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ORIGINAL ARTICLE

Tuber Lectin Extracted from *Xanthosoma violaceum* (XVL) Promotes Apoptosis and Suppresses Proliferation in A549 Lung Adenocarcinoma Cells

G T Ramya[1], E Sarathkumar[1], M Meghana[1], N Anitha[1], S J Prashanth[2], G J Sathisha[1*]

[1]*Department of Postgraduate Studies and Research in Biochemistry, Jnana Sahyadri, Kuvempu University, Shankaraghatta, Shivamogga- 577 451, Karnataka, India*

[2]*Department of Postgraduate Studies and Research in Food Technology, Jnana Sahyadri, Kuvempu University, Shankaraghatta, Shivamogga- 577 451, Karnataka, India*

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** Corresponding author.*
G J Sathisha
satishlec@gmail.com

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ABSTRACT

Background: Lung cancer accounts for major cancer related mortality worldwide. Among the subtypes, non-small cell lung cancer (NSCLC) considered to be the most prevalent subtype. Due inferiority of current treatment strategies such as toxicity, non-selectivity, high cost and resistance emphasizes the exploration for selective and safer anticancer agents. In view of their recognition of aberrant carbohydrate ligands on the cancer cell surface, various plant lectins including *Xanthosoma violaceum* lectin (XVL) exhibit antiproliferative and apoptosis-inducing activities. Despite this, its effects on lung cancer cells remain insufficiently investigated. **Methods:** *Xanthosoma violaceum* lectin (XVL), which was previously purified and characterized, was used to investigate for antiproliferative activity against human lung adenocarcinoma A549 cells and normal embryonic kidney cells (HEK293) were used as control. MTT assay, an *in vitro* assay, has been employed to check the cell viability. Varying concentrations of XVL (0.31–10 µg/100 µL) was used to treat the cells. Prolonged cytotoxicity and cell migration were investigated by clonogenic and wound healing assays respectively. The caspase-3, -8, and -9 activity, flow cytometry, and comet assays were adapted to induction of apoptosis, cell cycle arrest and DNA fragmentation respectively. **Results:** The cell viability of A549 was significantly reduced by XVL in a dose-dependent manner with an IC_{50} of 6.508 µg/100 µL. However, it exhibits minimal toxicity toward HEK293 cells. Colony formation and cell migration were markedly inhibited. G_2/M phase cell cycle arrest along with a moderate increase in Sub G_0/G_1 apoptotic cell populations were observed from the Flow cytometric data. DNA fragmentation and production of caspase-3, -8, and -9 upon treatment with XVL induces apoptosis with the involvement of both intrinsic and extrinsic apoptotic pathways. **Conclusion:** Overall, *Xanthosoma violaceum* lectin specifically retards proliferation, migration, and survival of A549 lung cancer cells with less toxicity to normal cells. The results of present investigation underpin the potential of XVL as a natural anticancer agent and require preclinical investigation.

Keywords: *Xanthosoma violaceum* lectin, Lung cancer, A549 cells, Apoptosis, Cell cycle arrest, Caspase activation

INTRODUCTION

Among the cancer types, Lung cancer remains one of the most fatal cancers worldwide, accounting for over 1.8 million cancer-caused deaths each year[1]. About 85% of all instances of lung cancer are non-small cell lung cancer (NSCLC), and the majority of patients are discovered at an advanced stage, when there are few therapeutic choices

and a poor prognosis[2]. Systemic toxicity, poor tumour selectivity, and the establishment of drug resistance are common shortcomings of current therapeutic therapies, such as chemotherapy and radiation therapy, emphasizing the urgent need for the creation of safer and more effective anticancer treatments. Lectins are a heterogeneous group of non-immune proteins or glycoproteins that specifically bind to glycan motifs on cell surfaces and are found across all organisms. Their unique sugar-binding properties facilitate participation in various biological processes, including cell proliferation, differentiation, immunological regulation, and death[3, 4]. Additionally, lectins have come to light as promising macromolecules for oncological study.

Plant-derived lectins, in particular, have been demonstrated to have a wide range of anticancer actions, including cancer cell growth inhibition, programmed cell death induction, metastasis suppression, and angiogenesis regulation[5].

Xanthosoma violaceum, belong to the Araceae family, widely employed its tuber as a folk medicine due its anti-inflammatory and antimicrobial activities. As part of our on-going research on understanding structure-function relationship of plant lectins, the lectin (XVL) purified from the tubers possess strong hemagglutinating activity, specificity towards mannose, and remarkable stability over a broad range of pH and temperature conditions[6].

In addition to these biochemical properties, XVL has been reported to possess antibacterial and antioxidant activities, projecting its potential as a multifunctional bioactive molecule[7]. With all these favourable characteristics, the anticancer potential of XVL, particularly its effects on lung cancer cells, is largely untouched.

