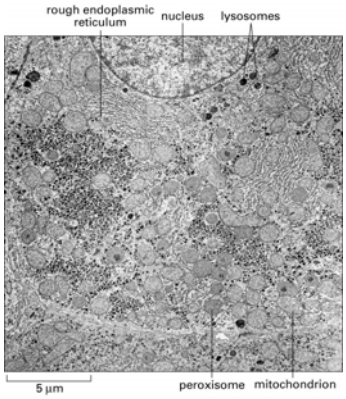


Figure 12–2. Molecular Biology of the Cell, 4th Edition.

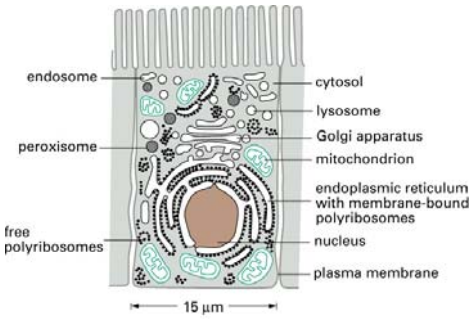
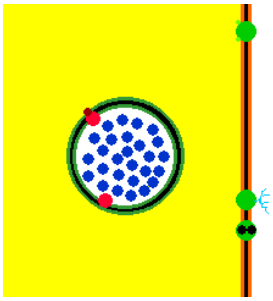


Figure 12–1. Molecular Biology of the Cell, 4th Edition.

Eksocytose



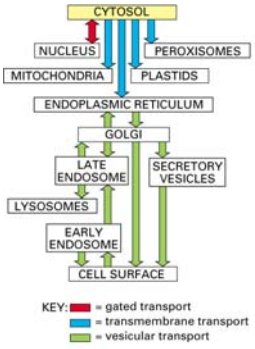


Figure 12–6. Molecular Biology of the Cell, 4th Edition.

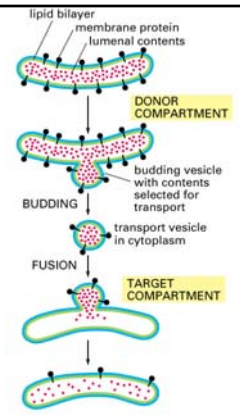


Figure 12–7. Molecular Biology of the Cell, 4th Edition.

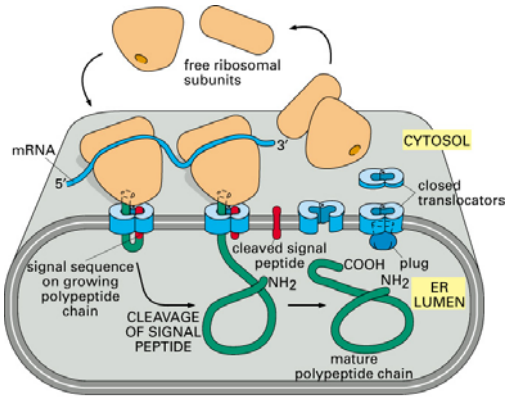


Figure 12–40. Molecular Biology of the Cell, 4th Edition.

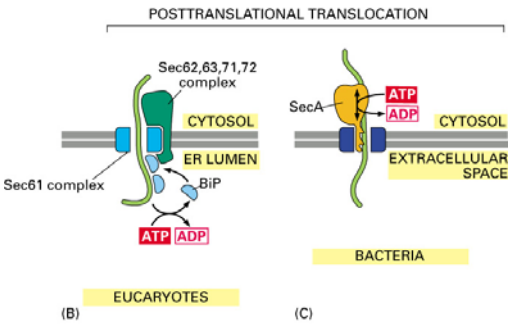


Figure 12–45 part 2 of 2. Molecular Biology of the Cell, 4th Edition.

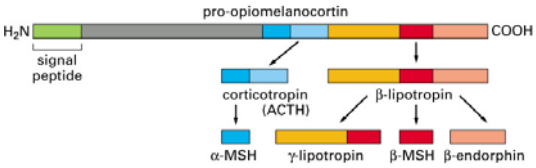


Figure 13–58. Molecular Biology of the Cell, 4th Edition.

