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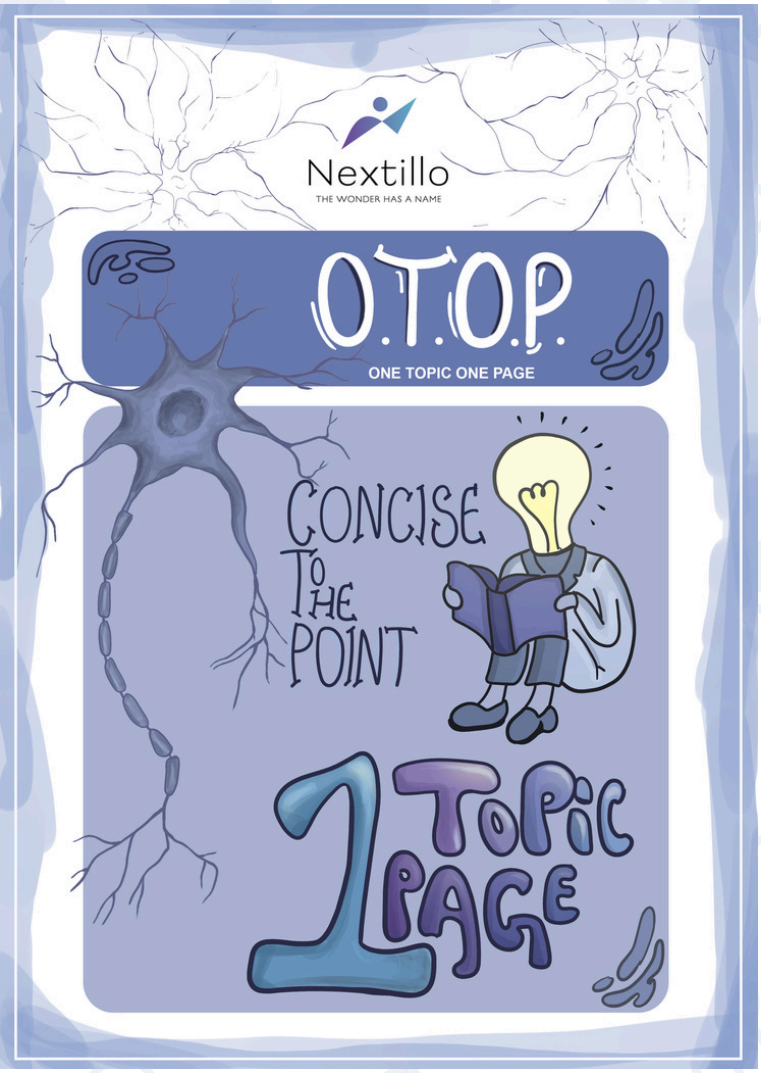

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PROSTAGLANDINS

Prostaglandins are lipid compounds derived from arachidonic acid metabolism. They are synthesized by cyclooxygenase enzymes (COX-1 and COX-2) and play diverse roles in physiological and pathological processes.

Synthesis:

- *Arachidonic acid, released from cell membrane phospholipids, is converted into prostaglandin H2 (PGH2) by cyclooxygenase enzymes.*
- *PGH2 is then converted into various prostaglandins by specific synthases.*

Types:

- *Prostaglandins include PGD2, PGE2, PGF2α,*
- *PGI2 (prostacyclin made via COX-2), and thromboxane A2 (TXA2 is made via COX-1).*

Functions:

- *Inflammation: Promote vasodilation, increased vascular permeability, and leukocyte chemotaxis.*
- *Pain Sensation: Sensitize pain receptors and contribute to pain perception.*
- *Fever: Induce fever by acting on hypothalamus.*
- *Gastric Protection: Enhance mucus production and bicarbonate secretion in the stomach, protecting against ulcer formation.*
- *Reproductive System: Regulate uterine contraction, ovulation, and cervical ripening.*

Effect on Platelets:

- *Prostaglandins exhibit a dual effect on platelets. PGI2 inhibits platelet aggregation and promotes vasodilation, reducing clot formation and blood vessel constriction. Conversely, TXA2 promotes platelet aggregation and vasoconstriction, enhancing clot formation and blood vessel constriction.*

