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Q1: Different between hypertrophy and hyperhyplasia?

Hypertrophy:

Hypertrophy is an increase and growth of muscle cells. Hypertrophy refers to an increase in muscular size achieved through exercise. When you work out, if you want to tone or improve muscle definition, lifting weights is the most common way to increase hypertrophy.

Hyperplasia:

Hyperplasia (from ancient Greek ὑπέρ huper, "over" + πλάσις plasis, "formation"), or hypergenesis, is an increase in the amount of organic tissue that results from cell proliferation. It may lead to the gross enlargement of an organ, and the term is sometimes confused with benign neoplasia or benign tumor.

1. Definition of Hypertrophy and Hyperplasia

Hypertrophy: The hypertrophy is an increase of the volume of a given tissue or organ due only to the enlargement of the cells.

Hyperplasia: The hyperplasia is an increase in the amount of a tissue, resulting from cell proliferation.

2. Genesis of Hypertrophy and Hyperplasia

Hypertrophy: The hypertrophy is mainly provoked by increased demand.

Hyperplasia: The hyperplasia is mainly provoked by excessive cell stimulation.

3. Process of Hypertrophy and Hyperplasia

Hypertrophy: Hypertrophy is a result from cell enlargement.

Hyperplasia: Hyperplasia is a result from cell proliferation.

4. Mechanism of Hypertrophy and Hyperplasia

Hypertrophy: Hypertrophy is a result of increased protein production in the cells.

Hyperplasia: Hyperplasia is a result of proliferation of mature cells, driven by growth factors.

5. Affected cells of Hypertrophy and Hyperplasia

Hypertrophy: Hypertrophy occurs in permanent cells (non-dividing, such as skeletal muscle, cardiac muscle, etc.).

Hyperplasia: Hyperplasia occurs in labile or stable dividing cells.

Q2. What is the difference between coagulative and liquefactive necrosis?

Coagulative necrosis is a type of accidental cell death typically caused by ischemia or infarction. In coagulative necrosis the architectures of dead tissue is preserved for at least a couple of days.

Causes:

Coagulative necrosis is most commonly caused by conditions that do not involve severe trauma, toxins or an acute or chronic immune response. The lack of oxygen (hypoxia) causes cell death in a localised area which is perfused by blood vessels failing to deliver primarily oxygen, but also other important nutrients. It is important to note that while ischemia in most tissues of the

body will cause coagulative necrosis, in the central nervous system ischemia causes liquefactive necrosis, as there is very little structural framework in neural tissue.

Macroscopic

The macroscopic appearance of an area of coagulative necrosis is a pale segment of tissue contrasting against surrounding well vascularised tissue and is dry on cut surface. The tissue may later turn red due to inflammatory response. The surrounding surviving cells can aid in regeneration of the affected tissue unless they are stable or permanent.

Liquefactive necrosis

Liquefactive necrosis (or colliquative **necrosis**) is a type of **necrosis** which results in a transformation of the tissue into a liquid viscous mass. Often it is associated with focal bacterial or fungal infections, and can also manifest as one of the symptoms of an internal chemical burn.

Q3. Write a note on labile and stable cells.

Definition:

In cellular biology, labile cells are cells that multiply constantly throughout life.[1] The cells are alive for only a short period of time. Due to this, they can end up reproducing new stem cells and replace functional cells.

Explanation:

Especially if the cells become injured through a process called necrosis, or even if the cells go through apoptosis. The way these cells regenerate and replace themselves is quite unique. While going through cell division, one of the two daughter cells actually becomes a new stem cell. This occurs so then that daughter cell can end up restoring the population of the stem cells that were lost. The other daughter cell separates itself into a functional cell in order to replace the lost, or injured cells during this process.[2] Labile cells are one type of the cells that are involved in the division of cells. The other two types that are involved include stable cells and permanent cells.

Each of these type of cells respond to injuries of the cells they occupy differently. Hepatocytes of the liver are thought to be a form of a labile cell because they can regenerate after they become injured. An example of this kind of regeneration can consist of performing a pediatric liver transplant. In which it consists of taking a piece of an adult's liver to replace a child's whole liver. Then the adult liver that was transplanted for the child's, would regenerate very quickly to around a normal size liver.[4] Other cell types that are thought to be cells that are constantly dividing include skin cells, cells in the gastrointestinal tract, and blood cells in the bone marrow. Acting as stem cells for these cell types.

In labile cells, it is not a speed-up in the segments of the cell cycle (i.e. G1 phase, S phase, G2 phase and M phase), but rather a short or absent G0 phase that is responsible for the cells' constant division.

