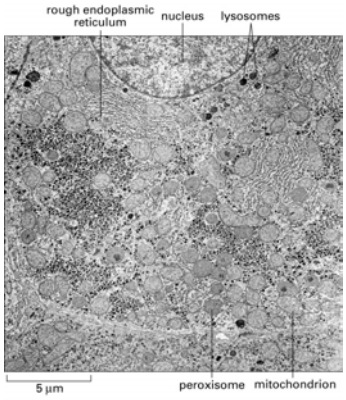


Figure 12–2. Molecular Biology of the Cell, 4th Edition.

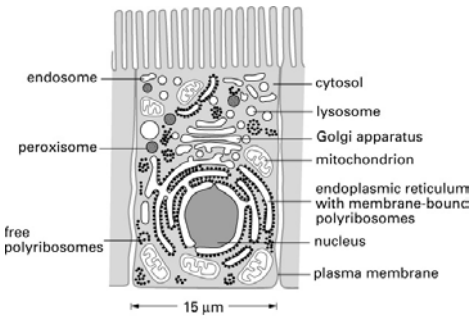
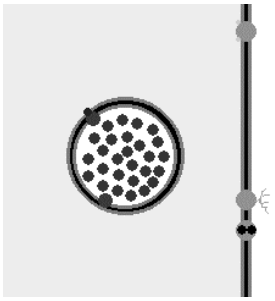


Figure 12–1. Molecular Biology of the Cell, 4th Edition.

Eksocytose



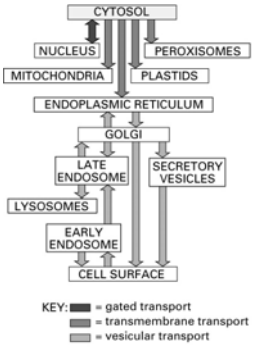


Figure 12–6. Molecular Biology of the Cell, 4th Edition.

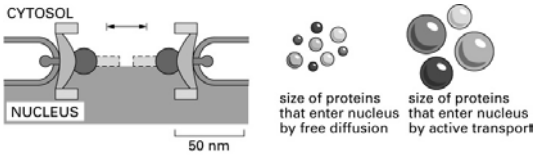


Figure 12–11. Molecular Biology of the Cell, 4th Edition.

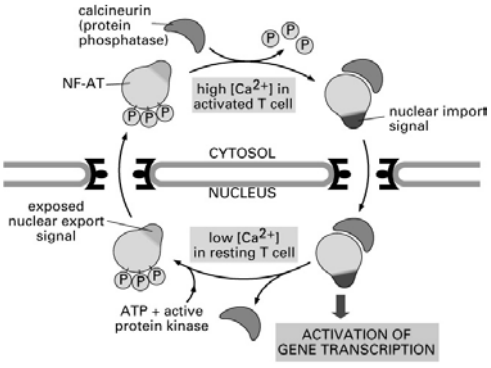


Figure 12–19. Molecular Biology of the Cell, 4th Edition.

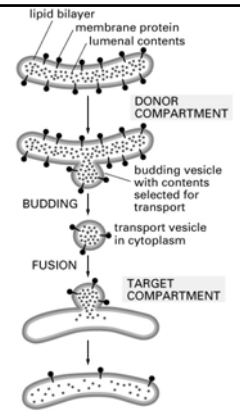


Figure 12–7. Molecular Biology of the Cell, 4th Edition.

Most of the exocytosed material comes from ER

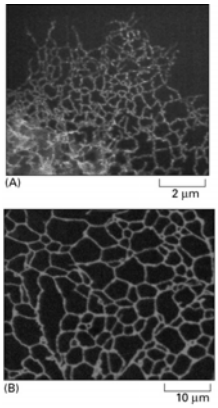


Figure 12–35. Molecular Biology of the Cell, 4th Edition.

