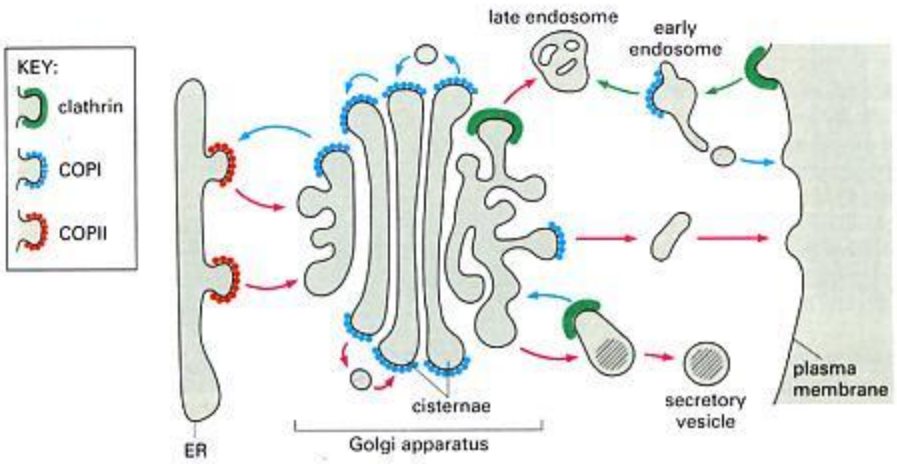
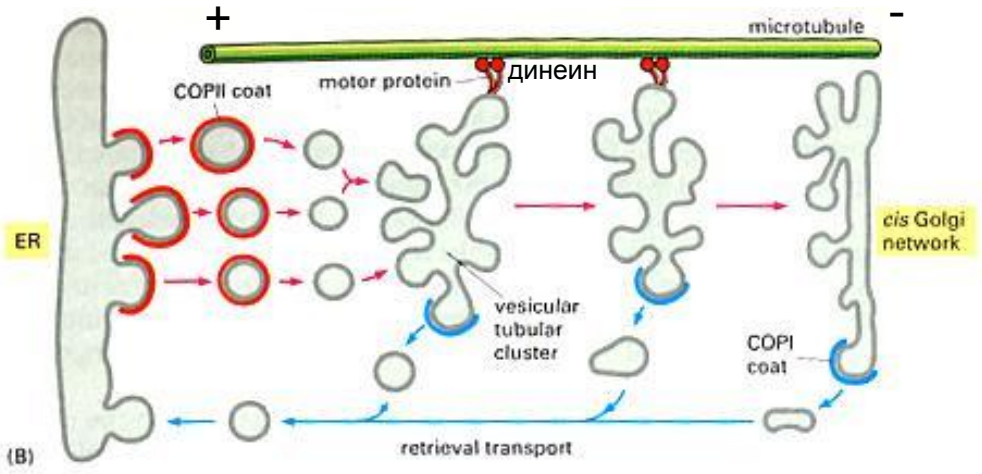
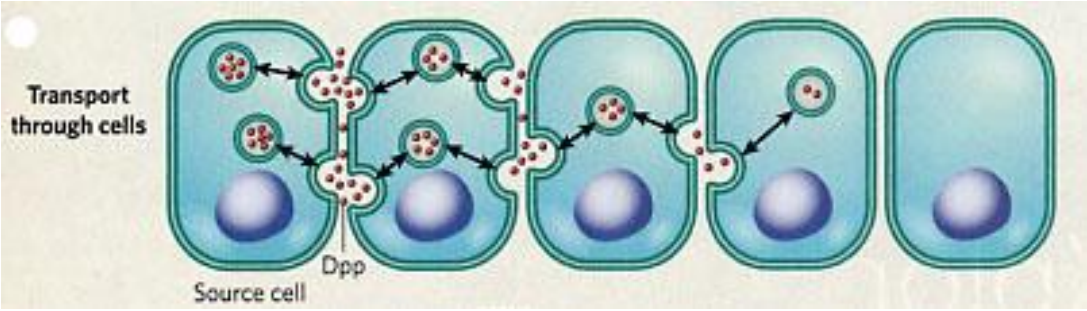
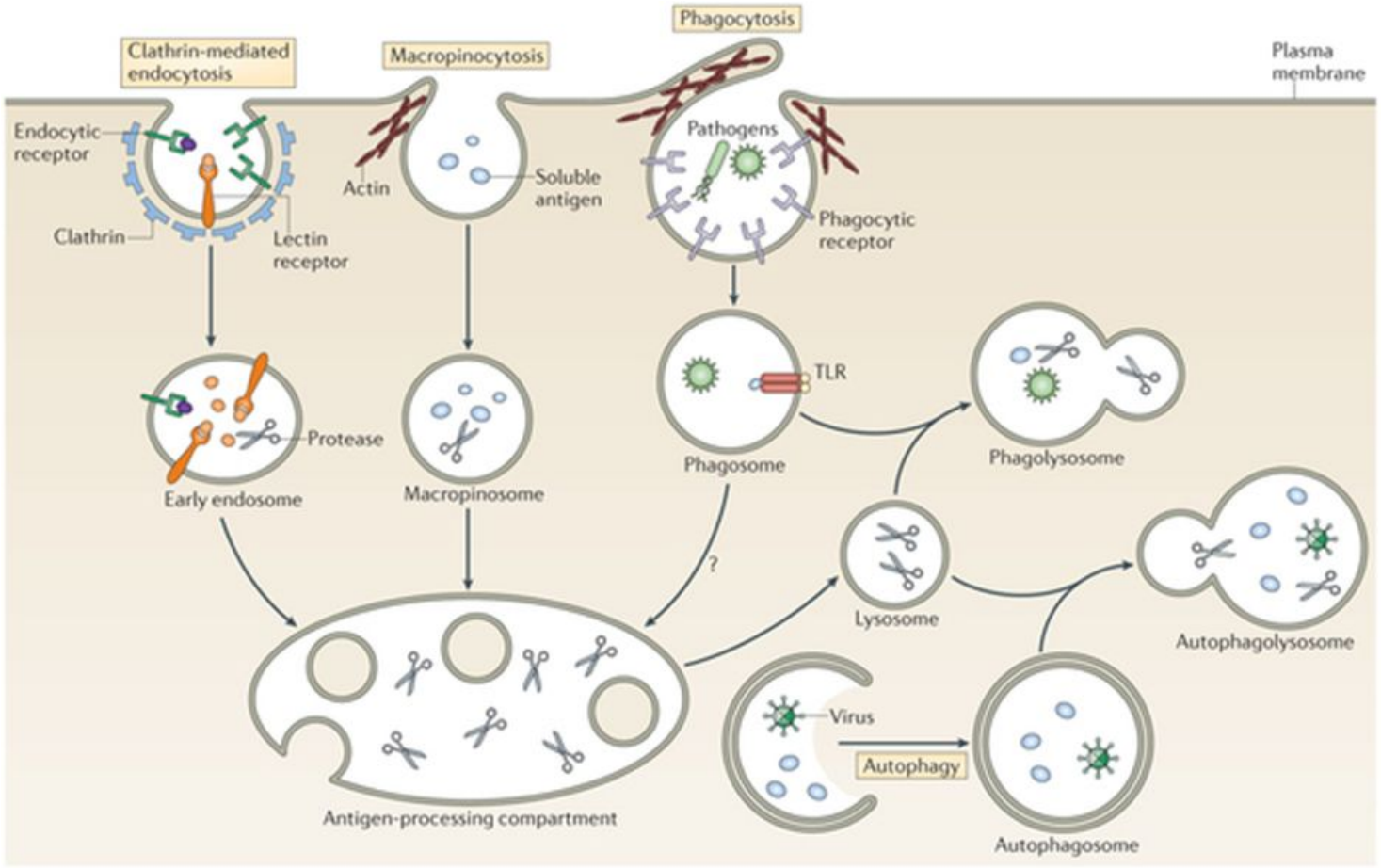

KEY:
clathrin
COPI
COPII
late endosome
early endosome
plasma membrane
cisternae
secretory vesicle
ER
Golgi apparatus


+
-
microtubule
динеин
motor protein
COPII coat
ER
cis Golgi network
vesicular tubular cluster
COPI coat
retrieval transport
(B)

ТРАНСЦИТОЗ Dpp (decapentaplegic, аналог TGF-β)





