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Protective effects of parecoxib on rat primary astrocytes from oxidative stress induced by hydrogen peroxide

Key words: Parecoxib, primary astrocyte, hydrogen peroxide (H_2O_2), Bax, Bcl-2, BDNF

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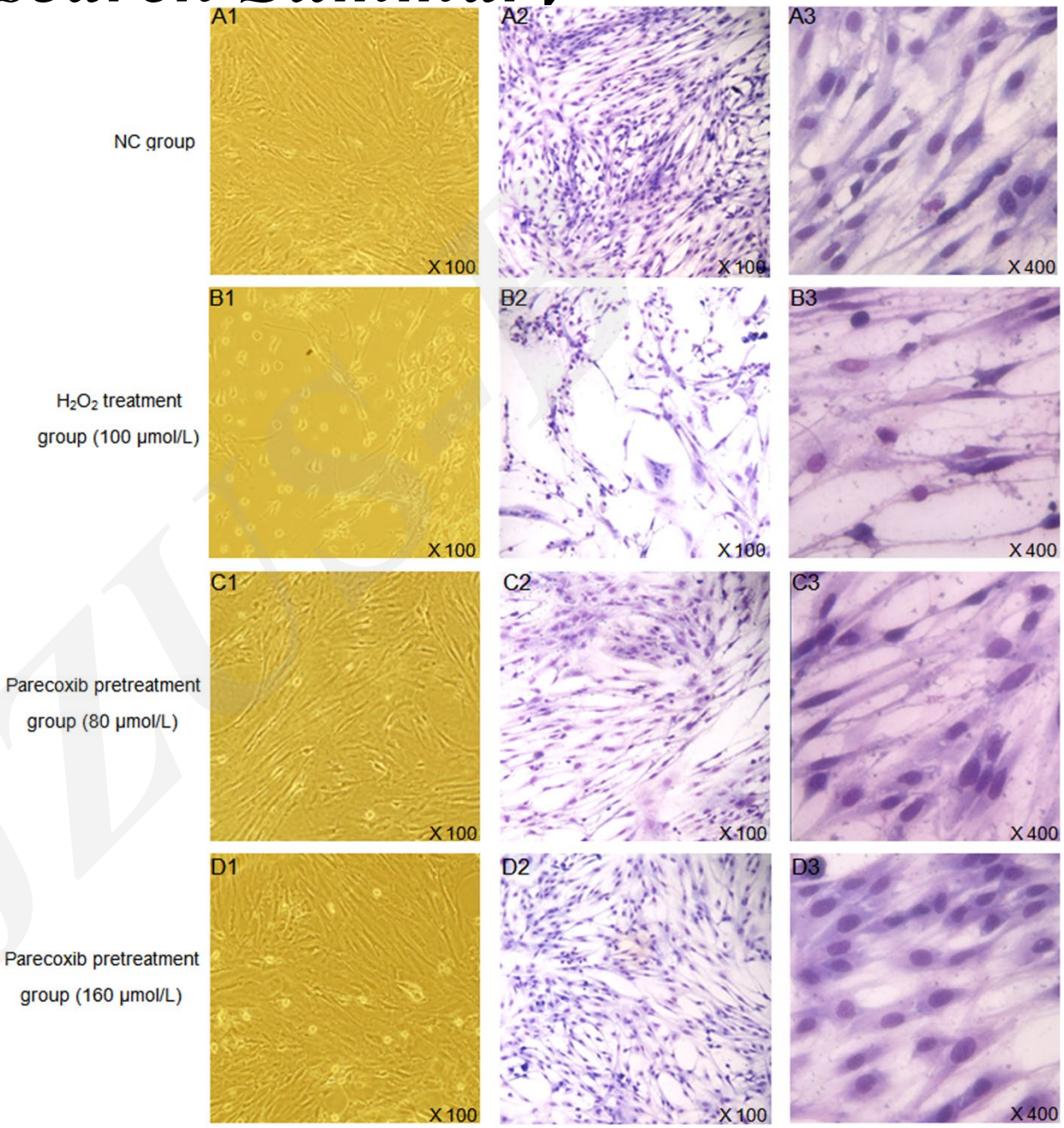
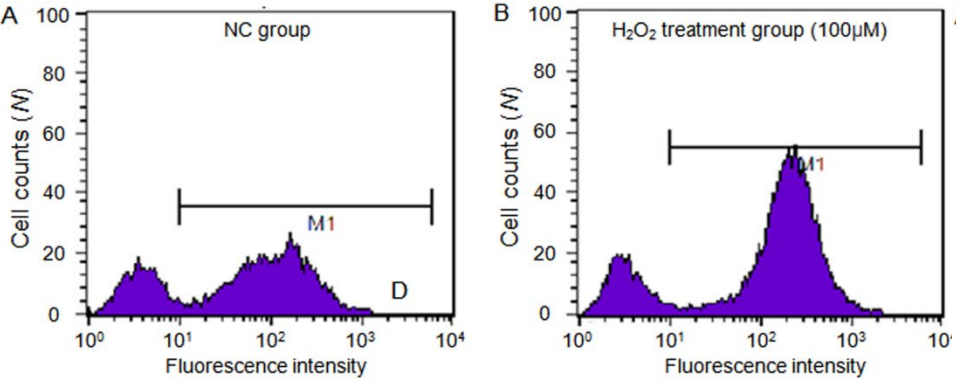
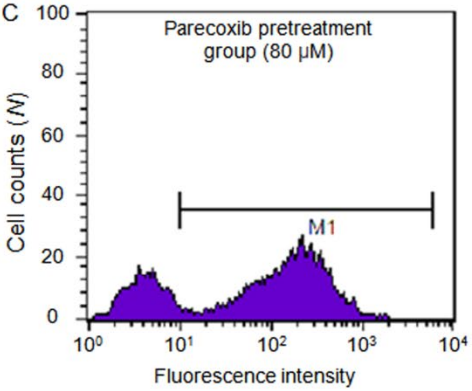
Research Summary