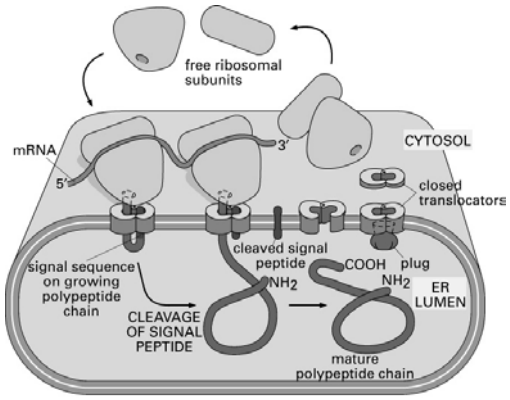


Figure 12–40. Molecular Biology of the Cell, 4th Edition.

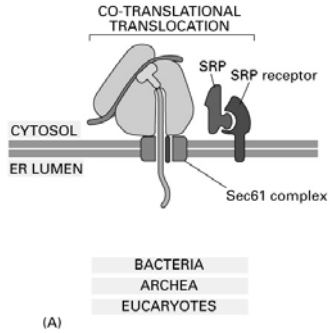


Figure 12–45 part 1 of 2. Molecular Biology of the Cell, 4th Edition

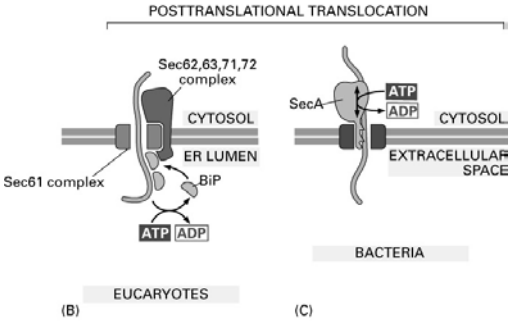


Figure 12–45 part 2 of 2. Molecular Biology of the Cell, 4th Edition.

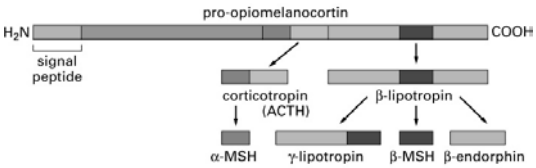


Figure 13–58. Molecular Biology of the Cell, 4th Edition.

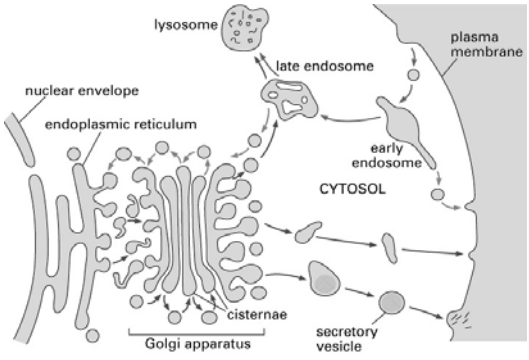
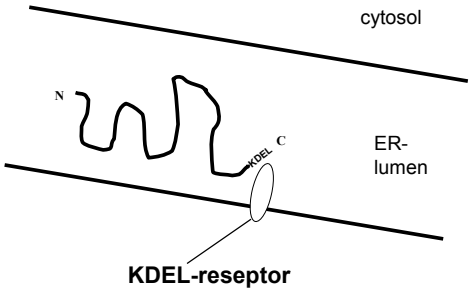


Figure 13–3. Molecular Biology of the Cell, 4th Edition.