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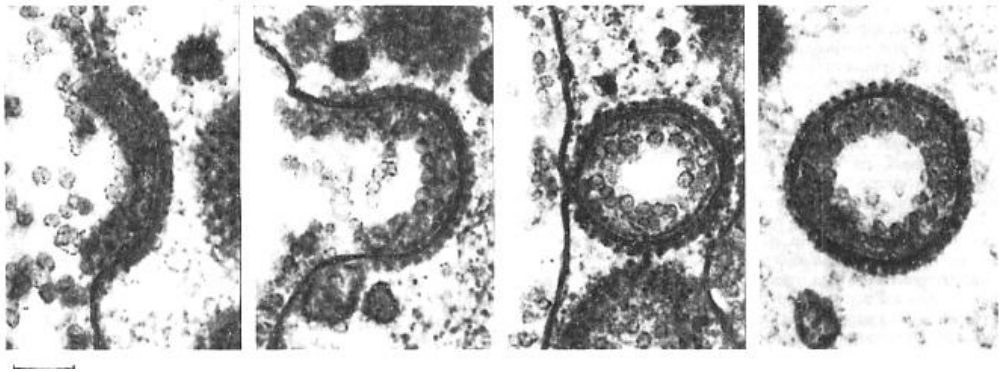
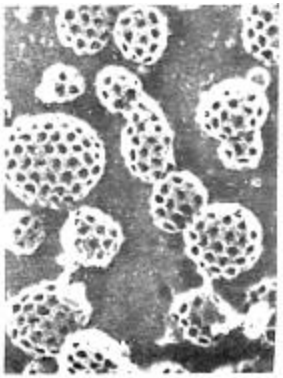
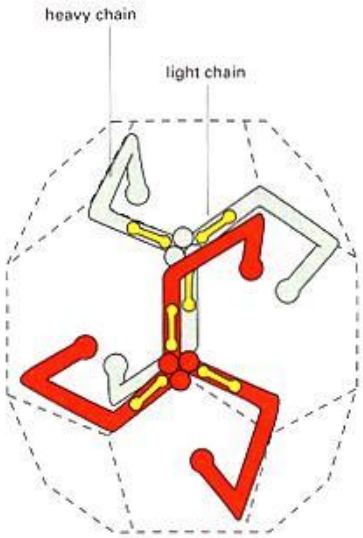
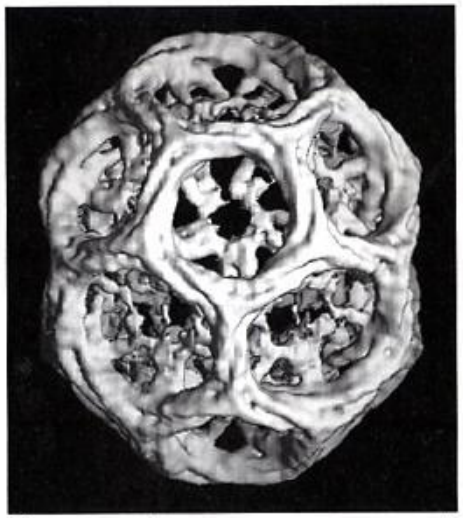






(B)



(C)

50 nm

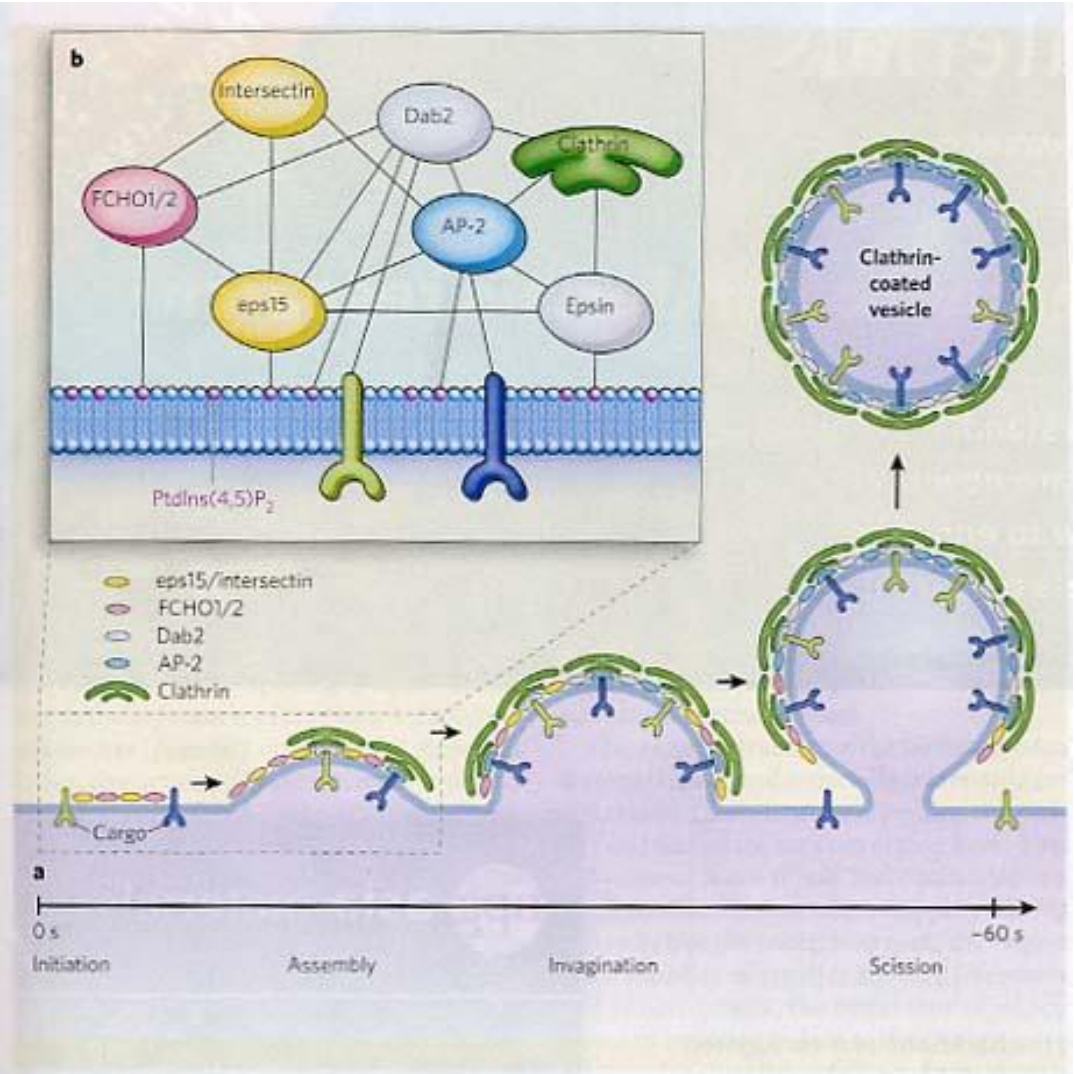


Figure 1 | Clathrin-coat assembly. **a**, Henne *et al.*[1] show that clathrin-coat formation at the cell surface begins as small assemblages of pioneer proteins called FCHO1 and FCHO2; recruitment of the endocytic proteins eps15 and intersectin probably coincide with this event. The region destined to become a bud then further expands laterally through the addition of components arriving later, such as AP-2, Dab2 and clathrin. Within a minute, it gently curves, invaginates deeply and constricts at the base. Finally, scission releases the coated vesicle. **b**, The initiation protein complex centres on core components with multiple common but weak and/or transient interactions (lines). Many of the early-arriving proteins bind to the lipid PtdIns(4,5)P_2. These proteins require AP-2 and Dab2 to link with clathrin and numerous classes of cargo.

