Malfunctions in the LINC (Linker of Nucleoskeleton and Cytoskeleton) complex machinery lead to varying cases of muscular dystrophy, cardiac and skeletal muscle pathologies, and progeria. Progeria is a premature aging syndrome characterized by the development of atherosclerosis in young patients. Although endothelial cell (EC) dysfunction contributes to cardiovascular impairments in progeria patients, the role of the LINC complex in EC is still poorly understood. Our study examines how the depletion of LINC complex proteins affects microtubule organization in endothelial cells. Specifically, we investigate the unique and shared functions of SUN1, SUN2, and SYNE1 (nesprin-1) proteins. Through siRNA-mediated depletion, re-nucleation assays, and orbital flow experiments, the research identifies how SUN1, SUN2, and SYNE1 influence centrosome positioning and microtubule nucleation in cultured endothelial cells. Results indicate that the depletion of *SUN1*, *SUN2*, or *SYNE1*, individually or in combination, impacts centrosome distance from the nucleus, induces Golgi apparatus dispersal, and delays microtubule re-nucleation. The findings suggest that the LINC complex components are critical for the communication between the nucleus and the centrosome, which has implications in endothelial cell polarization and signal transduction, contributing to vascular biology and pathology.