ЭЛЕКТРИЧЕСКИЕ ПОЛЯ РАСТИТЕЛЬНЫХ ЗАЧАТКОВ

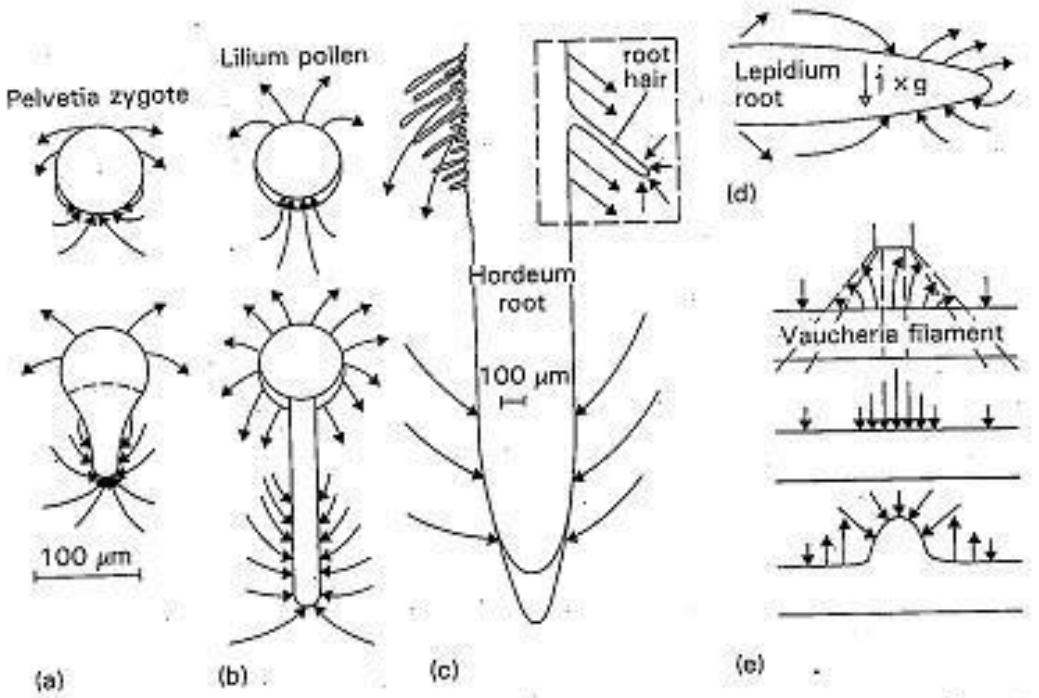


Figure 6.3 Natural ion currents generated by growing plant cells and organs. Current enters at the absorptive pole and exits proximal to the growing tip or at the photosynthetic pole. When roots are gravitropically stimulated by being turned on their sides (d), an outward current develops on the upper side of the tip within 3 min. (Weisenseel & Kicherer 1981; Behrens *et al.* 1982)

ЭЛЕКТРОФОРЕЗ МЕЖДУ КЛЕТКАМИ-КОРМИЛКАМИ И ООЦИТОМ

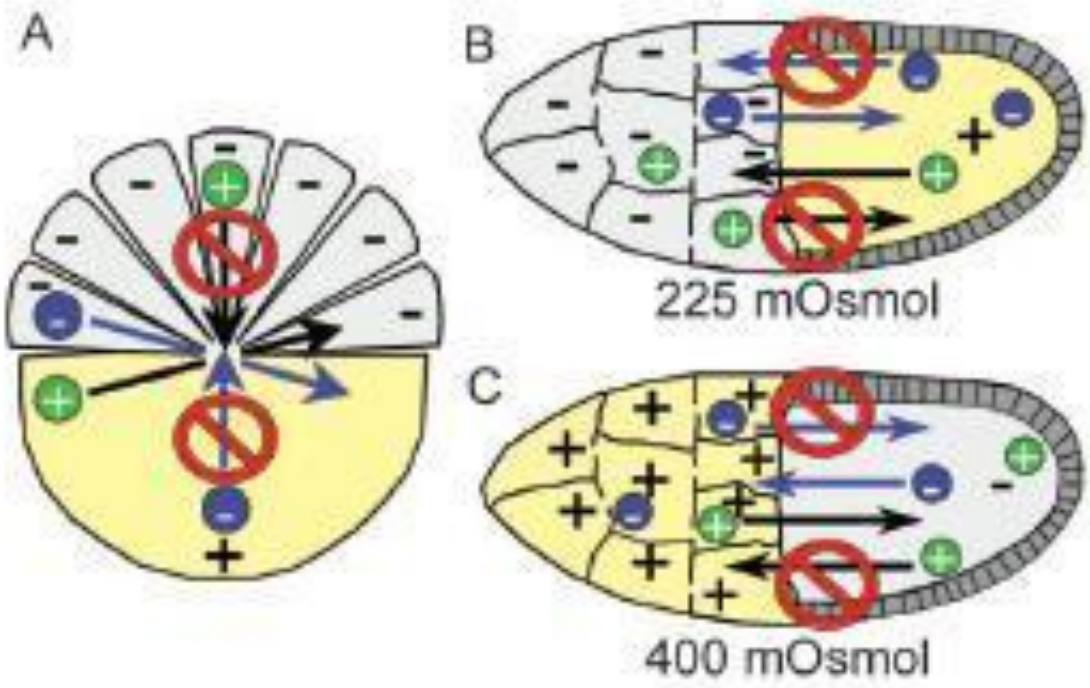


FIG. 13. Redistribution of charged molecules within the insect nurse cell (grey)/oocyte (yellow) syncytium. *A*: ovarian follicle of the moth *Cecropia*. Seven nurse cells are connected to the oocyte via a cytoplasmic bridge. *B*: ovarian follicle of the fruit fly *Drosophila*. Only 9 of the 15 nurse cells are shown. In *A* and *B*, the electrical potential of the oocyte is positive with respect to the follicle cells. Positively charged molecules injected into the oocyte move into the nurse cells as a result of the electric field across the cytoplasmic bridge, but when injected into the nurse cells, the same molecules did not move into the oocyte. Conversely, negatively charged molecules injected into the nurse cells move into the oocyte, but when injected into the oocyte they did not move into the nurse cells. The asymmetric distribution is a function of the potential difference in the two compartments. *C*: increasing the osmolarity of the extracellular medium from 250 mosM (physiological) to 400 mosM reverses the electrical potential of the *Drosophila* follicle. Two-dimensional gel electrophoresis of soluble proteins isolated from the cytoplasm nurse cells and oocytes indicates that charged endogenous proteins respond to the change in electrical potential in a manner consistent with their redistribution by the endogenous electrical gradient; that is, the direction of movement is opposite to that in *B*. (Based on Refs. 39, 209, 210.)

