AN OVER VIEW ON
PULMONARY ALVEOLAR MACROPHAGE

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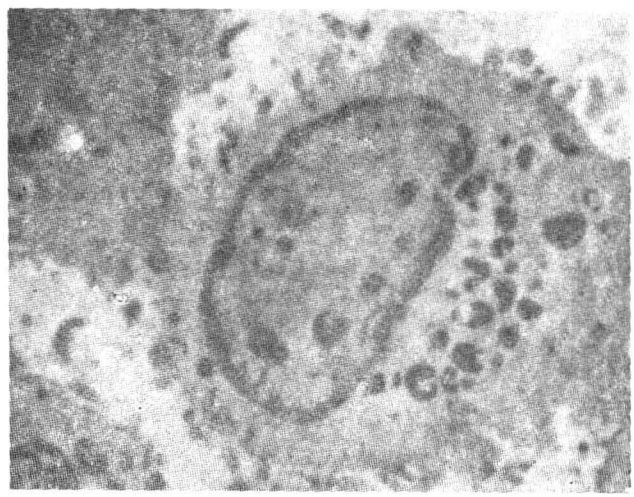

The pulmonary alveolar macrophage is the resident mononuclear phagocyte in the lung. It is a versatile cell. It has been recognised as a scavenger cell in the removal of non-cellular material in the alveoli. Its role in the destruction of microorganisms has been well established. More recently the participation of the alveolar macrophage in inflammatory and immune mechanisms have been unravelled. The pulmonary alveolar macrophage plays an important part in the afferent limb of the immune response being responsible for the presentation of antigen to the immunocompetent cells.

The pulmonary alveolar macrophage is the tissue equivalent of the circulating monocyte and like the latter is derived from the bone marrow. There are several differences between the pulmonary alveolar macrophages and macrophages derived from other sites such as the peritoneum. The distinctive biochemical features of the pulmonary alveolar macrophage are the result of adaptation to the unique environment in which the cell functions. Like most mononuclear phagocytes the pulmonary alveolar macrophage is a large cell (15-50 μm) even in the resting stage. The internal structure of the cell shows a large number of organcells and inclusions which contain a vast repertoire of enzymes which can be used to destroy organisms intracellularly.

The pulmonary alveolar macrophage possesses receptors for the FC portion of IgG and C_3 component of complement. These receptors aid the macrophage in the destruction of targets coated by immunoglobulin and complement. In addition pulmonary alveolar macrophages can respond to chemotactic factors elaborated by lymphocytes, bacteria or products of complement activation.

The anti-bacterial mechanisms of the pulmonary alveolar macrophage are triggered after the sequential occurrence chemotaxis and phagocytosis. Most of the antibacterial activity is achieved by the generation of powerful cidal agents like hydrogen peroxide. Other powerful bactericidal agents identified within the macrophage include catalases and superoxide ions. For effective destruction of microoganisms the macrophage also requires the presence of halide ions and various peroxidases. All these enzymes are released intracellularly to cause selective destruction of the bacteria. Recently surfactant related lipids have been shown to be necessary for effective microbicidal activity.

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The pulmonary alveolar macrophage like other macrophages can be converted into an energetic cell or angry cell when it is "activated". This can be achieved by antigenic stimulations, through the lymphokines sush as migration inhibition factor (MIF) and macrophage activating factor (MAF) which are elaborated by T lymphocytes. The activated macrophage has a vastly increased surface area and shows enhanced phagocytic properties. There is also striking increase in the hydrolytic enzyme content and metabolic rate of the macrophage. All these mechanisms result in acceleration of destruction of microorganisms.

The pulmonary alveolar macrophage elaborates a number of soluble products which can modulate inflammatory and immune mechanisms. The most significant of these are the factors chemotactic to the neutrophils. The cell is also known to elaborate certain growth promoting factors. The pulmonary alveolar macrophages also store a large quantity of elastases and antiproteolytic enzymes such as α_1-antitrypsin.

Pulmonary alveolar macrophages play an important role in defense mechanisms of the lung. Its part in the afferent limb of the immune system where it processes and presents the antigen to the lymphocytes has already been pointed out. This capacity of the pulmonary alveolar macrophages is crucial when antigens enter via the respiratory tract. It has been shown to elaborate lymphocyte activating factors (interleukins) which can stimulate the lymphocytes. On the efferent arc of the immune response the cell can destroy bacteria by using its microbicidal apparatus with the aid of immunoglobulins and complement. It also participates in cell mediated immune mechanisms by destroying virus infected cells by cytotoxic mechanisms. Its role in tumor immunity is not yet well established.

Defective alveolar macrophage functioning may underlie pathogenetic mechanisms in certain disease states. Smokers for example have abnormal pulmonary alveolar macrophages. Post viral bacterial infections of the lung have now been traced to defective macrophage functioning. In emphysema an inbalance of the enzyme systems of the macrophage has been cited as a possible pathogenetic mechanism.

The pulmonary alveolar macrophage enjoys the distinction of being the only accessible tissue macrophage in man and our knowledge of macrophages is to a large extent built upon studies on this cell. It is obviously the cell to watch in future years and a better understanding of its function may hold the key to the secrets of the defense mechanisms of the lung.