Commentary

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A Targeted Approach for Drug Development to Reduce Side Effects

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Description

Drug development is rarely a simple method. In fact, it's usually expensive, difficult, and time-consuming and it almost always ends in failure. Researchers often need years to identify the processes of disease before they can even consider developing therapeutic options and begin on the lengthy, laborious process, due to the enormous complexity of the cell and the remarkable diversity of diseases.

Safety testing is one of the most difficult challenges for drugs that make it to clinical trials. Before investigators can estimate pharmacological potency, potential toxicities and side effects must be assessed, and these critical characteristics must be deemed acceptable. Chemotherapy, for example, a powerful traditional strategy to killing fast-growing cancer cells, has the disadvantage of damaging good cells in the process, resulting in side effects such as pain, nausea, and hair loss.

Targeting interactions rather than individual proteins

Protein–protein interactions (PPIs) are highly specialised physical contacts formed between two or more protein molecules as a result of biochemical events controlled by electrostatic forces, hydrogen bonding, and the hydrophobic effect. Many are physical interactions between chains that take place in a cell or in a living organism in a specific biomolecular environment.

Proteins rarely act on their own since their functions are usually controlled. Many molecular activities in a cell are carried out by molecular machines made up of a variety of protein components that are structured by their PPIs. These physiological interactions make up the organism's interactomics, while abnormal PPIs are at the basis of aggregation-related disorders including Creutzfeldt–Jakob disease and Alzheimer's disease.

Improving the body's immune system

The complement system is a biochemical cascade that targets foreign cells' surfaces. It's made up of approximately 20 different proteins and gets its name from its capacity to "complement" antibody-mediated pathogen killing. The key humoral component of the innate immune response is complement. Many species, including non-mammals such as plants, fish, and some invertebrates, have complement systems. Complement binding to antibodies that have adhered to these microorganisms or the binding of complement proteins to carbohydrates on the surfaces of bacteria activates this response in humans. A quick lethal response is triggered by this identification signal. The speed of the response is due to signal amplification, which occurs after complement molecules, which are also proteases, are sequentially proteolytically activated. Complement proteins activate their protease activity after binding to the microbe, which in turn activates other complement proteases.

The adaptive immune system's cells are lymphocytes, which are a type of leukocyte. The two main types of lymphocytes are B cells and T cells, which are formed from hematopoietic stem cells in the bone marrow humoral and cellular immune response is mediated by B cells, whereas the cell-mediated immunological response is mediated by T cells. Helper T cells and regulatory T cells only recognise antigens that are attached to Class I MHC molecules, whereas killer T cells only recognise antigens that are coupled to Class II MHC molecules.

Role of DNA in cancer therapy

Several cancer chemotherapy medications act by causing excessive DNA damage, which results in cell death either directly or indirectly as a result of DNA replication. A number of DNA repair processes boost survival by repairing these damages.

Conclusion

Because tumour cells can survive DNA damage caused by chemotherapy treatments due to DNA repair mechanisms, inhibitors of certain DNA repair pathways may be effective when used in tandem with DNA-damaging chemotherapeutic agents. Furthermore, throughout tumour formation, changes in DNA repair pathways can make some cancer cells reliant on a smaller number of DNA repair pathways for survival. There is evidence that medications that block one of these pathways in malignant tumours could be useful as single-agent therapy, with the additional benefit of being selective for tumour cells and having fewer adverse effects.