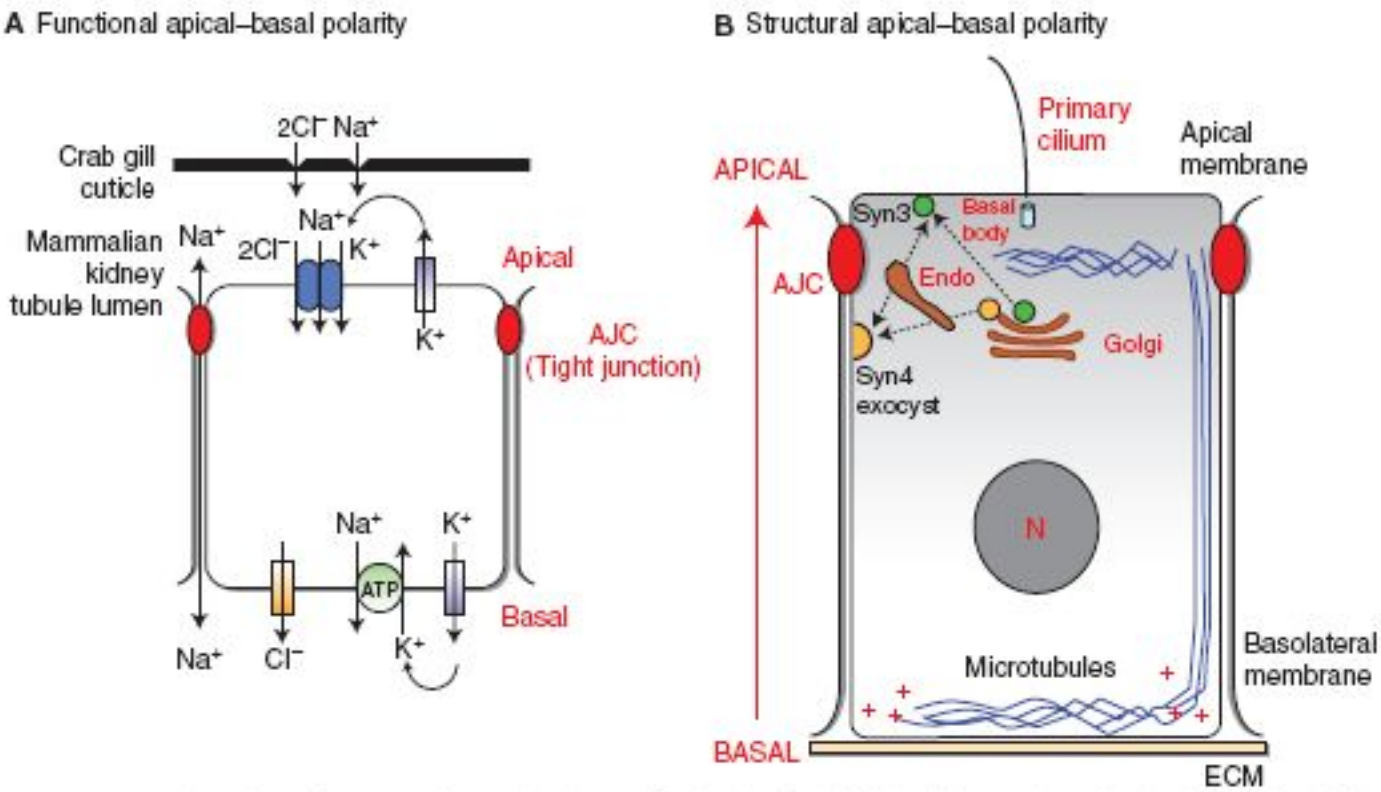
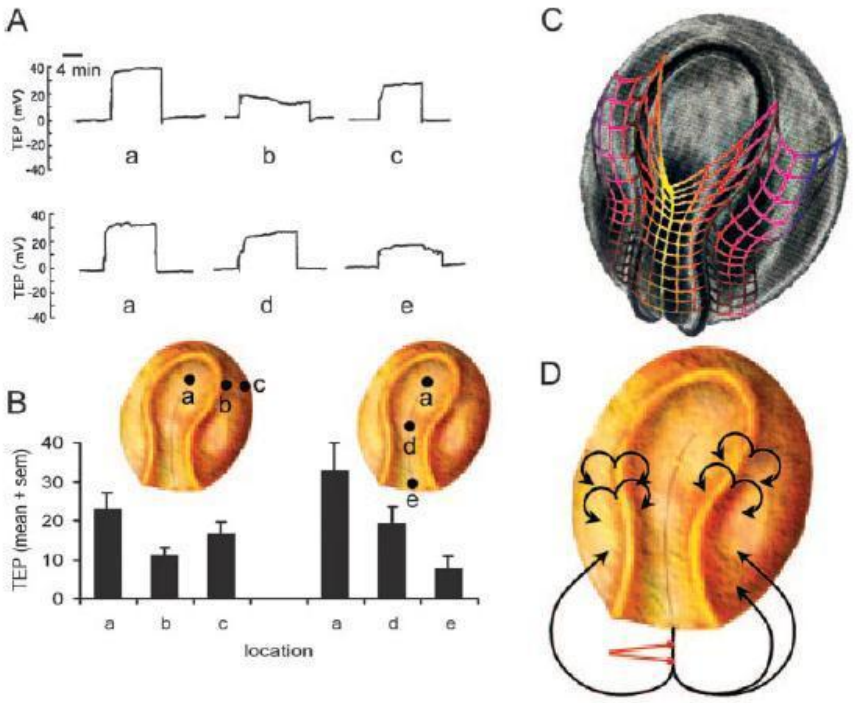
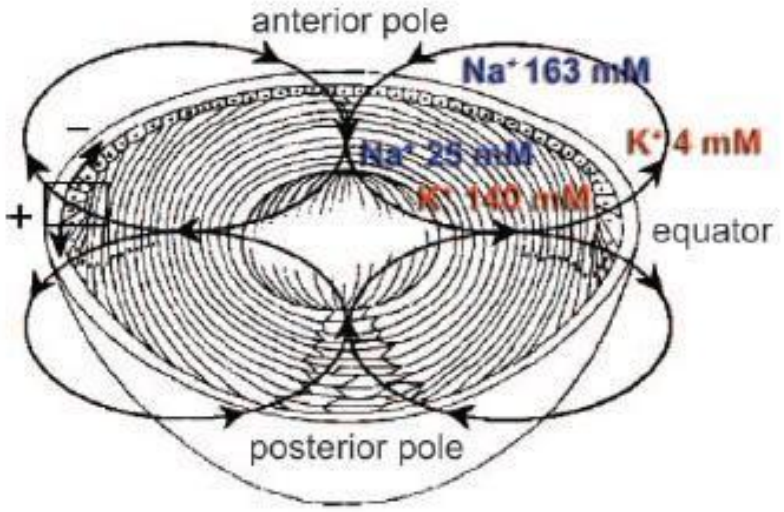


Figure 1. Functional and structural organization of polarized epithelia. (*A*) Functional apical–basal polarity. Physiological studies of transporting epithelia across the phyla (e.g., crab gill and mammalian kidney nephron) has revealed a remarkable conservation in the distribution of ion channels (Cl channel, K channel) transporters (Na,K,2Cl transporter) and pumps (Na/K-ATPase) between the apical and basolateral plasma membrane domains. The polarized distribution of these proteins generates an apical–basal sodium gradient that is used to move other ions and solutes across the epithelium. (Redrawn and adapted from Cereijido et al. 2004.) (*B*) Structural apical–basal polarity. Polarized epithelial cells have a distinctive apical–basal polarity in the orientation of cell–cell and cell–extracellular matrix (ECM). Major structures of these cells are also organized in the apical–basal axis: The organization of plasma membrane domains (apical and basolateral), junctional complexes (APC, apical junctional complex), the centrosome (basal body), microtubules and primary cilium, and the secretory pathway (Golgi). For details, see text.