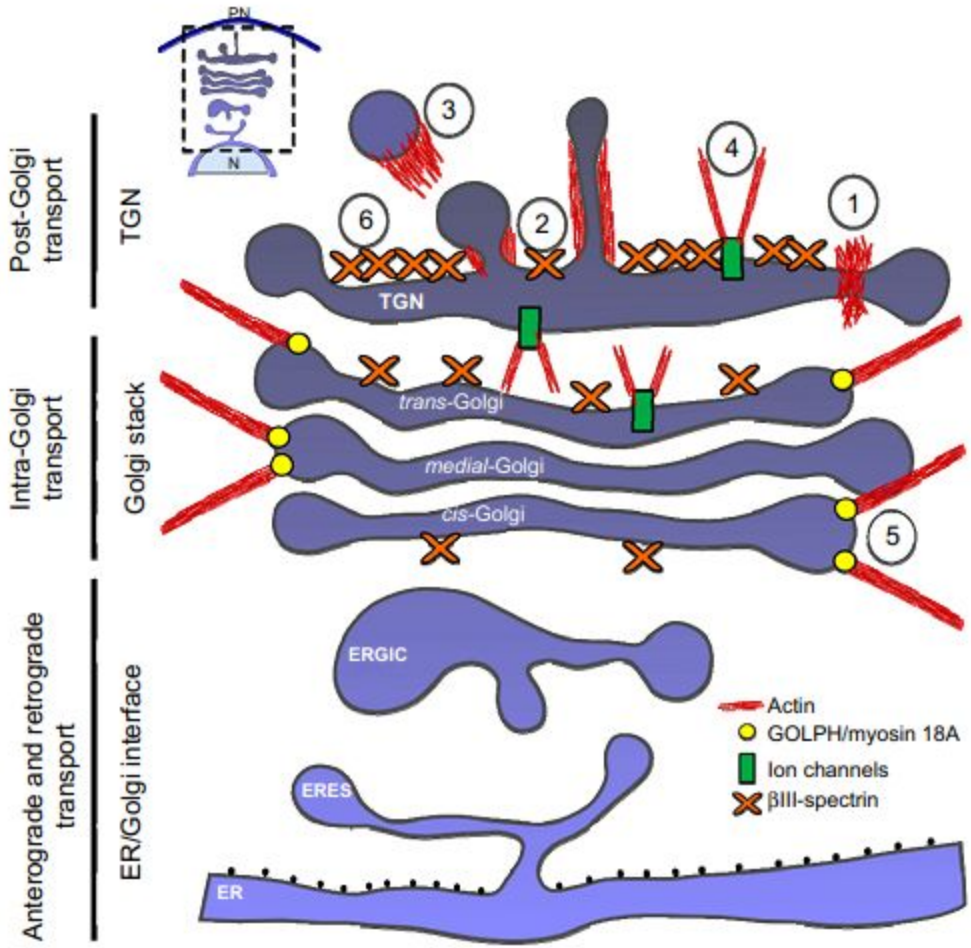


Figure 3 Actin–Golgi interaction II.

Notes: Diagram of the secretory membrane trafficking pathways and events in which actin is known to participate. Actin filaments, their polymerization, and dynamics could act as a force for the scission (1), pulling (2), and propelling (3) of the transport carrier generated in cisternae, and for maintaining the flattened shape of cisternae through the regulation of the activity on some ion pumps/channels (4) and/or being part of the spectrin-based cytoskeleton (6), and to keep the Golgi ribbon extended (5). Modified from Springer, *Histochemistry and Cell Biology*. Actin acting at the Golgi. 2013;140(3):347–360. Egea G, Serra-Peinado C, Salcedo-Sicilia L, Gutiérrez-Martínez E. Copyright © Springer-Verlag Berlin Heidelberg 2013, with kind permission of Springer Science+Business Media.[24]

Abbreviations: TGN, *trans*-Golgi network; ERGIC, endoplasmic reticulum–Golgi intermediate compartment; ERES, ER exiting sites.

СХЕМА ЭКЗОЦИТОЗНОГО ТРАНСПОРТА ВЕЗИКУЛ

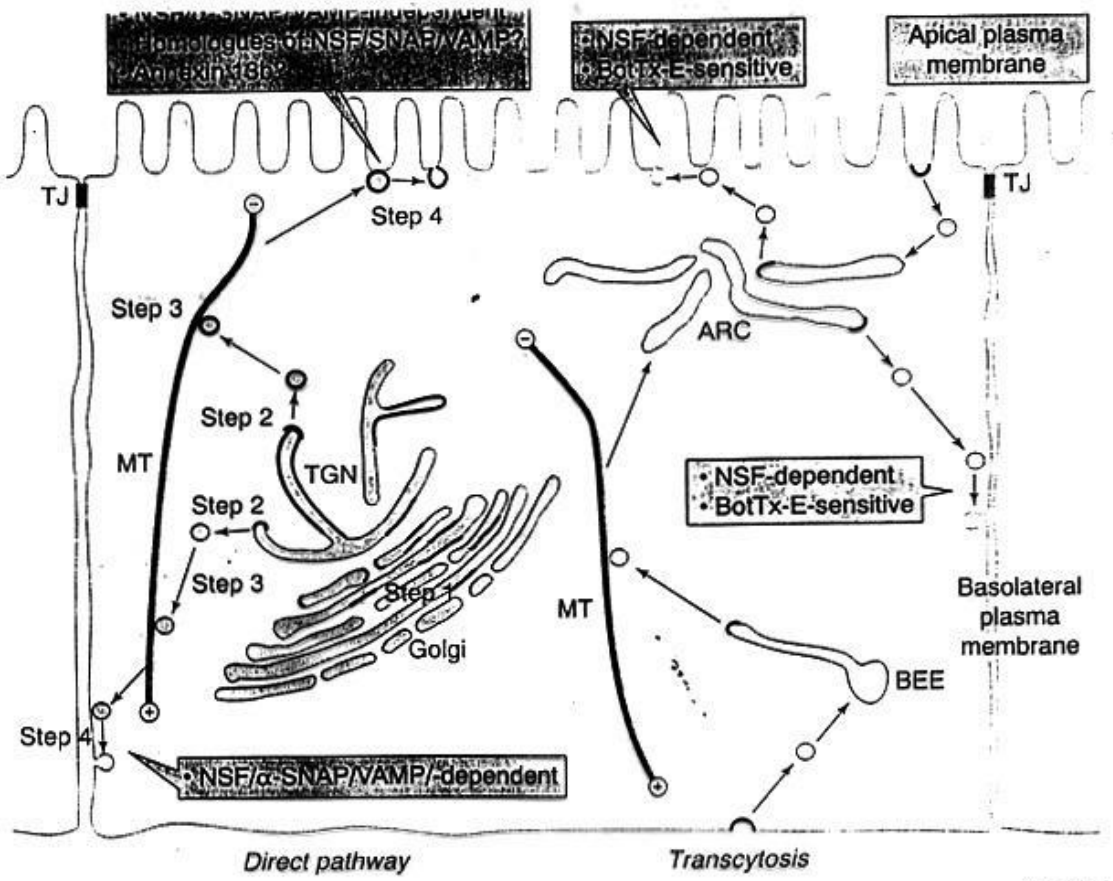
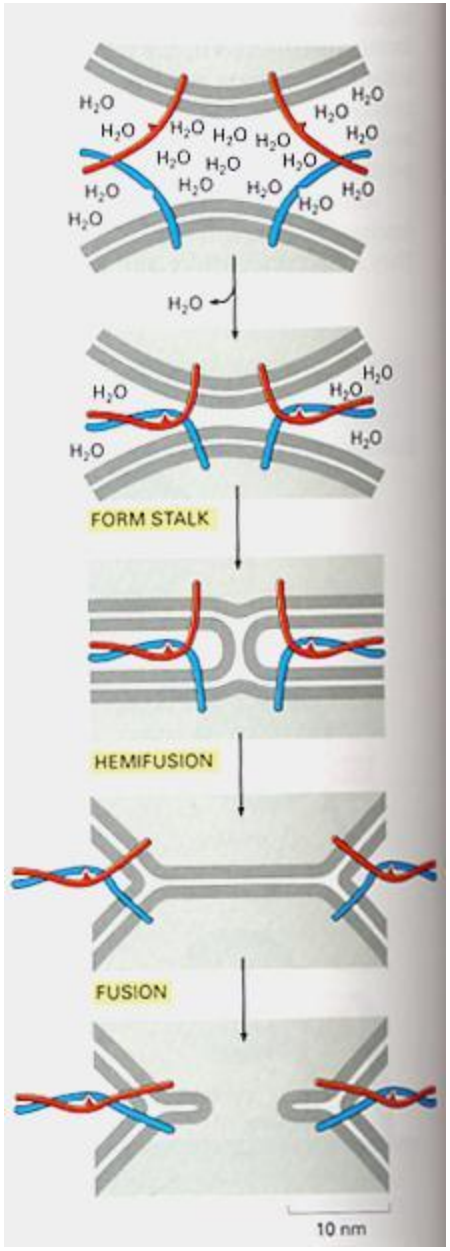
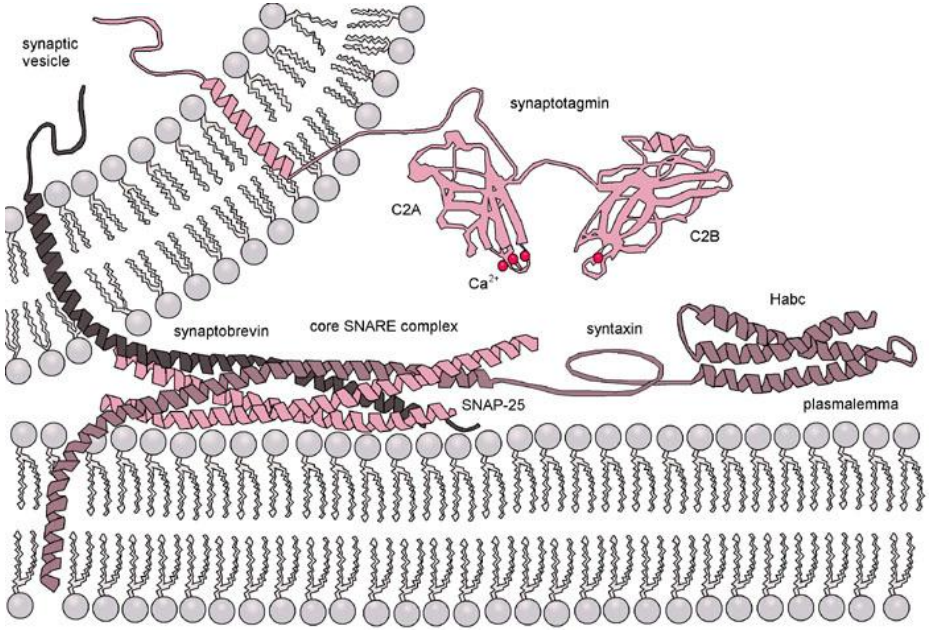


