Table 16.3 Immune mechanisms in some skin diseases[a]

Reaction and disease	Lesion	Association	Mechanism
Inactivation			
Pemphigus	Intraepithelial bullae	Autoallergic diseases	Autoantibody to desmosome protein
Pemphigoid	Subepidermal bullae	Neoplasm, autoallergy	Autoantibody to basement membrane
Epidermolysis bullosa	Subepidermal bullae	Autoimmunity	Autoantibody to basement membrane
Cytotoxic			
Dermatitis herpetiformis	Subepidermal bullae	Gluten enteropathy	IgA, alternate pathway activation?
Herpes gestationis	Subepidermal bullae	Pregnancy	Properdin, C3 in basement membrane
Cutaneous lupus	Basement membrane degeneration	Systemic and discoid lupus	Ig and C3 in basement membrane
Dermatomyositis	Microvascular injury	Myositis, lupus	C5b-C9 in vessels
Vitiligo	Depigmentation	Autoallergic diseases	Antibody to melanocytes
Alopecia areata	Focal hair loss	Endocrinopathy	Antibody to hair bulb capillaries
Immune complex			
Erythema nodosum	Subcutaneous vasculitis	Infection, systemic lupus erythematosus	Immune complex
Erythema marginatum	Subcutaneous vasculitis	Rheumatic fever	Immune complex
Cutaneous vasculitis	Subcutaneous vasculitis	Infections	Immune complex
Temporal arteritis	Temporal vasculitis	Actinic damage	Immune complex?
Anaphylactic reactions			
Mastocytosis	Whealing and anaphylaxis	Neoplasia	Increase in tissue mast cells
Cutaneous anaphylaxis	Wheal-and-flare	Allergic reaction	IgE-mediated mast cell degranulation
Giant urticaria	Large whealing	Allergy	Mast cell degranulation, IgE?
Angioedema	Nonpitting edema	Injury	Decrease in C1 esterase inhibitor
Hypocomplementemic urticarial vasculitis	Urticaria, vasculitis	Injury	C1q activation
Atopic dermatitis	Pruritic skin rash	Allergy	IgE, activation of macrophages
T_{CTL} cells			
Contact dermatitis	Spongiosis, vesiculation	Poison ivy, oak	T_{CTL}-cell reaction to hapten
Graft-versus-host	Intraepithelial necrosis	Bone marrow transplant	T_{CTL}-cell reaction to epithelial antigens
Viral exanthems	Intraepithelial necrosis	Virus infection	T_{CTL}-cell reaction to viral antigens
Erythema multiforme	Intraepithelial necrosis	Drugs, infection (herpes simplex virus)	T_{CTL} cells, antigens on endothelial cells
T_{DTH} cells			
DTH skin tests	Lymphocytic vasculitis	Skin test antigens	T_{DTH}-cell reaction to antigen, lymphokines
Syphilis	Perivascular and diffuse	*Treponema pallidum* infection	T_{DTH}-cell reaction to *T. pallidum* antigens
Psoriasis	Corneum parakeratosis	Rheumatoid arthritis	Th1, IFN-γ, TNF-α, keratinocyte lymphokines
Toxic epidermal necrosis	Epidermal necrosis	Drugs	Macrophage activation, TNF-α
Granulomatous reactions			
Leprosy	Subcutaneous granulomas	*Mycobacterium leprae* infection	T_{DTH} cells, granuloma formation
Gumma	Granulomas, various	*T. pallidum* infection	T_{DTH} cells, granuloma formation
Zirconium granuloma	Axillary granulomas	Stick deodorants	Granulomas
Sarcoidosis	Internal granulomas	Sarcoid	Granulomas, unknown

[a]Modified from S. Sell, p. 87–100, *in* R. E. Jordon, ed., *Immunologic Diseases of the Skin*, Appleton & Lange, Norwalk, Conn., 1991.

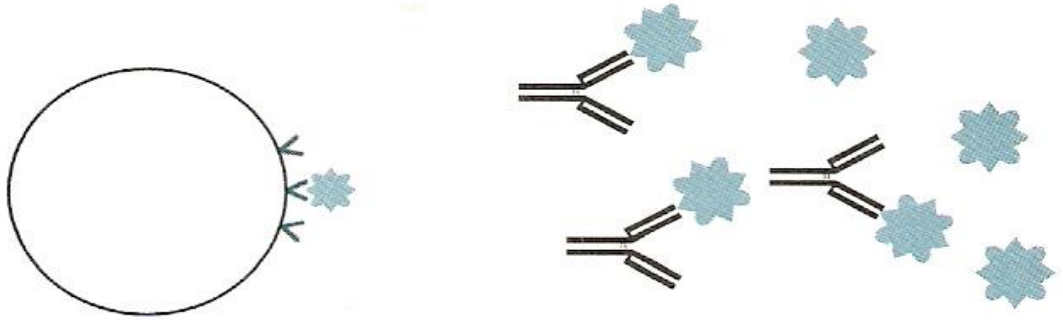
HOW VACCINES WORK: IMMUNE EFFECTOR MECHANISMS AND DESIGNER VACCINES. EXPERT REV. VACCINES 2019