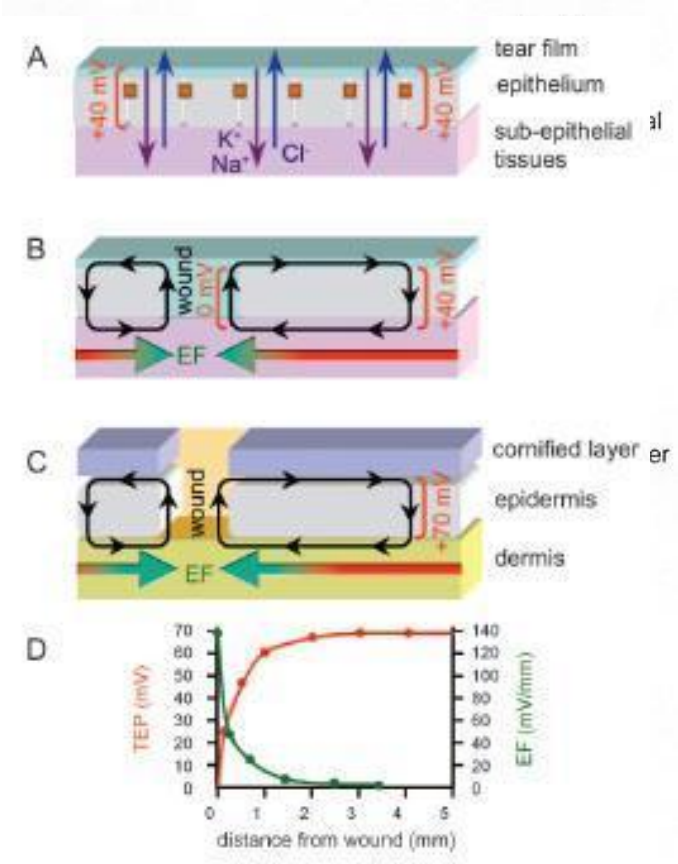
Электрические токи в эмбриональных тканях и органах

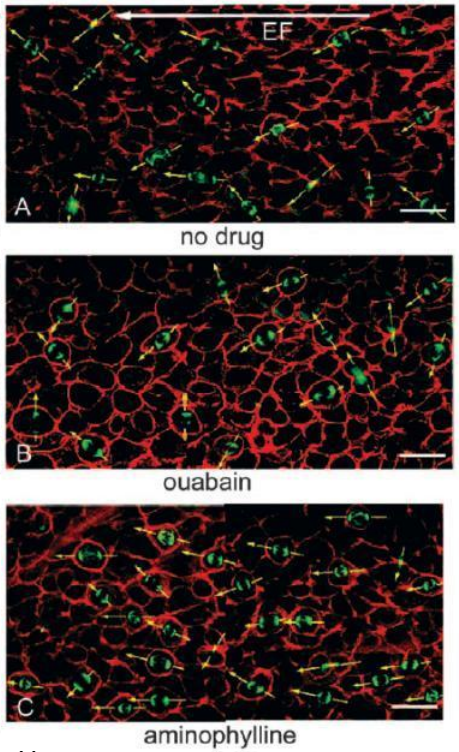


Глазной хрусталик



РОЛЬ РАНЕВЫХ ТОКОВ В ОРИЕНТАЦИИ КЛЕТОЧНЫХ ДЕЛЕНИЙ И АКСОНОВ





Усиление электрического сигнала

FIG. 10. Epithelial cell proliferation and the axis of cell division are controlled by naturally occurring wound-induced electrical signals. Mitotic profiles of corneal epithelial cells (green) in a whole mount rat cornea close to a wound edge (left margin). Wounding the cornea stimulates cell division near the wound edge, and the proportion of dividing cells drops off with distance back from the wound edge, as predicted if this were controlled by the wound-induced electrical signals. Enhancing these electrical signals pharmacologically, for example, with aminophylline, increased cell divisions (compare *A* with *C*), and suppressing the electrical signals with ouabain suppressed cell divisions (compare *A* and *C* with *B*). *A*: in untreated corneas, the long axis of the mitotic spindle (yellow arrows) was not oriented randomly, but lay significantly more parallel than perpendicular to the EF vector. *B*: in corneas where the electrical signal was suppressed with ouabain, the spindle axis was oriented randomly with respect to the EF vector. *C*: in corneas where the electrical signal was enhanced with aminophylline, the spindle axis was oriented strongly parallel to the EF vector. [Modified from Song et al. (184).]

