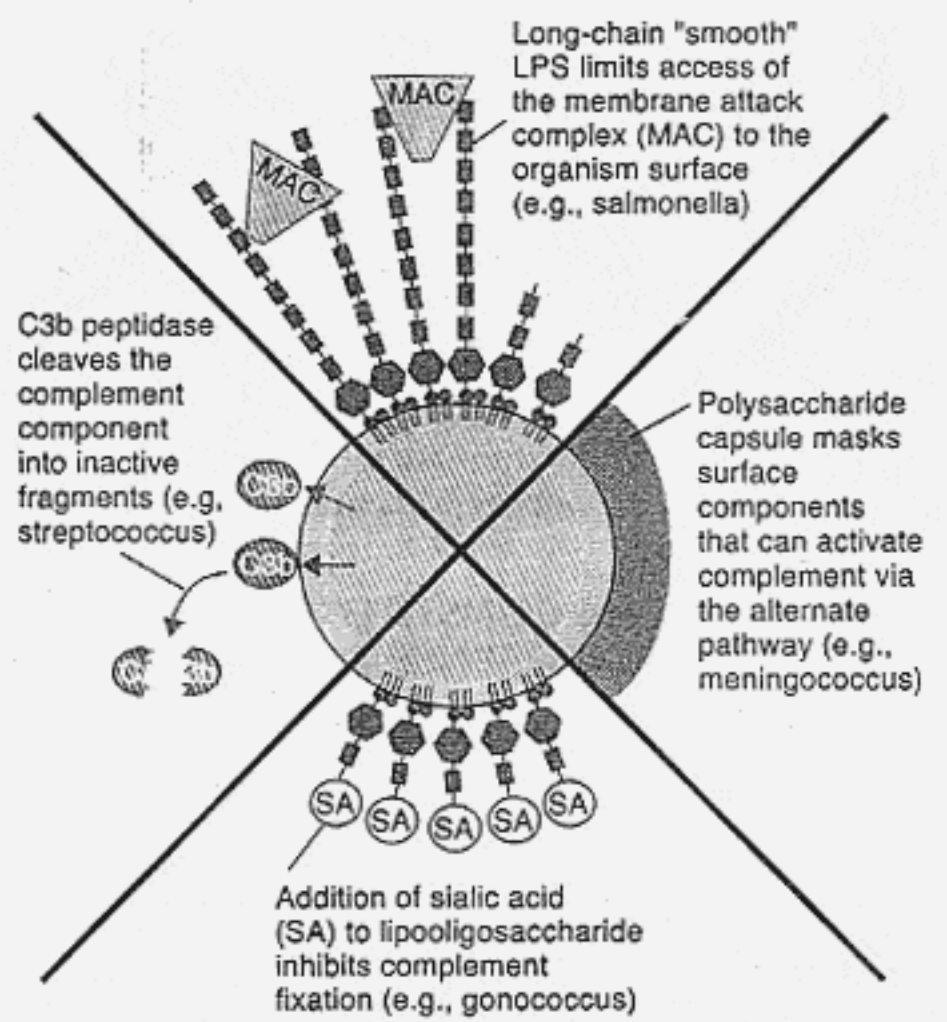
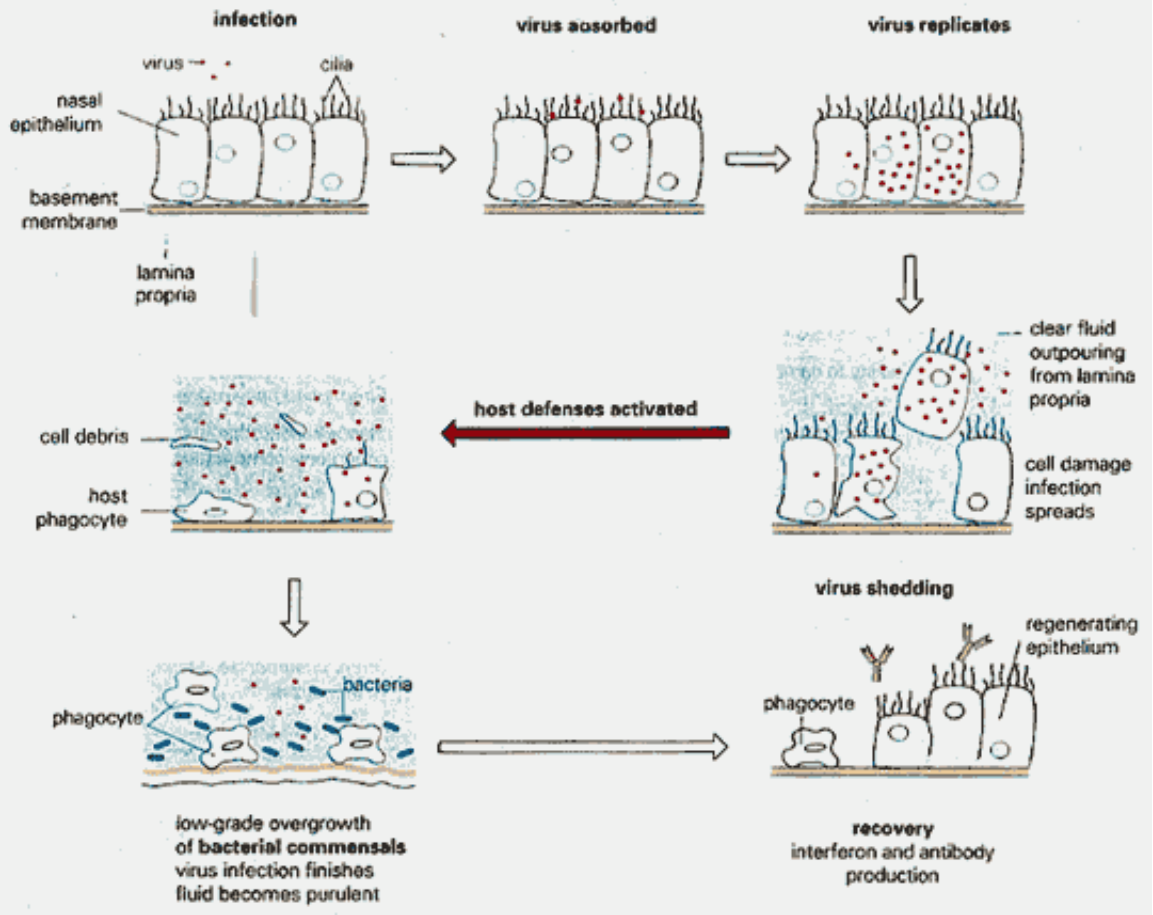


