Human breast carcinoma cells are induced to apoptosis by samsam ant venom through an IGF-1-dependant pathway, PI3K/AKT and ERK signaling

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Abstract:

In the present study we evaluated the anti-tumor potential of samsam ant venom (SAV) from Pachycondyla sennaarensis on the human breast carcinoma cell line MCF-7. We found that SAV induced growth arrest of MCF-7 cells without affecting the viability of MCF-10 (non-tumorigenic normal breast epithelial cells) and normal PBMCs. We then analyzed its impact on IGF-1-mediated MCF-7 cell proliferation and its effect on the underlying IGF-1 signaling pathways. Using flow cytometry analysis, we showed that the percentage of apoptotic cells was fourfold higher in SAV-treated cells as compared to untreated cells. More importantly, treatment with SAV induced a marked reduction in actin polymerization and a subsequent marked reduction in IGF-1-mediated cell proliferation. In addition to growth-inhibitory and proapoptotic effects, significant reductions were also observed in the phosphorylation of AKT and ERK, but not p38MAPK, in SAV-treated cells as compared to untreated cells. Our data reveal unique anti-tumor effects of samsam ant venom.

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