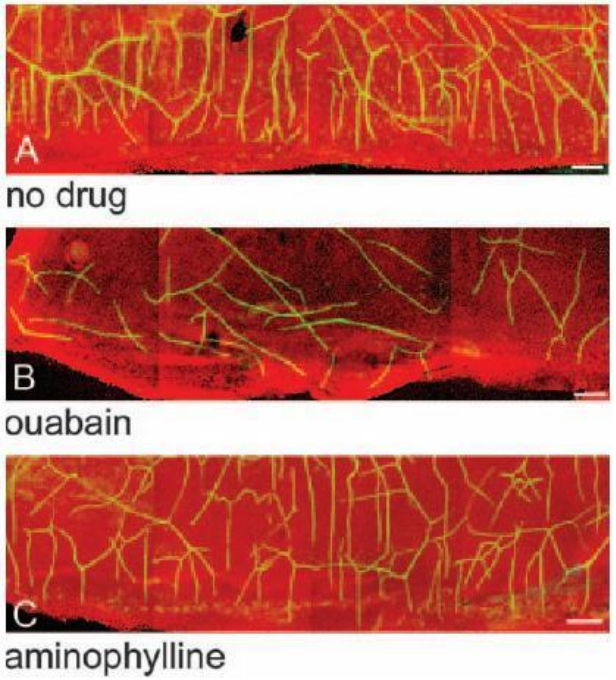
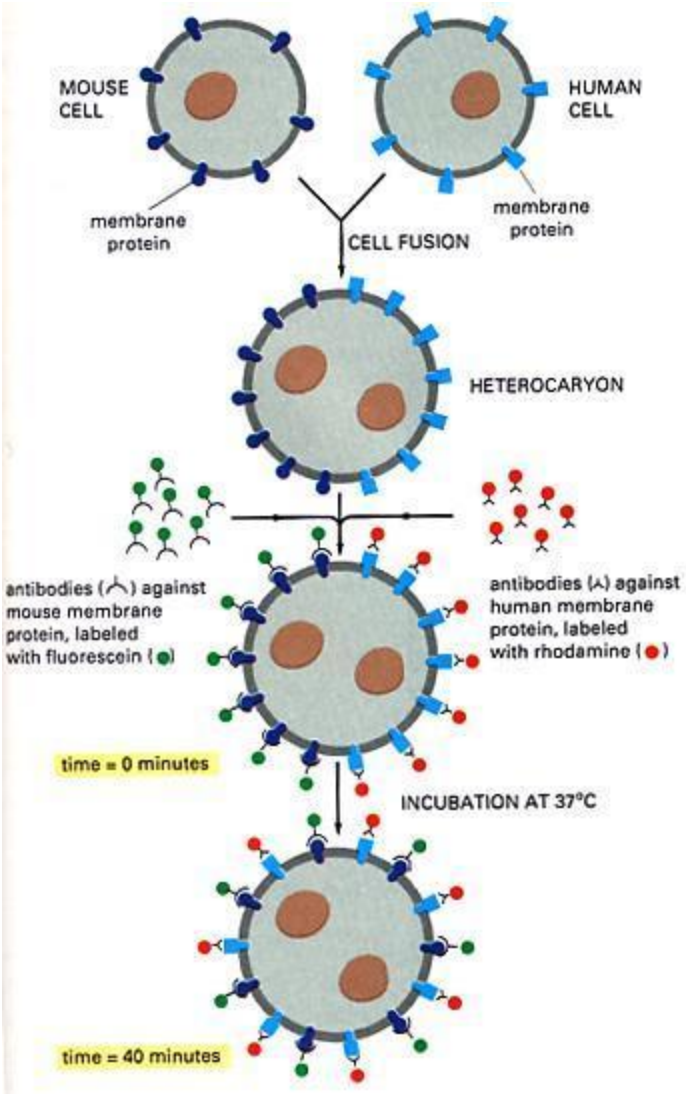
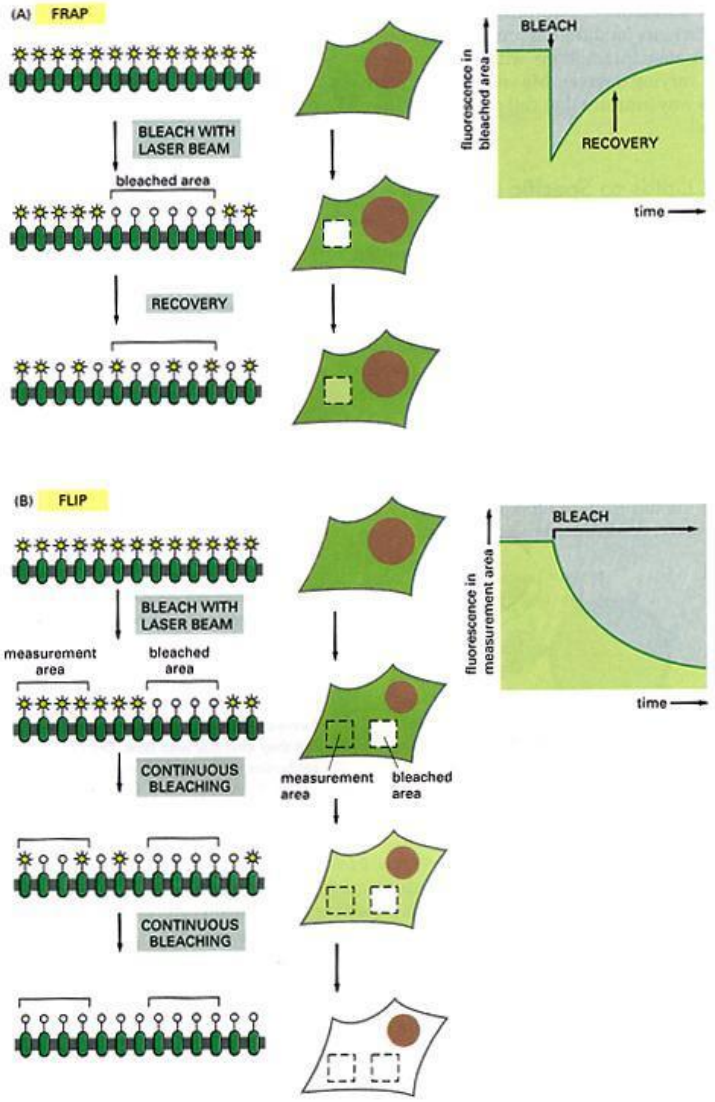


FIG. 11. Nerve sprouting is stimulated and directed by a wound-induced electrical signal in vivo. *A*: a wound in rat cornea attracts robust nerve sprouts within 24 h, and these are directed towards the wound edge. *B*: disrupting the wound-induced electrical field (with ouabain) did not prevent nerve sprouting, but sprouts were no longer directed by the EF vector towards the wound edge. *C*: enhancing the wound-induced electrical field (with aminophylline) increased and speeded up nerve sprouting along the EF vector towards the wound edge. [Modified from Song et al. (185).]

МЕТОДЫ ИЗУЧЕНИЯ ЛАТЕРАЛЬНОЙ ПОДВИЖНОСТИ КОМПОНЕНТОВ МЕМБРАНЫ

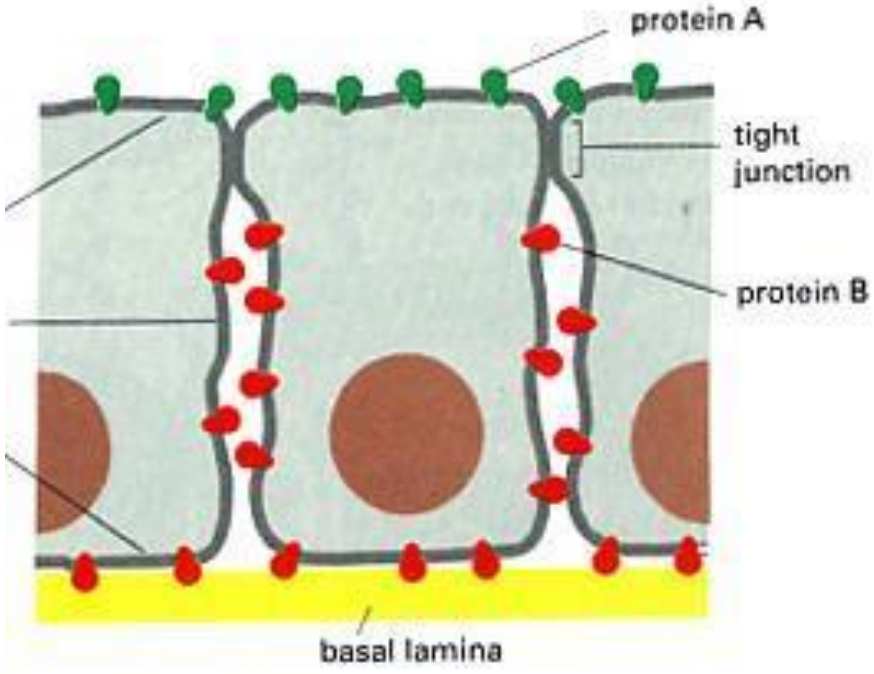


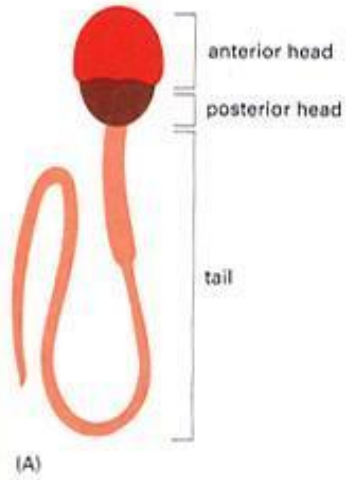


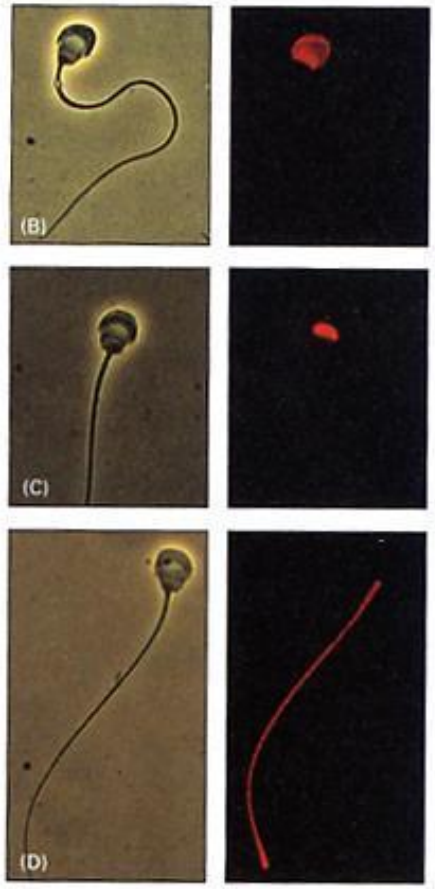
Латеральная диффузия липидов: 10^{-8} см^2 с$^{-1}$ (5-10 μm/s)