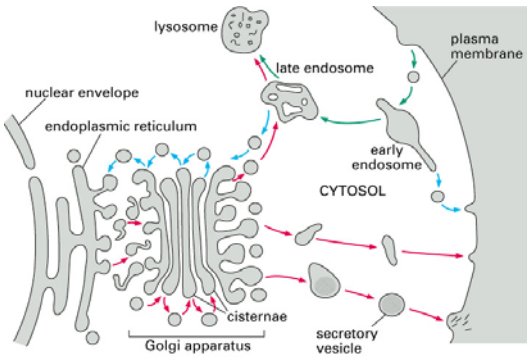
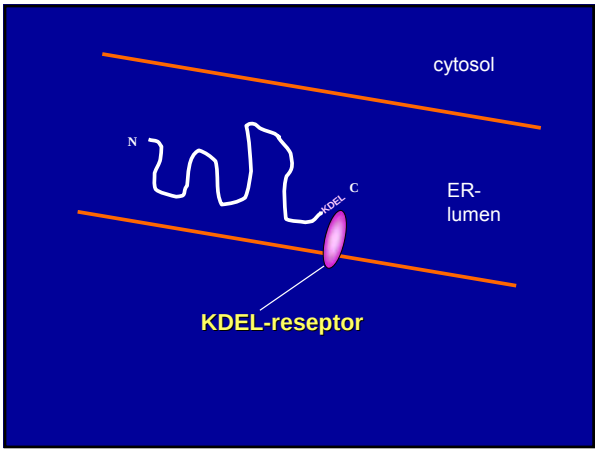


Figure 13–3. Molecular Biology of the Cell, 4th Edition.

Retensjon i ER-lumen

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



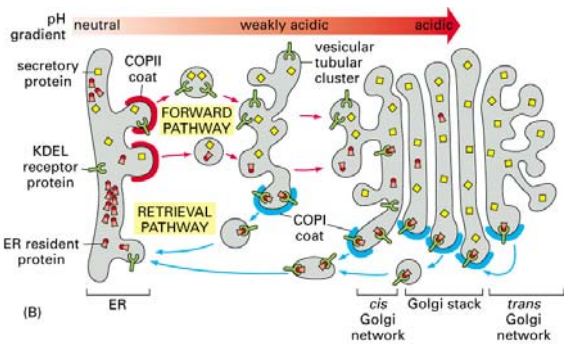
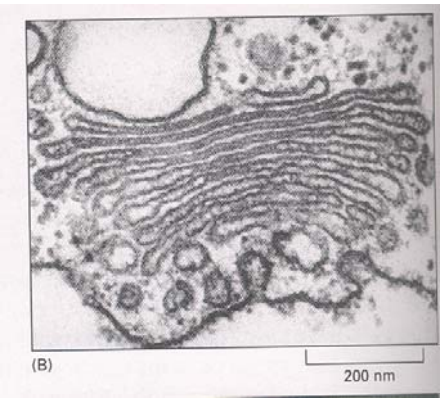
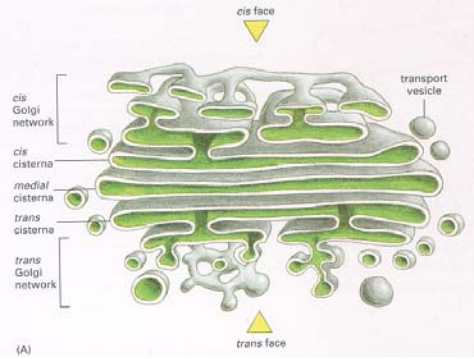


Figure 13–21 part 2 of 2. Molecular Biology of the Cell, 4th Edition.





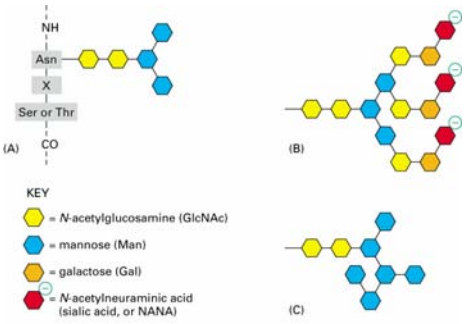
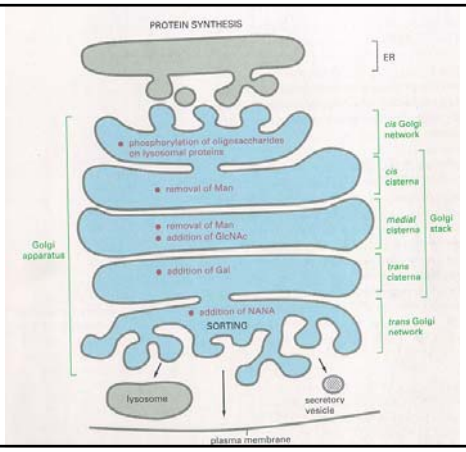


Figure 13–25. Molecular Biology of the Cell, 4th Edition.



