Supplementary information

Supplementary Table 1: Median estimated for the tumor cell viability, tumor outgrowth length and tumor outgrowth viability index in the different treatment conditions.

Treatment condition	Tumor cell viability (%)(IQR)	Tumor outgrowth length (%)(IQR)	Tumor outgrowth viability index (IQR)
Control 72h	79.4 (16)	54.7(26.9)	154.0(31.2)
Gem 1 µM	76.1(23.9)	67.8(39.5)	170.0(85.5)
Se 5 µM	52.4 (42.9)	51 (22.7)	118.0 (76.6)
Se 15 µM	6.1 (18.2)	18.6 (18.9)	27.5 (34.7)
Se 30 µM	1.9 (1.1)	12.6 (18.9)	14.4 (16.4)

Supplementary Table 2: Functions of genes that were altered by sodium selenite treatment.

Gene ID (Log 2-fc)	Function of the protein	References
CEMIP (-2.75)	Increased expression degrades hyaluronan (HA) into small fragments which promote metastasis, cell survival, tumor progression, ECM remodeling, invasion, migration and promotes angiogenesis. Upregulation correlates with a poor prognosis. It also regulates β-catenin. Downregulation reduces the migration of cancer cells, EMT, β-catenin (reduces fibrosis) and proliferation.	[1, 2]
LRP1 (-2.24)	Promotes tumor migration, progression, invasion and metastasis by increasing MMP2 and MMP9 through the ERK pathway. Increases angiogenesis and inhibits apoptosis by regulating insulin receptors. Overexpression of this gene correlates with lymph-node invasion. It is a P53 target gene. Downregulation reduces cancer cell progression, metastasis and induce apoptosis.	[3]
SCD (-2.39)	It promotes tumor progression by increasing monounsaturated fatty acid (MUFA) level to maintain redox imbalance, suffice cancer cell energy needs and activates UPR in ER. Downregulation results in tumor growth inhibition, reduce progression, reduced MUFA level, phospholipid synthesis and induces apoptosis.	[4-6]
LAMB1 (-2.04)	Poor prognosis in various cancer, induce cell proliferation, mortality, migration, and invasion of the tumor via the ERK/c-Jun pathway. It activates MMP2 and regulates ECM remodeling. Downregulation results in reduced tumor cell proliferation, mortality, migration, metastasis and invasion.	[7]
PLOD2 (-2.39)	Induced by hypoxia (HIF-1α) and involved in ECM-fiber remodeling in the stroma of PDAC thereby increases the cancer cell morality. Promotes EMT and cancer progression. It correlates with poor prognosis in various cancer types. PLODs highly expressed in cancer-associated fibroblasts (CAFs). Downregulation block the desmoplasia (fiber remodeling) in the stroma, collagen deposition thereby reducing tumor migration and invasion. It also suppresses tumor metastasis and reduces CAFs.	[8, 9]
DDR2 (-2.1)	It is activated by fibrillar collagens and is largely expressed in mesenchymal cells. It helps in cancer progression by inducing tumor cell-collagen interactions via Akt-mediated signaling pathways. Downregulation reduces tumor growth, invasion, fibrillar collagen and metastasis.	[10]
P4HA1 (-1.65)	Induced by hypoxic (HIF-1α) condition and it involved in HIF-1α stability to promote tumor progression. It promotes metastasis, chemoresistance, glycolysis and regulates ECM remodeling (collagen). Upregulation correlates with a poor prognosis. Downregulation of this gene inhibiting tumor cell proliferation, reduce expression of stemness markers, sensitizes cells to chemotherapy and suppressed the glycolytic activity of PDAC cells.	[11]

