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CORRECTION

Author Correction: metabolic signaling in T cells

Justin A. Shyer, Richard A. Flavell and Will Bailis

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Correction to: *Cell Research* https://doi.org/10.1038/s41422-020-0379-5, published online 24 July 2020

We apologize for an error we discovered in the review published online on 24 July 2020. The sentence discussing SERCA channel regulation by phosphoenolpyruvate (PEP) used wording that conflated the effect of thapsigargin and knockdown of enolase 1 on NFAT1 nuclear localization. In addition, the portion of the illustration in Fig. 1 on this topic was unclear. The corrected sentences and Fig. 1 are provided below.

The original sentence is "Inhibiting SERCA channels with thapsigargin or knockdown of the enzyme responsible for PEP production, enolase 1 (Eno1), is sufficient to restore calcium signaling and translocation of the Ca^{2+}-activated transcription factor NFAT1 to the nucleus in T cells with impaired glycolysis." The corrected one is "Inhibiting SERCA channels with thapsigargin is sufficient to restore calcium signaling and translocation of the Ca^{2+}-activated transcription factor NFAT1 to the nucleus in T cells with impaired glycolysis. Reciprocally, knockdown of the enzyme responsible for PEP production, enolase 1 (Eno1), impairs NFAT1 nuclear localization."

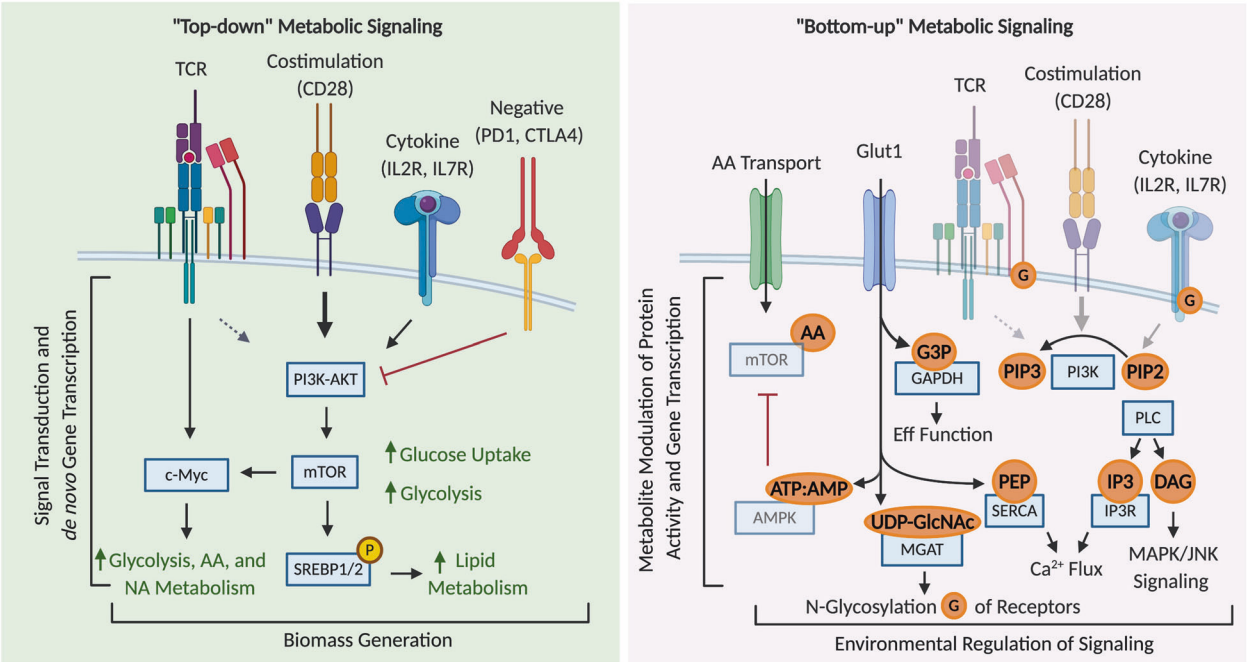

Fig. 1 "Top-down" vs. "bottom-up" metabolic signaling during T cell activation. "Top-down" signaling regulates the programming of T cell metabolism downstream of ligation of the TCR, co-stimulation, and cytokine signaling. Key metabolic regulators are engaged to meet the bioenergetic demands of effector T cells. Signal transduction pathways and de novo gene transcription lead to increased transcription and activation of mTOR and c-Myc, two master regulators of anabolism. mTOR and c-Myc are required to increase glucose uptake and metabolism. c-Myc is also critical for increasing amino acid (AA) and nucleic acid (NA) metabolism. mTOR activates increased lipid metabolism through SREBP1/2. "Bottom-up" signaling refers to metabolite regulation of signaling effectors. Increased rates of glucose and amino acid uptake and metabolism lead to the generation of metabolites that modulate the activity of several key signaling effectors, a process termed bottom-up metabolic signaling. Levels of glycolytic intermediates alter the activity of RNA-binding proteins, regulate posttranslational glycosylation, and activate the key metabolic regulator AMPK. Amino acid metabolism and uptake regulate mTOR activity though multiple mechanisms. Lipid species regulate the activity of several key signaling effectors of T cell activation.

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