Drug Interactions:

- *Nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit COX enzymes, reducing prostaglandin synthesis. Non selective COX inhibitors can lead to adverse effects such as gastrointestinal ulcers, renal dysfunction, and impaired platelet function due to decreased PGI2 and TXA2 production.COX-2 inhibitors are contraindicated in heart disease as they inhibit formation of PGI which inhibits platelet aggregation.*

Dysregulation:

- *Dysregulation of prostaglandin synthesis or signaling pathways can contribute to various pathological conditions such as inflammation, pain, fever, and cardiovascular diseases.*

Summary:

- *Prostaglandins are essential mediators of inflammation, pain, and fever. They also play critical roles in maintaining homeostasis in the gastrointestinal tract, regulating reproductive processes, and modulating vascular tone.*

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CYTOKINES

Cytokines are a diverse group of small proteins or glycoproteins that are important in cell signaling. They are produced by various cells in the body, including immune cells such as T cells, B cells, macrophages, and dendritic cells, as well as non-immune cells like fibroblasts and endothelial cells.

Interleukins (ILs):

- *Synthesis: Produced mainly by leukocytes, although other cell types can also produce them.*
- *Functions: Regulate immune responses, including inflammation, differentiation, and proliferation of immune cells.*
- *Diseases: Various autoimmune diseases, inflammatory disorders, and infectious diseases.*
- *Examples: IL-1 (pro-inflammatory) , IL-2, IL-4, IL-6, IL-10(anti-inflammatory).*

Tumor Necrosis Factor (TNF) Family:

- *Synthesis: Produced by activated macrophages, T cells, and other immune cells.*
- *Functions: TNF-alpha Regulates inflammation, immune system function whereas TNF beta plays an important role in lymphocytes activation and proliferation.*
- *Diseases: Rheumatoid arthritis, inflammatory bowel disease, psoriasis.*
- *Examples: TNF-alpha, TNF-beta (also known as LT-alpha).*

Interferons (IFNs):

- *Synthesis: Produced by various cell types, including leukocytes, fibroblasts, and endothelial cells, in response to viral infections and other stimuli.*
- *Functions: IFN-alpha has Antiviral activity, modulation of immune responses, inhibition of cell proliferation.*
- *Diseases: Viral infections, certain cancers, autoimmune diseases.*
- *Examples: IFN-alpha, IFN-beta, IFN-gamma.*

Chemokines:

- *Synthesis: Produced by various cell types, including leukocytes, fibroblasts, and endothelial cells.*
- *Functions: CCL2 (MCP-1) Regulate migration and activation of immune cells, inflammation, and angiogenesis.*
- *Diseases: Inflammatory diseases, autoimmune diseases, cancer.*
- *Examples: CXCL8 (IL-8), CCL2 (MCP-1), CXCL10 (IP-10).*

Transforming Growth Factor-beta (TGF-beta) Family:

- *Synthesis: Produced by various cell types, including immune cells, fibroblasts, and epithelial cells.*
- *Functions:TGF-beta1 Regulate cell growth, differentiation, apoptosis, immune responses, wound healing.*
- *Diseases: Cancer, fibrosis, autoimmune diseases.*
- *Examples: TGF-beta1, TGF-beta2, TGF-beta3.*

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WOUND HEALING

Wound healing is a complex process that involves various stages and mechanisms aimed at repairing damaged tissue. There are typically four overlapping phases of wound healing: hemostasis, inflammation, proliferation, and remodeling.

