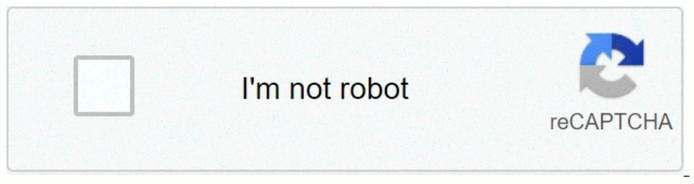

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T4 bacteriophage pdf

virus species Atomic structural model bacteriophage T4 Escherichia virus T4 Virus classification (unrated): Virus Realm: Duplodnaviria Kingdom: Heunggongvirae Phylum: Uroviricota Class: Caud Order: Caudovirales Family: Myoviridae Genus: Tequatrovirus Species: Escherichia virus T4 Strains[1] Enterobacteria phage C16 Enterobacteri phage F10 Enterobacteria phage Fs-alpha Enterobacteria phage PST Enterobacteria phage SKII Enterobacteria phage SKV Enterobacteria phage SKX Enterobacteria phage SV3 Enterobacteria phage T2 Enterobacteria phage T4 Enterobacteria phage T6 Synonyms[2] Enterobacteria phage T4 Escherichia virus T4 is a type of bacteriophage that infect Escherichia coli bacteria. It is a two-stranded DNA virus in the sub-family Tevenvirinae of the family Myoviridae. T4 is able to undergo only a lytic life cycle, not a lysogenic life cycle. This species was previously named T-i bacteriophage, a name that also includes, among others, strains (or isolates), Enterobacteria phage T2, Enterobacteria phage T4 and Enterobacteria phage T6. Bacteriophage means eating bacteria, and phages are well known for being mandatory intracellular parasites that multiply in the host cell and are released when the host is destroyed by lysis. These virulent viruses contain about 160 genes and are among the largest and most complex viruses known and one of the best studied model organisms. They played a key role in the development of virology and molecular biology. [3] [4] Used in research dating back to the 1940s and continuing today, T-i phases are considered to be the best studied model organisms. Model organisms are usually required to be simple with as few as five genes. Still, T-i rumors are actually among the largest and highest complexity virus in which these phases of genetic information consists of about 160 genes. By coincidence with their complexity, T-i viruses have been found to have an unimaginable feature of any other, the presence of unusual basic hydroxymethylcytosin (HMC) instead of nucleic acid base cytosine. In addition, HMC residues on Fág T-i are glucosylated in a specific way. This unique property enabled the formation of new enzymes that never existed in T-even infected cells or other cells, and modifying T-i phage DNA provided basic basic progress in viral and molecular levels. Another unique feature of the T-even virus is its regulated gene expression. [5] These unique qualities and other characteristics have given meaning to T-even Which includes transduction, which is responsible for the transfer of drug-resistant properties, lysogenous conversion is responsible for acquiring new properties, such as the formation of new enzymes, accidental insertion into the bacterial chromosome can provoke an injection mutation, epidemiological typical typing of bacteria (phage typing), phags are widely used in genetic engineering, where they serve as a cloning village. Library genes and monoclonal antibodies are kept in phases. In addition, they are responsible for the natural removal of bacteria from water bodies. [6] The genome and structure of the T4 virus's two-lemt GENOME is about 169 kbp long and encodes 289 proteins. The T4 genome is fatally redundant and is first replicated as a unit, then several genomic units are recombined end-to-end and form con contortions. When packaged, the stringing is cut in unspecified positions of the same length, resulting in several genomes representing circular permutations of the original. [8] The T4 genome carries eukaryote intron sequences. The translation of the Shine-Dalgarno GAGG sequence dominates the T4 virus's early genes, while the GGAG sequence is the target for the T4 endonuclease RegB, which initiates early mRNA degradation. [9] Virus particle structure Structural overview T2 phage T4 is a relatively large virus with a width of approximately 90 nm and a length of 200 nm (most viruses range from 25 to 200 nm in length). The DNA genome is held in the icosahedral head, also known as capsido. [10] The tail of T4 is hollow, so it can transfer its nucleic acid to the cell it infects after connection. Myoviridae phases like T4 have complex contractile tail structures with a large number of proteins involved in the tail assembly