SYNAPTIC PLASTICITY IN ADDITION YAN DONG AND R. SUZANNE ZUKIN pdf

1: Dopamine triggers Heterosynaptic Plasticity - Europe PMC Article - Europe PMC

Gompers A, Hwang J-Y, Monday HR, Buxbaum A, Yan J, Sawicka K, Castillo PE, Singer RH, Zukin RS The memory protein CPEB3 reduces synaptic incorporation of Ca2+-permeable AMPARs and loss of anti-Hebbian LTP at synapses onto inhibitory interneurons in Fragile X mice.

Abstract As a classic neuromodulator, dopamine has long been thought to modulate, rather than trigger, synaptic plasticity. In contrast, our present results demonstrate that within the parallel projections of dopaminergic and GABAergic terminals from the ventral tegmental area VTA to nucleus accumbens core NAcCo , action potential-activated release of dopamine heterosynaptically triggers LTD at GABAergic synapses, which is likely mediated by activating presynaptically-located dopamine D1 class receptors and expressed by inhibiting presynaptic release of GABA. Moreover, this dopamine-mediated heterosynaptic LTD is abolished after withdrawal from cocaine exposure. These results suggest that action potential-dependent dopamine release triggers very different cellular consequences from those induced by volume release or pharmacological manipulation. This dopamine-mediated LTD allows a flexible output of NAcCo neurons, whereas disruption of this LTD may contribute to the rigid emotional and motivational state observed in addicts during cocaine withdrawal. We further demonstrated that this LTD was triggered by heterosynaptic dopamine and was abolished after withdrawal from cocaine exposure. By pharmacological manipulations of dopamine receptors, extensive prior results firmly establish that dopamine is a modulator of plasticity. Our current results indicate that action potential-driven dopamine release is also a trigger of plasticity. This form of LTD relieves NAc neurons from inhibition upon receipt of phasic input from dopamine neurons that has been shown to be triggered by reward or reward-associated cues Fiorillo et al. Disruption of this LTD may contribute to the rigidity in reward-associated responses that occur during cocaine withdrawal. AAV-flexed-ChR2R expression was induced specifically in Cre recombinase expressing neurons, where the inverted expression cassette was flipped Atasoy et al. In this study, flexed virus was used in Gad-cre or TH-cre mouse lines. Intra-VTA viral injections were performed when animals were at the age of postnatal 26â€”34 days. After recovery, rats received i. Rats were placed back to the homecage for withdrawal. NAc slice preparations and electrophysiology As described previously Ishikawa et al. Slices were incubated in artificial cerebrospinal fluid aCSF containing in mM: The optical stimulation was given with 0. All procedures were performed by strictly following the standard procedures approved by the IACUC of listed institutions. Statistical significance was assessed using either one- or two-factor ANOVA with Bonferroni posttests or two-tailed t-test. One to four cells were recorded from each animal. Cell-based statistics were performed for all results. Three weeks later, we obtained horizontal brain slices, within which the VTA exhibited yellow fluorescent signals, indicating the expression of ChR2 Fig. By laser light-mediated activation of ChR2 0.

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2: - NLM Catalog Result

TY - CHAP. T1 - Synaptic plasticity in addiction. AU - Dong,Yan. AU - Zukin,R. Suzanne. PY - Y1 - N2 - Drug addiction, defined as compulsive drug use despite serious negative consequences, has been one of the major social problems facing modern societies.

