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Parallel processing in human audition and post-lesion plasticity

S Clarke

Abstract

Recent activation and electrophysiological studies have demonstrated that sound recognition and localization are processed in two distinct cortical networks that are each present in both hemispheres. Sound recognition and/or localization may be, however, disrupted by purely unilateral damage, suggesting that processing within one hemisphere may not be sufficient or may be disturbed by the contra lateral lesion. Based on the results of our research on acute and chronic lesions, patterns of recovery and functional imaging, evidence will be presented, which support the latter hypothesis.

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Neuronal basis of tactile sense in the rat whisker system

E Arabzadeh; E Zorzin; ME Diamond

Abstract

Using their whiskers, rats have tactile capacities rivaling those of the human with our fingertips. We have carried out experiments to explore how neurons encode touch signals to build up a central representation. Touch signals begin with the receptors in the follicle of each whisker and can be traced to a columnar module in somatosensory cortex that is connected with the same whisker: the well-known whisker-to-column topography. We first examine the spatial organization or tactile information and find that the columnar topography acts as a framework for the organization of sensory information in behaving rats. Then, we consider the messages carried by neurons. Because rats can sweep their whiskers across surfaces to identify texture with very fine capacities, we ask what the brain representation of texture is. We will present evidence that the surface features of an object induce vibrations in the whisker shaft with distinct kinetic "signatures." The brain encodes the kinetics of whisker vibration to achieve a reliable representation of texture.

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Why does the central nervous system not regenerate after injury?

J Nicholls

Abstract

A major problem for neuroscientists and clinicians is why the central nervous system shows ineffective regeneration after injury. Injured peripheral nerve fibers reform their connections, whereas those in injured spinal cord never re-grow. Insights into the mechanisms for repair and restoration of function after spinal cord injury have been obtained by experiments showing that injured nerve cells in the adult mammalian brain can indeed send out new fibers over a distance, if they are provided with appropriate tissue to grow along. Another major finding is that molecules exist in the CNS that inhibits the outgrowth of injured nerve cells. The aim of our experiments is to define more precisely changes that occur in devel¬opment, as the spinal cord changes from being able to regenerate to the adult state of failure. Our experiments show that after complete transection, spinal cords of newborn opossum pups regenerate provided that the animal is younger than about 12 days of age, after which the ability for repair becomes lost abruptly. We are now determining by subtractive suppression PCR which molecules decrease, and which inhibitory molecules increase during this critical period. Among growth-promoting genes we have found those for cadherin, catenin and molecules that affect transcription. Inhibitory genes include those associated with myelin and a different set of genes that affect transcription. Northern blots, reverse transcription PCR and in situ hybridization have confirmed these changes in gene expression. In spite of progress in many labs in recent years, major hurdles must be overcome before patients can be treated. Once a good understanding of mechanisms that promote and prevent regeneration is obtained, one will still have to de¬vise safe treatments for patients with spinal cord injuries, as well as methods for applying them. Nevertheless, the picture is not as bleak and dis¬couraging as it was: one can think today of strategies for doing research on spinal cord injury so as to promote regeneration and restore function.

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The role of glia in neurological disease

L Garey

Abstract

Glial cells form a network in the central nervous system to support neurons and interact with them. The glia consist essentially of astrocytes that help with the nutrition of neurons and react in some cases of injury, oligodendrocytes that produce myelin, and microglia that are derived from the haemopoietic system and are concerned with the immunological defense of the nervous system. Experimental allergic encephalomyelitis is an animal model of multiple sclerosis, characterized by infiltrates of T cells and demyelinisation. It is produced in genetically susceptible rats (DA-Dark Agouti) by inoculation of foreign protein. Other non-susceptible rats (AO-Albino Oxford) react by producing cellular infiltrates, but this disappears by apoptosis without clinical manifestation of the disease. In this study, OX-42 antibodies were used to identify microglia, and GFAP for astrocytes, in the cellular infiltrates in the lumbar spinal cord and the brainstem 10, 14, and 21 days after inoculation of myelin basic protein. There are cellular infiltrates in both DA and AO groups 10 days after inoculation, and at 14th day the cells are even more numerous in the DA. Obvious astrocytosis is evident in the DA at 10th, 14th and 21st days, but less in the AO. Microgliosis is pronounced in both strains at 10th day. At 14th day, it disappeared in the DA, but persisted in the AO. We propose the hypothesis that the infiltrates are dispersed by microglial activity, which persists in the AO and prevents astrocytosis. In the DA, the transitory microgliosis allows astrocytosis, demyelinisation due to oligodendrocytic lesions, and clinical disease. Schizophrenia is associated with glial changes in the cerebral cortex. We have studied astrocytes and microglia in the association cortex of chronic schizophrenics and non-schizophrenic controls. The number of astrocytes does not vary between schizophrenics and controls, but microglia increased. Therefore, it can be concluded that there is cortical microgliosis in schizophrenics that could be a response to a lesion early in life, with a protective function for neighboring neurons. In adults, these microglia might serve to maintain modified cortical microcircuitry. We propose that in an animal model and also in human diseases, microglia play a key functional role in the integration of glial and neuronal activity. They protect the nervous system against external aggression, and prevent the development of serious lesions of other glia and of neurons.

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Selective deficits in human audition: evidence from lesion studies

R Nilipour; S Clarke; B No'doust; G Tarighat Saber; A Najlerahim

Abstract

The human auditory cortex is the gateway to the most powerful and complex communication systems and yet relatively little is known about its functional organization as compared to the visual system. Several lines of evidence, predominantly from recent studies, indicate that sound recognition and sound localization are processed in two at least partially independent networks. Evidence from human studies on selective auditory deficits following brain lesions has indicated that auditory processing after brain damage relies on alternative neural networks. This paper is a report on the performance of five brain-damaged patients and six normal subjects in sound localization and sound recognition aptitudes, as assessed by the number of correct replies and reaction times on auditory tasks. Based on the results, brain-damaged patients can have slower reaction times for specific functions, reflecting most probably processing within alternative networks. In our study three out of five patients presented deficits in auditory spatial tasks or in sound identification and sound motion, confirming previous findings of such dissociations. Our data indicated that response times were significantly slower, as compared to normal subjects, in deficient domains. Each of the three patients had at least one domain in which normal performance in terms of correct replies was associated with response times within the normal range, speaking against a general slowness. The results of this research and the general approach presented in this paper are relevant to the evaluation of potential plasticity.

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Postnatal development of spatial coding in the gravity sensing system

YS Chan; CH Lai; SK Lai; C Li; YC Tse

Abstract

The critical maturation time of central otolith neurons in processing spatial orientations was examined in Sprague-Dawley rats. With the use of immuno-hybridization histochemical methods, we observed c-fos expression in vestibular nuclear neurons responding to transverse movement on the horizontal plane as early as P7 and those to antero-posterior stimulation as early as P9. In the inferior olive (IO) sub nuclei, downstream relays of the vestibulo-olivary pathway, c-fos expression was not observed until P11-13. These findings reveal a difference in critical maturation time between relays of the vestibulo-olivary pathway in the recognition of gravity-related horizontal orientations. Central recognition of vertical orientations also showed different critical maturation time between relay stations. Besides, progressive changes in resting and spatio-temporal neuronal properties as well as the expression of NMDA and AMPA receptor subunits on these otolith-related neurons were observed with age. We examined if otolith information along the horizontal plane is topographically represented in the vestibulo-olivary pathway. Our in vivo electrophysiological results demonstrated that the best response vectors of otolith-related vestibular nuclear neurons in P7 rats were restricted to the interaural axis while those of adults were multi-directional on the horizontal plane. Based on results from both approaches, we found that neurons differing in response vectors were randomly distributed in different vestibular subnuclei without any apparent topographic pattern. Evidence of a gravity-related topographic spatial organization was however observed in sub nuclei of the IO (viz. DMCC, IO) along its rostrocaudal dimension. Although neurons in the caudal IO displayed the adult topographic response pattern to antero-posterior movement as early as P13, those responsive to transverse movement progressively declined from P13 to adulthood. In contrast, neurons in the rostral IO progressively showed the adult pattern to movements in both directions by the third postnatal week. Taken together, these results reveal refinement of topography in the postnatal IO as a model for mapping the establishment of functional synaptic inputs in a plastic stage of growing axons.

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Action of brain-derived neurotrophic factor on function and morphology of visual cortical neurons

T Tsumoto

Abstract

Brain-derived neurotrophic factor (BDNF) is known to play a role in experience-dependent plasticity of the developing visual cortex. For example, BDNF acutely enhances long-term potentiation and blocks long-term depression in the visual cortex of young rats. Such acute actions of BDNF suggested to be mediated mainly through presynaptic mechanisms. A chronic application of BDNF to the visual cortex of kittens is known to expand ocular dominance columns in the cortex. With the method of direct intranuclear injection of plasmid cDNAs of BDNF tagged with green fluorescence protein (GFP) we have demonstrated that BDNF-GFP is transferred from presynaptic axon terminals to postsynaptic neurons in an activity-dependent manner, suggesting the possibility that BDNF also has chronic postsynaptic actions. In this study, however, it was not clear whether endogenous BDNF exerts such a postsynaptic action, because BDNF-GFP is a kind of artificial protein. In a subsequent study, therefore, we tested whether endogenous BDNF has any effect on dendritic morphology of postsynaptic neurons in igchimera culture of visual cortical neurons prepared from two types of transgenic mice, GFP mice and BDNF knockout mice. Neurons derived from the former mice have endogenous BDNF as well as GFP. We found that neurons derived from the latter mice, BDNF (-/-) neurons, have relatively poor dendrites if they were not contacted by GFP-positive terminals, whereas BDNF (-/-) neurons had complex dendritic morphology if they were directly contacted by GFP-positive terminals and thus supplied with endogenous BDNF. These results indicate that endogenous BDNF play a role in development of dendrites of visual cortical neurons in an activity-dependent manner.

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Short-latency category specific neural responses to human faces in macaque inferotemporal cortex

H Esteky

Abstract

In this article I would present evidence to show that timing of the flow of neural signals within the ventral visual stream is a crucial part of the neural code for categorization of faces. We recorded the activity of 554 inferotemporal neurons from two macaque monkeys performing a fixation task. More than 1000 object images including human and non-primate animal faces were presented up to 10 times, each for 105 ms without intervals. About one-third of the cells had selective responses to both human and non-primate animal face categories. The onset latencies were significantly shorter for responses to human faces than those to animal faces ($P<0.0001$, paired t-test, mean difference = 22 ms). In our results a relatively large number of face cells could not differentiate category of faces by rate code alone. Spike timing improves the neural information for discrimination of different stimuli that evoke similar firing rates. Response latency could play a role in organizing information about complex visual scene by perceptual segregation of object categories within the scene and by gating information flow in neural assemblies involved in different aspects of face recognition.

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Early and late consolidation and reconsolidation of memory in the prelimbic cortex

SJ Sara

Abstract

Rats can learn to forage among olfactory cues to associate one with reward in only 3 massed trials. The learning is achieved in less than 10 min and results in a memory trace lasting at least 1wk week. To study the neuro-anatomical circuits involved in the memory formation we used immunoreactivity to the immediate early gene c-fos as a marker for neuronal activity induced by the learning. The prelimbic cortex (PLC) was one of the few regions showing selective increases in c-fos immunoreactivity in trained rats compared to yoked pseudo-trained. In subsequent experiments, injection of an NMDA receptor antagonist directly into PLC immediately after learning induced amnesia 48h later, while similar injections into the hippocampus had no effect. Injections of the beta-adrenergic antagonist, timolol, 2h, but not 5 min after training also produced amnesia. This suggests that PLC is part of a neural circuit activated for an extended period during the post-acquisition consolidation of memory for odor-reward association. We studied reconsolidation processes after reactivation and found that injections of the NMDA receptor antagonist or the beta antagonist could block reconsolidation with similar temporal dynamics as during consolidation when the injections were made into the lateral ventricles, but not into the PLC. Thus, similar cellular mechanisms but different neural circuits appear to be involved in initial consolidation and reconsolidation.

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The emotive brain, the noradrenergic system, and cognition

SJ Sara; S Bouret

Abstract

Motivation and attention can have a profound influence on perception, learning and memory. Neuromodulatory systems, especially the noradrenergic (NE) system, co-vary with psychological states to modulate cortical arousal, influence sensory processing and promote synaptic plasticity. There is even some suggestion that the NE system might facilitate functional recovery after brain damage. Post-synaptic effects of NE in its ubiquitous projection regions have been well studied, but factors controlling activation of NE neurons are less known. The pontine nucleus locus coeruleus (LC) contains the entire population of NE neurons projecting to the forebrain and all cortical and thalamic regions receive NE input. Using single unit recording in freely moving rats, we have been studying neuroanatomical circuits, along with sensory stimuli within their cognitive contexts that control LC firing. Rats are implanted with movable microelectrodes to record activity during a variety of behavioral situations: exploration of novelty in a hole-board, response to tones or odors that predict reward or absence of reward, extinction or reversal of stimulus-reward association. We find that LC-NE neurons respond to novel or salient stimuli, habituate rapidly to respond anew when the stimulus is associated with reward (CS+), particularly in the early stage of learning. Recent experiments suggest that these responses are more related to reward expectancy than to the CS+. Importantly, there is robust LC response to any change in the predictive value of the stimulus, i.e. when new learning must occur. NE released by LC activation will promote the underlying synaptic plasticity.

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Global gene expression analysis using microarray to study differential vulnerability to neurodegeneration

V Ravindranath

Abstract

Neurodegenerative disorders such as Parkinson's disease, motor neuron disease and Alzheimer's disease is characterized by loss of specific cells within certain regions of the brain. One of the most compelling questions is to determine why specific cell populations are vulnerable to neurodegeneration. We addressed this question by studying global gene expression changes using an animal model of neurodegenerative disease. Human ingestion of "chickling peas" from the plant Lathyrus sativus containing an excitatory amino acid, L-?-N-oxalyl amino-L-alanine (L-BOAA) leads to a neurodegenerative disorder, neurolathyrism, a motor neuron disease characterized by spastic paraparesis that targets Betz cells in motor cortex (MC) and the anterior horn cells in the lumbosacral cord (LSC). Our laboratory has earlier shown that L-BOAA toxicity in mice is associated with mitochondrial dysfunction seen as loss of complex I activity in MC and LSC. While MC recovers after an initial insult, sustained injury is seen in LSC. We therefore, profiled global gene expression changes by microarray analyses in mice CNS regions during recovery from L-BOAA toxicity. Mouse cDNA arrays were hybridised with RNA from MC and LSC of control and L-BOAA treated mice. Data was analysed using Array vision and Spot-fire software. In concurrence with earlier results glutaredoxin, a thiol disulfide oxidoreductase was upregulated in both MC and LSC. Glutaredoxin is required for maintenance of complex I function. However, the up-regulation of NADH dehydrogenase (Complex I) was seen only in MC and not in LSC in agreement with the activity of complex I seen earlier. Upregulation of genes related to energy metabolism, antioxidant enzymes, MAP kinases and ubiquitin proteasomal pathways was seen in MC indicating their potential role in the recovery. In contrast in LSC, genes related to MAP kinases and anti-oxidant were not upregulated, instead programmed cell death gene was upregulated. These studies demonstrate the differential responses of CNS regions to a common excitotoxic insult and help identify factors responsible for differential vulnerability of CNS regions often seen in neurodegenerative diseases.

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Time course of dysregulation of calcium homeostasis in acutely isolated CA1 hippocampal pyramidal neurons after pilocarpine-induced Status Epilepticus

M Raza; S Pal; A Rafiq; R Delorenzo

Abstract

Glutamate induces excitotoxic damage to hippocampal pyramidal neurons in Status Epilepticus (SE) and epilepsy. In this study, we investigated time course of dysregulation of calcium homeostasis at various intervals after an episode of SE in acutely isolated CA1hippocampal pyramidal. For this purpose, male Sprague-Dawley rats (200 g) were subjected to pilocarpine-induced SE. The SE was blocked after 3 h with diazepam and MK-801. The CA1 pyramidal neurons were acutely isolated at 24 h, 48 h, 6 days, 10 days and one-month post-SE. The basal intracellular calcium levels [Ca++]i from individual neurons were determined by high speed image flourometery using Fura-2. Rise in neuronal [Ca++]i in response to 30 ?M glutamate (1 min) exposure was captured by Merlin image acquisition software. Calcium decay response curves were then constructed and compared with neurons from control animals at the same time points. The basal calcium levels in acutely isolated CA1 pyramidal neurons post SE were significantly higher (then control at different time points. The neurons studied at 24 h, 48 h, and 6 days were not able to restore calcium levels to normal after glutamate exposure. The neurons at 10 days and 1 month were gradually able to restore calcium levels to pre-glutamate levels. SE induced excitotoxic damage to the hippocampal pyramidal neurons that leads to neuronal death and disruption of calcium homeostasis. This is evidenced by an increase in basal [Ca++]i levels and inability to restore [Ca++]i to pre-glutamate levels after exposure. The neurons recover gradually over a period of 10 days after acute SE. This is the first direct evidence of dysregulation of calcium homeostasis in pyramidal neurons isolated from animals subjected to SE.

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The effect of hypothyroidism on the trigeminal calcitonin gene-related peptide containing motoneurons: an immunohisto-chemical study in late neonatal life

G Behzadi; F Ganji; F Golab

Abstract

CGRP, which coexists with acetylcholine in trigeminal motoneurons, could be one of the regulatory anterograde factors responsible for the enhanced expression of AChR subunits in neuromuscular junction. It has been also shown that low level of thyroid hormone plasma concentration has influence on the transitional development of muscle fibers and related motoneurons through a sever reduction in AChR density. Up today, no previous study has shown specific alteration of CGRP component of motoneurons in developing hypothyroid life. To this purpose, the chemical hypothyroid weaned pups (a %50 reduction in weight gain) were processed for immunohistochemistry, a semiquantitative evaluation was carried out to estimate the proportion of strongly, intermediately, and weakly labeled trigeminal motoneurons, as well as those totally lacking CGRP staining. Hypothyroid pups show a significant reduction in the number of strongly and intermediately CGRP reactive Vth motoneurons (%20 + %36 respectively), notably two times more unlabelled neurons were observed in normal weaned pups. On our preliminary observation on soma size, a three times increase in the number of small CGRP motoreurons reached to a three times decrease in large motoneurons existence. Certainly these obvious alterations emerging from direct or indirect thyroid hormone deficiency on both motoneuronal and muscular relation might be of physiological importance for a potential masticatory system during suckling period of nutrition and growth.

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T-type Ca2+ channels in thalamic sensory gating and affective Disorders

HS Shin

Abstract

Low threshold Ca2+ currents mediated by T-type channels underlie burst spike activities of relay neurons in the thalamus. We have previously reported that knock-out mice for T-type channels show an enhanced nociceptive response to visceral pain, accompanied by an increase in tonic spikes in the absence of burst spikes in thalamic relay neurons. These results raised a possibility that T-type channels are involved in thalamic sensory gating, blocking the relay of the pain signals to the cortex. We have tested this hypothesis by using several different sensory modalities: startle responses to auditory or tactile stimuli, and the response to chronic inflammatory pain. First, the mutant mice showed an enhanced startle response to auditory stimulations. The auditory brainstem recording (ABR) results indicated that the enhanced response was not due to increased input signals from the cochlear to the brainstem. Second, the mutant also displayed an enhanced startle response to tactile stimuli. Third, the mutant showed a selective increase in the late phase response to an intradermal injection of formalin into the hind paw, which known to be controlled by a supraspinal mechanism. Therefore, the mutant showed enhanced responses to sensory inputs of four different modalities, strongly supporting the idea that T-type channels are required in thalamic sensory gating. We suggest that the burst spikes induced by the low threshold Ca2+ currents are the key element in this thalamic function. Furthermore, the mutant mice showed mania-like behaviors, an increased alcohol preference, and resistance to alcohol-induced hypnotic effect. These behavioral consequences will be discussed with regard to the sensory gating failure of the mutant.

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C-terminal fragments of APP: Its neurotoxic mechanisms and involvement in gene transcription

YH Suh

Abstract

Several lines of evidence suggest that some neurotoxicity in AD is due to proteolytic fragments of APP. In this study, we compared the potency of neurotoxicity induced by CT with that of A-beta neurotoxicity and our results showed that various CT peptide fragments (CTFs; CTF99, AICD, CTF31) caused neurotoxicity in cultured cells and primary cortical neurons, induced strong non-selective inward currents in Xenopus oocytes, planar lipid bilayers, Purkinje cells, and blocked the later phase of LTP in rat hippocampus in vivo. And also we showed that CT peptide impaired calcium homeostasis, learning and memory impairment. Our results also suggest that CTF peptides triggered inflammatory reaction through MAPKs- and NF-kB dependent astrocytosis and iNOS induction. In addition, we found that CTF translocated into nucleus binding with Fe65 and CP2 and then affected transcription of genes, such as GSK-3beta, suggesting that neurotoxicities exerted by CTFs may be mediated by interactions of CTF with CP2 and Fe65 in the nucleus and their involvement in transcription of genes including GSK-3beta, leading to increase in tau phosphorylation and their inducing NFT and cell death. However, A-beta didn't enter the nucleus to affect gene transcription. Collectively these results imply that CT peptides themselves can much more greatly damage the neurons both in vitro and in vivo than A-beta and it is thought that both CT and A-beta may participate in the neuronal degeneration in Alzheimer's disease by different mechanisms.

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Neurochemistry and anatomy of the ventral medulla

PM Pilowsky; NS Costin; Q Li; T Lonergan; JM Makeham; T Miyawaki; EA Moon; JJ Neale; JR Padley; V Reja; M Seyedabadi; DS Springell; TA Verner; AK Goodchild

Abstract

The relationship between the anatomy and neurochemistry of neurons in the ventral medulla oblongata in regions that is responsible for cardiovascular, airways, and respiratory regulation was investigated. Microinjections of excitant amino acid (glutamate, 100 nl, 100 mM) were made throughout the ventral medulla in anesthetized rats. Arterial blood pressure, sympathetic nerve activity and phrenic nerve discharge was recorded. Injection sites were marked with albumin-colloidal gold or methylene blue. At the end of recording, brains were removed, fixed, sectioned and stained for gold spots, dopamine-beta-hydroxylase (DBH), phenylethanolamine-N-methyltransferase (PNMT) and serotonin. In the ventrolateral medulla (VLM), several interesting features emerged; first, the entire region corresponding to the pressor and depressor regions of the rostral and caudal VLM (RVLM and CVLM) lay in the area containing PNMT-immunoreactive neurons (C1 cell group). Caudal to the CVLM pressor sites were identified as far caudally as the spino-medullary junction. Activation of the B?tzinger region consistently inhibited phrenic amplitude and frequency whereas activation of the pre B?tzinger increased the basal level of phrenic nerve discharge as well as its frequency and amplitude. The PreBotzinger is found at the same level as the most rostral part of the CVLM. Medial sites in CVLM tended to be excitatory while lateral sites evoked inhibition. In the midline, few sites were found where excitation elicited large (±10%) changes in blood pressure or sympathetic nerve activity. However at one site (11.3 mm caudal to bregma) injection of glutamate elicited phrenic apnea in both paralyzed and non-paralyzed animals. The ventral medulla contains many areas from which responses that affect the control of airways, breathing and circulation exist; these responsive sites are coextensive with populations of neurons that are immunoreactive for many neurochemicals including amines and peptides. Functional studies are required to determine the individual roles played by these different neurotransmitters.

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Tonic and reflex control of the cardio-respiratory system by neurons in the ventral medulla

PM Pilowsky; NS Costin; Q Li; T Lonergan; JM Makeham; T Miyawaki; EA Moon; JJ Neale; JR Padley; V Reja; M Seyedabadi; DS Springell; TA Verner; AK Goodchild

Abstract

To investigate the channels and neurotransmitters in the ventrolateral medulla (VLM) oblongata that are responsible for the maintenance of sympathetic tone and cardio-respiratory reflex regulation. Microinjections of excitant amino acid (glutamate, 100 nl, 100 mM), calcium channel blockers, agonists and antagonists were made throughout the VLM in anaesthetized rats. Arterial blood pressure, sympathetic nerve activity and phrenic nerve discharge was recorded. Responses to activation of the baroreceptor, somatosympathetic, and chemoreceptor reflexes were recorded. Injection sites were marked with albumin-colloidal gold or methylene blue. At the end of recording, brains were removed, fixed, sectioned and stained for gold spots, and counterstained to localize injection sites. Bilateral microinjection of calcium channel antagonists into the rostral VLM (RVLM) had little effect on arterial blood pressure with the exception of Ni++ (an agent that blocks low voltage T-type calcium channels). Ni++ caused a profound fall in arterial blood pressure and sympathetic nerve activity, but did not affect reflex function. By contrast the high voltage channel (P, Q, L, N) blockers increased blood pressure and sympathetic activity following injection into the caudal VLM (CVLM). The agents applied were 8-OHDPAT; 5HT1a agonist, DAMGO; mu opioid agonist, DPDPE; delta opioid agonist and sar9-substance P; substance P agonist. In addition, the effect of changing the inhaled concentration of carbon dioxide was tested. In all cases except injection DAMGO, there was an attenuation of the somato-sympathetic reflex. DAMGO attenuated the baroreceptor reflex. None of these agents affected the chemoreceptor reflex. The data suggest that tonic maintenance of sympathetic activity is mediated, at least in part, by a Ni++ sensitive conductance in RVLM and that the neurochemical heterogeneity found within the inputs to sympathoexcitatory and inhibitory neurons in the RVLM has functional significance, with different neurotransmitters affecting cardiorespiratory reflexes differentially. Further functional studies are required to determine the precise connectivity between different neuronal populations that are subserving different functions in the RVLM.

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Syntaxin 1 is expressed in the trout saccular hair cells: RT-PCR and immunocytochemical observations

KM Khan; NA Ramakrishnan; MJ Drescher; JS Hatfield; DG Drescher

Abstract

Syntaxin is one of several proteins that may be involved in the docking of synaptic vesicles, synaptic vesicle recycling, and non-synaptic membrane trafficking. Presence of syntaxin has been reported in rat auditory and vestibular end organs. In the current study, we have examined the expression of message for syntaxin 1 in hair cells of the sacculus of the rainbow trout, Oncorhynchus mykiss, with RT-PCR using degenerate primers based on the sequence of zebrafish syntaxin. A PCR product of predicted size, 540 bp, yielded sequence with 94% amino acid identity to mouse, rat and human syntaxin and 96% identity to zebrafish syntaxin. An anti-syntaxin 1 primary antibody (S1172 Sigma) recognized a protein of 37 kDa, from the trout brain extract and the saccular hair cell homogenate in western blots. At the light microscopic level, immunoreactivity for anti-syntaxin 1 was observed within the sensory epithelium, in efferent fibers below the hair cells running parallel to the longitudinal axis of the saccule. In addition, sub- and supranuclear immunostaining was observed within select hair cells and immunoreactivity was concentrated at the base of stereocilliary arrays throughout. Immunogold electron microscopy further revealed that immunoreactivity was associated with the synaptic bodies and the basolateral and supranuclear regions of hair cells along with that present in the cuticular plate/stereocilliary regions of hair cells and efferent endings. These results suggest that syntaxin 1, expressed in the trout saccule, is associated not only with the docking of synaptic vesicles, but may be involved in vesicle recycling and in recycling of membrane.

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Oxidative stress and tardive dyskinesia: role of natural antioxidants

SK Kulkarni; PS Naidu

Abstract

Schizophrenia is a devastating psychiatric disorder that affects 1% of population worldwide. Neuroleptics are the major class of drugs used in the treatment of schizophrenia. Neuroleptics are associated with wide variety of extrapyramidal side effects such as akathesia, dystonia, neuroleptic malignant syndrome, Parkinsonism and tardive dyskinesia. tardive dyskinesia is a complex hyperkinetic syndrome consisting of choreiform, athetoid or rhythmic abnormal involuntary movements. The face, mouth and tongue are most frequently involved (orofacial type), but a variety of less frequent motor abnormalities of the upper and lower limbs and of the trunk may also occur. Estimates of the prevalence rate of TD in patients receiving neuroleptics range from 0.5%-70% with an average prevalence rate of 24%. Despite much research, the pathogenesis of TD remains elusive. So far various neurochemical hypothesis have been proposed for the development of TD. Those include dopaminergic hypersensitivity, disturbed balance between dopamine and cholinergic systems, dysfunctions of striatonigral GABAergic neurons and excitotoxicity. Similarly, different suppressive agents have been tried with limited success. Oxidative stress and products of lipid peroxidation are implicated in the pathophysiology of various neurological disorders including TD. Administration of single or chronic dose of haloperidol to animal led to decrease endogenous antioxidant, reduced glutathione levels in the striatum indicating generation of oxidative stress by the drug. Chronic haloperidol treatment also decreased antioxidant defense enzymes superoxide dismutase (SOD) and catalase levels. Natural antioxidant like vitamin E, melatonin, quercetin and Withania somnifera have shown to be beneficial in ameliorating tardive dyskinesia symptoms in animal models of TD, further supporting the pivotal role of oxidative stress in the pathophysiology of TD. A role of natural antioxidants has been implicated in the management of neurodegenerative disorders including tardive dyskinesia.

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Modeling storage and retrieval of memories in the brain

V Srivastava; SF Edwards

Abstract

We have proposed a neural network model that stores the incoming information after orthogonalizing it in the same manner as vectors are orthogonalized. The scheme enables the brain to compare a new informational system with those in the memory and store its similarities and differences with the old memories in an economical manner. This allows the brain to have an enormous capacity and yet the retrieval can be very accurate and efficient. Examples of how the idea is applied to the acquisition of words in the mental lexicon, and discrimination of contexts of motor actions by the cerebellum will be discussed. We will also describe an extension of the model to study how the memory might cope with trauma.

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Mechanisms of the effects of experimental diabetes on the development of tolerance to morphine antinociception in rat

M Jorjani; KH Joharchi

Abstract

Opiates, chiefly morphine, are commonly known as the best drugs for relief of pain, but due to development of tolerance and the physical or mental dependency the use of these drugs are restricted. Several neuroendocrine complications including diabetes change the antinociception of morphine and the development of tolerance and dependency to this drug. The withdrawal signs in diabetic rats significantly reduce in comparing to non-diabetic animals. The exact mechanism of these effects has not been known yet. Due to known role of nitric oxide in development of morphine tolerance this study was designed to evaluate the role of nitric oxide in the effects of diabetes on morphine tolerance. In this study the alloxan-induced diabetic male rats are used, and the changes in nitric oxide level is measured by spectrophotometric method. These changes are also measured in tolerant animals after the injection of repeated doses of morphine sulfate and by the use of inhibitors or producers of NO syntheses we are trying to clarify the role of this neurotransmitter in diabetic state.

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There is much scope for explorative learning and long-term memory in active teaching process

N Rassaian

Abstract

Introducing RCTM (Research-Centered Teaching Method) by the author showed the method provokes enthusiasm and anxiety, correlated with cognition: Cognition = (0.27) enthusiasm– (0.15) not interested + (0.08) anxiety. “The broaden-and-build theory” (Fredrickson, 2001) posits that positive emotions are useful in several ways. By linking this theory and emotional intelligence theory, it becomes apparent that the knowledge and use of positive emotions constitute an important skill set for effective functioning.” Renal Physiology was actively taught to 70 medical students, instructed to answer thoughtful questions asked in class. Reinforcing them by few grades and assuring them to be skillful physicians in future. It was emphasized that attending the Acid-Base Balance session is of great importance, due to the complexity and its management. Before the midterm exam, a questionnaire was distributed, asking to write down the topic they recall most precisely, and choosing the reasons. Acid-Base Balance was the mentioned topic, and teaching method was the most correlated factor among the reasons of their recollection ($r<0.773$). The normalized grades were compared: Student’s Question>Taxonomy3>Research Design and Taxonomy3 (Acid-Base Balance)>Taxonomy2 ~ Research Design ($P<0.02$). In the final exam the grades of Acid-Base Balance questions were raised ($P<0.015$). The results are possibly due to the context of reinforcing the students’ positive emotions.

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Fear and anxiety behavior in rats

P Rostami; MR Zarrindast; M Sadeghi Hariri; A Hosseini

Abstract

Fear can be considered as a functional defense behavior to protect living beings against dangerous situation. In our studies we have investigated the fear behavior in rats in elevated plus maze .The increase in two parameters percent of open arm entries (%OAE) and percent of time spent in the open arms (% OAT) and decrease in the percent of time spent in closed arm (%CAT) was considered as fear and anxiety effects .In our previous study we measured these parameters in control and prenatal stressed male rats before and after treating by different doses of testosterone enantate .Our data showed that prenatal stress increased fear and anxiety behavior compared with control , and testosterone treatment decreased this behavior dose dependently which is probably due to the inhibitory effects of testosterone on HPA and on some brain nuclei . Several central sites have been implicated in the modulation of fear and anxiety. Some researchers suggested the probable effects of GABA receptors in this modulation. we studied the effects of ICV and IP injection of GABAA and GABAB receptor agonists and antagonists on fear and anxiety. ICV injection of different doses of muscimol, a GABAA receptor agonist, induces anxiety profile in rats. Neither ICV nor IP injection of GABAB receptor agonist baclofen altered the three parameters. ICV injection of GABAB receptor antagonist CGP 35348 increased % OAE and % OAT and decreased % CAT. The response induced by injection of muscimol (0.5 ?g/rat ICV) or administration of CGP35348 (10 ?g/rat ICV) was reduced by i.c.v. (1, 2, and 4 ?g/rat) or IP (0.25, 0.5, and 0.75 mg/kg) injection of the GABAA receptor antagonist bicuculline, but the effect of CGP35348 on %CAT was not significantly altered by IP administration of bicuculline.IP administration of bicuculline (but not ICV) by itself reduced both %OAE and %OAT, but did not alter %CAT. None of the drugs altered the locomotor activity of the animals The current findings support our hypothesis that the anxiolytic effects of GABAB antagonist are mediated by autoreceptor blockade-induced release of endogenous GABA, which in turn activates postsynaptic GABAA receptors. The effects of morphine on fear and anxiety behavior are being investigated now.

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Activation of inwardly-rectifying k+ channels in hypothalamic POMC neurons: role in integrating synaptic and metabolic input

N Ibrahim; JL Smart; M Rubinstein; MJ Low; MJ Kelly

Abstract

Hypothalamic proopiomelanocortin (POMC) neurons are critical for controlling homeostatic functions in mammals. We used a transgenic mouse model in which the POMC neurons were labeled with enhanced-green fluorescent protein (EGFP) to perform visualized, whole-cell patch recordings from pre-pubertal female hypothalamic slices. The mouse POMC-EGFP neurons expressed the same endogenous conductance (IA and Ih) that has been described for guinea pig POMC neurons. In addition, the selective opioid receptor agonist DAMGO induced an outward current (maximum of 12.8 ± 1.2 pA), which reversed at EK+, in the majority (85%) of POMC neurons with an EC50 of 102 nM. This response was blocked by the opioid receptor antagonist naloxone with a Ki of 3.1 nM. In addition, the GABAB agonist baclofen (40 ?M) caused an outward current (21.6 ± 4.0 pA) that reversed at E K+ in these same neurons. The KATP channel opener diazoxide also induced an outward K+ current (maximum of 18.7 ± 2.2 pA) in the majority (92%) of POMC neurons with an EC50 of 61 ?M. The response to diazoxide was blocked by the sulfonylurea tolbutamide, indicating that the POMC neurons express both Kir6.2 and SUR1 channel subunits, which was verified using single cell RT-PCR. This pharmacological and molecular profile suggested that POMC neurons might be sensitive to metabolic inhibition, and indeed, we found that their firing rate varied with changes in glucose concentrations. Therefore, it appears that POMC neurons may function as an integrator of metabolic cues and synaptic input for controlling homeostasis in the mammal.

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Neurodynamic control of the heart of freely moving spiny lobster (Panulirus japonicus)

T Yazawa; K Tanaka; T Katsuyama

Abstract

The heart of the crustaceans has its own pacemaker neurons inside the heart, which are composed of 9 neurons. The neurons receive innervations of only three kinds of axons originated from the central nervous system; one pair of inhibitory and two pairs of acceleratory axons. Thus, in terms of the neural cardiac control from higher center, this system may have much more simplistic operation comparing to our hearts, because those vertebrate hearts receive hundreds of central nerve fibers so called the autonomic nervous system; sympathetic and parasympathetic nerves. This simplicity of the crustacean hearts may have a great advantage for physiological and physical understanding for the essential mechanism of the heartbeat control. Thus, we performed time series analysis of ECG data of Japanese spiny lobster, Panulirus japonicus. The ECGs were recorded in three conditions of the heart. The isolated hearts, which are obviously disconnected from the center but are well maintained at a steady condition, and the intact hearts, heartbeats of which were recorded at either a freely moving condition or a stressful condition receiving severe immobilizing stress. DFA (Detrended Fluctuation Analysis) revealed a difference of operation of the CNS. The three conditions of the hearts were found to have different scaling properties. Focusing on the difference between the scaling exponents of the isolated heart and the stressful heart, both of which had, intriguingly, almost identical power spectra, we will discuss the possibility to index the CNS operation by means of the scaling exponents.

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Relationship between neonatal testosterone and 5-hydroxytryptamine (5 HT) in controlling the pattern of LH release in adult rats

A Siddiqui; JF Murray; CA Wilson

Abstract

Relationship between neonatal testosterone and 5-hydroxytryptamine (5-HT) in controlling the pattern of LH release in adult rats was investigated in this study. Hypothalamic 5-HT concentrations are transiently lower in male as compared to female Wistar rats in the second week post-partum (pp). It has also been shown that pharmacologically potentiating 5-HT activity during this period feminizes certain aspects of sexually differentiated behaviors in adult males and androgenized females. In order to investigate whether neonatal testosterone and 5-HT interact to influence physiological and morphological brain sexual differences, intact females androgenized females and intact males were treated with the 5HT2 against (-)[2,5 dimethoxy-4-iodophenyl]-2-amino propane HCl [(-)] (DOI), over days 8-16 pp in androgenized females (250 mg testosterone propionate (TP), day 2 pp). DOI prevented the delay in vaginal opening, but did not prevent the androgen-induced constant oestrous in females treated with 100 ng TP at day 2 pp. DOI overcome the neonatal androgen effect in suppressing the positive feedback of ovarian steroids in a few males and androgenized females. DOI had a feminizing effect on the volume of the anteroventral periventricular nucleus (normally smaller in males, by significantly increasing its volume in male and androgenized females. It also had a significant antagonistic effect on the testosterone-induced increase in the volume of the sexually dimorphic nucleus of the preoptic area in males and androgenized females. These findings support the evidence that raised 5-HT activity in the second week of life antagonizes the masculinizng effect of neonatal testosterone.