and function. [11] Tail fibers are also important in recognizing the surface receptors of host cells, so they determine whether the bacteria is within the host range of the virus. [12] The structure of motherboard 6 megadalton T4, which contains 127 polypeptide chains of 13 different proteins (gene products 5, 5,4, 6, 7, 8, 9, 10, 11, 12, 25, 27, 48 and 53), was recently described in detail in atomic. An atomic model of the proximal region of the tail tube consisting of gp54 and the main tube protein gp19 was also created. Tape measuring protein gp29 is present in baseplate-tail tube complexes, but could not be modeled. [13] Infectious process T4 virus initiates infection of Escherichia coli by binding OmpC porin and lipopolysaccharide (LPS) proteins on the surface of E. coli cells with long tail fibers (LTF). [14] [15] A recognition signal is sent to the motherboard via LTF. This reveals short tail fibers (STF) that bind irreversibly to the surface of E. coli cells. The base plate changes the conformation and the tail shell shrinks, causing gp5 at the end of the tail tube to pierce the outer membrane of the cell. [16] The GP5 lysozymic domain is activated and degrades the periplasmic peptidoglycan layer. The rest of the membrane is degraded, and then the DNA from the head of the virus can travel through the tail tube and enter the E. coli cell. Reproduction Of the Lytic life cycle (from the entry of the bacterium to its destruction) lasts approximately 30 minutes (at 37 °C). Virulent bacteriophages multiply in their bacterial host immediately after entry. After the number of offspring reaches a certain amount, the lyse host causes the therefore, new host cells would be released and infected. [17] The process of hosting lyses and releasing is called a lytic cycle. The lytic cycle is a cycle of viral reproduction, which involves the destruction of the infected cell and its membrane. This cycle involves a virus that overtakes the host cell and its machines for reproduction. Therefore, the virus must go through 5 stages in order to multiply and infect the host cell: Adsorption and penetration (from immediate start) Arrest of the expression of the host gene (starting immediately) Enzyme synthesis (starting with 5 minutes) DNA replication (starting in 10 minutes) Formation of new viral particles (starting in 12 minutes) After the completion of the life cycle, the host cell opens and ejects newly built-in viruses into the environment , destroying the host cell. T4 has an explosion size of approximately 100-150 viral particles per infected host. Tests for replenishment, deletion and recombi combination can be used to map the rII gene loci using T4. These escherichia viruses infect host cells with their information and then blow up the host cells and thus spread themselves. Adsorption and penetration Diagram of the DNA injection process Like all other viruses, T-i rumors are not just randomly attached to the surface of their host; instead, they look for and bind to receptors, specific protein structures that are located on the surface of the host. These receptors differ with phage; teichoic acid, cell wall proteins and lipopolysaccharides, flagella and saws can serve as receptors for phages to bind to. In order for T-i phage to infect its host and begin its life cycle, it must enter the first process of infection, adsorption of the phage to the bacterial cell. Adsorption is a value characteristic of a phago-host pair, and the adsorption of the phague on the surface of the host cell is illustrated as a two-stage process: reversible and irreversible. This includes a phague tail structure that begins when the phague tail fiber helps bind the phage to the appropriate receptor of its host. This process is reversible. One or more components of the motherboard mediate the irreversible process of binding the phage to the bacterium. Penetration is also a value characteristic of phag-host infection, which involves injecting genetic material phages inside bacteria. Penetration of nucleic acid takes place after the irreversible adsorption phase. Mechanisms involving the penetration of phág nucleic acid are specific to each phase. This penetration mechanism may include electrochemical membrane potential, ATP molecules, enzymatic cleavage of the peptidoglycan layer or all three of these factors