Figure 8.1. Some mechanisms by which microorganisms may prevent harmful effects of complement.


infection
virus absorbed
virus replicates
virus
cilia
nasal epithelium
basement membrane
lamina propria
clear fluid outpouring from lamina propria
cell damage infection spreads
host defenses activated
cell debris
host phagocyte
virus shedding
regenerating epithelium
phagocyte
bacteria
low-grade overgrowth of bacterial commensals virus infection finishes fluid becomes purulent
recovery
interferon and antibody production

10b

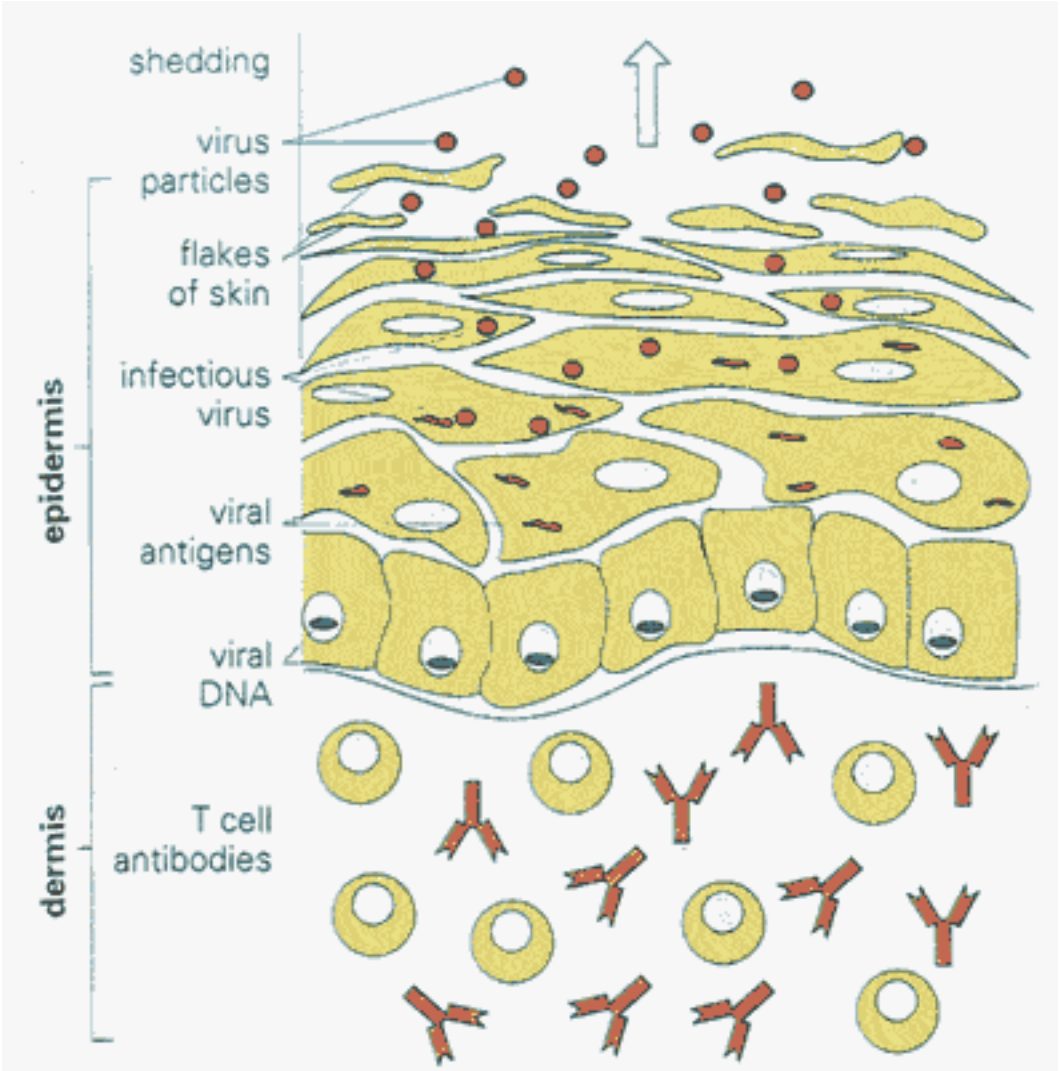


Fig. 11.2 Wart virus replication in epidermis – a privileged site? ell differentiation such as keratinization controls virus replication, and as a result virus matures when it is physically removed from immune defenses.

