

Supplemental Information

Vitamin C Suppresses Pancreatic Carcinogenesis through the Inhibition of Both Glucose Metabolism and Wnt Signaling

Ji Hye Kim [†], Sein Hwang [†], Ji-Hye Lee, Se Seul Im and Jaekyoung Son *

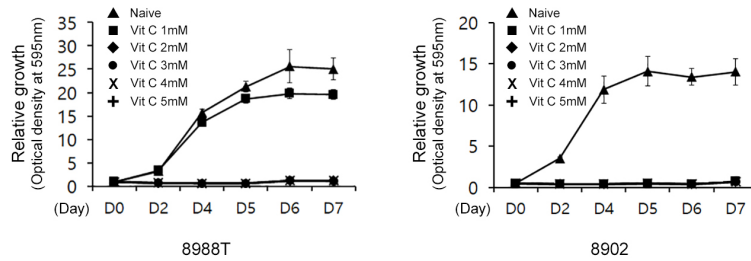
Department of Biomedical Sciences, Asan Medical Center, AMIST, University of Ulsan
College of Medicine, Seoul 05505, South Korea

* Correspondence: jaekson@amc.seoul.kr

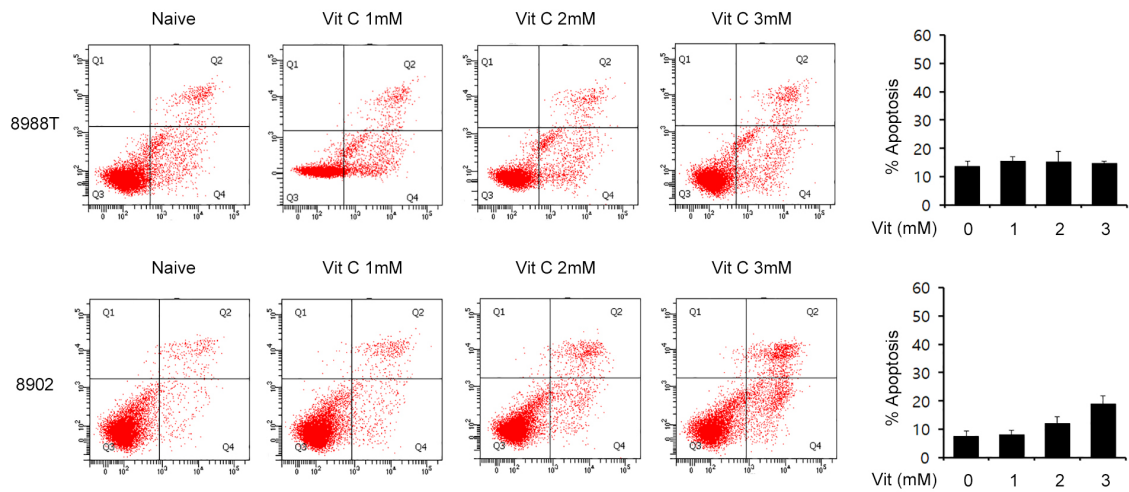
[†] These authors contributed equally to this work.

