Role of Cytokines in mediating of Ischemia/Reperfusion Injury in Liver.

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1997 Fiscal Year Final Research Report Summary

Role of Cytokines in mediating of Ischemia/Reperfusion Injury in Liver.

Research Project

Project/Area Number

08671424

Research Category

Grant-in-Aid for Scientific Research (C)

Allocation Type

Single-year Grants

Section

一般

Research Field

Digestive surgery

Research Institution

Kanazawa University

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Project Period (FY)

1996 – 1997

Keywords

ischemia / reperfusion injury / cytokine / KF-kappaB / tyrosin kinase / JNK / apoptosis / liver

Research Abstract

One of the most important complications after organ transplantation is graft damage caused by ischemia/reperfusion injury. The details of the mechanisms underlying organ injury under ischemia and reperfusion are not yet understood. We investigated the role of cytokines in mediating of ischemia/reperfusion injury and the intracellular signal transduction that modulates cytokine production.
The results from in-vitro experiments have shown that hypoxia induces the activation of NF-kappaB and tyrosine kinase inhibitors inhibits NF-kappaB activation by hypoxia. And the results from experiments using a mouse model for hepatic ischemia and reperfusion have shown that inflammatory cytokies affect liver injury following ischemia/reperfusion, and that pretreatment with a tyrosine kinase inhibitor, genistein, suppresses ischemia/reperfusion injury of the liver. Furthermore, it was also shown that JNK (c-Jun N-terminal kinase) was activated following hepatic ischemia and reperfusion. Interestingly, the activation of JNK and the number of apoptotic cells increased by shorter period of ischemia rather than longer period.
These results suggest that inhibition of cytokine production can suppress ischemia/reperfusion injury, and that JNK activation and apoptosis after short period of ischemia may play a protective role in tissue subjected to ischemia and reperfusion.

Research Products (6 results)

All | Other

All | Publications (6 results)

[Publications] Yamamoto, S., Shimizu, K., et al.: "Genistein suppresses cellular injury following hepatic ischemia/reperfusion." Transplantation Proceedings. 28・(2). 1111-1115 (1996)

[Publications] Muraoka, K., Shimizu, K, et al.: "Hypoxia,but not reoxygenation,induces interleukin 6 gene expression through NF-κB activation." Transplantation. 63・(2). 466-470 (1997)

[Publications] Onishi, I., Shimizu, K., et al.: "Activation of c-Jun N-terminalkinase during ischemia and reperfusion in mouse liver." FEBS Letters. 420. 201-204 (1997)

[Publications] Yamamoto, S., Shimizu, K., et al.: "Genistein suppresses cellular injury following hepatic ischemia/reperfusion." Transplantation Proceedings. 28-2. 1111-1115 (1996)

[Publications] Muraoka, K., Shimizu, K., et al.: "Hypoxia, but not reoxygenation, induces interleukin 6 gene expression through NF-kappaB activation." Transplantation. 63-2. 466-470 (1997)

[Publications] Onishi, I., Tani, T., et al.: "Activation of c-Jun N-terminal kinase during ischemia and reperfusion in mouse liver." FEBS Letters. 420. 201-204 (1997)

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