Hemostasis

- *Type: Initial response to injury involving vasoconstriction and platelet aggregation to form a clot.*
- *Activators: Exposure of collagen and tissue factor, leading to platelet activation and aggregation.*
- *Inhibitors: Antithrombin, protein C, tissue factor pathway inhibitor (TFPI).*
- *Function: Prevent excessive bleeding by forming a temporary plug (hemostatic clot) at the site of injury.*
- *Pathological Findings: Excessive bleeding due to coagulation factor deficiencies or platelet disorders can lead to impaired hemostasis.*

Inflammation

- *Type: Inflammatory cells migrate to the wound site to remove debris and prevent infection.*
- *Activators: inflammatory mediators such as histamine, prostaglandins, and cytokines.*
- *Inhibitors: Anti-inflammatory cytokines (e.g., IL-10), glucocorticoids.*
- *Function: Clean debris, bacteria, and damaged tissue; promote angiogenesis; and initiate the repair process.*
- *Pathological Findings: Chronic inflammation can delay wound healing and lead to excessive scarring or fibrosis.*

Proliferation:

- *Type: Phase characterized by tissue granulation, angiogenesis, and epithelialization.*
- *Activators: Growth factors such as transforming growth factor-beta (TGF-β), vascular endothelial growth factor (VEGF), and fibroblast growth factor (FGF).*
- *Inhibitors: TGF-β inhibitors (galunisertib, perfenidone), anti-angiogenic factors (thromobospondin-1)*
- *Function: Rebuilding of damaged tissue by deposition of extracellular matrix (ECM), proliferation of fibroblasts, and formation of new blood vessels.*
- *Pathological Findings: Excessive proliferation can lead to hypertrophic scars or keloids, while inadequate proliferation results in chronic wounds.*

Remodeling:

- *Type: Maturation and remodeling of the newly formed tissue.*
- *Activators: Metalloproteinases (MMPs), tissue inhibitors of metalloproteinases (TIMPs).*
- *Inhibitors: inhibitors of collagen synthesis like beta aminopropionitrile, doxycycline.*
- *Function: Reorganization and strengthening of collagen fibers, reduction in vascularity, and contraction of the wound.*
- *Pathological Findings: Dysregulated remodeling can result in abnormal scar formation, such as hypertrophic scars, keloids, or chronic wounds with impaired healing.*

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INTEGRINS

Integrins are a family of cell surface receptors that mediate cell-cell and cell-extracellular matrix interactions. They play essential roles in various cellular processes such as cell adhesion, migration, proliferation, differentiation, and signal transduction.

αvβ3 Integrin

- *Synthesis: Synthesized by various cell types, including endothelial cells, osteoclasts, and certain tumor cells.*
- *Function: Mediates cell adhesion to extracellular matrix proteins like vitronectin and fibronectin, involved in angiogenesis, bone resorption, and tumor metastasis.*
- *Site: Found on endothelial cells, osteoclasts, and certain tumor cells.*
- *Diseases Associated: Implicated in pathological angiogenesis, osteoporosis, and metastatic cancer.*

α5β1 Integrin

- *Synthesis: Synthesized by various cell types, including fibroblasts, endothelial cells, and smooth muscle cells.*
- *Function: Mediates cell adhesion to fibronectin and fibrinogen, involved in cell migration, wound healing, and angiogenesis.*
- *Site: Found on fibroblasts, endothelial cells, and smooth muscle cells.*
- *Diseases Associated: Implicated in diseases involving abnormal wound healing, fibrosis, and tumor progression.*

α4β1 Integrin (also known as VLA-4):

- *Synthesis: Expressed on various immune cells, including lymphocytes and monocytes.*
- *Function: Mediates cell adhesion to vascular cell adhesion molecule-1 (VCAM-1) and fibronectin, involved in leukocyte trafficking, homing, and inflammation.*
- *Site: Found on leukocytes, including lymphocytes and monocytes.*
- *Diseases Associated: Implicated in autoimmune diseases, inflammatory disorders, and cancer metastasis.*

αIIbβ3 Integrin (also known as GPIIb/IIIa):

- *Synthesis: Expressed primarily on platelets.*
- *Function: Mediates platelet aggregation by binding to fibrinogen and von Willebrand factor, essential for hemostasis and thrombosis.*
- *Site: Found on platelets.*
- *Diseases Associated: Implicated in platelet function disorders, thrombotic disorders, and cardiovascular diseases.*
- *Examples: Dysregulated αIIbβ3 integrin is associated with conditions like Glanzmann thrombasthenia.*