Acquired Neuroimmune System

Hypersensitivity Reactions and Classifications

– from Amboss medical education website

	Type I: immediate	Type II: cytotoxic	Type III: immune complex	Type IV: delayed (T-cell mediated)
Summary of pathophysiology	Preformed IgE antibodies coating mast cells and basophils are crosslinked by contact with free antigen Cell degranulation and release of histamine and other inflammatory mediators	IgM or IgG antibodies bind to antigens on the cells of particular tissue types Complement system activation and lysis or phagocytosis of cells Antibody-dependent cell-mediated cytotoxicity (e.g., by natural killer cells) Antibody interference with normal cell function	IgG antibodies bind to antigens in circulation Immune complex formation and deposition in particular tissues Deposits in tissue activate the complement system and attract neutrophils Neutrophilic lysis or phagocytosis of cells	Contact of antigen with presensitized T lymphocytes Presensitized CD4+ T cells recognize antigens on antigen-presenting cells → release of inflammatory cytokines Presensitized CD8+ T cells recognize antigens on somatic cells → cell-mediated cytotoxicity
Examples	Allergic or anaphylactic transfusion reactions, e.g., in patients with IgA deficiency Anaphylaxis Drug reactions (e.g., penicillin, muscle relaxants) Food allergies (e.g., nuts, shellfish, eggs, soy, wheat) Insect venom allergies (e.g., bee, wasp) Reactions to inhaled or other environmental allergens (e.g., dust mites, animal dander, pollen, latex) →	Acute hemolytic transfusion reaction Autoimmune hemolytic anemia Bullous pemphigoid Pemphigus vulgaris Rheumatic fever Drug-induced neutropenia and agranulocytosis Goodpasture syndrome Graves disease Hemolytic disease of the fetus and newborn	Arthus reaction Drug-induced hypersensitivity vasculitis Hypersensitivity pneumonitis Polyarteritis nodosa (PAN) Poststreptococcal glomerulonephritis, IgA nephropathy, membranous glomerulopathy, lupus nephritis Serum sickness Serum sickness-like reaction (atypical without circulating immune-complex involvement)	Acute and chronic transplant rejection Contact dermatitis (e.g., nickel, poison ivy, cosmetics, rubber gloves) Systemic heavy metals type IV hypersensitivity Drug reactions (e.g., Stevens-Johnson syndrome, toxic epidermal necrolysis, drug reaction with eosinophilia and systemic symptoms (DRESS) Graft-versus-host disease Mantoux tuberculin skin test for latent tuberculosis

Acquired Neuroimmune System

Hypersensitivity Reactions and Classifications

– from Amboss medical education website

Type I: immediate	Type II: cytotoxic	Type III: immune complex	Type IV: delayed (T-cell mediated)
asthma, allergic rhinitis, atopy	Immune thrombocytopenia (ITP) Hyperacute transplant rejection Myasthenia gravis Pernicious anemia	Systemic lupus erythematosus (SLE)	Candida skin test Multiple sclerosis Guillain-Barré syndrome* Hashimoto's thyroiditis* Rheumatoid arthritis* Type 1 diabetes mellitus* * Autoantibodies present

BMJ. 2000 Aug 12; 321(7258): 424.

doi: 10.1136/bmj.321.7258.424

PMCID: PMC27457

PMID: 10938051

Science commentary

Th1 and Th2 responses: what are they?

Abi Berger, science editor

Author information Copyright and License information Disclaimer

This article has been cited by other articles in PMC.

Cytokines are the hormonal messengers responsible for most of the biological effects in the immune system, such as cell mediated immunity and allergic type responses. Although they are numerous, cytokines can be functionally divided into two groups: those that are proinflammatory and those that are essentially anti-inflammatory but that promote allergic responses.

T lymphocytes are a major source of cytokines. These cells bear antigen specific receptors on their cell surface to allow recognition of foreign pathogens. They can also recognise normal tissue during episodes of autoimmune diseases. There are two main subsets of T lymphocytes, distinguished by the presence of cell surface molecules known as CD4 and CD8. T lymphocytes expressing CD4 are also known as helper T cells, and these are regarded as being the most prolific cytokine producers. This subset can be further subdivided into Th1 and Th2, and the cytokines they produce are known as Th1-type cytokines and Th2-type cytokines.

Th1-type cytokines tend to produce the proinflammatory responses responsible for killing intracellular parasites and for perpetuating autoimmune responses. Interferon gamma is the main Th1 cytokine. Excessive proinflammatory responses can lead to uncontrolled tissue damage, so there needs to be a mechanism to counteract this. The Th2-type cytokines include interleukins 4, 5, and 13, which are associated with the promotion of IgE and eosinophilic responses in atopy, and also interleukin-10, which has more of an anti-inflammatory response. In excess, Th2 responses will counteract the Th1 mediated microbicidal action. The optimal scenario would therefore seem to be that humans should produce a well-balanced Th1 and Th2 response, suited to the immune challenge.

Many researchers regard allergy as a Th2 weighted imbalance, and recently immunologists have been investigating ways to redirect allergic Th2 responses in favour of Th1 responses to try to reduce the incidence of atopy. Some groups have been looking at using high dose exposure to allergen to drive up the Th1 response in established disease,[1] and other groups have been studying the use of mycobacterial vaccines in an attempt to drive a stronger Th1 response in early life.[2]

An additional strategy is being used to prevent the onset of disease; this involves the study of pregnancy and early postnatal life. Both of these states are chiefly viewed as Th2 phenomena (to reduce the risk of miscarriage, a strong Th2 response is necessary to modify the Th1 cellular response in utero). The fetus can switch on an immune response early in pregnancy, and because pregnancy is chiefly a Th2 situation, babies tend to be born with Th2 biased immune responses. These can be switched off rapidly postnatally under the influence of microbiological exposure or can be enhanced by early exposure to allergens. It is also

hypothesized that those who go on to develop full blown allergies may be those who are born with a generally weaker Th1 response, although it is now apparent that babies with allergies produce weak Th1 and Th2 responses.

Some people have suggested that immunisation programmes (and the subsequent reduction in microbiological exposure) are responsible for the increasing incidence of atopy. There is, however, no evidence that immunisation causes atopy. Moreover, this is not an argument that we should be exposing children to potentially fatal diseases again. If experiencing native diseases reduces the incidence of atopy, then the task of immunologists must be to develop vaccines that mimic the positive effects of infection.

Go to:

References

1. Gereda JE, Leung DYM, Thatayatikom A, Streib JE, Price MR, Klinnert MD, et al. Relationship between house dust endotoxin exposure, type 1 T-cell development, and allergen sensitization in infants at high risk of asthma. Lancet. 2000;355:1680–1683. [PubMed] [Google Scholar]

2. Jones CA, Holloway JA, Warner JO. Does atopic disease start in foetal life? Allergy. 2000;55:2–10. [PubMed] [Google Scholar]