***Supplementary Figure: ICAM-1-LFA-1 interactions reverse the effect of PGE2 on CL4 CD8+ T cell proliferation.*** **A:** Naïve CD8+ T cells express LFA-1 with low affinity for ICAM-1. Temporary interaction of TcR with peptide/MHC class I complex on tumor cell stimulates Ca2+ influx inside CD8+ T cell, resulting in increased affinity of LFA-1 for ICAM-1. **B:** tumor cells express low levels of ICAM-1, a stabilized contact between tumor and CD8+ T cell cannot be maintained, and the T cell fails to receive sufficient signal to maintain Ca2+ influx. Under these situations, the increased cAMP levels in CD8+ T cell will interfere with Ca2+ influx in the T cell, resulting in reduced affinity of LFA-1 for ICAM-1. Eventually, the interaction between tumor and CD8+ T cell will be terminated and the two cells disassociate without resulting in productive activation. **C:** In contrast, tumor cells expressing high levels of ICAM-1, maintain contact with CD8+ T cells through interaction of ICAM-1 with LFA-1. Once a stable synapse is formed, the signals provided by both TcR–peptide & MHC class I complex and LFA-1 & ICAM-1 interactions activate the enzyme PLC-γ, resulting in increased Ca2+ influx in CD8+ T cell. The constant Ca2+ influx ensures a high affinity state of LFA-1 to be maintained for ICAM-1. Ultimately, signals provided by ICAM-1 & LFA-1 and MHC & TcR interactions result in IFN-γ production by the CD8+ T cell. In this situation the inhibition on Ca2+ influx, induced by PGE2-dependent increase in cAMP levels, fails to override the stimulatory signals maintained by TcR & LFA-1 ligation, and the T cell maintains its contact with tumor cell and undergoes productive activation.