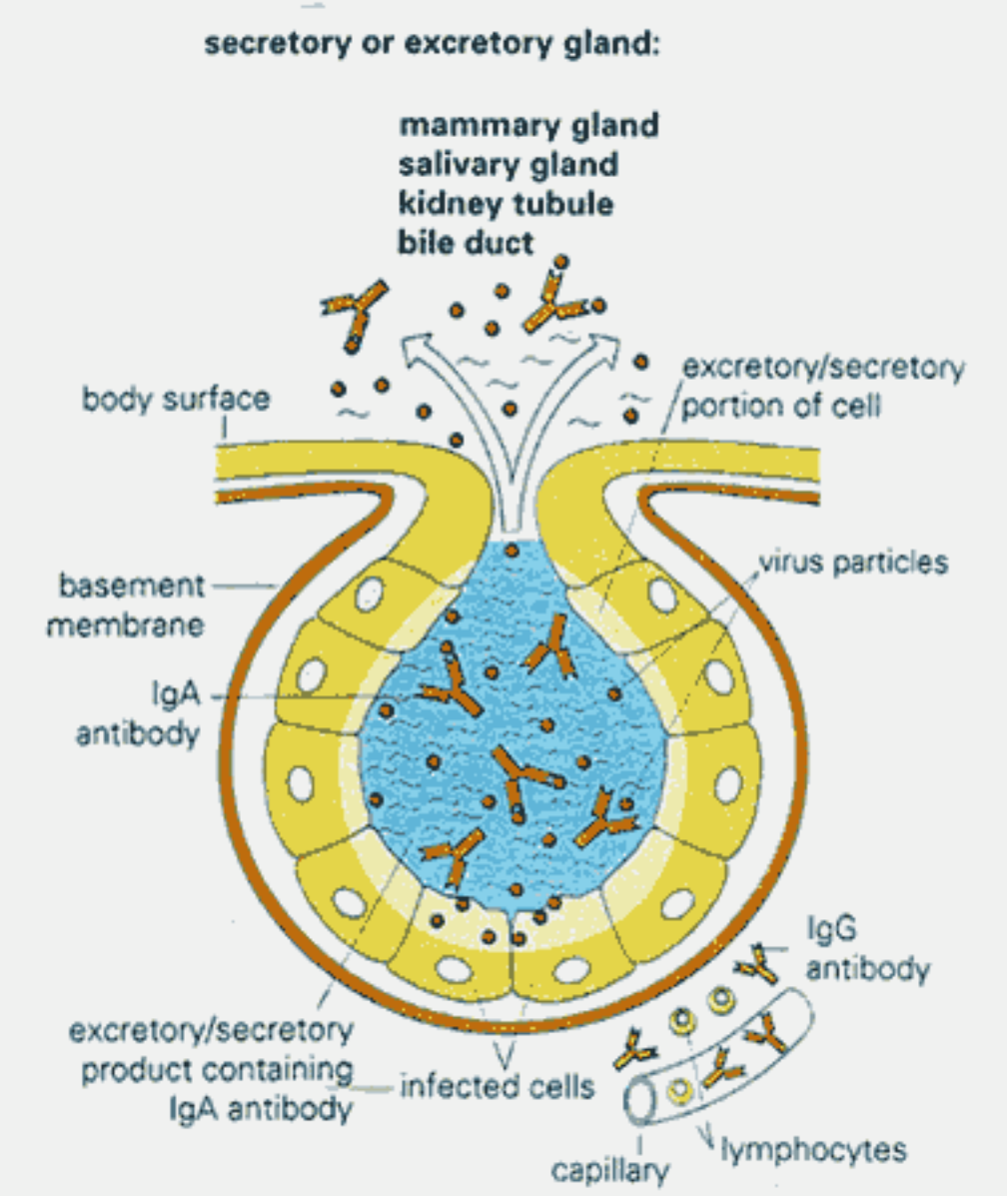


Fig. 11.1 Viral infection of cell surfaces facing the external world. Infection of the surface epithelium of, for instance, a secretory or excretory gland allows direct shedding of the virus to the exterior, as well as avoidance of host immune defenses.

10d

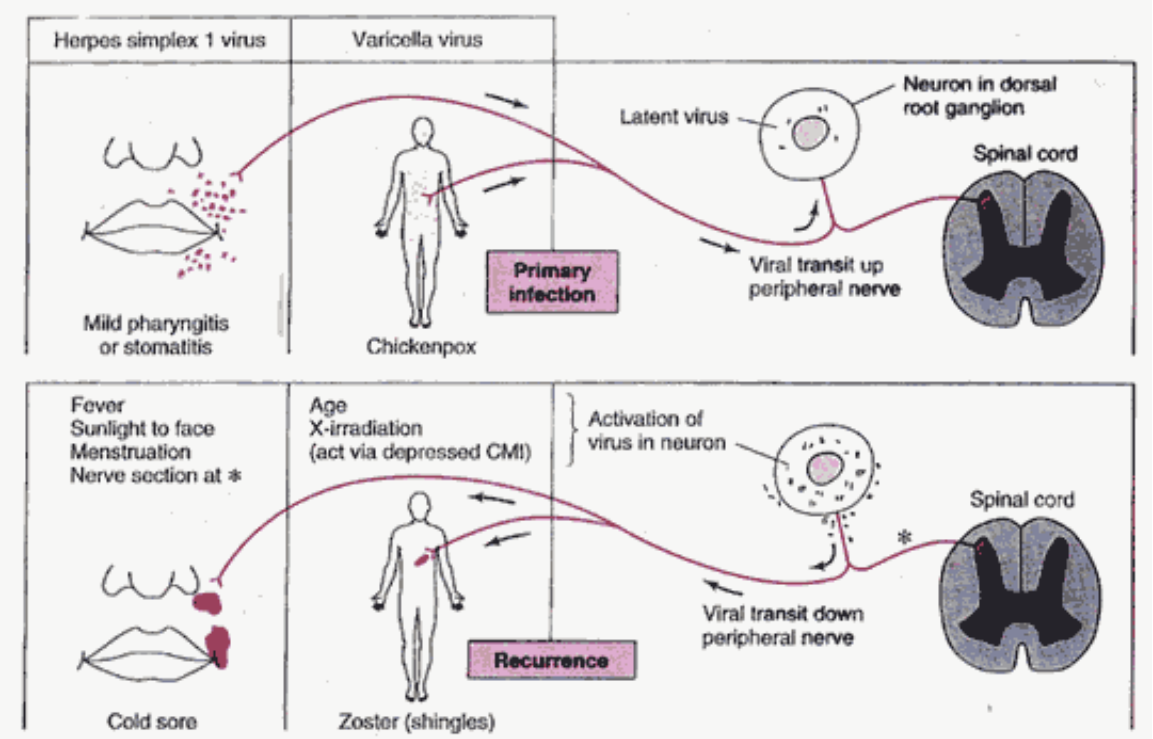


Figure 30–5. Latent infections by herpesviruses. Examples are shown for both herpes simplex and varicella-zoster viruses. Primary infections occur in childhood or adolescence, followed by establishment of latent virus in cerebral or spinal ganglia. Later activation causes recurrent herpes simplex or zoster. Recurrences are rare for zoster. (Reproduced, with permission, from Mims CA, White DO: *Viral Pathogenesis and Immunology.* Blackwell, 1984.)

