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Cucurmosin induces the apoptosis of human pancreatic cancer CFPAC-1 cells by inactivating the PDGFR-β signalling pathway

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Abstract:
Background: Pancreatic cancer treatment is limited and effective drugs are needed. We investigated cucurmosin (CUS)-induced apoptosis in cystic fibrosis pancreatic adenocarcinoma cells (CFPAC-1) and a possible mechanism of action to evaluate the clinical application potential of this new Type I ribosome-inactivating protein.
Methods: We analyzed the growth inhibition and apoptosis of CFPAC-1 cells *via* methylthiazol tetrazolium assay and fluorescence-activated cell sorting. Western blot was used to analyze the protein levels of caspase 3, bcl-2, caspase 9, platelet-derived growth factor receptor (PDGFR)-β, PI3K, Akt, p-Akt, the mammalian target of rapamycin (mTOR), p-mTOR, P70S6K-α, p-P70S6K-α, 4E-BP1, p-4E-BP1 and p-Bad after CUS intervention. The mRNA expression of PDGFR-β was analyzed using reverse transcription polymerase chain reaction.
Results: CUS inhibited the proliferation of pancreatic cancer cells. The induction of apoptosis depended on the CUS dose and incubation time. The drug inhibited all of the examined proteins in the PI3K/Akt/mTOR signalling pathway and induced the active fragments of caspase 3 and caspase 9. CUS downregulated PDGFR-β expression but no significant change was observed at the mRNA level.
Conclusion: CUS strongly inhibits the growth of CFPAC-1 by inducing cell apoptosis. CUS downregulated the expression of PDGFR-β at the protein level and induced the apoptosis of CFPAC-1 through the PI3K/Akt/mTOR signalling pathway.

Key words:
cucurmosin, pancreatic carcinoma, PDGFR, PI3K/Akt/mTOR, apoptosis

# These authors contributed equally to this work