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Lipopolysaccharide increase PD-L1 expression in pancreatic tumor via TLR4/MyD88/NF-κB pathway

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Introduction : The programmed death ligand 1(PD-L1) is essential for tumor cells to keep immune escape and related to poor prognosis. Previous studies have identified that bacteria lipopolysaccharide (LPS) can affect the expression of PD-L1 on immune cell, but its effect on the tumor cell hasn't been investigated. This study is aimed at whether lipopolysaccharide (LPS) can induce PD-L1 on pancreatic tumor.

Methods : The effect of LPS on the two pancreatic tumor cell lines (Panc1 and Bxpc-3) was analyzed in vitro. The level of PD-L1 and its relevant pathway were verified by real time PCR and western blot.

Results : The expression of PD-L1, Toll-like receptor 4(TLR4) and MyD88 was upgraded after LPS stimulation in a concentration-dependent way and the nuclear factor-κB（NF-κB） pathway is also activated. TLR4-sh plasmid and MyD88-sh plasmid was transfected into cell lines respectively. Correspondingly, PD-L1 expression was obviously downgraded comparing with their control groups. Meanwhile, the effect of LPS was neutralized when cells were pre-treated with NF-κB pathway specific inhibitor PDTC and Bay-11-7082 . Furthermore, we observed that p65 could bind to the PD-L1 promoter by Chip assay.

Conclusions : Overall, our study has shown that LPS can induce PD-L1 expression in pancreatic tumor via TLR4/MyD88/NF-κB pathway. Intervening LPS in vivo might be another way to reduce PD-L1-mediated immune escape.

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