

NEXTILLO OTOP
MAY MONTH

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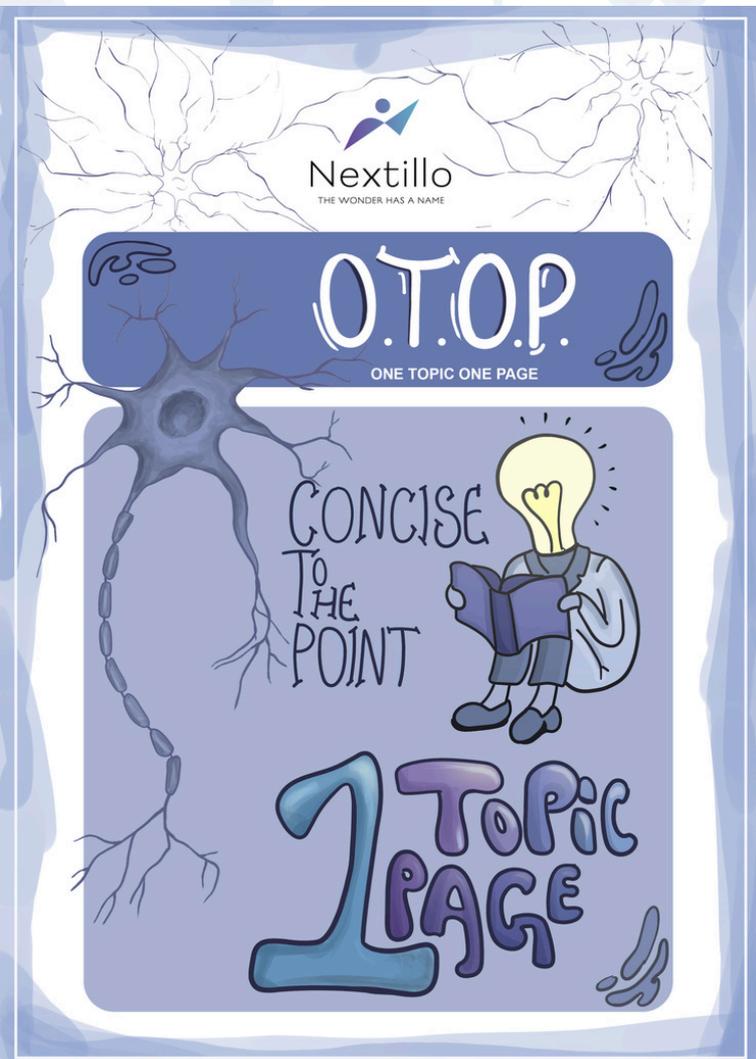
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BACTERIAL GENETICS

Genetic Organisation and Regulation of Bacterial Cell

All properties of a bacterial cell are determined by the genetic information contained within the cell genome. This information is normally encoded by the DNA of the cell. Most of the bacterial cell nucleus is arranged in the form of a circular chromosome of a double-stranded DNA molecule. The DNA is not associated with protein or histone molecules as it is in eukaryotic cell chromosomes.

Extrachromosomal Genetic Element

In addition to the single main chromosome, bacterial cells may also carry one or more small circular extrachromosomal elements termed plasmids. They often carry supplementary genetic information coding for beneficial properties (e.g., resistance to antibiotics) that enable the host cell to survive under a particular set of environmental conditions.

Properties of Plasmids

- Circular DNA in the cytoplasm
- Replicated independently
- Transferred from one cell to another
- Important tool in genetic engineering
- Not essential for the vital function of the host bacteria, but they may carry properties of drug resistance and toxigenicity

Classification of Plasmids

1. Based on Ability to Perform Conjugation

- *Conjugative Plasmids:* Possess the ability to transfer themselves to other bacteria by means of conjugation (self-transmissible or conjugative plasmids).
- *Non-conjugative Plasmids:* Cannot transfer.

2. Based on Compatibility Between the Plasmids

- *Compatible Plasmids:* Different plasmids can exist in a single bacterial cell only if they are compatible with each other.
- *Incompatible Plasmids:* If two plasmids are not compatible, one or the other will be rapidly lost from the cell.