Recognizing the well-known role among oxidative stress, chronic inflammation, and cancer progression, it was presumed that XVL may exhibit targeted cell growth inhibitory and apoptotic effects on lung cancer cells. Hence the aim of the present investigation was to evaluate the effects on human lung adenocarcinoma A549 cells with regard to cell viability, apoptosis induction, cell cycle regulation, DNA damage, and migratory potential by the XVL. This study endeavours to provide insight into the potential application of XVL as a natural therapeutic candidate for lung cancer.

MATERIALS AND METHODS

Source of Lectin (XVL)

Xanthosoma violaceum lectin (XVL), extracted from the tubers of *Xanthosoma violaceum*, was previously purified and characterized utilizing conventional analytical methods and subsequently assessed for its potential to suppress cell proliferation and induce apoptosis.

Cell Culture

The cell lines, such as A549 (human lung adenocarcinoma cells) and HEK293 (normal embryonic kidney cells), were procured from the National Centre for Cell Science (NCCS), Pune, India. Routinely cells were maintained in DMEM/F12 medium (Gibco, USA) supplemented with 10% heat-inactivated foetal bovine serum (FBS), 1% penicillin–streptomycin (100 U/mL penicillin and 100 µg/mL streptomycin), and 1% L-glutamine at 37°C in a humidified atmosphere containing 5% CO_2. At an interval of 2-3 days the culture medium was replaced, and cells were sub cultured at 80% confluency with 0.25% trypsin-EDTA. On a periodic basis, they were made for microbiological contamination and cell morphology.

MTT Assay (Cell Viability)

The MTT assay[8] was employed to examine the cell viability *in vitro*. Respective cells were seeded in 96-well plates at a density of 1×10^4 cells/well, followed by incubation for 24 hours. Later, 0.31, 0.62, 1.25, 2.5, 5, and 10 µg/100 µL concentrations of XVL were used to treat the cells. The treated cells were incubated for 48 h. Following incubation, MTT solution (5 mg/mL in PBS; 10 µL/well) was added to each well and incubated at 37°C for 4 h. The formed formazan crystals were dissolved in 100 µL DMSO, and the absorbance of the developed colour was measured at 570 nm (reference 640 nm) using a micro plate reader.

Cell viability (%) was calculated as:

Cell viability (%) = (Absorbance of treated cells / Absorbance of control cells) × 100

From the dose–response curves using nonlinear regression analysis, the IC_{50} of XVL was determined. All experiments were performed in triplicate and independently repeated three times.

Colony Formation Assay (Clonogenic Assay)

The extended cytotoxic effect of XVL on A549 cells was investigated using a clonogenic assay[9]. Five hundred A549 cells were seeded into each 35-mm plate with 2 mL of complete medium and allowed to adhere overnight. Cells were exposed to the IC_{50} concentration of XVL and incubated for 10 days, with medium refreshed every 3 to 4 days. Colonies were maintained in 3.7% formaldehyde for 15 minutes, stained with 0.2% crystal violet for 30 minutes, meticulously rinsed, air-dried, and then photographed. Colonies were solubilized in 1% SDS, and absorbance was measured at 570 nm to assess viability.

Surviving fraction (SF) was calculated as:

SF (%) = (Number of colonies in treated group / Number of colonies in control group) × 100.

Experiments were performed in triplicate and independently repeated three times.

Cell Migration Assay (Wound Healing Assay)

We employed the wound healing test[10, 11] to determine how XVL influenced A549 cells' ability to migrate. Cells (5×10^5 cells/well) were cultured in 6-well plates until they reached 100% confluence. A sterile 200 µL pipette tip was used to produce a linear scratch in the monolayer. To treat the cells, the IC_{50} concentration of XVL in serum-free media was used, after washing away any loose cells with PBS. Using a phase-contrast microscope, we observed the wound close at 0, 36, and 60 hours and photographed the same areas each time.

Percentage migration (%) was calculated as:

$$\text{Percentage Migration (\%)} = [(W_0 - W_t) / W_0] \times 100$$

Where *Wo* is the initial wound width at 0 h and *Wt* is the wound width at 36 h or 60 h. Experiments were performed in triplicate.

