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1: Biochim Biophys Acta. 2000 Dec 20;1498(2-3):273-80.

Mechanism of calcium oscillations in migrating human astrocytoma cells.

Ronde P, Giannone G, Gerasymova I, Stoeckel H, Takeda K, Haiech J.

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Numerous studies show that intracellular calcium controls the migration rate of different mobile cell types. We studied migrating astrocytoma cells from two human cell lines, U-87MG and A172, in order to clarify the mechanisms by which calcium potentially influences cell migration. Using the wound-healing model to assay migration, we showed that four distinct components of migration could be distinguished: (i) a Ca(2+)/serum-dependent process; (ii) a Ca(2+)-dependent/serum-independent process; (iii) a Ca(2+)/serum-independent process; (iv) a Ca(2+)-independent/serum-dependent process. In U-87MG cells which lack a Ca(2+)-dependent/serum-independent component, we found that intracellular Ca(2+) oscillations are involved in Ca(2+)-dependent migration. Removing extracellular Ca(2+) greatly decreased the frequency of migration-associated Ca(2+) oscillations. Furthermore, non-selective inhibition of Ca(2+) channels by heavy metals such as Cd(2+) or La(3+) almost completely abolished changes in intracellular Ca(2+) observed during migration, indicating an essential role for Ca(2+) channels in the generation of these Ca(2+) oscillations. However, specific blockers of voltage-gated Ca(2+) channels, including nitrendipine, omega-conotoxin GVIA, omega-conotoxin MVIIC or low concentrations of Ni(2+) were without effect on Ca (2+) oscillations. We examined the role of internal Ca(2+) stores, showing that thapsigargin-sensitive Ca(2+) stores and InsP(3) receptors are involved in Ca(2+) oscillations, unlike ryanodine-sensitive Ca(2+) stores. Detailed analysis of the spatio-temporal aspect of the Ca (2+) oscillations revealed the existence of Ca(2+) waves initiated at the leading cell edge which propagate throughout the cell. Previously, we have shown that the frequency of Ca(2+) oscillations was reduced in the presence of inhibitory antibodies directed against beta3 integrin subunits. A simple model of a Ca(2+) oscillator is proposed, which may explain how the generation of Ca(2+) oscillations is linked to cell migration.

PMID: 11108969 [PubMed - indexed for MEDLINE]

1: Arterioscler Thromb Vasc Biol. 2000 Sep;20(9):E34-40. Related Articles, Links

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Inhibition of type 4 phosphodiesterase by rolipram and Ginkgo biloba extract (EGb 761) decreases agonist-induced rises in internal calcium in human endothelial cells.

Campos-Toimil M, Lugnier C, Droy-Lefaix MT, Takeda K.

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The effects of Gingko biloba extract EGb 761 on 5 isolated, vascular, cyclic nucleotide phosphodiesterase (PDE) isoforms were evaluated. EGb 761 preferentially inhibited PDE4 (IC (50)=25.1 mg/L), the isoform that is mainly present in endothelial cells, in a competitive manner (K:(i)=12.5 mg/L). Because changes in cyclic nucleotide levels may affect intracellular calcium ([Ca(2+)](i)) levels in endothelial cells, we examined the effects of EGb 761 on both resting [Ca(2+)](i) levels and agonist-induced rises in [Ca(2+)](i) in single human umbilical vein endothelial cells (HUVECs) in culture. The effects of EGb 761 were compared with those of rolipram, a selective PDE4 inhibitor that increases cellular cAMP levels, and the cAMP analogue dibutyryl cAMP (db-cAMP). EGb 761 (20 and 100 mg/L), rolipram (50 micromol/L), and db-cAMP (100 micromol/L) significantly inhibited histamine-, ATP-, and thrombin-induced [Ca(2+)](i) increases in HUVECs without modifying resting [Ca(2+)](i) levels. Similar results were obtained by using a Ca(2+)-free bath solution. EGb 761 (100 mg/L), but not rolipram (50 micromol/L) or db-cAMP (100 micromol/L), also inhibited Ca (2+) influx into cells having thapsigargin-depleted internal Ca(2+) stores and bathed in a Ca (2+)-free external solution. Our results are consistent with an inhibition of PDE activity that causes a reduction of agonist-induced increases in [Ca(2+)](i) in HUVECs, mainly by inhibition of Ca(2+) mobilization from internal stores. It thus may be that the cardiovascular effects of EGb 761 involve inhibition of PDE4 activity and subsequent modification of Ca(2+) signaling in endothelial cells.

PMID: 10978267 [PubMed - indexed for MEDLINE]

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ELSEVIER FULL-TEXT ARTICLE

An adrenal slice preparation for the study of chromaffin cells and their cholinergic innervation.

Barbara JG, Poncer JC, McKinney RA, Takeda K.

