Metabolic requirements for cancer cell proliferation

Supplementary Figures

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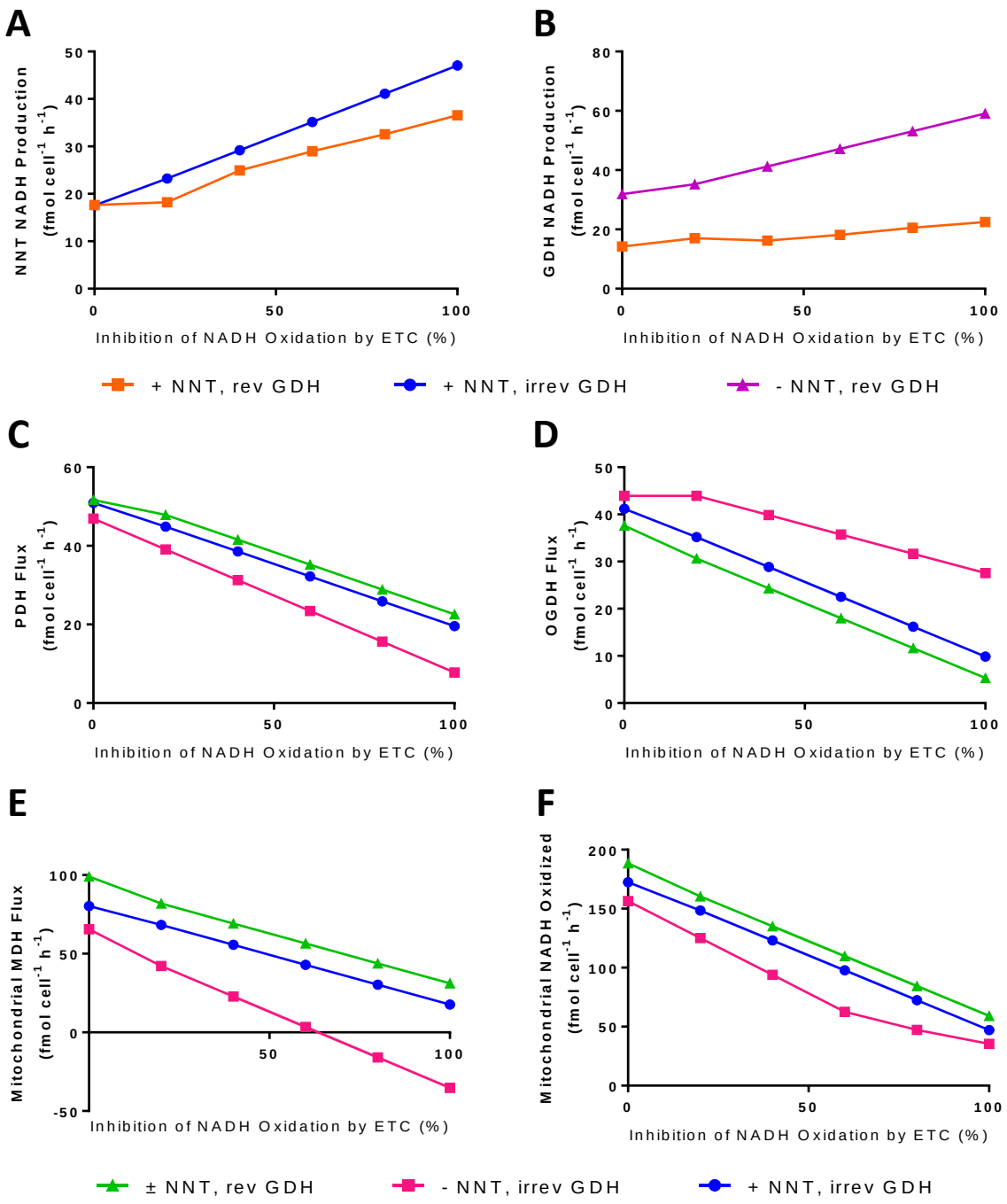

Figure S1. Mitochondrial NADH consumption and production flux predictions as functions of ETC inhibition for different nicotinamide nucleotide transhydrogenase (NNT) and glutamate dehydrogenase (GDH) conditions. (A) NADH production from NNT when GDH is either reversible (rev GDH) or irreversible (irrev GDH). NNT is included in both models. (B) NADH production from GDH when NNT is either included (+ NNT) or absent (- NNT). GDH is reversible in both models. (C) pyruvate dehydrogenase (PDH) flux, (D) oxoglutarate dehydrogenase (OGDH) flux, (E) mitochondrial malate dehydrogenase (MDH) flux, and (F) total mitochondrial NADH oxidized for different models. Both NNT-present and NNT-absent conditions gave effectively the same values when GDH was reversible (± NNT, rev GDH).