Quantification of Caspases (3, -8, -9) by Enzyme Colorimetric Assay

Following the previously stated 24-hour treatment of 2 x 10^6 A549 cells with doses of XVL at IC_{25}, IC_{50}, and IC_{100}, the cells were tested for the induction of caspase activity in accordance with the manufacturer's protocol. Trypsinization with a 0.05%/0.02% trypsin/EDTA solution, two rounds of ice-cold PBS washing, and centrifugation at 1,500 rpm were the methods used to harvest the cells. After that, the cells were reconstituted in 50 µl of cold cell lysis buffer and allowed to sit on ice for ten minutes. The BCA™ Protein Assay Kit (Pierce, Rockford, IL, USA) was used to measure the protein concentration of the cytoplasmic lysate following centrifugation at 13,800 rpm for 20 minutes at 4°C. Using cell lysis buffer, the protein content was brought to 100 µg/50 µl. Using the Caspase colorimetric test kit (Elabscience, Houston, Texas, USA), the activity of caspase-3, -8, and -9 was assessed in accordance with the manufacturer's instructions. In a microplate reader, the cleavage of synthetic caspase-3 (Ac-DEVD-pNA), caspase-8 (Ac-IETD-pNA), and caspase-9 (Ac-LEHD-pNA) substrates was measured spectrophotometrically at 405 nm.

Cell Cycle Analysis

In a 6-well plate, A549 cells ($2x10^5$ cells/well) were grown for 24 hours with and without XVL (IC_{50}). They were then trypsinized, washed with PBS, and fixed with 70% ice-cold ethanol. After centrifugation, the fixed cells were resuspended in 1 mg/mL propidium iodide and treated with RNase A solution (0.2 mg/mL RNase and 0.05% Triton X-100) in PBS[13]. A CytoFLEX flow cytometer (Beckman Coulter, Brea, California, USA) was used to analyse cell cycle distribution, and CytExpert software was used to collect and handle the data. Gated populations were quantified based on PI fluorescence intensity to assess the proportion of cells in each cell cycle phase (G_0/G_1, S, and G_2/M).

Percentage of cells in each phase was calculated as:

Percentage of cells = (Number of cells in a specific phase / Total analyzed cells) × 100

An increase in Sub G_0/G_1 indicates apoptosis, while G_2/M accumulation indicates cell cycle arrest. Experiments were performed in triplicate.

Assessment of DNA Damage by Single-Cell Gel Electrophoresis (Comet Assay)

To evaluate the degree of primary DNA damage caused by XVL (IC_{50}), the alkaline comet assay method was employed[14-16]. Control and treated cell suspensions were combined with 1% low melting point agarose (LMPA) and embedded onto microscopic transparencies pre-coated with 1% normal melting agarose (NMA) [HIMEDIA]. An additional layer of 0.5% LMPA was applied above the layer containing the cell. It was subsequently subjected to alkaline lysate (pH 10), followed by alkaline unwinding and electrophoresis (pH > 13) at 25 volts for 30 minutes. The samples were neutralized using 0.4M Tris-Cl (pH 7.5), stained with ethidium bromide, and examined under a fluorescence microscope (Lietz). The degree of DNA damage, indicated by tail DNA, was quantified using Komet 5.5 software.

Mean tail length (µm) was calculated as:

Mean Tail Length (µm) = (Σ Tail length of individual cells) / Number of cells analyzed

Increased tail length indicates DNA fragmentation. ≥50 cells/slide was analyzed. Experiments were performed in triplicate.

Statistical Analysis

The experiments were conducted in triplicate and independently repeated three times (n = 3). Data are reported as mean ± SD. One-way ANOVA was used to compare numerous groups (e.g., different XVL concentrations in MTT, caspase, and comet assays), followed by Tukey's post hoc test. Student's t-test was used to compare two groups (for example, treatment against control in clonogenic and migratory experiments). Statistical analyses were carried out using GraphPad Prism version 10 (GraphPad Software, USA). P-values < 0.05 indicated statistical significance. Statistical

significance is denoted by *p < 0.05, **p < 0.01, and ***p < 0.001.

RESULTS

Source of Lectin (XVL)

Xanthosoma violaceum lectin (XVL) from tubers was purified and characterized earlier using conventional analytical techniques.

XVL Induces Dose-Dependent Cytotoxicity in A549 Cells but Not in HEK293 Cells

Xanthosoma violaceum lectin (XVL) was tested to see how well it killed A549 human lung cancer cells and HEK293 normal embryonic kidney cells using the MTT assay after 48 hours of treatment.