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Thin slices (200-300 microm) of adrenal glands were prepared from Wistar rats. Patch-clamp recordings were made from visually identified chromaffin cells using the whole-cell and amphotericin B perforated-patch techniques. Electrophysiological properties of chromaffin cells in slices were similar to those in cultured cells. Catecholamine release from single chromaffin cells or cell clusters in slices was also measured by amperometry. Immunostaining of slices with an antineurofilament antibody revealed the presence of neuronal fibers. Acetylcholine release was stimulated either by raising external [K+] or by focally applying voltage pulses. Nicotinic excitatory postsynaptic currents (EPSCs) were detected, ranging from 20 pA to several hundreds of pA. Amplitude distributions of spontaneous EPSCs revealed clear equidistant peaks, supporting a quantal model for acetylcholine release onto chromaffin cells. The adrenal slice preparation therefore appears to be an excellent model for studying both the cholinergic innervation of chromaffin cells as well as catecholamine release from these cells.

PMID: 9667391 [PubMed - indexed for MEDLINE]

1: Bioconjug Chem. 1997 Jul-Aug;8(4):472-80. Related Articles, Links

Nicotinic acetylcholine receptor labeled with a tritiated, photoactivatable agonist: a new tool for investigating the functional, activated state.

Kotzyba-Hibert F, Kessler P, Zerbib V, Grutter T, Bogen C, Takeda K, Hammadi A, Knerr L, Goeldner M.

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Upon agonist activation, the nicotinic acetylcholine receptor undergoes allosteric transitions leading to channel opening and sodium ion influx. The molecular structure of the agonist binding site has been mapped previously by photoaffinity labeling, but most photosensitive probes used for this purpose interact only with closed receptor states (resting or desensitized). We have synthesized two novel photoactivatable 4-diazocyclohexa-2,5-dienone derivatives as cholinergic agonist candidates, with the objective of identifying structural changes at the acetylcholine binding site associated with receptor activation. One of these ligands, 9b, is a functional agonist at muscle acetylcholine receptors in human TE 671 cells. In photolabeling experiments with 9b, up to 35% inactivation of agonist binding sites was observed at Torpedo acetylcholine receptors. Tritiated 9b was synthesized, and photolabeling was found to occur mainly on the alpha-subunit in a partially protectable manner. This novel radiolabeled photoprobe appears to be suitable for future investigation of the molecular dynamics of allosteric transitions occurring at the active acetylcholine receptor binding site.

PMID: 9258443 [PubMed - indexed for MEDLINE]

: J Neurochem. 1996 Dec;67(6):2557-65. Related Articles, Links

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Novel photoactivatable agonist of the nicotinic acetylcholine receptor of potential use for exploring the functional activated state.

Kotzyba-Hibert F, Kessler P, Zerbib V, Bogen C, Snetkov V, Takeda K, Goeldner M, Hirth C.

Laboratoire de Chimie Bioorganique-URA 1386 CNRS, Illkirch, France.

The nicotinic acetylcholine receptor (AChR) exhibits at least four different conformational states varying in affinity for agonists such as acetylcholine (ACh). Photoaffinity labeling has been previously used to elucidate the topography of the AChR. However, to date, the photosensitive probes used to explore the cholinergic binding site photolabeled only closed or desensitized states of the receptor. To identify the structural modifications occurring at the ACh binding site on allosteric transition associated with receptor activation, we have investigated novel photoactivatable 4-diazocyclohexa-2,5-dienone derivatives as putative cholinergic agonists. Such compounds are fairly stable in the dark and generate highly reactive carbenic species on irradiation. In binding experiments using AChRs from Torpedo marmorata, these ligands had affinities for the ACh binding site in the micromolar range and did not interact with the noncompetitive blocker site (greater than millimolar affinity). Irreversible photoinactivation of ACh binding sites was obtained with the ligand 1b (up to 42% at 500 microM) in a protectable manner. In patch-clamp studies, 1b was shown to be a functional agonist of peripheral AChR in TE 671 cells, with the interesting property of exhibiting no or very little desensitization even at high concentrations.

PMID: 8931490 [PubMed - indexed for MEDLINE]

1: J Physiol. 1995 Nov 1;488 (Pt 3):609-22. Related Articles, Links

Voltage-dependent currents and modulation of calcium channel expression in zona fasciculata cells from rat adrenal gland.