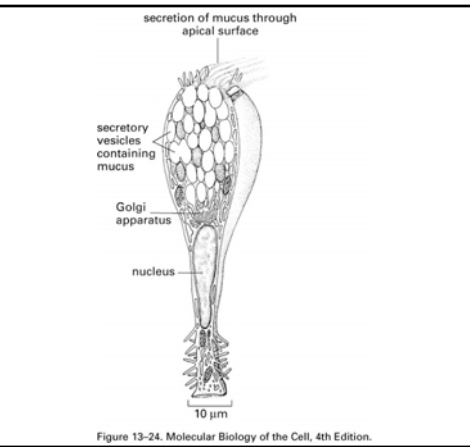


Figure 13–24. Molecular Biology of the Cell, 4th Edition.

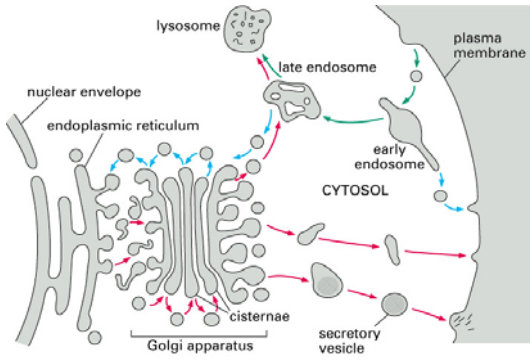
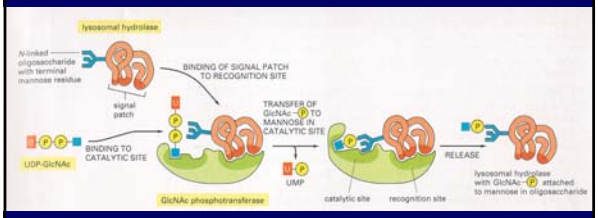


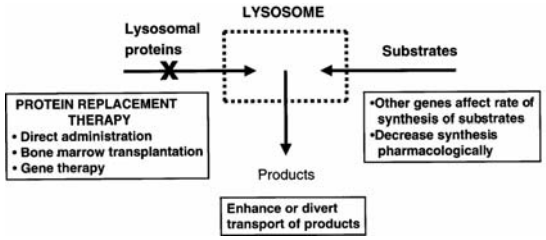
Figure 13–3. Molecular Biology of the Cell, 4th Edition.

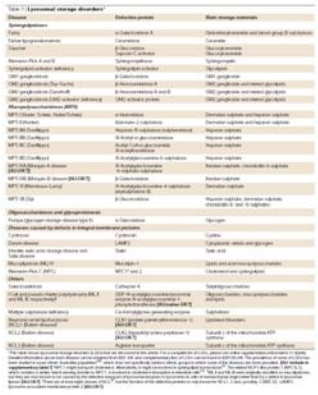
”Merking” av lysosomale enzymer med mannose-6-fosfat

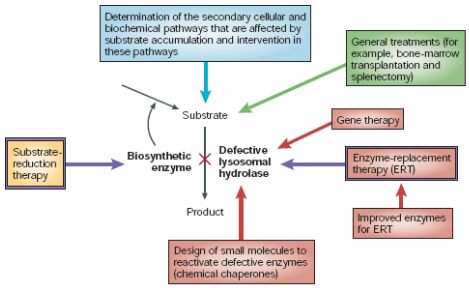
1. Trinn: Konjugering med N-acetylglukosaminfosfat



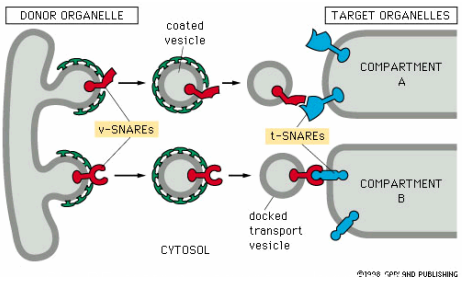
Over 40 different lysosomal storage diseases have been described in humans , with an overall prevalence of approximately 1 in 7000–8000 live births . Diagnosis is based on clinical symptoms followed by demonstration of a deficiency of a specific enzyme activity or transporter.

