Despite much progress over the last six decades, we are still far from understanding the mechanism of protein folding and aggregation in the cell. This lack of information poses tremendous challenges to progress in many areas of life sciences, and it severely impedes key efforts in biomedical research. Learning more about the interplay between protein folding and aggregation in bacterial cells has a direct impact on the development of strategies to treat microbial infection and on the optimization of protein-based drug production in the pharmaceutical industry. In bacteria, the majority of soluble cellular proteins fold or aggregate co-translationally and immediately post-translationally. The ribosome and molecular chaperones play a key role in this process. This lecture will report progress, failures and surprises on our molecular-level understanding of how the ribosome, ribosomal proteins and cotranslationally active molecular chaperones modulate the balance between protein folding and aggregation in the cell. By probing the structure and dynamics of nascent proteins by fluorescence depolarization in the frequency domain, multidimensional NMR, biochemical tools and kinetic simulations, we will attempt to recapitulate some fundamental concepts of general significance, and suggest how these concepts can be specifically exploited to reprogram bacterial cells and combat disease.