Retensjon i ER-lumen

* Spesifikk sekvens: **KDEL**
* C-terminal



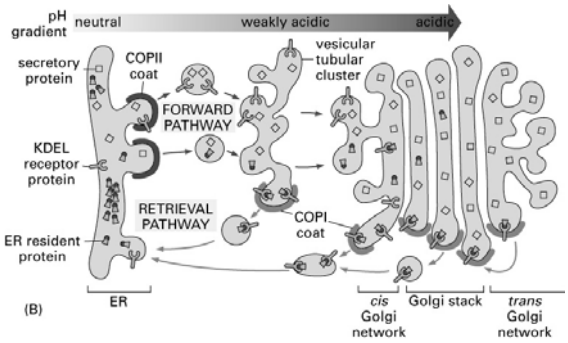
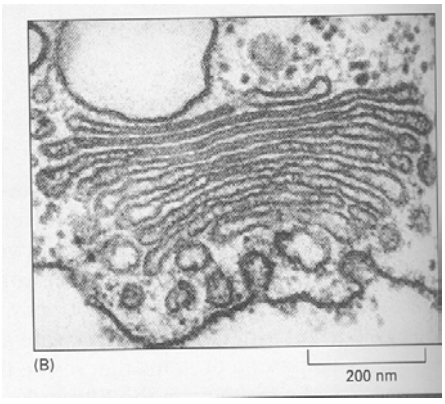
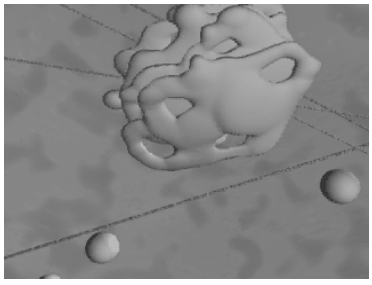
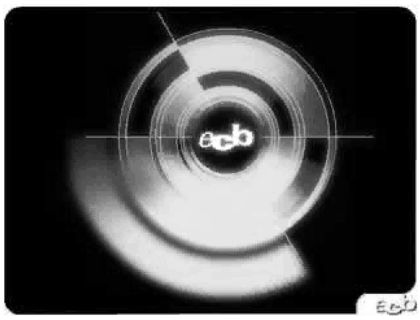


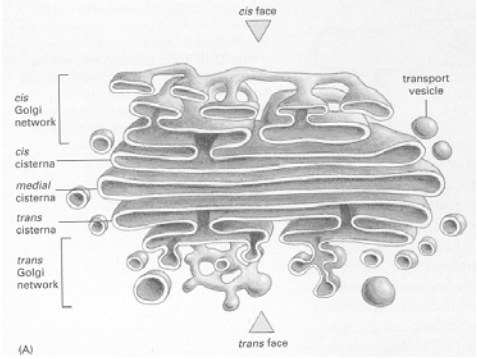
Figure 13–21 part 2 of 2. Molecular Biology of the Cell, 4th Edition.

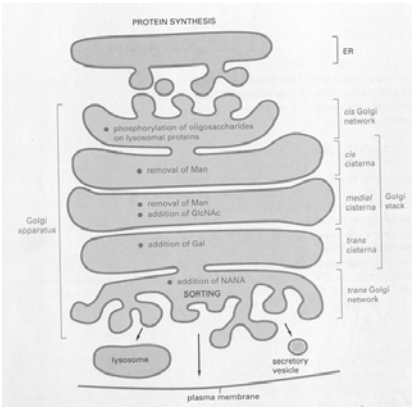


Golgi dynamics



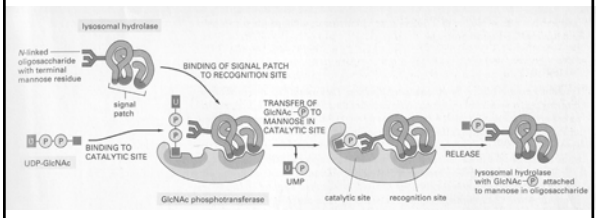




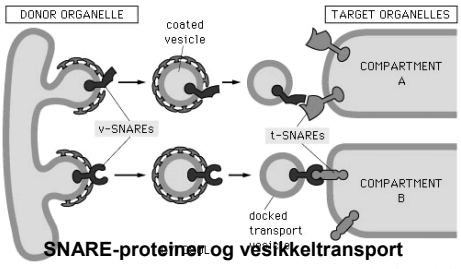


"Merking" av lysosomale enzymer med mannose-6-fosfat

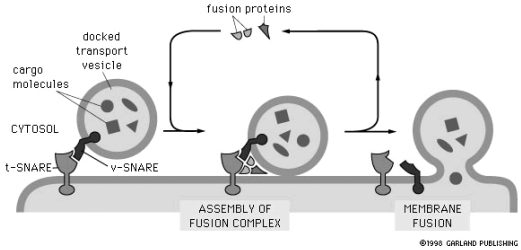
1. Trinn: Konjugering med N-acetylglukosaminfosfat

