The Gram-negative cell has a thinner murein wall than the Gram-positive bacterium. It does have an envelope with four components: The cytoplasmic membrane, the monolayered murein wall, the periplasmic space, and the outer membrane. Each of these has many functions to perform. However, only the murein wall is strong enough to give the cell shape and resist turgor pressure. The foremost questions about the wall are: How thick is it? How metabolically stable are the sidewall and the poles? How elastic is it? How stretched is the murein in a growing cell?

Because the wall of the Gram-negative cell is so thin, wall enlargement requires insertion of new murein units (disaccharide penta-muropeptides) into portions of the wall while it is under stress. This requires very special processes that need to be under the critical control of the bacterium. Most importantly, this requires “smart” autolysin action”. The job of autolysins is to cleave the murein to allow enlargement, but, of course, it has to be done very carefully. One suggestion to account for this is that the autolysins may be allosteric enzymes and their function has been arranged so that they only act if cleavage will not cause harm. A second model is that the autolysins are bundled with the synthetic part of a complex system for wall enlargement and result in new wall being formed as the old is being cleaved and/or turned over. Therefore very complex holoenzymes have been postulated. These holoenzymes would require a number of proteins to be exported through the cytoplasmic membrane and then aggregated into a multiprotein structure in order to function safely. A third, and recent, model (the Nona-muropeptide Stress Model) depends on the range of conformations achieved by a cross-linked nona-muropeptide under different intensities of tension. The nona-muropeptide on being newly linked into the stress-bearing wall is not stressed, but responds to increasing stress on it and elongates by altering its conformation. This change increases access to the “tail-to-tail” bond of the muropeptide as well as access of new penta-muropeptides to the unbounded groups on the nona-muropeptide. This model could function in a quite simple manner compared to the other two earlier models, mentioned above.
Although the cylindrical sidewall contains both old and new murein, the recent observation (see Chapter 11) is that the sidewall is composed of patches of old and new material. The size of the patches is not constant and ranges roughly to about 100 nm. Consequently, these patches consist of hundreds of oligoglycan chains both across and in length. In toto, this finding is inconsistent with the earlier models in the literature about how the sidewall and poles of the Gram-negative rod grow and divide.

The understanding of the mechanisms that allow the sacculus to grow and divide are important, however, it is the mechanisms that control (or are controlled by) the replication of the chromosome so that the process of cell growth and division and chromosome replication, which are equally important, will occur harmoniously.

THE GRAM-NEGATIVE WALL STRUCTURE

The structure of the Gram-negative wall is shown diagrammatically in Figure 13.1. Although we usually talk of three layers there really are several more. Starting from the inside these are:

(1) The two leaflets of the cytoplasmic membrane. Imbedded in this layer are proteins, some of which bridge the membrane and others that do not. Present are special molecules like the bactoprenol that aids the exporting of the penta-muropeptide from the cytoplasm and proteins for transport processes. There are several different processes, moreover, for protein export to the periplasmic space and beyond.

(2) The peptidoglycan layer. This has to completely surround the cell in a covalent network. It must also contain special proteins for export function to allow certain proteins and toxins to be extruded through the murein.

Figure 13.1 The Gram-negative wall in cross-section.