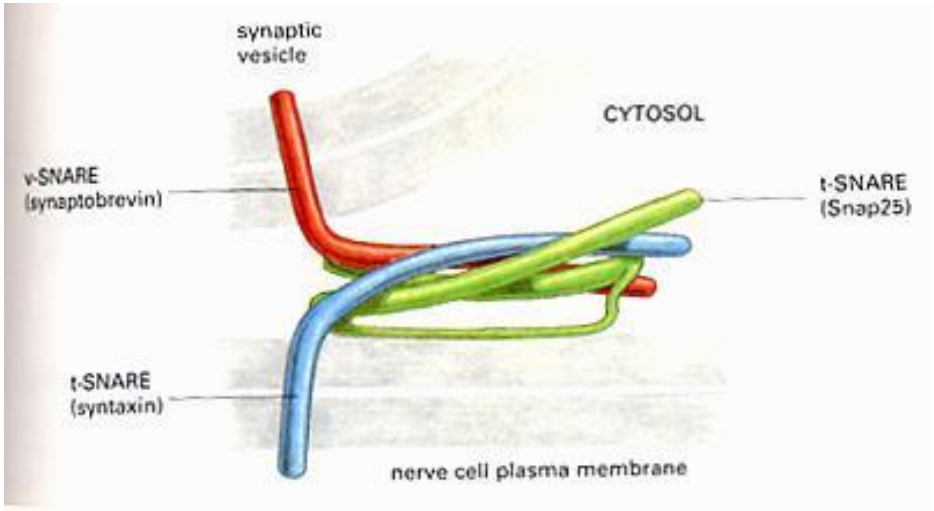
FIGURE 1

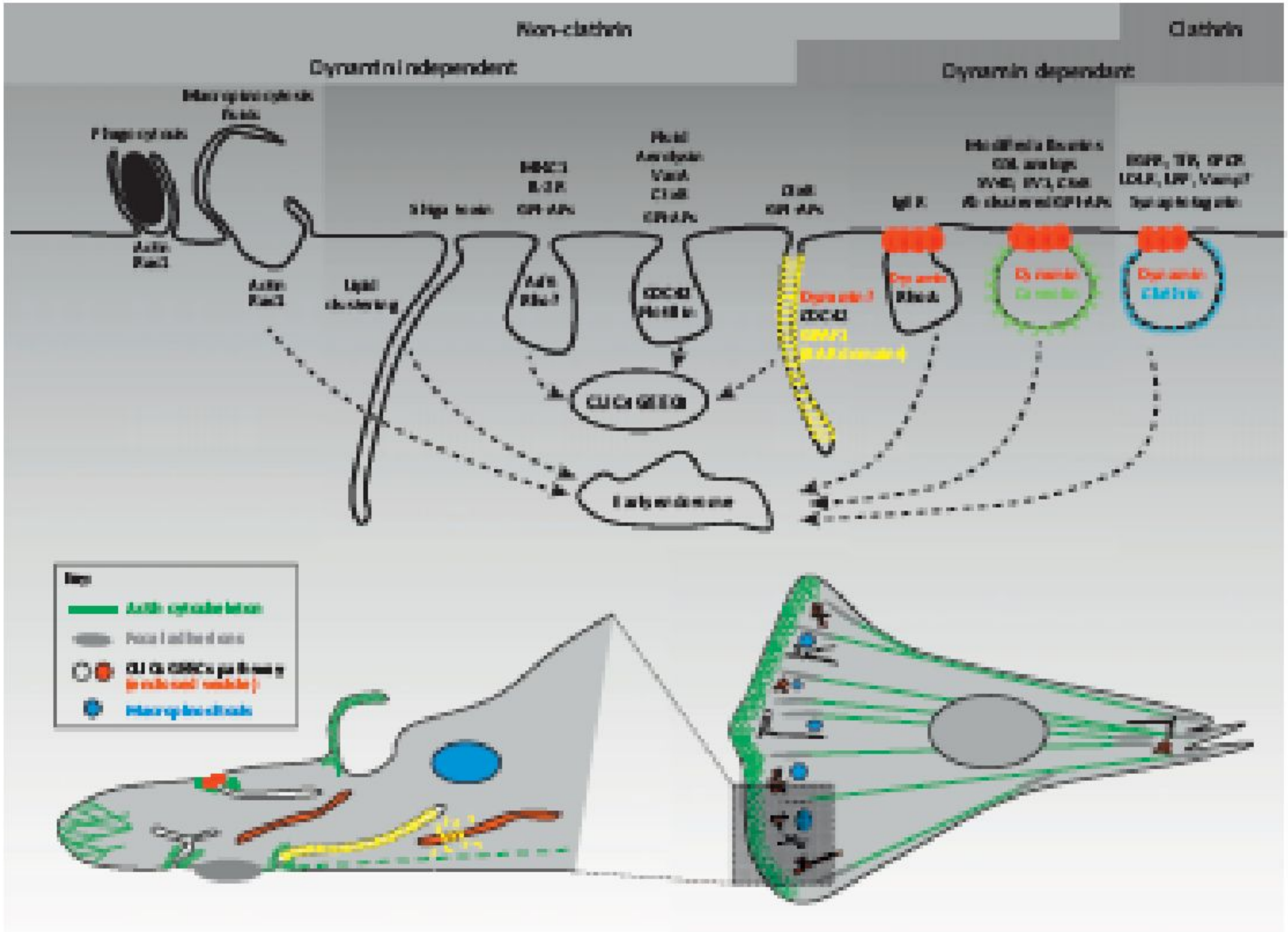
Summary of the vesicular membrane-trafficking pathways in a typical epithelial cell (e.g. MDCK). Epithelial cells possess two plasma membrane domains, apical and basolateral, separated by tight junctions (TJs). Two principal pathways exist for the targeting of plasma membrane proteins: in the 'direct' pathway, proteins are sorted in the Golgi apparatus, possibly by clustering into or exclusion from glycosphingolipid-rich membrane microdomains (rafts, step 1). Transport vesicles destined for the apical and basolateral membranes bud from the *trans* Golgi network (TGN), in a process probably mediated by coat proteins (step 2). Vesicles are transported directionally along microtubules (MTs) or other cytoskeletal elements using vesicle-associated motors (step 3). After reaching the plasma membrane, vesicles dock and fuse utilizing the SNARE machinery at the basolateral and possibly also at the apical surface (step 4, see text for explanations). In the 'indirect' pathway, newly synthesized membrane proteins are first transported from the TGN to the basolateral surface and are then endocytosed into basolateral early endosomes (BEE). From here, apical proteins are transported along microtubules to the tubovesicular 'apical recycling compartment' (ARC), which also receives proteins internalized from the apical surface. The final transport step to the apical plasma membrane involves the SNARE machinery since it is NSF-dependent and sensitive to botulinum toxin E (BotTx-E), which cleaves certain t-SNAREs[58].

