專利/技術名稱	一種具有抑制MZF-1與Elk-1交互作用之醫藥組合物 A pharmaceutical composition inhibiting interaction between MZF-1 and Elk-1		
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可利用範圍	提供一種新穎的胜肽，可以抑制內生性Elk-1或MZF-1表現，亦會中斷Elk-1與MZF-1的交互作用，減少蛋白激酶Cα表達，抑制癌細胞生長。		
摘 要	本發明是一種胜肽，特別是會中斷Elk-1與MZF-1交互作用，抑制癌症之胜肽。本發明在癌細胞發現MZF-1或Elk-1表現與PKCα的關聯性，MZF-1與Elk-1之交互作用位置為MZF-1之酸性區域與Elk-1之肝素結合區域結合，形成異二聚體後會與PRKCA啟動子結合，影響Elk-1入核、MZF-1蛋白質降解、DNA結合活性、PKCα表現，影響癌細胞之遷移、腫瘤形成、降低上皮細胞中胚轉化(epithelial-mesenchymal transition；EMT)。經由提供MZF-1胜肽、Elk-1胜肽或以TAT溶合胜肽表現MZF-1或Elk-1，可以佔據MZF-1或Elk-1之結合區域，中斷MZF-1與Elk-1之交互作用以治療癌症。 This invention discloses a peptide, which inhibits the interaction between of MZF-1 and Elk-1 and further inhibits cancers. Both myeloid zinc finger 1 (MZF-1) and Ets-like protein-1 (Elk-1) expressions correlate to PKCα expression in cancer cells. Furthermore, it is the interaction between the acidic domain of MZF-1 and the heparin-binding domain of Elk-1 which facilitated their heterodimeric complex formation before their binding to the PKCα promoter. Blocking the formation of the heterodimer changed Elk-1 nuclear localization, MZF-1 protein degradation, their DNA-binding activities, and subsequently the expression of PKCα in cancer cells. Thus, migration, tumorigenicity, and epithelial-mesenchymal transition potential of cancer cells decreased, suggesting that the Elk-1/MZF-1 heterodimer is considered as a mediator of PKCα in TNBC cell malignancy. The obtained data also suggest that the next therapeutic strategy in the treatment of cancer will come from the blocking of Elk-1/MZF-1 interaction through the saturation of Elk-1 or MZF-1 binding domains, such as through the application of cell-penetrating HIV transactivating regulatory protein-fused peptides.		