

## 论文 扇贝多肽经由aSMase-JNK通路抑制UVA诱导HaCaT细胞凋亡

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### 摘要:

建立紫外线A(UVA)辐射损伤HaCaT细胞的病理模型, 从酸性鞘磷脂酶-JNK信号通路的角度研究扇贝多肽(Polypeptide from *Chlamys farreri*, PCF)抑制UVA诱导HaCaT细胞凋亡的分子机制. 采用Hoechst 33258染色结合琼脂糖凝胶电泳分析细胞凋亡; 用RT-PCR法和细胞免疫荧光染色检测胞内酸性鞘磷脂酶(acid sphingomyelinase, aSMase)的表达; 蛋白印迹法检测细胞内JNK及磷酸化JNK的蛋白水平. 结果表明, PCF可明显地抑制UVA诱导的HaCaT细胞凋亡; aSMase抑制剂Desipramine和JNK抑制剂SP600125均可阻断UVA引起的细胞凋亡; PCF的浓度在1.42~5.68 mmol/L范围内可依赖性地抑制UVA辐射后细胞内aSMase的表达量以及JNK蛋白的磷酸化; 预先加入Desipramine则抑制UVA引起的JNK蛋白的磷酸化. 表明PCF通过阻断aSMase-JNK通路来抑制UVA诱导HaCaT细胞凋亡.

关键词: 扇贝多肽 酸性鞘磷脂酶 JNK 紫外线A 凋亡 HaCaT细胞

## Polypeptide from *Chlamys Farreri* Inhibits UVA-induced Apoptosis of HaCaT Cells Involving of aSMase-JNK Pathway

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### Abstract:

A pathological model of UVA-induced HaCaT cells was established to investigate whether polypeptide from *Chlamys farreri*(PCF) protects HaCaT cells from apoptosis induced by UVA through acid sphingomyelinase(aSMase)-JNK pathway and to explore its related molecular mechanism. Apoptosis of cells were determined by Hoechst 33258 staining and agarose gel electrophoresis. The expression of aSMase in HaCaT cells was detected via RT-PCR and immunofluorescence. Using Western Blot analysis, expression levels of JNK and phosphorylated JNK were assayed. The results showed that PCF can significantly protect against UVA-induced apoptosis of HaCaT cells. PCF can also inhibit expression of aSMase and phosphorylated of JNK in HaCaT cells radiated by UVA in a dose-dependent manner. ASMase inhibitor Desipramine and JNK inhibitor SP600125 had inhibitory effects on UVA-induced apoptosis, the former blocked phosphorylated of JNK. So it is concluded that PCF can protect HaCaT cells from apoptosis induced by UVA and its protective effects may attribute to blocking aSMase-JNK pathway.

Keywords: Polypeptide from *Chlamys farreri*(PCF) Acid sphingomyelinase(aSMase) JNK Ultraviolet A Apoptosis HaCaT cell

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