АДРЕСАЦИЯ ЭКЗОЦИТОЗНЫХ ВЕЗИКУЛ С ПОМОЩЬЮ БЕЛКОВ SNARE









TRENDS in Cell Biology

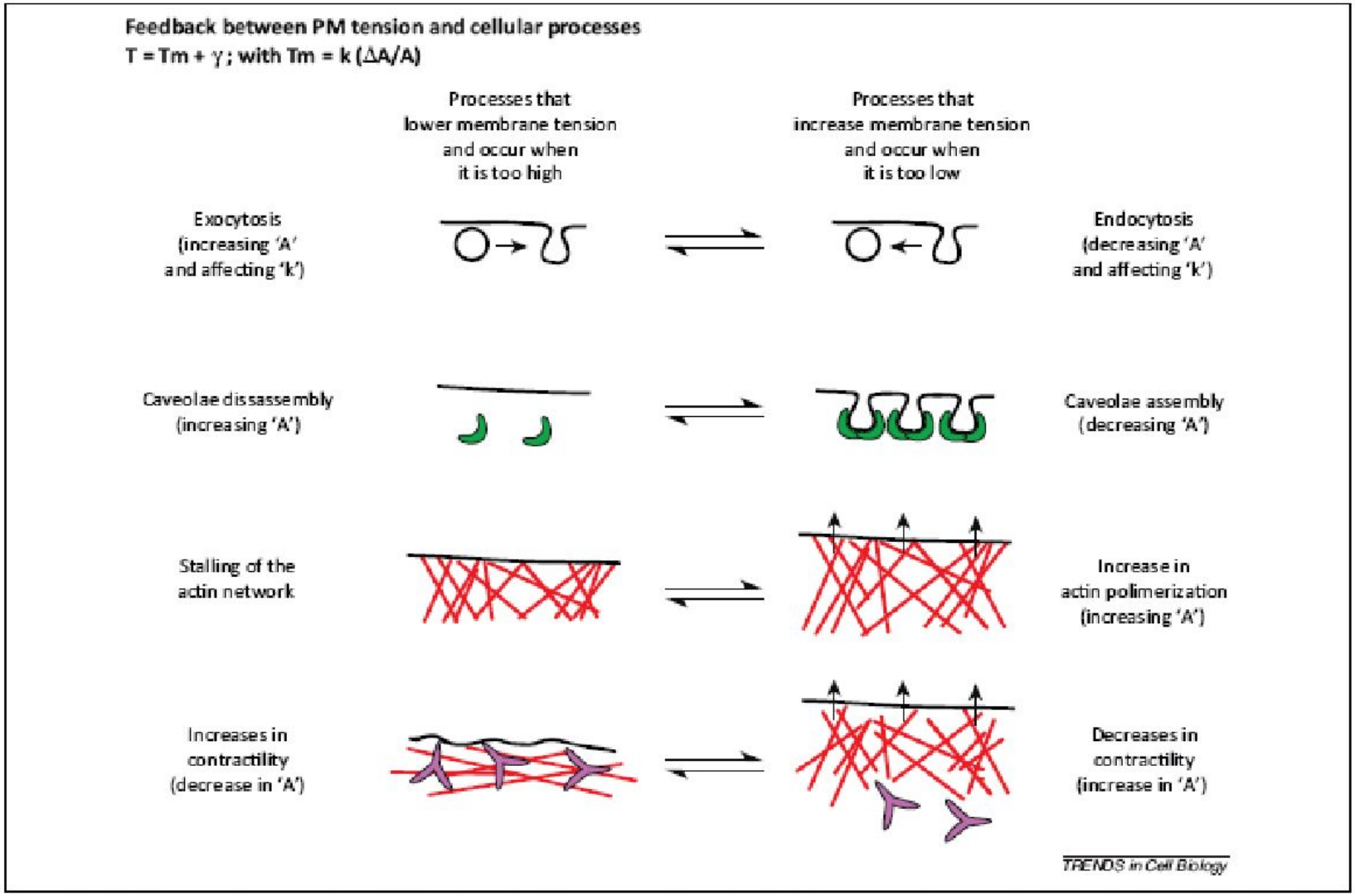
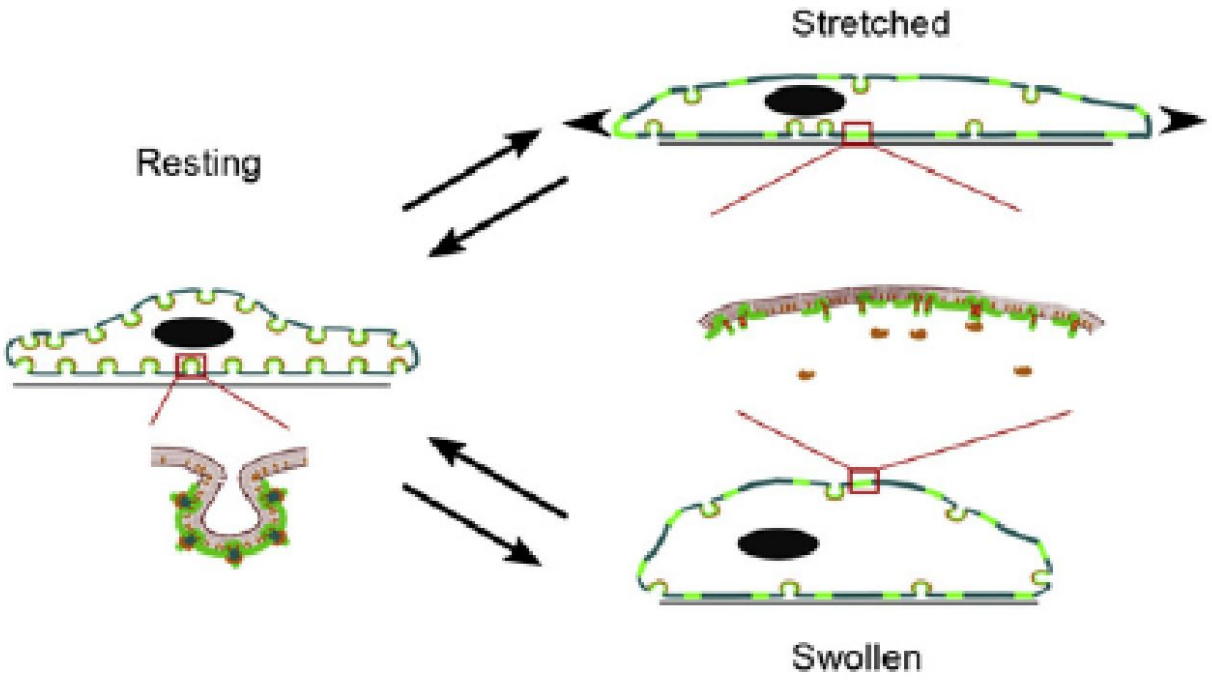


Figure 1. Feedback between plasma membrane (PM) tension and cellular processes. Examples of cellular processes that occur when PM tension is too high and that lead to its reduction (left) or that occur when PM tension is too low and lead to its increase (right) – vesicle trafficking, caveola formation, actin polymerization, and changes in myosin. In brackets we comment on the parameters of Equations [I] and [II] in Box 2 that are predicted to change in each of these processes.

РЕГУЛЯЦИЯ НАТЯЖЕНИЯ КЛЕТОЧНОЙ МЕМБРАНЫ ЧЕРЕЗ ПОСРЕДСТВО ВСТРАИВАНИЯ – РАЗБОРКИ И САМОСБОРКИ КАВЕОЛ



Натяжение
в 2-3 раза
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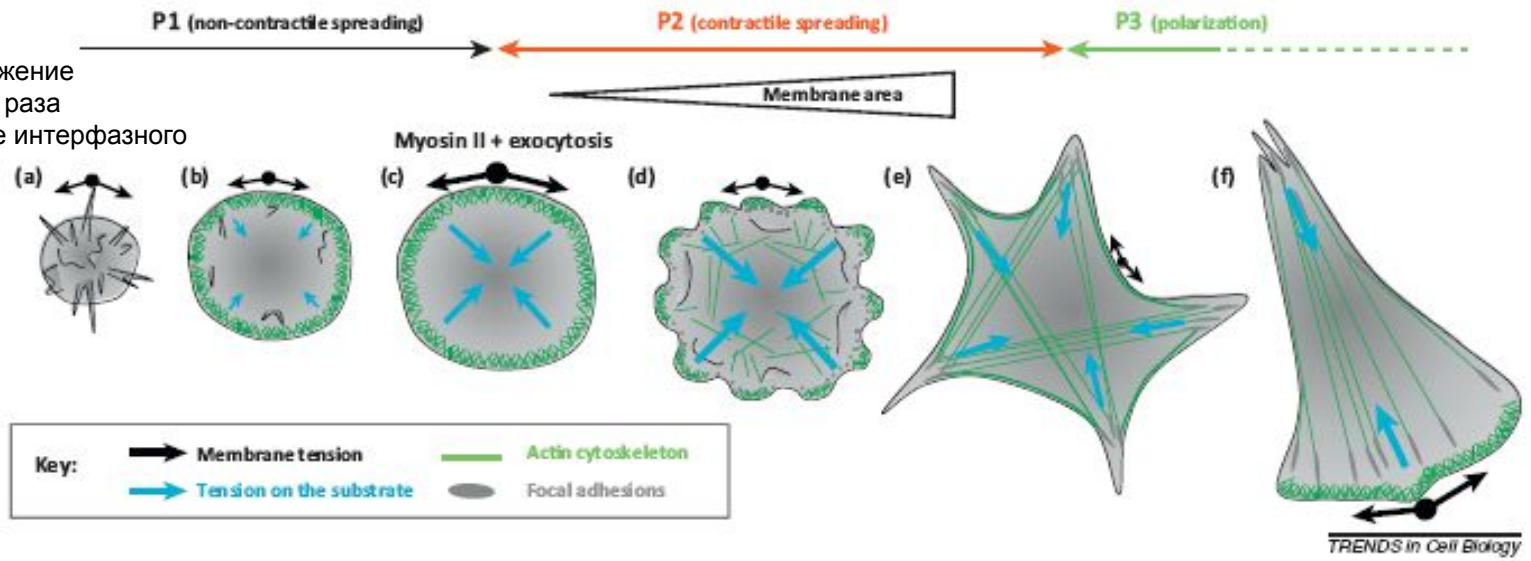

