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<b>Type</b>	Conference or Workshop Item
<b>URL</b>	This version is available at: <a href="http://usir.salford.ac.uk/id/eprint/9732/">http://usir.salford.ac.uk/id/eprint/9732/</a>
<b>Published Date</b>	2009

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The PAM-1 aminopeptidase regulates microtubule dynamics during meiosis and polarity establishment in the early *C. elegans* embryo. Pauline Greene<sup>1</sup>, Sara Marshall<sup>1</sup>, Lauren Brady<sup>1</sup>, Christopher Reeves<sup>1</sup>, Darren Brooks<sup>2</sup>, Elwyn Isaac<sup>3</sup>, **Rebecca Lyczak**<sup>1</sup>. 1) Biology Dept, Ursinus Col, Collegetown, PA; 2) Biomedical Sciences Research Institute, University of Salford, Salford, UK; 3) University of Leeds, Leeds, UK.

The PAM-1 aminopeptidase is required for numerous processes in the early embryo including meiotic exit, axis polarization, and chromosome segregation. As these developmental processes require distinct microtubule movements, we sought to examine the role of PAM-1 in microtubule dynamics. In localization studies we found that PAM-1 is largely cytoplasmic; however specific localization during meiosis and early polarity establishment was also observed around microtubules. During meiosis in *pam-1* mutants, we have found that the meiotic spindle wanders along and away from the cortex during meiosis II much more than in wild-type embryos. Additionally, during polarity establishment, the centrosomes move dynamically and prematurely away from the posterior cortex. We hypothesized that this centrosome movement may cause the polarity defect in *pam-1* mutants. To test this, we inactivated the microtubule motor dynein, DHC-1, and its regulator LIS-1, in an attempt to prevent centrosome movement from the cortex and restore anterior-posterior polarity. This was indeed what we observed, with *pam-1; dhc-1(RNAi)* embryos exhibiting normal pseudocleavage and localization of the P granules and PAR proteins, signs of polarity absent in *pam-1* mutants alone. We conclude that DHC-1 and LIS-1 are required for the abnormal centrosome movements in *pam-1* embryos. Additionally, we show that PAM-1's role in axis polarization is to prevent premature movement of the centrosome from the posterior cortex, ensuring proper axis establishment in the embryo. This coupled with the defects in meiotic spindle movement implicate PAM-1 in numerous microtubule dependent processes in the early embryo.