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Actions and release characteristics of secretin in the rat cerebellum

WH Yung

Abstract

Secretin, a peptide hormone of the gastrointestinal system, has been implicated in the etiology of autism. Our laboratory previously demonstrated the expression of secretin and its receptors in specific central neurons, and found for the first time that secretin is neuroactive in the cerebellum. We showed that bath application of secretin facilitated the release of GABA from terminals of basket cells. In the present report, we further characterize the actions of secretin in the cerebellum and provide evidence for the endogenous release of secretin in this brain area. First, by means of whole-cell patch-clamp recordings from Purkinje neurons in acutely prepared rat brain slices, we discovered that the facilitatory action of secretin on inhibitory inputs is partly dependent on the release of endogenous glutamate. In the presence of CNQX, an AMPA/kainate receptor antagonist, the facilitatory effect of secretin on GABA release was significantly reduced. Consistent with this idea, application of AMPA facilitates GABA release from inhibitory terminals, in a CNQX-sensitive manner. These data indicate that a direct and an indirect pathway mediate the action of secretin. Secondly, secretin is also active at the synapse between Purkinje neuron terminals and deep nuclei neurons. Thus, in voltage-clamped deep nuclei neurons, bath application of a low concentration of secretin increased the frequency but not the amplitude of miniature inhibitory post-ynaptic currents (mIPSCs). Therefore, secretin may act on presynaptic secretin receptors on the terminals of Purkinje cells. These data explain the fact that Purkinje cells express secretin receptor mRNA but do not respond directly to secretin, and indicate that secretin could act at multiple levels in the cerebellar circuits. Finally, to show that secretin is released endogenously, blocks of freshly dissected cerebella were challenged with 40 mM of KCl, or KCl in the presence of tetrodotoxin (TTX) or cadmium. The amount of secretin released was measured by commercially available EIA kits. Incubation with KCl almost doubled the rate of secretin release. This KCl-induced release was sensitive to TTX and cadmium suggesting the involvement of voltage-gated sodium and calcium channels. On the whole, these data give further and more solid evidence for the role of secretin as a neuropeptide in the central nervous system, and provide the rationale to further explore the relationship between secretin, cerebellum, and autism.

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Cellular SRC kinases and dsRNA dependent protein kinase (PKR) play key role in intracellular viral (CVB3) replication

N Maghsoudi; M Zeinoddini

Abstract

SRC kinases and PKR are intracellular protein kinases, which play key roles in intracellular viral replication. In this research, the effect of SRC kinase inhibition and PKR activation and inhibition on replication of coxsakievirus (CVB3), an entrovirus of the family picornaviridae – causative agents of fatal myocarditis, was studied. Vero and Hela cells were cultured and infected with CVB3 in the presence of herbimycin A (specific tyrosin kinase inhibitor), added before and after infection in various time interval, gamma interferon (PKR activator) and 2-aminopurine (PKR inhibitor). Viral replication was monitored by molecular methods (RT-PCR) as well as plaque forming unit (PFU) count using a set of forward and reverse primer for viral gene, which could detect positive and negative strand mRNA of the virus, as well as PFU count. It was observed that SRC kinase inhibitor, when added before addition of CVB3 to the medium, can inhibit intracellular viral replication, whereas if herbimycin is added after infection, had no effect on viral proliferation. Using set of primers for conserved region of PKR gene, it was possible to observe the presence of PKR mRNA in all cultures either interferon treated, or 2Ap treated, or control. PFU studies showed that ?-INF has inhibitory effect on viral replication, where as 2Ap treatment enhanced viral replication. Relevance of the results towards development of new molecules for viral infection treatment will be discussed.

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Interaction of NMDA and opioid receptors on thermal hyperalgesia and mechanical allodynia in two models of neuropathic pain

H Manaheji; Hamidi GhA

Abstract

The use of multiple loose ligations of the rat sciatic nerve has been proposed as a model for the study of allodynia and hyperalgesia. This pain hypersensitivity results from both an increase in the peripheral and central sensitization. The evidence indicating that the development of neuropathic thermal hyperalgesia and mechanical allodynia requires activation of spinal cord NMDA receptors. NMDA antagonists are capable of blocking central sensitization associated with persistent noxious stimulation in experimental mononeuropathy and opioid tolerance. In addition, it has been reported that treatment with MK801 prevents the thermal hyperalgesia and mechanical allodynia. In the present study we attempted to find the interaction of NMAD receptor antagonist MK801 and opioid receptor agonist morphine in two nerve injury models of neuropathic pain in attenuation of hyperalgesia and allodynia. The experiments were carried out on male Sprauge-Dawley rats weighing 200-250 g. Under anesthesia, the sciatic nerve were exposed unilaterally and ligated loosely with 4-0 chromic gut. In SNI procedure axotomy and ligation of tibial and common peroneal nerves, leaving the sural nerve intact. We observed that pretreatment with NMDA recepor antagonist MK801 (0.3 mg/kg) does significantly attenuated thermal hyperalgesia and mechanical allodynia.in both SNI and CCI. Preemptive injection of morphine (8 mg/kg) had to a much lesser than MK801 in hyperalgesia and allodynia in SNI and CCI models. Co-administration of morphine and MK801 could eliminate the hyperesthetic and allodynia state following sciatic nerve injury. In conclusion, the present study shows that the opioids alone could actually contribute to the development of neuronal plastic changes via interactions with NMDA receptors. Then morphine during the pre-operative period in rats failed to affect the development of post-operative hyperalgesia and allodynia.

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Crossing interaction of adrenergic, cholinergic, histaminergic and opioidergic systems on water intake in adult male rats

S Oryan; M Eidi; A Eidi; B Kohanrooz; L Sepehrara

Abstract

Several lines of evidence have indicated that many nuclei in the brain including preoptic nucleus, AV3V, subfornical organ, septal area and lateral hypothalamus are the targets of efferents from chemo-sensitive and pressure-sensitive systems. These areas may concern with regulation of fluid homeostasis. In the present study, intracerebroventricular injections were carried out in all experiments. After a 24 h deprivation of water, the volume of consumed water was measured for 1 h. Administration of pilocarpine (0.5-1 ?g/rat), prazocin (2 ?g/rat), histamine (40-80 ?g/rat) and naloxone (0.5-1 ?g/rat) increased, while scopolamine (5-10 ?g/rat), phenylepherine (30 ?g/rat), morphine (2.5 ?g/rat), pyrilamine (25-50 ?g/rat) and ranitidine (10-20 ?g/rat) decreased water intake in isolated rats. The activation of muscarinic cholinoceptors by pilocarpine attenuated the inhibitory effect induced by phenylepherine. Blockade of muscarinic cholinoceptors did not change the phenylepherine-induced response. Pretreatment of rats with prazocin decreased the pilocarpine-induced response. Pharmacological blockade of muscarinic cholinoceptors by scopolamine decreased the prazocin –induced effect on water intake. Blockade of histamine H1 and H2 receptors attenuated the histamine-induced response. Furthermore, pyrilamine, but not ranitidine, increased the inhibitory effect induced by morphine. Pharmacological blockade of H1 and H2 receptors decreased the naloxone-induced effect on water intake. It is concluded that the histaminergic system may have a close interaction with morphine and naloxone regarding drinking behavior. Also, muscarinic cholinoceptors and adrenoceptors may have an interactive effect in this respect.

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The effects of imidazoline compounds on nociception in animal pain model

M Sabetkasaei

Abstract

The discovery of imidazoline ligands has opened up a new field of study. The investigation of imidazoline actions independent of adrenoceptors started in the mid 1980s. Imidazoline receptors are classified in several subtypes, I1, I2 and I3 binding sites. Although imidazoline sites have been the subjects of research for several years, but there is still controversy about their actions especially their role in nociception. Therefore, during recent years, we have based our investigations on the study of antinociceptive effects of imidazoline and guanidinium compounds in different animal pain models. Visceral pain is one of the most common form of pain which is poorly understood. In this regard, we studied the influence of imidazoline/guanidinium compounds such as clonidine and guanfacine on visceral pain in the presence or absence of yohimbine and benazoline. We conclude that both imidazoline (I2) and ?2-adrenergic receptors may play a role in producing analgesia in visceral pain, although data remain fragmentary whether imidazoline (I2) receptors enhance or suppress nociception. Chronic pain is one of the most frequent reasons for patients to seek medical care. Since the involvement of imidazoline binding sites in the modulation of pain is still in debate, therefore, in our research, we investigated the anti-nociceptive effects of imidazoline compounds such as clonidine and rilmenidine in formalin test in rats. Formalin test is an animal model of chronic pain, which reflects both phasic and tonic responses to a subcutaneous injection of dilute formalin into the rodents paw. Finally, it may be concluded that clonidine and rilmenidine produce their anti-nociceptive activity via ?2–adreneoceptors rather than imidazoline (I1) receptors. However, the role of imidazoline (I1) binding sites in producing analgesia has not been well established yet.

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Artificial neural networks: applications in pain physiology

H Berenji; M Parviz; SH Gharibzadeh

Abstract

Artificial neural networks (ANNs) are intelligent systems that have successfully been used for prediction in different medical fields. In this study, the capability of ANN in predicting body behavior in pain-producing situations is evaluated. A three-layer back-propagation ANN is designed using MATLAB software. The inputs include the magnitude of stimulation in pain fibers, touch fibers and central anti-nociceptive fibers and output is the level of perceived pain. In other words, we modeled the gate control theory of pain. Important features of pain process were chosen and defined in 8 features and then were applied to the ANN. We examined the ANN to ensure that it can model the real situations. The result was acceptable (errors below 1%). Our model can be used for interpolation and extrapolation of pain-related data. This model is a useful tool in pain experiments to predict the behavior of the organism.

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Dynamics of alaninaminotransferase activity in subcellular fractions of different areas of brain cortex and hypothalamus in postnatal ontogenesis under protein-free feeding regime and after its withdrawal

TM Agaev; ER Salayeva

Abstract

Total and specific activities of alaninaminotransferase (Al-AT) were determined in general tissues, mitochondrial and cytosol fractions of visual, orbital, motor, limbic areas of brain cortex and hypothalamus of three-month old and one-year old rats under 10-20 days and 30 days protein deprivation and under recovery of normal food regime during the same terms. It was found out that Al-AT activities under impairment of feeding regime depend on several factors including morphofunctional peculiarities of the brain structure, terms of protein-free feeding, studied cellular substrates and animal age. On the basis of the experimental data the possible ways of enzyme involvement in the processes of biochemical adaptation occurring in the brain in nutritional protein deficiency were analyzed. The adaptive enzyme functions within the cell compartments are directed toward accomplishment of complex compensatory and adaptive reactions. On the level of mitochondrial fractions, the enzyme is involved into the complex of compensatory reactions via glucose-alanin cycle to support neurons with energetic substrates. In cytosol, the enzyme adaptive functions lie in maintaining of structural and synaptic cell plasticity on account of transamination reaction. The mentioned mechanism, according to the experimental results, is move developed in three-month old animals than in one-year ones.

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Adaptive changes of redox status in rat brain tissues due to decimeter microwave irradiation

LF Ismailova; AB Shabanova; Yusifov EYu; AM Hajiyev

Abstract

Electromagnetic waves affect living organisms and it is of great interest for wide interaction of new sources with a diversity of frequencies and powers to life of people. In the last few years, many authors have proposed that the biological effect of electromagnetic fields in both the high-frequency and low-frequency ranges are connected with oxidative processes in tissues. Studying the changes in redox balance, we could examine this idea. In the present work, we tried to elucidate how oxidant/antioxidant status in brain tissues responds to chronic irradiation by decimeter microwaves in rats. For this purpose, 460 MHz microwaves were used for whole body irradiation of rats at the power densities less than 0,1 mW/cm2 that are considered to be athermal. The levels of total thiols and easily accessible thiols, activity rate of antioxidant enzymes gluthatione reductase, superoxide dismutase, gluthatione peroxidase and thioredoxin reductase were measured in the tissues of visual cortex and hypothalamus of rats exposed to long-term irradiation during 1-4 weeks with a 20 min exposure/day. As an oxidant production index, we used lipid peroxidation product–malondialdehyde. Measurements were carried out in homogenates of tissues and particularly in mitochondrial fraction. The results showed that specific absorption rate (SAR) of 15 mW/kg displayed distinct changes of redox status depending on exposure time. Both ascorbate-dependent and NADPH-dependent induced lipid peroxidation experiments demonstrated the contributions of mitochondrial ETC and NADPH oxidase to oxidant production with following peroxide accumulation during microwave exposure. The studies showed that the changes in redox status of hypothalamus tend to return practically to control level after a 4-week irradiation, but visual cortex redox status remains sufficiently changed. More detailed studies are required to be conducted to determine the physicochemical mechanisms underlying the biological effects of decimeter irradiation.

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Impact of hypokinesia on dynamics of formation of evoked potentials in sensorimotor cortex in early postnatal ontogenesis

A Gaziev

Abstract

The analysis of evoked potentials of sensorimotor cortex in response to electrical stimulation of n. ischiadicus shows that the extremal factor hypokinesia has a negative impact on the dynamics of formation of amplitudal and temporal characteristics since eyes openning in 2-weeks old rats. The most vulnerable process to the impact of hypokinesia is the first-positive phase, which disappears in this case and the inhibitory effect of ascending excitation on the brain cortex is observed. This indicates that 2-weeks old rat lies in its most critical period and the onset stage of functional changes that will be eyes opening and formation of motor activity. Hypokinesia has a negative impact not only on the characteristics of the evoked potentials, but as well on biometric values (weight, length) and on the eyes-opening process. Therefore, the presented experimental data showed the impact of hypokinesia on the brain maturation, on the processes of excitation in the brain cortex, and in this way demonstrate the heterochronic morpho-functional maturation of its structures.

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A phase I/II clinical trial for adult recurrent glioma using 131i-tm-601, an iodinated peptide derived from scorpion venom

MB Khazaeli; VL Alvarez

Abstract

131I-TM-601 is a 36-amino acid peptide, called chlorotoxin (TM-601), derived from scorpion venom labeled with I-131. TM-601 binds a receptor on the surface of tumor cells, and not on normal cells. A single dose of 131I-TM-601 administered intracranially to human xenografted mouse models of glioma has been shown to extend survival up to 269% in multiple studies. 131I-TM-601 is in a multi-center phase I/II clinical trial for adult recurrent glioma. A total of 18 patients, six in each of three dosing panels (10 mCi/250 g 10 mCi/500 g, and 10 mCi/1000 g) were studied. Patients have a ventricular access device (VAD) placed in the tumor cavity at the time of resection. 14 to 28 days later, a single dose (2 ml) of 131I-TM-601is given via the VAD. Gamma, SPECT, and MRI/CT scans are taken over a six to eight day period. The primary endpoints are safety, toxicity, dosimetry, biodistribution and imaging and the secondary endpoints are tumor response and survival. Enrollment is complete. No dose limiting toxicities directly related to drug administration has been observed. Specific targeting of residual tumor in the cavity has been demonstrated. One to 10% of the radiation delivered specifically binds the tumor cavity. Biodistribution and dosimetry data indicate that targeting of 131I-TM-601 to the tumor cavity is very high and to critical organs very low. By 72 hours, 90% of the radiation has been eliminated from the body. Patients were followed for six months or until death. Studies were approved by Institutional Review Board under an IND from FDA. A single dose of 131I-TM-601appears to be safe, well tolerated, and to target tumor cells with high specificity. A phase II clinical trial using multiple doses of 131I-TM-601 with higher dose of radiation is planned.

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The septum modulates REM sleep-related penile erections in rats

KK Gulia; HN Mallick; VM Kumar

Abstract

Rapid eye movement sleep in males is characterized by penile erection along with EEG desynchronization, muscle atonia, ponto-geniculo-occipital waves, and rapid eye movements (REM). The central neural mechanisms regulating sleep related erections (SREs) are not known. Recently, the lateral preoptic area has been shown to contribute in sleep-related erectile mechanisms. The present study was conducted to examine the involvement of the septum for these SREs. Experiments were carried out in adult male Wistar rats (180-250 g) and electrodes were implanted for recording sleep parameters i.e. EEG, EMG and EOG under sodium pentobarbital anesthesia. Bulbospongiosus (BS) muscle EMG was recorded for monitoring penile activity. All the recording electrodes were connected to an IC socket, and the whole assembly was fixed to the skull with dental cement. After post-operative recovery, all the parameters were recorded for 24 hrs. The medial and lateral septi were bilaterally lesioned using NMDA (5 µg/ 0.2 µl), in two different sets of rats, and recordings were taken on days 6 and 15 after the lesion. At the completion of the experiments, the lesion sites were verified histologically. Lesion at the lateral septum produced a significant decrease in the BS muscle activity during REM as compared to pre-lesion control. The rat with lesion in the medial septum showed increase in the REM associated BS muscle activity after the lesion. These findings suggest that the septum plays important role in REM sleep related penile erection.

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Electrophysiological, pharmacological and behavioral studies of different physiological roles of the nucleus paragigantocellularis

S Semnanian

Abstract

The nucleus paragigantocellularis (PGI) is located in the rostral ventrolateral medulla and has noticeable connections with some other brain nuclei, such as locus ceruleus, nucleus raphe magnus and periaqueductal gray. In rats it is 3 mm in rostrocaudal and 1 mm in mediolateral and 1 mm in the dorsolateral aspect. Anatomically and functionally, PGI has been divided into two subnuclei, retrofacial and juxtafacial paragigantocellularis. PGI is consisted of small- (8-20 μm) and medium- (21-35 μm) sized neurons, which are adrenergic, serotonergic, and peptidergic. More recent cytochemical studies have also shown enkephalinergic, cholinergic and glutaminergic neurons in this brain structure. PGI's functions involve the regulation of blood pressure, heart rate, respiration, and pain sensation and control. In these series of studies, which has been conducted in our lab, we have evaluated different roles of PGI in blood pressure, nociception, and morphine tolerance and dependence through electrophysiological recordings, specific chemical and electrical lesioning techniques, pharmacological approaches and behavioral techniques.

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Pentylenetetrazol-kindling induced synaptic plasticity in the CA1 region of rat hippocampus

MR Palizvan; Y Fathollahi

Abstract

The impact of pentylenetetrazol-induced kindling on the effectiveness of theta pattern primed-bursts (PBs) for the induction of long-term potentiation (LTP) of field excitatory postsynaptic potentials (fEPSP) and population spikes (PS) were investigated in hippocampal CA1 of pentylenetetrazol-kindled rats in vivo. The results showed that shortly after kindling, control animals had normal LTP of fEPSP slope and PS amplitude in response to PBs, but kindled rats lack LTP of fEPSP slope and PBs induced LTP of PS amplitude in most of kindled animals. At days 30-33 after kindling, PB potentiation was not observed in the stratum radiatum of kindled animals but PBs induced LTP of PS amplitude, which was significantly greater than that of control animals. The effect is compatible with the hypothesis, which postulates kindling-associated functional deficit in hippocampus, especially CA1, as an explanation for the behavioral deficits seen with the kindling model of epilepsy.

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Role of adenosine receptors and protein phosphatases in the reversal of pentylenetetrazol-induced potentiation phenomenon by theta pulse stimulation in the CA1 region of rat hippocampal slices

A Omrani; Y Fathollahi

Abstract

The effect of theta pulse stimulation (TPS) on pentylenetetrazol (PTZ)-induced long-term potentiation of population spikes (PS) was studied in the hippocampal CA1 in vitro. A transient PTZ application produced a long-lasting enhancement of PS amplitude. A 3-min episode of TPS delivered at a higher intensity produced complete reversal of the PTZ potentiation when delivered during the last minutes of PTZ application. Prior application of high-intensity TPS also decreased the amount of PTZ potentiation, whereas it had no long-lasting effect on baseline synaptic responses. High-intensity TPS induced reversal was blocked by adenosine A1 receptor antagonist and, furthermore, was reduced by protein phosphatase 1 inhibitor. The results suggest that mechanism of PTZ-induced LTP reversal involves activation of adenosine receptors and protein phosphatases.

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Evaluation of anticonvulsant activity of N-(p-aminobenzoyl)-1,2,3,4-tetrahydro-4-methylquinoline

S Rodpaewpaln; C Patarapanich; MH Tantisira; B Tantisira

Abstract

Anticonvulsant activity of N-(p-aminobenzoyl)-1,2,3,4-tetrahydro-4-methylquinoline (NMQ), a newly synthesized ameltolide analogue, was investigated with regards to efficacy, lethality and neurotoxicity in mice. In vivo microdialysis experiments on freely moving rats were also performed in search for the possible effect of NMQ on cortical amino acid neurotransmitters. Similar to ameltolide, intraperitoneally given NMQ exerted it protective effect exclusively in maximal electroshock seizure (MES), but not in pentylenetetrazole test. However, in comparison with ameltolide which exhibited the median effective dose (ED50) of 1.08 mg/kg body weight (B.W.), NMQ was less effective (ED50 = 77.62 mg/kg B.W.). Based on the finding that no lethality was observed in mice being given NMQ up to 1000 mg/kg B.W. while the median lethality dose (LD50) of ameltolide was observed at 63 mg/kg B.W., NMQ appeared to be superior to ameltolide with regards to safety. The median neurotoxic dose (TD50) from the rotarod test was found to be 320 and 9 mg/kg B.W. for NMQ and ameltolide respectively, resulting in lower protective index (PI=TD50/ED50) of NMQ (4.12) than that of ameltolide (8.33). Thus, it could be anticipated that therapeutic dose of NMQ which seemed to be safe would produce more pronounced motor impairment than that of ameltolide. In microdialysis study on freely moving rats, it was found out that neither NMQ nor ameltolide was able to exert significant changes on the level of cortical brain amino acid neurotransmitters, namely, aspartate, glutamate, glycine and GABA. Therefore, some mechanisms other than alteration in brain amino acid neurotransmitters should underlie anticonvulsant activity exhibited by these two compounds in MES test. Furthermore, based on similarity in pharmacological screening profile of NMQ and ameltolide observed in the present study, it is suggestive that these two compounds may possess rather similar mechanism of action.

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Preclinical evaluation of anticonvulsant activity of N-(p-aminobenzoyl)-1,2,3,4-tetrahydroquinoline

B Bhuthabthim; P Leewanich; C Patarapanich; B Tantisira; MH Tantisira

Abstract

Anticonvulsant activity of N-(p-aminobenzoyl)-1,2,3,4- tetrahydro-quinoline (NTQ), a newly synthesized ameltolide analog, was investigated with regards to efficacy and toxicity in mice. Additionally, its effect on NMDA receptors was also performed on NR1a/NR2B expressed in Xenopus oocytes. NTQ, given intraperitoneally, was able to protect the animals in both maximal electroshock seizure and pentylenetetrazole (PTZ) tests, while ameltolide was effective only in the MES. NTQ was less potent than ameltolide, exhibiting the median effective dose (ED50) of 13.33 mg/kg body weight (B.W.), whereas the corresponding value of ameltolide was 0.96 mg/kg B.W. Ameltolide seemed to possess better safety profile than NTQ as indicated by the relative safety margin (LD50/ED50) of 65.28 for ameltolide and 44.97 for NTQ. Furthermore, based on the finding that the median neurotoxic dose (TD50) established by rotarod test was 37.28 and 7.18 mg/kg B.W. for ameltolide and NTQ respectively, ameltolide appeared to have more favorable protective index (TD50/ED50) than NTQ. Further experiment using Xenopus oocyte with NMDA receptor composing of NR1a and NR2B subunits was performed to probe for the effect of NTQ and ameltolide on NMDA-induced current. While NTQ and ameltolide did not induce any changes in current or a shift in membrane potential of oocyte, both of them significantly inhibited NMDA-induced current demonstrating the IC50 of 0.10 and 0.12 ?M respectively. Therefore, it is likely that inhibition of excitation of NMDA receptor may, at least in part, accounted for its anticonvulsant activity. In conclusion, the present study has identified NTQ as a broad-spectrum anticonvulsant agent, albeit its lower safety margin and protective index. Inhibition of NMDA receptor may partly contribute to anticonvulsant activity of these two compounds. Further structural modification of NTQ to improve its safety while preserving a broad-spectrum anticonvulsant activity should be carried out.

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Morphine releases glutamate through AMPA receptors in the ventral tegmental area: a microdialysis study in conscious rats

H Alaei; M Huotari; PT Piepponen; L Ahtee; O Hanninen; PT Mannisto

Abstract

Drug addiction has developed to a social illness. Changes in glutamate transmission have been recorded by the repeated administration of addictive drugs into VTA. In this investigation, In vivo microdialysis was used to study the effects of morphine on glutamate release from the ventral tegmentum area (VTA) in freely moving rats. Rats were anesthetized with chloral hydrate (350 mg/kg, i.p.) and placed in a Kopf stereotaxic apparatus. A vertical guide cannula was implanted through a burr hole and secured with dental cement held on the skull with small screws. The final co-ordinates for the tip of the microdialysis probe in right VTA relative to bregma were: anteroposterior (AP), 5.8; lateral (L), 0.5 and dorsoventral (DV), 9.0 according to atlas of Paxinos. One week after surgery, the microdialysis probe was inserted into the guide cannula and perfused with artificial cerebrospinal fluid (aCSF). After a 60 min wash out period, dialysate samples were collected in 20 min periods in vials and 20?m was used for glutamate HPLC analysis. Intraperitoneal (IP) injection of acute and repeated administration of morphine at increasing doses enhanced significantly glutamate release. Only a minor tolerance developed to this effect of morphine. AP-5 (2-amino-5-phosphonovaleric acid, 0.5 mg/kg i.p.), a NMDA receptor antagonist, given 20 min before each repeated morphine injection, did not have a significant effect on the stimulated glutamate release. Conversely, injection of CNQX (6-cyano-7-nitroquinnoxaline-2, 3-dione, 0.5 mg/kg i.p), an AMPA receptor antagonist, 20 min before each morphine dose was found to markedly inhibit morphine-induced glutamate release in the VTA. In all experiments, release of glutamate reduced by time. These results show for the first time that acute and repeated injection of morphine stimulates glutamate release in the conscious rat VTA via AMPA receptors.

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DT-MRI Tractography and its Application in Cognitive Neuroscience

M Zarei

Abstract

Recent advancement of MRI techniques and development of new methods of image analysis have allowed us to study large neural tracts within the human brain. This is based on the principle of diffusion tensor MRI that is similar to that of diffusion-weighted imaging but takes magnitude and direction of the diffusion of water into account. Using this technique we have been able to define large neural tracts within white matter of the human brain. In our centre, connections of thalamus, internal capsule, corpus callosum, and cerebral peduncle with different regions of cerebral cortex have been defined using Bayesian algorithm for DT-MRI tractography. Results of these studies are consistent with our knowledge of neuroanatomy regarding topography and orientation of fibers within human brain. This provides an exciting new way of exploring human brain particularly in relation to cognitive phenomena. In this talk, I will explain the principle of this method, recent discoveries in our centre and possible application of this method in cognitive neuroscience.

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Determining whether positively-charged channel-forming molecules of polyene antibiotic with aromatic groups affect muscle activity?

RG Gayibov; OG Kurbanov; KHM Kasumov

Abstract

This article evaluates the effect of membrane active channel-forming polyene antibiotic (PA) of levorin and its alkyl derivatives on the muscle performance. The membrane channels of muscle cells are capable to transport ions of potassium, sodium, and calcium. In the period of an intensive muscle exercise, the necessity for organic substrates increases and these channels start to work with the greater intensity. However, specific activity of native channels is limited, and, therefore they have no capacity to deliver organic compounds to muscle cells in indispensable quantity. The power engineering of muscle performance weakens rapidly and then, the muscle becomes fatigued. Here there is a necessity for activation of native cell channels induced by exogenic drugs. For this purpose, channel-forming substances like polyene antibiotics have been studied. To test the quality of such a system, BLM was used. In this technique, amphotericin B and levorin is incorporated in a lipid membrane to simulate the process of ionic permeability for such ions as potassium, sodium, calcium, and also transmembrane transport of carbohydrates and other low-molecular weight compounds. The results showed that amphotericin B and levorin enhance lipid membrane permeability for ions and monosaccharides and other neutral moleculas in the following order: water>urea>acetamide>glycerine>ribose>arabinose>glucose>saccharose. Thus, the use of PA and their derivatives with established chemical structure will create an indispensable condition for strengthening synthesis of energy-dependent substrates in muscle cells and may increase their membrane potential and can maintain energy potential of the organism at a high level. It was also demonstrated that aromatic heptayene antibiotic levorin increases the permeability of membranes for monosucrose and other neutral molecules in the following order: H2O>urea> acetamide>glycerine>ribose>arabinose>glucose>saccharose.

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Somatovisceral interactions in the rat dorsal column nuclei

HQ Zhang; PJ Rong; JL Zhang

Abstract

Recent studies have revealed that noxious visceral inputs travel in the dorsal column pathway, and interactions between colorectal noxious and tactile inputs occur in the ventrobasal thalamus. This investigation was to test whether the somatovisceral interactions also take place in the dorsal column nuclei (DCN). Forty-five single DCN neurons of anesthetized rats responsive to colorectal distension (CRD) all had excitatory responses to tactile stimuli, and their tactile responses were predominantly (32/45 units) enhanced by preceding CRD. Their responses to CRD were reduced in half (22/44) units when preceded by tactile stimulation. Despite these generally similar response features in comparison with the thalamic recordings, there were however different response properties in DCN neurons, suggesting that somatovisceral interactions take place at multiple levels in the dorsal column-medial lemniscus system.

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Developmental expression of tyrosine kinase b in rat vestibular nuclear neurons responding to horizontal and vertical linear accelerations

CH Lai; SK Lai; FX Zhang; YS Chan

Abstract

Brain-derived neurotrophic factor (BDNF) is known to be crucial for the development of peripheral vestibular neurons. However, the maturation profile of the BDNF signal transducing receptor, tyrosine kinase B (TrkB) in functionally activated otolith-related vestibular nuclear neurons of postnatal rats remains unexplored. In the present study, conscious Sprague-Dawley rats (P4 to adult) were subjected to sinusoidal linear acceleration along the vertical or horizontal axis. Neuronal activation in response to otolith stimulation was defined by the expression of c-Fos in the vestibular nucleus. Labyrinthectomized controls and normal controls showed only a few sporadically scattered c-fos-expressing neurons. In P4-6 test rats, no Fos-labeled neurons were found in the vestibular nuclei and immunostaining for TrkB was weak. The intensity for TrkB in vestibular nuclear neurons increased with age. From P7 onwards, Fos/TrkB double-labeled neurons responsive to vertical stimulation or horizontal interaural stimulation were detected; from P9 those to horizontal antero-posterior stimulation were also detected. These findings indicate a temporal disparity in the processing of gravity-related spatial orientations in space during development of central otolith neurons. At P9, Fos/TrkB double-labeled neurons responsive to horizontal interaural stimulation or vertical stimulation greatly outnumber those responsive to antero-posterior stimulation. The number of double-labeled neurons increased with age, reaching 80–85% of the total Fos-labeled vestibular nuclear neurons in the adult. Our results suggest that TrkB contributes to the mechanism of maturation in central otolith neurons.

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The role of acetylcholine muscarinic receptors in the rat basolateral amygdala on morphine-induced place preference

P Rostami; MR Zarrindast; Z Fatahi

Abstract

Some studies have shown that acetylcholine muscarinic receptors involved in the opiate reward. In the present study, the effect of intra-basolateral amygdale (BLA) acetylcholine muscarinic like receptor agonist (physostigmine) and antagonist (atropine) on the acquisition of morphine-induced place preference has been investigated in male Wistar rats. For this purpose, two 22 gauges guide cannulae were implanted into the BLA by using a sterotaxic instrument. After 7 days using biased method and the subcutaneous administration of morphine or intra-BLA injection of acetylcholinergic agents, the induction of conditioned place preference (CPP) was examined. Subcutaneous administration of the different doses of morphine sulfate (0.5-10 mg/kg) produced a significant CPP dose-dependently. The intra-BLA injection of physostigmine (1, 3, and 5 µg/rat) significantly potentiated the acquisition of morphine (0.5 mg/kg)-induced place preference. This potentiation reversed by atropine (7 µg/rat) pretreatment. The intra-BLA injection of different doses of atropine (1, 4, and 7 µg/rat) significantly reduced the acquisition of morphine (7.5 mg/kg)-induced place preferenc. Physostigmine or atropine by itself did not produce reliable CPP. Our studies demonstrated for the first time that acetylcholine muscarinic receptors in BLA have an important role in the acquisition of morphine-induced place preference.

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The effect of ascorbic acid on the acquisition and expression of nicotine-induced CPP in mouse

F Bahrami; H Sahraei; L Hosseinmardi; M Yari; N Faragi; H Ghoshooni

Abstract

Increased activity of mesolimbic dopaminergic system within ventral tegmental area (VTA) due to nicotine results in psychologic dependence. It has been argued that function of dopamine receptors could by affected by ascorbic acid (AA). Furthermore, AA can decrease the withdrawal syndrome signs in addicted animals and may postpone the development of nicotine dependence. In the present study, the possible role of AA on acquisition and expression of nicotine-induced CPP was investigated in mice. Female Swiss-Webster mice (20-25 g) were used and a biased method was performed for induction of CPP. The results showed that i.p. injection of nicotine (0.25-2 mg/kg) induced CPP in a dose-dependent manner. In addition, IP administration of AA (1-1000 mg/kg) on the test day reduced the nicotine effect, but not in a dose-dependent manner. The chronic administration of AA (1-1000 mg/kg) reduced the nicotine effect as well. Therefore, it can be concluded that motivational effects of nicotine are dramatically affected by AA.

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The effect of lamotrigin on stress-induced behavioral and biochemical changes in mice

J Shams; M Arbabi; MT Yasami; SH Goudarzi; F Moatamedi

Abstract

Stress may cause special behavioral and biochemical changes. Kindling is presumed to be the underlying mechanism for such changes. Lamotrigin, an anti-kindling agent which blocks glutamate release through inhibiting sodium channels may reverse these behavioral changes. The aim of this study was to explore the effects of lamotrigin in management of behavioral and biochemical changes caused by stress in an animal model. To provoke anxiety-like behavior, 19 mice were exposed to a brief electrical shock. Animals were then randomly divided into 2 groups. Behavioral changes were assessed in plus-maze at 24 hours,3rd week, and 6th week after exposure to stress. Plasma cortisol level, as an index of biochemical changes was also measured at 6th week after exposure. A single oral dose of lamotrigin (40 mg/kg) was administered to the case group in the first hour following stress,while the control group was given normal saline. Lower levels of anxiety-like behavior in lamotrigin-treated group were observed ($P<0.05$). Lamotrigin-treated group had also lower level of plasma cortisol ($P<0.005$). However, the difference in behavioral changes between groups at 24 hours and 3rd week was failed to reach a statistical significance. The behavioral changes at 24th hour and 3rd week as compared to each other were not significant. It can be concluded that lamotrigin may prevent biochemical and behavioral changes of stress.

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Nitric oxide mediation of morphine sensitization in the rat nucleus accumbens

H Ghoshooni; V Khoshbaten; F Bahrami; V Noroozzadeh; H Sahraei; S Bijani; S Oryan; M Eidi

Abstract

Previous studies have reported that morphine exerts its effects in part through the release of nitric oxide (NO). It has been pustulated that drug sensitization is a major cause of relapse to drug use in addicts. In the present study the effects of intra-accumbal injections of L-arginine, the NO precursor and L-NAME, the NOS inhibitor on the morphine sensitization in Wistar rats (250-300 g) were investigated. Male rats (n = 8/group) were anesthetized and bilateral cannulation in nucleus accumbens (23-gauge, AP = 1.7mm, L = ±0.8, V = 7.1 mm) was performed. Five days after cannulation, animals were trained in an Un-Biased conditioned place preference apparatus for five consecutive days. The NOergic drugs were injected to the animals in two ways: first; the animals were trained with morphine and were received L-arginine or L-NAME at 5th day of experiments just before the test. Second group received L-arginine or L-NAME before morphine injection. At the 5th day of the experiments, each animal was placed in the apparatus and its behavior was recorded for 10 min. The results showed that pretreatment of the animals with L-arginine augments the development, but reduced the expression of morphine sensitization. Pretreatment of the animals with L-NAME reduced both development and expression of morphine sensitization. In conclusion, present experiments showed that morphine sensitization is in part dependent on the activation of NO system within the nucleus accumbens and the role of this neuromodulator in morphine dependence must be considered in further treatments of morphine addicts.

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Inhibition of nicotine-induced behavioral tolerance by ascorbic acid in female mice

M Alghaci; H Sahraei; H Ghoshooni; AA Barzegari; AA Aliabadi; A Noroozzadeh

Abstract

Administration of nicotine leads to incentive and behavioral tolerance in human and animals. Ascorbic acid administration inhibits some effects of nicotine. In the present study, the effects of ascorbic acid administration on the expression and development of nicotine-induced behavioral tolerance in female Swiss-Webster mice (20-25 g) was investigated. Animals were injected with nicotine (0.5 mg/kg, i.p.) once daily for 12 days in order to induce tolerance. At 13th day, an effective dose of nicotine (1 mg/kg, i.p.) was administered to the mice and activity of the animals was recorded for 20 min in an activity monitoring system (30 x 30 x 20 cm). Different doses of ascorbic acid (1-1000 mg/kg, i.p.) were administered to the animals in two ways: first group; the animals received ascorbic acid at days 1-12 of the experiments 10 min before the administration of nicotine (development). Second groups received ascorbic acid only at 13th day of the experiments (Expression). The results showed that administration of nicotine (0.5 mg/kg, i.p.) for 12 days to the mice induced behavioral tolerance in the animals. Injection of ascorbic acid (1-1000 mg/kg, i.p.) inhibits the development of nicotine-induced behavioral tolerance in the female mice. Administration of ascorbic acid (1-1000 mg/kg, i.p.) was ineffective to alter the expression of behavioral tolerance induced by nicotine. In conclusion, administration of ascorbic acid may inhibit the development of nicotine-induced behavioral tolerance, but it is ineffective to inhibit the effect of the expression of nicotine-induced behavioral tolerance.

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Ascorbic acid inhibits nicotine-induced behavioral sensitization in male mice

AA Aliabadi; AA Barzegari; H Sahraei; H Ghoshooni; A Noroozzadeh; P Rostami

Abstract

Repeated administration of nicotine causes incentive and behavioral sensitization in animals. Ascorbic acid administration inhibits some effects of nicotine. In the present study, the effect of ascorbic acid administration on nicotine-induced behavioral sensitization in Male Swiss-Webster mice (20-25 g) was investigated. Animals were injected with nicotine (0.25 mg/kg, i.p.) once daily for seven days in order to induce sensitization. In 9th day, an ineffective dose of nicotine (0.1 mg/kg, i.p.) was administered to the mice and the activity of the animals was recorded for 20 min in an activity monitor (30 x 30 x 20 cm). Different doses of ascorbic acid (1-1000 mg/kg, i.p.) were administered to the animals in two ways: first group; the animals received ascorbic acid at days 1-7 of the experiments 10 min before the administration of nicotine (Development). Second group, the animals received ascorbic acid only at 9th day of the experiments (Expression). The results showed that: administration of nicotine (0.25 mg/kg, i.p.) for 7 days to the mice induced behavioral sensitization in the animals. Injection of ascorbic acid (1-1000 mg/kg, i.p.) inhibits the development of nicotine-induced behavioral sensitization in the mice. Administration of ascorbic acid (1-1000 mg/kg, i.p.) was ineffective to alter the expression of behavioral sensitization induced by nicotine. In conclusion, administration of ascorbic acid may inhibit the development of nicotine-induced behavioral sensitization but is ineffective to inhibit the effects of the expression of nicotine-induced behavioral sensitization.