Hazards:

Constantly dividing cells have a higher risk of becoming malignant and develop cancer, dividing uncontrollably.[6] This is why muscle cancer is very rare, even though muscle tissue accounts for ~50% of total body weight, since muscle cells are not constantly dividing cells, and therefore not considered labile.

\In addition, cytotoxic drugs, such as alkylating antineoplastic agents, used in treatment of cancer, work by inhibiting the proliferation of dividing cells, with the malignant cells as the desired target. However, this has the adverse effect of also striking against the cells normally dividing in the body, and thus impairing normal body function of hair, skin, GI tract and bone marrow.

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stable cells:

In cellular biology, stable cells are cells that multiply only when needed. They spend most of the time in the quiescent G_0 phase of the cell cycle but can be stimulated to enter the cell cycle when needed. Examples include the liver, the proximal tubules of the kidney and endocrine glands

Stable cell lines are crucial laboratory tools that can be used to express large amounts of a protein of interest. This can be necessary for a variety of reasons; high protein production may be necessary for the screening of experimental drugs, studying of gene functions, or production of therapeutic proteins (including recombinant antibodies). The cell lines indefinitely reproduce and continue to express the transgene consistently during that time period.

Perhaps the most important characteristic of stable cell lines is that they are, as the name implies, genetically homogenous in culture. This means that even after new generations have been produced, the same genetic characteristics are present in all cells. This is particularly relevant to long term genetic studies and industrial production of highly specific proteins. In research settings, the availability of stable cell lines allows for the evaluation of drug treatments and monitoring of the evolution of cellular behavior. Cancer cell lines, for example, must be stable in order for drug testing to be indicative of drug potential.

Stable cell lines contrast strongly with transiently-transfected cells, which do not have stable genetic and protein expression characteristics. Transient transfection results in temporary changes to cell lines, which may aid in one-time production of proteins or short-term experiments. This makes transiently-transfected cell lines ideal for simple experiments, but problematic for studies that last for several days or more. As such, genetic modifications of cells used for longer studies must be permanent (i.e be present in the genome), and hence require advanced transfection techniques.

Q4. Differentiate between healing by primary intention and healing by secondary intention.

PRIMARY INTENTION HEALING

Also known as “first intention healing” or “primary wound closure,” this type of healing is generally employed when there has been very little tissue loss and new blood vessels and keratinocytes need to migrate only a small distance. These clean wounds or surgical incisions are almost immediately closed with stitches, staples, glue such as Derma Bond, or tapes such as Steri Strips.

In some instances, closure of the wound or incision may be delayed and require the skill of a plastic surgeon or orthopedic surgeon if the wound is jagged, asymmetrical, or could interfere with movement in a joint. There may also be a delay if the wound is significantly contaminated or too much time has passed (generally more than 4-8 hours). In these instances, surgeons may opt for second intention healing.

healing by secondary intention:

Although most wounds and incisions are mended through primary intention healing, sometimes second intention healing (also known as “secondary closure healing”) is the chosen method. This occurs when there is a large tissue defect (possibly with infection), there has been extensive loss of cells and tissue, and wound edges cannot be approximated requiring new tissue to be formed to bridge the gap between wound edges or to “fill the wound.”

As you may expect, second intention healing is a much slower process as it requires granulation tissue formation for the wound to contract, and can often result in a large, unpleasant scar. Surgical removal of dead tissue (debridement) can help.

Q5. Write briefly about the cellular response to adverse effects.

The adaptive immune system in vertebrates has evolved to provide host resistance to infectious microorganisms and malignant disease. Normal immune function and the induction of specific immune responses require the orchestrated interaction between cells and molecules both within and outside the lymphoid system. Immunotoxicology can be defined as the study of adverse health effects that may result from the interaction of xenobiotics with the immune system. In general terms such effects can take one of two forms. The first of these is immunotoxicity (or immunosuppression) where there is a perturbation of, or damage to, one or more components of the immune system resulting in impaired immune function and reduced host resistance. The design and interpretation of experimental immunotoxicity studies and the investigation of clinical immunosuppression require consideration of the relationship between changes in the structure and/or function of discrete components of the immune system and holistic changes in the susceptibility to infectious and malignant disease. The other main way in which chemicals may cause adverse health effects secondary to interaction with the immune system is through stimulation of specific immune responses that result in allergic disease. Allergy to chemicals and proteins can take many forms, including allergic contact dermatitis, allergic sensitization of the respiratory tract (associated with rhinitis and/or asthma), systemic allergic reactions (associated frequently with drug treatment), and gastrointestinal disease. Here there is a need to distinguish between immunogenic responses per se and those immune responses that are of sufficient vigor and of the quality necessary to provoke allergic sensitization. The purpose of this article is to explore the extent to which distinctions can be drawn between adverse and nonadverse effects in the context of immunotoxicity and allergy.