B-Lymphocytes in Carcinogenesis

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I. Introduction

Cancer is not a single disease entity; the term is applied to a large number of diseases in which the abnormal and continued growth of cells of a given tissue is expressed. The immunologic mechanism responsible for the rejection of solid tumors in experimental animals is generally thought to be the same as that responsible for homograft rejection, i.e., delayed hypersensitivity. It has been demonstrated with the use of diffusion chambers that antibody and complement may kill some kinds of tumor cells. The cytotoxic reaction may be responsible for the destruction of tumor cells growing primarily in suspension and the delayed-type reaction may be responsible for the rejection of solid tumors. The mechanism of tumor cell destruction by specifically sensitized cells has
been studied in vitro. The major mechanism of destruction is the interaction of sensitized lymphocytes with tumor cell-surface antigens. Thereby, the sensitized cell may be either activated to become a killer cell or it may synthesize and/or release mediators (lymphokines) which activate other cells, such as macrophages, to participate in target cell killing (Sell, 1975).

Cell-mediated immunity can be demonstrated under three general aspects:

1. Destruction of target cells by lymphocytes specifically sensitized to the target cells.

2. Production of killer cells or appropriate cell products by normal unsensitized lymphocyte populations following treatment with nonspecific lymphocyte stimulants.

3. Target cell killing by normal unsensitized lymphocytes in the presence of humoral antibodies directed against the target cell (Sell, 1975).

The possible situations are adumbrated in Figure 1. Direct target cell killing by specifically sensitized killer cells and mitogen-stimulated killer cells or cell products...