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A study on the role of nitric oxide in morphine dependence using conditioned place preference

H Sahraei; F Bahrami; A Noroozzadeh; H Ghoshooni; MR Zarrindast; A Khoshbaten

Abstract

Drug abuse and dependence have progressively become a dramatic problem in human societies. Since the exact mechanism(s) of the effect of related drugs in the brain has not been explored, therefore, this problem has not been resolved yet. Several lines of evidence indicated that nitric oxide (NO) plays an important role in the process of drug dependence. The role of NO within the different regions of the brain has previously been reported in a condition of morphine dependence. The previous studies were carried out by means of conditioned place preference paradigm, a simple and useful method for evaluating the effect of drug abuse in an animal model. Therefore, the role of NO within hippocampus, central nucleus of amygdala, nucleus accumbens and ventral tegmental area was investigated in the process of drug dependence. For this purpose, L-arginine (0.3, 1, and 3 ?g/rat), the NO precursor and L-NAME (0.3, 1, and 3 ?g/rat), the NOS inhibitor were used as NOergic drugs, which were administered to male Wistar rats (250-300 g). Male rats (n = 8/group) were anesthetized and bilaterally cannulated above the mentioned regions. Five days after the cannulation, animals were trained in an un-biased conditioned place preference apparatus for five consecutive days. The NOergic drugs were injected into the animals in two ways: first; the animals were trained with morphine and received L-arginine or L-NAME at 5th day of experiments just before the test. Second group received L-arginine or L-NAME before morphine injection. At the 5th day of the experiments, each animal was placed in the apparatus and its behavior was recorded for 10 min. The results showed that NO plays a crucial role in the above-mentioned regions and the role of NO in morphine dependence should be considered in treatment of morphine addicts.

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The effect of GABAB receptor activation within the ventral tegmental area on morphine-induced incentive sensitization in female rats

L Etemadi; H Sahraei; H Ghoshooni; S Oryan; M Eidi; A Eidi

Abstract

Repeated administration of morphine sensitizes animals to the stimulant and rewarding properties of the drug. It may also alter the expression and function of GABA receptors within the ventral tegmental area (VTA), a midbrain region that is implicated in morphine action. In the present study, the effect of activation of GABAB receptor subtypes within the VTA on morphine-induced incentive sensitization in female Wistar rats (250-300 g) was investigated. Female rats (n = 8) were chronically implanted with a 21-gauge cannula in the VTA (V = 8.5, L = 0.9, and A = -4.8 mm from bregma). One week after the surgery, the animals received morphine (5 mg/kg, s.c.) in their home cage for three consecutive days. Five days later, the animals were conditioned with an ineffective dose of morphine in a two compartment un-biased apparatus. Different doses of GABAB receptor agonist, baclofen (1.5, 6, and 12 ng/rat) were administered to the animals 5 min before the test. The results indicated that morphine administration to the animals in three consecutive days produces sensitization in which low doses of morphine (0.5 mg/kg, s.c.) induced CPP. Furthermore, administration of low doses of baclofen (1.5 and 6 ng/rat, i.VTA) enhanced, while higher doses of baclofen (12 ng/rat, i.VTA) reduced the morphine effects. The results indicated that activation of GABAB receptors within the VTA in morphine-sensitized animals produces a biphasic response, which may be due to pre and post GABAB receptor activation.

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Individual typological characteristics of rat's behavior in open-field and passive avoidance learning

P Babaei; B Soltani

Abstract

The aim of this study was to compare the learning performance of Wistar rats in passive avoidance box and open field behavior. Animals were placed in the center of the open field and their behavior was manually recorded for 5 min. Two latency periods, first motion, and exit to the center, the number of inner and outer squares crossed, grooming activity, the number of rearing in the center of the open field. Animals with short latency period of motion (4.8±0.3sec) and high ambulatory activity (113±4) were classified as group 1(hyperactive), and ones with long latency to center (7.7± 0.5 sec) that crossed less (65.5±3.9) The square were selected as group 2 (hypoactive). Then animals were placed in passive avoidance box and received 2.5 mA electric foot shock for 5 sec. After 24 h animals were tested for 3min and the latency period and the total time spent on the platform was recorded. The data showed that the good learner rats exhibited a higher number of reactions in the open field test. Since in the passive avoidance experiments the environmental influences on rats were identical, so the differences observed among rats in learning performances might be due to internal state. In conclusion this study provides evidence that learning performance is different in rats with different behavioral responses in the open field.

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Sensitivity change of dopamine receptors in hippocampus (CA1) and its effect on morphine-induced condition place preference

P Rostami; MR Zarrindast; A Rezayof; M Nasehi

Abstract

In the present study, the effects of intra-cerebral hippocampus (CA1) injections of apomorphine D1, D2-like receptors agonists on morphine-induced place preference in male Wistar rats have been investigated. Subcutaneous administration of different dose of morphine sulphate (1, 3, 6 and 9 mg/kg) produced a dose-dependent conditioned place preference (CPP). Intra-cerebral hippocampus (CA1) administration of apomorphine (0.25, 0.5 and 1 ?g/rat) 5 days before measuring CPP (using a 3-day schedule at the specific time) with an ineffective dose of morphine (1 and 3 mg/kg) decreased a significant CPP. In other words, apomorphine (0.25, 0.5, and 1 ?g/rat) injection into hippocampus decreased CPP in combination with morphine (1 and 3 mg/kg). Since apomorphine is D1, D2-like receptors agonist of dopamine and has sensitized both receptors, the study of which of these receptors causes the significant CPP reduction is going on.

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Sciatic functional index following induction of injury in the sciatic nerve of rats

S Cheraghiyan; E Tamaddonfard; A Tashviginejhad

Abstract

Sciatic functional index (SFI) as a behavioral method was used to assess the functional recovery following experimentally-induced various injuries in the sciatic nerve, spinal cord, and brain. In the present study, the functional recovery after induction of injury in the sciatic nerve was investigated in rats using SFI. Twenty-four rats were divided into three groups including sham-operated (SS), Loose ligation (LL), and crushing with fine forceps (CF). For induction of sciatic nerve injury, rats were anaesthetized with a mixture of ketamine (180 mg/kg) and xylazine (3 mg/kg). Then, skin and muscles were incised and the sciatic nerve trunk was exposed. In SS group the same manipulation was performed. In LL group four ligatures (5.0 catgut) were loosely tied around the sciatic nerve about 1-mm spacing. In CF group four points of sciatic nerve were crushed using a fine forceps. The muscles and skin were then closed with catgut sutures. In the SFI test, rats walked along a 60 cm long runway lined with plain paper after first pressing their hindpaws onto an inkpad. The tracks left by the rats were then measured and four parametyers were taken: the length of each footprint, the distance to the next opposite footprint, the toe spread distance between 1st and 5th digits, and the distance between 2nd and 4th digits. The value for both injured and uninjured sides were entered into an empirical equation determined by de Medinaceli (1982). This test was conducted on rats in the 5th, 3 rd and 1st days before surgery and 1st, 2nd, 4th, 5th, 10th, 14th, 19th, and 25th days after the surgery. Data were analyzed by factorial ANOVA and Duncan test. The results showed that in the intact rats the average score of SFI was –5.4 ± 1.7. In SS group the SFI scores did not change during the course of study (-9.4 ± 4.6). In LL group SFI scores were more negative (-91.8 ± 11.2). In CF group the SFI scores were more negative till 14th day after the surgery, but in the 19th and 25th days after the surgery, SFI scores become less negative (-31.5 ± 5.5). From the results of this study it is concluded that in the various models of sciatic nerve injures, SFI method, especially using stamp and paper, might serve as a good tool in the investigation of functional recovery. Sciatic nerve injury induced by ligation produces more degenerative and regenerative changes than that of crushing.

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Deprenyl changes the expression of Trk-B and P75 NTR receptors in rat after sciatic nerve axotomy

M Heshmati; T Tarihi; SJ Mowla

Abstract

During development many of neurons die by the phenomenon named programmed cell death or apoptosis and this reaction is regulated by neurotrophin (BDNF, NGF, NT3 and NT4/5). These neurotrophins bind to two different classes of transmembrane receptor proteins, the Trks and P75 NTR. Axotomy can induce apoptosis after birth and deprenyl is a an inhibitor of monoamineoxidase type-B and seems to act as a neuroprotective factor and reduces apoptosis after sciatic nerve axotomy. The aim of this research was to study the effect of deprenyl (2.5 mg/kg, i.p.) following axotomy in newborn rat and to study the changes in expression of Trk-B and P75 NTR by RT-PCR. The segment of L4-L6 was studied after sciatic nerve axotomy. This study was conducted on 2 groups, control (normal saline injection) and experimental (deprenyl injection) and each group was classified into 3 subgroups on the basis of the time of injection and axotomy. In all of the 6 subgroups, 4 hours after injection, they were sacrificed and the segments L4-L6 of spinal cord processed by RT-PCR. The results showed that deprenyl increases expression of Trk-B, but decreases expression of P75 NTR as compared to control group.

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Deprenyl increases synaptophysin and choline acetyltransferase in rat after sciatic nerve axotomy

M Heshmati; T Tarihi; SJ Mowla

Abstract

Neuroprotective effect of deprenyl on motoneurons of spinal cord after axotomy of peripheral nerves such as sciatic has been well established. Deprenyl is an inhibitor of monoamine oxidase type-B (MAO-B). The main function of this agent is the release of neurotransmitters from pre-synaptic terminals. Acetylcholine is a neurotransmitter that is synthesized by choline acetyltransferase (ChAT) and is widely used as a marker to assess the function of neurons. Synaptophysin is one of the synaptic vesicle neuronal terminal residual proteins which is also used as a marker for some neurotransmitters.The aim of this research was to study the effect of deprenyl (2.5 mg/kg) injection on synaptophysin and ChAT immunoreactivity. The intraperitoneal injection of deprenyl was done following axotomy and continued for 21 days. Then, the animals were perfused with paraformaldehyde (4%) and spinal cord segment L4-L6 proccessed for immunohistochemistry. The results obtained in this study showed that deprenyl-treated axotomized mice had higher ChAT and synaptophysin expression as compared to untreated axotomized animals.

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A clinically oriented experiment on the effect of mixed culture of neonate spinal cord transplantation on recovery of spinal cord injury

M Firouzi; P Moshayedi; H Saberi; H Mobasheri; F Abolhassani; MA Oghabian

Abstract

In spinal cord injuries, direct trauma by edges of sublaxated or dislocated vertebrae and indirect ischemia as a result of vascular injury necrotize the neural tissue. After spinal cord injury, tissue loss appears as micro- or macrocavitation. Accumulations of non-neuronal cells substitute spared tissue and halts axon regrowth. Lack of supporting cells (secreting trophic factors and matrix) aggravates the condition. In this study, mixed culture of neonatal spinal cord cells were transplanted as potential source of trophic factors, matrix formers, new substitutional elements and myelinating units. Rats injured by impinging between the blades of a titanium clip (1.16 N closing force). Cells were grafted (2 * 10^6 cells in 8 microliters) subarachnoidally 7 days post-injury. Subjects were followed up for 2 months by behavioral (BBB score, Beam Balance Test, Foot Fault Test, Hopping Reflex, Pedal Withdrawal Reflex, Bracing Reflex, Lateral Tactile Reflex and Foot Static Index) and then were sacrificed and assessed by histological (axon density and demyelination distribution) and magnetic resonance imaging (sparing area and lesion length) techniques. Histological and imaging values significantly increased ($P<0.05$) in cell- treated group. Meanwhile, BBB score was significantly elevated between 15-39 days post-injury, and, then lost significantly. Clinical judgment was made according to the importance of each index.

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Morphological changes of lumbar spinal neurons after sciatic nerve transection in neonate rats

A Shams; B Jamei; TM Al-taraihi; M Rezazadeh Valujerdi; S Nazarpour; A Siadati

Abstract

Axotomy of the sciatic nerve have been documented to cause neuronal loss, especially in newborn rats. Few works have focused on time course of neuronal loss and the type of cell death, which occurs after axotomy. Forty rat pups were anesthetized by hypothermia and the right sciatic nerve transected at five days of their age and the left side was used as control. The operated animals were sacrificed 3, 5, 7, 10 and 14 days after axotomy. The animal were anesthetized with ether and perfused and fixed with paraformaldehyde and glutaraldehyde followed by osmium tetroxide as a post fixative. Forth to sixth lumbar spinal segments were used for light and electron microscopic processing and embedding. Transverse serial sections was cut and stained with Hematoxylin and Eosin as well as cresyl violet. Semi-thin sections was cut from resin embedded block and stained with Toluidine blue. The number of motoneurons (MNs) from experimental and control sides of lumbar spinal cord was counted by superimposing eyepiece graticule on the microscopic image. The comparison between the operated and control sides was done by student's t- test. The analysis of variance was also done to compare the variation among the groups. Ultrastructural features of dead MNs showed the classical features of apoptotic neurons. Other MNs showed the vacculation of cytoplasm. The conclusion of this study is that axotomy of sciatic nerve resulted in apoptotic cell death of motoneurons of spinal cord as well as a time-dependent reduction of cell number where the peak of the cell loss occurred at day 10.

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Heat shock protein 70 protects motor neuronal cells expressing mutant Cu/Zn superoxide dismutase (SOD1) against altered calcium homeostasis

HJ Kim; WM Cho; YH Hong; JJ Sung; M Kim; KW Lee

Abstract

Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disorder characterized by the progressive loss of motor neurons leading to paralysis and death. Mutations of the human Cu/Zn superoxide dismutase (SOD1) are found in some cases of familial ALS (fALS). Recent evidences suggest the accumulation of intracellular calcium is one of the primary mechanisms of motor neuronal degeneration. In this study, we attempted to test the protective effect of Hsp 70 on mutant SOD1 against altered calcium homeostasis. To test the effect of Hsp70 on constitutively expressing mutant SOD1, motoneuron-neuroblastoma hybrid cells (VSC4.1) co-expressing HSP70 and human mutant SOD1 (G93A, A4V) was established. Calcium mobilizer through cell membrane (4-bromo-calcium ionophore A23187) or endogenous calcium releasers (ryanodine, thapsigargin, 8-bromo-cyclic ADP) were treated and the viability determined by 3-(4, 5-dimethylthiazol-2-yl)-2, 5-diphenyltetrazolium bromide (MTT) assay. To determine the evidence of apoptotic cell death, treated cells were stained with Hoechst 33342 or assayed for caspase 3 activity. To confirm the suppression of SOD1 aggregates by Hsp70, VSC 4.1 cells expressing Hsp70 were transfected with mutant SOD1 gene (G93A), and then, A23187 or thapsigargin were treated for 1 day. Mutant SOD1 aggregates were quantified under the fluorescence microscope. The effect of exogenous NO was also tested with S-nitrosoglutathione (GSNO). Calcium ionophore A23187, thapsigargin and GSNO decreased viability of cells expressing mutant SOD-1 (A4V, G93A) in a dose-dependent manner. These cells showed dispersed chromatin or nuclear fragmentation by Hoechst 33342 staining. In addition, increased intracellular calcium levels by A23187 or thapsigargin increased caspase 3 activity and mutant SOD1 aggregates. Our data showed the protective effect of Hsp 70 against altered calcium homeostasis by inhibiting the caspase 3 activation. These findings suggest Hsp 70 may have beneficial effects on the SOD1-mediated motor neuronal degeneration in some forms of ALS.

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Cyclooxygense-1 inhibition delays hypersensitivity to nerve injury

SSV Padi; SK Kulkarni

Abstract

Despite the important role of both cyclooxygenase (COX) isoforms (i.e. COX-1 and COX-2) in maintenance of hypersensitivity following peripheral nerve injury, their role in the development of neuropathic pain is not clear. The present study was undertaken to determine the effect of COX inhibitors to address the potential role of COX isozymes in the development of neuropathic pain in rats after chronic nerve constriction injury. The paw withdrawal response to cold and mechanical stimulation was observed after every week for 4 weeks following nerve injury in rats. A single intraperitoneal administration of naproxen, a nonselective COX inhibitor (10 or 30 mg/kg) or rofecoxib, a selective COX-2 inhibitor (3 or 10 mg/kg) 2 h before nerve injury did not attenuate the development of neuropathic state. However, administration of naproxen (10 or 30 mg/kg, i.p.) on day 7 reversed hypersensitivity but did not attenuate its development. Chronic administration of naproxen, but not rofecoxib, 2 h before and daily for 7 days after nerve injury, significantly and dose dependently attenuated and further delayed the development of hypersensitivity for 21 days following nerve injury. These results indicate that prolonged inhibition of COX-1 but not COX-2, following peripheral nerve injury could prevent the development of neuropathic pain.

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The role of the desert hedgehog signaling pathway during degeneration and regeneration of peripheral nerves

S Naghibzadeh Bajestan; F Umehara; K Itoh; SS Namini; KR Jessen; R Mirsky; M Osame

Abstract

The desert hedgehog (Dhh) signaling pathway is involved in the development of peripheral nerves (PNs). Dhh-null mice show abnormal neuronal development and perineurial barrier function. As it was previously shown that dhh is mainly expressed in developmental nerves and Sonic hedgehog protein (dhh homologous) has therapeutic effects in neuronal survival, we attempted to investigate the possible role of dhh signaling pathway in the nerve maintenance and repair after injury. Using RT-PCR and immunohistochemistry, we investigated the expression patterns of Dhh and its receptors (Patched-1 and 2, Smoothened) in postnatal and adult nerves of Wild type (WT) mice. The mRNA expression patterns of dhh and its receptors were investigated on crushed sciatic nerves of adult mice at different time points after injury (day 2, 7, 14, 28, 42). The DDY mice were anaesthetized with interaperitoneal injection of sodium pentobarbital and inhaled ether. All experimental protocols involving animals were approved by the committee of life science resource development research center of Kagoshima University. The expression patterns of dhh and ptc-2 are similar to the other two Schwann cell proteins (ciliary neurotrophic factor and myelin associated proteins), which have been shown to play role in nerve repair and survival. In conclusion, our results suggest a potential role for the Dhh-Ptc2 signaling pathway in the maintenance of PNs and nerve regeneration after injury.

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Membrane fusion/repair in nerve cells: a biophysical application in spinal cord injuries regeneration

S Mousavidoust; H Mobasheri; M Firouzi

Abstract

Cell membrane has a critical and vital role in functioning and existence of nerve cells that form central nervous system (CNS) in mammals. Disruption of nerve membrane that normally occurs following an accident injuring spinal cord is known to be the major cause of paralysis. In most occasions, spinal cord injuries are not leading to complete cut in spinal cord fibers but are known to cause crush sites where the nerve membrane structure is destroyed. Parts of membrane in these fibers are later on interfused. In this study we are trying to employ different factors to promote membrane fusion, restoring the cytoplasm physicochemical condition and reconstructing microtubules networks so that the nerve fiber conduction is restored. The experiments are started in nerve cell culture medium where the application of various lipids in parallel to the implementation of electromagnetic field is meant to promote membrane repair/fusion. The base of this part of experiment is the introduction of specific lipids, compatible to those already exist in membrane, and are capable to reinitiate bilayer formation due to their particular phase transition characteristics. The nerve membrane healing recovery is monitored morphologically in culture medium. The follow up of the positive results is monitored in rats whose spinal cords have been injured experimentally. Behavior analysis of the rats, and their total scores obtained by means of know indices as well as somatosensory evoked potential (SSEP) tests form the in-vivo part of the study.

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Neuroprotection and restoration of the nigrostriatal dopaminergic system in 6-OHDA lesioned rat model of Parkinson's disease: Role of GDNF and TGF expressing Zuckerkandl's organ

RK Chaturvedi

Abstract

Zuckerkandl's organ (ZK) is an extra adrenal para-ganglion and has the ability to express glial cell line derived neurotrophic factor (GDNF) and transforming growth factor (TGF). It is also a source of dopamine and norepinephrine. In the present study, the neuroprotective and restorative potential of ZK was studied by transplanting it into the striatum of adult rats either before or after the intracerebral 6-OHDA administrations. Animals were analyzed to see the neuroprotection and restoration using neurobehavioral, neurochemical and immunohistochemical parameters at 12 weeks post-transplantation. In protective experiment, 6-OHDA lesioned rats receiving ZK before lesioning exhibited significant neuroprotection of dopaminergic neurons.

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Protective effect of adult olfactory ensheathing cells against 6-OHDA toxicity in PC-12 cells

S Shukla; AK Agrawal; RK Chaturvedi; N Srivastava; K Seth; C Sinha; PK Seth

Abstract

Olfactory ensheathing cells (OEC) have been successfully used to stimulate the growth of injured fibers and to promote functional recovery within the injured adult CNS. These cells exhibit functional properties, which are known to be involved in axonal elongation. OEC express high level of growth factors including NGF, GDNF, BDNF and NT-3, which are known to play an important role in nerve regeneration. In view of their axonal growth promoting properties, the present study is undertaken to further make an insight into the mechanism underlying the process of neuroprotection. In present study the effect of OEC conditioned media on PC-12 cells was investigated. OEC have a protective effect against 6-OHDA-induced toxicity as evident by MTT assay. Similarly, the expression of early response gene also reduced in 6-OHDA treated PC-12 cells in the presence of OEC-conditioned media. The results show that the factors secreted by the adult OEC can promote PC-12 cells from 6-OHDA-induced toxicity.

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The effect of prenatal restraint stress on the number and size of neurons in the rat hippocampal subdivisions

M Hosseini Sharifabad; NJ Randel

Abstract

Animal studies have shown that prenatal stress is able to induce long-lasting neurobiological and behavioral alterations in adult offspring. In spite of the facts that hippocampus is sensitive to early developmental influences and its known functional importance in learning and memory, few data are available on the effect of prenatal stress on the structure of hippocampus. Therefore, this study was carried out to evaluate the effect of repeated restraint stress during prenatal period on the total number and individual volume of neurons in granular and pyramidal layers in male adult rat. For this purpose, pregnant Wistar rats were randomly divided into two groups, stressed (n= 9) and non-stressed (n = 8). Pregnant dams in the stress group were placed in a Plexiglas restraint tube for 1 h/day from days 15-21 of gestation. Control rats were left undisturbed in their home cages. Then, 2-months old male offspring were anesthetized and transcardially perfused. The brains were removed and divided into hemispheres. One hemisphere was selected at random for estimating number of neurons and the other for estimating volume of individual neurons. The total number of granular and pyramidal cells (area CA1,3) in the hippocampus was analyzed using optical fractionator. The Rotator method was applied to estimate individual volume of neurons. Prenatally stressed rats showed a decrease in the size of granular and pyramidal neurons (CA1 and CA3) as compared to their non-prenatally stressed counterparts. This study also showed that there are no significant differences in the total number of granular layer neurons, CA1 pyramidal and CA3 pyramidal neurons of prenatally stressed and control animals. These data provide a neuroanatomical basis that may be relevant to the reported disturbances in behavior and learning in prenatally stressed offspring. In this respect, previous studies demonstrated that prolonged stress reduces mRNA levels for neurotrophins in the hippocampus. It is well known that a reduction in neurotrophin level can lead to neuronal atrophy without necessarily being accompanied by neuronal loss.

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The effect of sub-lethal doses of paraoxon on the growth of rat cultured hippocampal neurons in neurobasal/B27 medium

M Yousefpour; F Bahrami; Z Nourian; F Arabsalmani; A Khoshbaten; A Asgari

Abstract

The qualitative morphologic study of the effects of organophosphate agents on development of neural cells in culture is a promising approach as a primitive step for further physiologic, electro physiologic and molecular investigation. Parathion is an OP (Organophosphate) used as an agricultural insecticide. Improper handling of this agent causes poisoning characterized by convulsive seizures, which may sometimes lead to death. Since Parathion has been observed to be responsible for more cases of poisoning than any other OP insecticide, it is vitally important to investigate other mechanisms, besides cholinesterase inhibition, which can contribute to the neurotoxicity of paraoxon. In present study, hippocampus cells taken from rat's neonate were introduced into the neurobasal medium supplemented with B27 serum, which is an optimum medium for the growth of the hippocampus neurons, mainly due to its ability to prevent other neuroglial cell growth. Six hippocampi were dissected out of three rat brains. Cells were mechanically isolated by triturating in a dissociation buffer. After dilution and centrifugation, cells were plated on polylysine-coated pellets containing B27/neurobasal medium. Control pellets contained only neurons, while the research samples experimental contained 10, 30, 50, 100 & 150 ?m of paraoxon. The neuronal growth was morphologically followed from early hours of culturing up to one month after. Shape of the soma and arborizations were two main qualitative criteria of observation. Our results revealed that there was no clear morphologic difference between neurons in the control group and those exposed to 10?m paraoxon. In contrast, deformity of the soma and a decrease in the number of arborization were noticed in groups exposed to 100 & 150?m and changes were prominent with increasing paraoxon concentration. The present data requires a further quantification of our study by concentrating on doses between 30 ?m to 150 ?m.

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The effect of maternal hyperthyroidism on the formation of the cerebellar cortical layers in the rat embryo

Gh Kaka; SH Sadraie; F Fadaie; M Jallali Monfared; H Dashtnavard; M Mofid

Abstract

It has been shown that maternal hyperthyroidism can affects on neonatal outcomes such as spontaneous abortion, intrauterine growth retardation. The aim of this study was to investigate the influence of hyperthyroidism on formation of cerebellar cortical layers in the rat embryos. Thirty Sprague-Dawley female sexually mature rats weighting 200 ± 20 g were used in the study. Female rats were randomly divided into 2 groups. One group as control group did not received any injection. Another group as experimental group (hyperthyroid rats) received L-thyroxine (0.5 mg/kg per day) by IP injection. Serum concentrations of triiodothyronine (T3) and tetraiodothyronine (T4) increased in hyperthyroid rats after two weeks of the start of treatment. All female rats in both groups were mated with normal male rats. Hyperthyroid pregnant rats were received L-thyroxine (0.5 mg/kg per day) by IP injection during the gestation period. Animals were killed on 21th day of gestation and the embryos were taken and their head were cut and fixed in Boin's solution for 72 hours. Head of embryos were then dehydrated and embedded in paraffin. The paraffin blocks were sagitally sectioned at 5-micrometer thickness and then stained by hematoxylin-eosin method. Quantitative computer-assisted morphometric study was done on the cortical layer of the cerebellum including: External granular layer (EGL), Intermediate layer (IL), and maternal granular layer (IGL). The results showed the thickness of the EGL was decreased in experimental groups when compared to control group (P=0.06). The thickness of the IGL was also was no significant decreased in experimental groups when compared to control group (P=0.09). On the other hand, thickness of IL of experimental group was increased when compared to control group, but the differences were not significant. Our results showed that maternal hyperthyroidism can affects on the formation and neuronal migration in the cerebellar cortex of rat embryos. Therefore, pregnant women with hyperthyroidism should appropriately be managed.

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The effect of oral administration of morphine on the development of neural plate in Wistar rats

M Hashemi; H Sahraei; M Hosseini-Amin; M Shams-Lahijani

Abstract

Morphine administration produces several behavioral abnormalities in children and also in offspring animals, which is referred to as the effects of the drug on the development of the central nervous system. In the present study, the effect of maternal morphine consumption on the development of neural plate was investigated in Wistar rats (200-250 g). For this purpose, female rats (n = 8) were treated with morphine (0.01, 0.05, and 0.1 mg/ml of water) for 21 days. Then, the animals crossed with male rats and did not receive morphine until the 9th day of pregnancy. Thereafter, the animals were anesthetized by diethyl ether and the embryos were taken out surgically. The embryos were fixed in formalin (10%) for a week, and then, cross sectional procedure was performed. The sections were stained with H & E. The results showed that morphine administration to pregnant rats causes a delay in neural plate development in their embryos. In addition, morphine at a dose of 0.01 mg/ml produced the maximum effect. It can be concluded that morphine consumption before pregnancy delays neural plate development in rat embryos, which may also be true for humans.

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The effect of oral morphine administration on development of neural tube in Wistar rats

S Nasiraei-Moghadam; H Sahraei; M Sadooghi; H Bahadoran

Abstract

Morphine administration during pregnancy causes several behavioral abnormalities in offspring animals. In the present study the effects of maternal morphine consumption on development of neural tube in Wistar rats (250-300 g) were investigated. Female rats (n = 8 were crossed with male rats and pregnant ones were treated with oral morphine (0.01, 0.05 and 0.1 mg/ml of water) until the 10th day of pregnancy. On the day 10, the animals were anesthetized by diethyle ether and the embryos were taken out surgically. The embryos were fixed in formaline 10% for a week and then cross sectional procedure performed. The sections were stained with H&E. The results showed that: administration of morphine resulted in severe reduction in neural tube development in embryos. Morphine at a dose of 0.01 mg/ml showed the maximum effect. In conclusion, it is clear that morphine consumption in pregnant rats resulted in delay in neural tube development that may be true in humans.

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Uncontrolled stress produces severe defect in development of frontal cortex in the rat

F Abuali; A Mirdamadian; H Sahraei; H Bahadoran; H Dashtnavard; H Sadraei

Abstract

One of the most important factors in drug addiction is stress. It has been shown that animals with prenatal stress history are more risky for drug dependence. It have also been shown that frontal cortex play a role in drug dependence. In the present study the effects of two kinds of mild stress during pregnancy on the development of frontal cortex in Wistar rats (250-300 g) were investigated. Female rats (n = 8) were crossed with male rats and then were exposed to a random daily mild stress trial (5 min stress followed by five min rest in a 20 min period/day) until the 17th day of pregnancy. Two kinds of stress were applied, noise stress, by means of a tap on the animal cage with an iron tube and immobilization. On the day 17, the animals were anesthetized by diethyl ether and the embryos were taken out surgically. The embryos were fixed in formalin 10% for a week and then cross sectional procedure were performed. The sections were stained with H&E and evaluation was performed by motic program for the prefrontal cortex layers and laso cell counting. The results showed that thickness of the frontal cortex in the groups in which received stress was reduced. Cell counting also reveal that number of cells in the frontal cortex layers in experimental groups are lower than control one. In conclusion, present experiments showed that uncontrolled stress can change the structure and cytoarchitecture of frontal cortex which may be responsible for higher risk of drug abuse among the children of mothers with stress history during pregnancy.

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Differentiation of human embryonic stem cells into neurons

H Baharvand; M Hatami; M Soleimani; A Taei

Abstract

Human embryonic stem (ES) cells are undifferentiated pluripotent cells derived from the inner cell mass of blastocyst stage embryos. These unique cell lines have the potential to form virtually any cell type in the body and can be propagated in vitro indefinitely in an undifferentiated state. These cells are capable of forming embryoid bodies (EB) that contain cells from all three embryonic lingages. Here, we have characterized the neurogenic effects of retinoic acid (RA, 10-6 M) on human ES cells (Royan H1) in vitro. For morphological and imunocytochemical evaluation, antibodies against microtubules-associated protein 2 (MAP-2), neurofilament protein, neuron-specific tubulin-III and neuron-specific enolase (NSE) were used. The results showed that more than 70% of embryoid bodies differentiated into neuronal cells as compared to a 10% change in the control group and the cells expressed neuron specific molecules (MAP-2, Neurofilament protein, tubulin III and NSE). These findings show that human ES cells have great potential to become as an unlimited cell source for neurons in culture. These cells may then be used in transplantation therapies for neural pathologies.

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Alteration in postnatal development of masseter innervation in hypothyroid rats

F Ganji; G Behzadi

Abstract

The thyroid hormones have profound effects on the development of neuromuscular system. These hormones exert their influence on both muscle fibers and related motoneurons during development. Masseter is one of the most important muscles for mastication in mammals. Thyroid hormone deficiency 3 weeks after birth, the period in which an alteration from sucking to biting occurs, could influence the developmental pattern of the muscle. Horseradish peroxidase was injected into the masseter (0.5-5 µlit, 40%) of normal and prenatal hypothyroid pups at 1,7,15 and 23 postnatal days. After 24-48 h, the 50µm brainstem sections were processed for TMB histochemical procedure. Labeled motoneurons in Mo5 were counted, measured and on the basis of their tracing intensity classified into heavy (H), intermediate (I) and light (L) groups. In each age group motoneurons were divideded into small, intermediate and large populations. In 1-day old pups, there was no significant difference between the labeling intensity and size of motoneurons in normal and hypothyroid rats. From the day 7, a significant decrease in the number of H motoneurons was observed ($P<0.05$), I motoneurons remained unchanged,whereas the number of L motoneurons significantly increased in hypothyroid pups ($P<0.05$). A significant increase in the number of small motoneurons was observed in hypothyroid pups, medium sized neurons showed no change but large motoneurons significantly decreased in hypothyroid pups ($P<0.05$). These data suggest that at the end of sucking period (day 23), hypothyroid pups show a 6 days delay in their pattern of masseter innervation as compared to its normal development.

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The effect of morphine consumption by mother on brain development of rat offspring during lactation period

M Assaran; M Danaie; V Sheibani; P Rayegan; N Nematollahi Mahani

Abstract

According to some studies, the number of addicted females in reproductive ages has been increased. The present study was designed to investigate the effect of oral morphine consumption on the development of CNS and body growth in rats. For this purpose, female Sprauge-Dawley rats were divided into control and experimental groups (20 animals in each group). Maintenance dose of morphine (0/4 mg/ml) was administrated as the final dose and continued to the end of lactation period. After delivery, pups were investigated for mortality and their sex, congenital malformation, skull dimensions, and wet weight of the brain and volume of the brain. The duration of hair growth and eye opening was also considered. The results indicated that sex ratio (male/female) was 1.40 and 1.54 in experimental and control groups respectively. No congenital malformation was also observed. Meanwhile, weight in experimental group gradually significantly decreased to the end of lactation period. Duration of hair growing and eye opening of experimental group also showed a 1-day delay as compared to control group. At postnatal day 14, skull height in male significantly decreased, while skull width significantly increased in female. Wet weight of the brain significantly increased in both male and female rats. Volume of the brain also significantly decreased in male and female rats. At postnatal day 21, skull length in female significantly decreased. Volume of the brain also significantly decreased in male and female rats. It is concluded that the maximal effect of oral morphine administration on the developmental of the brain may appear at postnatal day 14 in rats.

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Microglia as a stem cell

F Sabouni; M Firouzi; Sh Abbasi

Abstract

Microglia is considered the only cell population of mesodermal origin, which comprises 10% of the cells in brain parenchyma. Recent neural stem cell (NSC) studies demonstrate that the brain has regenerative potential. NSCs do not give rise to microglial cells, however indicating that NSCs alone cannot complete the regenetion of the brain. Although the role of microglia is not fully understood, the recent study demonstrated that rat primary microglia cell expressed nestin, A2B5, and O4 antigens, which are markers for oligodendrocyte precursor cells. In our study, in purified primary cell culture microglia cells could be differentiated into other glial and neural cells.

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Stereological study of the cerebral and cerebellar cortex following short time exposure to morphine in rats

F Mohammad Ghasemy; A Dezfulian; B Shohany

Abstract

Drug abuse has been claimed to cause the loss of neurons and reduction of volume in both the adult and developing brain. This study examined cortex volumes in cerebrum and cerebellum of male rats that were dependent on morphine using unbiased stereological counting methods and systematic random sampling technique. Our goal was to test whether short time exposure to morphine can affect the cortex volume in the cerebrum and cerebellum. In this study, 30 NMRI male rats (3 month old and 110.36 ± 1.7 g) were used and animals randomly divided into three groups. The first group received no drug, while the second group received saline, and the third group received morphine + saline. Animals in the second and third groups were treated with saline or with an increasing dose regimen of morphine (5-25 mg/kg, twice a day) subcutaneously for 5 days. After confirming that the animals show withdrawal symptoms (12 h after the last injection), animals were anesthetized with an intraperitoneal injection with phenobarbital and killed by intracardiac perfusion with formalin in phosphate buffer. Then, the cerebellum and the cerebrum were carefully removed from the skull and embedded in 7% agar solution. The unbiased stereological procedure known as the Cavalieri principle was used to estimate the volume of cortex in the cerebellum and cerebrum of each animal. There were no significant statistical differences in volume of the cortex in cerebellum and cerebrum between groups. It is concluded that morphine has not toxic effect on the neurons body in the cortex of cerebrum and cerebellum after its short exposure.

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The role of spinal serotonergic system in morphine withdrawal syndrome in the rat

A Mohajjel Nayebi; D Pourfarhad

Abstract

Previous pharmacological studies have implicated serotonergic brain systems in opiate withdrawal syndrome. Increased brain 5-HT release is associated with the development of physical dependence to morphine. Specific serotonin reuptake inhibitors, such as fluvoxamine and sertraline reduce the severity of naloxone precipitated opioid withdrawal syndrome. Other studies have shown that 5-HT system is not directly responsible for the development of the withdrawal syndrome in morphine-dependent rats. In view of these evidences, the exact role of the spinal 5-HT systems remains unclear. This study aimed to investigate the effect of spinal serotonin on the morphine withdrawal syndrome. A total of 42 male Wistar rats weighing 220-250 g were used. Physical dependence was induced by i.p. injection of morphine at a schedule of 5 mg/kg for 3 days, 7.5 mg/kg for 2 days and 10 mg/kg for 2 days. Naloxone (1 mg/kg, i.p.) precipitated withdrawal behaviors such as jumping, tremor, abdominal writhing and wet dog shake were assessed 15 min after naloxone injection for 30 min. Results obtained from this study showed that 5-HT significantly attenuates ($P<0.01$) all of the above behaviors, both in chronic (200 µg/rat, i.t. for 7 days) and acute (200 µg/rat, i.t.) administration. Lesion of serotoninergic neurons by 5,7-DHT (100 µg/rat, i.t.) had not significant effect on jumping and wet dog shake behaviors, but decreased ($P<0.05$) severity of abdominal writhing and tremor. The results may indicate that morphine withdrawal syndrome is mediated through functional alterations in spinal cord serotoninergic system.

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The effect of ketamine and midazolam on morphine dependence and tolerance in mice

B Habibi Asl; K Hassanzadeh

Abstract

The aim of this study was to investigate the effect of ketamine and midazolam on preventing the development of morphine tolerance and dependence and withdrawal syndrome in mice. In the present study different groups of mice were received morphine (50 mg/kg, s.c.), morphine (50 mg/kg, s.c.) + ketamine (25, 50, 75 mg/kg, i.p.) , morphine (50 mg/kg , s.c.) + midazolam (0.5, 1, 2 mg/kg, i.p.) , morphine (50 mg/kg, s.c.) + [Ketamine (50 mg/kg, ip) + midazolam (1 mg/kg)] once a day for four days. Tolerance was assessed by administration of morphine (9mg/kg, ip) on fifth day. Withdrawal symptoms were assessed by administration of naloxone (4 mg/kg, i.p.) 2 hour after co-administration of morphine either with ketamine or with midazolam groups. The results showed that pretreatment with ketamine or midazolam decreases the degree of tolerance and withdrawal symptoms. On the other hand, co-administration of ketamine and midazolam before morphine treatment significantly decreased the tolerance and dependence and withdrawal symptoms. Ketamine and midazolam alone or in combination could also prevent the development of morphine tolerance and withdrawal symptoms. These effects can be attributed to NMDA-antagonistic behavior of ketamine and GABA-agonist behavior of midazolam. Furthermore, co-administration of ketamine and midazolam may exert its effect through two pathways, significantly preventing the development of morphine tolerance and withdrawal symptoms.