10e

MIMICRY AND UPTAKE OF HOST ANTIGENS

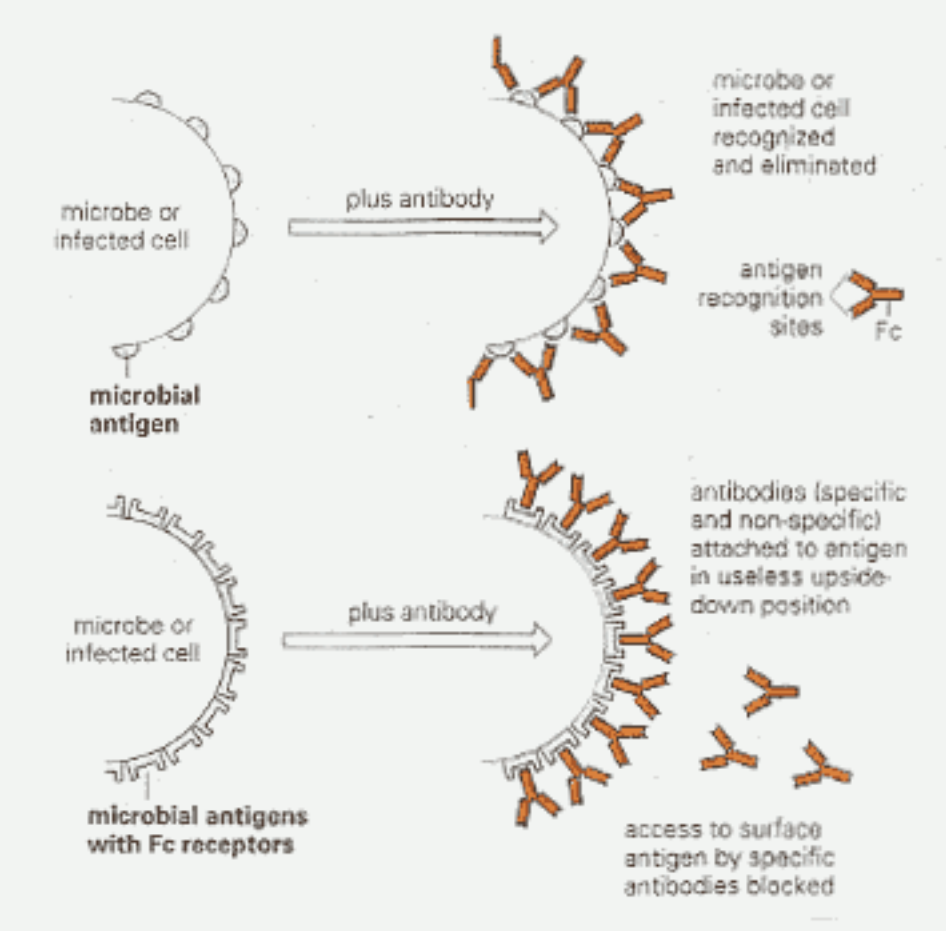
antigen	parasite	corresponding host antigen
mimicry	Epstein–Barr virus	human fetal thymus*
	streptococci	cardiac muscle (meromyosin)
	klebsiella	HLA-B27**
	Mycobacterium tuberculosis	65 kDa heat shock protein
	Neisseria meningitidis	embryonic brain
	treponema	cardiolipin†
	Mycoplasma pneumoniae	erythrocytes††
	plasmodia	thymosin-α_1
	Trypanosoma cruzi	heart, nerve
	schistosoma	glutathione transferase
antigen uptake	cytomegalovirus	β_2-microglobulin
	schistosoma	glycolipids, HLA, Ig etc.
	filarial nematodes	albumin

* also cross-reacts with erythrocytes of certain species and is the basis for the Paul Bunnell (heterophil antibody) test

