Title
2011 ASCB Annual Meeting abstracts

Permalink
https://escholarship.org/uc/item/7kw0k3d8

Journal
Molecular Biology of the Cell, 22(24)

ISSN
1059-1524

Authors
Xu, J
Reddy, B
Anand, P
et al.

Publication Date
2011-12-14

DOI
10.1091/mbc.E11-10-0886

License
CC BY 4.0

Peer reviewed
Kinesin-1 is a plus-end microtubule-based motor, and defects in kinesin-based transport are linked to diseases including neurodegeneration. Kinesin can auto-inhibit via a direct head-tail interaction, but is believed to be active otherwise. Here we report a tail-independent inactivation of kinesin, reversible by the disease-relevant signaling protein, casein kinase 2 (CK2). The majority of initially active kinesin (native or tail-less) loses its ability to bind/interact with microtubules in vitro, and CK2 reverses this inactivation (~ 4-fold) without altering kinesin's single motor properties. This activation pathway does not require motor phosphorylation, and is independent of head-tail autoinhibition. In cultured mammalian cells, reducing CK2 expression, but not kinase activity, decreases the force required to stall lipid droplet transport, consistent with a reduction in the number of active motors. These results provide the first direct evidence of a protein kinase up-regulating kinesin-based transport, and suggest a novel pathway for regulating the activity of cargo-bound kinesin.