Most bacterial cells divide with the help of a dynamic ring of polymerized tubulin (FtsZ), called the Z ring, along with numerous other membrane-associated proteins (cumulatively called the divisome). In *Escherichia coli*, a number of proteins regulate the assembly state of FtsZ. Some of these, such as MinC and SlmA, spatially modulate FtsZ assembly to ensure that the Z ring ends up at the precise middle of the cell. This results in the production of two equal-sized progeny cells. Once the Z ring is formed, there is a notable delay until constriction of the Z ring can be seen by microscopy, indicating that there is a setup phase followed by an active phase. Other regulators, such as ZipA and FtsA, a homolog of actin, are key for the setup phase. They anchor FtsZ polymers to the cytoplasmic membrane and modulate FtsZ protofilament bundling to create a stable divisome. In addition, FtsA seems to interact with proteins that arrive later at the divisome. We are studying the molecular mechanisms by which these regulators affect FtsZ assembly state, focusing particularly on SlmA, MinC, FtsA, ZipA and the late divisome protein FtsN. We also are investigating bacteriophage peptides that inhibit the divisome in order to understand the normal functions of their targets. In particular, we found that the Kil peptide of bacteriophage lambda targets FtsZ and inhibits FtsZ assembly. Finally, we are exploiting cells that lack MinC, as they form small minicells that are ideal hosts for cryo-electron tomography of surface structures such as chemoreceptor arrays and infecting phage.