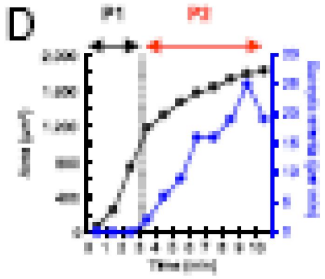

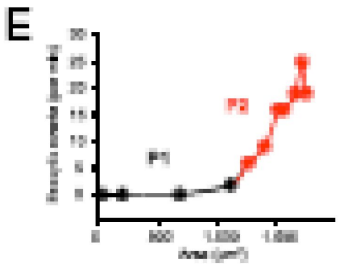

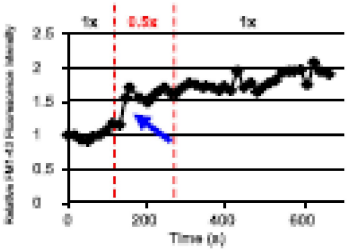

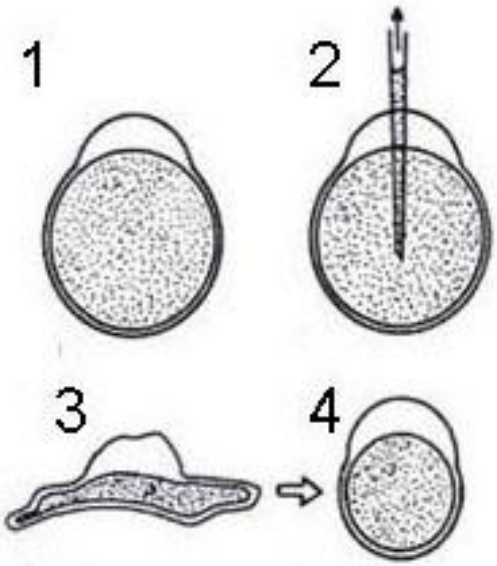
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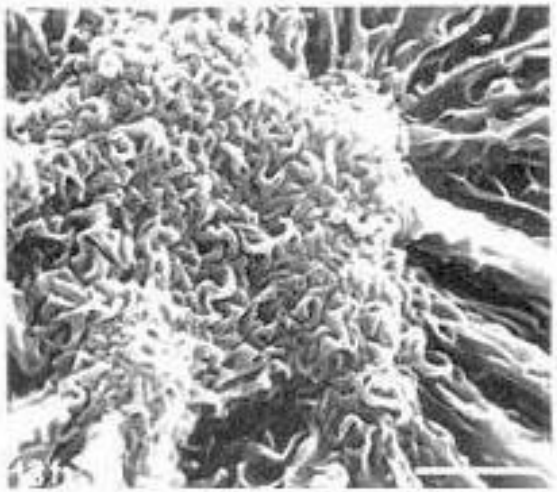
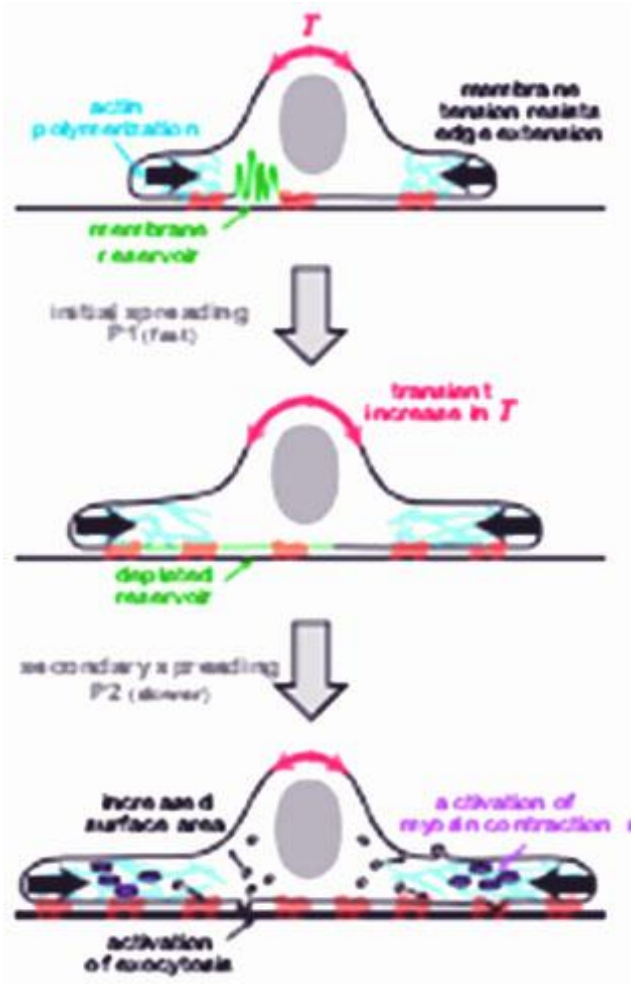



T
membrane tension resists edge extension
actin polymerization
membrane reservoir
initial spreading P1 (fast)
transient increase in T
depleted reservoir
secondary spreading P2 (slower)
increased surface area
activation of myosin contraction
activation of exocytosis

ДЛЯ ИНВАГИНАЦИИ МЕЗОДЕРМЫ У ЗАРОДЫШЕЙ ДРОЗОФИЛЫ НЕОБХОДИМО ПОДАВИТЬ ЭНДОЦИТОЗ МИОЗИНА II И СЕКРЕТИРУЕМОГО БЕЛКА FOG. У МУТАНТНОЙ РАСЫ ЭТО МОЖНО СДЕЛАТЬ МЕХАНИЧЕСКИМ РАСТЯЖЕНИЕМ ПОВЕРХНОСТИ В РЕЗУЛЬТАТЕ ТОЧЕЧНОГО ДАВЛЕНИЯ