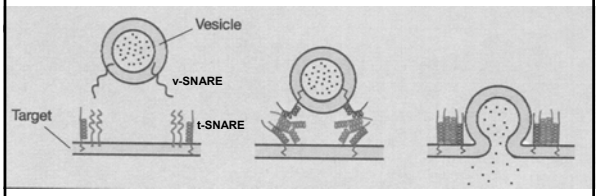


Both cargo and vesicles must be sorted



SNARE-proteiner og vesikkeltransport





SNARE-proteiner
* Spesifisitet i vesikkeltransport
* Katalyserer membranfusjon

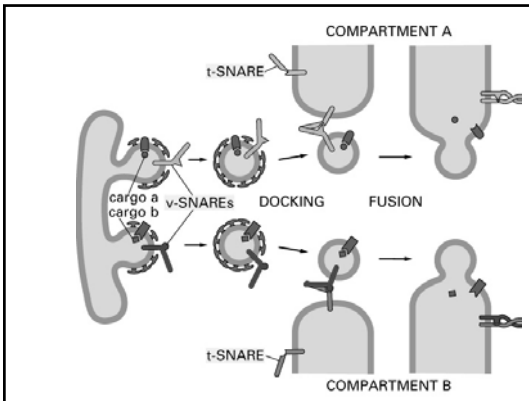


Figure 13–11. Molecular Biology of the Cell, 4th Edition.

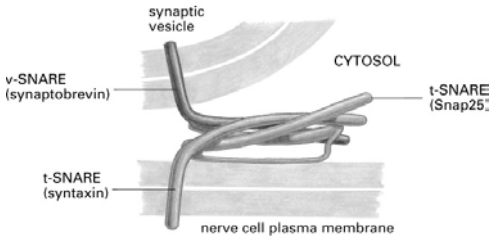


Figure 13–12. Molecular Biology of the Cell, 4th Edition.

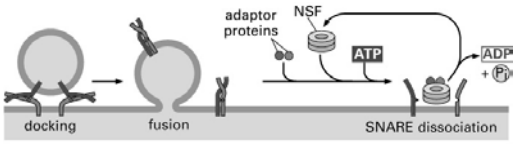


Figure 13–13. Molecular Biology of the Cell, 4th Edition.

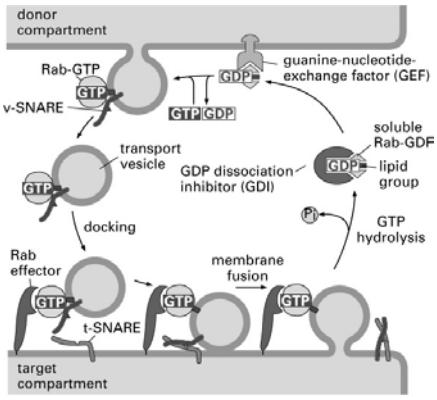


Figure 13–14. Molecular Biology of the Cell, 4th Edition.

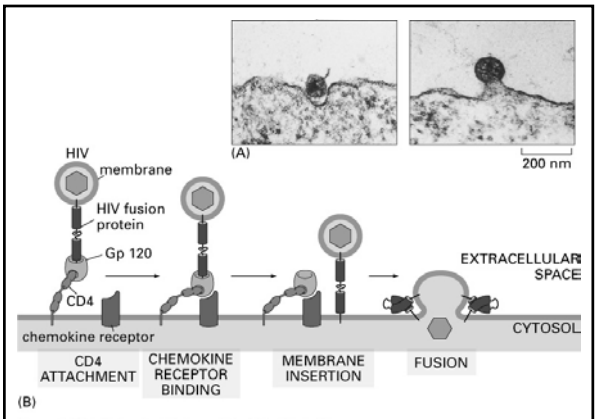


Figure 13–16. Molecular Biology of the Cell, 4th Edition.

