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Toxicodynamics - an overview and its importance

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Toxicodynamics, termed pharmacodynamics in pharmacology, describes the dynamic interactions of a toxicant with a biological target and its biological effects. A biological target, also referred to as the location of action, are often binding proteins, ion channels, DNA, or a spread of other receptors. When a toxicant enters an organism, it can interact with these receptors and produce structural or functional alterations. The mechanism of action of the toxicant, as determined by a toxicant's chemical properties, will determine what receptors are targeted and therefore the overall toxic effect at the cellular level and organismal level.

Toxicants are grouped together consistent with their chemical properties by way of quantitative structure-activity relationships (QSARs), which allows prediction of toxic action supported these properties. endocrine disrupting chemicals (EDCs) and carcinogens are samples of classes of toxicants which will act as QSARs. EDCs mimic or block transcriptional activation normally caused by natural steroid hormones. These sorts of chemicals can act on androgen receptors, estrogen receptors and hormone receptors. This mechanism can include such toxicants as dichlorodiphenyltrichloroethane (DDE) and polychlorinated biphenyls (PCBs). Another class of chemicals, carcinogens, are substances that cause cancer and may be classified as genotoxic or nongenotoxic carcinogens. These categories include toxicants like polycyclic hydrocarbon (PAHs) and carbon tet (CCl_4).

The processes of toxicodynamics are often useful for application in environmental risk assessment by implementing toxicokinetic-toxicodynamic (TKTD) models. TKTD models include phenomenas like time-varying exposure, carry-over toxicity, organism recovery time, effects of mixtures, and extrapolation to untested chemicals and species. thanks to their advantages, these sorts of models could also be more applicable for risk assessment than traditional modeling approaches.

While toxicokinetics describes the changes within the concentrations of a toxicant over time thanks to the uptake, biotransformation, distribution and elimination of toxicants, toxicodynamics involves the interactions of a toxicant with a biological target and therefore the functional or structural alterations during a cell which will eventually cause a toxic effect. counting on the toxicant's chemical reactivity and vicinity, the toxicant could also be ready to interact with the biological target. Interactions between a toxicant and therefore the biological target can also be more specific, where high-affinity binding sites increase the selectivity of interactions. If the biological target is critical and therefore the damage is severe enough, irreversible injury can occur first at the molecular level, which can translate into effects at higher levels of organization

Endocrine disruptors

EDCs are generally considered to be toxicants that either mimic or block the transcriptional activation normally caused by natural steroid hormones.[2] These chemicals include those working on androgen receptors, estrogen receptors and hormone receptors.

Effects of endocrine disruptors

Endocrine disrupting chemicals can interfere with the system during a number of the way including hormone synthesis, storage/release, transport and clearance, receptor recognition and binding, and postreceptor activation.

In wildlife, exposure to EDCs may result in altered fertility, reduced viability of offspring, impaired hormone secretion or activity and modified reproductive anatomy. The reproductive anatomy of offspring can particularly be affected if maternal exposure occurs. In females, this includes mammary glands, fallopian tubes, uterus, cervix, and vagina. In males, this includes the prostate, seminal vesicles, epididymitis and testes. Exposure of fish to EDCs has also been related to abnormal thyroid function, decreased fertility, decreased hatching success, de-feminization and masculinization of female fish and alteration of immune function.

Androgen-receptor mediated

Certain toxicants act as endocrine disruptors by interacting with the androgen receptor. DDE is one example of a chemical that acts via this mechanism. DDE may be a metabolite of DDT that's widespread within the environment.[1] Although production of DDT has been banned within the Western world, this chemical is extremely persistent and remains commonly found within the environment along side its metabolite DDE.[1] DDE is an antiandrogen, which suggests it alters the expression of specific androgen-regulated genes, and is an androgen receptor (AR)-mediated mechanism.[1] DDE may be a lipophilic compound which diffuses into the cell and binds to the AR.[1] Through binding, the receptor is inactivated and can't bind to the androgen response element on DNA.[1] This inhibits the transcription of androgen-responsive genes[1] which may have serious consequences for exposed wildlife. In 1980, there was a spill in Lake Apopka, Florida which released the pesticide dicofol and DDT along side its metabolites.[4] The neonatal and juvenile alligators present during this lake are extensively studied and observed to possess altered plasma hormone concentrations, decreased clutch viability, increased juvenile mortality, and morphological abnormalities within the testis and ovary