белков: 5x10^{-9} – 10-^{12} см^2 с$^{-1}$

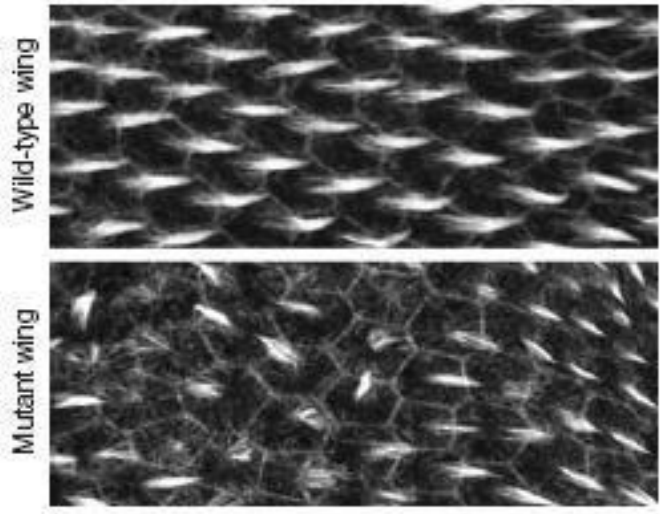
ОГРАНИЧЕНИЕ ЛАТЕРАЛЬНОЙ ПОДВИЖНОСТИ ИНТЕГРАЛЬНЫХ БЕЛКОВ КЛЕТОЧНЫХ МЕМБРАН

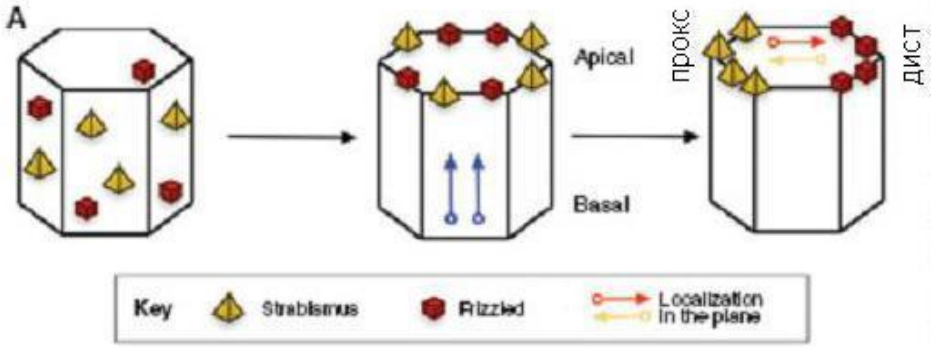


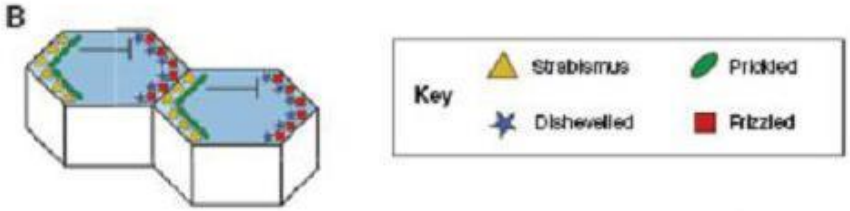


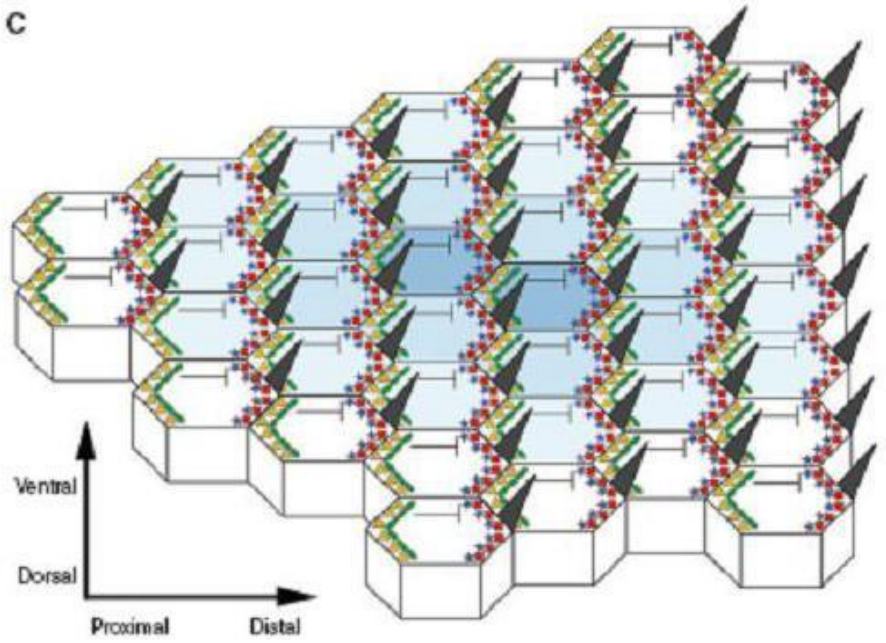