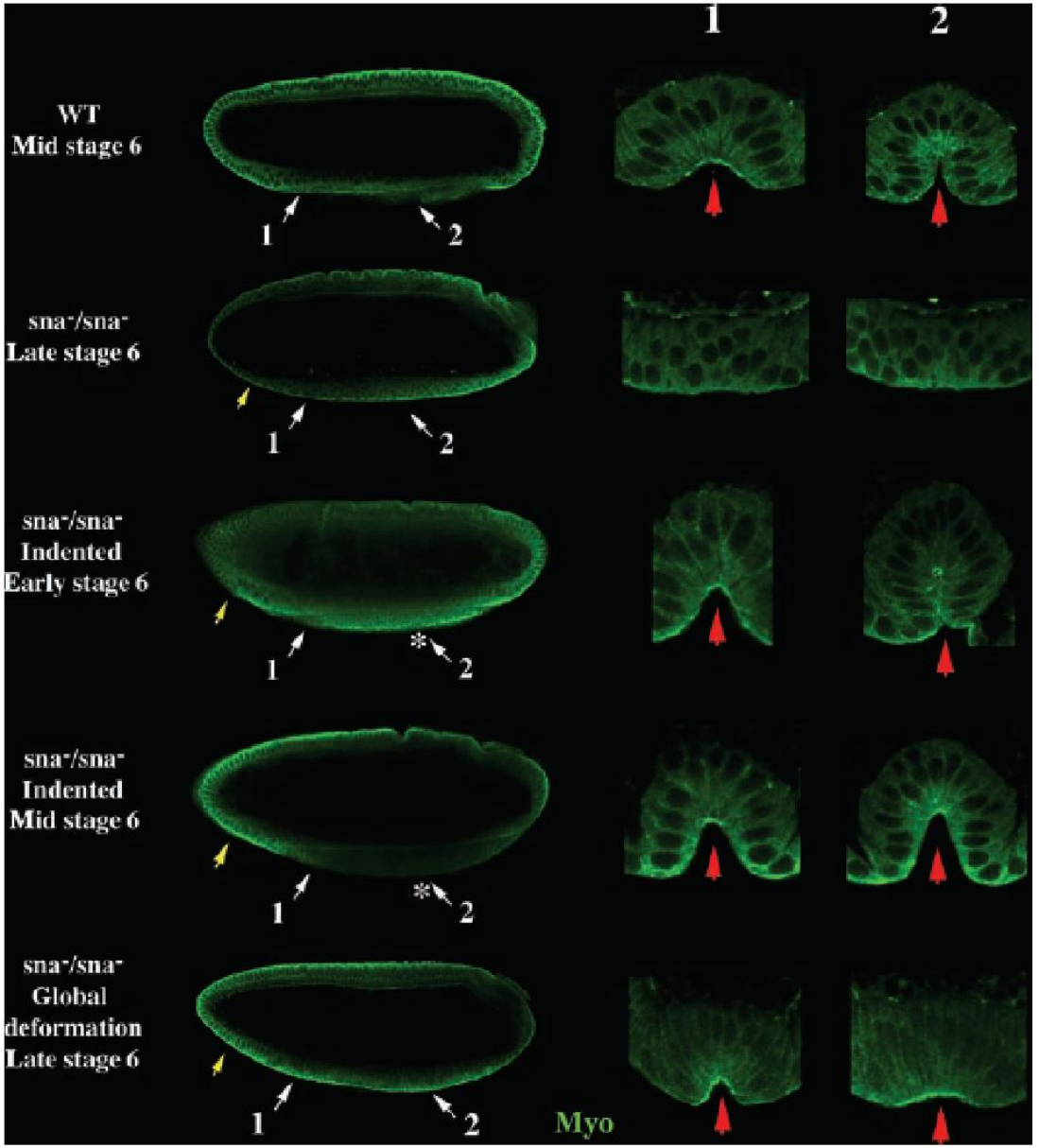
Дикий тип – нормальная инвагинация

Мутантная раса

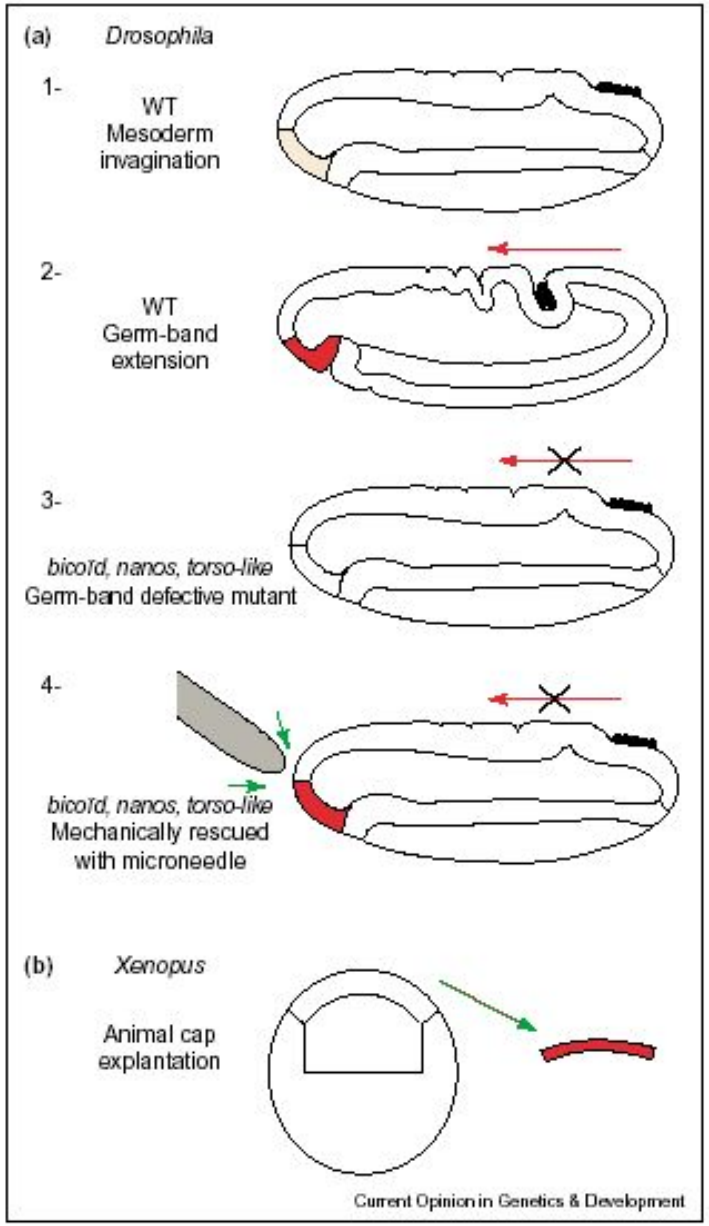
Мутантная раса – давление на ранней стадии

На средней стадии

«Глобальное» давление



МЕХАНИЧЕСКОЕ СЖАТИЕ СТИМУЛИРУЕТ ЭНДОЦИТОЗ β-КАТЕНИНА



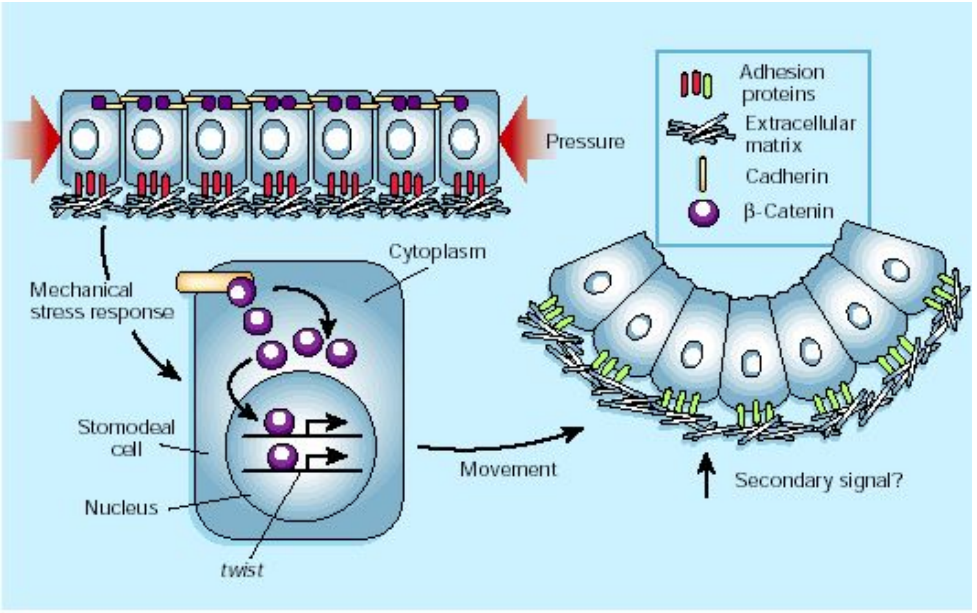
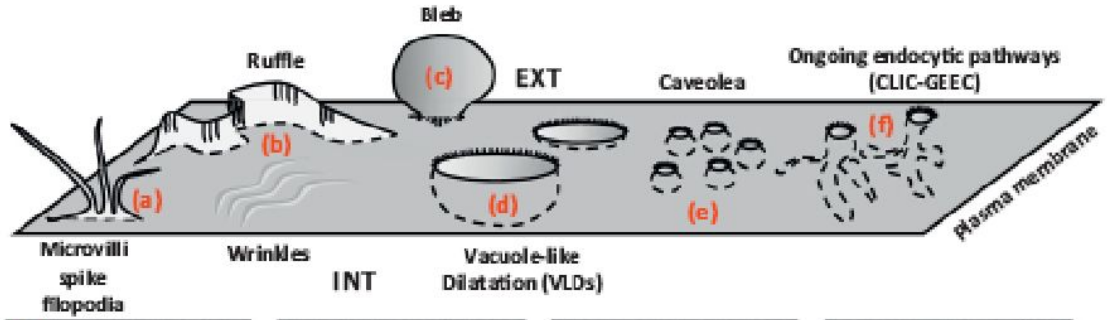
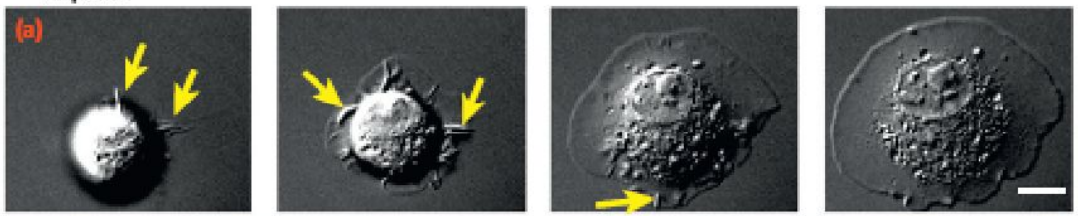
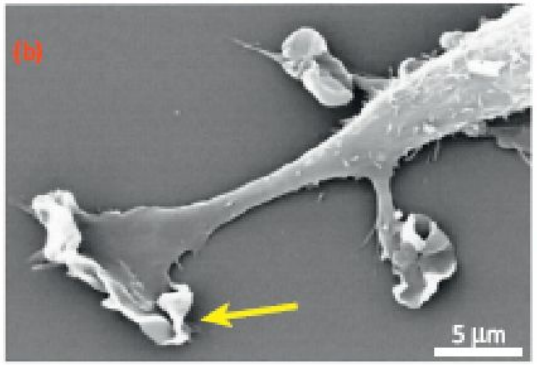
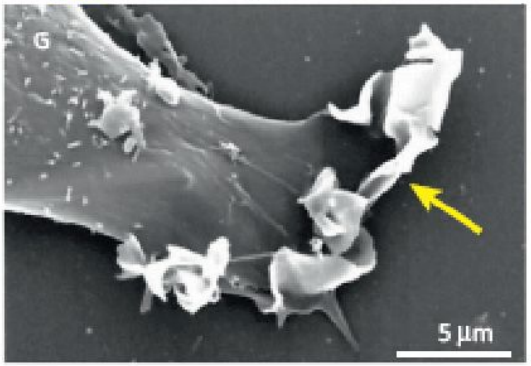