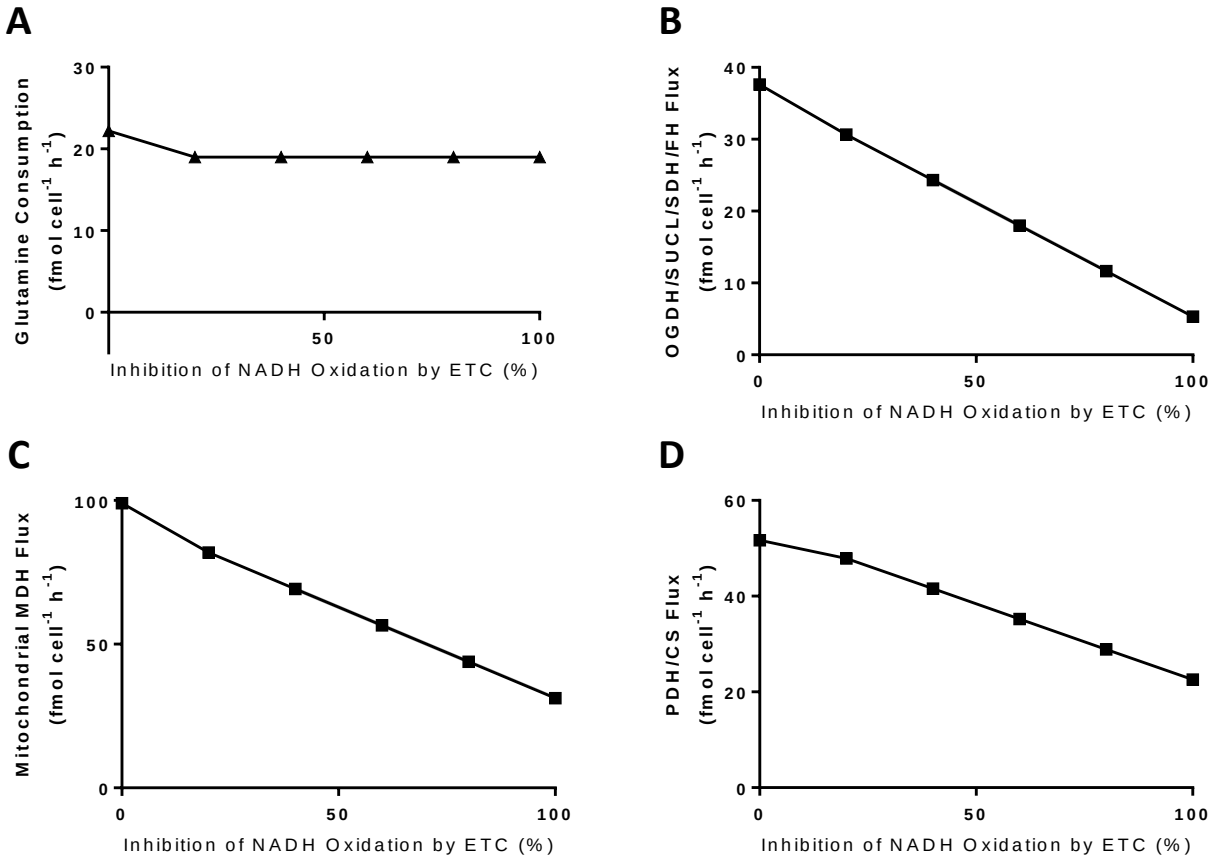

Figure S2. Additional metabolic flux alterations predicted to occur in response to ETC inhibition in simulations of metformin treatment. (A) Glutamine consumption; (B) oxoglutarate dehydrogenase (OGDH), succinate-CoA ligase (SUCL), succinate dehydrogenase (SDH), and fumarate hydratase (FH) flux; (C) mitochondrial malate dehydrogenase (MDH) flux; and (D) pyruvate dehydrogenase (PDH) and citrate synthase (CS) flux as functions of percent NADH oxidation inhibition.