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The effect of magnesium and bromocriptine on morphine induced dependence and withdrawal symptoms in mice

B Habibi Asl; K Hassanzadeh

Abstract

The aim of this study was to investigate the effects of magnesium as a N-Methyl–D-Aspartate (NMDA) receptor Antagnist and bromocriptine as a dopamine receptor agonist on morphine dependence and withdrawal symptoms. In the present study different groups of mice were received morphine (50 mg/kg, i.p.) for four days and on fourth day 1.5 hour after the last morphine administration they received different doses of magnesium (40, 60, 80 mg/kg, i.p.) or bromocriptine (5, 10, 20 mg/kg, i.p.) and 0.5 hour later naloxan (4 mg/kg, i.p.) was administrated and withdrawal symptoms was assessed during 30 min. This study shows that administration of magnesium or bromocriptine has decreased the morphine-dependence and withdrawal symptoms significantly.

Magnesium can prevent the development of morphine tolerance and dependence. The mechanism of this effect is related to the property of magnesium to block the NMDA receptors. On the other hand, bromocriptine decreased the withdrawal symptoms and this mechanism may be related to dopamine agonist and ?2 agonist behavior of bromocriptine.

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Interactive effect of two treatment methods on the reduction of withdrawal syndrome signs in male rats

H Jafari; E Abbasi; R Gharebaghi

Abstract

Drug addiction is a destructive misfortune that leads to recession in all social fields. For this reason, proper treatment of addiction should be seriously considered. Formerly, the effectiveness of TENS and ICWS methods on the reduction of withdrawal syndrome signs in the rat was evaluated separately. In this study the interaction of these two methods were analyzed. For this purpose, four groups of rats (Spruge-Dawley) made dependent using injection of morphine (s.c.). Then, rats were exposed to high frequency TENS and ICWS, alone or in combination. In all groups, naloxone was injected (i.p.) to induce withdrawal syndrome. Statistical analysis was performed using ANOVA, Kruskal-Wallis, and Mann-Witney. The results showed that TENS method was very effective to reduce jumping and ptosis. In contrast, using ICWS a significant reduction in diarrhea, weight loss, yawning and abnormal posture was observed. Using both methods, only few signs were reduced. It is recommended to use these methods in clinical trials.

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The effect of gabapentin on the withdrawal signs in morphine-dependent male rats

Gh Sepehri; M Shamsi Meimandi; MMobasher; N Ashrafgangui

Abstract

Acute morphine withdrawal is considered to be the physical manifestation of dependence, emerging when morphine administration is stopped or after the administration of an opiate antagonist, such as naloxone. Gabapentin, which is an antiepileptic, agent, enhances the analgesic effect of morphine in healthy volunteers, but its effect on morphine dependence and withdrawal signs has not determined yet. This study was performed to evaluate the effects of gabapentin on withdrawal signs in morphine dependent male rats. Four groups of male rats (n=32) weighing 220-250g tested for withdrawal signs: control, morphine, gabapentin and gabapentin-morphine treated. Rats received morphine (10 mg/kg, s.c.), gabapentin or both of them twice a day for 9 days. Control rats received normal saline. On the 9th day, two hours after the last dose of morphine the rats were challenged for withdrawal by administration of naloxone (2.5 mg/kg, i.p.) and observed for various withdrawal signs such as diarrhea, jumping, writhing, and fore paw tremor over a period of 20 minutes. All of the withdrawal signs were observed in morphine dependent rats after naloxone administration. In gabapentin-morphine treated rats, writhing and diarrhea were significantly increased while jumping, fore paw tremor and weight loss were attenuated as compared to saline treated rats. The results of this study showed that co-administration of gabapentin attenuated some withdrawal signs such as jumping, fore paw tremor and weight loss in morphine dependent rats, while diarrhea and writhing significantly increased.

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Noxious behaviors from subcutaneous injection of formalin and morphine tolerance effects on the responses of addicted male rats

N Gheibi; M Javdan

Abstract

Formalin as a chemical noxious stimulus evokes a biphasic pain that its second phase as a chronic inflammatory pain is similar to clinical pain. In this study, we used 40 NMRI male rats (250-300 g), and experiments were carried out considering ethical rules. Experiments were performed in normal and morphine addicted rats, the addiction was induced by Leung method. Behaviors of flinching and licking scored as quantifying pain after injection of 50 ?l formalin (2.5%) into plantar region of hind paw. Assessment of morphine tolerance was carried out by intraperitoneal injection of morphine (10 mg/Kg) 10 minute before formalin injection. In the control group (normal rats, n=10) we observed a biphasic model of formalin test, but in the other normal group (n=10) morphine injection depressed noxious behaviors. In the addicted groups (n=10) flinching and licking responses evoked by formalin showed a biphasic model and morphine did not affect their behaviors. Only licking scores in addicted rats have significant difference from normal rats. Addicted rats showed tolerance to morphine analgesia after formalin injection.

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The effect of parental morphine addiction on extracellular glutamate concentration of dentate gyrus in rat offspring: a microdialysis study

R Assaee; A Sarkaki; N Pajouhi; M Badavi; H Assaee

Abstract

Addiction is a major problem of society. The existing evidences have shown that parental addiction impairs CNS development, learning, and memory. With respect to the role of glutamate in dentate gyrus on learning and memory, in this study, the effect of parental addiction to morphine on extracellular glutamate concentration in dentate gyrus was evaluated. For this purpose, male and female rats were addicted following 5-days oral administration of morphine (s.c.) and subsequently exposed to non-addicted female and male rats respectively. Basal and perforant path electrical stimulating glutamate concentration in the dialysate from dentate gyrus was measured. The results showed that the extracellular basal and stimulated glutamate concentration decreases in both female- and male-addicted groups. The concentrations were lower in mother-addicted female offspring than male offspring. These results suggest that parental addiction may cause learning and memory deficits. Therefore, general attention should be paid to this issue.

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The effect of gabapentin on withdrawal syndrome, personality disorders and electroencephalogram of opium addicts during the detoxification period

M Mobasher; H Ziaaddini; A Hamzeii Moghaddam; F Sabzvari; S Sadeghipour

Abstract

Substance abuse is an important hygienic, psychic and social problem in the world. Gabapentin is a new antiepileptic drug that is used in neurological and psychiatric disorders. Moreover, the effects of gabapentin on increasing the analgesic effect of morphine and its inhibitory effect on dopamine release due to morphine in animal models have been proved. In the present study, the effect of gabapentin on withdrawal signs and symptoms in opium-addicted subjects and on psychiatric disorders and electroencephalogram of these patients during the detoxification period have been investigated. Two groups of patients were selected randomly. The first group (n= 36) received the current drugs based on their withdrawal symptoms and the second group (n= 35) received 300 mg gabapentin every 8 hours additionally. All patients were evaluated by electroencephalography and SCL-90 test on first and last days of hospitalization and their demographic characteristics were gathered by using a general questionnaire. During the hospitalization period (10 days) all subjects were assessed for withdrawal symptoms and signs. The analysis of data showed the excellent effect of gabapentin on all psychiatric symptoms and in decreasing signs and symptoms significantly. Gradual decrease of withdrawal symptoms and signs in the second group shows the efficacy of gabapentin. There was no significant difference between the two groups in regard to the electroencephalogram indices. The results showed that gabapentin improves the quality of therapeutic management in opium-addicts during the detoxification period.

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Inhibition of morphine tolerance within the rat nucleus accumbens by nitric oxide

A Noroozzadeh; A Khoshbaten; F Bahrami; H Ghoshooni; H Sahraei; S Bijani; S Oryan; M Eidi

Abstract

The role of glutamate receptors within the nucleus accumbens in morphine tolerance has been postulated. Previous studies have reported that glutamate receptors exert their effects in part through the release of nitric oxide (NO). In the present study, the effect of intra-accumbal injections of L-arginine, the NO precursor and L-NAME, the NOS inhibitor on the morphine tolerance in Wistar rats (250-300 g) were investigated. For this purpose, male rats (n = 8/group) were anesthetized and bilateral cannulation (23-gauge, AP=1.7mm, L=±0.8, V= 7.1 mm) was performed into nucleus accumbens. Five days after cannulation, animals were trained in an Un-Biased conditioned place preference apparatus for five consecutive days. The NOergic drugs were injected in two ways: first; the animals were trained with morphine and were received L-arginine or L-NAME at 5th day of experiments just before the test. Second group received L-arginine or L-NAME before morphine injection. At the 5th day of the experiments, each animal was placed in the apparatus and its behavior was recorded for 10 min. The results showed that pretreatment of the animals with L-arginine did not produce any effect on morphine tolerance. Pretreatment of the animals with L-NAME potentiated both development and expression of morphine tolerance. In conclusion, present experiments showed that morphine tolerance is in part dependent on activation of NO system within the nucleus accumbens and the role of this neuromodulator in morphine dependence must be considered in further treatments of morphine addicts.

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The effect of alloxan-induced diabetes on anti-nociception and on the development of morphine tolerance and dependence in rats

M Jorjani; Kh Joharchi

Abstract

Diabetes is one of the most common diseases in all societies including Iran. One of its important complications is the neuropathic pain, which can be relieved by opioid drugs such as morphine. Opioid therapy is restricted due to development of tolerance and physical or mental dependence. In this study, the effect of diabetes on morphine analgesia and development of morphine tolerance and dependence was investigated. Experimental diabetes was induced by alloxan (120 mg/kg, s.c.) in rats. Morphine sulfate (7 mg/kg, i.p.) application for 5 days developed tolerance in animals. On 5th day, 30 min after the injection of morphine, the acute and chronic pain was evaluated in diabetic and non-diabetic animals using hot plate and formalin test. In addition, withdrawal signs (jumping, chewing, urine and feces) were recorded for ten minutes using naloxone (2 mg/kg, s.c.). The results showed that the anti-nociceptive effect of morphine for acute pain markedly reduced, but slightly enhanced for chronic pain model. The withdrawal signs significantly decreased in diabetic animals. It seems that in diabetic state, an endogenous analgesic system is activated to potentiate the opioid effects. Further investigation is needed to clarify these mechanisms.

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Nitric oxide within the rat hippocampal CA1 area may play a role in morphine tolerance

A Harati-Kia; A Khoshbaten; F Bahrami; A Noroozzadeh; H Ghoshooni; H Sahraei; S Oryan; M Eidi

Abstract

Hippocampus as part of the limbic system plays an important role in abused drugs-induced memory. The role of glutamate receptor within the hippocampal CA1 area in morphine-induced memory has also been postulated. Previous studies indicated that glutamate receptors exert their effects in part through the release of nitric oxide (NO). In the present study, the effects of intra-CA1 area injections of L-arginine (0.3, 1, and 3 ?g/rat), the NO precursor and L-NAME (0.3, 1, and 3 ?g/rat), the NOS inhibitor on the morphine tolerance in Wistar rats (250-300 g) were investigated. Male rats (n = 8/group) were anesthetized and bilateral cannulation in nucleus accumbens (21-gauge, AP = -3.8mm, L = ±1.8, V = 2.5 mm) was performed. Five days after cannulation, animals were trained in an Un-Biased conditioned place preference apparatus for five consecutive days. The NOergic drugs were injected in two ways: first; the animals were trained with morphine and were received L-arginine or L-NAME at 5th day of experiments just before the test. Second group received L-arginine or L-NAME before morphine injection. At the 5th day of the experiments, each animal was placed in the apparatus and its behavior was recorded for 10 min. The results showed that pretreatment of the animals with L-arginine inhibit morphine tolerance. Pretreatment of the animals with L-NAME did not show any effect on morphine tolerance. In conclusion, present experiments showed that morphine tolerance is in part dependent to NO system activation within the hippocampus CA1 area and the role of this neuromodulator in morphine tolerance must be considered in further treatments of morphine addicts.

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Involvement of nitric oxide within the rat central nucleus of amygdala in morphine tolerance

F Zarei; A Noroozzadeh; A Khoshbaten; F Bahrami; H Ghoshooni; H Sahraei; S Oryan; M Eidi

Abstract

The role of glutamate receptor within the nucleus accumbens in morphine tolerance has been postulated. Previous studies have reported that glutamate receptors exert their effects in part through the release of nitric oxide (NO). In the present study the effects of intra-accumbal injections of L-arginine (0.3, 1, and 3 ?g/rat), the NO precursor and L-NAME (0.3, 1, and 3 ?g/rat), the NOS inhibitor on the morphine tolerance were investigated in Wistar rats (250-300 g). Male rats (n = 8/group) were anesthetized and bilateral cannulation in nucleus accumbens (23-gauge, AP = 1.7 mm, L = ±0.8, V = 7.1 mm) was performed. Five days after cannulation, animals were trained in an Un-Biased conditioned place preference apparatus for five conscutive days. The NOergic drugs were injected to the animals in two ways: first; the animals were trained with morphine and were received L-arginine or L-NAME at 5th day of experiments just before the test. Second group received L-arginine or L-NAME before morphine injection. At 5th day of the experiments, each animal was placed in the apparatus and its behavior was recorded for 10 min. The results showed that pretreatment of the animals with L-arginine have not showed any effects on morphine tolerance. Pretreatment of the animals with L-NAME potentiated both development and expression of morphine tolerance. In conclusion, present experiments showed that morphine tolerance is in part dependent to the activation of NO system within the nucleus accumbens and the role of this neuromodulator in morphine tolerance must be considered in further treatments of morphine addicts.

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Assessment of brain laterality among Iranian addicts and smokers

S Najafi; C Jalili; A Khodaei; M Yousefpour; S Riahi; H Sahraei; L Etemadi; S Hassankhani; M Karimi; H Moinipour; F Khaleghi; Sh Rahgozar; A Sabaei; S Rajezi

Abstract

Previous research supported that the prevalence of addiction is related to brain laterality. Brain laterality is often determined by left/right handedness and this phenomenon is considered as an important factor in addiction prevalence. The present study attempts to examine the relationships between left/right handedness with addiction and smoking on Iranian population. In this study, 2000 male and female non-addicted (age 31±3 years old) and 1000 male and female addicted (age 42±4 years old) were examined for left/right handedness. The results showed that: right versa left handedness percentages for normal subjects were 91.3% to 8.7% respectively, while for the addict and smoker group were 79.3% to 20.7% respectively. These data indicated that the prevalence of addiction and smoking among left-handed subjects was very higher than right-handed group ($P<0.001$). In conclusion, it is clear that the risk of addiction is higher in left-handed individuals, which is consistent with previous studies.

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Swim stress decrease the development of morphine tolerance apart from nitric oxide inhibition

FG Davoodi; A Ahmadiani; M Javan

Abstract

Stress and chronic pain have been shown to prevent the development of tolerance to morphine analgesia, which appears to be related to neuroendocrine activity and alternation in neurochemicals. Also the involvement of nitric oxide (NO) has been implicated in tolerance to morphine analgesia. In our pervious study, we showed that co-administration of swim stress (ss) with chronic morphine, prevents the development of morphine tolerance. In this study the probable interactions between swim stress and nitric oxide level on development of morphine tolerance were investigated. Adult male NMRI rats weighing 180-220 g were divided into control and experimental groups (N = 8) that received morphine 20 mg/kg (i.p.) for 4 days, swim stress 4 minutes for 4 days at 20 ?C water and combination of both swim stress and morphine injection for 4 days. Nitric oxide was measured as indicator of NO by Griess methods. Swim stress raised NO level ($P<0.001$). Combination of morphine injection and swim stress significantly decreased nitric oxide level compared to chronic morphine treated group ($P<0.001$). These data suggest that at least two parallel systems may be activated during stress: 1. inbibition of morphine tolerance may be mediated by stress via activation of HPA axis as described by other reports in case of pain stress, and HPA axis activation in tolerance prevention, and 2. suppression of nitric oxide synthase (NOS) activity.

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Evaluation of life quality in children with epileptic diseases

M Nobahar; AA Vafaei; A Samaei

Abstract

Epilepsy is one of the disorders with chronic, recurrent and sudden changes in neurological function due to electrical dysfunction of brain. Previous studies have indicated that this disorder may affect the quality of life and could change it. The aim of this study was evaluation of life quality in children with epileptic diseases. This is a cohort and clinical trial study. Demographic data include age, sex and… data is gathered regarding quality of life by questionnaire. The results indicated that the mean age was 13 years old, 57% were female and 27% had family history of epilepsy. Also 45% had psychological problems (Anxiety, Depression) 16% had familial problems (regarding brothers and sisters) and 35% had social problems (regarding classmates and teachers) and 16% had a physical problem (Fatigue, weakness). There was not significant correlation ($P>0.05$) between the quality of life and age, sex and other factors. Above findings show that epileptic children and their family are not able to adapt with their disease. Also they have a lot of problem in their life that can create stressful situation for them.

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Epilepsy and psychosis

M Al-Kureishi

Abstract

This study reports the difficulties in the treatment and follow-up of patients with epilepsy and psychotic disorder and the orientation of neurologists about lifelong chronic effect of epilepsy towards psychiatric morbidity. Six patients (2 females, 4 males) complaining of generalized epilepsy, where the disease had started in the adulthood, for more than ten years, and suffered more than ten seizures per year, developed one of psychotic disorders. They are visitors of private clinics between 1999- 2003 as well as admission to psychiatric hospitals. They were assessed by clinical diagnosis according to criteria of DSM-IV, positive and negative syndrome scale (PANSS), magnetic resonance imaging study, and electroencephalography. Neuroleptics and antiepileptics were used with close observation and good cooperation with referring neurologists and families of all patients. Good control of both epileptic seizures and psychotic symptoms obtained in five of cases by equilibrium of the effect of neuroleptics and antiepileptics. Three cases needed repetitive hospitalization in psychiatric units. All of them re-monitored by PANSS and showed score reduction and re-integrated well to their usual environment. The sixth patient (a case of previous head injury) developed a very severe paranoid schizophrenia with aggressive attitude ended with long hospitalization in asylum. It can be concluded that mixed cases of epilepsy and psychosis are a challenge for neurologists and psychiatrists. Thus, there is a great need for close cooperation between them to point out psychiatric results of epilepsy and to study the epileptogenic effect of anti-psychotic drugs. All of these efforts are the corner stone to improve the quality of life of the patient and perform reintegration to the environment.

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Retinol and ?-Carotene inhibit PTZ-induced kindling in mice

M Yousefi-Pour; M Sayyah; J Narenjkar

Abstract

Vitamin A and its derivatives have recently reported to be implicated in synaptic plasticity. The possible effect of vitamin A and its precursor, ?-carotene, on seizure acquisition was tested in PTZ kindling model of epilepsy. Vitamin A and ?-carotene were tested for their ability to 1) suppress seizures (clonic and tonic) and lethality induced by PTZ in PTZ-kindled mice (anticonvulsant effect) and 2) attenuate the development of sensitization to convulsive and lethal effects of PTZ in kindled mice (anti-epileptogenic effect) when given as a pretreatment prior to each PTZ injection during kindling acquisition. Diazepam was used as positive control group. All these drugs showed anti-epileptogenic effects against PTZ-induced tonic seizures and lethality. Vitamin A and ?-carotene had no effect on clonic seizures induced by PTZ in kindled mice. It can be concluded that vitamin A and ?-carotene possess anti-epileptogenic activity. Retinoids appear to be valuable for development of a new class of anticonvulsant drugs.

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Anticonvulsant effects of intrahippocacampal N6-cyclohexyladenosine on piriform cortex-kindled seizures

M Zeraati; J Mirnajafizadeh; Y Fathollahi; S Namvar

Abstract

In this study the role of adenosine A1 receptors of the CA1 region of hippocampus on piriform cortex–kindled seizures was investigated in rats. Obtained results showed that in kindled animals, bilateral microinjection of N6–cyclohexyladenosine (CHA), an adenosine A1 receptor agonist, at doses of 10 and 100 mM into the hippocampal CA1 region decreased the afterdischarge duration (ADD), stage 5 seizure duration (S5D) and seizure duration (SD), and increased the latency to stage 4 (S4L) significantly. CHA at the doses of 1 mM decreased only ADD and no changes were observed at the doses of 0.1 mM. Bilateral microinjection of 1,3-dimethyl-8-cyclopenthylxanthine (CPT), an adenosine A1 receptor antagonist, into the CA1 region of hippocampus increased ADD at the dose of 20 mM significantly, but has no effect on seizure parameters at the dose of 10 mM. Pretreatment of animals with CPT (10 mM) before CHA (10 and 100 mM), reduced the effect of CHA on seizure parameters significantly. Thus, it may be suggested that the hippocampal CA1 region may play a role in relaying or spreading of seizure spikes elicited from the piriform cortex, and activation of adenosine A1 receptors of this region can prevent this actions.

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Dose-dependent effects of morphine on hippocampal seizure

E Dodangeh; M Derchanski; SS Jahromi; P Carlen

Abstract

Opiates have complex effects on seizure thresholds as these substances have both anti and proconvulsive actions in the mammalian brain. A reduction of inhibitory synaptic activity or enhancement of excitatory synaptic activity would be expected to trigger a seizure. This study is designed to determine how morphine and naloxone affect seizures induced by a low Mg2+ perfusate in the whole, intact hippocampus of mouse in vitro. In this research, C57BL/ 6 mice (P11-17) were used. Intact hippocampal structures were carefully dissected and incubated for at least 1 hour prior to recording. The tissues were perfused by low Mg2+ (0.2 mM)-ACSF at a rate of 3 ml/min. Morphine sulfate was used in various doses (10, 30, 100, 200 µM) as an opioid receptor agonist, while naloxone (5 and 10 µM) was utilized as an antagonist of these receptors. Various concentrations of morphine sulfate showed variable effects on whole hippocampus activity: Morphine (30 and 100 µM) noticeably elongated the length of epileptiform activity, while decreasing the frequency of the tonic stage of seizures. Morphine (200 µM) attenuated the hippocampal seizure activities. Low concentrations (5 µM) of naloxone did not show any significant effect on hippocampal activity, but higher concentrations of naloxone (10 µM) depressed both interictal and ictal like events. When combining perfusion with morphine (10, 30, 100, and 200) and naloxone (10 µM), both interictal and ictal-like events were depressed. Our results indicate that morphine in therapeutic doses of 30 µM may enhance seizure activity and its use in epileptic patients should be considered with caution. Conversely, naloxone has anticonvulsant actions.

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Topographical evaluation of aphasia based on brain vascular territories

K Ghandehari; S Ghandehari

Abstract

Topographical evaluation of aphasic brain lesions can enhance our knowledge of cognitive physiology and plasticity. This prospective study was conducted on 100 stroke-afflicted patients with aphasia admitted in Valie-Asr Hospital (Khorasan, Iran) in 2003. Topography of infarct lesions was detected by a neurologist based on the map of brain vascular territories in CT-scan. Aphasic lesions categorized in anterior cerebral artery (ACA), posterior cerebral artery (PCA), middle cerebral artery (MCA), total MCA, anterior cortical MCA, Posterior Cortical MCA and deep MCA territories. The ethics committee of the university approved this research project. The results showed that of a total of 52 patients, global aphasia in MCA, ACA, PCA and MCA+ACA territories existed in 88%, 4%, 4%, and 4% respectively. All of the 40 patients with Broca aphasia had infarct in MCA territory and in 75% of the patient's anterior cortical MCA territory was involved. Furthermore, out of 6 patients evaluated, Wernicke aphasia with involvement of posterior cortical MCA and PCA territories existed in 66% and 16% of the patients respectively. Atypical topography of the lesions was found in 11% out of global and 27% out of Wernicke aphasias.

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Using functional magnetic resonance imaging (fMRI) to explore brain function: cortical representations of language critical areas

H Saberi; A Mahdavi; A Rezvanizadeh; MA Oghabian; N Riahi; A Mojebi; A Lavasani

Abstract

Pre-operative determination of the dominant hemisphere for speech and speech associated sensory and motor regions has been of great interest for the neurological surgeons. This dilemma has been of at most importance, but difficult to achieve, requiring either invasive (Wada test) or non-invasive methods (Brain Mapping). In the present study we have employed functional Magnetic Resonance Imaging (fMRI) to observe and delineate regional brain activations during execution of language related tasks. Our healthy volunteers comprised of 10 right-handed males (handedness being ascertained by "Snyder-Harris Handedness Inventory") all speaking Persian as their native language. All the subjects performed two consequent language tasks namely "Word Generation" and" Reverse Word Reading". The visual stimulation was performed employing a video projector and the presentation software, while the brain activity was monitored and studied by fMRI. The stimuli were given during the activation period, while the asterix had been used as the blank (rest period). The subject response was internal speech. The fMRI method employed, was Echo Planar Imaging (EPI) using FSL as the analyzing software. The Hardware comprised of 1.5 Tesla GE brand MRI scanner. The brain regions involved language processing could be successfully and prominently activated with the aforementioned tasks (percent activity 1.2% and $P<0.005$). In all of the ten subjects being examined, these regions were exclusively located in the left hemisphere corresponding to Broca, Wernicke, and Exner areas. These promising results may be of value to determine the dominant hemisphere by a non-invasive method, or as an adjunct to conventional methods of Electrocortical Mapping and Wada test. Besides, the presented visual tasks could specifically activate the traditional language centers even those known to be involved in writing (Exner area). Estimation of the exact sensitivity and specificity of the methods requires employing a gold standard methods and larger subject populations.

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Caffeine attenuates paradoxical sleep deprivation induced- memory impairment during paradoxical sleep windows in rats

E Nabai; Sh Mohammadian; F Motamedi

Abstract

There is considerable evidence to support the hypothesis of relationship between paradoxical sleep (PS) and learning–memory processing. It has been suggested that PS is important in memory retention at the specific time course called PS windows (PSW). The time of PSWs occurrence and duration of these PSWs following the training sessions and, the neurochemical nature of PSWs has not been well known. In this research the duration of PSWs and the effect of caffeine on memory retention in these periods has been investigated. For this purpose, male NMRI rats were trained in a two–way shuttle avoidance task (100 trials in one session) and their memory retention was tested one week after learning. In experiment 1, the baseline EEG and EMG activity 1-4 hr after training was recorded. In those animals that reached to 70% learning criteria (learner group), the total duration of PS increased significantly ($P<0.01$) 1-4 hr after training. This change was not observed in the non-learner group. In experiment 2, the learner group was made deprived of PS in the two periods of 1-4 hr and 5-8 hr after learning. The memory retention was significantly impaired in 1-4 hr group ($P<0.05$) but not in the 5-8 hr group. In experiment 3, caffeine (25 mg/kg) was injected i.p 1-4 hr after training to the learner group with or without PS deprivation. Injection of caffeine increased memory significantly ($P<0.05$), but in PS deprived rats caffeine had no effect on memory retention. In experiment 4, adenosine (7 and 50 mg/kg), physostigmine (0.1 mg/kg) and scopolamine (5 mg/kg) were administered i.p 1-4 hr after training. None of these drugs had a significant effect on memory retention. According to our data, it seems that 1-4 hr period after training could be considered as a PSW in this condition, in which caffeine can increase memory which is not due to cholinergic and adenosine systems. It is concluded that the memory processing that occurs in PSWs could not be attributed to one neurotransmitter system.

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Interaction of vitamin E and scopolamine on memory retention in male Wistar rats

S Oryan; A Eidi; M Eidi; G Mahmoodi

Abstract

Vitamin E (vit. E) is an antioxidant compound with different kinds including tocopherols and tocotrienols. The major sources of vit. E are vegetable and seed oils. The recommended dietary allowances vary with age, gender, and state of the person. There have been numerous experiments showing beneficial effects of vit. E on immune system, cancer, and coronary artery diseases in the elderly. Moreover, vit. E in general appears to play a beneficial role in maintaining human good health. The aim of this study is to define the interaction of vit. E and scopolamine on memory retention. For this purpose a permanent guide cannula was implanted stereotaxically within the right lateral ventricle of adult male rats. After recovery period, animals received habituation and training sessions by passive avoidance task. The drugs were injected after shock intracerebroventricularly. The effect of these drugs on memory retention was evaluated after 24h. The results showed that scopolamine, the antagonist of muscarinic receptor (1, 5 ?g/rat) decreases memory retention, while vit. E (50 ?g/rat) potentiates this response. The pretreatment of scopolamine (0.1, 1, and 5 ?g/rat) attenuated increasing response induced by vit. E (50 ?g/rat). The results indicated that there is a close interaction between scopolamine and vit. E. In addition, vit. E increases acetylcholine release in the brain and improves memory retention.

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The effect of vitamin E and prazocin on memory retention in adult male rats

S Oryan; A Eidi; M Eidi; B Ghorbanalizadeh

Abstract

Both aging and age-associated neurodegenerative diseases are related with various degrees of behavioral impairments, and among the prime candidates responsible for producing the neuronal changes mediating these behavioral deficits, appear to be free radicals and the oxidative agents which they generate. Free radicals such as vit. E may be important factors in maintaining neuronal integrity and preventing cell death. In this research, effects of vit. E on memory retention was evaluated. vit. E (10, 20, and 50 ?g/rat) and prazocin (0.1, 1, and 0.5 ?g/rat) were injected intracerebroventricularly. The effect of drugs on memory retention was studied by passive avoidance task and time of step-through latency (STL) was measured. The results indicated that vit. E (50 ?g/rat) increases and prazocin (0.5, and 1 ?g/rat) decreases memory retention in a dose - dependent manner. The pretreatment of prazocin (0.1, 0.5, and 1 ?g/rat) attenuated increasing response induced by vit. E (50 ?g/rat). The results showed that vit.E may induce its memory retention effects through activation of ?1-adrenoceptors.

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The effect of acute restraint stress and dexamethasone on retrieval of long-term memory in rats: an interaction with opiate system

; H Sadeghi; AA Taherian; AA Vafaei; Y Fathollahi

Abstract

This study investigated whether application of acute restraint stress or dexamethasone, as a glucocorticoid receptor agonist, impaired retrieval of long-term memory and if pretreatment with opiate antagonist naloxone blocked their effects on memory retrieval. Young adult male rats were trained in one trial inhibitory avoidance task (1 mA, 1.5 s foot shock). On retention test given 48 hr after training, the latency to re-enter dark compartment of the apparatus was recorded. Thirty min before retention test, the rats were exposed to 10 min of restraint stress in a Plexiglas tube or were injected with dexamethasone (1 mg/kg) with or without prior treatment of naloxone (1 or 2 mg/kg). The results showed that both acute restraint stress and dexamethasone impaired retention performance, and their effects were blocked by naloxone pretreatment. The applied stress increased circulating corticosterone levels as assessed immediately after the retention test, indicating that stress–induced impairment of memory retrieval mediates, in part, by increasing plasma levels of glucocorticoid. These findings further indicate that acute restraint stress and glucocorticoids impair retrieval of long-term memory, and provide evidence for the existence of an interaction between glucocorticoids and opiate system on this process.

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An inward current induced by a putative cyclic nucleotide-gated channel in rat cerebellar Purkinje neurons

SM Tsoi; WH Yung

Abstract

The roles of cyclic nucleotide-gated (CNG) channels in sensory transduction have long been recognized. More recent studies found that CNG channels are distributed in multiple brain regions involved in memory and learning, including the cortex, hippocampus and cerebellum. These findings suggest that their functions are not limited to sensory perception, but also to neuronal plasticity phenomena, such as long-term potentiation (LTP) and long-term depression (LTD). Our studies using immunostaining have shown that there is a prominent specific expression of CNG channels in cerebellar Purkinje neurons. In order to demonstrate and characterize these CNG channels, we employed whole-cell patch technique to probe their functional expression in the cerebellar Purkinje neurons in brain slices prepared from 3 weeks old Sprague-Dawley rats. Bath application of the membrane permeable analog of cGMP, 8-Br-cGMP (1mM), consistently elicited an inward current of 156±14pA (n=14) in these neurons which were voltage-clamped at –70mV. The current was washable and repeatable. An inward current could also be induced by 8-Br-cAMP (1mM), a membrane permeable analog of another cyclic nucleotide, cAMP. To eliminate the possible contribution of protein kinase G (PKG) in mediating the inward current, 8-Br-cGMP was applied in the presence of KT5823 (2 ?m), a specific PKG inhibitor, in the internal solution. In this case, a comparable inward current was still evoked (197±31pA, n=6, p>0.05). In conclusion, these characteristics are consistent with those of CNG channels. This finding strengthens the speculation that CNG channels are expressed in the cerebellum and may contribute to neuronal plasticity phenomena of the cerebellum such as LTP and LTD.

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The effect of ketamine on NMDA receptor-mediated LTP depends on ketamine effects on non-NMDA-mediated synaptic transmission in CA1 area of rat hippocampal slices

B Rahmati; Y Fathollahi; S Semnanian; MRVaez Mahdavi; M Shafeezade

Abstract

It has been reported that ketamine as an uncompetitive N-methyl-D-aspartate (NMDA) receptor antagonist has also non-NMDA receptor antagonist properties. We recently found that ketamine (20 ?M) affected differently induction of NMDA receptor-mediated long-term potentiation (LTP) when administered 30 min prior to tetanic Primed-Bursts (PBs) stimulation. On the other hand, ketamine also influenced non-NMDA-mediated synaptic transmission in different manner. In the present study, in order to find the cause of different effects of ketamine on NMDA-mediated responses, we examined the probable relationship between the effects of ketamine on non-NMDA-mediated baseline responses and NMDA-mediated responses. Population spikes amplitude (PSA) was measured before and after tetanic stimulation in ketamine (1-100 µM) treated slices. We found that ketamine failed to inhibit NMDA receptor-mediated LTP, when baseline PSA enhanced in presence of ketamine. On the other hand, when ketamine not changed or decreased beseline PSA, caused LTP inhibition or even LTD induction. These findings indicated that ketamine effects on NMDA receptor-mediated responses depends on, ketamine effects on non-NMDA receptor-mediated synaptic transmission.

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A comparative study on the effect of intrahippocampal CA1 area injection of esradiol benzoate and sesame oil on learning and memory in adult male rats

R Hoveyda; AA Moazedi; A Rasekh

Abstract

Estrogen has a vast and complex role on higher brain abilities such as learning and memory. On the other hand, sesame oil (SO) (as a vehicle in the steroids structure) has a different effect on body function. So, in this study the effect of intrahippocampal injection of estradiol benzoate (EB) and SO on spatial learning and memory in adult NMRI male rats was investigated. The animals were anesthetized by ketamine (78 mg/kg) and xzylazine (3 mg/kg) and bilaterally microcannulated into the CA1 region of hippocampus, and after recovery they were divided into the following 4 groups (n = 7): control group (no injection) that was only trained in Y-maze. The SO test, physiological saline sham and EB test groups received 0.5 µl of SO, 0.5 µl of physiological saline and 1µg/0.5µl of EB respectively. The injections were made bilaterally into the CA1 region of hippocampus immediately before training. Then, each rat was trained in 30 trials every day for a total of 5 days with Y-maze. After one month, all of the experimental groups were tested (one session) for memory test. Statistical analysis of data using repeated measure design and least significant difference test showed a significant difference between EB and saline groups ($p<0.001$), but no significant difference between SO and saline groups was noted. Also, no significant difference between EB and SO groups was found out. These results indicate that EB increases and SO improves spatial learning tasks. Regarding memory test, there were no significant differences between learning results of 5th day and a month later in different groups. This may indicate that their memory has not changed. In conclusion, it seems that EB increases learning task through an interaction with cholinergic system and enhancement of synaptic plasticity in hippocampal CA1 area and SO increases learning task through its unsaturated fatty acids that may change neuronal membrane fluidity and through lecithin as a precursor of acetylcholine that may interact with cholinerigic system.

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The effect of chronic oral administration of verapamil on learning and retrieval in rats using passive avoidance learning task

R Lashgari; MSh Safari; F Motamedi; S Zahedi-Asl

Abstract

Verapamil is a drug that blocks L-type calcium channels and selectively inhibits calcium ion entry into the central and peripheral nervous system cells. Verapamil is used acutely and chronically in treatment of some cardiovascular and central nervous system disorders. It has also been shown that acute administration of verapamil has no significant effect on learning and memory in rats, but the effect of chronic administration of verapamil on learning and memory has not been fully investigated. Therefore, the purpose of this study was to evaluate the effect of chronic oral administration of verapmil on learning and memory in rats. Male Wistar rats, weighing 150-200 g were used. Rats were treated orally with verapamil for 60 days using oral tube. They were divided into four groups consisting of control and 10, 20, 50 mg/kg of verapamil. After 60 days of treatment with verapamil, animals were trained in a passive avoidance shuttle box, and 24 hours later their memory retrieval were tested. The data indicated that verapamil in all groups had no significant effect on acquisition, but verapamil at a low dose (10 mg/kg) and at a high dose (50 mg/kg) and not at moderate doses (20 mg/kg) significantly reduced memory retrieval ($P<0.05$ and $P<0.01$ respectively). These results suggest that chronic administration of verapamil has no significant effect on passive avoidance learning, but at high (50 mg/kg) and low (10 mg/kg) doses and not at moderate dose (20 mg/kg) impairs long term memory in passive avoidance task. It is concluded that verapamil at moderate dose seems to be safe for treatment of cardiovascular and CNS disorders, which needs to be further investigated.

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Deficits on passive avoidance learning after exposure to electromagnetic field in male rats

Sh Babri; N Khalaji

Abstract

The interaction of electromagnetic fields (EMF) with living organisms presents an increasing source of interest nowadays. There are reports on the influence of EMFs on memory. In this study, the effect of a 50 Hz, 50G EMF was investigated on passive avoidance learning in male rats. For this purpose, male Wistar rats (n = 50, 6-8 weeks, 250-300 g) were randomly divided into five groups. Control group trained in passive avoidance box and tested 24 hours later. Other groups exposed to EMF immediately after training for 1, 4, 6 and 8 hours respectively, and 24 hours later were tested. Data obtained show that increasing the time of exposure to EMF from 1 hour to 4 hours causes a significant reduction in retention latency ($P<0.05$) as compare to control group. It is concluded that EMF can disturb memory consolidation, but this disturbance is closely related to exposure time.

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Cysteamine pretreatment reduces Mg2+-free medium-induced plasticity in the CA1 region of the rat hippocampal slices

M Rostampour; Y Fathollahi; S Semnanian; S Hajizadeh; J Mirnajafizadeh

Abstract

Extracellular population responses are largely preferred for the study of long-term potentiation (LTP). The effect of Mg2+-free medium on changes in activity and plasticity of Schaffer collateral-CA1 pyramidal cell synapses was examined. Hippcampal slices from cysteamine-treated (200 mg/kg, s.c.) and saline-injected (1 ml/kg, s.c.) albino rats were perfused with ACSF. Population spikes (PS) were recorded in CA1 cell body layer following Schaffer-collateral stimulation. The results showed that Mg2+-free medium enhances the amplitude of population spike (PSA). At 60 min following Mg2+-free medium washout, an increase in PSA was observed at both groups. The extent of LTP of PSA was significantly lower in cysteamine-treated rats in comparison with saline-injected ones. It is concluded that the effect of cysteamine (a somatostatin depletor) can entail lasting modifications in susceptibility of hippocampal CA1 for synaptic plasticity induced by Mg2+-free medium. The relevancy of the results to the facilitatory role of endogenous somatostatin in the function of Schaffer collateral-CA1 pyramidal cell synapses should also be considered.