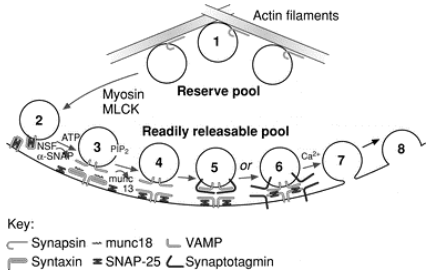
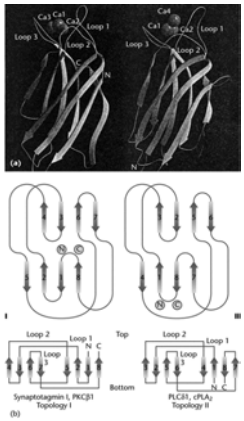
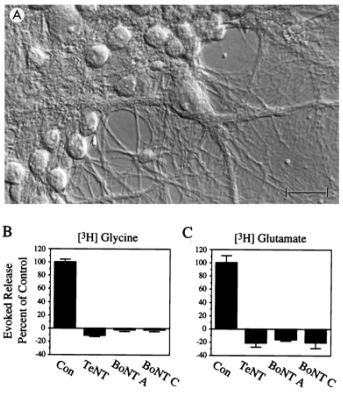


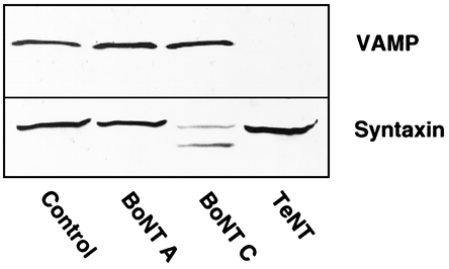
Figure 1 Molecular mechanisms underlying synaptic vesicle exocytosis. A reserve pool of synaptic vesicles (1), linked to actin by synapsin, is recruited to docking sites at the active zone by vesicle detachment followed by movement via myosin motors. Following vesicle docking at the presynaptic active zone (2), vesicles are rendered fusion-competent by a series of priming steps (2 → 5 or 6) that may involve ATP-dependent, NSF-mediated disassembly of *cis* SNARE complexes (2 → 3), ATP-dependent synthesis of $PI(4,5)P_2$ (2 → 3), munc18 dissociation from syntaxin possibly catalysed by munc13 (3 → 4), and the formation of loosely assembled (4 → 5) or fully assembled (4 → 6) *trans* SNARE complexes. Ca^{2+} triggering of fusion is depicted as mediated through synaptotagmins on either vesicle or presynaptic membrane. Ca^{2+} binding to synaptotagmins could promote formation of fully assembled *trans* SNARE complexes and fusion (5 → 7). Alternatively, a fully assembled *trans* SNARE complex that initiated hemi-fusion (6) could be acted upon by Ca^{2+}-bound synaptotagmin to promote transition to fusion pore formation and full fusion (6 → 7). Subsequent dilation of the fusion pore and release of neurotransmitter is depicted in (8).

Synaptotagmin is a
Ca sensor





Botulinum and Tetanus toxins cleave SNARE's



Proteins can be separated by gel electrophoresis

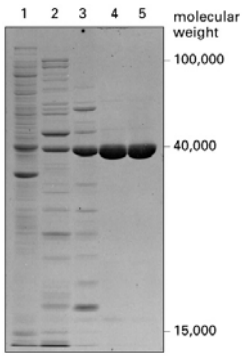


Figure 8–15. Molecular Biology of the Cell, 4th Edition

Proteins separated by electrophoresis can be detected by western blotting using specific antibodies

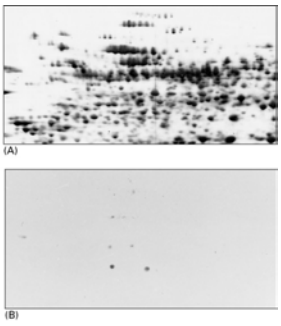
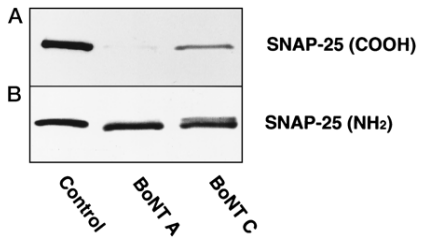


Figure 8–18. Molecular Biology of the Cell, 4th Edition.



