Aggressive lymphomas represent the most common type of lymphoid malignancies with a five-year survival rate of 60%. Despite effective initial treatment, one-third of all patients will experience a relapse, warranting more research to develop novel therapies. We recently detected a significant reduction of nuclear receptor *NR4A1* expression in aggressive lymphoma patients that correlated with poor cancer-specific survival. We observed that loss of *Nr4a1* leads to an accelerated lymphomagenesis *in vivo*, concomitant with increased expression of immune checkpoints. Immuno-competent, but not immune-deficient, mice transplanted with *Nr4a1*-deficient lymphoma cells exhibited rapid lymphoma development, reduced survival, and upregulation of immune checkpoints. To further further dissected the immunoregulatory function of *Nr4a1* in aggressive lymphoma., we used OT-1 CD8+ T cells expressing a T cell receptor targeting ovalbumin peptide (OVA) and EμMyc Nr4a1+/+ or EμMyc Nr4a1-/- lymphoma cell lines, which were pulsed with the OVA, in a co-culture cytotoxicity assay. Immune cell mediated lymphoma cell lysis was measured after 4h, 8h, 16h, and 24h, respectively. Interestingly, lymphoma cell killing was diminished in the EμMyc Nr4a1-/- setting after 16h and 24h. Our data suggest that Nr4a1 plays a critical role in regulating the licensing of immune evasion in aggressive lymphomas by regulating the immune checkpoint expression.