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Brief note on Immunotoxicity and Integrated Function of Natural Killer (NK)

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Editorial Note

Exposure to two types of immunotoxic agents results from their intentional use. The first of these are chemotherapeutic agents, which are widely used in cancer treatment. The second type of pesticide is one that is actively used to control unwanted plant and microbial growth (herbicides and fungicides), as well as vermin (insecticides, acaricides, and rodenticides). In both cases, these agents serve primarily to benefit humanity, but they also cause unintended consequences, either as a side effect or as a result of environmental contamination. Hundreds of chemotherapeutic agents are used to extend the lives of cancer patients. This chapter only looks at a few of the most common agents, which were chosen using the method described in Section 5·2, 5·3. Similarly, the number of pesticides is enormous; however, these agents can be classified based on their basic structure and then further classified based on the chemistry of the compound.

The main immunotoxic effects of nickel exposure are allergic reactions. Allergic skin reactions are the most common in nickel-sensitive humans. There have been few in-depth studies of nickel's immunotoxic effects. Despite the fact that sensitivities are the most well-known toxicological result of ingesting or being presented to unusually high groupings of nickel chloride and the subsequent divalent nickel, nickel openness can likewise result in other toxicological outcomes cause a variety of other immunotoxic effects. In one study, for example, nickel chloride caused an increase in superoxide anion generation and phagocytosis activity in crab hemolymph. In humans, inhalation is a common route of exposure in the workplace; as a result, skin allergies, lung fibrosis, and respiratory tract cancer have been observed. It should be noted, however, that these examples necessitated nickel-polluted environments.

A solitary or different intramuscular infusions of nickel chloride brought about a huge decrease in an assortment of safe cells and capacities in mice. In the spleens of nickel chloride–injected mice, lymph proliferative responses to T-cell mitogens, phytohemagglutinin and concanavalin A, were suppressed, and the number of theta-positive T-lymphocytes was reduced. In vitro and in vivo assays revealed suppression of natural killer cell activity, but no significant reduction in spleen cellularity or suppressor cell production was observed.

They are advantageous in that they investigate the entire context of NK function, and the effects of toxin exposure can be evaluated using endpoints relevant to human experience - infection and organism/tumor burden. Nonetheless, these assays have significant drawbacks. For starters, they are expensive in terms of time, money, and animals. Because the challenge organism affects the constitutional, histologic, and cytometric values that serve as the foundation for other screening assays, host-resistance assays necessitate a separate cohort of animals. Furthermore, because these studies frequently use mortality as an endpoint, a large number of animals may be required to achieve reasonable statistical analyses at toxin doses with mediocre immunologic effects. While increasing the challenge dose range may improve sensitivity, this necessitates more animal testing. Surrogates such as pathogen titer or tumour load may be used to avoid the problems associated with mortality endpoints. This has the disadvantage of being more labor-intensive.

Second, in lieu of in-depth investigation, their holistic nature renders host-resistance assays nonspecific for NK cells in general. The mouse CMV model, by far the best understood NK-related infection, exemplifies this. NK cells are the only terminal effectors required to protect mice from early death in mice with the Ly49H-activating receptor variant. However, as previously stated, the NK response is entirely dependent on functional TLR-dependent DC responses. As a result, immunotoxicity affecting any part of the DC component would have an effect on the outcome. A potential counter-argument to this argument is that host-resistance assays are used as 'tier II' studies after in vitro evidence of NK dysfunction has been obtained; thus, any reduction in host resistance must be due to the NK effect.

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