may be vital for the penetration of nucleic acid inside the bacterial cell. Studies have been conducted on the T2 bacteriophage (T4-like phage) penetration mechanism and it has been shown that the phague tail does not penetrate inside the bacterial cell wall and the penetration of this phage involves membrane potential on the inner membrane. [18] [19] Vir T4 genome replication and packaging is synthesized in the host cell using rolling circle replication. The time it took to replicate DNA in a living cell was measured as the rate at which the DNA of the virus T4 in E. coli infected with the virus was extended. [20] During the exponential increase in DNA at 37°C, the rate was 749 nucleotides per second. The mutation rate per base pair per replication during T4 DNA synthesis is 1.7 to 10-8,[21] a highly accurate MECHANISM of DNA copying, with only 1 error in 300 copies. The virus also encodes unique DNA repair mechanisms. The head of the T4 Phage is assembled empty around the scaffolding, which is later degraded. As a result, DNA must enter the prohead through the small pores, which is achieved by the hexamer gp17 interacting with the DNA first, which also serves as an engine and nuclease. The T4 DNA packaging engine was found to load DNA into virus pockets at speeds of up to 2,000 base pairs per second. The power, if increased, would be equal to that of the average car engine. [22] Transduction occurs within the lytic cycle, where bacterial DNA units are packaged in phage capsid. [23] Release The last step in virus reproduction and multiplication is determined by releasing virions from the host cell. The release of virions occurs after breaking the bacterial plasma membrane. Unseen viruses lyze the host cell, which is characterized by viral proteins attacking peptidoglycan or membrane. Lysis of bacteria occurs when the capsids inside the cell release the enzyme lysozyme, which break down the cell wall. Released bacteriophages infect other cells, and in these cells a viral multiplier cycle is repeated. The lysogenous cycle of Phage2 Lysogenic Phages multiplies in one of two ways; either by entering an inactive or latent state or by multiplying it by the lytic phase. Through a process known as lysogens, phág DNA is replicated with replication of the host chromosome by assimilation to the host chromosome itself. It is then passed to its daughter cells; that's why it's usually not recognized by the host. A process known as lysogenous or fage conversion alters the properties of cell bacteria; this is possible because the prophage itself contains genes that can represent new properties to the cell bacteria or host cells. These bacteria are considered lysogenized. Lysogenized bacteria are resistant to superinfection by the same or related phases. This is known as superinfective immunity. [24] Multiplicity Reactivation Survival curve for T4 virus with UV-damaged DNA (top) or MMC (bottom) after one T4 virus infecting host cells (monocomplexes) or two or more T4 viruses simultaneously infecting host cells (multicomplexes). Multiplicity reactivation (MR) is a process in which two or more virus genomes, each containing inactivation damage to the genome, can interact cells to create a viable virus genome. Salvador Luria, studying UV irradiated T4 virus in 1946, discovered an MRI and suggested that the observed reactivation of the damaged virus occurs using a recombinant mechanism. (see referee. [25] [26] [27]) Prior to that, DNA confirmation as genetic material in 1952 in a related T2 virus was preceded by the Hershey-Chase experiment. [28] As Luria recalled (1984,[29] p. 97], the discovery of the reactivation of the irradiated virus (known as multiple reactivation) immediately began a wave of activity in the study of radiation damage repair within the early phase group (reviewed by Bernstein[30] in 1981). It later emerged that repairing the damaged virus with the mutual help Luria had discovered was just one special case of DNA repair. Cells of all types, not only bacteria and their viruses, but all the organisms examined, including humans, are now known to have complex biochemical processes to repair DNA damage (see DNA repair). DNA repair processes are also now recognized as playing a key role in protecting against aging, cancer, and infertility. MRI is usually represented by survival