RAB26 (2.5)	Loss of MIST1 function largely results in cells susceptible to pancreatitis. MIST1 upregulates RAB26 to regulate lysosome coalescence and redistributing mitochondria into neighborhood cells.	12, 13
ATF3 (2.27)	It is an adaptive response gene, which is a master regulator in metabolic and immune homeostasis. It impedes migration, invasion, induces inflammation and apoptosis by stabilizing pro-apoptotic genes such as STAT1 and Bax. It also downregulates MMP1 transcription in primary monocytes and macrophages. ATF3 can downregulate SCD1 and proinflammatory cytokines such as IL6 and IL-12b.Downregulation results in increased tumor initiation, progression, and remission because of imbalanced metabolic homeostasis. Increased proinflammatory cytokines such as IL6 and IL-12p40 following TLR activation. Suppress pro-apoptotic genes such as Bax and Bak.	14
FOSB (2.88)	Suppress cancer cell proliferation, migration, and clone formation. Prevent lymph node metastasis in PDAC. Reduced expression correlate with poor differentiation, lymph node metastasis and advanced TNM stage, poor prognosis, and patient survival. Induce proliferation, malignant transformation, migration and acquiring malignant potential in PDAC.	15, 16
GADD45B (2.22)	It has a Pro-apoptotic function which is involved in DNA damage repair, cell cycle arrest and normal cell survival. It increases phosphorylation of MAP2K3 and active P38 and P53. It plays a role in cell cycle checkpoint by inhibiting cdc2/cyclinB1 kinase and inhibit metastasis. Downregulation promotes metastasis and tumor cell progression.	5, 17
PDK1 (-1.67)	It can phosphorylate several kinases pathways to induced tumorigenesis such as PI3K, Akt, SGK3, etc. It is important for T-cell receptor signaling to convey its signal for maturation and migration of endothelial cells, neutrophil chemotaxis and angiogenesis. Downregulation results in reduced cancer progression, invasion, migration, metastasis, angiogenesis, anchorage-dependent and anchorage-independent growth in PDAC and several other cancer types. It sensitizes the tumor cells to chemotherapy.	18, 19
PIK3AP1 (-1.62)	It is the upstream regulator of the PI3K/Akt/c-Myc pathway. It induces tumor progression and chemoresistance. It is important for tumor cell proliferation, metabolism and survival. Downregulation inhibits tumor progression and sensitize the cells to chemotherapy.	20
ZNF395 (-1.53)	It is induced by hypoxic conditions and increases the survival of PDAC. It promotes inflammation-mediated cancer progression via NFkB and HIF pathways. It induces IFNα stimulating genes. Downregulation reduces IFNα-mediated stimulating of innate immune response and reduces cancer cell progression.	21, 22
CSGALNACT1 (-1.33)	It promotes adhesion, invasion, metastasis, migration, EMT, angiogenesis, apoptosis, and ECM remodeling by increasing heparan sulfate (HS) and chondroitin sulfate (CS). It is also involved in hyaluronan synthesis which promotes tumor proliferation and migration. Downregulation of this gene inhibit metastasis, cell proliferation, EMT, invasion and migration	23
CD14 (-2.57)	In tumor cells with high CD14 is important in TLRs signaling pathways which stimulates tumor microenvironment for proliferation and progression. It also helps in IL6 production and is highly vascularized with myeloid cell infiltration and retention. It produces inflammatory factors that activate angiogenesis to maintain an immune-suppressive environment for tumor cells.	24
RAB31 (-1.57)	Increased RAB31 expression induces uncontrolled proliferation, tumor development, migration, and metastasis through ERK1/2 and PI3K/Akt pathways. Poor prognosis for PDAC patients.	25
SRPX2 (-1.82)	Considered an oncogene that promotes proliferation, migration and invasion of cancer cells. Poor prognosis for PDAC patients.	25

ACHE (1.72)	A low level of ACHE revealed a poor prognosis in HCC. ACh (acetylcholine) hyper-activate nAChRs (nicotinic acetylcholine receptor) to induce angiogenesis, migration, and proliferation of tumor cells. Increased expression induces apoptosis and inhibits cancer proliferative pathways such as PI3K/Akt (cancer stem cell maintaining pathway). Higher expression showed longer survival in patients. ACh level is regulated by AChE protein.	[26]
MAFA (3.39)	Downregulated in various cancer type. Expressed in β-cells and it is the positive indicator of health and function. It activates insulin transcription in high glucose conditions.	[27]
DUSP8 (1.97)	Attenuate the ERK/Akt pathway thereby reducing the tumor proliferation and migration in vivo. DUSP controls the MAPK pathway. DUSP8 might be a tumor suppressor gene.	[28, 29]

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