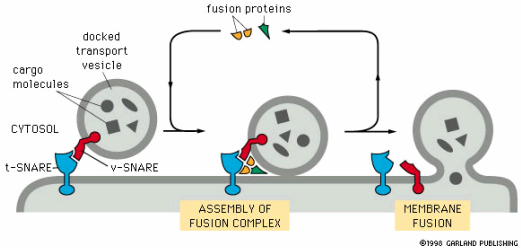






How do transport vesicles find their right target?





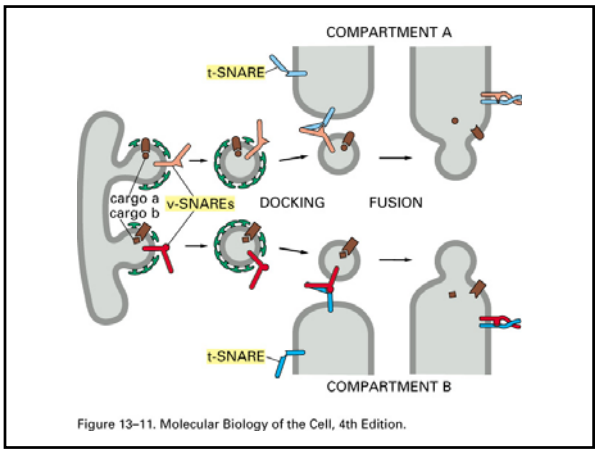


Figure 13–11. Molecular Biology of the Cell, 4th Edition.

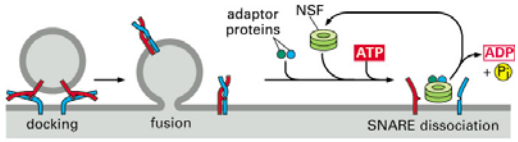


Figure 13–13. Molecular Biology of the Cell, 4th Edition.

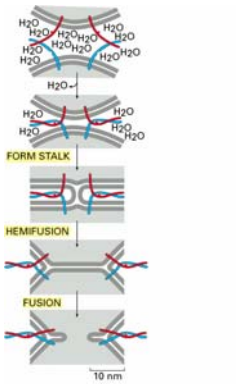


Figure 13–15. Molecular Biology of the Cell, 4th Edition.

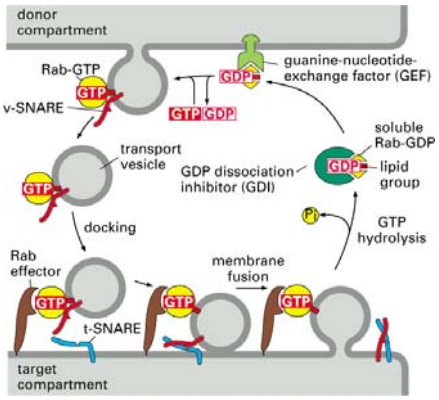


Figure 13–14. Molecular Biology of the Cell, 4th Edition.

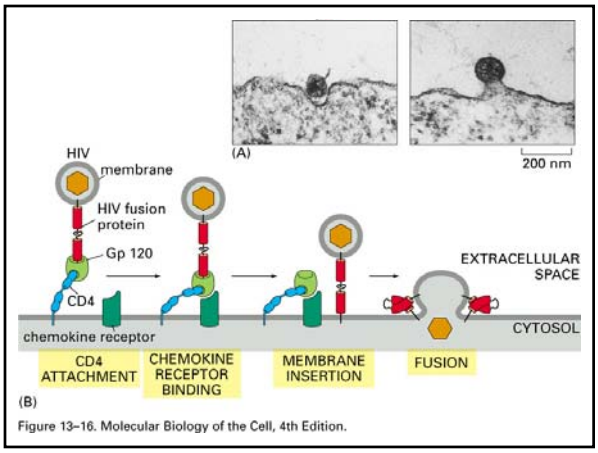
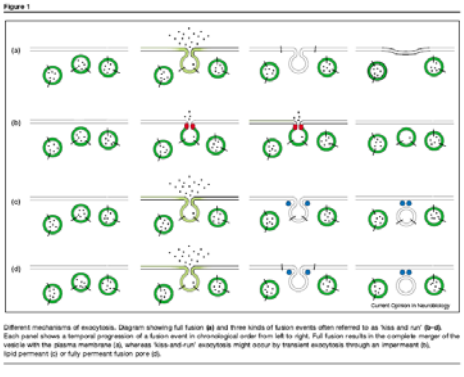
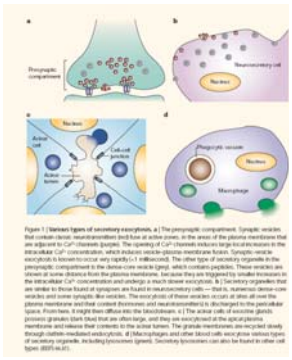


Figure 13–16. Molecular Biology of the Cell, 4th Edition.