We are testing a new system for linking publications to authors. If you notice any innacuracies, please sign in and mark papers as correct or incorrect matches. If you identify any major omissions or other inaccuracies in the publication list, please let us know. The emerging field of epigenetics in neurodegeneration and neuroprotection. Activation of autophagy rescues synaptic and cognitive deficits in fragile X mice. REST, a master transcriptional regulator in neurodegenerative disease. Current Opinion in Neurobiology. Aberrant Rac1-cofilin signaling mediates defects in dendritic spines, synaptic function, and sensory perception in fragile X syndrome. Global ischemia induces lysosomal-mediated degradation of mTOR and activation of autophagy in hippocampal neurons destined to die. Cell Death and Differentiation. The Journal of Neuroscience: Zukin RS, et al. PDE-4 inhibition rescues aberrant synaptic plasticity in Drosophila and mouse models of fragile X syndrome. Estradiol pretreatment ameliorates impaired synaptic plasticity at synapses of insulted CA1 neurons after transient global ischemia. The gene silencing transcription factor REST represses miR expression in hippocampal neurons destined to die. Journal of Molecular Biology. Casein kinase 1 suppresses activation of REST in insulted hippocampal neurons and halts ischemia-induced neuronal death. Survivin Is a transcriptional target of STAT3 critical to estradiol neuroprotection in global ischemia. Dysregulation of synaptic plasticity precedes appearance of morphological defects in a Pten conditional knockout mouse model of autism. Epigenetic mechanisms in stroke and epilepsy. Official Publication of the American College of Neuropsychopharmacology. Gintonin, a ginseng-derived novel ingredient, evokes long-term potentiation through N-methyl-D-aspartic acid receptor activation: Bidirectional control of mRNA translation and synaptic plasticity by the cytoplasmic polyadenylation complex. Estradiol attenuates ischemia-induced death of hippocampal neurons and enhances synaptic transmission in aged, long-term hormone-deprived female rats. Effects of protopanaxatriol-ginsenoside metabolites on rat N-methyl-d-aspartic Acid receptor-mediated ion currents. Repressor element-1 silencing transcription factor REST -dependent epigenetic remodeling is critical to ischemia-induced neuronal death. N-terminally cleaved Bcl-xL mediates ischemia-induced neuronal death. Genes, Brain, and Behavior. Dysregulation of mTOR signaling in neuropsychiatric disorders: Synaptic plasticity in addiction Synaptic Plasticity: Cell Biology, Regulation and Role in Disease. A silent synapse-based mechanism for cocaine-induced locomotor sensitization. Serotonin mediates cross-modal reorganization of cortical circuits. Neuroprotective actions of estradiol and novel estrogen analogs in ischemia: Pharmacological reversal of synaptic plasticity deficits in the mouse model of fragile X syndrome by group II mGluR antagonist or lithium treatment. Eradicating the mediators of neuronal death with a fine-tooth comb. Spatially restricting gene expression by local translation at synapses. Dysregulation of mTOR signaling in fragile X syndrome. Acute administration of non-classical estrogen receptor agonists attenuates ischemia-induced hippocampal neuron loss in middle-aged female rats. A single fear-inducing stimulus induces a transcription-dependent switch in synaptic AMPAR phenotype. Disease Encyclopedia of Neuroscience. Oestradiol and insulin-like growth factor-1 reduce cell loss after global ischaemia in middle-aged female rats. In vivo cocaine experience generates silent synapses. Estradiol rescues neurons from global ischemia-induced cell death: Proteomic analysis reveals novel binding partners of metabotropic glutamate receptor 1. Chronic estradiol treatment increases CA1 cell survival but does not improve visual or spatial recognition memory after global ischemia in middle-aged female rats. Ischemic preconditioning blocks BAD translocation, Bcl-xL cleavage, and large channel activity in mitochondria of postischemic hippocampal neurons. Epigenetic targets of HDAC inhibition in neurodegenerative and psychiatric disorders. Current Opinion in Pharmacology. CREB modulates the functional output of nucleus

accumbens neurons: The Journal of Biological Chemistry. NMDA receptor trafficking in synaptic plasticity and neuropsychiatric disorders. Failure of estradiol to ameliorate global ischemia-induced CA1 sector injury in middle-aged female gerbils. Ischemic insults promote epigenetic reprogramming of mu opioid receptor expression in hippocampal neurons. Effects of estradiol on cognition and hippocampal pathology after lateral fluid percussion brain injury in female rats. RNA trafficking and local protein synthesis in dendrites: Zinc-dependent multi-conductance channel activity in mitochondria isolated from ischemic brain. Ischemic insults direct glutamate receptor subunit 2-lacking AMPA receptors to synaptic sites. Acute and chronic estradiol treatments reduce memory deficits induced by transient global ischemia in female rats. Blockade of calcium-permeable AMPA receptors protects hippocampal neurons against global ischemia-induced death. Estrogen can act via estrogen receptor alpha and beta to protect hippocampal neurons against global ischemia-induced cell death. Induction of Dickkopf-1, a negative modulator of the Wnt pathway, is required for the development of ischemic neuronal death. Postsynaptic density protein regulates NMDA channel gating and surface expression. Electrical coupling and neuronal synchronization in the Mammalian brain. Pathophysiology, Diagnosis, and Management. Ischemic insults derepress the gene silencer REST in neurons destined to die. NMDA-receptor trafficking and targeting: Estrogen protects against global ischemia-induced neuronal death and prevents activation of apoptotic signaling cascades in the hippocampal CA1. Ischemic preconditioning acts upstream of GluR2 down-regulation to afford neuroprotection in the hippocampal CA1. Global ischemia-induced increases in the gap junctional proteins connexin 32 Cx32 and Cx36 in hippocampus and enhanced vulnerability of Cx32 knock-out mice. Activation of metabotropic glutamate receptor 1 accelerates NMDA receptor trafficking. Insulin promotes rapid delivery of N-methyl-D- aspartate receptors to the cell surface by exocytosis. Remodeling of alpha-aminohydroxymethylisoxazole-propionic acid receptor subunit composition in hippocampal neurons after global ischemia. Status epilepticus decreases glutamate receptor 2 mRNA and protein expression in hippocampal pyramidal cells before neuronal death. Protein kinase C potentiation of N-methyl-D-aspartate receptor activity is not mediated by phosphorylation of N-methyl-D-aspartate receptor subunits. AMPA receptor protein expression and function in astrocytes cultured from hippocampus. Journal of Neuroscience Research.

