

脳虚血における細胞のストレス応答の可視化とその制御

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2000 Fiscal Year Final Research Report Summary

Visualization and regulation of ischemia-induced stress response in brain.

Research Project

Project/Area Number

10480215

Research Category

Grant-in-Aid for Scientific Research (B).

Allocation Type

Single-year Grants

Section

一般

Research Field

Neurochemistry/Neuropharmacology

Research Institution

Kanazawa University (1999-2000)

Osaka University (1998)

Principal Investigator

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Project Period (FY)

1998 - 2000

Keywords

Heat shock protein / Energy metabolism / Stress response / Transgenic mice / Stress reporter / Gene therapy

Research Abstract

An integral component of the cellular response to environmental challenge is expression, usually by de novo protein synthesis, of stress-associated polypeptides, such as heat shock proteins (induced by high temperature), glucose-regulated proteins (GRPs ; induced by glucose deprivation), and oxygen-regulated proteins (induced by oxygen deprivation). These biosynthetic responses are well preserved from prokaryotes to mammals, and have been hypothesized to contribute importantly to maintenance of cellular homeostasis as cellular adaptation to altered environmental conditions is under way.

Astrocytes are strategically positioned to exert cytoprotective effects on neurons, the latter known for their vulnerability to changes in the local environment. Such neuro-

Research Products (17 results)

All Other
All Publications

[Publications] Bando Y (ほか): "The 150 kDa Oxygen Regulated Protein (ORP150) functions as a novel molecular chaperone in the protein transport of the MDCK cells."Am.J.Physiol. (Cell Physiol.). 278. C1172-1182 (2000) ▼

[Publications] Taguchi A (ほか): "Blockade of rage-amphoterin axis suppresses tumor growth and metastases."Nature. 405. 354-360 (2000) ▼

[Publications] Kobayashi T (ほか): "bundant expression of 150-kDa oxygen-regulated protein in mouse pancreatic beta cells is correlated with insulin secretion."Biochem.Biophys.Res.Commun.. 267. 831-837 (2000) ▼

[Publications] Che YH (ほか): "Changes in mRNA of protein inhibitor of neuronal nitric oxide synthase following facial nerve transection."J.Chem.Neuroanat. 17. 199-206 (2000) ▼

[Publications] Tamatani M (ほか): "ORP150 protects against hypoxia/ischemia-induced neuronal death."Nature Med.. 7. 317-323 (2001) ▼

[Publications] Tsukamoto Y (ほか): "Expression of a novel RNA splicing factor, RA301/Tra2beta, in vascular lesions and its role in smooth muscle cell proliferation."Am.J.Pathol. (In press). (2001) ▼

[Publications] Yamaguchi A, et al.: "Stress-associated endoplasmic reticulum protein 1 (SERP1)/Ribosome-associated membrane protein 4 (RAMP4) stabilizes membrane proteins during stress and facilitates subsequent glycosylation."J.Cell Biol.. 147. 1195-1204 (1999) ▼

[Publications] Niitsu Y, et al.: "Exposure of cultured primary rat astrocytes to hypoxia results in intracellular glucose depletion and induction of glycolytic enzymes."Brain Res.Mol.Brain Res.. 74. 26-34 (1999) ▼

[Publications] Tamatani M, et al.: "Tumor necrosis factor induces Bcl-2 and Bcl-x expression through NFkappaB activation in primary hippocampal neurons."J.Biol.Chem.. 274. 8531-8538 (1999) ▼

[Publications] Ozawa K., et al.: "ORP150 (150kDa oxygen-regulated protein) suppresses hypoxia-induced apoptotic cell death."J.Biol.Chem.. 274. 6397-6404 (1999) ▼

[Publications] Yan S.D.et al.: "Role of ERAB/L-3 Hydroxyacyl-coenzyme A dehydrogenase type II activity in Ab-induced cytotoxicity."J.Biol.Chem.. 274. 2145-2156 (1999) ▼

[Publications] Bando Y et al.: "The 150 kDa Oxygen Regulated Protein (ORP150) functions as a novel molecular chaperone in the protein transport of the MDCK cells."Am.J.Physiol.(Cell Physiol.). 278, (6). C1172-1182 (2000) ▼

[Publications] Taguchi A, et al.: "Blockade of rage-amphoterin axis suppresses tumor growth and metastases."Nature. 405. 354-360 (2000) ▼

[Publications] Kobayashi T, et al.: "Abundant expression of 150-kDa oxygen-regulated protein in mouse pancreatic beta cells is correlated with insulin secretion."Biochem.Biophys.Res.Commun.. 267. 831-837 (2000) ▼

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