Slide 1

During this presentation I will talk about the different infection cycles a phage can follow.

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Some phages have a strict lytic lifestyle, meaning that upon infection the bacteriophage will hijack the host's machinery for production of new virions. These kind of viruses are called virulent phages and will inevitably kill their host cell.

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A different approach is opted by temperate phages. Besides following the lytic cycle, these viruses can also opt to enter the lysogenic cycle. The phage then integrate its DNA into the host chromosome via recombination. As the phage genome is replicated in synchrony with the host chromosome, it will be passed on from one generation to the next. Bacterial cells harboring a phage genome are termed lysogens while the inserted phage genomes are called prophages.

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During lysogeny the temperate virus does not exist as a virus particle in the cell but is integrated linearly in the host chromosome and all the genes encoding for enzymes and proteins associated with lytic cycle will be repressed. This control is typically due to a phage-encoded repressor protein. During this lysogenic state the repressor is expressed and will repress the left and the right promoter by binding to it.

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The virus repressor protein does not only inhibit transcription from the genes located on its own prophage, it also prevents gene expression by any identical or closely related virus that tries to infect the same host cell. This results in the lysogens having immunity to superinfection by the same type of virus or related viruses.

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Although phages usually proliferate by depletion of their host's resources followed by cell lysis, they are also utterly dependent on them for survival and replication. It therefore lies in the best interest of both parties that the bacterium thrives well. Mutually beneficial interactions evolved in which the resident phage contributes to the virulence and/or fitness of the host. The cholera toxin of *Vibrio cholera* and the SopE effector protein of *Salmonella enterica* are two examples of such beneficial interplay. These pathogeny increasing contributions are typically caused by the expression of virulence factors encoded on the prophage.

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If the phage repressor is inactivated or if its synthesis is prevented the prophage is induced. New virions are produced and the host cells is lysed. Altered environmental conditions, especially damage to the host cell DNA by UV, induces the lytic pathway in some cases. However, spontaneous induction remains a rather rare event. If the virus loses the ability to leave the host chromosome is called a cryptic phage.

Slide 8

Phages with a strict lytic lifestyle inevitably kill the infected cell, leading to a drastic reduction of available hosts. A safer approach is opted by temperate phages that can decide to insert their DNA into the host chromosome, although this protection comes at the expense of an impairment in spread of progeny. A possible solution to circumvent this dilemma is the formation of a phage carrier state or pseudolysogeny in which a delay of the integration event allows the growth of a phage free subpopulation. Due to overexpression of immunity factors in the phage carrier state, these phage free siblings will be transient resistant to superinfection. However these factors will be diluted out over the next generations and the cells will become susceptible again for infection. This strategy allows these farming phages to foster a reservoir of bacterial cells that afterwards can be “harvested”.