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1. Whole-cell voltage-activated currents from single zona fasciculata (ZF) cells from rat adrenal glands were studied. T- and L-type Ca2+ currents and a slowly inactivating A-type K+ current were the three major currents observed. 2. In freshly isolated cells, the A-type K+ current and the T-type Ca2+ current were predominant. The A-type current was activated at -50 mV and inhibited by 4-amino-pyridine with a half-maximal block (IC50) at 130 microM while the T-type current was activated at -70 mV and blocked by Cd2+, Ni2+ and amiloride with IC50 values of 24.1, 132.4 and 518.9 microM, respectively. 3. Under current clamp, depolarizing current pulses produced a single Ca2+ action potential with Cs+ in the pipette internal solution. Upon replacement of Cs+ by K+, the half-amplitude width of the action potential was shortened and membrane potential oscillations were seen after the spike. 4. In freshly isolated cells and during the first 24 h after plating, the T-type current was observed in all cells, with L-type current being observed in < 2% of cells, even in the presence of (+)SDZ 202,791, a dihydropyridine Ca2+ channel agonist. With time in culture, the T-type current disappeared, and a high-voltage-activated L-type current became increasingly apparent. In cells tested after > 2 days in culture, (+)SDZ 202,791 potentiated L-type current by 407 +/- 12% and the antagonist (-)SDZ 202,791 blocked this increase. The L-type current was activated between -30 and -20 mV and was sensitive to nitrendipine and omega-conotoxin GVIA. 5. Pre-incubation of cultured ZF cells with adrenocorticotrophic hormone (ACTH) or vasoactive intestinal peptide (VIP) for 3 days resulted in a high, sustained level of expression of T-type current, with a mean amplitude of 34.2 +/- 5.5 pA pF-1 for ACTH-treated cells compared with 3.4 +/- 1.8 pA pF-1 for untreated cells. Cycloheximide strongly inhibited this effect. Neither treatment affected L-type current expression. 6. The expression of both Ca2+ current types was unaffected by pre-incubation with 8-bromo-cAMP or forskolin. The protein kinase A antagonist, H89, did not inhibit the ACTH-induced upregulation of T-type Ca2+ currents. 7. It is concluded that the main voltage-dependent currents involved in cell excitability and steroidogenesis in rat adrenal ZF cells are an A-type K+ current and a T-type Ca2+ current. The physiological role and control of expression of L-type Ca2+ channels in rat ZF cells remain less clear.

PMID: 8576852 [PubMed - indexed for MEDLINE]

☐ 1: J Physiol. 1990 Sep;428:545-60. Related Articles, Links

Nicotinic cholinergic modulation of voltage-dependent calcium current in bovine adrenal chromaffin cells.

Klepper M, Hans M, Takeda K.

Universite Louis Pasteur de Strasbourg, Laboratoire de Pharmacologie Cellulaire et Moleculaire-CNRS URA600, Illkirch, France.

1. The effects of cholinergic agonists on voltage-dependent calcium current (ICa) were studied in cultured chromaffin cells from bovine adrenal medulla. 2. Application of both acetylcholine (ACh) and nicotine resulted in inward nicotinic current from a holding potential of -90 mV, and at the same time reversible decreases in depolarization-activated ICa. Both of these effects were blocked by d-tubocurarine, while atropine pre-treatment was ineffective. 3. Internal accumulation of neither Na+ nor Ca2+ seems likely to explain the nicotinic-agonist-dependent decrease in ICa, as the modulation was observed with symmetrical Na+ solutions, with Ca2 (+)-free Ba2(+)-containing external solutions, from holding potentials of both -90 and -40 mV, and when the internal Ca2+ buffer capacity was increased. 4. Isodihydrohistrionicotoxin, an open-channel blocker which does not compete for the agonist binding site, completely inhibited inward cholinergic currents while the agonist-dependent decrease in ICa was seen in only two of fifteen cells. 5. The nicotinic agonist-mediated decreases in ICa were not voltage-dependent. 6. No changes in voltage-dependent INa were seen with the nicotinic agonists. 7. Muscarine, with or without GTP in the pipette solution, produced neither modulation of ICa nor any changes in steady holding currents. The nicotinic current and the reversible decrease in ICa induced by ACh and nicotine were not affected by including GTP, or the guanine nucleotide analogues GDP-beta-S and GTP-gamma-S, in the pipette solution. 8. A 10 min pre-incubation of the cells in a high-K+ solution optimal for catecholamine secretion did not affect the nicotinic agonist-mediated decreases in ICa.

PMID: 2172525 [PubMed - indexed for MEDLINE]

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Voltage-dependent and agonist-activated ionic currents in vascular endothelial cells: a review.

Takeda K, Klepper M.

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Vascular endothelial cells produce a variety of substances known to modulate the tone of surrounding smooth muscle, but the initial steps involved in receptor-response coupling are poorly characterized in these cells. Because the stimulated release of endothelium-derived relaxing factor depends on the presence of external calcium, ion channel-mediated calcium influx might represent an essential first link. Furthermore, agonist-induced endothelial cell hyperpolarization has been widely described, although the ion channels involved and the functional significance of this response remain uncertain. A review of the available literature to date concerning voltage-dependent and agonist-activated ionic currents obtained using patch clamp techniques in vascular endothelial cells is presented here. A discussion of the possible functional roles of the underlying ion channels is included.

Publication Types:
- λ Review
- λ Review, Tutorial

PMID: 1700734 [PubMed - indexed for MEDLINE]

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