EBREMER

ERICH BREMER

- ... *(Array, 0 elements)*
  Krumo version 0.2.1a | **http://krumo.sourceforge.net**
  Called from **/mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc**, line **257**
- ... *(Array, 0 elements)*
  Krumo version 0.2.1a | **http://krumo.sourceforge.net**
  Called from **/mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc**, line **257**
- ... *(Array, 0 elements)*
  Krumo version 0.2.1a | **http://krumo.sourceforge.net**
  Called from **/mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc**, line **257**
- ... *(Array, 0 elements)*
  Krumo version 0.2.1a | **http://krumo.sourceforge.net**
  Called from **/mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc**, line **257**
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- ... *(Array, 0 elements)*
  Krumo version 0.2.1a | **http://krumo.sourceforge.net**
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- ... *(Array, 0 elements)*
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  Called from **/mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc**, line **257**
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  Called from **/mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc**, line **257**
- page =>

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  Called from **/mnt/exodus/disk1/www/ebremer/includes/module.inc**, line **1022** **Krumo version 0.2.1a** | **http://krumo.sourceforge.net**

- *Notice*: Undefined index: optionals in *sparql_views_plugin_query_sparql->build_graph_pattern()* (line 257 of */mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc*).
- *Notice*: Undefined index: triples in *sparql_views_plugin_query_sparql->build_graph_pattern()* (line 258 of */mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc*).
- *Notice*: Undefined index: optionals in *sparql_views_plugin_query_sparql->build_graph_pattern()* (line 260 of */mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc*).
- *Warning*: Invalid argument supplied for foreach() in *sparql_views_plugin_query_sparql->build_graph_pattern()* (line 260 of */mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc*).
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- *Notice*: Undefined index: triples in *sparql_views_plugin_query_sparql->build_graph_pattern()* (line 258 of */mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc*).
- *Notice*: Undefined index: optionals in *sparql_views_plugin_query_sparql->build_graph_pattern()* (line 260 of */mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc*).
- *Warning*: Invalid argument supplied for foreach() in *sparql_views_plugin_query_sparql->build_graph_pattern()* (line 260 of */mnt/exodus/disk1/www/ebremer/sites/all/modules/sparql_views/plugins/sparql_views_plugin_query_sparql.inc*).

Navigation

Person

- Build a GMap macro
- Import core RDF vocabularies
- Import external RDF vocabulary
- Node locations
- User locations

eBremer

- eMail

Who's online

There is currently 1 user online.

