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Title: Long-Term Exposure to Decabromodiphenyl Ether Promotes the Proliferation and Tumourigenesis of Papillary Thyroid Carcinoma by Inhibiting TRß

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Polybrominated diphenyl ethers (PBDEs) have been reported to possess endocrine-disrupting and tumour-promoting activity. However, the effects of long-term exposure to decabromodiphenyl ether (BDE209) on thyroid tumourigenesis of papillary thyroid carcinoma (PTC) and the underlying mechanisms remain poorly defined. In this study, functional assays in vitro and mouse models in vivo were used to evaluate the toxic effects of long-term exposure to environmental concentrations of BDE209 on the pathogenesis and progression of PTC. MTS, EdU and colony-forming assays confirmed the chronic toxicity of BDE209 on the proliferation of human normal follicular epithelial cell line (Nthy-ori 3-1) and PTC-derived cell lines (TPC-1 and BCPAP). Wound and Transwell assays showed that BDE209 exacerbated the aggressiveness of PTC cells. BDE209 significantly promoted cell proliferation during the S and G2/M phases of the cell cycle. Mechanistically, BDE209 altered the thyroid system by acting as a competitive inhibitor of thyroid receptor beta (TRß) expression and function, which was further proven by public databases and RNA-seq bioinformation analysis. Taken together, these results demonstrated that BDE209 has chronic toxicity and potential tumourigenic effects on the thyroid by inhibiting TRß.

Biography

Xinpei Wang is a postgraduate student now. She has passion in improving the health and wellbeing. Together with her mentor, she worked on the study of the causes of thyroid cancer. Recently the problem of environmental pollution has gradually attracted their attention.