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3: R. Suzanne Zukin, Ph.D. â€“ Publications â€” Albert Einstein College of Medicine

Note: Citations are based on reference standards. However, formatting rules can vary widely between applications and fields of interest or study. The specific requirements or preferences of your reviewing publisher, classroom teacher, institution or organization should be applied.

Stefanik, Mike Milovanovic, Yanhua H. Epub Dec 8. Opposing mechanisms mediate morphine- and cocaine-induced generation of silent synapses. Re-silencing of silent synapses unmasks anti-relapse effects of environmental enrichment. Sleep Regulates Incubation of Cocaine Craving. Silent Synapses Speak Up: Epub Mar Dong Y, and White FJ. Dopamine D1-class receptors selectively modulate a slowly inactivating potassium current in rat prefrontal cortex pyramidal neurons. Dopamine modulates inwardly rectifying potassium currents in medial prefrontal cortex pyramidal neurons. Adaptations in Potassium Currents. Drugs of abuse and stress trigger a common synaptic adaptation in dopamine neurons. Cocaine-induced potentiation of synaptic strength in dopamine neurons: A schizophrenia-related sensorimotor deficit links alpha 3-containing GABAA receptors to a dopamine hyperfunction. CREB modulates excitability of nucleus accumbens neurons. Galanin and GALP differentially modulate the neuronal activity in rat hypothalamic arcuate nucleus neurons. Dong Y, Zukin RS. Synaptic Plasticity and Addiction. A critical role of synaptic NMDA receptors. GABA B receptors are required for galanin modulation of membrane properties of neurons in the arcuate nucleus of rats. Glucocorticoid signaling mediates stress-induced synaptic adapatation in midbrain dopamine neurons. Selective Receptor Agonists for the Treatment of Obesity. Reducing hippocampal cell proliferation in the adult rat does not prevent the acquisition of cocaine-induced conditioned place preference. Lee BR, Dong Y. Cocaine-induced metaplasticity in Nucleus Accumbens: Silent synapse and beyond. Positive effective vocalizations during cocaine and sucrose self-administration: A model for spontaneous drug desire in rats. CB1-expressing neurons in the nucleus accumbens. Presynaptic enhancement of glutamatergic transmission within the nucleus accumbens following cocaine exposure. Dopamine triggers Heterosynaptic Plasticity. Exposure to cocaine regulates inhibitory synaptic transmission from the ventral tegmental area to nucleus accumbens. Differential roles of PSD isoforms in regulating synaptic transmission. Maturation of silent synapses in amygdala-accumbens projection contributes to incubation of cocaine craving. An unusual suspect in cocaine addiction. Dong Y, Nestler EJ. Increased excitability of lateral habenula neurons in adolescent rats following cocaine self-administration. Neumann PA, Dong Y. Molecular and Cellular Mechanisms of Addiction. Encyclopedia of Addictive Behaviors.

SYNAPTIC PLASTICITY IN ADDITION YAN DONG AND R. SUZANNE ZUKIN pdf

4: R. Suzanne Zukin - Publications

(Yan Dong, R. Suzanne Zukin, Program in Neuroscience, Department of Vet and Comparative Anatomy, Pharmacology and Physiology, Washington State University, Pullman, Washington, USA, and others) Involvement of Zinc via Crosstalk with Calcium in Synaptic Plasticity and Neurodegeneration in the Hippocampus.*

