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Editorial Open Access

Anticancer Activity and Mechanism of Alpha-Helical Antimicrobial Peptide

Yibing Huang*

Key Laboratory for Molecular Enzymology and Engineering of the Ministry of Education, College of Life Science, Jilin University, China

***Corresponding author:** Yibing Huang, Associate Professor at Key Laboratory for Molecular Enzymology and Engineering of the Ministry of Education, College of Life Science, Jilin University, China, Tel: 86-431-8515-5245; E-mail: huangyibing@jlu.edu.cn

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Editorial

Anticancer peptides (ACPs) have become promising molecules as new anticancer agents due to the unique mechanism and several extraordinary properties, such as smaller size, high activity, low immunogenicity, good biocompatibility, etc. [1].

HPRP-A1 is an 15 amino acid residues antimicrobial peptide (AMP), derived from the N-terminus of ribosomal protein L1 (RpL1) of *Helicobacter pylori*. It can be induced to form an amphipathic α-helical structure in hydrophobic medium and exhibited good antimicrobial and antifungal activity [2,3]. Based on the mechanism of antimicrobial peptides, they can descript the integrit of cell membrane by the electrostatic interactions and hydrophobic interaction. HPRP-A1 has been also found that can induce HeLa cell apoptosis by both the extrinsic and intrinsic pathways of the caspase cascade [4]. In order to improve the penetrability and specificity against cancer cell lines, a new hybrid peptide of HPRP-A1-TAT has been designed and synthesized by linked a cell-permeating peptide TAT to the C-terminus of HPRP-A1 [5]. Compared to HPRP-A1, the hybrid peptide of HPRP-A1-TAT exhibited higher anticancer activity against HeLa cells with lower toxicity against human RBC by increasing early apoptosis of HeLa cells and inducing caspase activity. Furthermore, we explored combinational anticancer therapy using HPRP-A1 with the chemical drugs doxorubicin (DOX). The data showed that the anticancer activity of these drugs against HeLa cell lines was synergistically increased both *in vitro* and *in vivo* [6]. We think this strategy of combination therapy appears to have great clinical potential.

Recently, we are working on the targeting modification of HPRP-A1 and the polymer modification to improve the selectivity and stability. We believe that ACPs as novel anticancer drugs will be play an important role for the clinical practices.

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