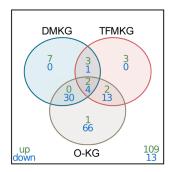
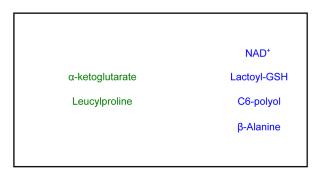
SUPPLEMENTARY FIGURES

A Complete Medium





DMKG TFMKG

DMKG TFMKG

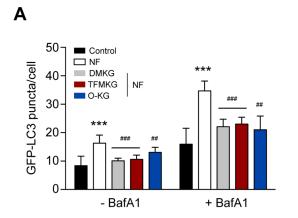
16
16
7
0
7

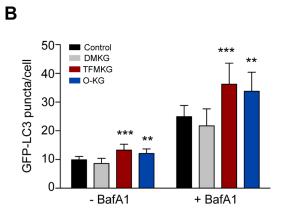
O-KG

29 48

ſ	α-ketoglutarate	Inositol	NADH
١	2-hydroxyglutarate	P Cae(15:0)	NADPH
١	Sphingosine	P Cae(16:0)	IVADEII
١	N1-Methyladenosine	P Cae(16:1)	Cytidine
١	Succinic acid	P Cae(18:0)	Pyruvate/OAA
١	Sarcosine	P Cae(18:1)	Heptanoic acid
١	Glutamylleucine	P Cae(20:4)	
	S-Adenosylmethionine	P Cae(22:6)	Caproic acid

Supplementary Figure 1 (related to Figure 1). Shared vs. private effects of α -ketoglutarate precursors on intracellular metabolome. Venn diagrams depicting the number of metabolites (FDR < 0.1) up- or down- regulated upon administration of α -ketoglutarate precursors in complete medium (A, left panel) or nutrient free medium (B, left panel). Metabolites that are convergently modulated (p < 0.05) by the three compounds are indicated in A and B (right panels).





Supplementary Figure 2 (related to Figure 2). Modulation of autophagy by α -ketoglutarate precursors in H4 cells. (A) Inhibition of starvation-induced autophagy by DMKG, TFMKG and O-KG. H4 cells stably expressing the autophagic markers GFP-LC3 were incubated in HBSS medium (NF) and left untreated or incubated with α -ketoglutarate precursors. Co-treatment with Bafilomycin A1 (BafA1) was used to assess autophagic flux. Data represent mean \pm S.D. (one representative experiment, n=3). *** p < 0.001 compared to Control; *** p < 0.001 compared to NF (unpaired t test). (B) Induction of autophagy by TFMKG and O-KG, but not DMKG, in complete medium. *** p < 0.01, **** p < 0.001 compared to Control (unpaired t test).