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Journal
Biophysical Journal, 102(3)

ISSN
00063495

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Publication Date
2012

DOI
10.1016/j.bpj.2011.11.2012

Peer reviewed

1869-Pos Board B639

Casein Kinase 2 Reverses Tail-Independent Inactivation of Kinesin-1

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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.