Color Atlas of Allergic Diseases

Translation by Walter Burgdorf

Bearbeitet von
Jürgen Behr, Martin Rocken, Gerhard Grevers, Walter Burgdorf, Martin Röcken

1. Auflage 2003. Buch. 256 S.
ISBN 978 3 13 129191 2
Format (B x L): 12,5 x 19 cm

Weitere Fachgebiete > Medizin > Sonstige Medizinische Fachgebiete > Psychosomatische Medizin, Schlafmedizin

Zu Inhaltsverzeichnis

schnell und portofrei erhältlich bei

beck-shop.de
DIE FACHBUCHHANDLUNG

Die Online-Fachbuchhandlung beck-shop.de ist spezialisiert auf Fachbücher, insbesondere Recht, Steuern und Wirtschaft. Im Sortiment finden Sie alle Medien (Bücher, Zeitschriften, CDs, eBooks, etc.) aller Verlage. Ergänzt wird das Programm durch Services wie Neuerscheinungsdienst oder Zusammenstellungen von Büchern zu Sonderpreisen. Der Shop führt mehr als 8 Millionen Produkte.

Roecken, Grevers, Burgdorf. Color Atlas of Allergic Diseases (ISBN 3131291915) © 2003 Georg Thieme Verlag

Basic Immune Functions

The immune system is designed to defend the body in a safe and efficient way against a variety of potentially dangerous materials including toxins and infectious organisms. There are many components to this defense system. Mechanical and biological barriers prevent the penetration of exogenous materials into the body (p. 1). Only after these barriers have been breached and cells have been directly attacked does the immune system come into play.

Certain immune cells can directly phagocytose and destroy many pathogens by a variety of mechanisms. To accomplish this, they require the close cooperation of the somatic cells, which both alert the immune system through alarm signals and then later participate in the effector phase. These signals can be grouped together as "stress signals." This initial, nonspecific immune response is known as the innate immune response. Apparently, all cells can participate to an extent in this process. The major players are those cells that are first or most often attacked. Thus, in the skin it is the keratinocyte; in the mucosa it is the epithelial cell; in the liver it is the hepatocyte. Other cells, generally thought of as structural and not active, such as glial cells and fibroblasts, are also involved in innate immunity.

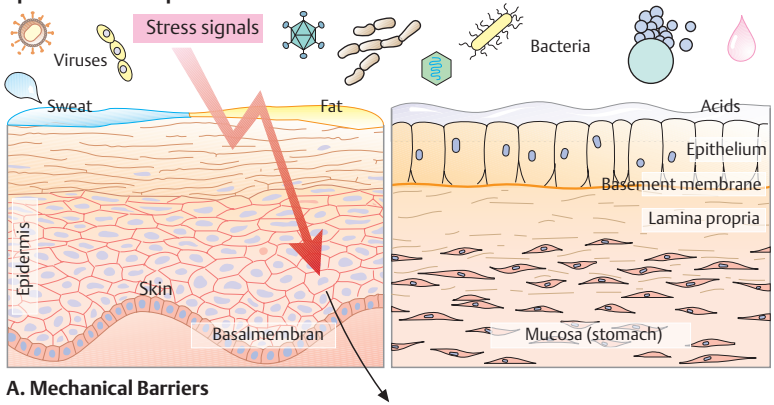
In response to stress signals, these cells respond with the release of a variety of messengers (p. 18f) that attract and activate phagocytic antigen-presenting cells (APC) (p. 12f) and thus initiate the specific immune response. Once activated, APC carry processed foreign antigens from the site of injury and migrate via the lymphatics to the regional lymph nodes, where they secrete chemokines to attract naive CD4 and CD8 T cells (p. 14f). The APC present the foreign antigen peptide to these T cells. Those naive cells that recognize the foreign peptide on the basis of their receptor structure are in turn stimulated and differentiate into blast cells. These activated T cells now express different surface antigens that make them capable of responding to growth factors, leaving the lymph nodes and themselves secreting cytokines. Exactly which mediators are involved and how the T cells in the end interact with their target cells and antigens is quite dependent on how they are activated (p. 14f).