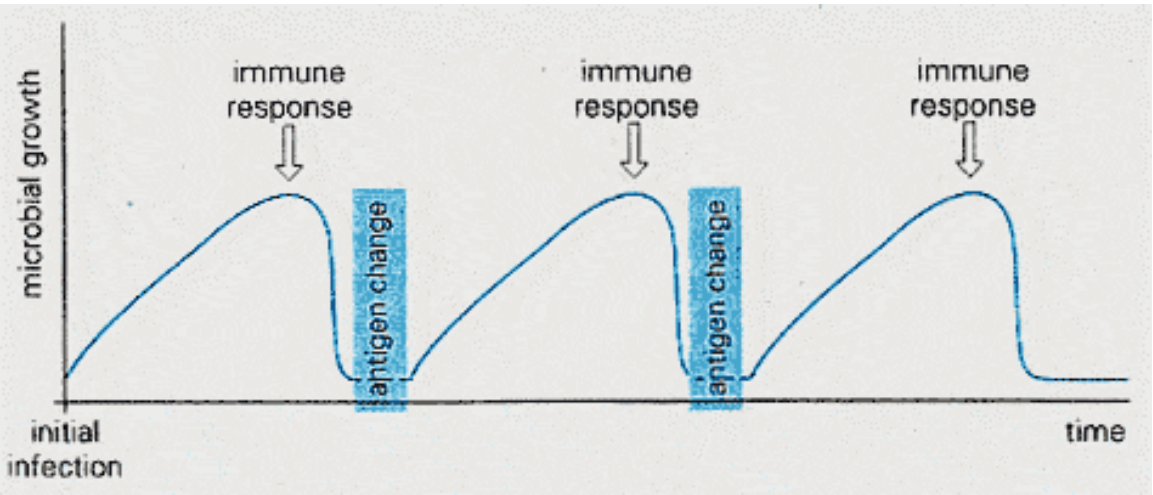
** possible basis for ankylosing spondylitis