Different mechanisms of exocytosis. Diagram showing full fusion (a) and three kinds of fusion events often referred to as 'kiss and run' (b–d). Each panel shows a temporal progression of a fusion event in chronological order from left to right. Full fusion results in the complete merger of the vesicle with the plasma membrane (a), whereas 'kiss-and-run' exocytosis might occur by transient exocytosis through an impermeant (b), lipid permeant (c) or fully permeant fusion pore (d).



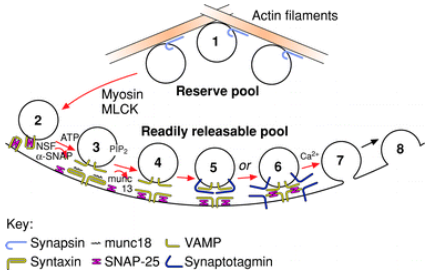
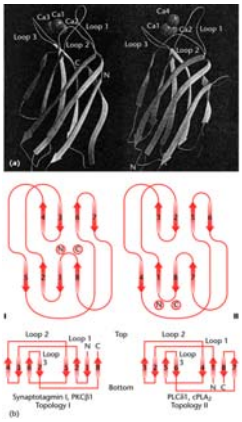
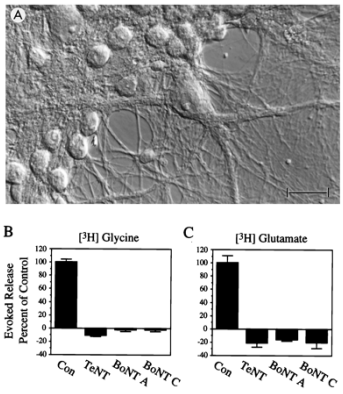


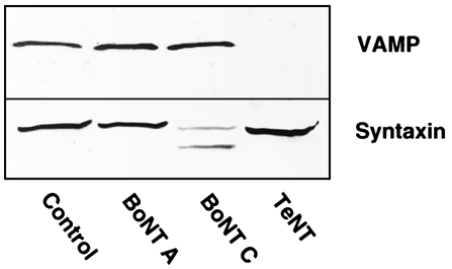
Figure 1 Molecular mechanisms underlying synaptic vesicle exocytosis. A reserve pool of synaptic vesicles (1), linked to actin by synapsin, is recruited to docking sites at the active zone by vesicle detachment followed by movement via myosin motors. Following vesicle docking at the presynaptic active 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).