Supplementary Figure S1

A

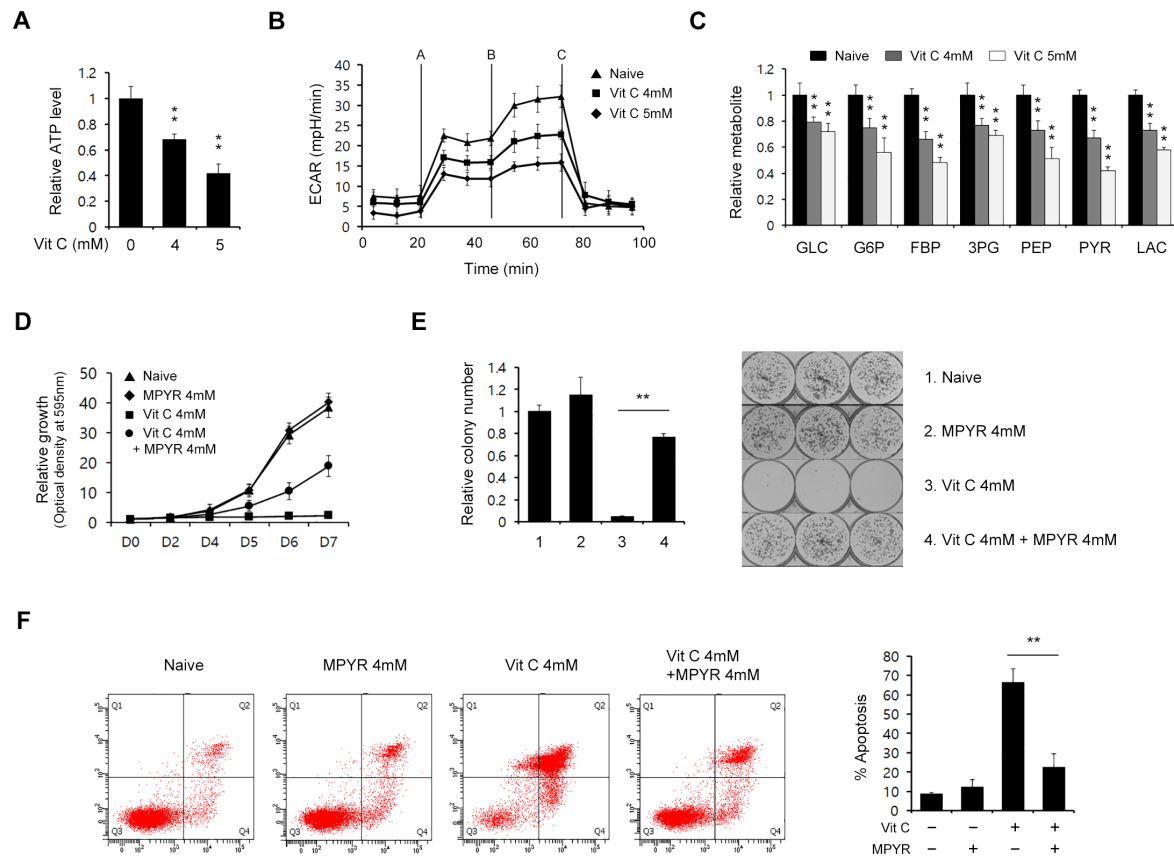


B



Supplementary Figure S1. Anti-tumor effects of vitamin C in PDAC cells. **(A)** Growth assay for PDAC cells treated with vitamin C for the indicated days. **(B)** PDAC cells were treated with vitamin C for 24 h and assayed for apoptotic cell death by annexin V/PI staining and flow cytometry. Error bars represent s.d. of triplicate wells from a representative experiments.

Supplementary Figure S2



Supplementary Figure S2. Effect of high-dose vitamin C on glycolytic flux. **(A)** 8902 cells were treated with vitamin C (4 or 5 mM) for 24 h and assayed for intracellular ATP. **(B)** 8902 cells were treated with vitamin C (4 or 5 mM) for 24 h and the extracellular acidification rate was measured. **(C)** 8902 cells were treated with vitamin C (4 or 5 mM) for 24 h and analyzed for glycolysis metabolite pools via LC/MS-MS. **(D)** Cell growth assay for 8902 cells treated with vitamin C (4 mM) for 8 days with or without methyl-pyruvate (4 mM). **(E)** Clonogenic assay for 8902 cells treated with vitamin C (4 mM) for 8 days with or without methyl-pyruvate (4 mM). **(F)** 8902 cells were treated with vitamin C (4 mM) for 8 days with or without methyl-pyruvate (4 mM) and assayed for apoptotic cell death by annexin V/PI staining and flow cytometry. Error bars represent s.d. of triplicate wells from a representative experiment; ** $p < 0.01$.