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Editorial

Biological Target and Its Mechanism

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EDITORIAL NOTE

Biological target is whatsoever within an alive organism to which some other entity (like an endogenous drug or ligand) is absorbed and binds, subsequent in a modification in its function or behaviour. For examples of mutual biological targets are nucleic acids and proteins. Definition is context dependent, and can refer to the biological target of a pharmacologically active drug multiple, the receptor target of a hormone like insulin, or some other target of an external stimulus. Biological targets are most commonly proteins such as ion channels, enzymes, and receptors. The term "biological target" is regularly used in research of pharmaceutical to describe the nastive protein in body whose activity is altered by a drug subsequent in a specific effect, which may a desirable therapeutic effect or an unwanted adverse effect. In this setting, the biological target is referred to as a drug target.

Mechanism: The external stimulus like the ligand or drug physically binds to the biological target. The interaction between the substance and the target may be:

Non-covalent: A relatively weak interaction between the target and the stimulus where any chemical bond is formed between the two interacting partners and hence the interaction is totally reversible.

Reversible covalent: A chemical reaction occurs between the target and the stimulus where the stimulus chemically binds to target, but also the reverse reaction readily occurs in which the bond could be broken.

Irreversible covalent: The stimulus is enduringly bound to the target through irreversible chemical bond formation.

Depending on the stimulus nature, the following can occur:

1. There is no direct change in the biological target, but the binding of the substance stops other endogenous substances (such as triggering hormones) from binding to the target. Contingent on the nature of the target, this effect is mentioned as receptor antagonism, ion channel blockade or enzyme inhibition.

2. A conformational change in the target is induced by the stimulus which results in a change in target function. This change in function can mimic the effect of the endogenous substance in which case the effect is referred to as receptor agonism or be the opposite of the endogenous substance which in the case of receptors is referred to as inverse agonism.

Conservation Ecology: These biological targets are conserved across species, making pharmaceutical pollution of the environment a danger to species that possess the same targets. For example, the synthetic estrogen in 17-R-ethinylestradiol, human contraceptives, has been shown to increase the feminization of fish downstream from sewage treatment plants, thereby unbalancing reproduction and creating an additional selective pressure on fish survival. Pharmaceuticals are usually found at ng/L to low-µg/L concentrations in the aquatic environment. Adverse effects may occur in non-target species as a consequence of exact drug target interactions. Therefore, evolutionarily well-conserved drug targets are probable to be related with an amplified risk for non-targeted pharmacological effects.

Metabolism: Living organisms are single in that they can extract energy from their environments and use it to carry out doings such as growth, movement, development, and reproduction. Hundreds of multistep reactions, coordinated, fueled by energy obtained from nutrients and solar energy, ultimately convert readily available materials into the molecules required for growth and maintenance.

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