

Abstract Submitted
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Casein Kinase 2 Reverses Tail-Independent Inhibition of Kinesin-1¹ JING XU, ZHANYONG SHU, PREETHA ANAND, BABU REDDY, SILVIA CERMELLI, THOMAS WHISENANT, UC Irvine, STEPHEN KING, University of Missouri-Kansas City, LEE BARDWELL, LAN HUANG, STEVEN GROSS, UC Irvine — Kinesin-1 is a plus-end microtubule-based molecular motor, and defects in kinesin transport are linked to diseases including neurodegeneration. Kinesin can auto-inhibit via a direct head-tail interaction, but is believed to be active otherwise. In contrast, this study uncovers a fast but reversible inhibition distinct from the canonical auto-inhibition pathway. The majority of the initially active kinesin (full-length or tail-less) loses its ability to bind/interact with microtubule, and Casein Kinase 2 (CK2) reverses this inactivation (up to 4-fold) without altering kinesin's single motor properties. Motor phosphorylation is not required for this CK2-mediated kinesin activation. In cultured mammalian cells, knockdown of CK2 level, but not kinase activity, was sufficient to decrease the force required to stall lipid droplet transport, consistent with a reduction in the number of active motors. We propose that CK2 forms a positive regulating complex with the motor. This study provides the first direct evidence of a protein kinase positively regulating kinesin-transport, and uncovers a pathway whereby inactive cargo-bound kinesin can be activated.

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