Figure S1. Diagrammatic summary of dynamic T and B cell interaction in the TLS associated with the TME of gastric cancer. Naïve T and B cells are recruited to TLS upon interaction between CD62L, which is expressed on naïve lymphocytes, and PNAd, which is expressed on HEVs. $CD4^+$ T_h and T_{FH} can promote B cell activation, germinal center formation, and plasma cell differentiation. The plasma cells secrete antibodies and participate in humoral antitumor immune responses. $CD4^+$ and $CD8^+$ T cells can also be activated upon engaging tumor-antigen-peptides/MHC presented by DC and become memory or effector T cells. Effector $CD8^+$ T cells in the TME upregulate PD-1. Based on the results from studies of chronic HIV or LMCV infection, we hypothesize that $PD1^+CD8^+$ TILs can be further divided into $CXCR5^+$ and $CXCR5^-$ subsets, which are regulated by id2/E2A axis. The $CXCR5^+$ subset located in the B cell follicle and the germinal center. The follicular $CXCR5^+$ $CD8^+$ T cells subset can undergo proliferation and differentiate into the $CXCR5^-$ subset upon id2 upregulation. Based on previous studies, we further hypothesize that germinal center $CXCR5^+$ $CD8^+$ T cells can exert a suppressive effect on germinal B cell responses and inhibit the generation of plasma cells. In contrast, ample evidence has supported the notation that B cells and plasma cells can inhibit $CD8^+$ T cell-mediated antitumor effect. TME, tumor microenvironment; TLS, tertiary lymphoid structures; SLO, secondary lymphoid organ; APC, antigen presenting cell; FDC, follicular dendritic cells; T_h, helper T cell; T_{FH}, follicular helper T cell; SHM, somatic hypermutation; CRS, class switch recombination; PNAd, Peripheral Node Addressin.