- ebremer

Abstract: The junctions of the red blood cell membrane skeleton are formed by interactions between spectrin and actin protofilaments. A spectrin tryptic peptide of 16.5-kDa apparent molecular mass (based on sodium dodecyl sulfate-polyacrylamide gel electrophoresis) which binds to F-actin in cosedimentation experiments has been identified. The peptide has been partially purified by gel filtration, anion-, and cation exchange chromatography. Intact spectrin heterodimer causes half-maximal inhibition of the 16.5-kDa peptide/F-actin interaction at a concentration of 5 microM. Comparison of the two-dimensional iodopeptide maps of the 16.5-kDa peptide with maps of alpha- and beta-spectrin, demonstrate that the peptide is generated from the beta subunit. It shows no significant relationship to the peptide maps of the beta-spectrin domains I-IV. Protein sequencing indicated that this actin-binding domain represents a stretch of amino acids at the N terminus of the beta subunit from alanine 47 probably through lysine 186. The sequence derived molecular weight of this actin-binding domain is 16,290 g/mol. The sequence presented represents the region of greatest homology among the spectrin supergene family (spectrin, dystrophin, alpha-actinin).
PMID: 19064946
Abstract: Using two-dimensional difference gel electrophoresis (2D DIGE) we have analyzed monocytes derived from 10 sickle cell disease patients (5 males and 5 females ages 12-18) to generate hypotheses regarding signature proteins that appear most positively and negatively correlated with vasoocclusive event rate. Signature proteins have been identified by tandem mass spectrometry. Based on the limited number of samples analyzed, the most negatively correlated proteins related to crises rate were transketolase and coronin in the membrane fraction and heat shock 70 kDa protein cognate 4, and adenylate kinase isoenzyme 2, mitochondrial found in the cytosolic fraction. The protein spots that were most positively correlated with crisis rate in the cytoplasmic fraction were far upstream element-binding protein and Alpha actinin 1 or Alpha actinin 4. Utilizing StepSIM analysis, vinculin was able to classify all samples from the combined set and the membrane-only set, and cytosolic leukotriene A-4 hydrolase and phosphoglycerate kinase were also identified as important indicators for differentiating between low and high vasoocclusive event rates.
PMID: 7757495
Abstract: This article reviews our current knowledge of the structure of alpha spectrins and beta spectrins in the brain, as well as their location and expression within neural tissue. We discuss the known protein interactions of brain spectrin isoforms, and then describe results that suggest an important role for spectrin (alpha SpII sigma 1/beta SpII sigma 1) in the Ca(2+)-regulated release of neurotransmitters. Evidence that supports a role for spectrin in the docking of synaptic vesicles to the presynaptic plasma membrane and as a Ca2+ sensor protein that unclamps the fusion machinery is described, along with the Casting the Line model, which summarizes the information. We finish with a discussion of the value of spectrin and ankyrin-deficient mouse models in deciphering spectrin function in neural tissue.
PMID: 6572929
Abstract: IgG autoantibodies in human serum selectively bind to a glycopeptide antigen that appears on senescent and damaged cells in situ. We identified the membrane protein from which the senescent cell antigen is derived by using a phagocytosis-inhibition assay and immunoautoradiographic gel staining and electroblotting techniques. Results of the phagocytosis-inhibition assay revealed that only the purified transmembrane glycoprotein designated band 3 and senescent cell antigen inhibited the phagocytosis of erythrocytes induced by IgG eluted from senescent erythrocytes. Purified spectrin, syndein, band 4.1, actin, glycophorin A, and intact or desialylated sialoglycoprotein periodic acid/Schiff (PAS) staining bands 1-4 containing glycophorins A, B, and C did not inhibit phagocytosis. Specific antibodies against the senescent cell antigen and erythrocyte band 3 were used to identify the membrane protein from which the senescent cell antigen is derived. Band 3-related polypeptides (MrS approximately equal to 60,000, 42,000, and 18-26,000) were identified in erythrocyte ghosts prepared in the presence of diisopropyl fluorophosphate, phenylmethylsulfonyl fluoride, and EDTA by immunoautoradiography with antiband 3. Antibodies to senescent cell antigen reacted with band 3 and the same lower Mr band 3-related polypeptides. Thus, the senescent cell antigen is immunologically related to band 3.
PMID: 16816129
Abstract:
PMID: 16816128
Abstract:
PMID: 6403381
Abstract:
PMID: 3121803
Abstract: An immunoreactive, structural, and functional analog of erythrocyte protein 4.1 is present in neuronal cell bodies and dendrites. Other investigators have described the isolation of a 4.1 analog in brain with structural characteristics suggesting that its identity was synapsin I, a neuronal phosphoprotein localized in the presynaptic terminal in association with small synaptic vesicles. In this report we demonstrate that the cell body/dendritic form of brain protein 4.1, which we have named amelin, is distinct from that of synapsin I on the basis of subcellular localization, migration in 2-dimensional gel electrophoresis, and structural criteria. We also demonstrate that amelin, like synapsin I, can bind brain spectrin on nitrocellulose paper. Neither amelin nor synapsin I binds calmodulin, as determined by a blot binding assay. We hypothesize that there exists in brain a family of 4.1-related proteins with distinct subcellular localization and function.
PMID: 16254212
Abstract: Store-operated calcium (SOC) entry represents the principal Ca2+ entry pathway into nonexcitable cells. Despite intensive investigation, mechanisms underlying activation of SOC entry have remained elusive. The endothelial ISOC channel is a Ca2+-selective SOC entry channel to which the transient receptor potential (TRP) proteins TRPC1 and TRPC4 contribute subunits. Activation of ISOC is specifically regulated by the spectrin-actin membrane skeleton; however, the nature of coupling between the ISOC channel and membrane skeleton is unknown. Here we demonstrate that protein 4.1 is an essential component of the ISOC channel gating mechanism. Protein 4.1 interacts with TRPC4 and the membrane skeleton. Deletion of the protein 4.1 binding domain on TRPC4 or peptide competition to the protein 4.1 binding domain prevents ISOC activation. These findings reveal that interaction of protein 4.1 with TRPC4 is required for activation of the endothelial ISOC channel.
PMID: 2899449
Abstract: The binding of [3H] [D-Ala2, MePhe4, Gly-ol5]enkephalin ([3H]DAGO), [3H]D-Ala2,D-Leu5]enkephalin ([3H]DADLE) and (+/-)-[3H]ethylketocyclazocine ([3H]EKC) to neurotumor tissues derived from S20Y neuroblastoma cells transplanted into A/Jax mice was examined. Specific and saturable binding to [3H]DADLE and [3H]EKC was detected, and the data fit a single homogeneous binding site for each ligand. Scatchard analysis for [3H]DADLE and [3H]EKC yielded Kd values of 0.65 and 0.45 nM, respectively, and Bmax values of 9.2 and 116 fmol/mg protein. Binding was dependent on time, temperature, and pH, and was sensitive to Na+ and guanine nucleotides. Pretreatment of the tumor homogenates with trypsin markedly reduced binding to both ligands, suggesting that the binding sites were proteinaceous in character. Displacement experiments indicated that delta (delta) receptor related compounds (e.g. DPDPE, ICI 174,864) avidly displaced [3H]DADLE, whereas kappa (kappa) related compounds (e.g. U50,488, dynorphin) markedly competed with [3H]EKC. Mu (mu) receptor drugs (e.g. DAGO, beta-FNA, morphine) were not potent in displacing either [3H]DADLE or [3H]EKC. These results are the first to characterize opioid binding sites in tumor tissue. The function of these sites is unclear, but previous evidence as to the growth regulatory properties of endogenous opioid systems may suggest that either one, or both, binding sites may be involved in carcinogenic events.
PMID: 21237293
Abstract: Mitochondrial structural and functional alterations appear to play to an important role in the pathogenesis of Alzheimer's disease (AD). In the present study, we used a quantitative comparative proteomic profiling approach to analyze changes in the mitochondrial proteome in AD. A triple