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Chapter 1 : EP4: Prostaglandin E receptor 4

Prostaglandin-mediated signaling may have increased activity in schizophrenia (Smesny The main assumptions of David Horrobin's MPC hypothesis of.

Altered gyrification in schizophrenia and its relation to other morphometric markers. Schizophrenia Research, in press. Potential Brain Age Reversal after Pregnancy: Younger Brains at Weeks Postpartum. In vivo biomarkers of structural and functional brain development and aging in humans. Neurosci Biobehav Rev, in press. Front Aging Neurosci, Common and distinct structural features of schizophrenia and bipolar disorder: Prostaglandins Leukot Essent Fatty Acids, in press. Premature brain aging in humans exposed to maternal nutrient restriction during early gestation. Brain structural correlates of irritability: Findings in a large healthy cohort. Hum Brain Mapp, 38 Hippocampal metabolism and prefrontal brain structure: Associations between urban upbringing and cortical thickness and gyrification. J Psychiatr Res, Keeping brains young with making music. Brain Struct Funct, 1: Volume versus surface-based cortical thickness measurements: A comparative study with healthy controls and multiple sclerosis patients. Brain Struct Funct, 12 7: BrainAGE score indicates accelerated brain aging in schizophrenia, but not bipolar disorder. Front Aging Neurosci, 9: Cortical complexity in bipolar disorder applying a spherical harmonics approach. Effects of subclinical depression, anxiety and somatization on brain structure in healthy subjects. J Affect Disord, Estimating brain age using high-resolution pattern recognition: Younger brains in long-term meditation practitioners. Voxel-based morphometry in opera singers: Increased gray-matter volume in right somatosensory and auditory cortices. Front Cell Neurosci, 6: Glutamatergic dysfunction linked to energy and membrane lipid metabolism in frontal and anterior cingulate cortices of never treated first-episode schizophrenia patients. Schizophr Res, Brain structural correlates of schizotypy and psychosis proneness in a non-clinical healthy volunteer sample. Prefrontal gyrification in psychotic bipolar I disorder vs. Eur Neuropsychopharmacol, 25 Brain structure in schizophrenia vs. Changes of individual BrainAGE during the course of the menstrual cycle. Patterns of cortical thinning in different subgroups of schizophrenia. Br J Psychiatry, 6: Klein Standardized evaluation of algorithms for computer-aided diagnosis of dementia based on structural MRI: A step user guide for analyzing voxel-wise gray matter asymmetries in statistical parametric mapping SPM. Brain structure in people at ultra-high risk of psychosis, patients with first-episode schizophrenia, and healthy controls: Schizophrenia Res, Brain structure in narcissistic personality disorder: Psychiatry Res, 2: ZNFA genetic variation rs affects brain grey but not white matter in schizophrenia and healthy subjects. Psychological Medicine, 45 1: Voxel-based MRI intensitometry reveals extent of cerebral white matter pathology in amyotrophic lateral sclerosis. Gender-specific impact of personal health parameters on individual brain aging in cognitively unimpaired elderly subjects. Front Aging Neurosci, 6: Individualized Gaussian process-based prediction and detection of local and global gray matter abnormalities in elderly subjects. Neuroprotective effects of testosterone treatment in men with multiple sclerosis. NeuroImage Clinical, 4 Regionally accentuated reversible brain grey matter reduction in ultra marathon runners detected by voxel-based morphometry. Accelerated Brain Aging in Schizophrenia and Beyond: A Neuroanatomical Marker of Psychiatric Disorders. Schizophrenia Bulletin, 40 5: Cortical surface complexity in frontal and temporal areas varies across subgroups of schizophrenia. Human Brain Mapping, 35 4: Advanced BrainAGE in older adults with type 2 diabetes mellitus. Front Aging Neurosci, 5: Structural basis of the fronto-thalamic dysconnectivity in schizophrenia: Meditation effects within the hippocampal complex revealed by voxel-based morphometry and cytoarchitectonic probabilistic mapping. Frontiers in Psychology, 4 1: Fully Bayesian inference for structural MRI: BrainAGE in mild cognitive impaired patients: White-matter lesions drive deep gray-matter atrophy in early multiple sclerosis: Multiple Sclerosis Journal, 19 The visual cortex in schizophrenia:

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Chapter 2 : Publications at Structural Brain Mapping Group

Muscarinic receptors in schizophrenia: Prostaglandin-mediated signaling in schizophrenia. S Smesny. Pages

