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Filament depolymerisation by motor proteins GERNOT KLEIN, KARSTEN KRUSE, FRANK JUELICHER, Max Planck Institute For The Physics Of Complex Systems, Noethnitzerstr. 38, 01187 Dresden, Germany — Many active processes in cells are driven by highly specialized motor proteins, which interact with filaments of the cytoskeleton. Members of the Kin-13 kinesin subfamily are able to interact specifically with filament ends and induce depolymerisation of the filaments ends. Recent in vitro assays and single molecule studies have shown, that MCAK accumulates at both ends of stabilized microtubules and induces depolymerisation while at the same time MCAK molecules do not generate directed motion along the microtubules [1]. We analyse both, a stochastic model and a generic mean-field description of this process. We discuss conditions under which motors dynamically accumulate at the filament end. Such a dynamic accumulation occurs for processive cutting, which implies, that the motor can remain attached to the shrinking edge after subunit removal. For processive cutting, the depolymerisation speed as a function of the bulk motor concentration can exhibit a maximum for intermediate motor concentration. For high motor processivity a dynamic instability can occur. We discuss our results in relation to recent experiments on Kin-13 motor proteins [2]. [1] A.W. Hunter, et al., Mol. Cell **11,** 445 (2003) [2] G.Klein, et al., submitted

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