Perturbative metabolic fluxes in CK2-overexpressed colon cancer cells resolved by carbon labeling experiment.

<u>박민호</u>, 임대균, 양경미¹, 김건홍¹, 오민규[†]

1Department of Chemical & Biological Engineering Korea University, Seoul 136-713, Korea;

12Department of Biochemistry and Molecular Biology, Yonsei University College of Medicine, Seoul, Republic of Korea

(mkoh@korea.ac.kr[†])

CK2 is a serine/threonine kinase that is constitutively active in multiple cancer cells. Previously, we reported that stable increase of CK2 in cancer cells could induce EMT. Using colon cancer cell lines such as HT29 and SW620, CK2 also induced EMT and the transitioned cells became more proliferative than control cells. We assumed that CK2 could affect cancer cell growth by modulating energy metabolism. Therefore, we examined reprogrammed metabolic fluxes of CK2 on glucose metabolism. To measure intracellular metabolite, we fed U-13C6 glucose, U-13C5 glutamine to glucose, glutamine free DMEM respectively, and cultured until cells reached to metabolic steady state. The results of isotope analysis showed that continuous activation of CK2 in colon cancer cell lines facilitates the Warburg effect and anaplerotic pathways. This study suggests that because oncogenes regulate important metabolic enzymes and metabolism signaling pathways, CK2 is excellent metabolic target for cancer therapy.