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Ischemic preconditioning induces neuroprotection cause by a transient global ischemia via maintaining the expression of P63

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Introduction

p63 is a transcription factor of p53 gene family, which are involved in development, differentiation and cell response to stress; however, their roles in ischemic preconditioning (IPC) in the brain are not clear.

Objectives

In the present study, we investigated the effect of IPC on p63 immunoreactivity caused by transient cerebral ischemia, which was induced by 5 min of transient ischemia, in gerbils, and IPC was induced by subjecting the gerbils to 2 min of ischemia followed by 1 day of recovery.

Methods

The animals were randomly assigned to 4 groups (sham-operated-group, ischemia-operated-group, IPC plus (+)-sham-operated-group and IPC+ischemia-operated-group).

Results

The number of viable neurons in the stratum pyramidale of the hippocampal CA1 region (CA1) was significantly increased by IPC+ischemia-operated-group compared with that in the ischemia-operated-group 5 days after ischemic insult. We found that strong p63 immunoreactivity was detected in the CA1 pyramidal neurons in the sham-operated-group, and the immunoreactivity was decreased with time after ischemia-reperfusion. In addition, strong p63 immunoreactivity was newly expressed in the microglial cells of the CA1 region from 2 days after ischemia-reperfusion. In all the IPC+sham-operated-group, p63 immunoreactivity in the CA1 pyramidal neurons was similar to that in the sham-operated-group, and the immunoreactivity was well maintained in the IPC+ischemia-operated-groups after cerebral ischemia.

Conclusions

In brief, our present findings showed that IPC dramatically protected the reduction of p63 immunoreactivity in the pyramidal neurons of the CA1 region after ischemia-reperfusion, and this result suggests that the expression of p63 may be necessary for the neurons to survive after transient cerebral ischemia.

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