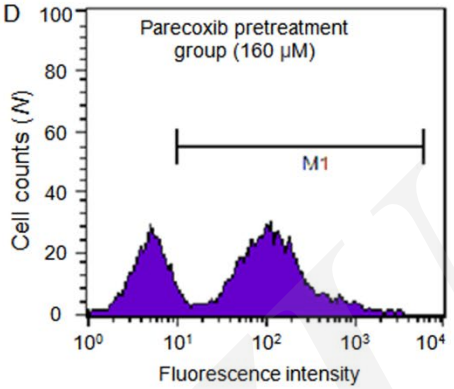
This study aimed to to investigate the protective effects of parecoxib on oxidative stress induced by hydrogen peroxide (H_2O_2) in rat astrocytes *in vitro*.

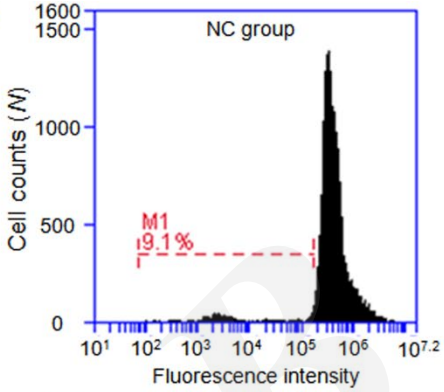


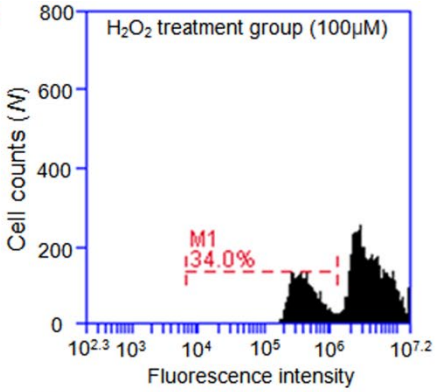

A
NC group
Cell counts (N)
M1
D
Fluorescence intensity

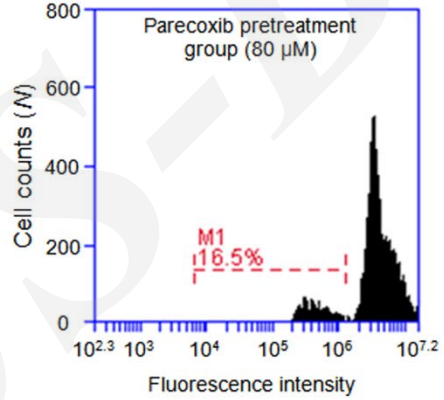
B
H2O2 treatment group (100μM)
Cell counts (N)
M1
Fluorescence intensity