Regulation of synaptic function and plasticity in response to external cues including neuronal insults, maternal deprivation, and stress, via epigenetic mechanisms. Altered signaling at the synapse in mouse models of autism. A particular focus centers on epigenetic mechanisms that underlie neuronal death in animal models of stroke. We found that REST is activated in response to neuronal insults such as global ischemia and orchestrates epigenetic remodeling of synaptic AMPA receptors. We found that REST is causally related to ischemia-induced neuronal death. Using an epigenome-wide search, we identified novel REST targets in the context of ischemia. Neuron-specific microRNAs silence networks of non-neuronal genes in neurons. We expect this research to have a major impact on amelioration of neuronal death and impaired cognition associated with stroke and AD. To perform this research, we use genetics, electrophysiology, behavior and delivery of engineered cDNA and shRNA constructs directly into the brains of living animals via the lentivirus expression system. We use clinically relevant models of stroke and global ischemia, and transgenic mice in which REST can be conditionally knocked out. In another direction, we are studying epigenetic mechanisms that regulate synaptic proteins during postnatal brain development. We found that REST is causally related to the developmental switch. Importantly, adverse experience early in life in the form of maternal deprivation disrupts activation of REST and acquisition of the mature NMDA receptor phenotype. These findings document a role for REST in experience-dependent fine-tuning of genes involved in synaptic plasticity. We recently found that REST abundance in neurons is controlled at the level of protein stability and identified casein kinase 1 as an upstream signal that negatively regulates REST in neurons. Findings from this research are expected to enhance our understanding of NMDAR function as it pertains to memory, synaptic stabilization, and cognitive information flow and how adverse experience acts via epigenetics to impair brain development. We are extending studies to mouse models of schizophrenia. A rotation project we are offering involves rescue of brain development in maternally-deprived pups by communal nesting of female mice and their litters. In a third direction, we are studying mouse models of autism. Fragile X Syndrome is the most common inherited cause of intellectual disabilities and a leading cause of autism. New directions include a focus on mTORC2 and its role in aberrant spine structure and number. We showed that the Rho GTPase Rac1, a downstream target of mTORC2 and its downstream target LIMK are also elevated and causally related to elevated phosphorylation and blunted activity of the actin depolymerizing factor cofilin, a major determinant of spine morphology. We rescued impaired cofilin signaling and spine abnormalities pharmacologically by administration of a small molecule inhibitor of PAK1 and genetically by viral delivery of a constitutively active cofilin mutant cofilin-S3A into the somatosensory cortex of neonatal Fragile X mice. These findings suggest a causal relationship between enhanced Rac1-cofilin signaling, synaptic defects, and impaired sensory processing in FXS and uncover a previously unappreciated role for impaired Rac1-cofilin signaling in the aberrant spine morphology and spine density associated with FXS. A rotation project could examine the impact of dysregulated cofilin signaling on contextual learning and cognition and a role for mTOR complex 2 in regulation of spine structure and cognition. The switch in synaptic AMPAR phenotype could potentially alter the inhibitory to excitatory balance, an important theme in autism spectrum disorders. New directions for rotation students include the impact of CPEB3 on synaptic plasticity at inhibitory synapses and autism-relevant behaviors. Nat Neurosci, in review, Cell Death Differ Estradiol pretreatment ameliorates impaired synaptic plasticity at synapses of insulted CA1 neurons after transient global ischemia. J Mol Biol Casein kinase 1 suppresses activation of REST in insulted hippocampal neurons and halts ischemia-induced neuronal death. Survivin is a transcriptional target of STAT3 critical to estradiol neuroprotection in global

ischemia. Dysregulation of synaptic plasticity precedes morphological defects in a Pten conditional knockout mouse model of autism. Bidirectional control of mRNA translation and synaptic plasticity by the cytoplasmic polyadenylation complex. Repressor element-1 silencing transcription factor REST -dependent epigenetic remodeling is critical to ischemia-induced neuronal death. Genes Brain Behav N-terminally cleaved Bcl-x L mediates ischemia-induced neuronal death. A single fear-inducing stimulus induces a transcription-dependent switch in synaptic AMPA receptor phenotype. In vivo cocaine experience generates nascent synapses. NMDA receptor trafficking in synaptic plasticity and neuropsychiatric disorders. Nat Rev Neurosci 8:

5: Synaptic plasticity in addiction â€" Albert Einstein College of Medicine

Book Description: Cognitive science is most simply defined as the scientific study either of mind or of intelligence. It is an interdisciplinary study drawing from relevant fields including psychology, philosophy, neuroscience, linguistics, anthropology, computer science, biology, and physics.

6: R. Suzanne Zukin, Ph.D. | Albert Einstein College of Medicine

Collectively, our results show that in vivo experience can generate silent synapses de novo, and these newly-generated silent synapses may transiently provide highly-efficient plasticity substrates (Marie et al.,) for subsequent experience-dependent, long-lasting synaptic plasticity.

7: Recent Publications - YAN DONG LABORATORY

Studies over the past decade have enunciated silent synapses as prominent cellular substrates for synaptic plasticity in the developing brain. However, little is known about whether silent synapses can be generated postdevelopmentally.

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