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Studies of Toxic Effects on the Hereditary Genotoxicity

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DESCRIPTION

Genotoxicity is the property of chemicals that damage the genetic information in cells and cause mutations that lead to cancer. Genotoxicity is often confused with mutagenicity, although all mutagens are genotoxic, some genotoxic substances are not. This change can have direct or indirect effects on DNA. Improperly timed mutagenesis Direct DNA damage leads to activating events and mutations. Permanent, genetic changes can affect either the somatic cells of an organism or the germ cells intended to be passed on to future generations. Cells prevent the expression of genotoxic mutations by either DNA repair or apoptosis. However, the damage is not always repaired and mutagenesis occurs. To test genotoxic molecules, researchers test for DNA damage in cells exposed to toxic substrates. This DNA damage can take the form of single and double-strand breaks, loss of excision repair, cross-links, alkaline labile sites, point mutations, and structural and numerical chromosomal abnormalities. Cancer is known to occur when the integrity of genetic material is compromised. As a result, many sophisticated techniques, such as the Ames assay, *in vitro* and *in vivo* toxicology tests, and the comet assay, have been developed to assess the potential of chemicals to cause DNA damage that can lead to cancer. Genotoxins are chemicals or substances that can cause DNA or chromosomal damage. Such damage to germ cells can lead to inherited altered traits (germ cell mutations). DNA damage in somatic cells can lead to somatic mutations that can lead to malignant transformation (cancer). Numerous *in vitro* and *in vivo* assays for genotoxicity addressing various endpoints, DNA damage, or its biological effects in prokaryotic cells (such as bacteria) or eukaryotic cells (such as mammalian, avian, or yeast) are being developed. These assays are used to assess the safety of environmental chemicals and consumer products and to study the mechanism of action of known or suspected carcinogens. Many chemical carcinogens/mutagens undergo metabolic activation to reactive species that covalently bind to DNA, and the DNA adducts so formed can be detected in cells and human tissues by a variety of sensitive techniques. Detection and characterization of DNA adducts in human tissues provide clues to the pathogenesis of human cancers. Characterization of genetic mutations in human tumors, together with the known mutagenicity profile of genotoxins in experimental systems, may provide further insight into the role of environmental mutagens in human cancer. This is a very rapid test for mutagens. It uses bacteria of the Salmonella typhimurium species that have mutations in the histidine operon and are unable to synthesize histidine. We test compounds to see if they can induce the reversal of this mutation. This test is very easily performed by plating His (negative) mutants on agar plates containing only trace amounts of histidine. Place the crystals or filter disc containing the solution of the compound to be tested on the surface of the agar. Bacteria only grow for a short time before their histidine is used up. After this point, the only bacteria that can continue to grow colonies are those that undergo reversion and synthesize their own histidine, histidine prototroph (His+). If the test compound is not mutagenic, several revertant colonies will be randomly scattered across the agar plate. If mutagenic, the number of colonies will cluster around the point where the compound was added to the plate. Note that a more quantitative method can be constructed to obtain a dose-response curve for each compound tested.

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CONFLICT OF INTEREST

Author declares that there is no conflict of interest.

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