† basis for Wassermann-type antibody test for syphilis

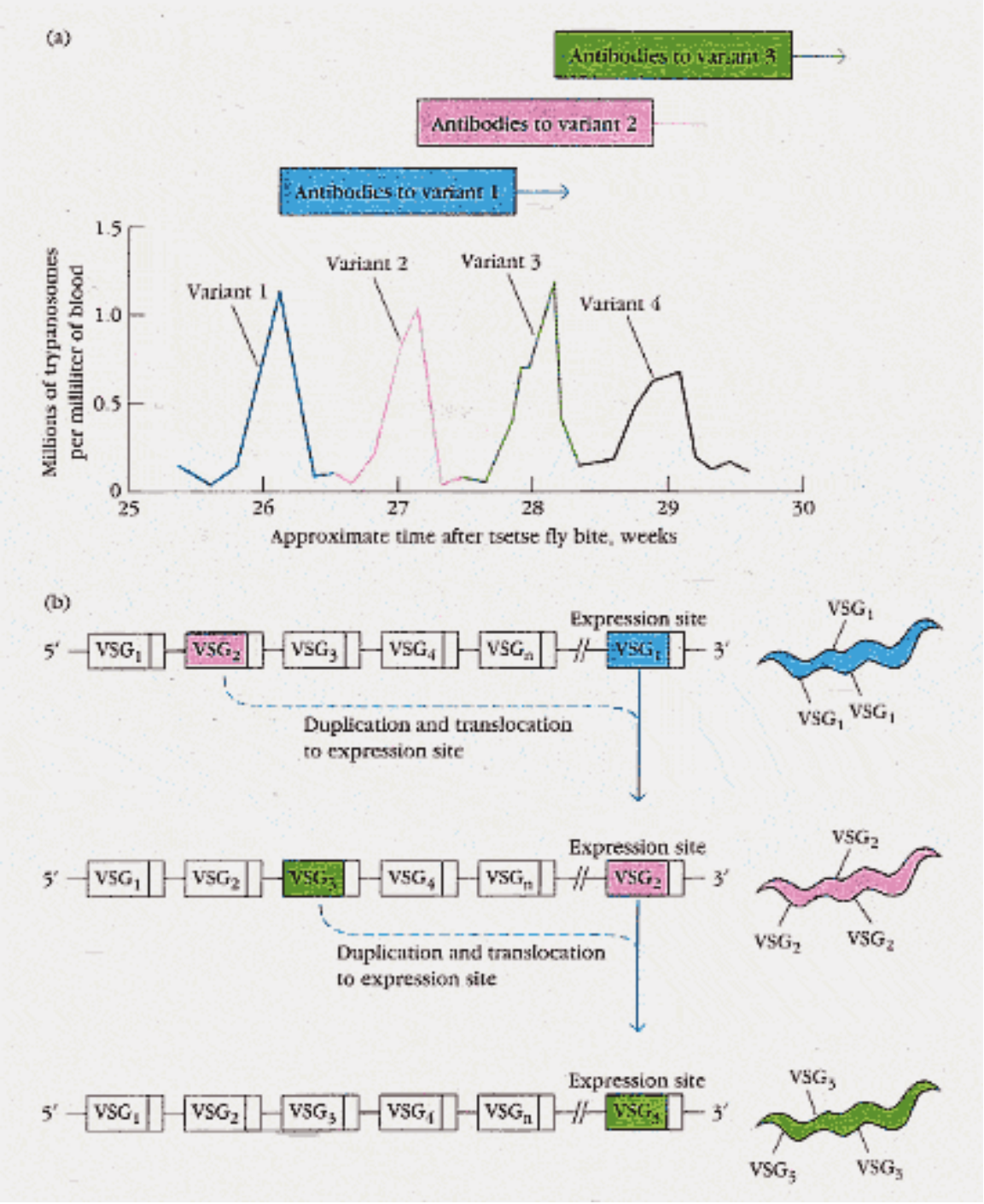
†† basis for cold agglutinin test


microbe or infected cell
plus antibody
microbe or infected cell recognized and eliminated
antigen recognition sites
Fc
microbial antigen
antibodies (specific and non-specific) attached to antigen in useless upside-down position
microbe or infected cell
plus antibody
microbial antigens with Fc receptors
access to surface antigen by specific antibodies blocked

10g


microbial growth
immune response
immune response
immune response
antigen change
antigen change
initial infection
time

10h


(a)
Antibodies to variant 3
Antibodies to variant 2
Antibodies to variant 1
Millions of trypanosomes per milliliter of blood