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Morphine sensitization and state-dependent learning in mice

A Rezayof; MR Zarrindast

Abstract

In the present study, the effects of morphine sensitization on morphine-induced impairment of memory formation and the state-dependent retrieval of a passive avoidance task learned under morphine influence have been investigated in mice. Pre-training administration of morphine (0.5, 2.5 and 5 mg/kg) dose dependently suppressed the learning of one-trial passive avoidance task. Pre-test administration of morphine (0.5, 2.5 and 5 mg/kg) induced state-dependent retrieval of the memory acquired under pre-training morphine influence which these effects of morphine is named state-dependent learning. The inhibitory effect of morphine (5 mg/kg) on memory formation was significantly antagonized by pretreatment administration of naloxone (0.025, 0.5 and 1 mg/kg) before the pre-training morphine. The pre-test administration of naloxone dose dependently inhibited the restoration induced by morphine (5 mg/kg). Amnesia induced by pre-training morphine significantly inhibited in morphine-sensitized mice, which had previously received once daily injections of morphine (20 and 30 mg/kg, s.c.) for 3 days. Morphine sensitization did not affect on morphine state-dependent memory of passive avoidance. The inhibition of morphine-induced amnesia in morphine-sensitized mice suppressed by once daily injections of naloxone (0.5, 1 and 2 mg/kg) 30 min prior to s.c. injection of morphine (20 mg/kg/day for 3 days). These results suggest that morphine sensitization affect the impairment of memory formation, but not the facilitation of retrieval induced by morphine.

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Attenuation of reserpine-induced perioral movements and memory dysfunction by natural antioxidants

PS Naidu; A Singh; SK Kulkarni

Abstract

Neuroleptics are widely prescribed drugs for the treatment of schizophrenia and related psychiatric disorders. Tardive dyskinesia (TD) is a late complication of prolonged neuroleptic treatment. Despite much research, the pathogenesis of TD remains elusive. It may be caused by loss of dopaminergic cells, due to free radicals as a product of high synaptic dopamine levels. Tardive dyskinesia has been associated with cognitive deficits. Reserpine-induced perioral movements (Vacuous chewing movements and tongue protrusions) were recently suggested as a new animal model for tardive dyskinesia. Natural antioxidants were administered for a period of 7 day prior to the reserpine administration and were co-administered along with reserpine for the next 5 days. Repeated treatment with reserpine alone (1 mg/kg) on each other day for a period of five days (1st, 3rd and 5th day) significantly induced perioral movements in rats. Antioxidants like melatonin (2.5 and 5 mg/kg), quercetin (50 and 100 mg/kg) or Withania somnifera (Ws)(100 and 200 mg/kg) significantly reduced the reserpine-induced perioral movements. Memory impairment was assessed by elevated plus maze and passive avoidance paradigms. Reserpine treated rats showed poor performance on elevated plus maze and passive avoidance paradigms indicating memory impairment. Chronic treatment with melatonin, quercetin or WS significantly shortened the TL latency and step down latency on elevated plus maze and passive avoidance paradigms respectively as compared to reserpine alone treated rats. Biochemical analysis revealed that reserpine treatment significantly induced lipid peroxidation and decreased glutathione, superoxide dismutase and catalase levels. Chronic treatment with antioxidants significantly reduced the lipid peroxidation and restored the reserpine-induced decrease in glutathione, SOD and catalase levels. The findings of the present study highlighted the fact that reactive oxygen species and oxidative stress play an important role in the pathophysiology of reserpine-induced perioral movements and memory impairment. Antioxidants could be useful drug candidates for the treatment of reserpine-induced perioral movements and memory impairment.

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The state dependency effect of morphine on memory by behavioral and electrophysiological methods in freely moving rats

M Noorbakhshnia; MR Zarrindast; A Haeri Roohani; F Motamedi

Abstract

Endogenous opioid system agonists exert amnestic effects in different models of memory. It has been suggested that these amnestic effects may be linked indirectly to state-dependent learning. Accordingly pre-training administration of morphine can impair the retrieval of learned tasks in a state dependent manner, which is reversible by pre test morphine administration. In this study, state dependency effect of morphine was investigated by behavioral and electrophysiological methods in freely moving rats. In behavioral studies the step-down passive avoidance paradigm for examining long term memory was used. Also, because of involvement of hippocampus in memory, the possible role of hippocampal dentate gyrus neurons in morphine state dependency was assessed using electrophysiological method. In electrophysiological studies, field excitatory post-synaptic potential (fEPSP) and population spike (PS) were recorded from dentate gyrus of freely moving rats. Results of step-down showed that different doses of morphine (1, 5, 10, and 15 mg/kg, i.p.) could induce significant state dependency in rat, but could not induce significant changes in the slope of fEPSP and PS amplitude of freely moving rats in contrast to the control group. In conclusion, it seems that granular cells of hippocampal dentate gyrus are not involved in morphine state dependency, and other areas of hippocampus or other brain structures may be involved in this phenomenon, which needs further investigation.

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The effect of reversible inactivation of raphe nuclus on learning and memory in rats

M Hosseini; H Alaei; AR Haghshenas

Abstract

The role of raphe nucleus (R.N) and serotonin in some behaviors such as sleep, cognition, mood, and memory has previously been reported. The median raphe (MR) nucleus is a major serotonin-containing cell group within the brainstem and is one of the main sources of projections to the septum and hippocampus. The hippocampus is widely believed to be essential for context-conditioning learning. Moreover, the hippocampus is thought to have a temporary function in the storage of memory, because when the hippocampus is damaged, recent, but not past memories are impaired. In the present study, the effect of reversible inactivation of R.N on memory and learning was investigated using passive avoidance method. For this purpose, Wistar rats (220-250 g) were anesthetized with ketamine and cannula was implanted above the R.N according to atlas of Paxinos. Ten days after surgery, animals were divided into 4 groups. In three of the experimental groups, lidocaine (2%) was injected 0, 60, and 120 minutes after acquisition period and in 1 group, saline used as control. Forty-eight hours later, animals were placed in shuttle box and latency time before entering into the dark chamber and total duration being spent in darkness and lightness was recorded. The results indicated that the latency period before entering into the dark chamber increases up to 350%, 450% and 475% at 0, 60, and 120 minutes respectively, as compared to control group. In addition, time duration being spent in dark space reduced up to 46%, 31%, and 87% and time duration being spent in light space increased up to 64%, 100% and 90% at 0, 60, and 120 minutes as compared to control. It could be proposed that the interactions between the septo-hippocampal 5-HT and ACh in the modulation of learning and memory may be related with 5-HT-ergic origin (DRN or MRN). This study showed that injection of lidocaine (2%) into the raphe nucleus could improve learning and memory.

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Characterization of spontaneous network-driven synaptic activity in rat hippocampal slice cultures

MH Mohajerani; E Cherubini

Abstract

A particular characteristic of the neonatal hippocampus is the presence of spontaneous network-driven oscillatory events, the so-called giant depolarizing potentials (GDPs). GDPs depend on the interplay between GABA and glutamate. Early in development, GABA, acting on GABAA receptors, depolarizes neuronal membranes via a Cl- efflux. Glutamate, via AMPA receptors, generates a positive feedback needed for neuronal synchronization. The depolarizing action of GABA ensures calcium entry through NMDA receptors and voltage-gated calcium channels. The whole cell configuration of the patch clamp technique (in current and voltage clamp) was used to assess whether GDPs were present also in hippocampal slice cultures. This would constitute a good model to be used for imaging and genomic approaches. GDP-like activity started appearing during the second week in culture and disappeared during the third postnatal week. GDPs occurred at the frequency 0.14 ± 0.03 Hz (n = 15). GDPs were synchronous in pair recordings from pyramidal neurons (CA1 and CA3, CA3 and CA3) and pyramidal neurons and interneurons. They reversed polarity at –35 ± 2 mV (n = 9) and were blocked by bicuculline (10 µM) or DNQX (20 µM). In the presence of bicuculline, interictal burst occurred at the frequency of 0.013 ± 0.003 Hz (n = 6). They reversed polarity at -6 ± 3 mV (n = 6) and they were blocked by DNQX (20 µM), suggesting that they were mediated by AMPA/kainate receptors. Bicuculline also blocked spontaneously occurring spike-like events observed in cell attach recordings. In additional experiments performed with gramicidin-perforated patch, the reversal potential of GDPs was –42 ± 3 mV (n = 3). These data suggest that GDP like activity recorded from organotypic slice cultures displays a pattern very similar to that observed in acute slices obtained from neonatal rats and suggest that, at least in the majority of neurons, the action of GABA is still depolarizing and excitatory.

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Light deprivation related changes of strategy selection in the radial maze

M Salami; Z Aghanouri

Abstract

During the early postnatal age environmental signals underlie development of sensory systems. The visual system is considered as an appropriate system for evaluation of the role of sensory experience in postnatal development of sensory systems. In this study we evaluated the effect of visual deprivation on the usage of visuospatial cues in navigation of radial arm maze. Light (LR) and dark reared (DR) rats were trained for correct entries and adjacent arms tasks. Our results indicated that both the LR and DR animals equally entered correct arms. In the adjacent arms task, however, the control group significantly outperformed the DR animals. Also, while the LR males and females differently performed the tasks the DR group represented no sex differences in their performances. These findings suggest that the lack of visual experience is likely to influence the strategy selection and sex differences as well. Also the difference between performances of the LR and DR animals is probably due to the males rather than females behaviour.

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Interaction of sensory experience and age in spatial memory performances

M Salami; M Noureddini

Abstract

During a critical period of postnatal age sensory experience has a profound effect on maturation of visual cortical wiring. Electrophysiological evidence is indicating a substantial effect of visual deprivation on the visual cortical response properties. In this study we evaluated effect of light deprivation during a limited time of postnatal age on two aspects of spatial (working and reference) memory. Light (LR) and dark (DR) reared Wistar rats at age of 40 (P40) and 60 (P60) postnatal days were trained in an open eight arm radial maze. Number of the trials required for two consequent successful navigations to reach our criterion and number of the errors within each session were assessed. During the working memory tests the P40 DR rats made performances superior to their LR counterparts. On the other hand, in the reference memory experiments the P40 LR group showed higher performances compared to their DR counterparts. While the P60 LR and DR rats appeared no difference in the maze solving for the working memory tasks both groups at P60 outperformed those at P40. Regarding the reference memory, the two groups at P40 showed superiority to those at P60. Our results suggest that the working and reference memory performances are differently influenced by the sensory deprivation and age as well.

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The possible analgesic effect of Beta vulgaris in streptozotocin-diabetic rats using formalin test

M Roghani; T Baluchnejadmojarad; Z Sohrabi; M Sadeghi; B Sabouri; R Mohebi; N Nahavandi

Abstract

Diabetic rats in long-term display an augmented nociceptive response to chemical, mechanical, and thermal stimuli. Furthermore, hyperalgesia is one of the major symptoms of diabetic neuropathy in some patients. Considering the antidiabetic potential of chard, this study was carried out to evaluate the possible analgesic effect of chard-mixed food intake in male streptozotocin-induced diabetic rats. For this purpose, male Wistar rats (n = 45) were randomly divided into control, chard-treated control, salicylate-treated control, diabetic, and chard-treated diabetic groups. At the end of the experiment, nociceptive response was evaluated in both acute and chronic phases of the standard formalin test. The results showed that there was a significant increase in the number of pain scores in both acute and chronic phases in diabetic rats ($P<0.05$), and administration of chard for one month did not significantly reduce the pain scores in both phases of the test. Meanwhile, sodium salicylate as positive control only reduced this score in the second phase ($P<0.05$). It can be concluded that oral administration of chard for one month despite its anti-diabetic potential could not attenuate the nociceptive responses in diabetic rats.

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The analgesic effect of oral administration of Artemisia dracunculus in diabetic rats: a behavioral analysis using formalin test

M Roghani; T Baluchnejadmojarad; Z Sohrabi; M Sadeghi; B Sabouri; R Mohebi; N Nahavandi

Abstract

Hyperalgesia is one of the major symptoms of diabetic neuropathy in some patients and could affect their life quality. Considering the antidiabetic potential of tarragon in traditional medicine, this study was conducted to evaluate the analgesic effect of oral administration of tarragon in male streptozotocin-induced diabetic rats. For this purpose, male Wistar rats (n = 43) were randomly divided into control, tarragon-treated control, salicylate-treated control, diabetic, and tarragon-treated diabetic groups. All treatments continued for one month. At the end of the experiment, nociceptive response was evaluated in both acute and chronic phases of the standard formalin test. The results showed that there was an increase in the pain scores in both phases of the test in diabetic group ($P<0.05$) and administration of tarragon for one month did produce a significant reduction in nociceptive scores for both phases, especially in the second phase of the formalin test ($P<0.05$ and $P<0.01$ respectively). In contrast, sodium salicylate as positive control only reduced pain scores in the second phase. It can be concluded that oral administration of tarragon for one month has a moderate analgesic effect in diabetic rats and this may be considered as a potential treatment for diabetic neuropathy.

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The effect of Capsicum frutescens on formalin-induced flinching behavior in streptozotocin-diabetic rats

M Roghani; T Baluchnejadmojarad; Z Sohrabi; M Sadeghi; B Sabouri; R Mohebi; N Nahavandi

Abstract

Diabetic rats in long-term display an augmented nociceptive response to chemical, mechanical, and thermal stimuli. Furthermore, hyperalgesia is one of the major symptoms of diabetic neuropathy in some patients. Considering the antidiabetic potential of pepper, this study was conducted to evaluate the possible analgesic effect of pepper-mixed food intake in male streptozotocin-induced diabetic rats. For this purpose, male Wistar rats (n = 46) were randomly divided into control, pepper-treated control, salicylate-treated control, diabetic, and pepper-treated diabetic groups. In this study, pepper was mixed with standard pelleted food. After one month, number of formalin-induced flinches/h was counted. The results showed that there was an increase in the number of flinches in both acute and chronic phases in diabetic rats ($P<0.05$), and administration of pepper for one month did significantly reduce the flinching behavior in both phases of the test ($P<0.01$). In contrast, sodium salicylate as positive control only reduced this score in the second phase. It can be concluded that oral administration of pepper through central and peripheral mechanisms could attenuate the nociceptive response in diabetic rats.

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The possible acute analgesic effect of pepper, tarragon, and chard in streptozotocin-diabetic rats using hot plate test

M Roghani; T Baluchnejadmojarad; Z Sohrabi; M Sadeghi; B Sabouri; R Mohebi; N Nahavandi

Abstract

Diabetic rats in long-term display an augmented nociceptive response to thermal stimuli. Furthermore, hyperalgesia is one of the major symptoms of diabetic neuropathy in some patients. Considering the antidiabetic potential of medicinal plants chard, pepper, and tarragon, this study was undertaken to evaluate the possible analgesic effect of chard-, pepper-, and/or tarragon-mixed food intake in male streptozotocin-induced diabetic rats. For this purpose, male Wistar rats (n = 83) were randomly divided into control, treated controls, diabetic, and treated diabetic groups. At the end of the experiment, nociceptive response was evaluated using hot plate test. In this respect, the latency for the first response of the animal (either paw licking or jumping) was recorded as the pain response latency (PRL) in seconds. Meanwhile, only one determination was performed for each animal and 30 s of exposure with no response was established as the cut-off time. The results showed that although there was no significant reduction in PRL in diabetic rats as compared to control group, but pepper-treated diabetic rats have a significant higher PRL as compared to untreated-diabetic group ($P<0.05$). It can be concluded that oral administration of pepper for one month could attenuate the acute nociceptive response in diabetic rats.

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The analgesic effect of Capsicum frutescens in streptozotocin-diabetic rats using formalin test

M Roghani; T Baluchnejadmojarad; Z Sohrabi; M Sadeghi; B Sabouri; R Mohebi; N Nahavandi

Abstract

Diabetic rats in long-term display an augmented nociceptive response to chemical, mechanical, and thermal stimuli. Furthermore, hyperalgesia is one of the major symptoms of diabetic neuropathy in some patients. Considering the antidiabetic potential of pepper, this study was carried out to evaluate the possible analgesic effect of pepper-mixed food intake in male streptozotocin-induced diabetic rats. For this purpose, Male Wistar rats (n = 46) were randomly divided into control, pepper-treated control, salicylate-treated control, diabetic, and pepper-treated diabetic groups. At the end of the experiment, nociceptive response was evaluated in both acute and chronic phases of the standard formalin test. The results showed that there was a significant increase in the number of pain scores in both acute and chronic phases in diabetic rats ($P<0.05$), and administration of pepper for one month did significantly reduce the pain score in both phases of the test ($P<0.01$). In contrast, sodium salicylate as positive control only reduced this score in the second phase ($P<0.05$). It can be concluded that oral administration of pepper for one month through central and peripheral mechanisms could attenuate the nociceptive response in diabetic rats.

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Anti-inflammatory effect of alcoholic Datura steramonium seed extract in acute inflammation induced by formalin injection in hind paws of male NMRI rats

M Khalili; MRVaez Mahdavi

Abstract

In the present study, the effect of Datura steramonium (DS) seed extract on acute inflammation induced by formalin injection was investigated. For this purpose, two control and treatment groups were selected and in order to induce pain, formalin (50 µl, 2.5%) was applied to the plantar surface of hind paws. In treatment group, 20-30 min before formalin injection, the DS seed extract was used i.p. at an ED50 of 50 mg/kg. At the same time, Evans-blue dye (30 mg/kg) was injected via left ventricle. One hour after Evans-blue injection, the rat's paw was detached and after crushing, it was mixed with aceton + sulfate sodium solution. The mixture set aside 24 h at room temperature, and then, the solution was centrifuged. Absorption of the solution was measured by spectrophotometer. Our results from control and treatment groups were analyzed by Student's t-test. The results showed that there was a significant ($P<0.001$) difference between control and treatment groups. In addition, our experiment showed that DS seed extract could have a marked anti-inflammatory effect following formalin injection. However, more studies are warranted to evaluate the efficacy of DS extract on other kinds of inflammation.

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Evaluation of antidepressant activities of rose oil and geranium oil in the forced swim test in mouse

D Farzin; M Zarghami; L Khalaj

Abstract

Depression is one of the most common psychiatric disorders that may result in significant consequences in active population of the society. In traditional manuscripts there are many indications about the antidepressant effects of rose oil, but so far no scientific research has been done about this subject. The purpose of present study was to determine the antidepressant effects of rose oil and geranium oil. Geranium oil is similar to rose oil due to the effective components. All experiments were carried out on male Swiss-Webster mice (25-30 g). The antidepressant activities of rose oil and geranium oil were assessed using the forced swim test according to the method published by porsolt (6). This test is the most widely used tool for antidepressant activity preclinically. In this test, mice were placed into a cylindrical glass (25 cm height, 12 cm in diameter) containing a column of 17 cm of water at 25 ±1 °C. After 30 min (for the injection route) or 2 weeks (for the oral route) of the rose oil and geranium oil administrations, the mice were subjected to forced swimming test for 8 min. Acute subcutaneous injection or chronic orally administered of rose oil and geranium oil significantly decreased the immobility time in the mouse forced swim test. The geranium oil response was biphasic. Pretreatment of animals with amphetamine and nortriptyline also reduced the immobility time. The inhibitory effects elicited by rose oil, geranium oil and amphetamine but not nortriptyline were antagonized by reserpine. The results suggest that the antidepressant activities of rose oil and geranium oil may be mediated through a presynaptic mechanism.

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The effect of crocin (a derivative of Crocus sativus L.) on neural development and regeneration of rat: in vivo and in vitro study

M Firouzi; P Moshayedi; F Sabouni; Kh Parsa; M Keshavarz

Abstract

In this study, the effect of crocin, a substance found in Crocus sativus L. stigmata on nervous system development and regeneration was investigated. Crocin is a glycosyl ester of the polyene dicarboxylic acid crocetin and is one of the few families of carotenoids found in nature, which are freely soluble in water. In the first experiment, the effect of crocin (30 microM, 60 microM and 120 microM) on dorsal root ganglia (DRG) outgrowth was investigated quantitatively. Presence of crocin made a 2.4-fold increase ($P<0.03$) in DRG outgrowth within 48 hrs and caused a 2.1-fold increase ($P<0.03$) in the stimulatory effect of fetal calf serum (FCS) on DRG outgrowth, over an 18-hour period. In another experiment, crocin was injected subcutaneously to rats whose spinal cord was hemisectioned. Animals were followed for one month by BBB score, by various reflexes (Hopping Reflex, Pedal Withdrawal Reflex, Bracing Reflex, Lateral Tactile Reflex), Foot Static Index and electromyography. Behavioral results showed more rapid recovery in crocin treated group while there was no significant change in final recovery level. Moreover, a trend in treated group was noted. In EMG study, injured side of treated subjects showed less delay time in comparison to intact side of treated group and both sides of control animals. This study reveals various aspects of crocin effect on nervous system that can be described with antioxidant, differentiating, nucleic acid production inhibition and anti-apoptotic property of crocin.

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Anticonvulsant effect of extract and essential oil of Coriandrum sativum seed in concious mice

M Emamghoreishi; G Heidari-Hamedani

Abstract

Coriandrum sativum L. (coriander) has been indicated for a number of medial problems in traditional medicine such as loss of appetite, convulsion, and nervousness. We have previously shown that the aqueous extract of coriander has depressant effects on motor coordination and spontaneous activity, suggesting of a possible sedative and muscle relaxant effect. These findings suggest that coriander seed may have anticonvulsant effect as indicated in traditional medicine. Therefore, the aim of this study was to examine whether the extract or essential oil of coriander seeds have anticonvulsant effect. Mice were administered the aqueous, or 70% ethanolic extract or essential oil of coriander seed (200, 400, 600 and 800 mg/kg) or vehicle intraperitoenally 30 minutes before the injection of pentylenetetrazole (90 mg/kg). Diazepam (3 mg/kg) was used as a reference drug. Duration of time before onset of myoclonic, clonic and tonic convulsions, and number of animals that show convulsion and the percentage of mortality were recorded. The aqueous extract, hydroalcoholic extract and essential oil of coriander significantly increased the latency of myoclonic and clonic convulsions at doses of 600 and 800 mg/kg. Chi square for a linear trend showed that there is a significant linear relationship between the doses of coriander extract and essential oil and the protection against pentylenetetrazole-induced tonic convulsion and death. These findings strongly suggest that Coriandrum sativum seed possesses anticonvulsant activity and may have a value in the treatment of absence seizure.

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The effect of aqueous extract of Coriandrium sativum seed on acute and chronic pain in formalin test in mice

AA Taherian; A Rashidy-Pour ; AA Vafaei; M Emami-Abarghoei; H Miladi-Gorgi; M Jarrahi; H Sadeghi

Abstract

Previous investigations have shown that Coriandrium sativum (CS) modulates pain in both animals and human. The aim of this research was to assess the role of CS on acute and chronic pain in formalin test in mice. In this study, male albino mice (n=35) in 5 groups (25-30 g) were used. CS (100, and 200 mg/kg) and saline were injected 30 min before formalin test. Indexes of signs were licking and foot elevation for assessment of acute pain (5 min) and chronic pain (15-40 min) after injection of formalin 5% (25 µl) into right paw. Results indicated that CS has analgesic effect by decreasing the duration of acute and chronic phases in comparison with control groups at both doses ($P<0.01$). Also, the higher dose of the CS was more effective ($P<0.01$). Above findings show that CS extract has a modulatory effect on peripheral pain in formalin test.

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The effect of Ferula persica on modulation of withdrawal syndrome sign in morphine-dependent mice

AA Vafaei; AA Sajadi; AA Taherian; M Emami-Abarghoi ; H Miladi-Gorgi; M Jarrahi

Abstract

Tolerance and dependence are two main problems that have limited morphine administration. There are several reports on the effect of some medicinal plants on morphine-induced tolerance and dependence. In this study we investigated the effect of Ferula persica (FP) extract on modulation of withdrawal syndrome sign (WSS). For this purpose, male Albino mice (25-30 g, n=30) were used. Morphine was used to produce drug dependency according to Marshall method. The criteria for measuring intensity of morphine dependency were number of jumping and weight loss. Test groups received extract of FP at two doses (i.p.) and control group received vehicle (i.p.) 10 min before injection of naloxone for assessment of WSS. The results showed that FP extract significantly modulates withdrawal syndrome signs only for weight loss and not for number of jumping in morphine-dependent mice ($P<0.01$). These findings indicated that FP extract has a modulatory effect on withdrawal syndrome in morphine-dependant rats. Therefore, it is warranted to reveal its underlying mechanisms of effect in future research studies.

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Antioxidative effect of aqueous Date fruit extract in PC12 cell line

M Asadi Shekari; S Rajabalian

Abstract

Cell damage induced by free radicals has been implicated as causal factor in a wide variety of human diseases such as cancer, neurodegenerative disorders. Scientific recognition of natural antioxidants may be helpful in preventing many diseases. Aqueous Date fruit extract (ADFE)-PC12 cell line. ADFE at concentrations of 0.1-10% containing 0.005% H2O2 and H2O2 at concentrations of 0.01-1% containing 10% ADFE were prepared. 100µl from a cell suspension equal to 106 cells/ml of PC12 cells were added to centrifuge tubes and incubated for 24 hours. Cell suspension rinsed twice with medium and incubated in 96-multidish for 72 hrs. Then, cell growth was assayed by XTT-assay. Aqueous Date fruit extract (ADFE) at concentrations of 0.1, 1 and 10% comparing to 0.005 H2O2 showed significant increase in PC12 cell growth (by 11, 57 and 74% respectively) ($P<0.05$ to 0.001). ADFE at concentration of 10% showed protective effects against different concentrations of H2O2 (0.01-1%)($P<0.05$ to $P<0.001$). Date fruit contains compounds with potent antioxidant activity and daily consumption may be helpful in preventing many diseases, especially neurodegenerative disorders such as Alzheimer's disease and Parkinson's disease.

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The effect of Aloe vera on morphine withdrawal signs in mice

E Abbasi; H Jafari; M Shahidi; M Ahmadkhah

Abstract

Aloe vera is a herbal medicine for skin therapeutical objects. Many products like pomade, cream, gel, and shampoo for skin injuries have been made from this plant. Sometimes, the powder of this plant has also been used by mothers to keep out their infants from breast-feeding. In some of the books, this plant has been introduced as an opioid–receptors antagonist. In this study, 30 male mice (45-50 g) were randomly selected and divided into 5 groups (n = 6). All of them received morphine sulphate (120 mg/kg, s.c., 3 times daily) for 3 days. Control group only received morphine. Treated-groups received Aloe vera (5, 10, 20 mg/kg, IP) and morphine daily for 3 days. clonidine group received clonidine (3 mg/kg, IP) and morphine daily for 3 days. At the end of session (4th day), all of the groups received naloxone (5 mg/kg, IP) and, then morphine withdrawal signs (jumping, climbing, diarrhea) were studied for 30 minutes. Our results show that 10 mg/kg of Aloe vera is the most effective dose for treatment of morphine withdrawal signs, especially for reduction of diarrhea and its effect on diarrhea was better that clonidine effect.

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The analgesic effect of aqueous extract of Viola odaratan in mice

H Jafari; M Shahidi; E Abbasi; R Gharebaghi

Abstract

Pain is a general symptom of many diseases. The medicinal plant Viola odarata (V.O.) has a particular position as an analgesic in traditional medicine. In this study, the analgesic effect of aqueous extract of this plant was studied. The extract was prepared using suxhlet method. The extract was than concentrated. Then, the extract was dissolved in normal saline to produce the desired concentrations. The analgesic effect was evaluated using formalin test in male mice. The average weight of mice was 25 g. In this test, formalin 5% (s.c.) was used. The extract and ASA was injected i.p. ASA was injected at a dose of 300 mg/kg and extract was administered at doses of 10, 50, 100, and 200 mg/kg. The results indicated that extract at a dose of 100 mg/kg had maximum analgesic effect ($P<0.01$) and the extract at this dose was more effective in attenuation of chronic pain as compared to ASA ($P<0.05$). Meanwhile, it was ineffective in acute phase of formalin test.

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Analgesic effect of aqueous extract of Olea europaea L. in experimental models of pain in male rat

J Verdi; H Komeilizadeh; M Kamalinejad; Sh Sharif

Abstract

The medicinal plant Olea europaea L. is a valuable one whose fruit and its oil are considered as food. There are some reports in ancient medical literature that aqueous extract of its leaves has analgesic effects. This prompted us to investigate the analgesic effect of aqueous extract of its leaves using formalin and tail-flick tests. For this purpose, male NMRI rats weighting 220-260 g were used in all experiments. The aqueous extract of this plant was intraperitoneally injected at doses of 200, 400, and 600 mg/kg. In this study, sodium salicylate (300 mg/kg) and distilled water were used as positive and negative controls respectively. The results obtained were analyzed using one-way ANOVA and Tukey-Kramer tests. Our data showed that intraperitoneal injection of aqueous extract of the leaves of this plant (200, 400, and 600 mg/kg) produces a significant analgesia in both phases of the formalin test ($P<0.001$). Meanwhile, different doses of this plant did not produce any analgesic effect in tail-flick test. On the other hand, sodium salicylate (300 mg/kg, i.p.) induced analgesia in the second phase of the formalin test. Aqueous leaf extract of Olea europaea at doses of 200, 400, and 600 mg/kg had a more potent anti-nociceptive effect in comparison with sodium salicylate (300 mg/kg, i.p.) in formalin test. Since the aqueous extract of Olea europaea l. exert analgesic effects in both phases of the formalin test. Therefore, it may be concluded that it produces anti-nociception through the central mechanisms. In this respect, flavonoid and steroid compounds of the plant may be involved in its anti-nociceptive effects in rats.

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The effect of flavonoid from the leaves of Araucaria bidwilli in reversing LPS-induced memory deficit in rats

KFH Nazeer Ahamed; AM Wahile; V Kumar; BP Saha; PK Mukherjee

Abstract

There is growing interest in flavonoids for their wide array of biological actions. In neurodegenerative disorders these naturally occurring polyphenolic compounds, are known to inhibit proinflammatory mediators such as in TNF–? and IL-6 in lipopolysaccharide (LPS) stimulated neuronal degeneration, which are associated with Alzheimer's and Parkinson's disease. Recent studies on flavonoids have yielded an impressive array of novel structures, which acts as neuroprotective agents. The current study investigated the ability of flavonoid rich fraction from the leaves of plant Araucaria bidwilli on the cognitive performance in LPS-treated rats using passive avoidance step through behavior and elevated plus maze paradigm. Administration of LPS (100 mg/kg b.wt.) in rats exhibited impaired acquisition and retention memory in step-through passive avoidance and in elevated plus-maze tasks. Chronic administration of the flavonoid-rich fraction (50-200 mg/kg per oral) of the plant Araucaria bidwillii hook reversed the LPS-induced retention deficits in both behavioral paradigms. Furthermore, the extract was found to rejuvenate the antioxidants defense system in different brain regions. The present investigation showed that treatment with the flavonoid-rich fraction reverses cognitive deficits in LPS-intoxicated rats and are found to rejuvenate the brain antioxidants thereby preventing the memory deficits, which is found to be one of the symptoms related to AD.

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The effect of HHKV and TCTN1 extracts on malonyl dialdehyde (MDA) level of the brain

TVP Lien; HT Bich; LT Phuong; TH Hang

Abstract

The present research was conducted on 170 rats dividing into two groups. One group received HHKV extract at a dose of 2 g/kg/day continuously for 14 days. The other group received TCTN1 extract at a dose of 3 g/kg/day for 30 days. After 14 and 30 days of experiment, a half of each group received an electrical shock and the remainder didn't receive such treatment Malonyl dialdehyd (MDA) of rat's ground brain was determined using Jadwiga Robax method. Obtained results showed that two above-mentioned extracts have inhibiting effects on lipid peroxidation in the rat brain. Thus, treated rats showed a higher endurance with electrical shock in comparison with rats receiving only Giloba and water.

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The effect of HHKV extract on conditional reflex of mice and rats

HT Bich; TVP Lien; LT Phuong; TH Hang

Abstract

HHKV includes 4 herbal plants. In this study, we used an extract from HHKV for the rats and mice orally. All mice were divided into three groups, group 1: drinking HHKV at a dose of 2 g/kg body weight/day, group 2: drinking water, group 3: drinking Giloba at a dose of 2 mg/kg body weight/day. All rats divided into four groups, two controls receiving drinking water or receiving Giloba at a dose of 4 mg/kg body weight/day, and two tests drinking HHKV at a dose of 5 g/kg body weight/day or 10g/kg body weight/day respectively. This treatment was applied continuoslty for 14 days. After that, mice and rats were trained for conditional reflex. The results showed that extract from HHKV increase the memory of mice and rats. However, the effect of HHKV is weaker than Giloba.

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The effect of nucleus tractus solitarius inactivation on blood pressure in diabetic rats

M Kourosh Arami; A Sarihi; B Heshmatian; SM Malakouti; I Amiri

Abstract

The role of nucleus tractus solitarius (NTS) in cardiovascular system regulation is controversial. On the other hand, the problem of hypertension in diabetic animals is the subject of many research programs. The aim of present study was to determine whether inactivation of NTS could affect blood pressure and heart rate in diabetic rats. To this end, stereptozotocin induced diabetic rats were anesthetized with urethane and a cannula was inserted above NTS. Blood pressure and heart rate were monitored by using an intra-arterial cannula. Lidocaine (0.5 ml, %2) was injected unilaterally through a single injection cannula aimed at the NTS for functional ablation. Sham control diabetic group received saline. The results indicated that inactivation of NTS in diabetic rats has no effect on systolic and mean arterial pressure but enhances diastolic pressure ($P<0.05$). There was no significant difference in heart rate between sham control and test groups. It is concluded that NTS has a decremental effect on diastolic pressure in diabetes.

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Amygdala centralis cardiovascular response to angiotensin I microinjection in Goldblatt hypertensive rats

B Heshmatian; A Sarihi; M Zarei

Abstract

Previous studies have shown that induction of 2kidney -1clip Goldblatt hypertension (2K-1C) induction in rats eliminates hypertensive response after inactivating of the central nucleus of the amygdale (ACe). The present study investigated the possibility of alteration in local renin angiotensin system (RAS) activity in ACe after hypertension induction. Clamps were placed on the left renal arteries of a group of Wistar rats to induce 2K-1C Goldblatt hypertension. A similar surgery without clipping was done in sham group. Rats were studied six weeks after surgery. The mean arterial blood pressure (MAP) and heart rate (HR) were measured directly with indwelling arterial cannulas under urethane anesthesia. Rats received bilateral cannulation of the ACe for angiotensin I (AngI) microinjection. Before and 5,10,20,40 and 60 minutes after bilateral microinjection of Ang I (1 µL, 100 µM) into ACe, MAP and HR measured and compared between groups. In normotensive rats, significant changes in MAP or HR were not seen. Meanwhile, in hypertensive rats MAP significantly increased 5, 10, and 20 minutes after ($p<0.05$, $p<0.05$, $p<0.01$) though HR remained unaltered. MAP alteration range by microinjection in hypertensive rats has significantly been different in comparison with normotensive ones at mentioned times ($p<0.05$, $p<0.01$, $p<0.05$). This data shows that inhibitory effect of Ang I on ACe has increased after hypertension induction. This process may contribute in hypertension development. Increased Angiotensin II (Ang II) receptors or accelerated converting process of AngI to AngII may altere Ang I microinjection in ACe outcome.

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The effect of reversible inactivation of the central amygdaloid nucleus on cardiovascular responses in rats with renal hypertension

M Zarei; B Heshmatian; A Sarihi

Abstract

The brain rennin-angiotensin system (RAS) has an important role in the regulation of cardiovascular function. The aim of the present study was to determine the effect of reversible inactivation of the central amygdaloid nucleus (Ace) in normotensive rats and rats with renal hypertension (2K-1C). Two groups of normotensive rats were selected for this study. In one group, hypertension was induced by Goldblatt method and the other group subjected to a sham procedure. After 6 weeks, tests were performed on anesthetized rats with urethane (1 g/Kg). To perform bilaterally reversible inactivation, lidocaine (2%) was injected into Ace. Then, direct measurements of femoral blood pressure and heart rate were recorded just before and 5, 10, 20, 40, and, 60 min after injection of lidocaine. The degree of change was determined in relation to baseline. Our results revealed that sham group with Ace inactivation had a significant increase of blood pressure at 5 min after injection ($P<0.05$). There was also no significant difference between blood pressure at different times before and after injection in hypertensive group. The percentage of change had also a significant increase at 5 min following inactivation in sham-operated group as compared to 2K-1C rats ($P<0.05$). However, concerning heart rate there was no differences between groups. It seems that the tonic inhibitory effect of amygdala on blood pressure has disappeared due to hypertension. Thus, inactivation of Ace has no effect on blood pressure in hypertensive rats. It can be concluded that the change in Ace activity during induction of hypertension is due to the change in RAS activity.

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GABAergic receptors in rostral ventrolateral medulla mediates the cardiovascular responses to activation of bed nucleus of the stria terminalis in the female rat

M Ganjkhani; M Hatam; CVR de Oliveira; J Ciriello

Abstract

The bed nucleus of the stria terminalis (BST) is known to contain estrogen (E)- concentrating neurons. In addition, injections of E into BST have been reported to potentiate the sympathoinhibitory arterial pressure (AP) and heart rate (HR) responses elicited by glutamate (Glu) stimulation. In this study, the effect of GABA-A antagonist receptors, bicuculline methiodide (BMI), in the rostral ventrolateral medulla (RVLM) on the cardiovascular responses to Glu stimulation of BST (0.25 M; 20 nl) was investigated in the chloralose anaesthetized, ovariectomized (OVX; n = 6) or OVX+E treated (30 pg/ml plasma E, n = 7) female Wistar rats. Glu stimulation of the BST elicited decreases in AP (-28.1 ± 3.7 mmHg) and HR (-14.2 ± 3.5 bpm) in the OVX+E animals and in OVX only animals (AP, -22.48 ± 2.68 mmHg; HR, -12.2 ± 2.1 bpm). Microinjection of GABA-A receptor antagonist, BMI (1.0mM, 100nl) into the ipsilateral RVLM reversibly attenuates glutamate induced bradycardia and depressor responses to BST stimulation in both group of animals (OVX; n=6 AP,-8.03 ± 3.15 and HR,-5.8 ± 4.2) but in OVX+E animals only attenuates glutamate induced bradycardia but not depressor response (OVX+E;n=7 AP,-16.0 ± 2.4 and HR, -5.84 ± 2.0). These data suggest RVLM sympathetic premotor neurons contain GABA receptors, which mediate the sympathoinhibitory responses to stimulation of BST in OVX animals.

