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Autocoids as Regulators of Inflammation and Immune Responses

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DESCRIPTION

Autocoids (histamines, prostaglandins, and beta-mimetic catecholamines), once considered only inflammatory mediators, are formed during specific early and late stages of immunity. They require sufficient concentrations to affect immune cells and can modulate immunity; usually by inhibition. Autocoid receptors on the immune cells are not randomly distributed. Instead, as these cells mature, they develop autocoid receptors. With one exception, immune cell function is inhibited by the autacoid action. Again, in all but one case, this inhibitory regulatory effect is mediated and directly proportional to intracellular levels of cyclic adenosine monophosphate produced by autacoids. The clinical significance of these observations is slowly being evaluated. One is that pharmacological antagonists of autacoids may have predictable but hitherto unexpected effects on immune function. It is the inconceivable that these effects are without clinical value.

Autocoids (primarily histamine, β-adrenergic catecholamines, prostaglandins E and A) have only recently been recognized as the key regulators of many immune functions. If autacoids are to be considered as potential therapeutic immunomodulatory agents, their effects on T cell subsets in the contact and in non-contact should be understood. This report shows that the autacoid receptor is not randomly distributed among the phenotypically and functionally distinct subsets of human T cells. Each human T-cell subset responded to both histamine and isoproterenol, although dose-response curves and peak effects differed significantly between subsets. After mitogen stimulation, responses to histamine, but not to isoproterenol, are significantly increased only in a subset of the helper/inducer and cytotoxic T cells, and this effect may be regulated by the suppressor T cells I understand. Accumulation in response to histamine in mitogen-treated helper/inducer and cytotoxic T cells was completely blocked by the H2 antagonists, but not by H1 antagonists. These findings demonstrate an immunologically unaffected subgroup's response to selected drugs and control of basal and autacoid-induced production, as well as increased qualitative and quantitative selectivity induced by mitogens. If these experiments were performed only on undissociated cells, we would not have observed the significant selectivity of the autacoid effect for subsets of the T cells.

When these cells are exposed to the above agents, intracellular cyclic concentrations increase. The biological implications of such increases were initially difficult to the grasp. We now know that it can modulate the immunocompetence of some mouse spleen cells and inhibit the release of lysosomal enzymes and histamine from human leukocytes. Stimulation by the E series and these agents reverses suppressor. Pre-treatment of cells with the histamine increased IL 2 secretion. Reversal appears to be quantitatively dependent on cyclic accumulation. Amine and prostaglandin receptors are found on T cell precursors of cell-mediated immunity. The effect of each autacoid was additive on the cytolytic T cells alone. The isoproterenol and histamine-stimulated adenylate cyclase pools of cytolytic T cells may be the independent of each other, but further studies are needed to prove this point. They occur in some T effector cells in the selected models of allogeneic target cell lysis. Receptors also appear to arise on selected B cells once these cells begin to produce antibodies.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

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