curves, where the survival of plaque forming the ability to multiply infected cells (multicomplexes) is plotted against the dose of the genome of the harmful agent. By comparison, the survival of viral plaque forming the ability of individually infected cells (monocomplexes) is also rendered against the dose of the genome of the harmful agent. The top image shows survival curves for T4 multicomplexes and monocomplexes with increasing doses of UV light. Since survival is carried out on the log scale it is clear that the survival of multicomplexes exceeds monocomplexes by very large factors (depending on the dose). The UV inactivation curve for multicomplexes has an initial arm. Other DNA-damaging substances of the T4 virus with arms in multicomplex survival curves are X-rays[31][32] and ethylmethane sulfonate (EMS). [30] The presence of the arm has been interpreted as using two recombinant processes. [33] The first one corrects DNA with high efficiency (in the shoulder), but is saturated in its ability as the damage increases; The second path works at all damage levels. Surviving T4 virus released from multicomplexes shows no increase in mutation, suggesting that MR UV irradiated virus is an accurate process. [33] The bottom image shows the survival curves for inactivation of the T4 virus by mitomycin C (MMC). In this case, the survival curve for multicomplexes has no initial arm, indicating that only the second process of recombinant repair described above is active. The effectiveness of repair by this process is indicated by observing that the dose of MMC, which allows only 1 in 1000 monocomplexes to survive, allows for the survival of about 70% of multicomplexes. Similar multicomplex survival curves (without arms) were also obtained for DNA-damaging substances P32 decay, psoralen plus almost UV irradiation N-methyl-N'-nitro-N-nitrosoguanidine (MNNG), methylmethane sulfonate (MMS) and nitric acid. [30] Several genes that were necessary for MRI in the T4 virus turned out to be ortologists of genes necessary for recombi combination in prokaryots, eukaryots and archaea. This includes, for example, the T4 uvsX gene[34], which specifies a protein that has a three-dimensional structural homology for RecA from Escherichia coli and the homologous protein RAD51 in eukaryotes and RadA in the archa. It has been suggested that effective and accurate recombinant repair of DNA damage during MRI may be similar to the recombinant repair process that occurs during meiosis in eukaryotes. [35] The history of the Bacteriophages was first discovered by english scientist Frederick Twort in 1915 and Félix d'Herrell in 1917. In the late 1930s, T.L. Rakieten proposed either a mixture of raw sewerage or lysate from E.coli infected raw sewers to two researchers Milislav Demerec and Ugo Fano. These two researchers isolated T3, T4, T5 and T6 from E.coli. Also, in 1932, researcher J.Bronfenbrenner studied and worked on T2 phage, in which T2 phage was isolated from the virus. [36] This insulation was made of fecal material rather than sewerage. In any case, Delbruck was involved in the discovery of T i phages. It included the designation of bacteriophages in type 1 (T1), type 2 (T2), type 3 (T3), etc. The specific time and place of isolation of the T4 virus remains unclear, although they are likely to have been found in wastewater or fetal material. T4 and similar viruses were described in Thomas F. Anderson, Max Delbrück, and Milislav Demerec in November 1944. [37] A number of Nobel laureates have collaborated with viruses similar to T4 or T4 viruses, including Max Delbrück, Salvador Luria, Alfred Hershey, James D. Watson, and Francis Crick. Other prominent scientists who have worked with T4 include Michael Rossmann, Seymour Benzer, Bruce Alberts, Gisela Mosig Richard Lenski and James Bull. See also Viruses portal T4 rII system T2 phage T6 phage Bacteriophage Virology References ^ ICTV 9th Report (2011) Myoviridae. International Committee on Virus Taxonomy (ICTV). December 26, 2018. ↑ ICTV Taxonomy history: Escherichia virus T4. International Committee on Virus Taxonomy (ICTV). 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(T4-like phag insulation, including phage Ox2) External Links Viralzone: T4-like Viruses Animation T4 Bacteriophage infect E.coli Animation T4 Bacteriophage DNA Packs Obtained from