3. Based on Function

- Fertility or F-plasmids
- Resistance (R) plasmids
- Col plasmids
- Virulence plasmids
- Metabolic plasmids



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BACTERIAL GROWTH

Binary Fission

Bacteria reproduce by binary fission, which is a form of asexual reproduction and cell division.

Bacterial Cell Division

- Firstly, the bacterial cell replicates its DNA.
- The cytoplasmic membrane extends, and nucleic acids separate.
- Invagination of the membrane occurs.
- A septum-type wall forms in between.
- Separation and formation of daughter cells.

Generation Time

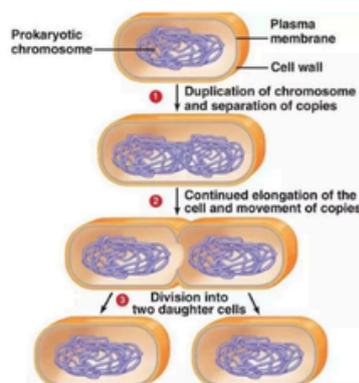
It is the time taken for the number of bacteria to double or the duration of the growth cycle. On average, it is usually about 20 minutes.

Some exceptions:

- *Lepra bacilli*: 12 to 13 days
- *Mycobacterium tuberculosis*: 14 hours

Bacterial Count

- **TOTAL COUNT**: Total bacteria (live + dead).
- **VIABLE COUNT**: Number of living (viable) cells.





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TUMOR ANTIGENS

Tumor antigens are molecules present on the surface of tumor cells that can be recognized by the immune system. tumor antigens is important for developing targeted therapies and vaccines against cancer. Immunohistochemistry (IHC) and molecular techniques are often used to detect specific tumor antigens from biopsy samples.

Tumor-associated antigens (TAAs)

- These antigens are present on both tumor cells and some normal cells but are overexpressed in tumor cells. They include:
- Differentiation antigens: Antigens expressed during normal differentiation but overexpressed in certain tumors.
- Mutated antigens: Products of mutated genes specific to tumor cells.
- Viral antigens: Antigens of oncogenic viruses that can integrated into the host genome.
- Oncofetal antigens: Antigens normally expressed during fetal development but re-expressed in certain tumors.

Tumor-specific antigens (TSAs)

- These antigens are unique to tumor cells and are not found in normal tissues. They arise only from mutations, gene rearrangements, or viral integration events specific to that tumor.

Neoantigens

- They arise from mutations in tumor cells and are **recognized as foreign by the immune system**. They are specific to individual patients and their tumors

Neoantigen	Associated Cancer
KRAS G12D	Pancreatic cancer, colorectal cancer, lung cancer
BRAF V600E	Melanoma, colorectal cancer
EGFRvIII	Glioblastoma multiforme (GBM)
TP53 mutations	Various cancers including breast cancer, lung cancer
PTEN mutations	Prostate cancer, glioblastoma, endometrial cancer
APC mutations	Colorectal cancer
Human papillomavirus (HPV) antigens	Cervical cancer, head and neck cancer
Epstein-Barr virus (EBV) antigens	Nasopharyngeal carcinoma, Hodgkin lymphoma

Tumor Antigen	Associated Cancer
HER2/neu (ERBB2)	Breast cancer, gastric cancer
PSA (Prostate-specific antigen)	Prostate cancer
CEA (Carcinoembryonic antigen)	Colon cancer, pancreatic cancer, breast cancer
CA-125	Ovarian cancer
AFP (Alpha-fetoprotein)	Hepatocellular carcinoma, germ cell tumors
Melan-A (Melanoma antigen)	Melanoma
WT1 (Wilms' tumor 1)	Wilms' tumor (nephroblastoma), leukemia
MUC1 (Mucin 1)	Breast cancer, pancreatic cancer
NY-ESO-1	Melanoma, synovial sarcoma, multiple myeloma



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LYMPHOMA

Lymphomas are cancers of the lymphatic system, involving lymphocytes. They are broadly classified into Hodgkin lymphoma (HL) and non-Hodgkin lymphoma (NHL). imaging studies (CT, PET-CT), biopsy (excisional or core needle), and immunophenotypings are used to determine cell lineage and subtype.

