et al. (11), tumors grow in an immunoprivileged site, that is, they reside in an environment that cannot be scrutinized by the immune system. However, anticancer therapy can disrupt this immunoprivileged condition.

a. Mechanisms of Immune Response Against Tumors

Chemotherapy or radiation can kill cancer cells, where the mechanism of cell death is apoptosis or necrosis of the cancer cells, and where cell death results in the release of tumor antigens into interstitial fluids. Fragments of killed tumor cells, and released tumor antigens, can be taken up by dendritic cells (DCs) (12).

Although tumor cells and tumor antigens may be taken up by DCs, this does not necessarily mean that the DCs will subsequently present tumor antigens to T cells in an effective manner, that is, in a manner that will stimulate the T cells to kill tumor cells.

In the absence of an activating environment, the DC that takes up tumor antigens may cause tolerance to the tumor. In studies of tumors in animals, den Brok et al. (13) and Van Oosten and Griffith (14) inflicted physical damage on tumors. The damage was inflicted in the presence and absence of a TLR-agonist. The TLR-agonist was CpG-oligonucleotide. While tumor debris in the animal was an effective antigen depot for DCs, an effective immune response requires an immune adjuvant, such as a TLR-agonist. According to Cuenca et al. (15), where DCs capture tumor antigens but without stimulation, the DCs will travel to the lymph nodes and communicate with T cells, but instead of causing the T cells to be active against tumors, the T cells will be caused to be tolerogenic T cells. Human cancers often contain $CD8^+$ T cells, that is, tumor-infiltrating $CD8^+$ T cells, that are antigen-specific but do not lyse tumor cells (16).

Immune adjuvants useful for treating cancer can take the form of a TLR-agonist such as CpG-oligonucleotide, polyinosinic:polycytidylic acid, an adjuvant that binds to TLR3 (17,18), imiquimod, bacillus Calmette-Guerin

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