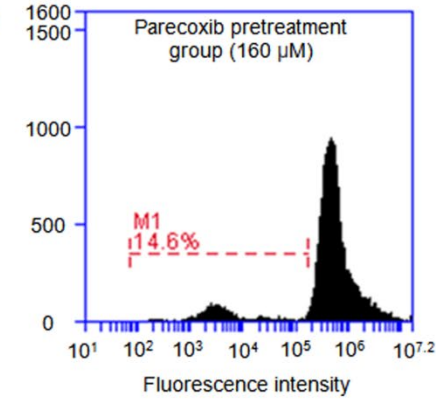

C
Parecoxib pretreatment group (80 μM)
Cell counts (N)
M1
Fluorescence intensity

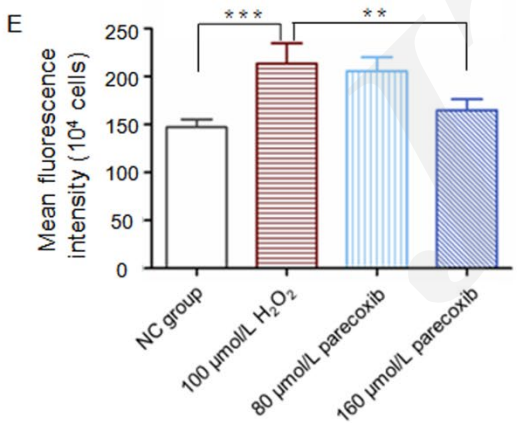

D
Parecoxib pretreatment group (160 μM)
Cell counts (N)
M1
Fluorescence intensity

