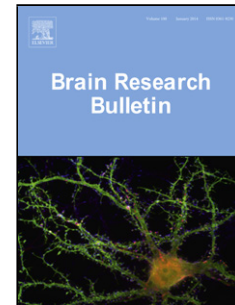


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Highlights

- ER α /ER β and GPR30 was observed in TDP-25 cells, a new cellular model of ALS;
- ER α and GPR30 differentially localize in TDP-25 cells compared with control E cells;
- Ral and E2 promote cell viability through ER- and GPR30-dependent mechanisms;
- Ral and E2 enhance autophagy and suppress apoptosis through ER and GPR30.

Abstract

Amyotrophic lateral sclerosis (ALS) is a fatal adult-onset neurodegenerative disease, and at present, therapies for ALS are limited. Estrogen is a potential therapeutic agent

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