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Early evidence of stress in immortalized neurons exposed to diesel particles: the role of lipid reshaping behind oxidative stress and inflammation

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Highlights

- • HT22 cells viability was not affected by 3h-24h of DEP exposure.
- DEP treatment induced oxidative stress-related proteins increase in HT22 cells.
- DEP treatment promoted inflammation-related proteins increase in HT22 cells.
- 24h of DEP treatment caused lipid reshaping and membrane rigidity in HT22 cells.
- 24h of DEP treatment affected APP processing-related proteins in HT22 cells.

ABSTRACT

Diesel combustion is the major source of fine particle road emission, whose solid fraction is represented by diesel exhaust particles (DEP). Many studies indicate the contribution of DEP to the onset of different neurological diseases, such as Alzheimer's disease (AD), identifying oxidative