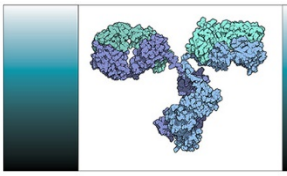
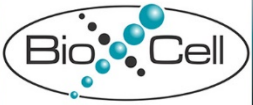


Research Highlight



IL-2 and α-ketoglutarate-sensitive metabolic changes drive T cell differentiation gene programs

For stem cells to differentiate into families of bone cells, muscle cells, blood cells, neurons and others, differing gene programs must be turned on or off. Metabolic states dynamically change during cellular differentiation, but it is currently unclear how changes in metabolism mechanistically regulate differentiation gene programs.

Using an *in vitro* T cell model Dr. Amy S. Weinmann and her colleagues from University of Alabama determined that high IL-2 concentrations induced α-ketoglutarate accumulation, a metabolite of glutamine, which in turn drove the expression of genes associated with effector potential, antigen response, IL-2-STAT5 signaling, and glycolysis. Further analysis revealed that α-ketoglutarate is responsible for part, but not all, of IL-2's impact on T cell gene programming.

Using ChIP-seq the authors found that α-ketoglutarate acts as a cofactor for DNA and histone demethylation which was required for IL-2 and α-ketoglutarate-sensitive T cell gene programming. Additionally, the authors found that α-ketoglutarate promoted the binding of CCCTC-binding factor to genomic sites associated with genes from developmental pathways. These sane genes displayed α-ketoglutarate-sensitive expression in embryonic stem cells.

This study identifies one mechanism by which changes in metabolism play a role in regulating cellular differentiation programs. These types of studies help us to understand how each cell knows how to be what it needs to be at each point in time. The authors used Bio X Cell's affordable anti-CD16/23 FcBlock (catalog #: CUS-HB-197).

Read the full article in Immunity: http://www.cell.com/immunity/abstract/S1074-7613(17)30322-9