1.5
1.0
0.5
0
Variant 1
Variant 2
Variant 3
Variant 4
25
26
27
28
29
30
Approximate time after tsetse fly bite, weeks
(b)
Expression site
5′
VSG1
VSG2
VSG3
VSG4
VSGn
3′
Duplication and translocation to expression site
VSG1
VSG2
VSG3

10i

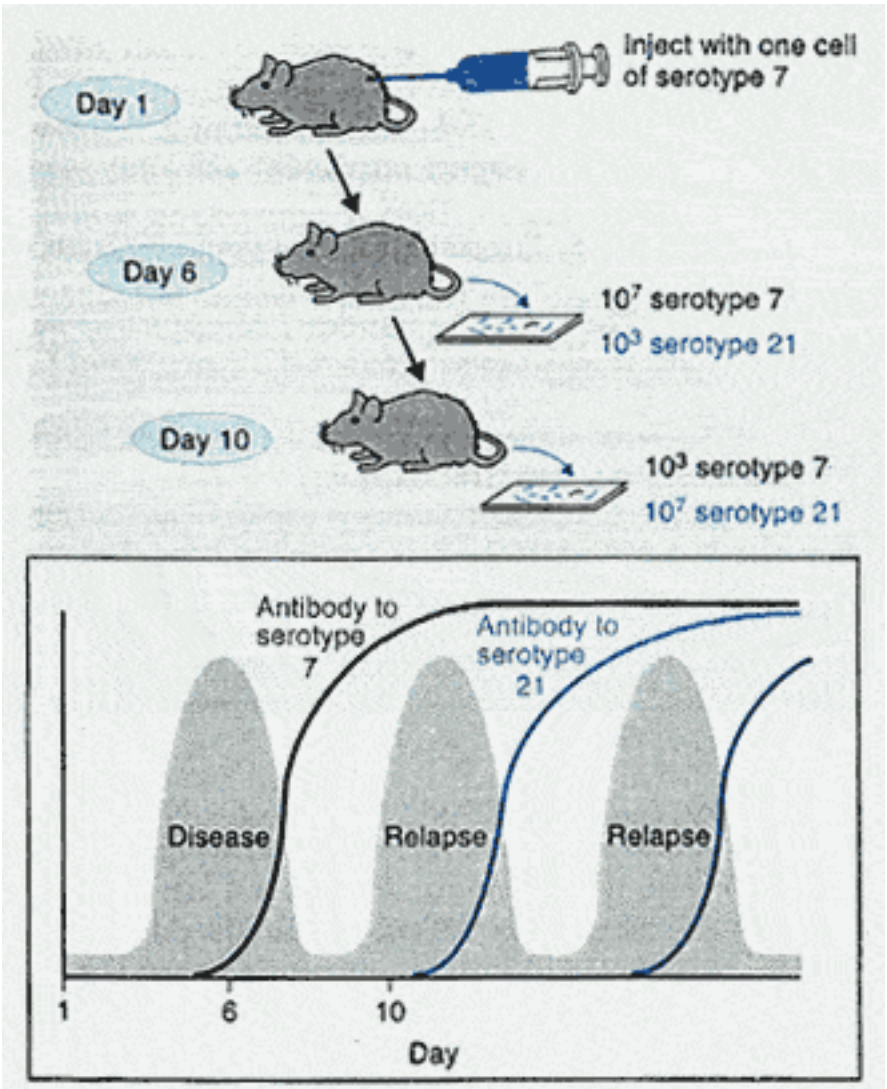


Figure 8.4. An experimental infection in which the development of type-specific antibodies against *Borrelia* sp. is associated with the emergence of new antigenic types. The emergence of each new type is associated with a relapse of disease; hence the common name for these borrelia infections—"relapsing fever." The rapid emergence of the new antigenic types is mediated by successive DNA rearrangement (see the paradigm in Chapter 14, "Neisseria" for more details).

