Activation of T cell receptors (TCR) by agonist peptide major histocompatibility complex (pMHC) molecules is the front line of the adaptive immune response. T cells discriminate among different pMHC based on molecular binding dwell time between pMHC and TCR, and this discrimination is widely considered to utilize a kinetic proofreading mechanism. After TCR activation, activated ZAP70 kinase at the TCR phosphorylates the scaffold molecule, LAT. It has recently been realized that LAT undergoes a protein condensation phase transition on the membrane surface, in a phosphorylation dependent manner. In this talk I will focus on the mechanism of LAT condensation and possible roles for this phase transition in the context of T cell signaling.