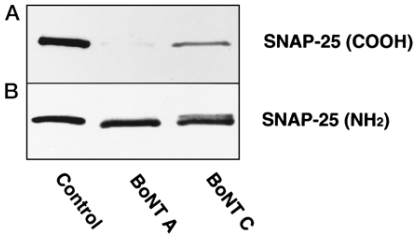
Synaptotagmin is a
Ca sensor

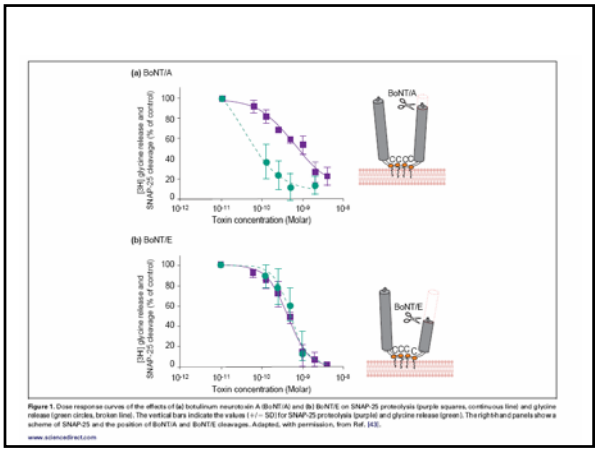




Botulinum and Tetanus toxins cleave SNARE's







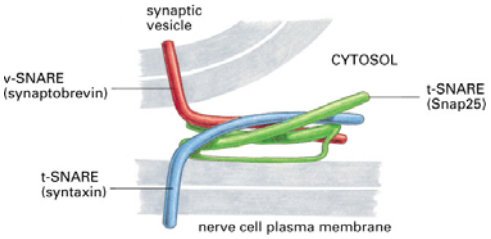
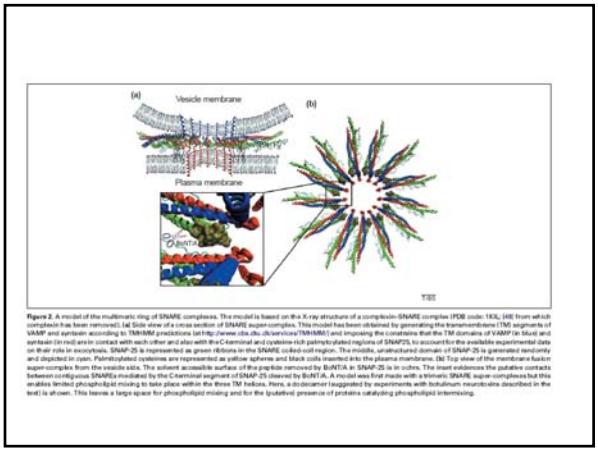
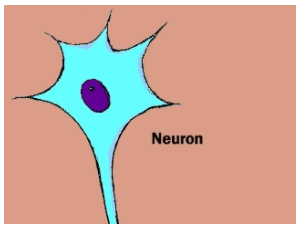


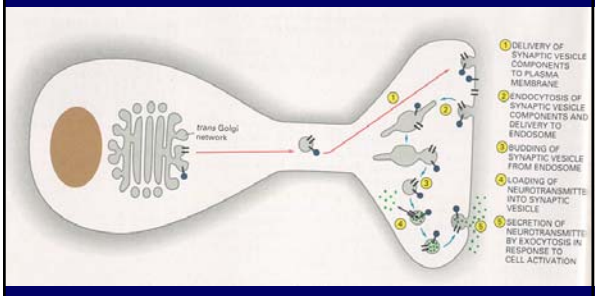
Figure 13–12. Molecular Biology of the Cell, 4th Edition.

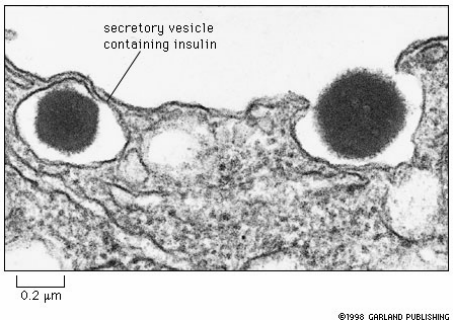


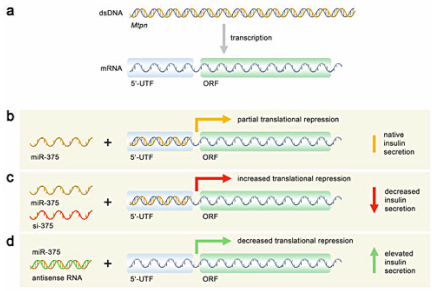
Regulert eksocytose



Eksocytose av synaptiske vesikler

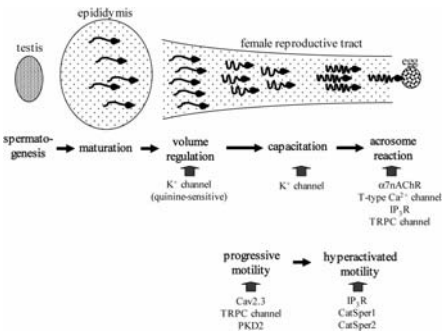






Panel 1. Translational repression of myotrophin by miR-375. - (a) The dsDNA gene for myotrophin (*Mtpn*) and its transcribed mRNA, showing both the 5'-unstranlated region (UTF) and open reading frame (ORF). (b) In the wild type setting, the single-stranded miR-375 microRNA anneals to a portion of the 5'-UTF and represses downstream translation. (c) Upon the addition of small interfering (si) RNAs (siRNA), specifically ones (si-375) that are sequence analogues to miR-375, the effective concentration of miR-375 increases and anneals to more *Mtpn* 5'-UTFs, further repressing Mtpn expression. However, when an antisense RNA (antisense to miR-375) is added, it anneals to miR-375 and prevents it from annealing to the 5'-UTR, thereby enhancing *Mtpn* expression. Mtpn is a component of insulin exocytosis.