The first important task for the activated T cells is clonal proliferation to ensure that enough cells are available to react in a specific fashion against the alarm-triggering noxious or infectious agent. Such specialized T cells can increase their number by a factor of 10,000 over a period of a few days under the influence of the cytokine interleukin 2 (IL-2) (p. 14f). Activated T cells have many other tasks. They interact closely in the lymph nodes with B cells, giving them signals for immunoglobulin production (p. 16f). Finally, they leave the lymph nodes and return to the initial site of injury. This return is made possible because the innate immune response has already started an inflammatory process at this site, leading to the production and expression of a variety of adhesion molecules. Certain vascular adhesion molecules specifically allow the activated T cells to attach to the vessel wall, migrate through the wall, and then reach the inflamed tissue. There the activated T cells are stimulated again—most likely by antigen-laden monocytes and macrophages—to produce a wide variety of pro-inflammatory mediators.

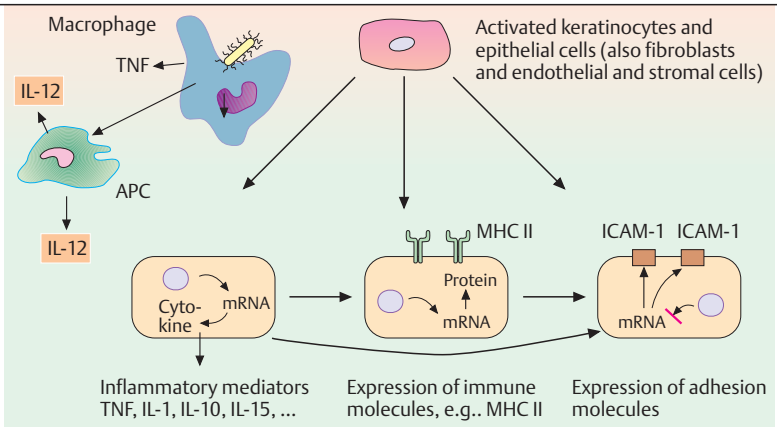
Interferon γ (IFN-γ) plays a major role at this stage. It triggers the macrophages to produce inflammatory mediators, which in turn further stimulate the neighboring cells to produce free radicals, tumor necrosis factor (TNF), and other factors with which they had initially triggered the immune response. Compared to the initiation phase, at this stage both the magnitude of the response and the spectrum of cytokines secreted is much greater (p. 18f). The initial nonspecific immune response is so effectively increased by the appearance of these microbe-specific or toxin-specific T cells that the local inflammatory response becomes capable of destroying the infected cells. This inactivates the invading micro-organisms and may expel or control foreign material.

Roecken, Grevers, Burgdorf. Color Atlas of Allergic Diseases (ISBN 3131291915) © 2003 Georg Thieme Verlag

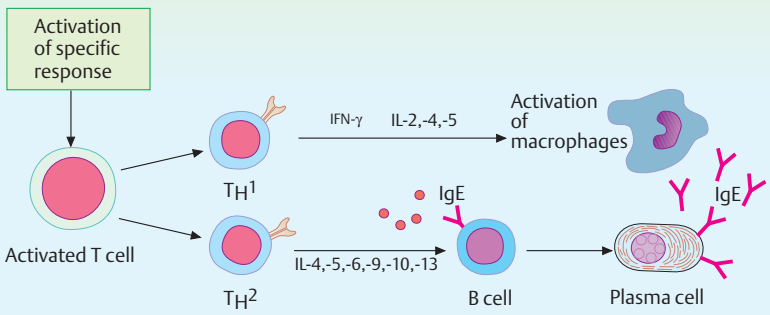
Specific and Nonspecific Defenses



A. Mechanical Barriers



B. Nonspecific Immediate Response



C. Specific or Adaptive Immune Response