Editorial

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The Role of the Immune System in Destroying or Managing Cancerous Cells

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Editorial Note

For a short review, the immune system is traditionally thought to be made up of two arms: innate and adaptive. However, this is a simplification because these arms have overlapping functions and are tightly related. Dendritic cells, Natural Killer cells (NK), macrophages, neutrophils, eosinophils, basophils, and mast cells are all part of the innate immune system. Innate immune cells are the initial line of defence against foreign antigens and do not require antigen stimulation. B lymphocytes, CD4+ helper T lymphocytes, and CD8+ Cytotoxic T Lymphocytes (CTLs) make up the adaptive immune system, which is activated by Antigen-Presenting Cells (APCs). Antigen-specific T- and B-cell lymphocytes are produced by the adaptive immune system. Within a given person, the immune system is extremely varied amongst individuals yet generally constant over time. Every day, about 20,000 DNA damaging events are predicted to occur in each cell, which are routinely repaired by particular DNA repair mechanisms with no long-term consequences. The tumour immune-surveillance system normally recognises and kills cells that are not fixed and develop malignant or possibly malignant alterations. This mostly involves cell-mediated systems that distinguish between self and non-self-antigens. Many novel Tumour-Associated Antigens (TAAs) may be expressed because a malignant cell might have over 11,000 genetic alterations. TAAs include mutant proto-oncogenes, tumour suppressor genes, overexpressed or aberrantly expressed proteins, onco-fetal antigens, altered glycolipids and glycoproteins, and cell type-specific differentiation antigens. The Major Histocompatibility Complex (MHC) molecules exhibit these novel TAAs, or parts of them, on cell surfaces. However, recognition of an antigen-MHC complex by a T-cell antigen receptor is insufficient for naive T-cell activation, necessitating additional costimulatory signals provided by the engagement of the CD28 receptor on the T-cell surface with B7 ligand molecules (two of which are CD80 and CD86) on APCs. This "immunological synapse," or CD28 receptor/B7 ligand pair, increases T-cell proliferation and activity. Many other receptor/ligand combinations exist between activated T-cells and other cells, including tumour cells, and some of these interactions, such as PD-1/PD-L1 and CTLA-4/B7 are inhibitory. Some malignant cells can manipulate their own properties as well as the cells in their surroundings to become "successful" tumours, evading the tumour immune surveillance system; these evasive processes are a key focus of contemporary Clinical research. The hypothesis was initially dismissed, but it is now recognised as a component of cancer immune editing, in which the monitoring system can determine or "shape" the immunogenicity of tumour cells that have not been eliminated. Cancer cells that are not destroyed and survive may do so by expressing fewer antigens on their surfaces or by reducing MHC class I expression. They may also demonstrate the ability to defend themselves against T-cell attack by expressing Immune Checkpoint (IC) molecules on their surfaces in the same way that normal cells do; these IC molecules are upregulated by cytokines produced by activated T-cells and are part of a normal negative feedback loop that controls excessive tissue damage from inflammation by downregulating or suppressing T-cells. The "cancer-immune set point" refers to an individual's inherent immunological ability to fight cancer, which is modified by a complicated combination of elements involving the tumour, the host, and environmental factors. Clinical studies are attempting to better describe these characteristics in order to predict a person's immunotherapy response.