Electronic Supplementary Material (ESI) for Soft Matter. This journal is © The Royal Society of Chemistry 2015

Supplementary Information (Smith et al.)

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Experimental details

Genetic perturbations: For the motor mutants, the cin8 and kip1 deletion strains were tested in several different ways: PCR for the presence of the KAN gene in the cin8 and kip1 locus; PCR for the absence of a wildtype copy of CIN8 and KIP1; and the most telling, a functional experiment: synthetic lethality of the cin8/kip1 double mutant, having one marked with NAT, the other with KAN. In crossing the cin8 strain to kip1, no spore was recovered that carried both mutations, of the approximately 60 tetrads tested, while all the other expected genotypes show up as expected, excluding the presence of a suppressor in either of the strains. Taken together, the PCR results verify that the deletions are present and that there is no wild type copy of either kip1p or cin8p present; and the crosses verify that these strains are synthetically lethal.

Fig. S1 shows that when we remove either of the two types of kinesin-5 tetrameric motors, kip1 or cin8, using complete deletion of the gene, we find no statistically significant change in the observed spectrum.

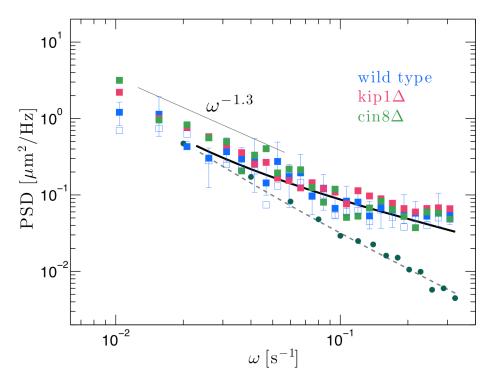


Figure S1. The *in vivo* PSD determined in metaphase for separate populations of cells lacking the gene for one or the other of the two types of kinesin-5 tetrameric motors, kip1 and cin8, plotted together with the observed spectrum from unperturbed metaphase cells (Fig. 2) for comparison.