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Nerve agents: Lessons from the Iranian experiences

A Foroutan

Abstract

On September 22, 1980 Iraqis attacked Iran's air bases and simultaneously entered Iran with six army divisions on three fronts and occupied a vast part of Iranian territories. At that time, Iraqis did not have chemical warfare. But in time, they began to develop an intensive research program to produce and store chemical weapons and used the war fields to test and perfect their chemical warfare. They began with mustard and then added different kinds of nerve agents like tabun sarin and GF. We witnessed two different clinical pictures of nerve gas poisoning; fulminant and classic (severe, moderate and mild). Treatment was based on 4 steps: 1- Restoring respiration. 2- Atropine. 3- Oxime. 4- Diazepam (and other drugs).Respiratory arrest was the main factor of death in nerve gas victims. In severe cases the treatment was as following: Ventilation and administration of oxygen (resuscitation). Injection of enough atropine (see below). Application of 250 mg of obidoxime IV (our oxime of choice at that time). Administration of 10 mg of Diazepam IM. Successful administration of" high dosed atropine"had an important role on recovery of thousands nerve gas victims. Method of rapid and effective atropinization: 1- Injection of a test dose of 4 mg of atropine IV in 1-2 minutes. Examination of the patient's reaction after two minutes. Further atropinization is indicated if there are no signs of atropine intoxication. 2- 25 mg of atropine IV within five minutes accompanied by continuous monitoring of the pulse rate. The injection rate was reduced when the pulse was 20 to 30 beats above the initial value. The injection rate was increased while bradycardia was still present. If there was no sign of atropinization (dry moth and/or tongue) the „third dose“ was given. 3. The administration of atropine (step2) was repeated until the mouth and tongue were dry.The administration of atropine was also discontinued when miosis became absent or mydriasis appeared regardless of whether the mouth was completely dry or not. This method allowed the treatment with atropine to be completed within 10 to 15 minutes on average. In many instances we needed 100-200 mg atropine (for initial atropinization) to overcome life threatening cholinergic crisis.

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The reversal effect of mefenamic acid in the sporadic model of Alzheimer's disease in rat: a behavioral analysis

T Baluchnejadmojarad; M Roghani; S Hosseinzadeh

Abstract

Alzheimer's disease (AD) is a chronic neurodegenerative disease causing progressive impairment of memory and cognitive function. Streptozotocin (STZ) injection into the brain is known to cause cognitive impairment in rats and is similar to sporadic AD in humans. Several lines of evidence have indicated that an inflammatory process contributes to the pathology of AD. On the basis of the results of epidemiological studies, nonsteroidal anti-inflammatory drugs (NSAIDs) are currently considered to be the most promising approach for anti-inflammatory treatment of AD. The present study was performed to investigate the effect of mefenamic acid after intracerebroventricular (i.c.v) administration of STZ on learning and memory in adult rats in passive avoidance task. The results showed that i.c.v. injection of STZ decrease step through latency (STL) on the passive avoidance test as compared to control group. The intraperitoneal administration of mefenamic acid (30 mg/Kg body weight, daily) before training trial for 10 days significantly increased STL. These results showed that i.c.v. administration of STZ is able to make a model of Alzheimer's disease without affecting animal's motor activity. In addition, the treatment with mefenamic acid could improve the impaired acquisition and/or consolidation of passive avoidance learning.

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AGE proteins as a causative factor in Alzheimer's Disease

MR Khazaey; M Habibi-Rezaei; S Safarian; Z Karami; F Sabouni

Abstract

The reaction between reducing sugars and protein free amines, known as the Maillar reaction results in the formation of advanced glycation endproducts (AGEs). AGE modification changes the structure of proteins to amyloid cross-beta structure. These protein structures can activate receptors known as RAGE on glial cells (microglia and astrocytes), and induce the expression of inducible nitric oxide synthase (iNOS). Activation of inducible nitric oxide synthase in glial cells is assumed to contribute to oligodendrocyte degeneration in demyelinating diseases (e.g. multiple sclerosis) and neuronal death during Alzheimer's Disease (AD). Our goal was to study AGE-activated signal transduction pathways involved in the induction of iNOS in the rat microglial cells. In vitro prepared AGE-BSA used as model AGE, induces nitric oxide (NO). We treated the Cultured Microglial cell with different range of glycated BSA (low-glycated-BSA to Advanced Glycated End-BSA) and BSA which conserved against glycation in the presence of some antiglycation agents. So, after we characterized the effects of these treatments on the activation of iNOS in microglia and on microglial cell proliferation and morphology. Secreted Nitric Oxide was determined spectrophotometrically by using the Griess reaction. According to our result we found that AGE-BSA increase the iNOS expression and NO secretion which can lead to AD, but BSA which was conserved against glycation couldn't induce NO production in microglial cells.

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Neuroprotective effect of caffeine in an early model of Parkinson's disease in rat: behavioral and histochemical evidence

MT Joghataie; M Roghani; F Negahdar; L Hashemi

Abstract

Parkinson's disease (PD) is the second most common neurodegenerative disorder affecting 1 to 3% of individuals over the age of 65 years. Both retrospective and prospective epidemiological studies have consistently demonstrated an inverse association between coffee consumption and PD. This study was designed to investigate the beneficial effect of caffeine in an early model of PD. For this purpose, unilateral intrastriatal 6-hydroxydopamine (6-OHDA)-lesioned rats were pretreated with caffeine (20 mg/kg) 1 hour before and treated twice a day for one month. Apomorphine-induced rotations and number of Nissl-stained neurons of substantia nigra pars compacta (SNC) were measured. Statistical analysis of the total net number of rotations made over a 60-min period 4 weeks after the surgery showed that apomorphine caused a very significant contralateral turning in the rats of the lesion (L) group ($P<0.001$) and induced less significant rotations in the caffeine-treated lesion (L+C) group ($P<0.005$) in comparison with the SH group. The results of histological studies demonstrated that although there was no significant difference for the number of Nissl-stained neurons on the right and left sides of SNC in SH group, but a significant reduction was observed for L ($P<0.001$) and L+C ($P<0.05$) groups. Meanwhile, there was no significant difference between SH and L+C groups when comparing number of Nissl-stained neurons on the left side of SNC. It can be concluded that caffeine administration attenuates rotational behavior and protect the neurons of SNC against 6-OHDA toxicity.

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A study on striatal local electrical potential changes in an animal model of Parkinson's disease

A Sarkaki; SM Alavian; SMT Mansouri

Abstract

Parkinson's disease (PD) is a neurodegenerative disorder that does not develop spontaneously in some animal species. PD can be induced experimentally in some laboratory animals including mouse, rat and horse. Globus pallidus (GP) and substantia nigra pars compacta (SNc) are damaged in patients with PD. The hallmark of PD is a progressive impaired control of movement, an alteration of autonomic functions, cognitive disorders, behavioral changes, and dementia. Loss of basal forebrain nucleus cholinergic neurons, appearance of Lewy bodies, reduced striatal dopamine, norepinephrine and 5-HT contents, loss of glutamate decarboxylase, leu- and met-enkephalins, and substance-P have been observed in SNCc and ventral tegmentum (VTM) in PD. In order to study the possible changes of field potentials of striatal neurons in the experimental model of PD, 20 male NMRI rats (200-250 g) were used and two guide cannulae (0.7 mm in diameter) were implanted bilaterally into SNC and a bipolar metal wire electrode were implanted into the striatum under stereotaxic surgery. The striatal electrical activity was amplified and recorded. To induce the model of Parkinson's disease, halopridole (10 µg/µl /rat in each hemisphere) was injected into SNC bilaterally and 25 minutes after injection, striatal local EEG was recorded. EEG amplitude changes were analyzed statistically. Results showed that amplitude of striatal local EEG significantly ($P<0.01$) decreases after injection of halopridole in SNC. These data suggest that in patients with PD, electrical activity of striatal neurons reduces and, thereby it may reduce the activity of GP neurons.

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The effect of intrastriatal injection of estrogen on pallidal field potential and rigidity in Parkinsonian -ovariectomized rats

N Hosseiny; A Sarkaki; M Badavi; MK Gharibnaseri

Abstract

The major pathological feature of Parkinson's disease (PD) is a progressive loss of dopamine-producing neurons of the substantia nigra pars compacta (SNc), resulting in a reduction of dopamine (DA) content in the target field of these neurons, the striatum (STR). The present evidences suggest that female sex hormones may influence the onset and severity of PD symptoms. PD is more prevalent in men than in women by an approximate ratio of 3:2, suggesting a possibly protective influence of estrogen on predisposition to the disease. In this work, the effect of intrastriatal injection of 3 µg/3µl of estrogen on globus pallidus (GP) extracellular field potential and muscular stiffness before and after lesioning the SNc by 6-hydroxydopamine (6-OHDA) in 12 ovariectomized Wistar rats (180-220 g) has been tested. Results showed that decreased GP local EEG amplitude significantly improved after estrogen therapy in Parkinsonian-ovariectomized rats in both pre-and post- SNc lesioning ($P<0.01$). Meanwhile, the GP local EEG amplitude in pretreated-lesioned ovariectomized rats and then treated by estrogen was significantly ($P<0.01$) higher than post- lesion treated ones. These findings suggest that estrogen pretreatment in female rats suffering from PD has an important defense mechanism by which estrogen modulates DA function. Recent studies suggest that estrogen alter the function of both pre-synaptic SNc neurons and their post-synaptic targets in the STR, thereby exerting a potent modulatory influence over dopaminergic transmission within the basal ganglia. It seems that pretreatment by estrogen may prevent the reduction of GP local EEG amplitude and can attenuate muscular stiffness induced by 6-OHDA as a selective neurotoxin for dopaminergic system.

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Clioquinol-induced ordered conformational behavior in alpha-synuclein: promising relevance for therapeutic approach to Parkinson's disease

B Bil; ML Hedge; KSJ Rao

Abstract

Parkinson?¦s disease (PD) is a devastating and an intricate complex neurological disorder that results from the progressive degeneration of nerve cells in Substantia nigra that controls movement. The pathological hallmark of PD is the formation of insoluble protein aggregates known as lewey bodies. Alpha-synuclein is the major constituent of these fibrillar structures. Alpha-synuclein a 140 amino acid pre-synaptic protein is natively unfolded, random coiled conformation, but n Lewey bodies and neuritic plaques it has cross beta conformation. There is a great need to look for biomolecules that favors and stabilizes ordered conformation in alpha-synuclein and thus of great therapeutic application. The formation of alpha-helix is a protective mechanism against beta sheet and prevents aggregation. In this perspective, we investigated the role of a metal chelator, clioquinol, in inducing ordered structure in alpha-synuclein. Interestingly, circular dichroism spectroscopic studies on alpha-synuclein-clioquinol interaction revealed that there is a characteristic minimum at 198 nm and at a short amplitude of 222 nm, which confers the formation of alpha-helix. The formation of alpha-helix in alpha-synuclein by clioquinol is verified by fluorescence spectroscopic studies like 8-Anilino NitrosulphonicAcid binding, Intrinsic Tyrosine Fluorescence and Acrylamide quenching studies. The stability of alpha-helix of synuclein was characterized by CD-Tm studies and found an increase in Tm values (Syn alone-41 degree, Syn+CQ-49.8 degree) compared to Synuclein alone Hence, the alpha-helix formed by clioquinol is thermodynamically stable. This is further supported by studies carried out at lower pHs, melting temperature studies and chemical denaturation studies using Urea and GdHcl. To the best of our knowledge, this is the first study to show the relevance of clioquinol in developing of therapeutic strategies in Parkinson’s disease by understanding the conformational pattern of alpha-synuclein.

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Cholinergic neuropathology in a mouse model of Alzheimer's disease

P Pasbakhsh; D German; N Omidi

Abstract

Transgenic mice over-expressing mutant human amyloid precursor protein (PDAPP mouse) develop several Alzheimer's disease (AD)-like lesions including an age-related accumulation of amyloid-?-containing neuritic plaques. Although aged, heterozygous PDAPP mice also exhibit synaptic and glial cell changes, that is characteristic of AD pathology, no evidence of neurodegeneration has been observed. The present study sought to determine whether homozygous PDAPP mice, which express very high levels of amyloid-? peptide, exhibit AD-like cholinergic degenerative changes, and whether the changes paralleled the deposition of amyloid-? plaques. Mice were examined at months 2 and 4 and years 1 and 2 of their age. There was an age-related increase in the density of amyloid-??plaques in the cortex and hippocampus of the PDAPP animals, but at months 2 and 4, there were very few plaques. There was also an age-related reduction in the density of cholinergic nerve terminals in the cerebral cortex (as early as 4-months of age), there were over a 50% reduction at year 2, and a 15% reduction in the number of basal forebrain cholinergic somata, which innervate the cerebral cortex and hippocampus. These data indicate that the homozygous PDAPP mouse exhibits cholinergic degenerative pathology similar to that observed in AD, and the neurodegenerative changes occur prior to the deposition of amyloid-?-containing neuritic plaques.

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Minocycline blocks c-terminal fragments of amyloid precursor protein-induced neurotoxicity by inhibition of cytochrome c release and caspase-12 activation

Y Choi; EM Kim; YH Suh

Abstract

Minocycline is a second-generation tetracycline that effectively crosses the blood-brain barrier. It has remarkable neuroprotective qualities in models of cerebral ischaemia, traumatic brain injury, Huntington's and Parkinson's diseases. However, there is no evidence about neuroprotective effects of minocycline on AD. Alzheimer's disease (AD) is a neurodegenerative disorder characterized neuropathologically by the presence of neuritic plaques containing amyloid fibrils and neurofibrillary tangles whose main component is paired helical filament composed of hyperphosphorylated tau. There are numerous lines of evidence that some of the neurotoxicity associated with AD is due to proteolytic fragments of the amyloid precursor protein (APP). In this study, it was found out that minocycline reduces neurotoxicity induced by various C-terminal fragments of APP through inhibition of cytochrome c release and caspase-12 activation.

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Function of mitochondrial complex-I and -IV in normal human and Parkinson's disease cybrids

M Varghese; KP Mohanakumar

Abstract

Mitochondrial dysfunction has been implicated in the dopaminergic neurodegeneration, which characterizes Parkinson’s disease (PD). The activities of mitochondrial complexes I and IV were found to be reduced in the brains of PD patients (n = 4) as compared to age-matched controls (n = 4). This is tested in SH-SY5Y cell lines, transformed Rho0 cells, and in normal and PD cybrid cell lines. Cybrids were created by fusion of the Rho0 cells with platelets from PD patients and age-matched normal humans. The reduction in activities was found to be stably transferred to PD cybrids. By Western blotting, it was found that the expression of the subunits of the two complexes was also aberrant in the patient brain samples. Most of the PD cybrids showed a decreased expression of the subunits of complexes I and IV. While administration of coenzyme Q10 was found to increase complex I activity in the control cybrids, no such recovery was found in the PD cybrids. These results indicate decline of both complex-I and -IV activities and their subunits expression in most cases of sporadic PD. Cybrids could be a useful tool for investigating mitochondrial gene abnormalities in PD. While coenzyme Q10 was effective in cybrids from the aged, it was found to be ineffective in sporadic PD. These results suggest an active involvement of the components of the electron transport chain in PD, and offer scope for meaningful investigation into neuroprotective strategies relevant to humans.

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Striatal dopamine levels and changes in mitochondrial function following chronic 3-nitropropionic acid treatment in rats

M Pandey; KP Mohanakumar; R Usha

Abstract

An irreversible inhibitor of complex II in the mitochondria, 3-nitropropionic acid (3-NP), induces bilateral striatal lesions with many neuropathological features of Huntington's disease (HD) in rats. It is widely used as a model of HD. Chronic systemic treatment of 3-NP for 4 days in rats (10, 15 and 20 mg/kg) caused a significant dose-dependent reduction in succinate dehydrogenase activity, which was evident in histochemical as well as in biochemical assays. Splayed toes, and moderate to severe immobility in the hind limbs marked 3-NP administration in adult rats. A dose-dependent rise in striatal dopamine level, as measured employing HPLC-electrochemistry, was also evident. The rise in dopamine level was consistent in adult (4-6 months), but inconsistent in younger rats (2-3 months). A significant rise in NADH:ubiquinone reductase activity was indicated in the mitochondrial fractions prepared from the cerebral cortex region of rats treated with 3-NP. This was equally visible in the rotenone sensitive and insensitive fractions of the enzyme. Our results strongly suggest involvement of Complex I dysfunction in the behavioral and dopaminergic aberrations as seen in HD.

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Polymorphism in the interleukin-10 promoter affects both provirus load and the risk of human t lymphotropic virus type I (HTLV-I) associated myelopathy/tropical spastic paraparesis

AH Sabouri; M Saito; K Usuku; CRM Bangham; M Osame

Abstract

To investigate candidate genes that influence the risk of HTLV-I associated myelopathy/tropical spastic paraparesis (HAM/TSP), we analyzed 6 single nucleotide polymorphisms (SNP) in the interleukin-10 (IL-10) promoter region. METHODS: 280 cases of HAM/TSP patients and 255 HTLV-I seropositive asymptomatic carriers (HCs) from Kagoshima, Japan were studied. All subjects gave written informed consent and the study approved by local ethics committee. PCR-RFLP method used for SNP typing. HTLV-I provirus load was measured by quantitative PCR. RESULTS: The IL-10 -592A/C SNP showed a significant difference in allele frequency between HAM/TSP patients and HCs (p=0.014). Possession of the IL-10 -592 A allele was associated with a >2-fold reduction in the odds of HAM/TSP (2p=0.011, OR=0.50). This effect remains significant even after taking into account the other two known predictors such as provirus load and HLA-A*02 genotype. Given the OR and the observed frequency of the IL-10 -592 A allele, we can estimate that IL-10 -592 A allele prevents 44.7%(13.1%SD) of potential cases of HAM/TSP in the study population. The presence of the IL-10 592 A allele is also a significant predictor of lower provirus load in the entire cohort (p=0.001). Furthermore, HTLV-I Tax transactivates the IL-10 promoter in the human T-cell line Jurkat and this transactivation was higher with the IL-10 -592 C allele than the A allele. Among more than 90 non-HLA candidate gene loci we have so far examined, IL-10 -592 A/C SNP is the only non-HLA candidate gene locus associated with a significant reduction in both the provirus load and the risk of HAM/TSP. These data demonstrate that the IL-10 -592 A allele influences the provirus load in HTLV-I infected individuals, and defines a further component of the genetic susceptibility to HAM/TSP.

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Alpha-synuclein induced apoptosis and proliferation interacted with CD44 in human lymphocytes

CJ Heo

Abstract

Human ?-synuclein is a 140 amino acid protein with little or no secondary structure. The ?-synuclein is expressed at high levels in the brain and enriched in neural synaptic terminals but its physiological function remains largely unknown. More recently, ?-synuclein has been shown to be one of the principal componets of Lewy bodies, neuronal inclusions that are found in diverse human neurodegenrerative disorder including the Lewy body variant of Alzeheimer's disease, diffuse Lewy body disease, Parkinson's disease, multiple system atrophy. Therefore, ? -synuclein and its abnormal protein aggregation have been thought to play a role in the pathogenesis of these neurodegenerative diseases known as ?-synucleinopathies. In order to understand their etiology and pathogenesis, it is crucial to identify the normal function of ?-synuclein. It was previously reported that the splicing variant of ? -synuclein is expressed in heart, skeletal muscle, and pancreas. In our study, we identified the expression in spleen and confirmed the expression of ?-synuclein in isolated human PBMC. The physiological role in lymphocytes showed induction of apoptosis with Caspase-8 and Caspase-9 activation. And also they showed enhanced expression of CD44, a multifunctional cell surface glycoprotein, on human lymphocytes. We further found that the stimulation of CD44 triggered intracellular ?-synuclein expression, reversly. There was involvement of multiple signaling pathways, the activation of PKC, Ras oncoprotein. These Results suggest that ?-synuclein is participates in cell adhension and viability in lymphocytes.

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Cortisol secretion in adult male rats

A Eidi; S Oryan; M Eidi; A Nazar

Abstract

As a neurotransmitter or neuromodulator, brain histamine has a variety of physiological roles in brain functions such as hypothalamic- pituitary- adrenal (HPA) activity. Histamine induces the release of ACTH through the activation of hypothalamic neurons containing vasopressin and CRH. Histamine induces the activity of HPA axis directly or indirectly. Endogenous opioids modulate the (HPA) axis activity as well. In order to consider the interaction between morphine and pyrilamine in HPA axis, the adult male Wistar rats were administered morphine (0.5, 1, 1.5, 3 mg/kg), and pyrilamine (1, 5, 10, 20 mg/kg) respectively. Thirty minutes after injection, the rats were anesthetized and blood samples were collected for hormonal investigations. The result showed that morphine dose-dependently and also pyrilamine decreased the cortisol concentration in the serum. The treatment of morphine and pyrilamine together decreased cortisol level significantly and there is a close correlation between pyrilamine and morphine in cortisol secretion.

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Evaluation of changes in testosterone concentration of the rat central nervous system following progesterone administration

H Amini; A Ahmadiani

Abstract

Neurosteroids are steroids that are produced in the central nervous system (CNS). While progesterone and dehydroepiandostendione (the precursors of testosterone) are among the identified neurosteroids, it is not clear that testosterone could be considered as a neurosteroid. The testosterone synthesis has been demonstrated in the brain of castrated frog, but not in the rat brain. In the present work, we tried to demonstrate testosterone synthesis in the rat brain and spinal cord by administration of progesterone. Endogenous level of testosterone was measured in the whole brain, spinal cord, and plasma of mature male, female and castrated Sprague-Dawely rats. Progesterone (5 mg/kg) was injected subcutaneously and testosterone was measured after 3 hours. Testosterone was significantly ($P<0.001$) lower in the CNS and plasma of female and castrated male rats in comparison with male rats. Administration of progesterone failed to alter testosterone concentrations in all groups. The results of this study could not support testosterone synthesis in the rat CNS. This may show that unlike the frog brain, testosterone synthesis in the rat brain has a minor importance.

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The effects of long term handling stress on thyroid function in male rats

M Shabani; S Zahedi-Asl; H Manaheji

Abstract

Inappropriate responsiveness of the stress system may cause numerous alterations in endocrine, metabolic and autonomic systems. Many studies have been carried out on the effects of acute stress on thyroid function but the effects of chronic stress have not been fully investigated. The purpose of this study is to evaluate the effects of long term handling stress on thyroid function. Study was performed on two groups of male Wistar rats. Experimental group received distilled water by oral tube for two months once a day, while control group were not handled or did not received distilled water. At the end of the period animals were anesthetized, abdomen was opened and blood samples were obtained from abdominal aorta. The samples were centrifuged, sera separated and stored at – 20 ?C until the time of the assay. Total triiodothyronine (TT3), total thyroxine (TT4), free triiodothyronine (FT3), free thyroxine (FT4) and T3 uptake were assayed by ELISA and thyroid stimulating hormone (TSH) was determined by radio-immunoassay using DRG kits. Total T4 level was significantly lower in experimental group (3.49 ± 0.1 ?g/dl) than in control groups (4.5 ± 0.34). Free T3, free T4 and T3 uptake were also significantly lower in experimental group ($P<0.005$) as compared to control group. There were also no significant differences in the TSH, TT3, cortisol levels, T3/T4 ratio and body weights between the groups. The alterations in thyroid function of the stressed group suggest that the effect of handling stress must be considered in all neuroendocrine investigations.

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The effect of chronic psychological stress on carbohydrate metabolism in rat

H Zardooz; S Zahedi-Asl; MK Gharibnaseri

Abstract

Several studies have shown that stress has major effects on metabolism. There is also some evidence indicating that stress may induce diabetes mellitus. The present study has investigated the role of chronic psychological stress on carbohydrate metabolism in male Wistar rats (250 ± 50 g). Animals were divided into control and test groups (n = 8/group). Blood samples were obtained by tail snipping from fasted animals, then oral glucose tolerance test was performed on the 1st, 15th and 30th days of experimental period. Different restrainer stressors were used twice daily and to minimize habituation the sequence of the stressors were randomized during the experiment. Glucose concentration was measured by a glucose oxidase method. Results showed that fasting plasma glucose concentration in the control group did not significantly change during the experiment. On the other hand, in the test group fasting plasma glucose concentration increased on 15th (157 ± 5 mg%) and 30th (157 ± 4 mg%) days of experiment as compared to the 1st day (127 ± 8 mg%). The comparison of fasting plasma glucose concentration between control and test groups showed a significant rise on 30th day in the test group. Glucose tolerance test in the test group exhibited a significant reduction in glucose tolerance just on 15th day with respect to the 1st day. The comparison of plasma glucose concentration between the test and control group after performing glucose tolerance test showed no significant difference. In conclusion, the results of this study indicate that psychological stress may induce diabetes types I and II or at least can set a basis for the disease.

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Stress-related effects on neuronal morphology and choline acetyltransferase activity in the hippocampus

M Parviz

Abstract

The effects of semi-chronic stress on neuronal morphology and choline acetyltransferase were studied by injecting rats with dexamethasone. It was found that in the dexamethasone-treated rats the choline acetyltransferase activity had increased in the area where the fibers from the perforant pathway synapse with the granular cell layer of the dentate gyrus and also where the mossy fibers of the granular cell layer leave the dentate gyrus. In the fibers of same area, an increase in their intensity and amount was detected in the dexamethasone-treated rats. The elevated levels of cortisol or (corticosterone in rats) when one is under semi-chronic stress could have an affect on the acetylcholine synthesis and mossy fiber plasticity in the hippocampus.

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The effect of pinna reflex and dynamic stretch on spike discharge of single ?-axons and spindle afferents in caudal muscle spindles in rat

E Barghi; H Mohammadi

Abstract

The ?-axons supplying muscle spindle (MS) induce a particular pattern of activity in spindle Ia and II afferents on contraction. From a dynamic viewpoint, the Ia shows increasing spike rates, but II-afferents do not show any activity. For evaluation of nerve fiber activity, spike amplitude (SA) is suggested to measure it as well as its benefit of diagnosing clinical symptoms. This paper aims to find relative activity between ? axons and spindle afferents and to measure their SAs in both mechanical stimuli and background discharge (BD) for determination of their activity. For this purpose, 15 rats were anaesthetized with urethane (170 mg/kg, i.p.). Spinal dorsal and ventral roots of L3 to L5 were exposed by laminectomy and tails skin was removed. The ?-axons and spindle afferents of caudal MS were isolated from ventral and dorsal roots respectively. Then, ?-axons were examined to determine the affected caudal MS. A single mechanical pinna reflex (PR) and one dynamic stretch (DS) (1-second at 50 Hz) were used to stimulate ? and spindle afferents. The means of peak-to-peak SA computed in PR, DS, and also BD for comparison of means together. Meanwhile, 15 single ?-axons and also corresponding spindle afferents were tested in this study. During PR, the late compound Ia and II spikes elicited later than ?-spikes, as Ia-SAs (193µv±5.39SD) calculated higher than II-SAs (60 µv ± 3.87 SD), and ?-SAs (92 µv ± 4.88 SD) were lower than Ia-SAs. There were also significant differences ($P<0.001$) in the SAs mean values of Ia, II, and ? between PR and BD and also DS and BD, whereas in DS, the SAs of Ia (245 µv ± 5.35 SD) were strongly higher than II (65 µv ± 3.56 SD), and ?-SAs were obviously suppressed (13 µv ± 2.16 SD). There was a strong increase in SA mean values of Ia and II in DS with respect to PR, but ?-activity was inhibited in DS. In PR, the Ia and II spikes elicited later than ?-spikes but it was dependent on ?-activity.

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How are postural strategies modified along the time?

HA Hoseini; M Shaikh; S Talebian; H Bagheri

Abstract

Postural control is critical for effective performance of all goal-directed activities. For maintenance of postural equilibrium against gravity and environmental disturbances, central nervous system produces centrally organized patterns of muscle activity based on initial conditions, perturbation characteristics, learning and intention. These organized strategies are called "postural strategies". The purpose of this study was to examine how are these strategies modified along the time. For this purpose, eighteen healthy male subjects participated in this study. Subjects were randomly placed (with wide & narrow base of supports) on a tilt board that could be locked in two positions (toe-up & toe-down) at a specified angle. Electromyography (EMG) activity of tibialis anterior, pronea and gastrosoleus muscles was recorded for 10 seconds. The first and last 2 seconds were eliminated to reduce technical error. For every second, integrated electromyography (IEMG) was calculated using special software. Then, data were normalized according to the IEMG of the first second. For all muscles and in all positions, there were significant differences in muscle activity in different times periods ($P<0.05$). Also, analysis of data revealed a negative correlation between muscle activity and time ($0.001<P$ value<0.02 and $-0.87<r<-0.98$). It seems that reduction of EMG activity along the time can be explained by the presence of passive structures like ligaments, an increase in passive stability of the musculoskeletal system, and a decrease in active neural control needed for equilibrium maintenance.

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The effect of NDT and BTX-A injection on muscle spasticity

M Shaikh; HA Hoseini; GH Olyaee; S Talebian; R Abolfazli

Abstract

The aim of this study was to compare the effect of neuro-development treatment (NDT) technique and NDT plus botulinum toxin type A (BTX-A) injection on the reduction of spasticity and improvement of the quality of movement patterns of lower limb in hemiplegic patients. For this purpose, 15 hemiplegic (8 female and 7 male cases) with an age range of 27-70 years participated in this study. The patients were in spastic phase and able to walk with or without assisting device. The severity of spasticity was measured by the original Ashworth scale (OAS) and was assigned from 1 to 3. Five patients were treated using NDT method (Group A) and ten patients were treated using combination method (NDT plus BTX-A injection, Group B). BTX-A injection was performed in gastrocnemius, soleus and tibialis posterior muscles. NDT technique was performed in both groups for 10 sessions. Baseline assessments consisted of measuring the severity of spasticity in plantar flexors according to OAS, active and passive range of motion of ankle joint and the quality of movement patterns of lower limb in the standing position according to NDT. In both groups, reduced spasticity, increased active and passive range of motion and improved quality of movement patterns were observed, while the mean changes of these parameters was significantly ($P<0.05$) higher in group B. In each group, the rate of recovery according to the improvement of the quality of movement patterns was correlated with the severity of spastisity ($P<0.05$). Meanwhile, NDT technique may reduce spasticity and may increase range of motion and quality of movement patterns through an inhibitory effect on the abnormal movement patterns and facilitation of normal responses and increasing the ability of patients to perform discrete movements in spite of the presence of synergic movement patterns. Direct attenuating effect of BTX-A on muscle tone and spasticity may increase the effectiveness of NDT technique.

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Praoxon-induced changes in the function of chicken biventer cervices nerve-muscle preparation and the reversal of such changes by pralidoxime

G Poorheidari; N Khodaei; H Sahraei; M Saberi; A Shahriary; A Noroozzadeh; G Pirzad; A Khoshbaten

Abstract

One of the most toxic effects of organophosphate (OP) poisoning has been the paralysis of skeletal muscles that can lead to paralysis of respiratory muscles and death. However, oximes are the only antidotes available to reverse or prevent such toxic effects of OP insecticides and nerve chemical warfare agents. In the present research work, the effect of different concentrations of paraoxon (as an OP) on the function of skeletal muscle and reversal or prevention of these effects by an oxime (pralidoxime, 2-PAM) were studied in chicken biventer cervices nerve-muscle preparation using twitch tension recording technique. For this purpose, twitches of the biventer cervices were evoked by stimulating the motor nerve at 0.1 Hz with pulses of 0.2 msec duration and a voltage of greater than that required to produce the maximum response. Twitches and contractures were recorded isotonically using Narco Biosystems. The results showed that paraoxon (10 nM) induces a significant increase (more than 100%) in the twitch amplitude, while higher concentrations (30 and 100 nM) could induce partial or total contractures. In this study, paraoxon at a concentration of 10 nM was used to examine the capability of pralidoxime to reverse or prevent its effects. Pralidoxime at doses of 300 and 100 µM almost fully reverse (when it was used as post treatment) or prevent (when it was used as pretreatment or at the same time as toxin) the effect of paraoxon. While oxime at doses of 30 and 10 µM could only reverse or reduce this effect to about 25 and 70% respectively, pralidoxime alone had no significant effect on the function of the muscle. These results suggest that this applied method is of high value in studying the functional effects of OPs on skeletal muscle tissues and the reversal effects of antidotes, and pralidoxime by itself may fully reverse such effects.

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The effect of different dendrotoxins on neurotransmission in chicken biventer cervices nerve-muscle preparation

G Poorheidari; A Shahriary; AL Harvey

Abstract

In recent decades natural biological products (including components of animals and plants) have increasingly been investigated to discover new drugs. In this study, different dendrotoxins from Eastern green mamba (Dendroaspis aungusticeps) venom were examined for their effects on neurotransmitter release in chicken biventer cervices nerve-muscle preparation using twitch tension recording technique. Twitches of the biventer cervices were evoked by stimulating the motor nerve at 0.1 Hz with pulses of 0.2 msec duration and a voltage of greater than that required to produce the maximum response. Twitches and contractures were recorded isotonically using polygraphs (Grass model 79 and Grass model 79B) and Grass FT03 force transducers. Alpha- and ?-dendrotoxin (10 ?g/ml ? 1.4 ?M) produced a significant augmentation in twitch amplitude, whereas ?- and ?-dendrotoxin did not appear to be effective at this concentration. These results were consistent with other reports using binding competition assays to demonstrate that ?- and ?-dendrotoxin are more potent than ?- and ?-dendrotoxin in their affinity for the binding sites of radiolabelled ? -dendrotoxin. Overall, it is concluded that the potency of different dendrotoxins are as follows: ?>?> ?>?.

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The effect of HI-6 on reversal or prevention of changes induced by paraoxon in the function of Chicken biventer cervices nerve-muscle preparation

N Khodaei; G Poorheidari; G Pirzad; A Khoshbaten

Abstract

Paralysis of skeletal muscles, which can lead to paralysis of respiratory muscles and death, is one of the most toxic effects of organophosphates, and oximes are almost the only known antidotes that can reverse or prevent such toxic effects. In the present research work, possible reversal or preventive effect of different concentrations of the relatively new oxime (HI-6) on changes induced by paraoxon on the function of skeletal muscle were studied in chicken biventer cervices nerve-muscle (CBC) preparation using twitch tension recording technique. For this purpose, twitches of the CBC muscle were evoked by stimulating the motor nerve at 0.1 Hz with pulses of 0.2 msec duration and a voltage of greater than that required to produce the maximum response. Twitches were recorded isotonically using Narco Biosystems. Our prior findings revealed that paraoxon at a concentration of 10 nM induces a significant increase (more than 100%) in the twitch amplitude, and therefore, this concentration was used to examine the efficacy of HI-6 to reverse or prevent such effects. HI-6 at 1000 µM could almost fully reverse (when it was used as post treatment) or prevent (when it was used as pretreatment or at the same time as toxin) the effect of paraoxon. It could also reverse or reduce this effect to about 25, 50 and 75% at 300, 100 and 30 µM respectively. Furthermore, HI-6 at 10 µM produced no significant preventive or reversal effect. However, HI-6 alone at 1000 µM increased the twitch amplitude by about 20%. These data indicated that HI-6 could be recognized as an antidote of paraoxon, although it may have other effects at high concentrations.

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The effect of obidoxime on reversal or prevention of paraoxon-induced changes in the function of Chicken biventer cervices nerve-muscle preparation

N Khodaei; G Poorheidari; A Noroozzadeh; A Khoshbaten

Abstract

Paralysis of skeletal muscles, which can lead to paralysis of respiratory muscles and death, is one of the most toxic effects of organophosphates (OPs), and oximes are the only available antidotes that can reverse or prevent such toxic effects. In the present study, the possible reversal or preventive effect of different concentrations of obidoxime (toxogonin) on changes induced by paraoxon (as an OP) on the function of skeletal muscle were investigated in chicken biventer cervices nerve-muscle (CBC) preparation using twitch tension recording technique. For this purpose, twitches of the CBC muscle were evoked by stimulating the motor nerve at 0.1 Hz with pulses of 0.2 msec duration and a voltage of greater than that required to produce the maximum response. Twitches were recorded isotonically using Narco Biosystems. From prior studies, it was obtained that paraoxon at 10 nM induces a significant increase (more than 100%) in the twitch amplitude, and therefore, this concentration was used to examine the possible capability of obidoxime to reverse or prevent such effects. Obidoxime at 300 µM could almost fully reverse (when used as post treatment) or prevent (when used as pretreatment or at the same time as toxin) the effect of paraoxon. However, oxime at 100, 30 and 10 µM could only reverse or reduce this effect to about 25, 50 and 75% respectively. Oxime alone had also no significant on the function of the muscle at concentrations used. These data suggest that obidoxime alone may fully reverse the effect of paraoxon on skeletal muscle tissues.

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Cold, season, and incidence of bells palsy

D Savadi Oskoyi; A Abedi; Kh Rostami

Abstract

Bell's palsy is a relatively common disease characterized by the sudden onset of unilateral facial paralysis. Using a centralized system that contains demographic data, the authors estimated rates, trends, and demographic risk of Bell's palsy during a 2-year period. There were 140 incident cases of Bell's palsy among patients referring to private clinics (a total of 6500 patients). The crude incidence rate was 2.15%. Incidence rates were high in an age range between 20-30 years and were equal among females and males. Incidence of BP was higher in cold season than warm ones and higher in cold regions. The results are consistent with hypotheses regarding viral etiologies (e.g., reactivation of herpes simplex) of Bell's palsy in which cold can activate this virus.

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Spike timing dependent plasticity: mechanisms, significance, and controversies

B Babadi; MH Kazemi; K Moradi; M Sadat Safavi; Sh Gharibzadeh

Abstract

Long-term modification of synaptic strength is one of the basic mechanisms of memory formation and activity-dependent refinement of neural circuits. This idea was purposed by Hebb to provide a basis for the formation of a cell assembly. Repetitive correlated activity of pre-synaptic and post-synaptic neurons can induce long-lasting synaptic strength modification, the direction and extent of which depends critically on the temporal order of pre-synaptic and postsynaptic activity. This process, “spike timing dependent plasticity” (STDP), is well suited for creating neural networks with predictive behavior. The cellular mechanisms of STDP are not well understood. But it is believed that a transient increase in postsynaptic intracellular calcium plays a central role and downstream to calcium, several protein kinases and phosphatases signal for the changes in synapse. In most cases, induction of LTD and LTP depends on activation of NMDA receptors that seems to act as coincidence detector by the virtue of their particular property that channel opens only when glutamate binds to its receptor and magnesium block is removed by coincidence depolarization. The degree of channel opening will then determine the amount of calcium passing through the pore. At first look it seems that high levels of calcium induce LTP and moderate calcium levels favor LTD. If this was the case, we were to observe an additional LTD window in positive spike timing range. However, such an additional LTD was not observed in most studies that have mapped out the asymmetric spike timing window. It seems that other spatial and temporal patterns of calcium transient are also important in synaptic modification. Another interesting feature of STDP is its effect on the behavior of neural networks. According to modeling studies, STDP causes a balanced irregular firing regime in networks of neurons sensitive to the pre-synaptic action potentials. Molecular biology and computational tools are now beginning to interpret synaptic plasticity in terms of quantitative and spatiotemporal rules, which are likely to bridge the gap between synaptic physiology and neural network behavior. This review will try to represent a perspective of the latest findings in this field and current opinion and possible future vista.

