Figure S1. Pancreatic cancer cell proliferation was measured using MTT assay. Data are presented as the mean ± SD (n=3). Data were analyzed using one-way ANOVA followed by Tukey's post-hoc test. VNN1, Vanin-1; PV, PANC-1 cells with the stable overexpression of VNN1; PE, PANC-1 cells transfected with empty vector; CV, CFPAC-1 cells with the stable overexpression of VNN1; CE, CFPAC-1 cells transfected with empty vector.

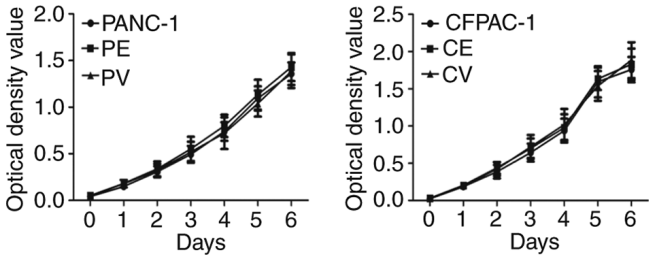

Figure S2. High-performance liquid chromatography analysis of cysteamine. (A) The mobile phase. (B) Mobile phase contained the standard sample of cysteamine. The peak with a retention time of 6.0 min stood for cysteamine. (C) The mobile phase contained the mixed conditioned medium (conditioned mediums of PV and CV cells were mixed together). Only one peak emerged prior to 6.0 min. (D) The mobile phase contained the mixture of mixed conditioned medium and cysteamine (standard). Only one more prominent peak emerged prior to 6.0 min, indicating that the peak prior to 6.0 min in panel C stood for cysteamine.

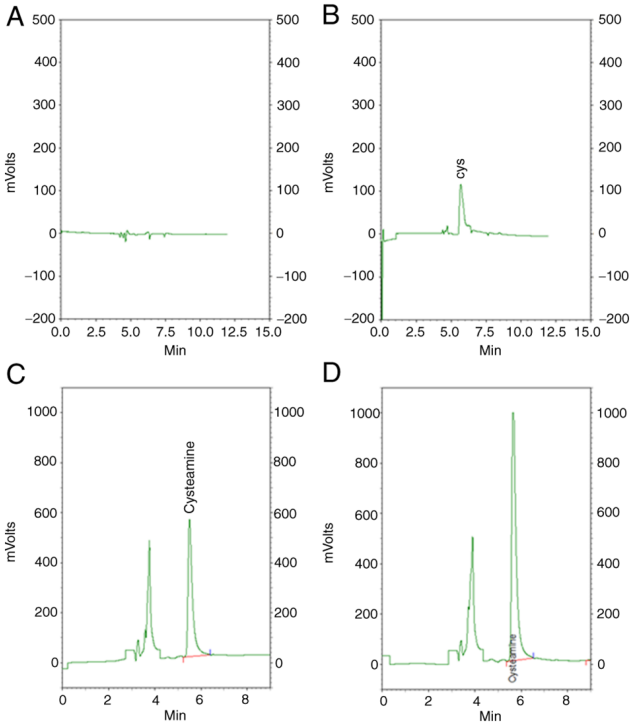

Figure S3. Schematic diagram illustrating the mechanisms through which VNN1 in PC cells inhibits the activity and function of paraneoplastic islets by inducing oxidative stress. (A) VNN1 hydrolyzes pantetheine to produce cysteamine in PC cells. (B) Intracellular cysteamine is secreted to extracellular. (C) Extracellular cysteamine is absorbed by β-cells. (D) Cysteamine increases the ROS levels, and decreases the GSH and PPARγ concentrations in β-cells. (E) The oxidative stress is increased in β-cells. (F) The viability and insulin secretion of β-cells are inhibited. PC, pancreatic cancer; VNN1, Vanin-1; ROS, reactive oxygen species; PPARγ, peroxisome proliferator-activated receptor gamma; GSH, glutathione.