Bombesin-like receptor 3 expression induced by bisphenol A is likely associated with reduced cell proliferation by inhibiting DNA synthesis and inducing inflammation in liver cells

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Background

Bisphenol A (BPA) is an exogenous endocrine disruptor mimicking hormones closely associated with health complications, such as cancer progression. BPA is also related to an increase in the prevalence of obesity-related diseases due to its obesogenic action. Bombesin-like receptor 3 (BRS3) is an important factor that should be considered in the adipogenic gene network, as depletion of this gene alters adiposity.

Methods

Therefore, the present study aimed to investigate the messenger ribonucleic acid (mRNA) expression of BRS3 in human liver THLE-2 cells post-BPA treatment by real-time polymerase chain reaction. The effects of BPA on the levels of pro-inflammatory proteins, interleukin 6 (IL6) and CC motif chemokine ligand 2 (CCL2), in conditioned media of BPA-treated THLE-2 cells and deoxyribonucleic acid (DNA) synthesis in replicating BPA-treated THLE-2 cells during the cell cycle were also examined by enzyme-linked immunosorbent assay (ELISA) and flow cytometry, respectively.

Results

The study found that the mRNA expression of BRS3 was increased in THLE-2 cells treated with BPA. The study also showed that the expression levels of IL6 and CCL2 reached an optimum level in the conditioned media of BPA-treated THLE-2 cells after 48 h of treatment. Subsequently, the DNA synthesis analysis showed that bromodeoxyuridine/propidium iodide (BrdU/PI) stained positive cells were decreased in BPA-treated THLE-2 cells at 72 h of treatment.

Conclusion

The study demonstrates that BRS3 expression induced by BPA is likely associated with reduced cell proliferation by inhibiting DNA synthesis and inducing cellular inflammation in liver cells.