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Role of STDP in regulation of neural timing networks in human: a simulation study

M Sadat Safavi; B Babadi; K Moradi; MH Kazemi; Sh Gharibzadeh

Abstract

Many physiological events require an accurate timing signal, usually generated by neural networks called central pattern generators (CPGs). On the other hand, properties of neurons and neural networks (e.g. time constants of neurons and weights of network connections) alter with time, resulting in gradual changes in timing of such networks. Recently, a synaptic weight adjustment mechanism has been docu¬mented in some synapses, based on timing of pre- and post-synaptic electrical activities, called s spike-timing-dependent plasticity (STDP). We present a model in which the accuracy of the timer network sig¬nificantly improves by using this mechanism. In our model, based on leaky integrate and fire elements, we used 30 timer neurons (time constant=0.0005, resting voltage=-60 mv and threshold=-50 mv) and one feedback neuron (Time constant=0.001, resting voltage=-60 mv, threshold=-50 mv). Some simulated noise was applied to the synaptic connections (random deviation up to 20% of the default synaptic weight). We applied STDP to feedback neuron-timer neurons connections (for both long term depression and long term potentiation: dw=0.01×e-(ISI/100+1) and examined the inter-spike interval (ISI) of the feedback neuron as our model output. There was a significant reduction in ISI variation with (Mean ISI=230 and SD=2.21) and without (Mean ISI=204, SD=10.1) applying STDP. In this simulation, weak synapses will be strengthened because their post-synaptic timer neuron will fire after feedback neuron (long term potentiation) and strong synapses will be weakened because their post-synaptic timer neuron will fire before feedback neuron (long term depression). Therefore, the effect of noise would be partially compensated.

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Evaluation of the effect of dendritic branching on signal processing in hippocampus pyramidal cells

A Ertiaee; A Salehi Omran; M Parviz; Sh Gharibzadeh

Abstract

Since branching region of an active nerve fiber is an abrupt widening of the structure, two concepts emerge: first, the stimulating current must be sufficient to raise the outgrowing fibers above the thresh¬old, and secondly, the stimulating current will be divided in proportion to the characteristic admittance of the branches. On the other hand, blocking of the nerve impulse in this region is an important issue that has been evaluated in several different ways including Hodgkin-Huxely, Markin-Chizmadzhev, general leading edge, cubic leading edge, and piecewise linear leading edge models. Each of these models has its advantages and disadvantages, but none of them can be completely accounted. Meanwhile, hippocampus is a part of the nervous system that is involved in sophisticated functions including memory. Hence, we have evaluated the behavior of branching dendrites in pyramidal cells of the hippocampus not only to elucidate the effect of branching on signal processing, but also to understand more precisely the function of pyramidal cells. In this study, MATLAB software (6.5) was used to design a program that evaluates the effect of branching point on propagation or blockade of impulse. In order to execute this program, we have modeled the nerve fiber as an electrical circuit and exerted some neurobiological features of pyramidal cell to this model. Finally, a MATLAB program was introduced that can be used as a model of pyramidal cells in experimental researches.

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Artificial neural networks: applications in predicting pancreatitis survival

B Saboori; R Azadi; SM Aghdaee; Sh Gharibzadeh

Abstract

Artificial neural networks are intelligent systems that have successfully been used for prediction in different medical fields. In this study, the efficiency of a neural network for predicting the survival of patients with acute pancreatitis is compared with days-of-survival obtained from patients. A three- layer back-propagation neural network was developed for this purpose. Clinical data (e.g. patient's age, white cell count, blood sugar level, enzyme levels, etc.) were introduced as input to the network and the survival of patients was obtained as output. The weights of all layers were randomly assigned and were modified according to clinical data obtained from patient's records. The network was trained until the error rate fell below five percent. After training, data from another set of patients (not introduced to the network before) were presented to the neural network and its output was compared to the patient's days-of-survival. The results showed that the network significantly outperformed clinical criteria used for this purpose. Due to the importance of identifying patients with acute pancreatitis who are at a high risk of death and the inefficiency of precise predicting of clinical criteria, it could be concluded that neural networks are efficient in predicting illness severity in patients with acute pancreatitis.

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Glutaminergic receptors in rostral ventrolateral medulla mediate the cardiovascular responses to activation of bed nucleus of the stria terminalis in female rats

M Hatam; M Ganjkhani; CVR de Oliveira; J Ciriello

Abstract

The bed nucleus of the stria terminalis (BST) has been known to contain estrogen (E)-concentrating neurons. In addition, injections of E into BST have been reported to potentiate the sympathoinhibitory arterial pressure (AP) and heart rate (HR) responses elicited by glutamate (Glu) stimulation. In this study, the effect of glutamate antagonist receptors in the rostral ventrolateral medulla (RVLM) on the cardiovascular responses to Glu stimulation of BST (0.25 M; 20 nl) was investigated in the chloralose anaesthetized, ovariectomized (OVX; n=6) or OVX+E-treated (30 pg/ml plasma E; n=10) female Wistar rats. Glu stimulation of the BST decreased AP (-15 ± 5 mmHg) and HR (-25.23± 2 bpm) in the OVX+E animals and in OVX only animals (AP, -29.78 ± 4.17 mmHg; HR, -17.3 ± 3.1 bpm). Microinjection of glutamate antagonist receptors, kynurenic acid (5.0 mM, 100nl) into the ipsilateral RVLM reversibly attenuated glutamate-induced bradycardi and depressor responses to BST stimulation in OVX only animals (OVX; n=8 AP,-13.7±2.6 and HR,-8.1±4.3) but in OVX+E animals only attenuated glutamate induced bradycardia, but not depressor response (OVX+E;n=7 AP,-11.7±3.8 and HR, -16.7±2.3). These data suggest that RVLM sympathetic premotor neurons contain glutaminergic receptors mediate the sympathoinhibitory responses to stimulation of BST in OVX animals.

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Functional role of GABA in the nervous system under impact of testosterone-propionate in different seasons

DK Rustamov; MI Safarov

Abstract

The contents of GABA, glutamine (Glu) and asparagine (Asp) acids as well as activities of glutamatdecarboxylase (GDK; CF 4.1.1.15) and 4-aminobutirat: 2-oxoglutarataminotransferase (GABA-T: CF 2.6.1.19) in brain cortex, cerebellum, brainstem and hypothalamus under impact of testosterone propionate at a dose of 5 mg/100 g of alive weight of adult albino male rats in spring and autumn were studied. It was revealed that under effect of testosterone-propionate at a dose of 5 mg/100 g of alive weight in tissues and mitochondrial fractions of studied brain structures the level of components of GABA system (GABA, GDC and GABA-T) and Glu and Asp contents in different seasons (spring and autumn) decreases. These changes are more prominent in spring period than in autumn.

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Non-cholinergic effects of paraoxon on [3h]-GABA release from rat cerebellar giant synaptosomes

SM Hosseini; A Asgari; HA Mehrani; A Koshbaten

Abstract

Diethyl p-nitrophenyl phosphate (paraoxon) is the active toxic metabolite of parathion. Some evidences indicate that OPs affect the GABA system via noncholinergic mechanisms. The purpose of this study was to investigate the effects of paraoxon on K+-evoked [3H]-GABA release from cerebellar synaptosomes. Adult male rats (200 ± 30 g; 3-4 months old) were sacrificed by decapitation and the cerebellum was removed immediately and homogenized. Homogenate was centrifuged twice at 1000 × g for 5 min (all in 0-4 ?C). Synaptosomes were incubated with [3H]-GABA (S.A 99 Ci/mmol, 0.1 µm). Then, aliquots of the synaptosomal suspension were layered on microporous filters at the bottom of superfusion chambers (14900 Superfusion System, Raiteri,s Method, UGO BASILE, Italy). Following 34 minutes of superfusion (time required to equilibrate the system, t = 0), fractions were collected every minute and the radioactivity in the different samples was quantified by liquid-scintillation counting. At t = 8 (s1) and t = 28 (s2), synaptosomes were depolarized with KCl (30 mM). Paraoxon was added to the superfusion medium concomitantly with the second stimulus (s2) and the ratio of s2 /s1 release was compared between the control and test groups. Present data indicate that paraoxon increases spontaneous and K+-evoked [3H]-GABA release from rat cerebellar giant synaptosomes, possibly via noncholinergic mechanisms.

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The effect of morphine on some electrophysiological parameters of paragigantocellularis and locus coeruleus nuclei interconnections

F Ghaderi Pakdel; S Semnanian; Y Fathollahi

Abstract

As one of the most important diffused brain modulatory systems, the nucleus locus coeruleus (LC) receives most of its afferents from the nucleus paragigantocellularis (PGi) and plays a major role in the control of drug dependence and some emotional and exciting states. For detailed investigation of the effect of morphine on relationship between these two brain stem nuclei, the activity of the rat (Sprague-Dawley) LC neurons was examined after being dependent on morphine in comparison with independent group. The activity of the nucleus LC neurons with and without the activity of nucleus PGi neurons was recorded using extracellular single unit recording technique. Lidocaine microinjection (1 ml, in 1-2 min, 2.5%) was used for inactivation of PGi. The PSTH (Peri-Stimulus Time Histogram) of the neuronal activity showed a decrease in neuronal firing rate of LC by 85.8±1.7% in morphine-dependent group (from 2.95 spike/sec to 0.64 spike/sec, n = 4) and by 48.3±4.3% (from 3.5 spike/sec to 1.94 spike/sec, n = 6) for independent one. The difference was significant ($P<0.001$). The time period of the decrease in activity was 120±9 min for dependent group and 44.8±4.5 min for independent one. The difference was also significant ($P<0.001$). It seems likely that development of morphine dependence entails some changes in neurons of the nucleus LC, which may cause an increase in its neuronal firing rate in the presence of nucleus PGi neuronal activity.

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The role of D1/DA1 dopamine receptors on histamine-and carbachol-induced gastric acid secretion in male rat

T Ghasemi Bossejine; A Eliassi

Abstract

Dopamine has an effective role on gastric acid secretion (GAS). The role of peripheral and central dopamine D1/DA1 receptors on stimulated GAS are unknown. The objective of the present study was to use SKF38393 and SCH23390 on stimulated GAS. For all gastric sampling, animals (Sprague Dawley rats) were anesthetized and a polyethylene tube was introduced into the stomach through esophagus and a cannula was inserted into the pylorododenal junction and passed up into the stomach. After washing, the stomach was perfused with physiological saline and perfusate was titrated with NaOH. Intravenous (jugular vein) infusion of histamine- and carbachol-induced marked increased in GAS with a peak response that started from 20 and 30 min respectively up to the experiments. Administration of SKF (1mg/kg) produced a decrease (50 %) in histamine - stimulated GAS. This effect completely removed by SCH23390 (0.1 and 0.01 mg/kg). The acid suppressant effect of SKF was not observed on carbachol-induced GAS. In conclusion, there is probably a regulatory mechanism of GAS by D1 receptors interaction with H2 but not M3 receptors.

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Modulatory role of nitric oxide releasing NSAIDs in aging- and lipopolysaccharide-induced cognitive dysfunction in mice

CS Patil; NK Jain; J Lehmann; SK Kulkarni

Abstract

Inflammatory processes play a critical role in the pathogenesis of the neurodegenerative disorders and are associated with cognitive impairments. Nonsteroidal anti-inflammatory drugs (NSAIDs) and nitric oxide releasing NSAIDs are reported to be effective in reducing the risk of developing AD or cognitive impairments. Present experiments were performed to study the possible effect of nitro naproxen (NO-naproxen) on cognitive performance of young, aged and lipopolysaccharide (LPS) treated mice (an animal model of AD) using one trial step–through type of passive avoidance and in elevated plus maze task. Chronic administration (15 days) of naproxen (ED50 dose) and NO-naproxen (molar equivalent of naproxen) significantly reversed the age-induced retention deficits in both test paradigms but the effect of NO-naproxen was pronounced as compared to naproxen. In young and LPS- treated mice, chronic administration of NO-naproxen produced a significant increase in cognitive performance as compared to naproxen. Further, NO-naproxen did not produce any alteration in gastric function since naproxen was associated with gastric toxicity (ulcer index, pH and free/ total acidity). Based on this the study indicates that chronic treatment with nitric oxide releasing NSAIDs reverses the cognitive deficits in age- and LPS treated mice. These findings indicate that NO-NSAIDs besides inhibiting the caspase activity and pro-inflammatory cytokines in CNS are less toxic to the gastrointestinal tract and may prove to be suitable for the treatment of AD.

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GABAergic system for Ptychodiscus brevis toxin-induced depression of synaptic transmission elicited in isolated spinal cord from neonatal rats

JN Singh; SB Deshpande

Abstract

The involvement of inhibitory transmitters for Ptychodiscus brevis toxin (PbTx)-induced depression of spinal synaptic transmission in neonatal rats was investigated. Stimulation of a dorsal root evoked monosynaptic reflex (MSR) and polysynaptic reflex (PSR) potentials in the segmental ventral root. The PbTx depressed the reflexes in a concentration-dependent manner and this depression was blocked by GABAA antagonist, bicuculline (1 M). GABA also produced depression of the reflexes in a concentration-dependent manner. Simultaneous application of sub maximal concentrations of PbTx (28 M) and GABA (30 M) enhanced the depression (>75%). In contrast, PbTx alone (28 M) depressed the MSR and the PSR by 33 and 47%, respectively, and GABA (30 M) alone depressed the reflexes by 30%. The N-methyl- -aspartate receptor antagonist, -2-amino-5-phosphono-pentanoic acid (10 M), blocked the PbTx-induced depression of MSR and also the enhancement of GABA response by PbTx. A glycine receptor antagonist, strychnine (1 M), failed to block the depression by the toxin up to 28 M; however, the depression was attenuated significantly at 84 M of the toxin. The results indicate that PbTx depressed the spinal reflexes via GABAA receptors. Furthermore, the potentiation of GABAergic action by PbTx requires the N-methyl-D-aspartate-dependent mechanism.

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Does dietary fish oil improve nerve conduction velocity via regulating of blood glucose level in diabetic rat?

M Jarrahi

Abstract

Although it is a well-established fact that dietary fish oil can improve nerve conduction velocity in diabetic rat, but the exact mechanism of its action is unknown. Therefore, in this study we measured the blood glucose level of all animals for evaluating the possible effect of fish oil in improvement of nerve conduction velocity in diabetic neuropathy via regulation of blood glucose level. For this purpose, 30 male albino rats were randomly assigned to one of the three treatment protocols as follows: control, diabetic and fish oil groups. Blood glucose of animals was determined in all groups at the beginning and at different days after the experiment. Diabetic and fish oil-treated diabetic groups received alloxan (180 mg/kg, s.c.) at the beginning of the experiment. Animals with blood glucose higher than 300 mg/dl were considered diabetic. From the 10th day, Menhaden fish oil was used (by 10% of daily food consumption) through orogastric tubing for ten days. After 20 days, motor nerve conduction velocity (MNCV) of tibial nerve was measured. It was found out that there was a 17.1% ($P<0.01$) reduction in MNCV of diabetic group in comparison with control group. There was also a 6.5% ($P<0.05$) increase in MNCV of fish oil-treated diabetic group in comparison with untreated-diabetic group. Results also showed that there were no significant differences between the groups regarding blood glucose level. On this basis it can be concluded that pretreatment of diabetic rats with fish oil does not act through changes of blood glucose level, and it may act through other unknown pathways.

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Evaluation of relation of hyperlipidemia and polycythemia with incidence of cerebral stroke

M Nobahar; AA Vafaei; S Masoumi; A Samaei

Abstract

Cerebral stroke is a leading cause of serious disability and is the third leading cause of death in most countries and its prevention is a key to reduction of morbidity, mortality, and disability. This study investigated the relationship between hyperlipidemia (cholesterol and triglyceride), polycythemia, and incidence of cerebral stroke. Data was obtained from 70 participants that hospitalized for a one-year period and recorded in checklists in Fatemiah hospital (Semnan, Iran). In this respect, levels of serum cholesterol and triglyceride and hematocrit were determined. Results indicated that the mean age of patients was 72 years old. The percentage of risk factors was 34% (hypercholesterolemia), 20% (hypertriglyceridemia), 9% (polycythemia) and other risk factors were 67% (hypertension), 40% (diabetes), 44% (heart diseases), 19% (smoking) and 24% only reported a history of stroke in their family. It may be concluded that hyperlipidemia and polycythemia are two major risk factors with involvement in incidence of cerebral stroke. Therefore, control of these factors can reduce the prevalence of stroke.

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Central effect of histamine and peripheral effect of histidine on food intake in rabbits

G Vafaye Saiah; E Tamaddonfard

Abstract

Histidine is an amino acid precursor of neuronal histamine. It is evident that peripheral injection of histidine produces effects that resemble to centrally administered histamine. The aim of the present study was to compare the effects of intracerebroventricular (ICV) injection of histamine and inraperitoneal (IP) injection of histidine on food intake in rabbits. ICV injections of histamine (12.5, 25, 50,100 and 200 ?g/rabbit) were performed through a permanent cannula, which was implanted into the right lateral ventricle of brain. IP injection of histidine (31.25, 62.5, 125, 250, and 500 mg/kg) were injected using a 23-gauge injection needle. Cumulative food intake was measured at 1, 2, 3, 6 and 24 h after injections. The results showed that histamine (ICV, 12.5 and 25 ?g) and histidine (IP, 31.25 and 62.5 mg/kg) had no effect on food intake. A short-time (2 h) reduction of food intake was observed when the doses of 50 ?g of histamine and 125 mg/kg of histidine were applied. The highest reduction (total 24h) of food intake occurred at the 200 ?g of histamine and 500 mg/kg of histidine. Based on the results of the present study it is concluded that activation of brain histamine in rabbits induces anorexia. Histidine, a precursor of histamine, mimics effects of the centrally administered histamine. This may be due to conversion of histidine into histamine in the brain.

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Central effect of histamine and antihistamines on food intake in freely feeding and food-deprived rabbits

G Vafaye Saiah; E Tamaddonfard

Abstract

Several lines of evidence suggest that brain histamine may be involved in the central control of food intake. The effect of histamine on feeding is mediated through three kinds of receptors (H1, H2, and H3). The present study was designed to investigate the effect of intracerebroventricular injection of histamine, promethazine (H1 antagonist) and ranitidine (H2 antagonist) on food intake of freely feeding and food-deprived (for 16 h) rabbits. For intracerebroventricular injections, a 23-gauge, 18 mm long stainless steel guide cannula was surgically implanted into the left lateral ventricle of brain. Ten days after cannulation, intracerebroventricular injection of normal saline (control), histamine (25, 50, 100, and 200 ?g/rabbit) promethazine and ranitidine at the same doses of 100 and 200 ?g/rabbit alone or before injection of histamine were performed using a 25?l Hamilton's syringe. Food intake was measured at 1, 2, 3, 6 and 24 h after injections. Data were analyzed by factorial ANOVA and Duncan test. The results showed that 16 h food deprivation increases 1, 2 and 3 h food intake. The 6 and 24 h food intake was not affected by food deprivation. In freely feeding rabbits, histamine (25 ?g) had no effect, but at doses of 50, 100 and 200 ?g decreased food intake. Promethazine (100 and 200 ?g) alone increased food intake, and pretreatment with promethazine prevented the histamine-induced anorexia. Ranitidin alone had no effect, and pretreatment with ranitidine did not inhibit the effect of histamine. In the 16 h food-deprived rabbits, histamine (25 and 50 ?g) had no effect, but histamine (100 and 200 ?g) decreased food intake. Promethazine and ranitidine alone had no effects. Pretreatment with promethazine blocked the histamine-induced anorexia of histamine on food intake. Based on the findings of the present study it is concluded that the activation of brain histamine system produces anorexia in the freely feeding and food-deprived rabbits. The anorectic effect of histamine is mediated through its central H1, but not H2 receptors.

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Interaction of intracerebroventricular injection of captopril and histamine on water intake in adult male Wistar rats

S Oryan; P Rostami; MR Zarrindast; M Navaeian

Abstract

Several evidences have indicated that many nuclei in the brain have critical role in water intake. Angiotensin II is one of the most important neurotransmitters involved in regulation of fluid homeostasis. In this study, the interaction of i.c.v. injection of captopril and histamine on water intake in adult male water-deprived rats was investigated. Rats were implanted with guide cannula aimed toward lateral ventericle. After recovery period, the animals deprived of water for 24 h, and then, drugs were injected and the volume of water intake was measured for 1 hour. The results showed that captopril (7 and 10 µg/rat) decreased, while histamine (40 and 80 µg/rat) increased the rate of water intake. Meanwhile, captopril pretreatment blocked the responses induced by histamine. These data suggest that histamine and angiotensin systems may have an interaction on water intake mechanism.

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Malnutrition and development among rural and urban school children in southern province of Hormozgan in Iran

A Aminpour; M Habibi; M Molavian

Abstract

The association between chronic severe malnutrition and poor mental development as a social and health problem that may lead to a behavioral dysfunction has been reported in several studies. This study has attempted to investigate the relation between stunted children and cognitive measures (IQ). In this study, 1945 school children (845 urban, 1100 rural) have randomly selected by using variable probability and cumulative frequency methods on 1.5 percent of whole school children population (130702) in southern province of Hormozgan. The nutritional status of children were determined by Index of Wt/Age, Ht/Age and Wt/Ht and intellectual quotient (IQ) measured by using good-enough Harriss Test on children attending the first year of school in the studied regions. The IQ test has been normalized for the studied population. The results showed that the percentage of stunting and Ht/Age (Z score) were higher in rural than urban population (32.5% in rural and 28.3% in urban children). The percentage of IQ scores below 80 was also greater in rural than urban children (63.8% in rural and 47% in urban children). Also, the number of children with IQ score above 110 were higher in urban than rural children (16.2% in urban and 9.1% in rural children). Stunting has also been diagnosed as the past-chronic malnutrition among children, mainly due to inadequate dietary intakes. The IQ scores of children were also affected as a consequent factor, which could lead to their limited school achievement.

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The effect of constant magnetic field on the formalin-induced pain in mice

M Fereidoni; E Navid-ahmadi

Abstract

Magnetic field (MF) has had many therapeutic usages in traditional medicine. Today, it is used to accelerate the recovery period after surgical operations by some medical centers and in some horse-training farms in the world. There are some scientific reports on the alternative effect of MF on nervous system function, convulsion, pain, neurons, embryonic cells and immune system cells. For this reason, the effect of constant MF on formalin-induced pain was investigated in the present project. For this purpose, two magnets (80 and 120 militesla (mt) at 15 centimeter distance from each other) were used. It was 4 mt in the middle of 30 cm distance between two magnets when they were opposed to each other by different poles. These three MF powers were used to perform the tests. Tests were designed in two categories; i.e. acute and chronic. In the acute experiment, formalin test (FT) was performed in three groups (n = 6) during exposure MF power. In the chronic case, groups (n = 9) were exposed to MF for two hours a day for 10 days and then FT was performed. Results of the acute tests showed additive effect of MF on pain sensation, as MF with 80 mt and 120 mt powers had 15.5 % and 19.5% additive effects ($P<0.01$) in the first phase, and 3.7% and 14.9% additive effects ($P<0.05$) in the second phase of FT respectively. Results of the chronic tests showed two opposite effects in the first and second phases of FT. In this respect, MF with 4, 80, and 120 mt power had 8% ($P<0.05$), 1.5%, and 3.5% additive effects in the first phase, and 14.9%, 16.7% and 31% attenuating effect ($P<0.01$) in the second phase respectively. Some reports have shown MF acutely evokes calcium channels and increases intracellular concentration of calcium and so affects the cell excitability. It may also increase the excitability of pain receptors and its pathway in the central nervous system (CNS), so it is a probable reason for acute additive effect of MF. It has also indicated that chronic MF increases release of serotonin and enkephalins. Also, the inhibitory effect of long-term high intracellualr calcium concentration on the cell activity has been proved. These two mechanisms may be responsible for chronic attenuation of pain sensation at the second phase of FT.

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Gabapentin increases analgesic effect of morphine in male rats

M Shamsi Meimandi; M Mobasher; Gh Sepehri; N Ashrafgangui

Abstract

The clinical usefulness of morphine for treatment of chronic pain is limited by development of tolerance. So many authors have proposed the co-administration of other drugs. This study was performed to evaluate the role of gabapentin on analgesic effect of morphine. Four groups of male rats (220-250 g) were examined for time latency using tail flick test: control, morphine (M), gabapentin (GB), and gabapentin-morphine (GB-M)-treated groups. Rats received morphine (10 mg/kg, s.c.) or gabapentin (75 mg/kg, i.p.) or both of them twice a day for 10 days. Control rats received normal saline as scheduled time. Tail flick latency time was recorded 3 times (5 minute interval) before drug injection and at 60, 65 and 70 minutes after drug injection. In this respect, %MPE was calculated for all groups. Analysis of variance showed no significant difference of %MPE in control and GB groups, while in M and GB-M groups, %MPE changed significantly during the study. GB had no analgesic effect, while M and GB-M had significant analgesic effect as compared to control. %MPE of GB-M was also significantly higher than M at days 5, 7 and 9. It can be concluded that co-administration of gabapentin enhances the analgesic effect of chronic use of morphine. Precise mechanism(s) should be investigated in future.

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Role of hypocretin-1,2 (orexin A and B) in pain perception

JI Mobarakeh; MH Heidari; H Akbari; MH Asadi; AK Khamaneh; M Bakhtiyari; S Nishino; K Yanai

Abstract

Hypocretins/orexins are primary excitatory neuropeptides located exclusively in neurons of the lateral hypothalamic area, which send projections to most monoaminergic nuclei. It has been reported that i.c.v. injection of hypocretin 1 (orexin A) enhances wakefulness in rats and mice. The present work was carried out to examine the roles of hypocretins in nociception in mice. The presence of robust projections from the hypothalamus to laminae I and II of the spinal cord strongly suggests the role of hypocretins in the nociceptive pathways. Localization of the fibers of hypocretin-containing neurons in the hypothalamus, locus coeruleus, thalamus and periaqueductal gray is also consistent with their central roles in sensory processing. To investigate the role of these hypothalamic peptides, C57BL/6 mice were administered with hypocretin 1 and 2 (i.c.v.) and intrathecally (I.T.), and examined for pain thresholds using three kinds of nociceptive tasks. These included assays for thermal (hot-plate, tail-flick, paw-withdrawal), mechanical (tail-pressure), chemical (formalin, capsaicin and abdominal stretch) nociceptions and behavioral responses. The I.C.V. and I.T. administration of hypocretins produced morphine-like anti-nociceptive effects in mice. The anti-nociceptive effects of hypocretins 1 (orexin A) were more remarkable than those of hypocretins 2 (orexin B). The effects of hypocretin 1 were completely blocked by 3-dipropyl-8-cyclopentylxanthine (DPCPX), a nonselective adenosine receptor antagonist but not by naloxone. No motor impairments were observed with both of the compounds at the doses studied. The present findings suggested that the hypocretin-containing neurons in the hypothalamus have a potential role in the modulation of nociceptive transmission.

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Deletion of histidine decarboxylase (HDC) enhances the antinociceptive effects of orexin A in the central nervous system

JI Mobarakeh; MH Heidari; H Akbari; MH Asadi; AK Khamaneh; M Bakhtiyari; S Nishino; K Yanai

Abstract

It has long been established that histamine plays a role as a mediator of inflammation. From numerous studies, it has been well known that the amine has many pharmacological actions on a variety of organs. To evaluate the role of histamine in pain perception, we generated HDC knockout mice using a gene targeting method. Histamine is a hydrophilic autacoid, and in most tissues it is stored and synthesized by mast cells, basophils, parietal cells endothelial cells and neurons. In these deficient mice, the number of mast cells and histamine contents are dramatically decreased. The effects of deletion of histidine decarboxylase on orexin A-induced antinociception were examined on various assays for thermal nociception (tail-flick), mechanical nociception (tail-pressure) and chemical nociception (capsaicin tests) using HDC knockout and their wild-type mice. In these nociceptive assays, intracerebroventricularly (ICV)- administered morphine produced significant anti-nociceptive effects in wild-type mice. The anti-nociceptive effects produced by ICV- administered orexin A were enhanced in HDC knockout mice. These results indicate that antinociception by orexin A was enhanced by decrease in histamine contents. Furthermore, our report demonstrated the possible interactions of orexin A with histaminergic neurons in pain perception, which provides theoretical support for the use of histamine receptor antagonists and orexin A as analgesics in the pain study.

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Antinociceptive effect of acetaminophen in low frequency tail shock vocalization test in male NMRI rats

MR Vaez Mahdavi; M Khalili

Abstract

The present study was a protocol for investigation of the effect of acetaminophen (i.p.) on pain induced by electrical stimulation. We used tail shock vocalization (TSV) test for measurement of the pain. In this test, the animals were restrained and stimulated by two electrode implanted on the middle of the animal's tail. The latency from stimulation of the tail till vocalization of the animal was measured and considered as pain scoring. We used high (80 Hz) and low (3 Hz) frequency stimulation in TSV test. Application of the acetaminophen at doses of 200 and 400 mg/kg could not produce a significant anti-nociceptive effect in high frequency TSV, but the pain induced by low frequency TSV markedly diminished at doses of 200 and 400 mg/kg of acetaminophen. Therefore, it is concluded that acetaminophen as an analgesic drug can attenuate low-frequency (3 Hz) TSV-induced pain

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The efficacy of intraperitoneal administration of acetaminophen on pain produced by formalin test in mail NMRI rats

M Khalili; MR Vaez Mahdavi

Abstract

In this study, IP administration of acetaminophen and its half-life were investigated. Acetaminophen as an analgesic drug ordinarily is used in tablets or syrup form. Therefore, there is no report about administration of acetaminophen through injection. Acetaminophen in ED50 dose (200 mg/kg) was injected into the animals intraperitoneally, and its analgesic effect was investigated by formalin test. We used acetaminophen 10 and 30 min before and at the same time of formalin injection. Our results showed that the usage of acetaminophen 10 min prior to formalin injection could exert a significant analgesic effect in chronic phase of formalin test. Also, a marked analgesic effect was obtained in the acute and middle part of chronic phase (15-40 min) of formalin test. However, the late phase (35-60) of chronic pain was diminished significantly, when acetaminophen administered at the same time of formalin injection. In addition, our data show that acetaminophen can be used as injection form, and through IP route has a half-life of nearly 1 hour.

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Peripheral effect of phenylephrine and prazocin on phasic pain during estrus cycle in rats

M Taherianfard; R Mazlomi

Abstract

The neuromodulatory interaction of sex steroids with the opioid system may involve in sex differences in pain sensitivity. Also, sex steroids undergo fluctuations during estrus cycle. On the other hand, adrenergic system can influence pain sensitivity. Thus, the aim of the present study was to investigate the effect of phenylephrine and prazocin on pain sensitivity during estrus cycle. For this purpose, 6 adult rats weighing 190-220 g were kept in a room with a controlled temperature (21-24 ?C) exposed to daily light from 6:00 a.m. to 6:00 p.m. Animals were divided into two control groups (intact and/or sham animals which received i.p. injection of saline) and one experimental group which received phenylephrine at doses of 3, 5, and 8 mg/kg and prazocin at doses of 1, 2, and 3 mg/kg via i.p. route. Then, tail flick test was performed before injection, and 15 and 30 minutes after injection. Data was presented as percentage of analgesic indices and were compared statistically using ANOVA test. It was shown that phenylephrine significantly ($P<0.05$) decreased pain sensitivity in all phases of estrus cycle. This analgesic effect was higher in diestrus and lower in proestrus rats. Prazocin significantly ($P<0.05$) increased pain sensitivity. This hyperalgesic effect was higher in diestrus and lower in proestrus rats. The presented data indicated that pain sensitivity after phenylephrine and prazocin administration was different during estrus cycle. It seems that fluctuations in ovarian hormones is responsible for these differences in pain sensitivity.

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The effect of desmopressin infusion into dorsal raphe nucleus on pain modulation and morphine analgesia in rats tail flick reflex

A Sarihi; B Heshmatian

Abstract

Recent neuroanatomical and behavioral evidence has indicated that vasopressin (VA) increases pain threshold. The dorsal raphe nucleus (DRN) is an important nucleus in pain modulation. Anatomical studies have shown that DRN receives vasopressinergic fibers originating in the hypothalamic paraventricular nucleus. The aim of the present study was to examine the effects of intra-DRN injection of desmopressin (DES) on pain threshold and morphine antinociception in tail flick reflex. DES (0.01 mg/ml) was injected through a single cannula aimed at the DRN for activation of VA receptors of DRN neurons; control group received saline. In experiment 1, rats received intra-DRN injection of DES or saline 10 min before tail flick reflex. Injection of DES into DRN decreased tail flick latency at 45 and 60 min ($P<0.05$, $P<0.01$) but not at 15 and 30 min. In experiment 2, to perform morphine analgesic test, rats were treated with 3mg/kg S. C. morphine 30 min before tail flick. Intra cerebral injection of DES and saline were done in the same way as experiment 1. Infusion of DES into DRN decreased tail flick latency at 15, 30, 45 and 60 min ($P<0.01$, $P<0.01$, $P<0.001$, and $P<0.05$) during morphine analgesia test. It is concluded that VA receptors in the dorsal raphe have an excitatory role in pain modulation and VA can inhibit morphine analgesia in the tail flick reflex.

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The effect of stress and glucocorticoids on modulation of pain in mice: Interaction with activation of voltage dependent Ca2+ channel

AA Vafaei; AA Taherian; H Miladi-Gorgi

Abstract

Previous studies indicated that stress and glucocorticoids have modulatory effects on acute pain. The aim of present study was to determine the interaction between stress and glucocorticoids with activation of voltage dependent Ca2+ channel on modulation of acute pain in mice. Male albino mice (25-30 g) were used for this experiment. Tail flick and hot plate were used for evaluation of analgesic effect of the drug. We used subcutaneous injection of vehicle or dexamethasone (DEX) 30 min before or 1 min swimming stresses 5 min before observation period. Also, we used verapamil (10 mg, i.p.) as a voltage Ca2+ channel blocker 10 min before of DEX and stress. Results indicated that DEX and stress have analgesic effects as compared to control group and verapamil can significantly increase this effect ($P<0.01$). These findings provide further evidence for an important role of stress and glucocorticoids on modulation of pain and probably interaction with these effects and activation of voltage dependent Ca2+ channel.

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Anti-nociceptive effect of cimetidine in mice: the role of ATP-sensitive potassium channels

H Miladi-Gorgi; A Rashidy-Pour ; AA Vafaei

Abstract

Recent studies have shown that intracerebroventricular administration of cimetidine (CIM) induces anti-nociceptive and anti–inflammatory effects in rats. However, the underlying mechanism of CIM effect has not been determined yet. This study was planned to determine the anti-nociceptive effect of CIM (50 mg/kg, i.p.) in male mice (25-30 g, n = 80) using tail flick test. Also, the role of ATP-sensitive potassium channels was assessed using glybenclamide (5 mg/kg, i.p.) and minoxidil (2 mg/kg, i.p.) in CIM anti-nociceptive effect. The results showed that CIM significantly induces anti-nociception in mice ($P<0.05$) and its effect is not dependent on the ATP-sensitive potassium channels. Anti-nociceptive effect of CIM is not related to H2–receptors and K+ channels. However CIM is able to increase brain and plasma morphine levels and can inhibit morphine metabolism.

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The effect of blocking of dorsal and lateral paragigantocellularis muscarinic receptors on pain scores in rats

A Sarkaki; SMT Mansouri; SM Zarei; AV Amari

Abstract

Previous studies have shown that paragigantocellularis nucleus (PGI) neurons contain some neurotransmitters such as GABA, neuropeptides, monoamines, substance P, enkephalin and serotonin. It has been suggested that PGI is a main cholinergic source in the rat's brain stem. There are few evidences on the presence and importance of cholinergic receptors on PGI neurons with regard to nociception. In the present study, 38 NMRI young male rats (200-300 g) were used. They were aneasthetized with ketamine hydrochloride (110 mg/kg, i.p.) and a guide cannula was implanted into dorsal or lateral subdivisions of left PGI of both test and sham-operated animals. Then, 1 microliter of the solution containing 781 micromoles of scopolamine (0.3 mg/ml) was injected into dorsal or lateral (DPGI or LPGI). After the recovery period, acute and chronic pain was evaluated by formalin test (50 ?l of formalin, %2.5 injected into right hind paw subcutaneously) ten minutes after the injection of scopolamine. Sham animals received same volume of normal saline. Results showed that injection of scopolamine in DPGI or LPGI reduces pain score in both acute and chronic phases significantly ($P<0.001$, $P<0.05$ respectively). In addition, injection of scopolamine into LPGI induced longer analgesic effect. These data suggest that PGI neurons contain cholinergic muscarinic receptors that may involve in pain processing and the injection of scopolamine into LPGI has longer effects on the chronic pain.

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The effect of blocking of medial raphe nucleus Ca+2-channels on pain in male rats using formalin test

A Sarkaki; SM Alavian

Abstract

Midbrain periaqueductal gray mater area (PAG) receives some afferents from anterolateral system, frontal cortex, and hypothalamus that inhibit pain through its descending pathway that synapses medial raphe nuclus (mrph) and finally terminates on spinal cord enkephalinergic interneurons in layers II and III. In the present study, the role of Ca+2–channels located on medial raphe nucleus neurons was studied. For this purpose, thirty male NMRI rats weighting 185-240 g were used and a stainless steel guide cannula (0.7 mm in diameter) was implanted into mrph nucleus under stereotaxic surgery (AP = -7.8, ML = 0, DV = 8). Then, one microliter of deltiazem(0.1 mg/kg) was injected into mrph. Five minutes after injection, the pain was evaluated using formalin test (50 µl of 2.5% formalin solution was injected into hind paw, s.c.). Every 15 seconds of each 5-minute blocks was scored by 0-3 scores. Sham- treated animals received the same volume of ACSF into mrph nucleus. These results show that intra mrph nucleus administration of diltiazem could not significantly change pain threshold in male rats.

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The role of ?1-adrenergic antagonists in an experimental model of neuropathy: chronic constriction injury (CCI) and CCI along with saphenoctomy

HA Safakhah; H Manaheji

Abstract

In this study the behavioral effects of sectioning the saphenous nerve at the time of application of the loose ligature and the effect of a selective ?1-adrenergic antagonist for two weeks after tying ligatures was examined. Male Sprague-Dawley rats were used in this study. Animals were divided into six groups: Sham-operated, sciatic nerve ligation (CCI), saphenous nerve section (Saph), CCI + Saph, CCI with IP injection of prazocin (CCI+Pra) and CCI + Saph with IP injection of prazocin (CCI+Saph+Pra). Two weeks after induction of neuropathy, animals tested for thermal allodynia, mechanical allodynia, thermal hyperalgesia, and mechanical hyperalgesia. Animals that underwent CCI all had signs of the neuropathic pain. The group CCI + saphenoctomy showed analgesia except in mechanical allodynia. Injection of prazocin in CCI group relieved thermal allodynia and mechanical hyperalgesia, but had no effect on mechanical allodynia and thermal hyperalgesia. Injection of prazosin to CCI+Saph group only relieved mechanical allodynia as compare to CCI+Saph. Meanwhile, comparison between CCI+Saph+Pra with CCI+Pra showed a significant increase in thermal allodynia and mechanical hyperalgesia and CCI group showed a relief in mechanical allodynia and hyperalgesia and thermal allodynia. It may be hypothesized that the hyperesthesia of the sciatic territory, as induced by ?1-adrenergic antagonists, could mediate saphenous collateral sprouts that invade a partially-denervated territory.