ЛАТЕРАЛЬНАЯ ПОЛЯРНОСТЬ КЛЕТОК









ЛАТЕРАЛЬНАЯ ПОЛЯРИЗАЦИЯ ЗИГОТЫ C.elegans

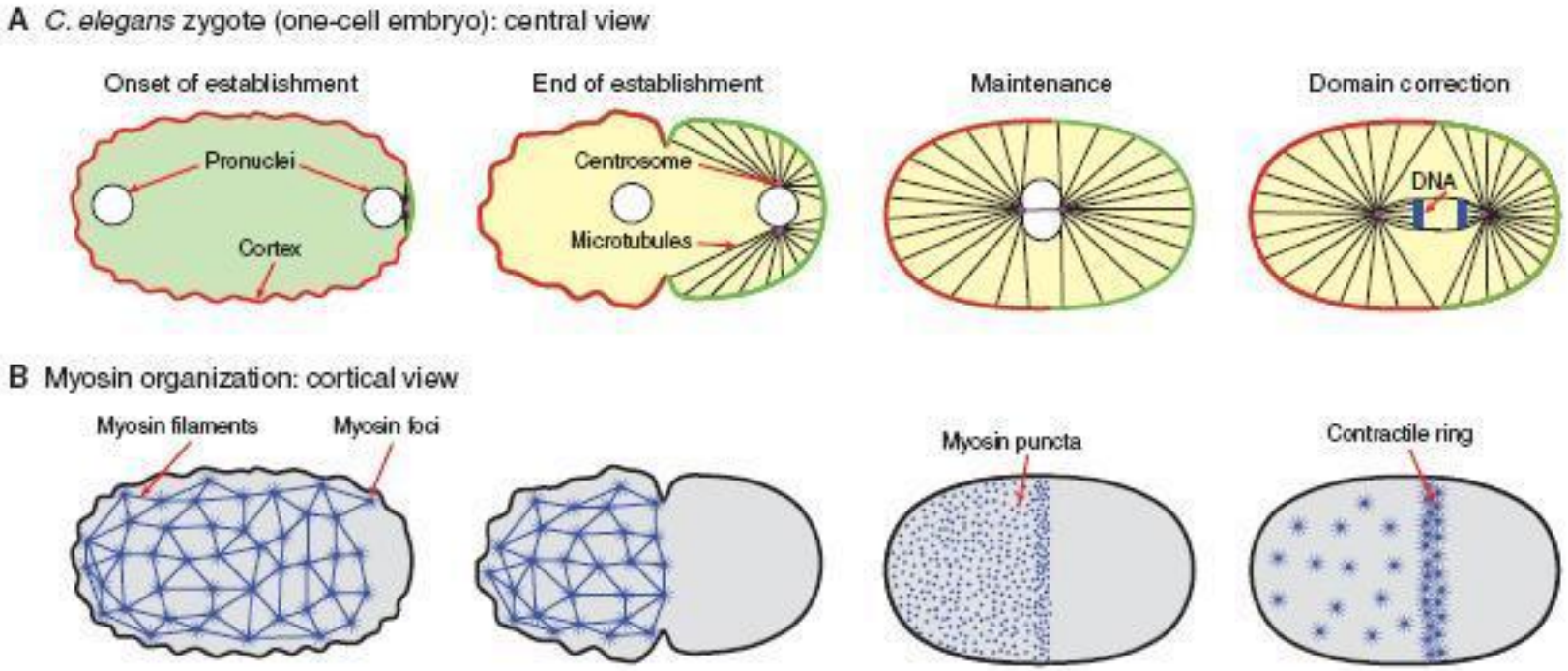
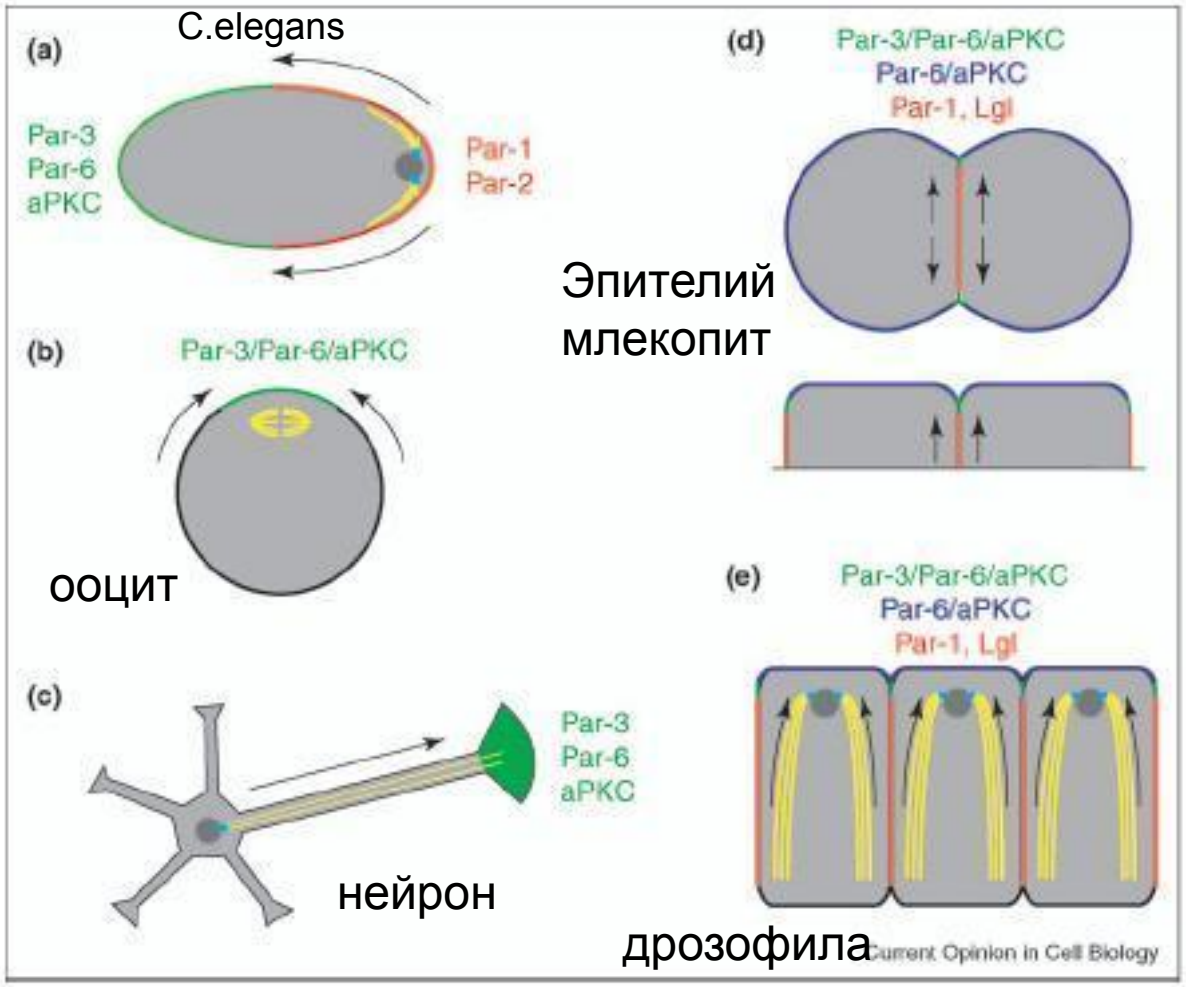
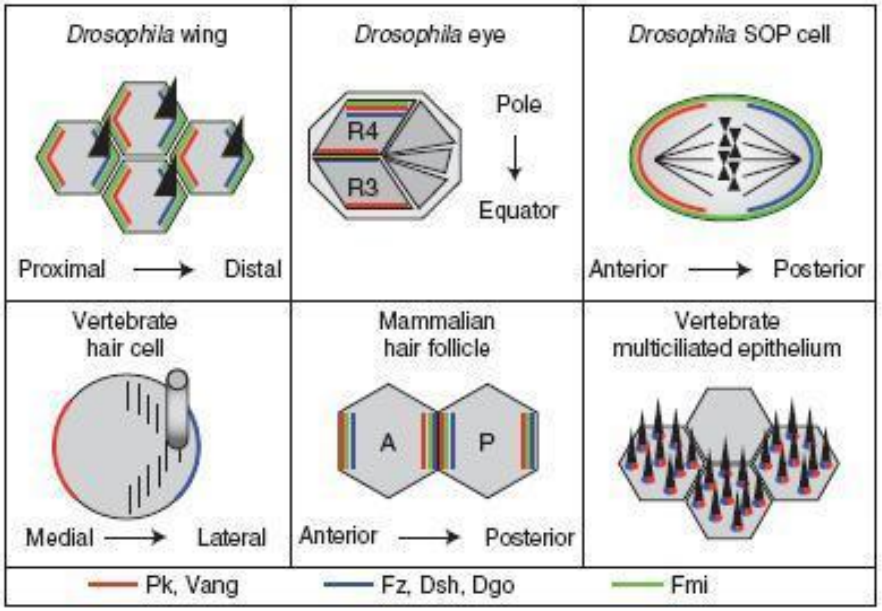
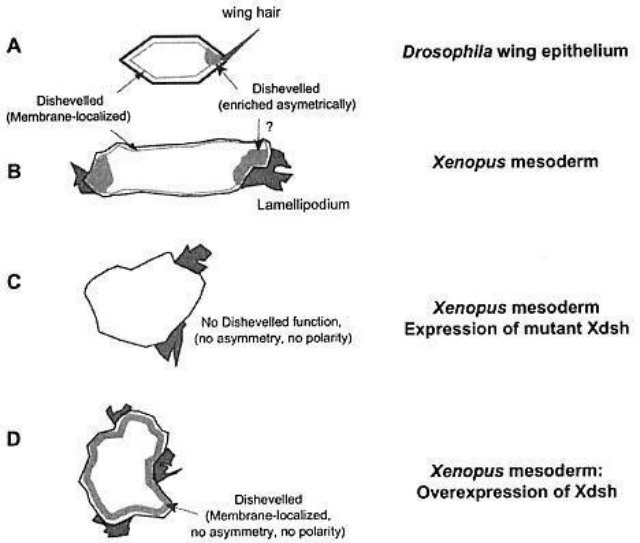


