IFM contract rapidly (~ 200 Hz), the sarcoplasmic reticulum of this muscle type is scarce. In its place are numerous mitochondria. The stretch activation response allows the high muscle contraction frequencies independent of direct nervous control.

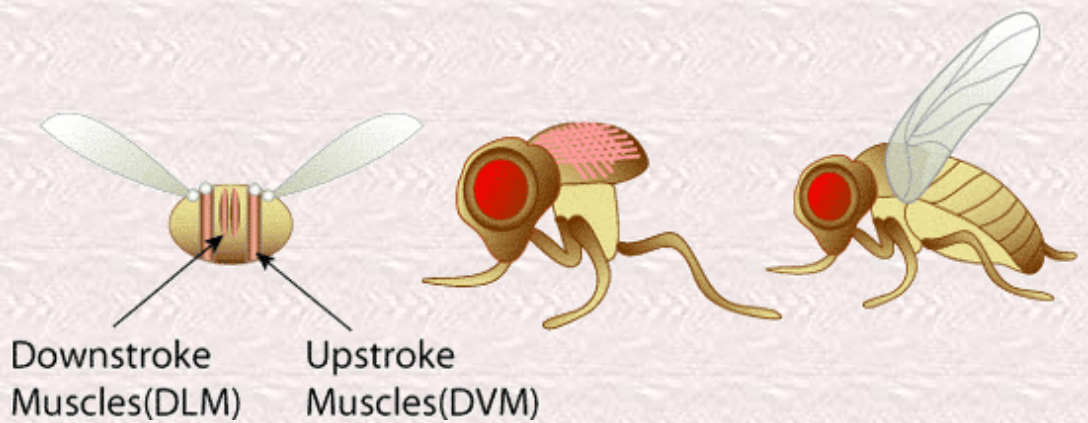


### The *Drosophila* thoracic muscular system.

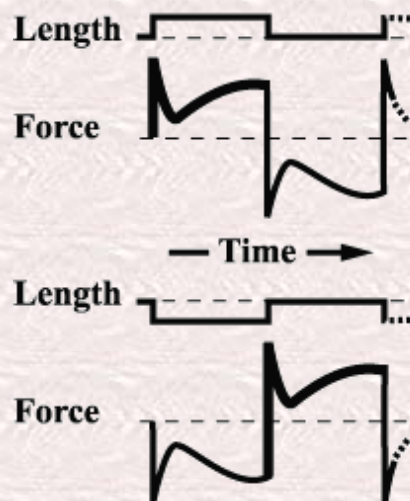
Wing movement in *Drosophila* is powered by two antagonistically oriented muscle fibers that are attached to the thoracic cuticle from which the wings extend. There are 14 dorsal ventral muscle fibers (DVM) that are located toward the outsides of the thorax. The dorsal longitudinal muscles (DLM) can be seen flanking the mid-sagittal plane. The tergal depressor of trochanter (TDT), or “jump” muscle is shown lateral to the the DVMs.



### Wing Movement



The wing-thorax-flight muscle system of Dipterans is capable of highly tuned resonance. It is believed that contraction of the jump muscle deforms the thoracic cuticle in such a way that causes the both the wings to move through their upstroke and to stretch the DLMs. Stretching of the DLMs activates them and their contraction deforms the cuticle the opposing way and stretches the DVMs. Stretching of the DVMs leads to their contraction which stretches the DLMs. If the timing between the delay in activation matches the characteristic frequency of the flight system a resonating oscillator will be formed.



### Stretch Activation

**DLM** It is important to note that the IFM are activated by intracellular calcium via sporadic nervous stimulation. Only with this fluctuating (but sufficiently high) calcium level does the muscle contract alternately in response to a stretch produced by the other. In isolated IFM the response to stretch consists of a rapid, spring-like, increase in tension followed by a

decay that is eventually overcome by a significant increase in tension (the stretch activation response). Stieger (1977) and Pringle (1978) observed that the delayed tension rise was a property of all muscles that happens to be enhanced in muscles that drive oscillating systems like cardiac and indirect flight muscle. Basically, if the delay matches the timing of the muscle extension and shortening then the muscle will perform oscillatory work and thus feed the resonating system. Several models have been proposed to explain stretch activation. [Click here](#) for more information.

[Research](#) ● [Techniques](#) ● [Publications](#) ● [Opportunities](#) ● [Other Sites](#) ● [Contact Us](#) ● [Home](#)

**Boston University School of Medicine**

715 Albany Street, Boston, MA 02118 • ph 617-638-4251 • fax 617-638-4273 • email [jxmoore@bu.edu](mailto:jxmoore@bu.edu)



**Research**

**Techniques**

**Publications**

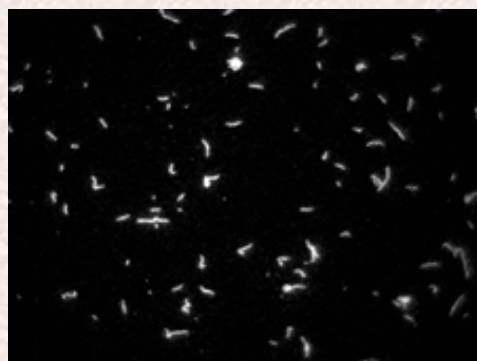
**Opportunities**

**Other Sites**

**Contact Us**

**Home**

We use a variety of techniques to study the biochemical and mechanical properties of molecular motors, the cytoskeleton and biopolymers.