Inhibitors of the acrosomal reaction may serve as male contraceptives



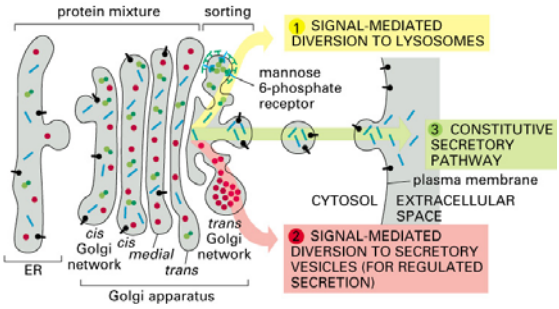


Figure 13–55. Molecular Biology of the Cell, 4th Edition.

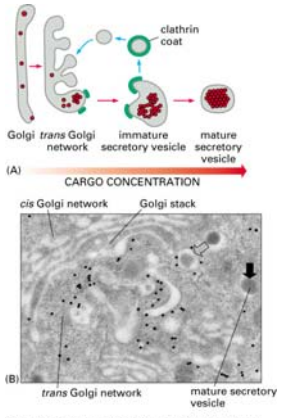


Figure 13–56. Molecular Biology of the Cell, 4th Edition.

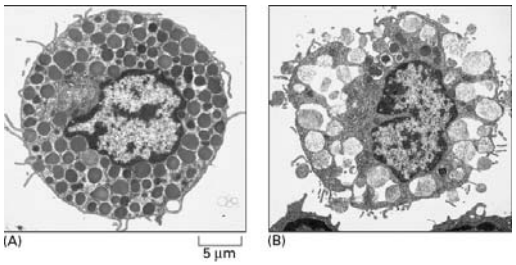


Figure 13–59. Molecular Biology of the Cell, 4th Edition.

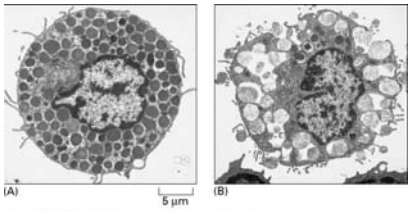


Figure 13–59. Molecular Biology of the Cell, 4th Edition.

Parameter	MC	Neuron
Granule type	Secretory lysosome	Specialized secretory granule
Granule size	300–1000 nm	50 nm
Granule number	≤1000 per cell	200–500 per synaptic terminal
Calcium requirement	~1 μM	>20 μM
PKC requirement	PKCβ and/or PKCδ	No
Kinetics	Minutes	Milliseconds
Recycling (granule)	Long (hours to days)	Very short (minutes)
Release (type)	One single release of ≤100% (compound or multigranular mode)	Multiple release, one granule per fusion event

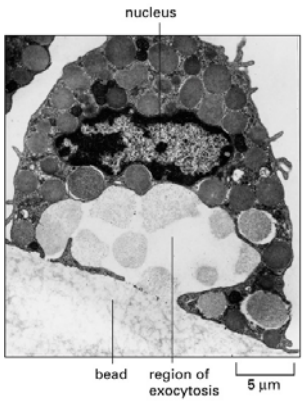


Figure 13–60. Molecular Biology of the Cell, 4th Edition.

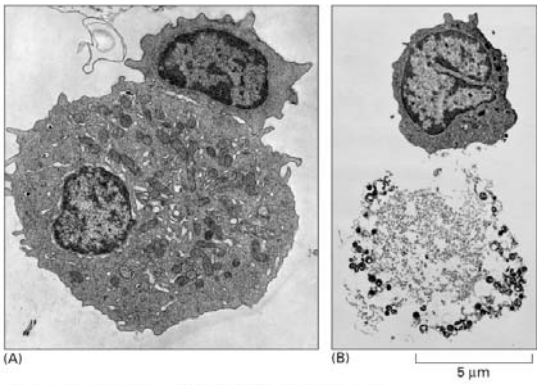


Figure 24–45 part 1 of 2. Molecular Biology of the Cell, 4th Edition.