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Comparison of behavioral pain responses in two neuropathic models in rat

GhA Hamidi; H Manaheji

Abstract

To study the neuropathic pain mechanism, various behavioral responses of animals in to different neuropathic models were considered. Adult male Sprague-Dawley rats weighing 230-280 g were used. Anesthesia was initially induced with pentobarbital I.P. (50 mg/kg). These models included (1): chronic constriction injury (CCI) by loose ligation of the sciatic nerve (2) Spared nerve injury (SNI) by a lesion of two of the three terminal branches of the sciatic nerve. The animals were tested for behavioral responses, thermal and mechanical allodynia and hyperalgesia at 1, 2, 3, and 4 weeks after nerve injury. On day 7: mechanical allodynia was significantly increased in CCI group compared with SNI group. On day 14: mechanical and thermal hyperalgesia were not significantly different in CCI group compared with SNI group. On day 21: mechanical and thermal allodynia and hyperalgesia did not significantly differ in CCI and SNI groups. On day 28: a significant difference was seen between mechanical hyperalgesia and heat allodynia between CCI and SNI groups. The results of the present study demonstrated that both neuropathic models, CCI and SNI produce disturbance of sensory nerves after 2 days and reach to a maximum within 13-14 days. The mechanical sensitivity and thermal responsiveness also increased in ipsilateral limb.

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The effect of nifedipine and baclofen on spinal anesthesia induced by local anesthetics

M Sabetkasaei; F Masoudnia

Abstract

The primary mode of action of local anesthetics is through sodium channel and axonal conduction blockade. Local anesthetics have also extensive effects on pre-synaptic calcium channels that must function to stimulate the release of neurotransmitters. Thus, interference with calcium channel conductance may enhance spinal anesthesia with local anesthetics. The present study was designed to investigate the effect of the intrathecal calcium channel blocker, nifedipine, or GABAB agonist, Baclofen, which is a blocker of pre-synaptic calcium channels on the spinal anesthesia induced by lidocaine. Male Sprague-Dawley rats were chronically implanted with lumbar intrathecal catheters. Tail-flick (TF) test was used to assess thermal nociceptive threshold, motor functions were assessed using a modified langerman's scale. Intrathecal lidocaine alone showed the prolongation of (TF) latency, and the increase in motor function scale in a time-and dose-dependent manner. But intrathecal nifedipine (50-200 ?g), or baclofen (10, and 20 ?g) alone demonstrated neither sensory nor motor block. The combination of lidocaine (10, 20, 50, 100, and 200 ?g, i.t.) and nifedipine (50 ?g, i.t.) or baclofen (10 ?g, i.t.) produced more potent and prolonged antinociception and motor block when compared with local anesthetics alone. We interpreted these results to indicate that the intrathecal calcium channel blocker, nifedipine, or GABAB agonist, baclofen, potentiate anesthesia with local anesthetics.

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Prevention of migraine headache attacks: enalapril or valproat sodium

S Babazadeh; A Hoseinzadeh; A Tabatabaei; Sh Sharifzadeh; F Ataei; A Baghaki

Abstract

The pharmacological treatment of migraine may be acute or preventive. Frequent, Severe, complicated and long lasting migraine attacks, more than 2 attacks in month require prophylaxis. Traditional preventive drugs such as ?-Blockers, antidepressants, calcium blockers and anticonvulsants, despite their documented efficacy, have many side effects. This study was conducted to determine the effect of enalapril in prevention of migraine headache attacks. Seventy patients aged 19 to 59, who had migraine headache attacks, two to six times in month for at least one month were studied. They were randomly divided into two groups. One group received 5mg enalapril tablet twice a day (totally 10 mg per day) and the second group received 200 mg sodium valproate tablet twice a day (totally 400 mg per day). Both groups received the drugs for eight weeks. At the end of each month all the patients were asked about the number and the duration of headache attacks, accompanying complaints, and analgesic use during the study period. In enalapril group, total number of migraine headaches reduced to 2-3 times per month after two month totally, 42.8 percent of the enalapril group experienced two attacks and 48.5 percent experienced six attacks per month. In valproate sodium group 20 percent had four attacks and 8.5 percent experienced six attacks per month. The total number of migraine attacks, analgesic use and photophobia were statistically lower in enalapril group ($P<0.001$). The results of this study reveal that enalapril is more effective than valproate sodium in prevention of migraine headache attacks. We suggest that the efficacy of long period prevention with enalapril (more than 2 month) deserves further investigation.

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The effect of fluoxetine on thermal hyperalgesia in STZ-induced diabetic mice: possible involvement of 5-HT1/2 receptors

A Muragundla; C Kanwaljit

Abstract

Diabetic neuropathic pain, an important micro vascular complication in diabetes mellitus, has been recognized as one of the most difficult types of pain to treat. Lack of understanding of etiology involved, inadequate relief, development of tolerance and potential toxicity of classical anti-nociceptive agents warrants the investigation of newer agents to relieve this pain. The aim of the present study was to explore the anti-nociceptive effect and possible mechanism of action of a serotonin reuptake inhibitor, fluoxetine, in streptozotocin (STZ)-induced diabetic mice. Diabetes was induced in mice by intraperitoneal injection of streptozotocin (STZ, 200 mg/kg). After 4 weeks of a single intraperitoneal injection of STZ, diabetic mice were subjected to tail-immersion and hot plate assay. Diabetic mice exhibited a significant hyperalgesia as compared to control mice. When diabetic mice were injected with fluoxetine (10 and 20, but not 5 mg/kg, i.p.) produced anti-nociceptive effect in both tail immersion and hot plate assays. The %MPE produced by fluoxetine (20 mg/kg/i.p) was significantly less in diabetic mice as compared with that in control mice. The antinociceptive effect of fluoxetine (20 mg/kg) in diabetic mice was dose dependently potentiated by pindolol (5 and 10 mg/kg, i.p., a selective 5-HT1A/1B receptor antagonist), attenuated by ritanserin (1 and 2 mg/kg, i.p., a selective 5-HT2A/2C receptor antagonist) and remained unaffected by ondansetron (1 and 2 mg/kg, i.p., a selective 5-HT3 receptor antagonist) in both test systems. These results suggest that fluoxetine-induced antinociception primarily involves serotonin pathway modulation through 5-HT1 and 5-HT2 receptors, but not through 5-HT3 receptors in chronic pain associated with STZ-induced diabetic neuropathic pain. Furthermore, the potentiation of anti-nociceptive effect of fluoxetine by pindolol indicates the usefulness of combination of an antidepressant and a 5-HT1A/1B receptor antagonist in therapy of human diabetic neuropathic pain.

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Intrathecal transplantation of cultured calf chromaffin cells attenuate sensory motor dysfunction in a rat model of neuropathic pain

F Nasirinezhad; J Sagen

Abstract

The potential usefulness of chromaffin cells as a source of neuroactive agents for transplantation in the CNS is based on several promising features, including the diversity of biologically active neurotransmitters, neuropeptides and trophic factors produced by the cells. The purpose of this study was to test the possibility that motor as well as sensory dysfunction is reduced by cultured chromaffin cells. For this reason chromaffin cells were isolated from calf adrenal gland and were kept in DMEM-F12 at 37? C for two weeks. One week before transplantation, peripheral neuropathy was induced by a chronic constriction injury of the sciatic nerve of rats. At the time of transplantation, each rat received 100000-150000 cells. Behavioral tests included radiant heat, paw pinch, and, acetone. Motor function was tested by grasping reflex. The experiments illustrate that chromaffin cells can alleviate sensory and motor dysfunction consequence to peripheral nerve injury. These suggest that adrenal medullary chromaffin cells function as a local long-lasting agent at the spinal segment and further support the potential use of this approach for the treating of chronic pain.

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Rate and severity of depression in patients with Parkinson's disease

NS Ali; IK Taha

Abstract

Depression is common and may occur at any stage in the long history of Parkinson's disease, markedly affecting a patient's threshold to discomfort, but it is particularly difficult to assess before clear clinical features are evident. The rate of depression in patients with Parkinson's disease vary, partly because of differences in the definition of depression, difficulties in distinguishing between the features of depression and those of Parkinson's disease, methods of assessment, and the patient population studied. To measure the rate and severity of depression among patients with Parkinson's disease (PD) and its correlation with motor disability, age, sex, and other variables, 54 patients with PD were matched for age and sex with 52 healthy controls. Depression was diagnosed by using ICD-10 criteria and its severity rated by Beck Depression Inventory, while the motor disability of PD was made according to Hoehn and Yahr scale. Patients with PD were significantly more depressed than the control group (42.59% vs 7.69%, $P<0.001$), and there was a positive correlation between the severity of depression and severity of motor disability ($P<0.05$). In addition, 47% of depressed patients were in the age group 50-59 years, and the rate of depression was nearly equal between males and females. It is concluded that there is a high rate of depression in patients with Parkinson's disease than in the control group, and there exists a positive correlation between the severity of depression and the severity of motor disability.

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Brain complexity increases during the manic episode of bipolar mood disorder type I

B Babadi; B Bahrami; R Seyedsadjadi; M Noroozian

Abstract

Fractal dimension of the electroencephalographic (EEG) signal has been argued to reflect the complexity of the underlying brain processes. To this date, conventional studies of EEG in mood disorders have not been able to distinguish between patients and normal individuals. Here we show that, compared to normal subjects, EEG fractal dimension is significantly augmented in the manic episode of bipolar mood disorder type I (BMD I) with an incremental trend towards the right hemisphere. In addition, EEG power spectrum analysis showed that in the delta bandwidth, signal amplitude increases significantly from left to right hemisphere in patients. It is concluded that during the manic episode of BMD I, asynchronous neuronal activity increases in multiple cell assemblies predominantly located in the right hemisphere. As a psychopathological hypothesis, this conclusion encompasses clinical features of the manic episode, studies of post-traumatic mania and the relevant neuroimaging findings. Implications of our findings in understanding the neurophysiological basis of the converging trends in the pharmacology of epilepsy and mood disorders are also discussed.

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Neuropsychological study of ex-veterans injured by chemical weapons using Bender-Gestalt test

S Momtazi

Abstract

Tens of thousands of Iranians were killed and more were physically and/or mentally injured in Iran-Iraq war (1980-1988). During this war the most extensive use of chemical weapons in recent years by Iraq was reported against Iranian veterans and also Iranian and Iraqi civilians. In this study we aimed at finding probable neuropsychological injuries in victims of chemical weapons comparing other veterans. 27 ex-veterans with history of injury due to exposure to chemical weapons during Iran-Iraq war (1980-1988) were examined by Bender-Gestalt neuropsychological test (group A). We compared this group with two other groups of ex-veterans, a group of 25 people with post-concussion syndrome with normal brain imaging (group B) and another group consisted of 27 veterans with other non specific physical war injuries (group C). We excluded any person with specific brain injury and/or brain imaging problem. The mean age of all groups was 38 and the mean years after injury was19 years. No specific abnormality was found in Bender–Gestalt test results of chemical weapons injured group (group A). There were abnormalities in Post Concussion group (group B) and it was significantly different from non-specific injury group (group C)($P<0.005$). Exposure to chemical weapons did not lead to an abnormal neuropsychological finding in Bender-Gestalt test. Post concussion syndrome can be associated with abnormal finding in that test.

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Perceived afterimage size in depth cue-conflict condition

A Lak

Abstract

In depth cue-conflict conditions, various depth cues could represent different extents of depth. Previous studies have investigated the perceived size of negative afterimage in depth cue-correlated conditions in which different cues introduce almost the same amounts of depth to the visual system. This study examined the perceived size of the afterimage in the human observers in a condition that depth cues of texture gradient, linear perspective and gradient of disparity entered into a conflict with cues of absolute binocular disparity, accommodation and convergence. On a 2-D flat screen, a picture of a room that had two cubes inside was shown to seven observers. They were asked to fixate their gaze on the center of a red circle, which was shown randomly on the front surface of one of these cubes for 50 seconds. Observers then should shift their gaze toward the front surface of the other cube and report perceived size of afterimage by adjusting the size of an adjustable blue ring. The result of this experiment shows that the perceived afterimage size has significant difference with adaptation stimulus size. Note that a control experiment has done and its result confirms the above-mentioned finding. Considering that neural correlates of texture gradient, linear perspective and gradient of disparity are located in higher level visual area than neural correlate of absolute binocular disparity, our results point out that in this cue-conflict condition, the afterimage size is determined mainly by higher level depth cues and therefore, consistent with modified weak fusion theory (MWF), in the depth cue averaging procedure, these higher level cues have more weight than other cues that are processed in lower level visual areas.

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The effects of the rotatory maneuver on ENG results of patients with directional preponderance and a history of peripheral vestibular vertigo

N Rassaian; B Sabetazad; N Sadeghi Ghandehari

Abstract

This study was performed following favorable results from a previous research, which studied the response of acute vestibular patients to the rotatory maneuver, to investigate the effects of the rotatory maneuver on chronic vestibular patients. In an attempt to normalize the patient's directional preponderance (DP), and hence their vestibular symptoms, the study was designed to reduce the imbalance between the vestibular systems on the two sides. By rotating the subjects with a rotating chair towards the opposite direction of dominant side, the dominant side would be inhibited and simultaneously the subordinate side would be stimulated. Five patients with a history of peripheral vestibular vertigo and an increased DP of at least one year's duration, originally confirmed by ENG and rotatory test, were subjected to a rotatory stimulus, the effect of which was evaluated by rotation test. The maneuver was performed 6 times over 4 weeks. The DPs of all patients were compared before the stimulus and 70 minutes after that for the 6 sessions of maneuver. The results indicate a significant difference (P=0.043) between the DP mean values before the stimulation and after that. By minimizing the inequality between the vestibular discharges on the two sides, a reduction in vestibular symptoms is expected.

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The effect of ibotonic acid lesion of the nucleus basalis of Meynert (NBM) on the response of cortical neurons in the rat barrel cortex

F Goshadrou; H Esteky

Abstract

In the present study, the effect of NBM lesion on the temporal characteristics of response integration evoked by multiple whisker stimulations in the barrel cortex of rats was studied. Nucleus basalis of Meynert (NBM) projects to widespread areas of the cortex and provides the major cholinergic input (80%) to the cerebral cortex. In this study we examined the effects of NBM lesion on the responses properties of rat barrel cortical cells male Wistar rats weighing 250-350 g were used. The animals were randomly distributed into two groups: One group received bilateral NBM lesions (5 ?g ibotonic/5ul phosphate-buffered saline solution); rats in the other group were sham-operated. Extracellular single-unit recording and controlled whisker stimuli were used to compare response properties of cells in barrel cortex between the two groups. Two neighboring principal and adjacent whiskers in the same row were deflected alone with varying inter-stimulus intervals. Result showed that response magnitude was significantly lower in NBM-lesioned rats as compared to the sham group ($P<0.05$). Unit discharge to subsequent deflection of adjacent whiskers was reduced in a time-dependent fashion in both groups. But the magnitude of responses significantly reduced at each inter-deflection interval in NBM lesioned group ($P<0.001$). Our results suggest that NBM has an important role in regulating the balance of excitation and inhibition in the barrel cortex and can influence the temporal integration of tactile inputs in the cortex.

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Weber's law orthogonal to the psychometric function

P Pakarian

Abstract

Psychometric function plots the percentage of the correct responses among an entire pool of responses (cumulative probability) in a psychophysical task versus the amount of change in an independent variable. These changes in the independent variable are made with reference to a constant initial value. If this initial value is altered, the psychometric function will change according to Weber's law. In other words, in a task such as the detection of the luminance increment, the psychometric function is stretched to the right for greater initial stimulus intensities and is compressed to left for smaller initial intensities. All of this behavior can be plotted in a 3-D coordinate system. The amount of change in the stimulus intensity, the initial reference intensity, and the above-described probability would be plotted on the X, Y, and Z axes respectively. The formula would be: [Z=1/(1+exp((KY-X)/aKY))]. The intersection of the plot of this formula with any plane parallel to the XY plane would be a line plotting the Weber's law, and that with any plane parallel to the XZ plane, would be a line plotting the psychometric function. That is why these two functions are called orthogonal in this article. K in the above formula is the same as appears in Weber's law for just noticeable difference (JND). The coefficient "a" has an arbitrary value that should be at most equal to 0.34 to set the probability of change detection at 5 percents or less when veridically there is absolutely no change in the stimulus intensity. Further implications have been discussed.

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Postnatal expression of EAAC1 and glutamate receptor subunits in vestibular nuclear neurons responsive to vertical linear acceleration

SK Lai; CH Lai; YS Chan

Abstract

Both glutamate receptors and transporters are known to be important in the postsynaptic regulation of glutamate neurotransmission. However, the maturation profile of glutamate transporter EAAC1 and glutamate receptor subunits (NR1, NR2A and NR2B; and GluR 1-4) in functionally activated saccule-related vestibular nuclear neurons of postnatal rats remains unclear. In the present study, conscious Sprague-Dawley rats (P4 to adult) were subjected to sinusoidal linear acceleration along the vertical plane. Neuronal activation was denoted by the expression of Fos protein. In control experiments, labyrinthectomized animals subjected to stimulation and normal animals that remain stationary showed only sporadically scattered Fos labeled neurons. Functionally activated neurons were also studied for co-localization with NMDA receptor subunits, AMPA receptor subunits or EAAC1. During postnatal development, the proportion of Fos/EAAC1 double-labeled neurons within individual vestibular nuclei progressively decreased with age and constituted about one-third of the Fos-labeled neurons in adult rats. However, comparable proportions of Fos/NMDA or Fos/AMPA double-labeled neurons were observed in each age group. About 70-80% of Fos-labeled neurons in the spinal vestibular nucleus, medial vestibular nucleus and group x subnucleus expressed either NMDA or AMPA receptor subunits. However, in subnucleus y 70% of the total Fos-labeled neurons expressed NMDA while only 10-30% of Fos-labeled neurons expressed AMPA receptor subunits. Triple-immunofluorescence data further showed that some Fos-labeled neurons co-expressed EAAC1 and NMDA or AMPA receptor subunits. Our results suggest that neurons in the different vestibular subnuclei differ in post-synaptic processing of gravity-related vertical spatial information.

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Music therapy and sleep cycle

A Ranjbar; L Mosalanejad; S Shahsavarie

Abstract

Sleep is a basic behavior in humans. There are some research reports on the cyclical patterns of different types of sleep and their relationship to breathing, heart rate, brain waves, and other physical function. More recently, scientific research has begun to show how music therapy interacts with relaxation and sleep cycle. This research is focusing on the following hypotheses: 1. Music could influence physiological variables including blood pressure, heart rate, respiratory rate, EEG measurements, body temperature, as well as biochemical parameters within relaxation and sleep cycles. 2. Music therapy causes hormone secretion that results in relaxation. In this respect, melatonin is a hormone that contributes to the regulation of the human sleep cycle. Therefore, it is to be predicted that increased level of this hormone will cause relaxation. This descriptive cross-sectional study determined the relationship between music therapy, relaxation, and sleep cycle. Based on the obtained results, it can be suggested that light music promotes relaxation and regular sleep up to 75% of its value. This conclusion is especially supported by epidemiological studies.

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Sleep promoting effects of BR-16A: interaction with GABAergic modulators

A Kumar; SK Kulkarni

Abstract

The aim of the present study was to investigate the sleep promoting effect of BR-16A, a polyherbal formulation along with GABAergic drugs. Pentobarbitone-induced hypnosis test was used as an animal model to explore its role in sleep. Pentobarbitone produces quick the sleep latency (onset) and prolongation of total sleep time (duration). Seep latency and total sleep time were used as parameters for the evaluation. BR-16A potentiated the effect of triazolam (0.1 mg/kg, ip) and alprazolam (0.25 mg/kg, ip). Melatonin (5.0 mg/kg, i.p.) and zolpidem (0.5 mg/kg, i.p.) did not produce any significant effect on sleep parameters ($P<0.05$). However alprazolam (0.25 mg/kg, i.p.) potentiated the effect of BR-16A (100 mg/kg, p.o.) only at higher doses. Sleep promoting effect of BR-16A, a polyherbal formulation in combination with GABAergic drugs (triazolam and alprazolam,) suggested that these drugs have common mechanism in sleep promoting effect of pentobarbitone and could be used along with other GABAergic hypnotics for the treatment of insomnia. This may reduce the applied dose of the latter drug(s). BR-16A can also be used for the treatment of sleep and related disorders.

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The effect of hypernatremic status on anesthesia

F Heydarpour

Abstract

Hypernatremia is defined as plasma Na+ concentration above 145 meq/L (often due to absolute body water loss and not to total sodium excess). Nevertheless, when kidney damage is present, as occurs in renal diseases, hepatic cirrhosis and congestive heart failure, total body sodium can be increased. The present study evaluates the relationship between hypernatremia and response to anesthetic drug ketamine. For this purpose, 3 groups of rats, each consisting of 5 male Wistar rats ,weighing 200 ± 20 g, were chosen for the experiment. The control group used Zanjan city water during the study while the other two groups were maintained on 1% and 2% NaCl solutions. After 2 weeks, the response to anesthetic drug, ketamine (125 mg/kg) was studied. Results: The average serum sodium level in the rats of control group was 131 meq/L. For those on 1 and 2 percent solutions it was 148 meq/L and 165 meq/L. Sensitivity to the anesthetic drug, the speed of recovery from different stages of anesthesia and its total time was significantly increased in the test groups in comparison with the control group. This rise was quite conspicuous in those using 2 percent solutions. The mortality rate in the group using 1 percent solution was 20 percent and that of 2 percent solution was 60 percent. When hypernatremia develops, due to dehydration or any other reason, the anesthetic drug dosage should be reduced because hypernatremia increases the sensitivity to anesthesia and lengthens the recovery time from different stags of it. If the dose reduction is not practiced in hypernatremia, the increased mortality and drug side effects will most probably be encountered.

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Retrospective study of skin neurofibromatosis (SN) of twenty years period (1991 -2000) in 4 Iranian pathology centers

SMH Noori Mugahi; M Jamali; B Minaii; AS Abdolghader

Abstract

The present study was designed to assess the frequency and stage of skin neurofibroma in the samples of patients with age, gender and biopsy locations. Data obtained from annual record files of four different pathology centers with considering factors of diagnosis, age, and gender and biopsy location, examined for statistical difference. Each diagnosis has been coded by ICD method. Statistical examination has been done in 4186 samples of skin related to 52907 cases. The results of this experiment showed that SN distributed in 52.9% of male cases and 47.1% of female cases and among all cases, trunk was the most common site of biopsy with 691 cases (24.7%). 632 cases (22.6%) were in face and 438 cases (15.8%) were in head & neck. Three samples (5.3%) were taken from the eyelids, 9 (15.8%) were taken from the faces, 11(19.3%) were taken from the heads and necks, 9 (15.8%) were taken from the trunks, 9 (15.8%) were taken from the upper limbs, and 19 samples (28.1%) were taken from the lower limbs showed neurofibroma. In addition, among all cases, the third decades of life was the most common time of SN appearance with 860 cases (21.7%). 648 cases (16.4%) were in 4th decade & 592 cases (14.9%) were in 5th decade. These results may indicate that we should work more about (SN) and its effects on our life story.

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Is a negative cranial CT-scan adequate to support a diagnosis of pseudotumor cerebri

Z Adwan

Abstract

In a patient with features indicating the presence of pseudotumor cerebri, a negative cranial CT-scan is not adequate to rule out less benign pathology and an MRI of brain should be performed. In this study, a 10-year old boy with daily headaches for one month and diplopia for one week was found to have a partial abducens palsy and bilateral papilledema; otherwise, his examination was normal. A cranial CT-scan was normal. Lumbar puncture showed a markedly elevated opening pressure of greater than 550 mmHg in CSF. He was then begun on medical therapy with acetazolamide for presumed pseudotumor cerebri. MRI of brain, done several days later because of continuing symptoms, very expectedly showed multiple white matter lesions. He was placed on high dose IV steroids but his symptoms persisted. Because of his failure to improve and the uncertainty of the nature of his white matter lesions, he had a brain biopsy. The biopsy disclosed a multicentric cerebral oligodendroglioma. It was found out that in a patient with features indicating the presence of pseudotumor cerebri, a negative cranial CT-scan is not adequate to rule out less benign pathology and an MRI of brain should be performed. A revised definition of pseudotumor cerebri could better include "normal MRI of brain" rather than more general supportive finding of "normal neuroimaging".

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Acute nicotine treatment accelerates photochemically induced platelet aggregation in cerebral arterioles of mice: an in vivo study

S Singh; MA Fahim

Abstract

When tobacco is smoked, chewed or snuffed, nicotine is absorbed by the lungs or mucous membrane and quickly moved into the bloodstream, where it is circulated throughout the brain. In fact nicotine is highly dangerous to be consumed in any form. The present study was conducted to know the adverse effects of nicotine on the platelet aggregation in cerebral microvessels of mice. Male mice of average weight 33 g were injected with Saline (control) or Nicotine (1 mg/kg, 0.1 ml/10 g) one hour before the experiment. Animals were anaesthetized and trachea was intubated. Craniotomy was performed and a window was opened on the skull. Layer of dura was removed. Brain surface microvessels were exposed and animal was placed on the microscope stage. Microscope was connected to a monitor and VCR to record all events. Exposed brain surface was continuously irrigated with ACSF solution. After the body temp was maintained at 37°C, Sodium fluorescein (2%, 0.1 ml/10 g) was injected i.v. through tail injection. High intensity mercury light was switched on to induce photochemically induced platelet aggregation. Appearance of the 1st platelet aggregation and total blood flow stop were timed in seconds. Our results showed that in the animals treated with nicotine, venule did not show any alteration in the platelet aggregation timings in comparison to the controls. But in arterioles platelet aggregation timings were significantly accelerated ($P<0.001$) in nicotine treated animals. [(Control: venule 1st aggregation 22.87 ± 1.45, flow stop 161.25 ± 19.9; Arteriole: 1st aggregation 104.37 ± 19.64, flow stop 167.25 ± 23.54; Nicotine: venule 1st aggregation 23.57 ± 4.79, flow stop 142.15 ± 25.9 Arteriole: 1st aggregation 50.0 ± 13.08, flow stop 100.71 ± 33.70)]. Data shows the adverse influence of nicotine on the susceptibility to thrombosis in the arterioles, in vivo. The enhanced initiation of thrombosis activity may be attributed to the facilitated damage to the arteriolar endothelium caused by nicotine.

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Temperature-dependent model of human cardiac sodium channel

B Keshavarzi; H Mobasheri; MJ Abdekhodaie; D Bastani

Abstract

Cardiac sodium channels are integral membrane proteins whose structure is not known at atomic level yet and their molecular kinetics is still being studied through mathematical modeling. This study has focused on adapting an existing model of cardiac Na channel to analyze molecular kinetics of channels at 9-37°C. Irvine et al developed a Markov model for Na channel using Neuronal Network Model as a framework. They produced state diagram for channel with 5 close, 2 open, and 5 close-inactivated and one inactivated states. Transitions are expressed as ?H and ?S and an effective valence (z) terms. Almost all of rate constants are voltage and temperature dependent. The model fails to reproduce kinetics at temperature higher than 25°C. A practical approach would be to scale up each rate constant by its own Q10. As temperature is increased, time constants and recovery rate decrease and increase respectively. First latencies decrease with temperature at (–100)-20 mV. Open probability of channels does not follow temperature suggesting that at higher temperature a larger fraction of channels are inactivated before they reach open state. This study exhibits a reasonable model for single channel ionic current and its recovery from inactivation for a voltage range of (–100)-20 mV at 9-37°C. Our approach may lead to more detailed molecular kinetics of Na channels resulting in better understanding of arrhythmias and Na channel based nervous system malfunction.

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Immunohistochemistric study of the intra-cardiac ganglia of the pig heart

A Shams; I Nour Mohammadi; F Aghaee; D Hopkins; S Nazarpour; A Siadati

Abstract

Heart contains a variety of neurons known to influence cardiac functions. Little is known about the neurochemistry of these neurons. In this study, neurochemistry of intra cardiac neurons of the intrinsic cardiac ganglia was investigated. The technique used allows studying the morphology of the ganglionated nerve plexus found within the atria and great cardiac vessels as well as of individual neurons. The epicardial surface of pig hearts obtained postmortem using immunohistochemically staining in conjunction with confocal microscopy neurons in the cardiac intrinsic ganglia immunoreactive for PGP 9.5 identified in the epicardial fat pad around the aortic artery and pulmonary vein. Immunocytochemistry with the marker Protein gene product PGP9.5 was used to study the presence of neurons within clusters of intrinsic cardiac ganglia. Briefly, Tissues were fixed with neutral buffered formalin. Then, they were dissected, dehydrated, cleared, washed and incubated overnight at room temperature with PGP 9.5 (1/1000 in phosphate buffered saline with 0.2 Triton X-100). The slides were washed in PBS and incubated with a mixture of Cy3 labeled Donkey anti-rabbit antibody IgG for 2 hour at room temperature. The slides were evaluated and photographed with a confocal microscope. The results of this study indicate a moderate level of chemical diversity within the intra cardiac neurons of the pig such chemical diversity may reflect functional specialization of neurons in the intra cardiac ganglia. A distinct population of ganglia was seem to arise from in the endocardial plexus of the atria and was predominantly protein gene product 9.5 positive. The small black depressions on the neurons represented satellite cells. Neurons of the intrinsic cardiac ganglia are distributed more widely than has been described in experimented animals. Intra cardiac ganglia formed a complex plexus around the origin of pulmonary veins and were connected by nerve fibers. All neurons included in intrinsic cardiac ganglia contained immunoreactivity to PGP 9.5. Neurons express PGP 9.5 immunoreactivity, suggesting that catecolamine may be present in autonomic nerves in the heart.

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Role of pentoxyphiline in stroke prevention

K Gharagozli; M Rafi; F Fallahi; S Abadian; B Azimi; B Salarian

Abstract

Anti-platelet agents such as aspirin and dipyridamole are main factors to prevent stroke in high-risk people. Pentoxyphiline has been reported as a red blood cell aggregation antagonist to prevent stroke. We evaluated the role of pento-xyphiline as an adjunct therapy to decrease risk of stroke in patients who were under treatment with aspirin or dipyridamole. A 5-years open labeled clinical trial was performed on 4465 patients with a history of thrombotic cerebrovascular accident. The patients divided into 2 groups. Group 1 (a total of 2230 patients, 1032 females and 1198 males) were under treatment by aspirin alone, and group 2 (a total of 2235 patients, 991 females and 1244 males) received aspirin + penthoxyphiline 400 mg/day. Subjects were followed prospectively for 5 years. The primary assessment was the proportion of patients who suffered another stroke. 193 patients in aspirin group and 213 patients out of the other group were excluded because of bleeding complication. After 5 years, 45.6% of the patients who received aspirin alone remained stroke-free as compared to 60.5% of those given aspirin + pentoxyphiline ($P<0.007$). In high risk patients for atherosclerotic stroke, combination treatment of aspirin and pentoxyphiline have better prognostic outcome.

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Lithium changes due to biorhythms and changing activity of epiphysis

IA Omarov; RA Huseinova

Abstract

Considering the physiological role of epiphysis in functional activity and defense reactions of the body, this study was carried out to evaluate changes of lithium level due to biorhythms of epiphysis. For this purpose, albino mice (6-month old) were used. The animals were kept in light (resulting in inhibition of the activity of epiphysis) and dark (which increases the activity of epiphysis) environments (March, April, and May months). After a week, the dynamics of the changes of the lithium level in their blood and in plasma were evaluated. The level of lithium was measured according to Voynar method and expressed as mkq/ml. The results showed that during these periods (March, April, May), the inhibition of the epiphysis due to light factor could increase the level of lithium in blood and plasma. With prolongation of the light regime, this difference increases. This may continue up to 14 p.m. In contrast, keeping animals in darkness led to different results. The observed effects may be due to changes of metabolism following alteration of lithium level. These results may be useful to define the role of the epiphysis in the microelement metabolism.

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EEGs and autonomic changes during and after acupuncture stimulation

Sakai; Umeno; Tabuchi; Hori; S Ono; H Nishijo

Abstract

A "Qi" feeling (a kind of comfortable sense) during and after acupuncture is one of the important determinants to decide quantity (trial number) of acupuncture stimulation. It has been reported that various autonomic responses were evoked when patients felt "Qi" and when acupuncture stimulation had significant effects on EEGs. However, quantitative relationships among acupuncture stimulation, EEGs, and autonomic responses remain unclear. In the present study, autonomic functions [ECGs, heart rate (HR), blood pressure (BP)] and EEGs were analyzed during and after acupuncture stimulation. Acupuncture stimulation was given to the neck of the subjects. The subjects were required to push a button when they felt "Qi". The results indicated that acupuncture significantly decreased HR, and increased systolic BP (SBP). Spectral analysis indicated that acupuncture significantly decreased low frequency components (LF) of HR variability (HRV) and SBP variability (SBPV), and also significantly reduced ratio of LF to high frequency component (HF) of HRV (indices of sympathetic activity). Furthermore, there were significant negative correlation between changes in LF/HF ratio of HRV and number of acupuncture trials, and significant positive correlation between HF of HRV (index of parasympathetic activity) and number of acupuncture trials. Analyses of EEGs data indicated increases in all EEG bands during acupuncture. Furthermore, HF of HRV was positively correlated with power of all EEG bands, while LF of SBP and LF/HF of HRV were negatively correlated with these spectral data. These results suggest that physiological effects of acupuncture are mediated through the central nervous system.

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Endosulfan induced expression of early response genes/ oxidative injury in PC12 cell line

K Seth; AK Agrawal; C Sinha; S Shukla; RK Chaturvedi; Y Shukla; PK Seth

Abstract

Recently use of endosulfan has aroused a great concern among clinical and basic neuroscientists as besides adult population even the children residing in some areas of touristic state of Kerala, India which are under constant spraying of endosulfan had shown symptoms of CNS dysfunctioning including cerebral palsy, retardation of mental and physical growth, epilepsy and various congenital anomalies. In spite of several studies demonstrating neurotoxic potential of endosulfan, very little is known about the mechanism of its neurotoxicity. In the present study an attempt has been made to delineate the mechanism by which endosulfan a chlorinated hydrocarbon insecticide exerts its neurotoxic potential at cellular level using PC12, a dopaminergic cell line. Cells exposed to endosulfan (0-100 µM) for 1, 24 and 48 h showed significant loss in cell viability /mitochondrial functioning in a dose- and time- dependent manner following MTT assay and LDH leakage assessment. An oxidative stress mediated neurotoxicity was evident where endosulfan (1uM, a non cytotoxic concentration) exposure is accompanied by increase in lipid peroxidation and lowering in antioxidant defense as malonaldehyde (MDA), a product of lipid peroxidation was enhanced by 49 and 62 % following 24 and 48 hours endosulfan exposure. A decrease in levels of GSH (41 & 68 %) along with catalase (39 and 52%) and SOD (26 & 44 %) activities was also evident 24 & 48 h post exposure. Induced expression of early response gene protein (c-Fos, $P<0.001$; c-Jun $P<0.05$ and GAP-43, $P<0.05$) during 1-24 h endosulfan (1 ?M) exposure was accompanied by PKC activation. The present results suggest possible role of PKC/early response gene mediated oxidative injury and mitochondrial dysfunction in endosulfan neurotoxicity.

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Target-controlled infusions of remifentanil and propofol during laparoscopic cholecystectomy

A Agzamov; AM Al Qattan; AY Dubikaitis

Abstract

We have had this study to evaluate the clinical profile of target-controlled infusion-based anesthesia using remifentanil and propofol. 116 ASA I-II patients undergoing elective laparoscopic cholecystectomy (LCH) were enrolled. TCI Remifentanil was set at 8 micrograms. L - 1 as target and TCI propofol at 4 mcg/ml throughout the whole procedure. The hemodynamics during induction of anesthesia and recovery profiles were recorded. Arterial blood samples for analysis of remifentanil were taken 15 min after infusion, 20 min after infusion and at time of emergence. After induction of anesthesia, systolic blood pressure (SBP) decreased from (140 ± 24) mm Hg to (101 ± 16) mm Hg ($P < 0.05$), mean blood pressure (MBP) decreased from (103 ± 14) mm Hg to (75 ± 11) mm Hg ($P < 0.05$) and heart rate (HR) decreased from (78 ± 14) beats.min-1 to (65 ± 10) beats.min-1 ($P > 0.05$). SBP, MBP and HR remained stable after intubations for 3 min. No patient showed haemodynamic stress to tracheal intubations. Times from stopping administration of anesthetics until full spontaneous respiration, eye opening, tracheal extubation, orientation and discharging from the postanesthetic care unit (PACU) were (10±6), (7±4), (11±6), (12±5) and (18±7) min respectively. Measured drug values of remifentanil were (4.5±7.5) microgram. L-1, (6.4±11.4) microgram. L-1, (1.1±7.6) and microgram. L-1 respectively. Remifentanil/propofol TCI-based anesthesia achieved the optimal hemodynamic stability during anesthesia induction and maintenance, and better recovery profile from anesthesia. Measured drug values of remifentanil showed a considerable inter individual variation and more lower than the set target.

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GABA-mediated membrane oscillations as coincidence detectors for enhancing synaptic efficacy in the developing hippocampus

E Cherubini

Abstract

Spontaneously occurring neuronal oscillations constitute a hallmark of developmental networks. They have been observed in the retina, neocortex, hippocampus, thalamus and spinal cord. In the immature hippocampus the so-called 'giant depolarizing potentials' (GDPs) are network-driven membrane oscillations characterized by recurrent membrane depolarization with superimposed fast action potentials. Usually, they last hundreds of ms and are separated by intervals of several seconds. GDPs depend on the synergistic action of GABA and glutamate acting on GABAA and (RS)-a-amino-3-hydroxy-5-methyl-4-isoxadepropionate (AMPA) receptors, respectively. Early in postnatal life, GABA depolarizes and excites the postsynaptic cells due to high intracellular [Cl-] which results mainly from the unbalance of two Cl- co-transporter systems, the NKCCl and KCC2 that enhance and lower intracellular [Cl-], respectively.

In accord with the Hebb postulate on activity-dependent synaptic strengthening, here the hypothesis has been tested that GDPs may act as coincident detector signals between pre and postsynaptic activity. This assumption has been verified at poorly developed mossy fiber (MF)-CA3 connections. These synapses are particularly interesting because they can release GABA in addition to glutamate.

We found that during the first postnatal week correlated pre (MF) and postsynaptic (GDPs) activity persistently enhances synaptic strength at MF-CA3 connections. This effect was usually restricted to the activated synapse. When the interval between GDPs and MF stimulation was increased, the potentiating effect progressively declined and disappeared. The potentiation depended on the activation of voltage-dependent calcium channels and calcium flux. Moreover, it was clear from our experiments that at this developmental stage GABA is the main neurotransmitter released from the MF. This is consistent with the sequential expression of functional GABA and glutamatergic synapses found in the hippocampus at early developmental stages.

Synchronous membrane oscillations may therefore contribute to the refinement of neuronal connectivity before the establishment of the adult neuronal circuit. Later in development when the degree of functional connections is sufficiently high, GDPs would be replaced by more subtle types of signal synchronization such as theta or gamma activity characteristic of the adult network.