Botox produces temporary chemical denervation by blocking the presynaptic release of acetylcholine (Ach) at the neuromuscular junction (NMJ).[4] Botox binds irreversibly to presynaptic cholinergic neuroreceptors, which sets in motion a cascade of events leading to Botox endocytosis. Once in the cytoplasm, a subunit of botulinum toxin (light chain) facilitates the zinc-dependent enzymatic cleavage of a synaptosomal associated membrane protein (SNAP-25).[5][6] SNAP-25 is one of several proteins required for Ach exocytosis and release into the NMJ. Thus, by inactivating SNAP-25, Ach release into the NMJ is prevented and local neuromuscular transmission interrupted. The ensuing localized paralysis typically takes 24 to 48 hours to become fully effective, which reflects the time needed for cellular metabolism of the toxin. In some cases, several days may pass before local paralysis is complete. The localized effects of standard Botox injections last for □90 days.

Regulert eksocytose

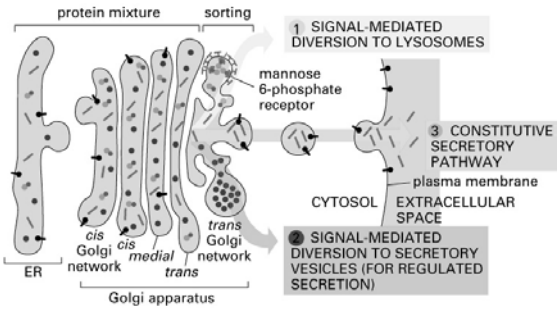


Figure 13–55. Molecular Biology of the Cell, 4th Edition.

Formation of secretory vesicles

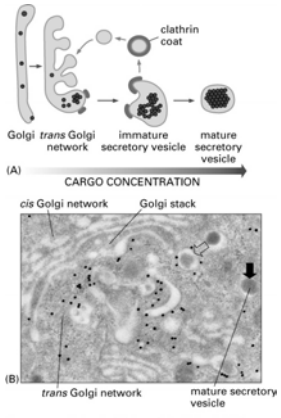
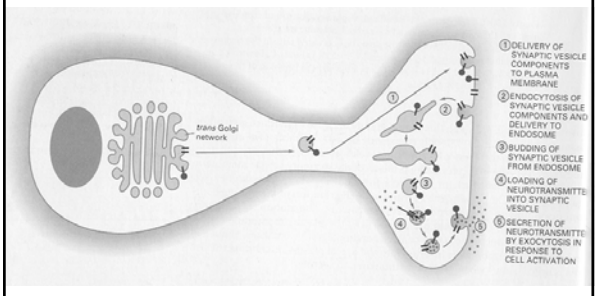
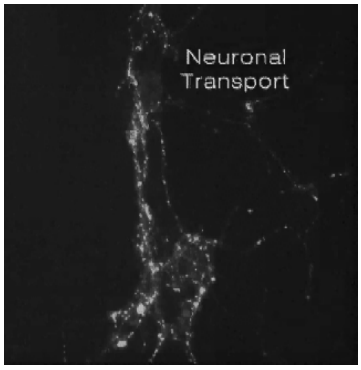
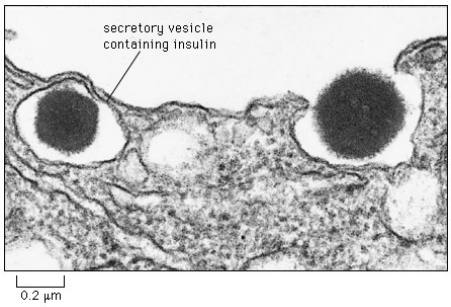


