International Scholars Journals

African Journal of Immunology Research ISSN 2756-3375 Vol. 8 (2), pp. 001-002, September, 2021.
Available online at www.internationalscholarsjournals.com © International Scholars Journals

Perspective

Cytotoxic effector cell mechanisms

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Accepted 15 September , 2021

INTRODUCTION

Different host mechanisms for destroying target cells have evolved, resulting in a diverse collection of cytotoxic cells capable of responding against a wide variety of foreign cells and organisms. A common feature among them, however, is that cytotoxicity may be a regulated process. They are generally activated as results of cell surface ligands on the target cells binding and triggering selected cell surface receptor molecules on the effector cells.

Significant sources of cytotoxic effector cells that have been concentrated widely are CTLs, NK lymphocytes, and activated macrophages.

CYTOTOXIC T LYMPHOCYTES

CTLs result from classic T-cell immune responses: 1) The CTL response is started by exposure to antigen, 2) generation and regulation of the response relies upon a complex MHC-dependent interaction of antigen-processing cells and T cells, 3) the T-cell Antigen Receptor (TCR) provides MHC-dependent antigen specificity for the interaction between CTLs and target cells, and 4) memory responses commonly follow reexposure to the antigen, resulting in more rapid, longer, and higher-level responses. CTLs do not have a characteristic morphology. They might show up as small, resting, a granular lymphocytes or as large, blastic, granular cells or as gradations in between.

NATURAL KILLER CELLS

NK cells are a relatively small population of lymphocytes distinct from T and B lymphocytes. They generally are large, granular lymphocytes that originate in the bone marrow. NK cells share a common progenitor with T cells, and NK cell precursors have been recognized in the thymus; however, they do not need the thymus for maturation, and they probably diverge from the T-cell lineage at a beginning stage of differentiation. Several markers are particularly helpful in distinguishing between NK cells and CTLs. Human NK cells are primarily TCR/CD3–, CD5–, CD56+, FcR+. These cell surface features are used both for physically separating the cells and for functionally distinguishing between NK cell and CTL activity.

LYMPHOKINE-ACTIVATED KILLER CELLS

Both CTLs and NK cells cultured with relatively high doses of IL-2 show enhanced nonspecific cytotoxic activity, as revealed by their ability to lyse selectively a broad spectrum of fresh autologous, syngeneic, or allogeneic tumor cells that are relatively insensitive to normal NK-mediated cytotoxicity. They are referred to as lymphokine-activated killer cells. The more cytotoxic activity of these cells appears to result, in part, from their activation-enhanced expression of surface molecules that contribute to target cell binding and to triggering cytotoxic activity.

MACROPHAGES

Activated macrophages, as with NK cells and CTL cultured with relatively high doses of IL-2, can be nonspecifically cytotoxic for cancer cells *in vitro*. Macrophages can be activated by numerous extra factors, including cancer cell contact. As with NK cells, the basis for triggering cytotoxicity after contact between activated macrophages and cancer cells has not been established. Macrophages and monocytes are clearly distinct from CTL and NK cells. In contrast to CTL or NK cells, they may be phagocytic; express CD14, CD36, or CD68; have a comparatively abundant cytoplasm lacking enormous azurophilic granules; and are generally adherent to culture vessel surfaces *in vitro*. Although they may express CD4, they do not express the TCR, CD3, or CD8. Mediators of cytotoxicity, triggered by the binding of activated macrophages to cancer cells, include the following products: cytolytic proteases; TNF-a and related factors; IFN-a,g; IL-1;

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reactive oxygen intermediates, such as H_2O_2; reactive nitrogen intermediates, like nitric oxide; arginase; thymidine; and lysosomal enzymes. A serine protease has also been implicated. Many of the cytotoxic mediators may also act on cancer cells that are not in direct contact with macrophages, but most of the cytotoxic effects on bystander cells can be against both normal and tumor cells. Several mechanisms limit bystander effects, including short half-lives of the cytotoxic factors.