Study Guide for Biofilms and Quorum Sensing

We covered this stuff quickly and briefly, so the expectations are similar.

Biofilms - communities of bacteria usually at surface-liquid interfaces. Usually are mixtures of different bacteria. Free (individual) bacteria are called planktonic.

Steps in biofilm development (from review article on food biofilms):

Stage 1: reversible attachment of bacterial cells to a surface.

Stage 2: irreversible attachment - exopolymeric material

Stage 3: formation of microcolonies.

Stage 4: mature biofilm with a 3-dimensional structure containing cells packed in clusters with channels between the clusters that allow transport of water and nutrients and waste removal

Stage 5: detachment and dispersion of cells from the biofilm.

Because of limited nutrients, most cells in the biofilms are not replicating. Therefore, they are resistant to antibiotics, environmental stress, host defenses, etc.

Biofilms are a form of developmental biology for organisms. They go from planktonic to biofilms to planktonic. Because the bacteria change their physiology so much, there must be some serious gene regulation going on. This can be regulated by quorum sensing.

Here is the definition from the Annual Review article: "Quorum sensing is a cell-cell communication process in which bacteria use the production and detection of extracellular chemicals called autoinducers to monitor cell population density." It can regulate many things from light production, to virulence genes, to biofilms. Quorum sensing can turn things off, as well as turning things on.

Quroum sensing is somewhat different between gram-positive and gram-negative bacteria. G+ use peptides as autoinducers, G- use other small molecules. Autoinducer-1 molecules tend to be species specific and are acyl homoserine lactones (I will not ask you to draw them out!). AI-1s are used to ask, "How many of my type are here?". AI-2s are more conserved and are sensed among diverse species. AI-2s are used to ask, "How many bacteria in general are here?".

Let's focus on the Vibrio harveyi model for terminology and concept. It is in the Annual Review article and likely in the text (I don't have it with me right now).

The autoinducers are produced by different genes, whose names are not important. For *V. harveyi*, the autoinducers are recognized by membrane receptors. In a low AI state (low bacterial density), they act as kinases to phosphorylate in succession two intermediate proteins: LuxU and LuxO. LuxO stimulates the production of small regulatory RNAs, sRNAs, that repress the quorum sensing-regulated genes by decreasing the synthesis of LuxR, usually an activator of QS-regulated genes. The sRNAs are aided in their binding to the luxR mRNA by the Hfq protein, and this leads to destabilization of the luxR mRNA. So note that this is phosphorylation cascade that leads to repression.

Conversely, when AI levels are high, the membrane proteins act as phosphatases, and LuxO is not phosphorylated. The sRNAs are not made, so LuxR is made and turns on (or off) the appropriate genes.