Figure 13-56. Molecular Biology of the Cell, 4th Edition

Eksocytose av synaptiske vesikler







Regulert eksocytose av insulin

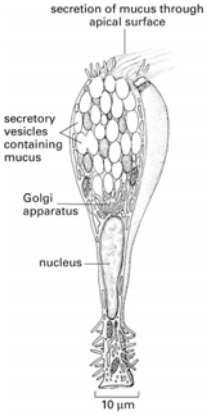


Figure 13–24. Molecular Biology of the Cell, 4th Edition.

Exocytosis of histamine from mast cells

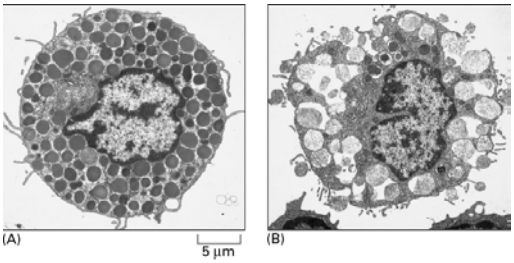


Figure 13–59. Molecular Biology of the Cell, 4th Edition.

Exocytosis can be localized

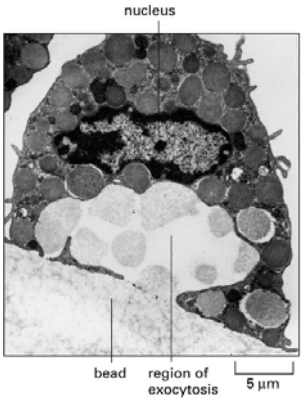


Figure 13–60. Molecular Biology of the Cell, 4th Edition

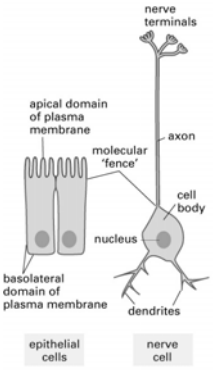


Figure 13–61. Molecular Biology of the Cell, 4th Edition.

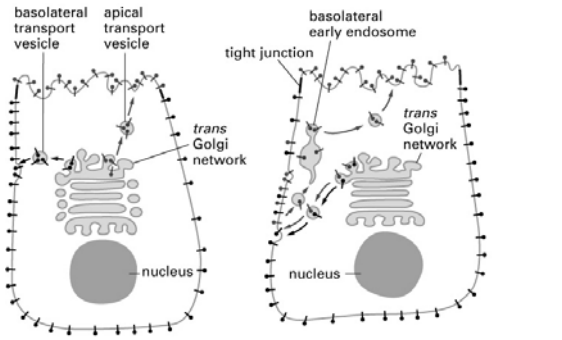


Figure 13–62. Molecular Biology of the Cell, 4th Edition.

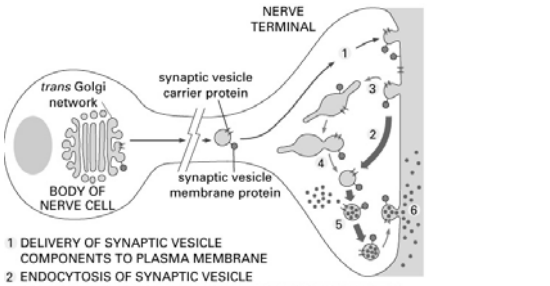
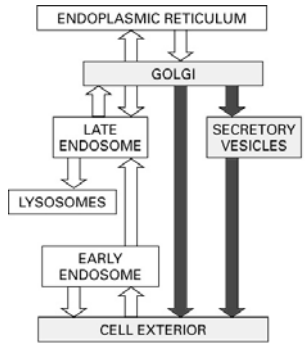


Figure 13–64. Molecular Biology of the Cell, 4th Edition.