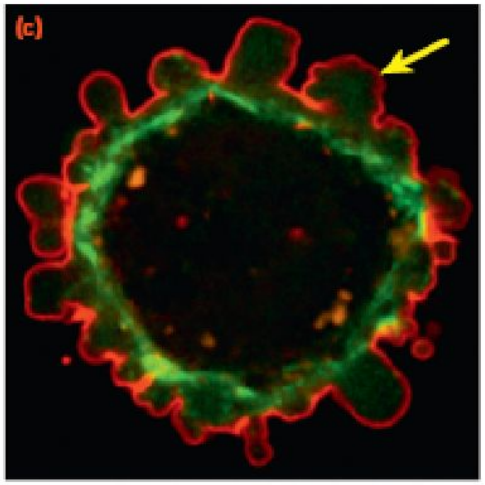
Figure 1 The mechanics of shaping the digestive tract. New work by Farge[1] shows that mechanical compression of stomodeal precursor cells (which will form the part of the digestive tract called the stomodeum) can affect the expression of the *twist* gene. Pressure causes the β-catenin protein to move from the cell membrane (where it associates with cadherin) to the cytoplasm, increasing its concentration there. This movement in turn allows β-catenin to accumulate in the nucleus, where it activates *twist* and probably other developmental genes. *twist* is required for the invagination and development of the stomodeum. Possible 'downstream' responses may include changes in the contacts made between cell adhesion molecules and the extracellular matrix, facilitating invagination. The reaction to mechanical stress might also be 'permissive', allowing the stomodeal cells to interpret a secondary signal originating from surrounding tissues.

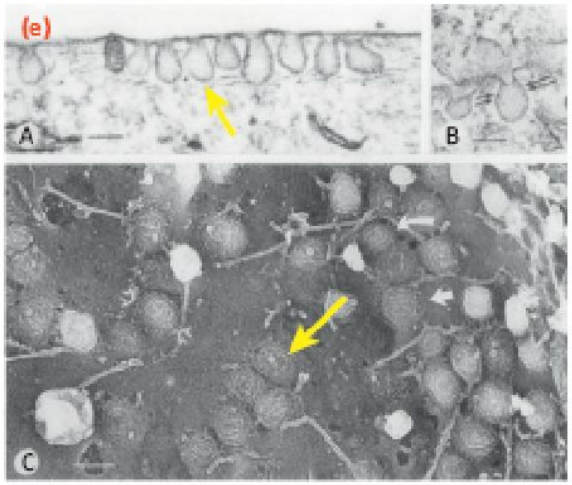

Bleb
Ruffle
(c)
EXT
Caveolea
Ongoing endocytic pathways
(CLIC-GEEC)
(f)
(b)
(a)
(d)
(e)
Plasma membrane
Microvilli
spike
filopodia
Wrinkles
INT
Vacuole-like
Dilatation (VLDs)


(a)


(b)
5 µm


G
5 µm


(c)


(e)
A
B
C

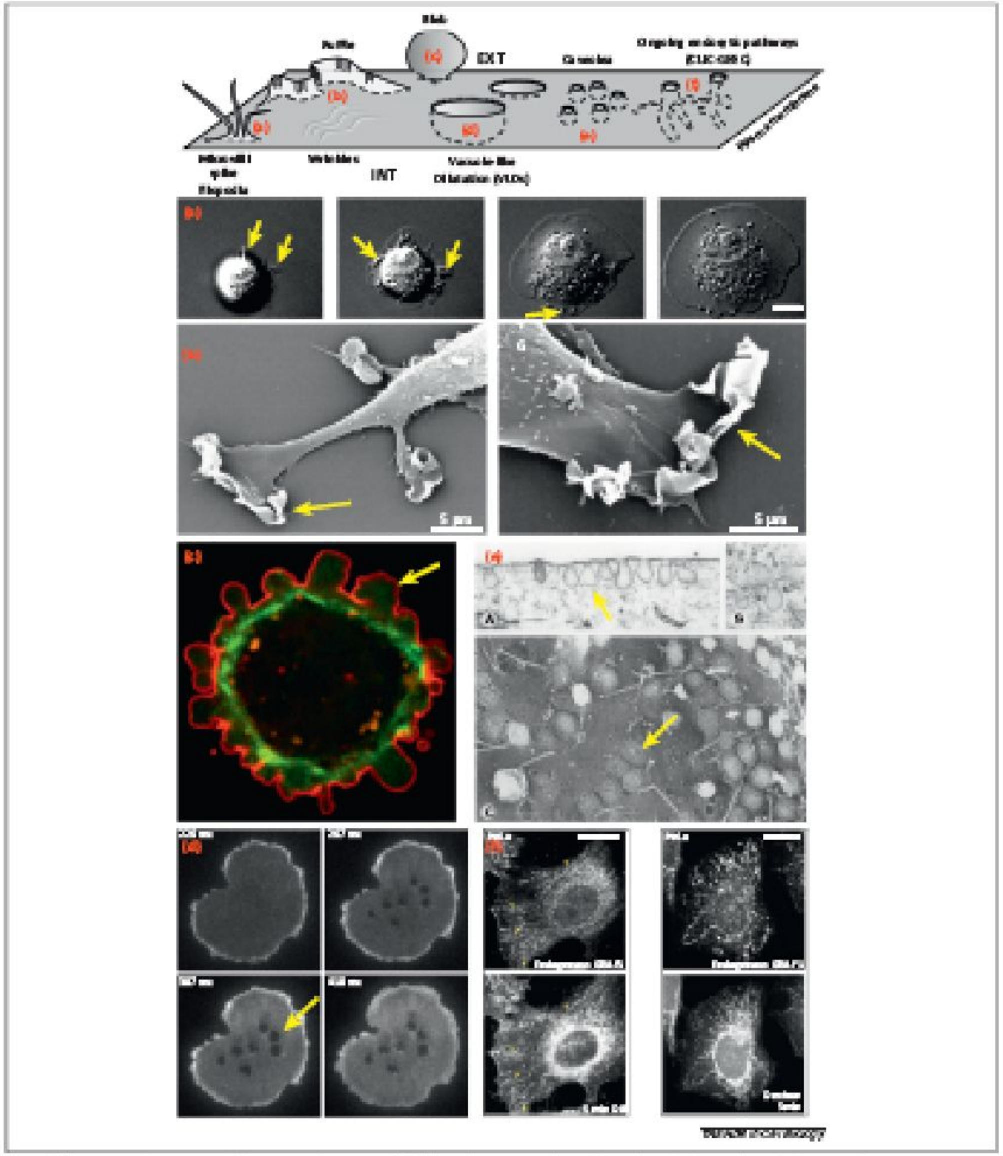


Figure 1. Plasma membrane area and plasma membrane reservoirs. Schematic representation and experimental observations of plasma membrane superstructures regulating membrane area and tension. (a) Microvilli and spikes, as observed during rapid cell spreading [illegible]. (b) Membrane ruffles: flat sheets of membrane sustained by the cytoskeleton with high surface to volume ratio. Scanning electron micrographs of a fibroblast stimulated with growth factor. The cell harbors large ruffles. Reproduced, with permission, from [illegible]. (c) Membrane blebs, as observed in dying cells or amoeboid migration. This confocal image shows myosin (green) recruited to fusiform membrane blebs (red). Reproduced, with permission, from [illegible]. (d) Vacuole-like dilatations, as observed forming on the basal membrane of spreading cells after isotonic stimulation following hypotonic stretch [illegible]. (e) Caveolae, as observed on thin section and rapid-freeze, deep-etch views of a fibroblast. Reproduced, with permission, from [illegible]. (f) The clathrin-independent carrier (CLIC) and glycosylphosphatidylinositol-anchored protein enriched in early endosomal compartments (GEEC) pathways, which represent high membrane area capacities, as observed with Binamphiphysin/Rvs (BAR) and Rho GTPase-activating protein (GAP) domain-containing protein GTPase regulator associated with focal adhesion kinase-1 (GRAF1). Reproduced, with permission, from [illegible].

ЭКЗОЦИТОЗ И ТРАНСЦИТОЗ ПРИ ПЕРЕДАЧЕ СИГНАЛОВ