Table SI. Pathways or molecular mechanisms related to the antitumor effects or DCs in CCA.

Pathway/molecularmechanism	Results	(Refs.)[a]
JAK-STAT3, TGF-β/Smad	Knockdown of TGF-βRII and IL-10RA mRNA by transducing self-differentiated DCs with a short-hairpin RNA lentiviral increases the antitumor activity of T-cells.	(99)
	Inhibition of the IL-10 and TGF-β receptors on DCs significantly enhances cytolytic activity of effector T-cells.	(100)
LAG-3, PD-1/PD-L1	Co-blockade of LAG-3 and PD-L1 promotes antigen cross-presentation by DCs and subsequently potentiate $CD8^+$ T-cell cytotoxicity.	(101)
CD40-CD40L	Activation of the CD40-CD40L pathway upregulates Th1-cytokines, leading to the immune regulatory function of DCs, promoting tumor-specific effector cell proliferation and cytotoxicity towards bile duct tumors.	(104)
CD40-CD40L, PD-1/PD-L1	Activation of the CD40-CD40L pathway can stimulate DCs and increase the response to PD-1 antagonist in murine iCCA growth.	(175)

[a]The cited references can be found in the reference list in the main manuscript. DCs, dendritic cells; PD-1, programmed cell death protein 1; PD-L1, programmed cell death ligand 1;LAG-3, lymphocyte-activation gene 3; iCCA, intrahepatic cholangiocarcinoma.