IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

Evaluation Of Caspase-3 Expression In A549 Lung Cancer Cells Treated With AXA Electrohomeopathic Product: A Flow Cytometry-Based Study

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Abstract

Apoptosis is a vital mechanism in cancer therapy. Caspase-3, a crucial executioner enzyme, serves as a marker for apoptotic activity. This study aimed to evaluate the apoptotic effect of AXA, an electrohomeopathic formulation, on human lung adenocarcinoma (A549) cells using flow cytometry. Cells were treated with AXA (1% v/v) and compared with Cisplatin (10 µg/mL) for 24 hours. AXA treatment significantly elevated Caspase-3 activation (81.1 ± 0.5%) relative to control (3.5 ± 0.2%), comparable to Cisplatin (83.8 ± 0.3%). The mean fluorescence intensity (MFI) also increased approximately 4.8-fold versus control. These results indicate that AXA induces Caspase-3-mediated apoptosis effectively in A549 cells, suggesting its potential as a complementary anti-cancer agent.

Keywords: Apoptosis, Caspase-3, Electrohomeopathy, AXA, Flow Cytometry, A549, Cisplatin

1. Introduction

Cancer remains a major cause of death globally. Conventional chemotherapeutic drugs such as Cisplatin are effective but limited by their cytotoxic side effects. Apoptosis, a programmed cell death process, provides a controlled mechanism for eliminating cancer cells. Caspase-3 is one of the principal effectors in this process. Electrohomeopathic preparations like AXA are believed to act through bio-potentized natural compounds with lower toxicity. This study evaluates AXA's potential to activate Caspase-3 and induce apoptosis in A549 lung cancer cells.

2. Materials and Methods

A549 human lung adenocarcinoma cells were cultured in DMEM containing 10% FBS and 1% antibiotics, maintained at 37°C and 5% CO_2. Cells were divided into three groups: Control (untreated), Cisplatin (10 µg/mL), and AXA (1% v/v). After 24 hours of treatment, cells were fixed, permeabilized, and stained with FITC-anti-active Caspase-3 antibody (BD Biosciences, Cat. No. 559341). Flow cytometry analysis was performed on a Cytomics FC500 instrument and analyzed using FlowJo software.

3. Results

Treatment	MFI (Mean ± SD)	% Caspase-3+ (± SD)	Fold Change	p-value
Control	2604 ± 16	3.45 ± 0.25	1.00	-
Cisplatin (10 µg/mL)	13445 ± 96	83.8 ± 0.3	5.15	<0.001
AXA (1% v/v)	12564 ± 89	81.1 ± 0.5	4.83	<0.001

4. Discussion

The increased Caspase-3 activity confirms apoptosis induction by AXA, comparable to Cisplatin's effect. These results demonstrate that AXA may offer an effective, low-toxicity alternative in cancer management. Further mechanistic and animal model studies are needed to validate these findings.

5. Conclusion

AXA, developed by AERC India, activated Caspase-3-mediated apoptosis in A549 lung cancer cells. These findings highlight AXA's promise as a complementary anti-cancer formulation requiring additional preclinical testing.

6. References

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Acknowledgement

The authors thank AERC India, Mumbai, for laboratory facilities and technical assistance.

Funding

This research was supported internally by AERC India, Mumbai.

Conflict of Interest

The authors declare no conflict of interest.

Ethical Statement

No human or animal subjects were used; only authenticated A549 cell lines were employed.