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*A Study of the Function of Cohesin at
Centrosomes in Human Cells*

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Cohesin is a multi-subunit protein complex classically known to hold sister chromatids together during mitosis and meiosis. Separation of sister chromatids is regulated by the cleavage of the cohesin's Scc1/Rad21 subunit by the proteolytic enzyme separase. Experiments demonstrating cohesin's localization to polar centrosomes and ability to bind with specific centrosome components, such as γ -tubulin, have cued towards a novel function of cohesin at the centrosomes. Depleting individual subunits of the cohesin complex in HeLa cells (a laboratory human cell line) by RNA interference results in multipolar spindle formation during mitosis as visualized by immunostaining. As cancer cells commonly have mutations in the genes encoding the cohesin complex and are clinically characterized as having an irregular number of chromosomes, the multipolar state that arises upon disrupting formation of the cohesin complex may offer insight to how chromosomes are miss-sorted during cancer cell division. I used live cell imaging to study the dynamics of the development of multipolar mitotic cells in two HeLa cell lines after Scc1/Rad21 depletion by RNA interference. Specific proteins within the cell lines were tagged with a green fluorescent protein (GFP) to visualize both the mitotic spindle (α -tubulin-GFP) and the interphase centrosome (centrin-1-GFP). Overall, 60% of experimental α -tubulin-GFP depleted cells exhibited extended mitotic periods and aberrant spindle formation while interphase centrin-1-GFP knockdown cells showed increased numbers of centrosome structures and less centrosome separation.



Poster Number: Session: