Supplementary Figure 1. (related to Figure 1). Quantification of the cell nuclei size. (A) The cell nuclei size was quantified with Image J software.

Supplementary Figure 2. (related to Figure 1). Correlation of key SASP factor, IL-8 with CPT1C expression in pancreatic cancer patients. (A) IL-8 negatively correlates with CPT1C mRNA expression in pancreatic cancer patients.
Supplementary Figure 3. (related to Figure 2). Metabolomics profiles reveal significantly decreased acylcarnitines in senescent vector PANC-1 cells. (A) PCA score plots of HILIC-ESI-MS metabolomics profiles obtained from HILIC-ESI-MS, n = 6/group. (B) S-plot of OPLS/DA models of HILIC-ESI-MS data. (C–F) Validation of cell metabolite biomarkers by LC-MS/MS analysis. Corresponding MS/MS spectra of authentic chemicals (up) and cell samples (bottom) are shown.
Supplementary Figure 4. (related to Figure 3). Vector PANC-1 cells exhibited higher glycolytic function. (A) Glycolytic function in the forms of ECARs (mpH.min⁻¹) in senescent vector PANC-1 cells. Data are presented as the mean ± S.E.M, n = 3.

Supplementary Figure 5. (related to Figure 5). Cell sensitivity to metabolic stress. Cell sensitivity to metabolic stress from (A) glucose withdrawal and (B) glycolytic inhibition in senescent vector PANC-1 cells at the indicated concentrations is shown. Data are presented as the mean ± S.E.M, n = 5 (*p < 0.05, **p < 0.01).

Supplementary Figure 6. Reduced CDKN1A (P16) mRNA in the senescent vector PANC-1 cells. (A) Quantitative RT-PCR analysis for P16 was performed in the senescent vector PANC-1 cells.
Supplementary Figure 7. (related to Figure 6). Correlation of crucial signaling components of cellular senescence with CPT1C expression in pancreatic cancer patients. (A) Correlation analysis between PPARα, TP53, and CDKN1A with CPT1C mRNA expression in pancreatic cancer patients (data from the Collisson Pancreas cohort, n = 27).