10j

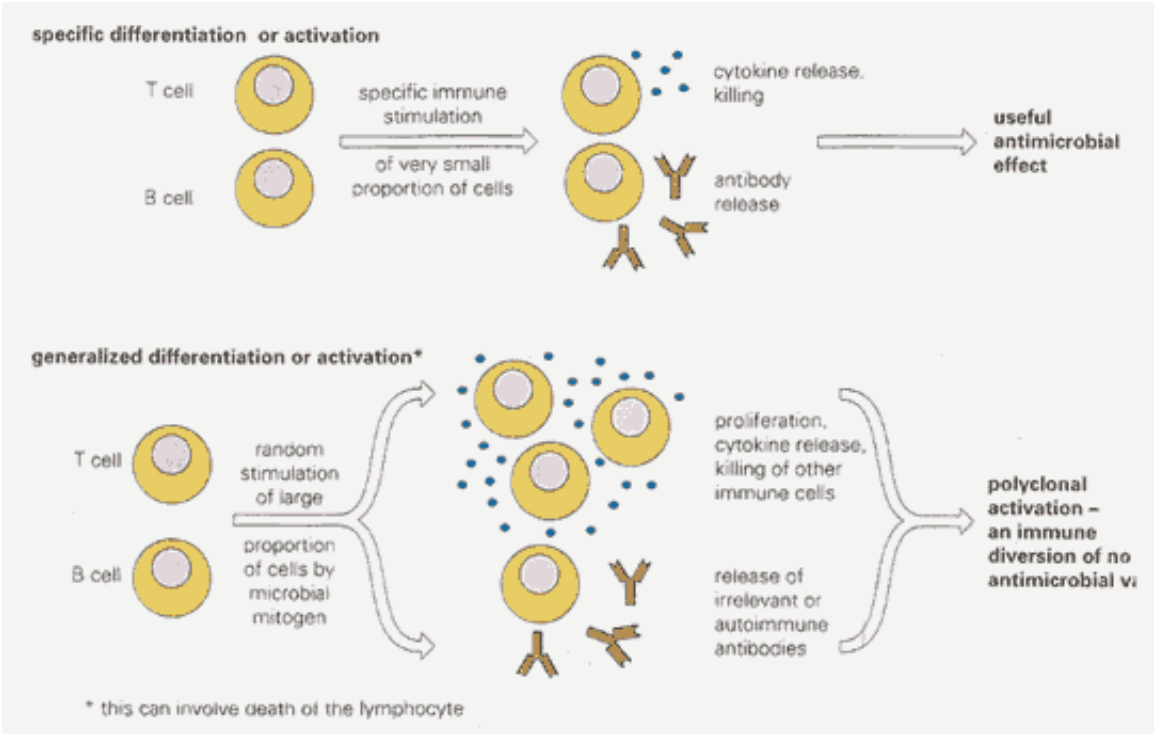
DEPRESSED IMMUNE RESPONSES CAUSED BY MICROBIAL INFECTIONS

parasite		feature of immunosuppression	mechanism
viruses	HIV	↓Ab ↓CMI long lasting	↓CD4⁺ T cells immunosuppressive molecule (gp41) ↓ antigen presentation by infected APC polyclonal activation of B cells
	Epstein–Barr virus	↓CMI temporary	includes polyclonal activation of infected B cells
	measles	↓CMI temporary	differentiation blocked in infected T and B cells
	cytomegalovirus	↓CMI temporary	unknown; infection of very occasional mononuclear cells
	varicella–zoster virus mumps	↓CMI temporary	infection of T cells
bacteria	*M. lepraе* (lepromatous leprosy)	↓CMI	polyclonal activation of B cells induction of suppressor T cells
protozoa	*Trypanosoma* *Plasmodia* *Toxoplasma* *Leishmania*	↓Ab ↓CMI	?

Ab, antibody; CMI, cell-mediated immunity; APC, antigen-presenting cell

Fig. 11.8 Depressed immune responses in microbial infections. In most cases the mechanisms are unclear, but possible important factors are listed. For HIV, the depressed responses are seen later, after initial neutralizing antibody and cytotoxic cell responses. There are at least nine possible mechanisms involved in HIV immunosuppression, but decreased numbers of CD4⁺ T cells is probably the most important.

10k



10l