Fig. 1. PAR protein and myosin asymmetry during *C. elegans* zygote polarization. (**A**) Central view of a *C. elegans* zygote showing PAR-3 (red) and PAR-2 (green) domains during polarization. Anterior is to the left. Microtubules (black lines) are shown emanating from centrosomes (magenta). The centrosome triggers anterior movement of the cortical PAR-3 domain during the establishment phase, the period during which the PAR domains form. The cortical PAR-2 domain fills in the posterior cortex devoid of PAR-3. The border between the PAR-3 and PAR-2 domains stabilizes and remains in the middle of the zygote during the maintenance phase. The domains are adjusted to align with the cytokinesis furrow during the domain correction phase. (**B**) Cortical view of myosin (blue) organization during polarization at the same stages as shown in A. Anterior is to the left. Myosin foci and filaments are present in a contractile network at the onset of polarization, and during the establishment phase the network contracts asymmetrically to the anterior. The myosin network breaks down to form smaller puncta during the maintenance phase. During the domain correction phase, larger puncta of myosin form in the nascent contractile ring and anterior region. Myosin organization is based on Munro et al. (Munro et al., 2004) and Werner et al. (Werner et al., 2007).



Modes of PAR localization and transport in different cell types. **(a)** In *C. elegans*, actomyosin-based cortical flows transport Par-3/Par-6/aPKC away from the sperm MTOC, allowing Par-1 and Par-2 to associate with the posterior cortex. **(b)** In vertebrate oocytes, Par-3/Par-6/aPKC forms a cap over the meiotic spindle in an actomyosin-dependent fashion. **(c)** In hippocampal neurons, Par-3/Par-6/aPKC accumulates at the tip of the axon through an association with Kif-3a (Kinesin II) and APC. **(d)** In cultured mammalian epithelial cells, nascent cadherin-based junctional structures move in an actomyosin-dependent way towards the edges of cell contacts and towards the apical surface, where they recruit Par-3/Par-6/aPKC, either directly or through intermediates like Jam-1 or the nectins. **(e)** In *Drosophila* embryos during cellularization, Par-3 accumulates at the apicolateral boundary, in part through dynein-based transport towards the apically localized centrosomes, although other mechanisms may also be involved. In all these cases, cross-regulatory interactions (see Figure 2) will shape spatial distributions of Par-1, Par-2 and Lgl in response to active redistribution of Par-3/Par-6/aPKC.





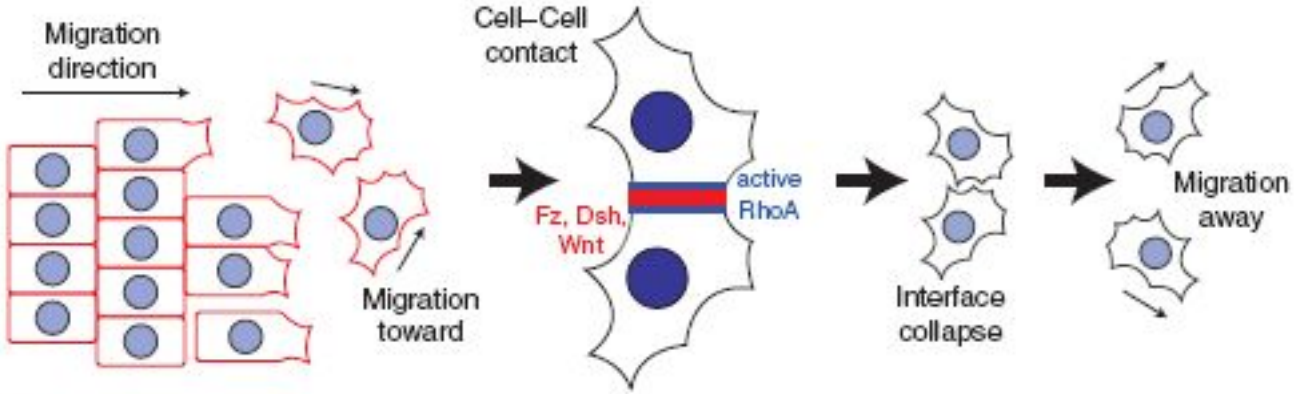
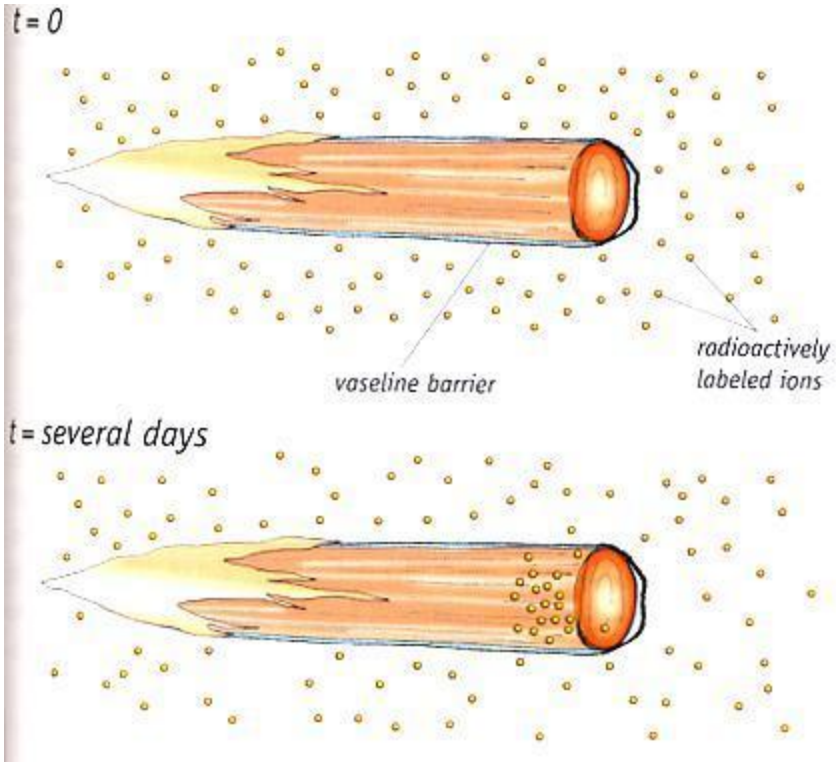
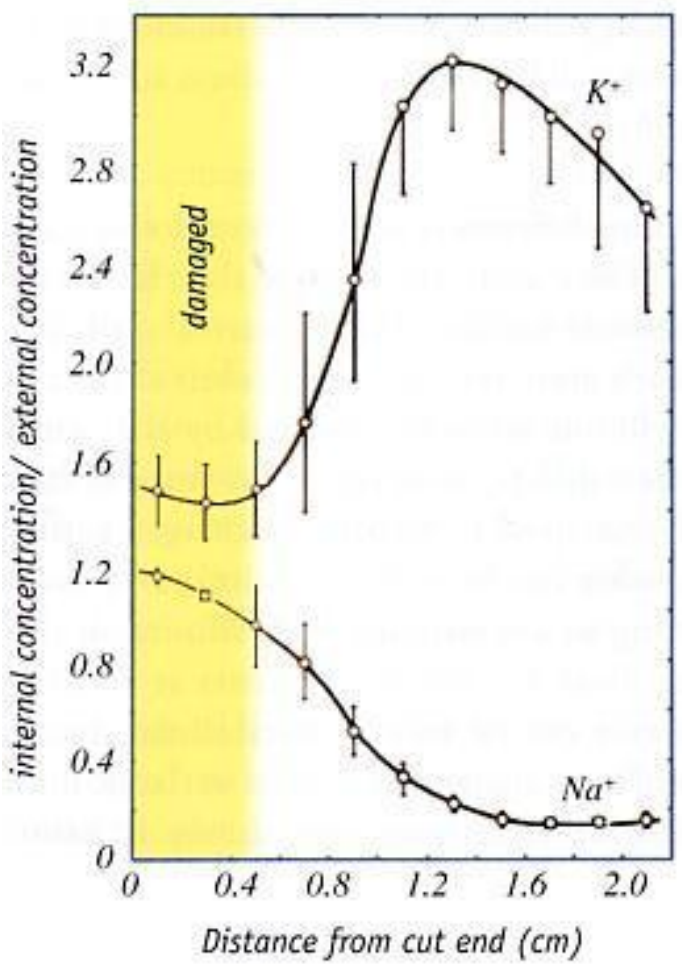


