Cytokinesis constitutes the final stage of cell division, where a parent cell with a duplicated genome is divided into two daughters receiving a complete genome each. To accomplish this, dividing cells undergo unparalleled morphological transformations that are driven by distinct cytoskeletal machineries. Careful spatio-temporal control of these processes is required to maintain the fidelity of genome transmission. A critical juncture in the cytokinetic programme is the formation and stabilization of an intercellular bridge between the dividing cells. As cytokinesis progresses, this structure undergoes an elaborate maturation process involving its elongation and thinning before it is finally abscissed to physically separate daughter cells. Important molecular details of these complex operations remain unknown. In this thesis, I endeavored to decipher the mechanisms of two aspects of intercellular bridge biology: first, the molecular determinants of its formation and maturation, and second, the regulation of the final abscission event. With regards to the former, I discovered that biochemically distinct populations of septin cytoskeletal filaments participate in the bridge’s formation and maturation, requiring the adaptor proteins anillin and CIN85 to exert their coordinated functions. For the latter, I discovered that the PP2A-B56ε phosphatase complex promotes abscission by dephosphorylating the regulatory abscission factor CHMP4C. Further, I demonstrate that this phosphatase is dysregulated by a replication stress-like pathway in response to DNA segregation errors, an insult known to activate a purported cell cycle checkpoint that delays abscission. Lastly, I report that this ‘abscission checkpoint’ appears to bias dividing cells with serious segregation errors towards cell cycle arrest. Together, these investigations broaden our understanding of the molecular mechanisms and biological rationale at play in the intercellular bridge.