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Casein Kinase 2 Reverses Tail-Independent Inactivation of Kinesin-1[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 head-tail interaction, but is believed to be active otherwise. Here we report a tail-independent inactivation of kinesin, reversible by the disease-relevant signalling protein, casein kinase 2 (CK2). The majority of initially active kinesin (native or tail-less) loses its ability to interact with microtubules in vitro, and CK2 reverses this inactivation (approximately fourfold) without altering kinesin's single motor properties. This activation pathway does not require motor phosphorylation, and is independent of head–tail auto-inhibition. In cultured mammalian cells, reducing CK2 expression, but not its kinase activity, decreases the force required to stall lipid droplet transport, consistent with a decreased number of active kinesin motors. Our results (Nat. Commun., 3:754, 2012) provide the first direct evidence of a protein kinase upregulating kinesin-based transport, and suggest a novel pathway for regulating the activity of cargo-bound kinesin.

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