Advanced Search Abstract The skin flush response to niacin is abnormally blunted among a subset of patients with schizophrenia SZ , preferentially associates with SZ compared to other mental illnesses, occurs frequently in nonpsychotic members of SZ-affected families, appears heritable, and shows evidence of genetic association. From the dose-response curves, we calculated the concentration of methylnicotinate required to elicit a half-maximal blood flow MBF response EC 50 value and MBF value for each subject. Moreover, the NRA was not influenced by age, gender, race, and cigarette smoking. In summary, the NRA may define a SZ subtype with a clinically significant phospholipid signaling defect. Understanding its molecular origins may shed light on the pathophysiology of SZ and suggest new tools for its early diagnosis and treatment. The mechanism of niacin-induced skin flushing adapted from Messamore et al View large Download slide The mechanism of niacin-induced skin flushing adapted from Messamore et al We here describe the use of quantitative laser Doppler flowmetry to ascertain the prevalence of this subtype, as well as its specificity for SZ compared to BP and HC reference groups. HC subjects were recruited through the local Pittsburgh community. Inclusion Criteria for SZ. Inclusion Criteria for BP. Inclusion Criteria for HC. Regardless of subject type, those with the following characteristics were excluded: In order to increase the statistical power of sensitivity and specificity estimates, and to assess the extent to which this method can be replicated, we include in our analyses data obtained from an independently funded study conducted by Dr Messamore at the Portland VA Medical Center. Quantification of Niacin Response The cutaneous blood flow responses to graded topical doses of MN was measured according to Messamore et al. The dose-response data were analyzed by nonlinear curve fitting to calculate the EC 50 value for MN-induced blood flow as well as the maximal blood flow MBF response to MN. Statistical Analyses Data Preparation and Assessment. The distribution of the log 10 EC 50 and MBF data was confirmed to be approximately normal by univariate histogram, quantile-quantile plots, a univariate correlation test of normality, 17 bivariate scatterplots, and chi-square quantile-quantile plots and the Henze-Zirckler test 18 for the bivariate data. Covariates considered were Smoke, Race, Gender, and Age. After the SZ group was divided into 2 subgroups based on their Log 10 EC 50 and MBF measurements, the distribution of the covariate values between these 2 subgroups was examined. For categorical covariates Smoke, Gender, Race , the null hypothesis of equal proportions of the covariate in each of the SZ subgroups was tested with Fisher Exact or chi-square tests, while the null hypothesis of equal Age distributions in the 2 SZ subgroups was tested with a Kolmogorov-Smirnov test. Confirmation of Group Differences. Results Normality Quantile-quantile plots and the tests of univariate and multivariate normality confirmed that log 10 EC 50 and MBF both were distributed with approximate univariate normality as well as bivariate normality for all subject groups, HC, SZ, and BP.

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Chapter 3 : Disorders of synaptic plasticity and schizophrenia (edition) | Open Library

Prostaglandin Deficiency and Endorphin Excess in Schizophrenia: Prostaglandin-mediated signaling in schizophrenia. S Smesny; View. Show abstract. Recommended.

Biosynthesis of eicosanoids Prostaglandins are found in most tissues and organs. They are produced by almost all nucleated cells. They are autocrine and paracrine lipid mediators that act upon platelets , endothelium , uterine and mast cells. They are synthesized in the cell from the fatty acid arachidonic acid [2]. Arachidonic acid is created from diacylglycerol via phospholipase-A2 , then brought to either the cyclooxygenase pathway or the lipoxygenase pathway. The cyclooxygenase pathway produces thromboxane , prostacyclin and prostaglandin D, E and F. Alternatively, the lipoxygenase enzyme pathway is active in leukocytes and in macrophages and synthesizes leukotrienes. Release of prostaglandins from the cell[edit] Prostaglandins were originally believed to leave the cells via passive diffusion because of their high lipophilicity. The discovery of the prostaglandin transporter PGT, SLCO2A1 , which mediates the cellular uptake of prostaglandin, demonstrated that diffusion alone cannot explain the penetration of prostaglandin through the cellular membrane. The release of prostaglandin has now also been shown to be mediated by a specific transporter, namely the multidrug resistance protein 4 MRP4, ABCC4 , a member of the ATP-binding cassette transporter superfamily. Whether MRP4 is the only transporter releasing prostaglandins from the cells is still unclear. The classic dogma is as follows: COX-1 is responsible for the baseline levels of prostaglandins. COX-2 produces prostaglandins through stimulation. However, while COX-1 and COX-2 are both located in the blood vessels , stomach and the kidneys , prostaglandin levels are increased by COX-2 in scenarios of inflammation and growth. Several prostaglandin E synthases have been identified. To date, microsomal prostaglandin E synthase-1 emerges as a key enzyme in the formation of PGE2. Other terminal prostaglandin synthases[edit] Terminal prostaglandin synthases have been identified that are responsible for the formation of other prostaglandins. A thromboxane synthase TxAS has also been identified. Functions[edit] There are currently ten known prostaglandin receptors on various cell types. Prostaglandins ligate a sub-family of cell surface seven-transmembrane receptors, G-protein-coupled receptors. The diversity of receptors means that prostaglandins act on an array of cells and have a wide variety of effects such as:

Chapter 4 : Cannabinoids influence lipid-arachidonic acid pathways in schizophrenia.

Disorders of synaptic plasticity and schizophrenia by, Mitogen-activated protein kinase signaling Prostaglandin-mediated signaling in schizophrenia / S. Smesny.

Chapter 5 : Prostaglandin - Wikipedia

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Chapter 6 : - NLM Catalog Result

Cannabinoids influence lipid-arachidonic acid pathways in schizophrenia. Smesny S(1), Rosburg T, indicating disturbed prostaglandin-mediated processes.