Hodgkin Lymphoma (HL):

- Characterized by the presence of Reed-Sternberg cells, large abnormal B lymphocytes, within a background of inflammatory cells.
- Subtypes include nodular sclerosis (MC), mixed cellularity, lymphocyte-rich (good prognosis), and lymphocyte-depleted (rare subtype) with poorest prognosis.
- Diagnosed through lymph node biopsy, often presenting with painless lymphadenopathy, fever, night sweats, weight loss (B symptoms), and pruritus.
- Reed-Sternberg cells are large, multinucleated cells with prominent nucleoli and characteristic "owl-eye" appearance due to their binucleation.

Non-Hodgkin Lymphoma (NHL):

- Heterogeneous group of lymphoid malignancies with various histological subtypes.
- Can originate from either B cells, T cells, or natural killer (NK) cells.
- Subtypes include diffuse large B-cell lymphoma (DLBCL), follicular lymphoma (FL), mantle cell lymphoma (MCL), and peripheral T-cell lymphoma (PTCL).
- Presentation varies depending on subtype which commonly involving lymphadenopathy, B symptoms (fever, night sweats) and extranodal involvement.

Classification	Subtypes	Examples
Hodgkin Lymphoma	Classical Hodgkin Lymphoma (cHL)	Nodular sclerosis, Mixed cellularity, Lymphocyte-rich, Lymphocyte-depleted
	Nodular Lymphocyte-Predominant Hodgkin Lymphoma (NLPHL)	
Non-Hodgkin Lymphoma	B-cell Neoplasms	Diffuse large B-cell lymphoma (DLBCL), Follicular lymphoma (FL), Mantle cell lymphoma (MCL), Burkitt lymphoma
	T-cell Neoplasms	Peripheral T-cell lymphoma (PTCL), Anaplastic large cell lymphoma (ALCL), Adult T-cell leukemia/lymphoma (ATLL)
	Precursor lymphoid neoplasms	Acute lymphoblastic leukemia (ALL), Acute myeloid leukemia (AML) with lymphoid differentiation



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MEDIATORS OF INFLAMMATION

Mediators of inflammation are a diverse array of molecules produced in response to tissue injury, infection, or immune stimulation. Mediators of inflammation include cytokines, chemokines, lipid mediators (such as prostaglandins and leukotrienes), histamine, complement proteins, oxygen-derived free radicals, and platelet-activating factor.

Cytokines:

- Signaling molecules secreted by various cells, including immune cells, to regulate inflammation. Examples include:
- Interleukins (IL): IL-1, IL-6, IL-8, IL-12, IL-17, IL-18, etc., involved in immune cell activation, recruitment, and differentiation.
- Tumor necrosis factor-alpha (TNF- α): Promotes inflammation, induces fever, and activates endothelial cells.
- Interferons (IFN): IFN- α , IFN- β , IFN- γ ; involved in antiviral defense, macrophage activation, and immune regulation.

Chemokines:

- Small cytokines that induce chemotaxis and guide leukocyte migration to sites of inflammation. Examples include:
- CXCL8 (IL-8): Chemotactic for neutrophils.
- CCL2 (MCP-1): Attracts monocytes/macrophages.
- CXCL10 (IP-10): Induces migration of T cells.

Prostaglandins:

- Lipid mediators derived from arachidonic acid metabolism. Examples include prostaglandin E2 (PGE2) and prostacyclin (PGI2), which promote vasodilation, pain, and fever.

Leukotrienes:

- Lipid mediators synthesized from arachidonic acid by leukocytes. Examples include leukotriene B4 (LTB4), which attracts neutrophils, and cysteinyl leukotrienes (LTC4, LTD4, LTE4) which induce bronchoconstriction and vascular permeability.

Histamine:

- Released by mast cells and basophils, causes vasodilation, increased vascular permeability, and smooth muscle contraction.

Complement system:

- Series of plasma proteins that enhance inflammation, opsonization, and cell lysis. Activation products, including C3a, C5a (anaphylatoxins), and the membrane attack complex (C5b-9), promote inflammation.

Oxygen-derived free radicals:

- Generated by activated neutrophils and macrophages, cause tissue damage and contribute to inflammation.

Platelet-activating factor (PAF)

- Phospholipid mediator that induces platelet aggregation, leukocyte activation, and endothelial cell injury.