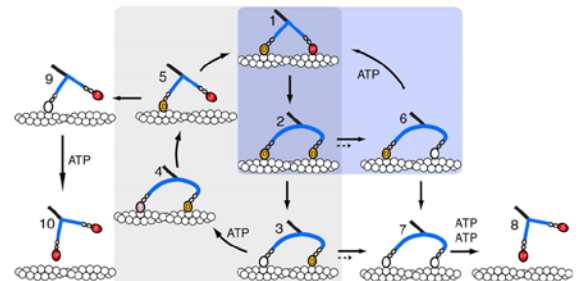


Howard D. White, PhD

Research Interests

The goal of the research in my laboratory is to determine the detailed molecular mechanism of how myosin motor proteins convert the free energy from ATP hydrolysis into movement and work. There are 40 genes for myosin in the human genome, 10 of the genes code for myosins in muscle and the other 30 are involved in either the movement or production of tension in various non muscle cells and organs. Defects in myosin motor function can produce such diverse affects as cardiac failure, deafness, blindness and neurological abnormalities. Non muscle myosins have also contributed enormously to our basic understanding of the molecular mechanism of myosin function. Work from my laboratory has been fundamental in determining the important roles of phosphate and ADP dissociation with respect to the powerstroke and shortening velocity of actomyosin. A sabbatical in John Trinick's laboratory sponsored by a Fogarty international fellowship resulted in an extensive collaboration which combined the rapid kinetic methods from my laboratory with the em expertise in Trinick's laboratory to develop various approaches to time resolved electron microscopy, which have been utilized in our work and by the wider scientific community. My laboratory has been funded by NIH and various private foundation grants (MDA, AHA, ACS) since I became an independent investigator. Current work in my laboratory includes determination of the structure and mechanism of the reverse directed myosinVI motor and the role of myosin binding protein C on the regulation of the cardiac thin filament. This is a truly exciting time in the molecular motors field as we now have the technology and insight to truly understand function at the molecular level.

Diagram of the principal states of the ATP hydrolysis pathway of actin-Myo5-2IQ-SAH



Baboolal T G et al. PNAS 2009;106:22193-22198