

*Poster Abstracts*

Biology / Epidemiology 8

**Decrease of PKC alpha expression by a TAT-fused peptide in human triple-negative breast cancer cells**

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**Background:** It is known that PKCN1 is involved in many human diseases and modulate its expression may improve the symptom.

**Methods/Aim:** Because PKCN1 was regulated by the MZF-1/Elk-1 heterodimers, we selected a peptide matching a peptide from the fragment of myeloid zinc finger 1 (MZF-1), as it is the binding site for Elk-1. Saturation of the Elk-1 binding site with this peptide decreases the chances of endogenous MZF-1/Elk-1 binding and decreases the number of endogenous MZF-1/Elk-1 heterodimers binding to the PKCN1 promoter.

**Results:** When we transfect this peptide to triple-negative breast cancer (TNBC) cells, PKCN1 expression, as well as cell migration and tumorigenicity, decreased. Through micro-array and western blotting analysis. We found that the EMT-related genes were also altered. Moreover, using TAT-fused peptide method to deliver the peptide directly to cells, similar results were found in TNBC cells, but not found in lung and bladder cancer cells.

**Conclusions:** We suggest that the TAT-fused peptide, which is derived from a fragment of MZF-1, can inhibit PKC alpha expression and reduce cancer cell migration and tumorigenicity, particularly in human triple-negative breast cancer cells.