Targeted Metabolomic Profiles of Serum Amino Acids and Acylcarnitines Related to Gastric Cancer

Running title: Metabolomic Profiles in Gastric Cancer

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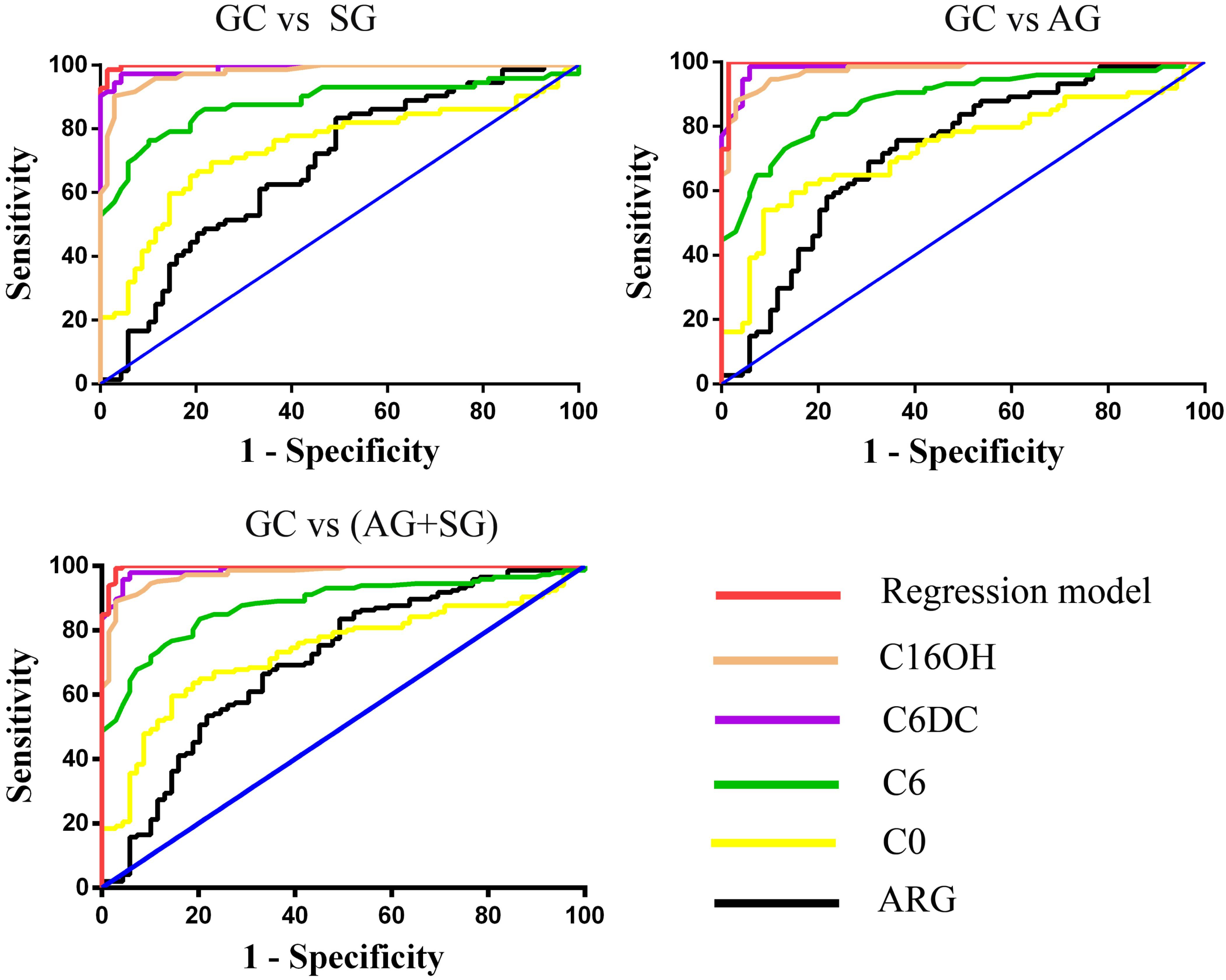
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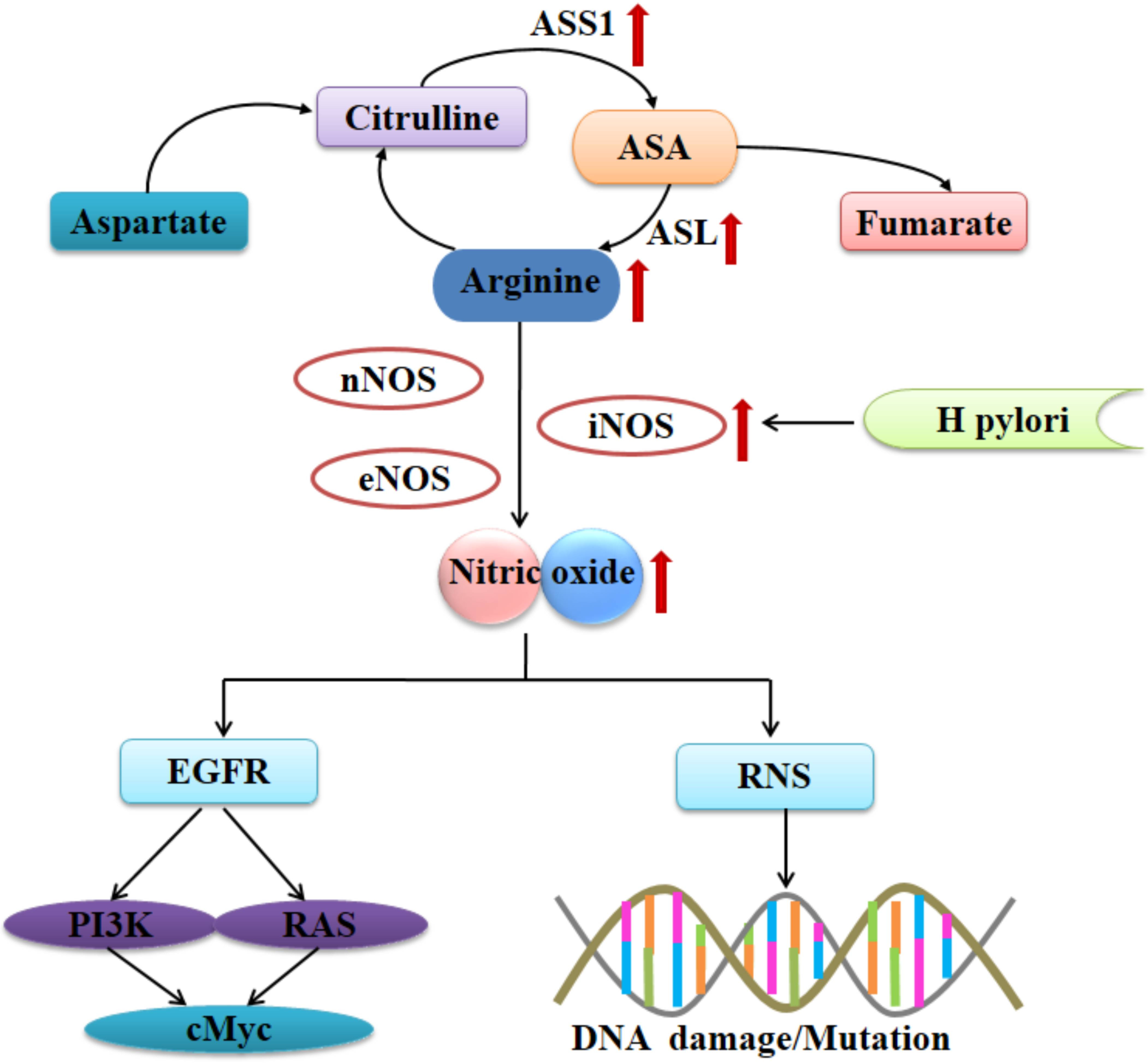
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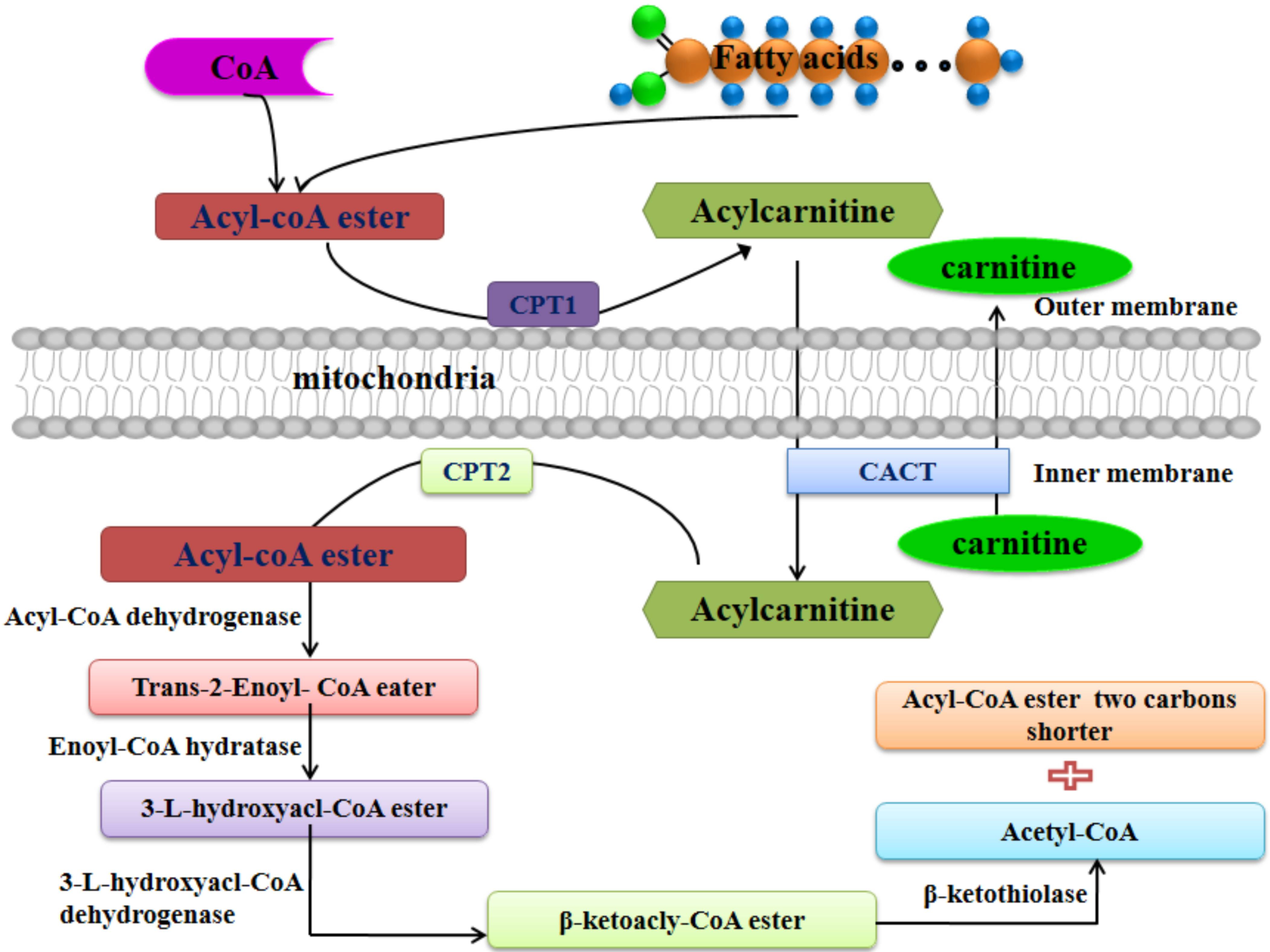


**Figure.S1.**Receiver operating characteristic (ROC) curves for five metabolites and the regression model. AUC, area under the curve.



**Figure.S2.Molecular mechanisms of arginine involvement in gastric cancer**. Red arrows denote cancer-related upregulation of proteins contributing to arginine metabolism, causing a net increase in nitric oxide (NO) synthesis. Cancer cells showed enhanced NO amounts by upregulating iNOS, ASS1 and ASL, which increase arginine availability for nitric oxide synthesis.

ASA, argininosuccinic acid; ASL,argininosuccinatelyase; ASS1, argininosuccinate synthase 1; NOS:nitricoxide synthase;RNS:reactive nitrogen species



**Figure.S3.Mitochondrial fatty acid β-oxidation.** Upon transport across the cytosolic membrane, fatty acids are transformed into acyl-CoAs in the cytosol. CPT1 transforms acyl-CoAs into respective acylcarnitines, which are then transported across the mitochondrial membrane by CACT. CPT2 converts the seacylcarnitines back into acyl-CoAs, which are degraded into acetyl-CoAs via an inducible set of enzymes.

CPT:carnitinepalmitoyltransferase; CACT: carnitine-acylcarnitinetranslocase