Figure 3. PCP components regulate contact inhibition of locomotion in migrating neural crest cells. Schematic of neural crest cell migration showing cells breaking away from the epithelial sheet leading edge, migrating toward each other, and forming an interface, which then collapses and prompts cells to migrate in opposite directions. Initially, PCP components have uniform distributions, but then relocalize to the site of cell–cell contact. PCP components are required to arrest and then alter the migration path, possibly through activation of RhoA.

Восстановление нормальных соотношений K^+ и Na^+ в перерезанной мышце





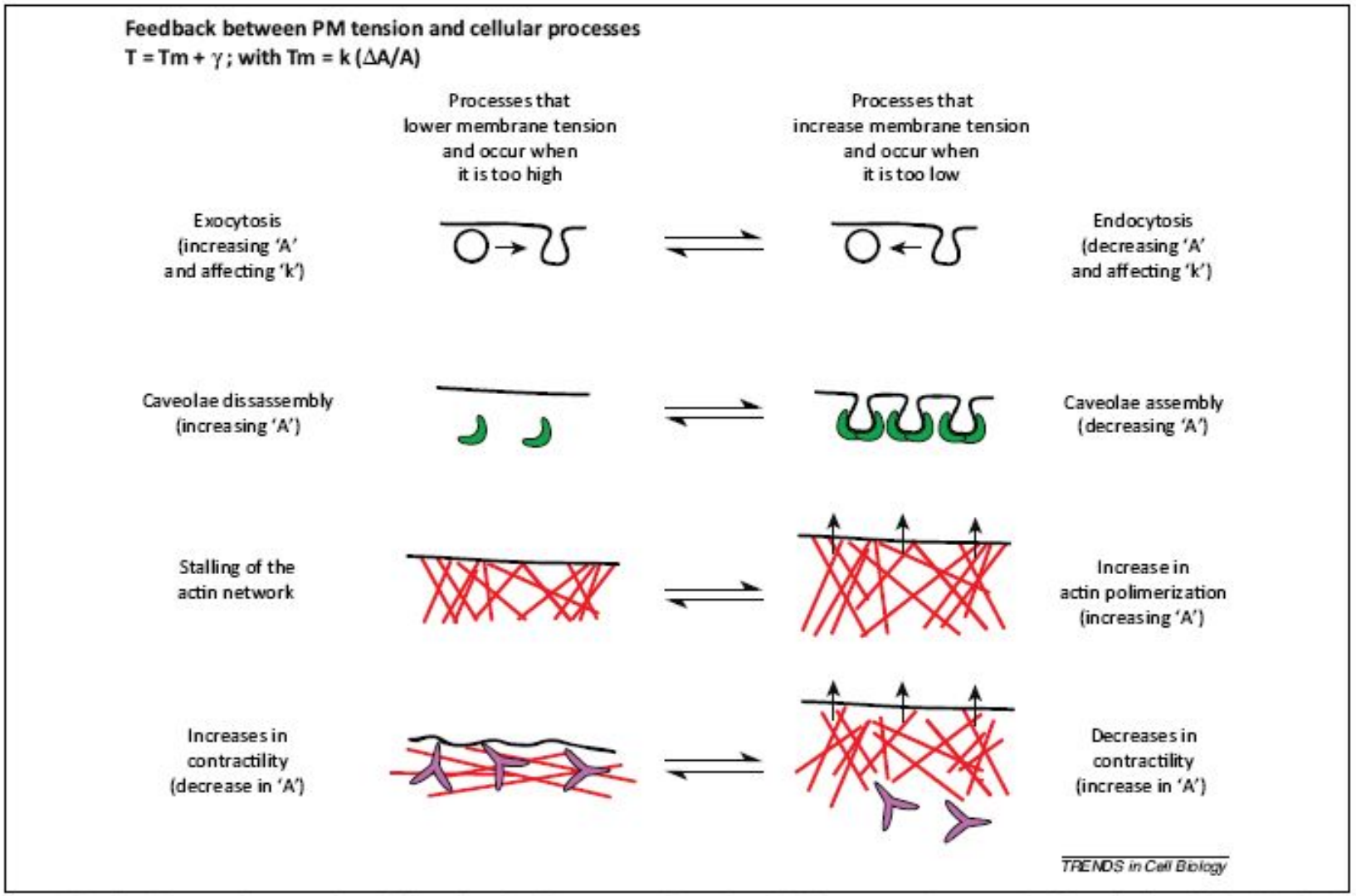


Figure 1. Feedback between plasma membrane (PM) tension and cellular processes. Examples of cellular processes that occur when PM tension is too high and that lead to its reduction (left) or that occur when PM tension is too low and lead to its increase (right) – vesicle trafficking, caveola formation, 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.