MECHANISM	PREVENTIVE	THERAPEUTIC
Toxin Neutralization		
Diphtheria	Toxin Inactivation	
Tetanus	Toxin Inactivation	
Receptor blockade and toxin neutralization		
Pertussis	Receptor blockade	Toxin Inactivation, T-CTL DTH
Cholera	Receptor blockade	Toxin Inactivation
Anthrax	IgA Receptor blockade	Toxin inactivation
Receptor blockade and immune complex reaction		
H. Influenza	Receptor blockade	Immune complex
Meningococcus	Receptor blockade	Immune complex
Pneumococcus	Receptor blockade	Immune complex
Receptor blockade and T-CTL		
Viral Exanthems		
Smallpox	Receptor blockade	T-CTL
Measles	Receptor blockade	T-CTL
Rubella	Receptor blockade	T-CTL
Varicella	Receptor blockade	T-CTL
Influenza	Receptor blockade	T-CTL
Mumps	Receptor blockade	T-CTL
Ebola	Receptor blockade	T-CTL
Rotavirus	IgA Receptor blockade	T-CTL
Rabies	Receptor blockade	T-CTL
Hepatitis	Receptor blockade	T-CTL
Polio	IgA Receptor blockade	T-CTL
Chagas disease	Receptor blockade	T-CTL

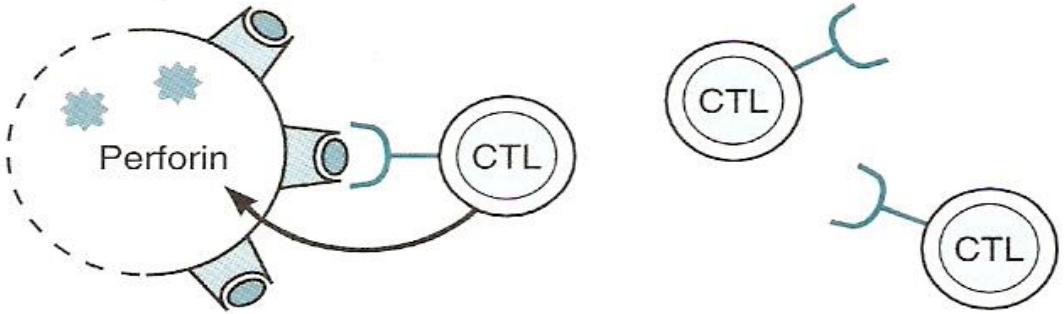
Receptor blockade and DTH			
Yellow Fever	**Receptor blockade**		**DTH**
Japanese Encephalitis	**Receptor blockade**		**DTH**
Dengue	**Receptor blockade**		**DTH**
West Nile	**Receptor blockade**		**DTH**
Zika	**Receptor blockade**		**DTH**
Typhoid	**IgA Receptor blockade**	**DTH**	
Cytolytic			
Malaria	**Cytolytic antibody**		**T-CTL**
Atopic			
Helminths	**Atopic**		**DTH**
Schistosomiasis	**Atopic**		**DTH**
Immune complex			
Staphylococcus aureus	**Immune complex**		**toxin inactivation**
Lyme disease	**Immune complex**		**DTH**
T-CTL			
Papilloma	**T-CTL**		
DTH			
Tuberculosis and Leprosy	**DTH**		**DTH**
Syphilis	**H. Influenza**	**Receptor blockade**	
Meningococcus	**Receptor blockade**		**Immune complex**
Pneumococcus	**Receptor blockade**		**Immune complex**
Receptor blockade and T-CTL			
Viral Exanthems			
Smallpox	**Receptor blockade**		**T-CTL**
Measles	**Receptor blockade**		**T-CTL**
Rubella	**Receptor blockade**		**T-CTL**
Varicella	**Receptor blockade**		**T-CTL**
Influenza	**Receptor blockade**		**T-CTL**
Mumps	**Receptor blockade**		**T-CTL**

HUMORAL AND CELLULAR DEFENCE MECHANISMS AGINST VIRAL INFECTIONS.

Antibody to virus binds to viral receptors and blocks attachment to cell



$CD8^+$ T-CTL reacts with viral antigens on surface of infected cell; perforin release causes lysis of infected cells



$CD4^+$ T-DTH reacts with viral antigens on surface of infected cell; lymphokines attract and activate phagocytosis by macrophages