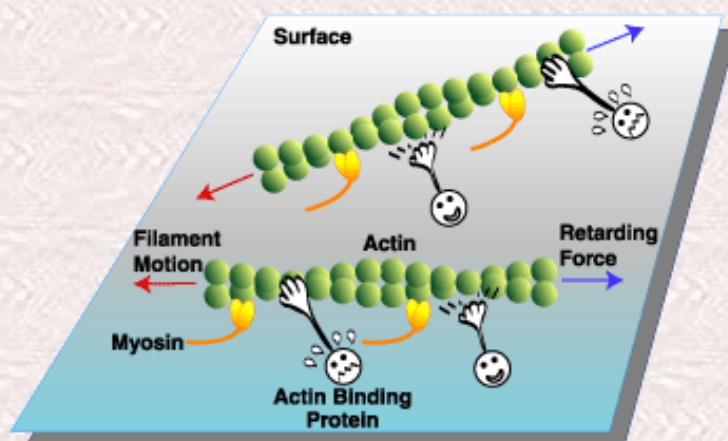
## Unloaded in vitro motility assay

The unloaded in vitro motility assay has all the advantages of solution biochemistry (i.e., temp, ionic conditions, substrate concentrations etc. are easily manipulated); however,

movement, one of the fundamental properties of molecular motor function, can also be quantified. In this assay myosin is adsorbed to a silanized coverslip surface. Fluorescently labeled actin filaments are then allowed to bind to the surface-bound myosin. Movement is initiated by the addition of ATP and recorded with an ICCD camera.

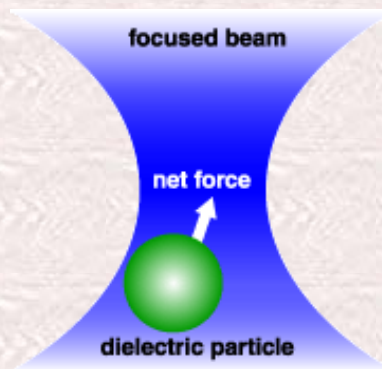
## Frictional loading in vitro motility assay

The unloaded motility assay gives important information about the motion generating capacity of molecular motors. However, motor molecules also produce force. We assess the effects of myosin or thin filament regulatory protein mutations on relative (to wild-type) force generation. In this assay, load is applied to the myosin motor by applying it to the surface along with small amounts of non motile actin binding proteins (e.g., alpha-actinin). The alpha-actinin will introduce a frictional load that resists filament motion. The amount of alpha-actinin required to stop filament motion is an indicator of



of alpha-actinin required to stop filament motion is an indicator of

myosin force generating capability. Since the load is applied to all of the filaments in a field of view, this assay is useful for rapidly screening a variety of experimental conditions (e. g.: regulated actin, unregulated actin, submaximal calcium activation, etc.).

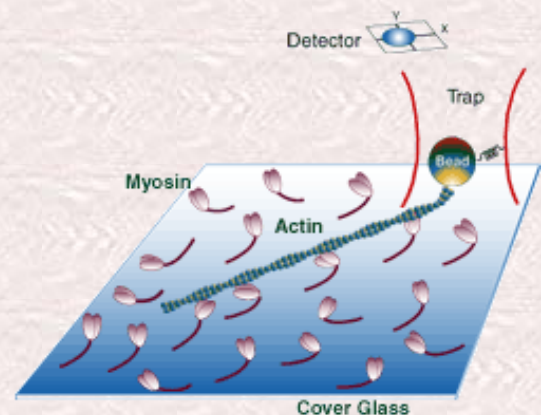


### Optical trap

Optical traps use photon momentum to capture and hold small transparent particles with a higher refractive index than the medium. As a trapped particle moves away from the laser focus there is a restoring force that is proportional to the distance the particle moves. Therefore, an optical trap can be used as a very sensitive force transducer that can measure forces in the piconewton range.

### Laser trap loading assay

We use both optical trap and frictional loading based assays to measure the inherent force generating capacity of mutant myosin molecules. Frictional loading assays provide an indication of relative force while optical trap based assays measure the absolute isometric force generating capability of the myosin mutants one filament at a time.



[Research](#) ● [Techniques](#) ● [Publications](#) ● [Opportunities](#) ● [Other Sites](#) ● [Contact Us](#) ● [Home](#)

**Boston University School of Medicine**

715 Albany Street, Boston, MA 02118 • ph 617-638-4251 • fax 617-638-4273 • email [jxmoore@bu.edu](mailto:jxmoore@bu.edu)