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GSK-3 regulates transport of kinesin-1 driven cargos *in vivo* CHRISTINA LEIDEL, Department of Physics, University of Texas at Austin, CAROLE WEAVER, LUKASZ SZPANKOWSKI, LAWRENCE S.B. GOLDSTEIN, HHMI, Department of Cellular and Molecular Medicine, School of Medicine, Univeristy of California, GEORGE T. SHUBEITA, Department of Physics, University of Texas at Austin, CENTER FOR NONLINEAR DYNAMICS, DEPARTMENT OF PHYSICS, UNIVERSITY OF TEXAS AT AUSTIN COLLABORATION, HHMI, DEPARTMENT OF CELLULAR AND MOLECULAR MEDICINE, UNIV. OF CALIFORNIA COLLABORATION — The Glycogen Synthase Kinase 3 (GSK-3) has been linked to many aspects of the development of Alzheimer's disease and was proposed to play a role in the transport of the Amyloid Precursor Protein (APP) by kinesin-1 motors. Using *Drosophila* embryos and larvae with altered GSK-3 expression, we characterize motor transport of cargos including APP and lipid droplets using DIC microscopy, high-resolution video tracking, fluorescence, and *in vivo* stall force measurements with optical tweezers. By comparing cargo velocities and run lengths we find that GSK-3 is a required negative regulator of *in vivo* transport. Stall force measurements on lipid droplets reveal that enhanced transport under conditions of reduced GSK-3 is a result of a larger number of active motors hauling the cargo. Our findings have implications on the use of GSK-3 inhibitors in treatment of Alzheimer's disease.

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