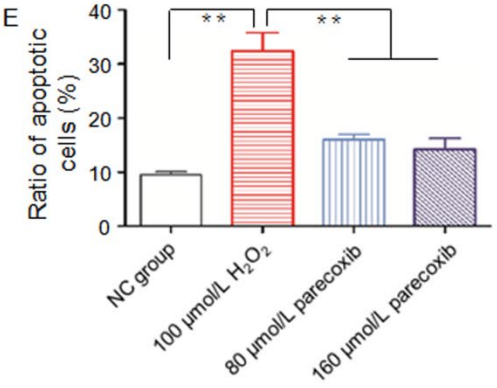

A
NC group
Cell counts (N)
M1
9.1%
Fluorescence intensity

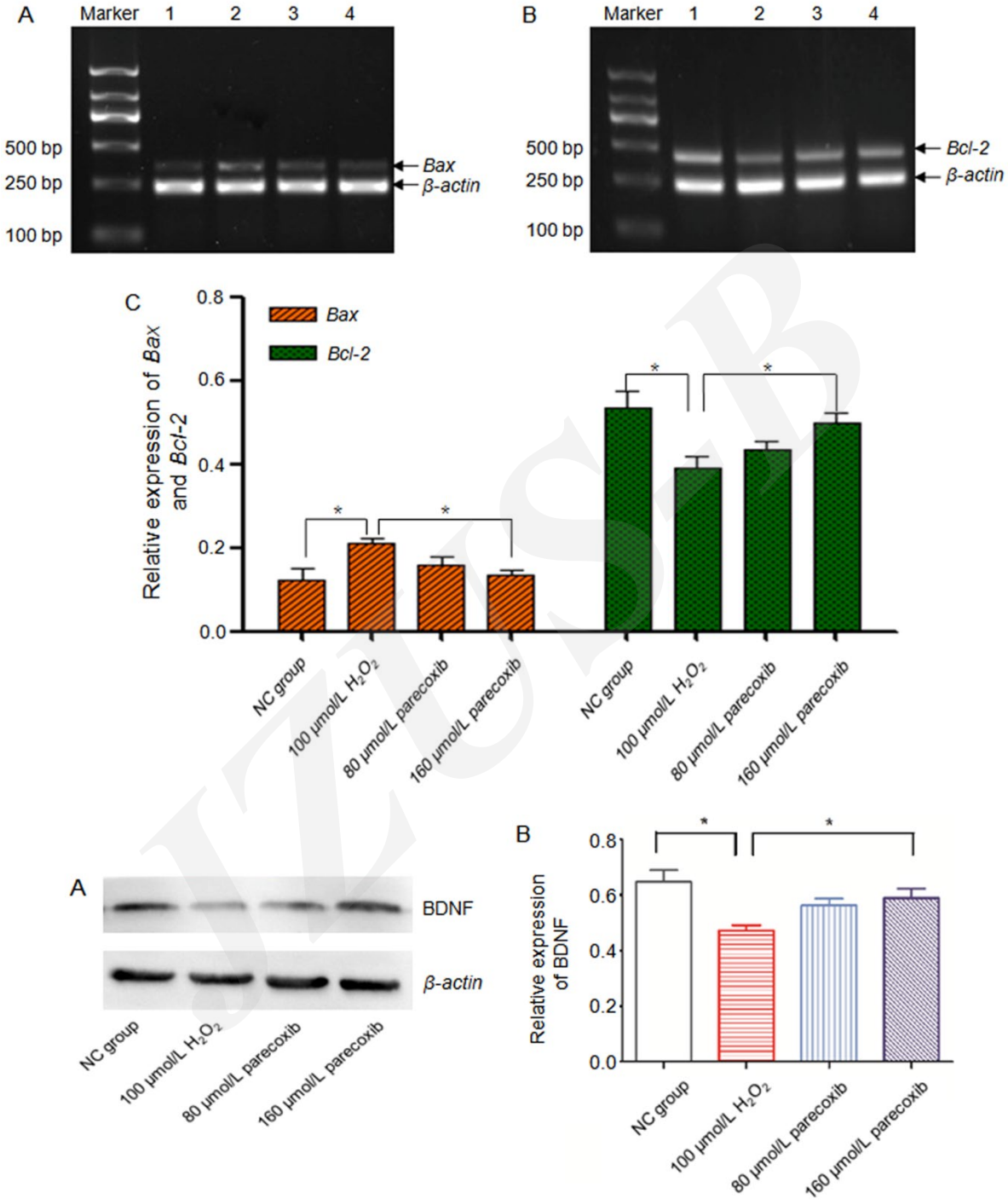

B
H2O2 treatment group (100μM)
Cell counts (N)
M1
34.0%
Fluorescence intensity


C
Parecoxib pretreatment group (80 μM)
Cell counts (N)
M1
16.5%
Fluorescence intensity


D
Parecoxib pretreatment group (160 μM)
Cell counts (N)
M1
14.6%
Fluorescence intensity


E
Mean fluorescence intensity (10⁴ cells)
***
**
NC group
100 μmol/L H2O2
80 μmol/L parecoxib
160 μmol/L parecoxib


E
Ratio of apoptotic cells (%)
**
**
NC group
100 μmol/L H2O2
80 μmol/L parecoxib
160 μmol/L parecoxib


A
Marker
1
2
3
4
500 bp
250 bp
100 bp
Bax
β-actin
B
Marker
1
2
3
4
500 bp
250 bp
100 bp
Bcl-2
β-actin
C
Relative expression of Bax and Bcl-2
0.8
0.6
0.4
0.2
0.0
Bax
Bcl-2
*
*
*
*
NC group
100 μmol/L H2O2
80 μmol/L parecoxib
160 μmol/L parecoxib
NC group
100 μmol/L H2O2
80 μmol/L parecoxib
160 μmol/L parecoxib
A
BDNF
β-actin
NC group
100 μmol/L H2O2
80 μmol/L parecoxib
160 μmol/L parecoxib
B
Relative expression of BDNF
0.8
0.6
0.4
0.2
0.0
*
*
NC group
100 μmol/L H2O2
80 μmol/L parecoxib
160 μmol/L parecoxib

Innovation points

A serial of experiments, including cell viability assay, apoptosis assay, ROS level detection and mRNA/protein expression, were performed.

This study concluded that parecoxib pretreatment could reverse the changes on rat primary astrocytes induced by H2O2 treatment.

Mechanical investigation showed that dysregulated Bax, Bcl-2 and BDNF could be implicated in these changes.

JZUS-B