Page 757. Molecular Biology of the Cell, 4th Edition

Cytotoxic T-cells killing cancer cells

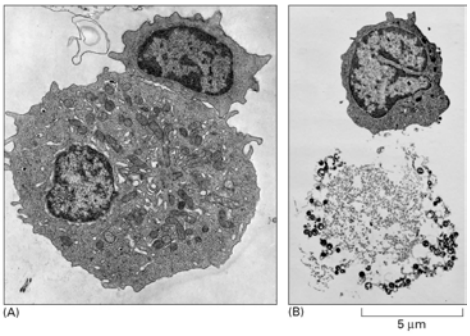


Figure 24–45 part 1 of 2. Molecular Biology of the Cell, 4th Edition.

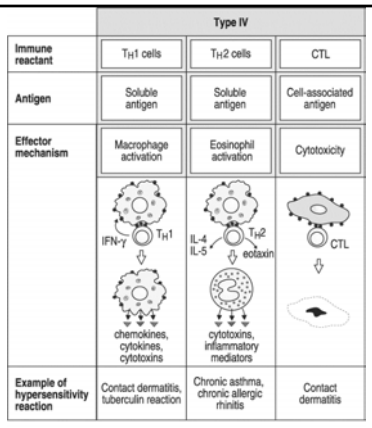


Fig 12.2 part 2 of 2 © 2001 Garland Science

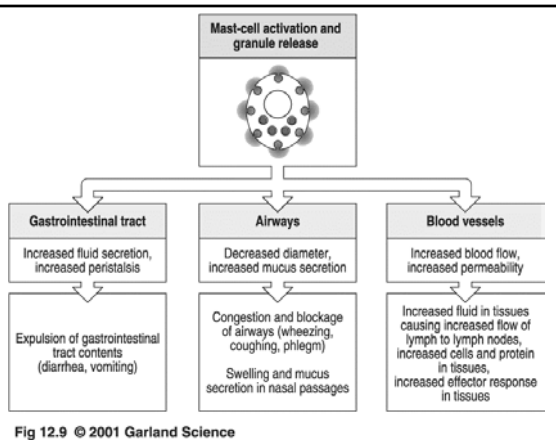


Fig 12.9 © 2001 Garland Science

Class of product	Examples	Biological effects
Enzyme	Tryptase, chymase, cathepsin G, carboxypeptidase	Remodel connective tissue matrix
Toxic mediator	Histamine, heparin	Toxic to parasites Increase vascular permeability Cause smooth muscle contraction
Cytokine	IL-4, IL-13	Stimulate and amplify T_H2 cell response
	IL-3, IL-5, GM-CSF	Promote eosinophil production and activation
	TNF-α (some stored preformed in granules)	Promotes inflammation, stimulates cytokine production by many cell types, activates endothelium
Chemokine	MIP-1α	Attracts monocytes, macrophages, and neutrophils
Lipid mediator	Leukotrienes C4, D4, E4	Cause smooth muscle contraction Increase vascular permeability Stimulate mucus secretion
	Platelet-activating factor	Attracts leukocytes Amplifies production of lipid mediators Activates neutrophils, eosinophils, and platelets

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Class of product	Examples	Biological effects
Enzyme	Tryptase, chymase, cathepsin G, carboxypeptidase	Remodel connective tissue matrix
Toxic mediator	Histamine, heparin	Toxic to parasites Increase vascular permeability Cause smooth muscle contraction
Cytokine	IL-4, IL-13	Stimulate and amplify T_H2 cell response
	IL-3, IL-5, GM-CSF	Promote eosinophil production and activation
	TNF-α (some stored preformed in granules)	Promotes inflammation, stimulates cytokine production by many cell types, activates endothelium
Chemokine	MIP-1α	Attracts monocytes, macrophages, and neutrophils
Lipid mediator	Leukotrienes C4, D4, E4	Cause smooth muscle contraction Increase vascular permeability Stimulate mucus secretion
	Platelet-activating factor	Attracts leukocytes Amplifies production of lipid mediators Activates neutrophils, eosinophils, and platelets

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