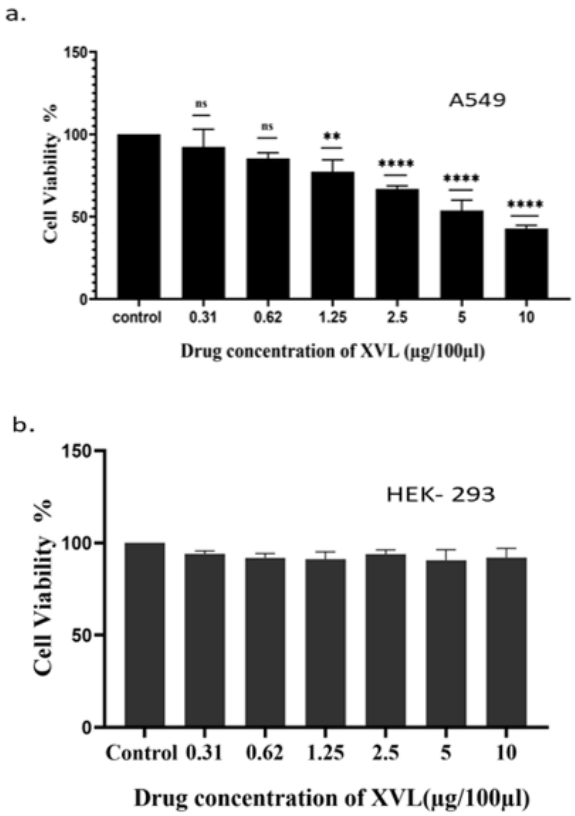

Fig. 1: Dose-dependent cytotoxicity of *Xanthosoma violaceum* lectin (XVL) on A549 and HEK293 cell lines

The vitality of A549 human lung adenocarcinoma cells (a) and HEK293 normal embryonic kidney cells (b) was evaluated after 48 hours of treatment with escalating concentrations of XVL (0.31–10 µg/100 µL) by the MTT test. Data are presented as percentage cell viability compared to untreated control and represent the mean ± SD of three independent experiments (n = 3).

Fig. 1 shows that A549 cells became less viable when they were treated with XVL. This effect was dose-dependent. As the concentration of XVL rises, cell viability falls. The lowest level of cytotoxicity was found at 0.31 µg/100 µL, and the highest level of reduction was seen at 10 µg/100 µL. This graph shows that at 6.508 µg/100 µL, 50% of the cells are still alive, which is its half-maximal inhibitory concentration (IC_{50}). But in HEK293 cells, even at higher concentrations of XVL, there was no significant loss of viability. There was no statistically significant drop compared to controls that were not treated ($p > 0.05$, $n = 3$). There is no doubt that these findings show that XVL selectively kills cancer cells while having little effect on healthy cells. The half-maximal inhibitory concentration (IC_{50}) of XVL against A549 cells was found to be 6.508 µg/100 µL, which means it had moderate antiproliferative action. HEK293 cells, on the other hand, stayed alive at all concentrations that were tried. These results show that XVL only kills cancer cells and not normal cells, which suggests that it might be useful as a therapeutic. This might be possible because glycans are expressed differently in cancer cells than in normal cells.

XVL Supresses Colony Formation ability of A549 Cancer Cells

A clonogenic assay was used to assess the impact of XVL on the capacity of cancer cells to form colonies. This examination evaluates a single cell's capacity to endure, divide, and establish colonies after therapy. A concentration of XVL (6.508 µg/100 µL) was applied to the cells. When compared to the untreated control cells, XVL demonstrated a discernible decrease in clonogenic survival (Fig. 2). The microscopic findings showed that treated cells had substantially fewer and smaller colonies than control cells, which had dense and well-defined colonies. The fixed colonies' colorimetric assessment at 570 nm using crystal violet shows a significant drop in absorbance, suggesting that the clonogenic survival was lower in the XVL-treated group than in the untreated control group ($p < 0.01$, $n = 3$). These results demonstrate the long-lasting antiproliferative action of XVL by showing that it considerably reduces the clonogenic potential of A549 cells at the treatment dose.

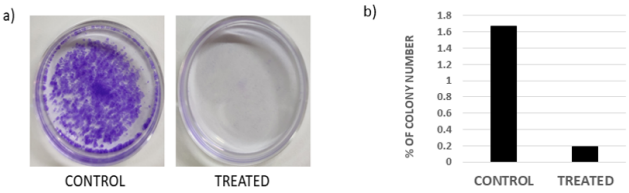

Fig. 2: Effect of XVL on clonogenic survival of A549 cells

(a) Representative images of colony formation in untreated control and XVL-treated (IC_{50}) A549 cells. (b) Quantitative analysis of surviving colonies measured at 570 nm after crystal violet staining. Data are expressed as mean ± SD of three independent experiments ($n = 3$); $p < 0.01$ vs. control

XVL Inhibits Migration of A549 Cells

Compared to untreated cells, significant migratory inhibition of A549 cells was noted in those treated with IC_{50} concentration of XVL at both 36 and 60 hours (Fig. 3). Microscopic images revealed a noticeably larger and wider wound gap in untreated cells compared to those treated with XVL over the incubation period. After 60 hours of incubation, untreated cells exhibited around 80-90% wound repair, whereas XVL-treated cells demonstrated just 50-60% closure, as assessed using ImageJ software. The quantitative and qualitative findings substantiate that XVL significantly impedes migration, suggesting its possible involvement in cancer cell migration and invasion.

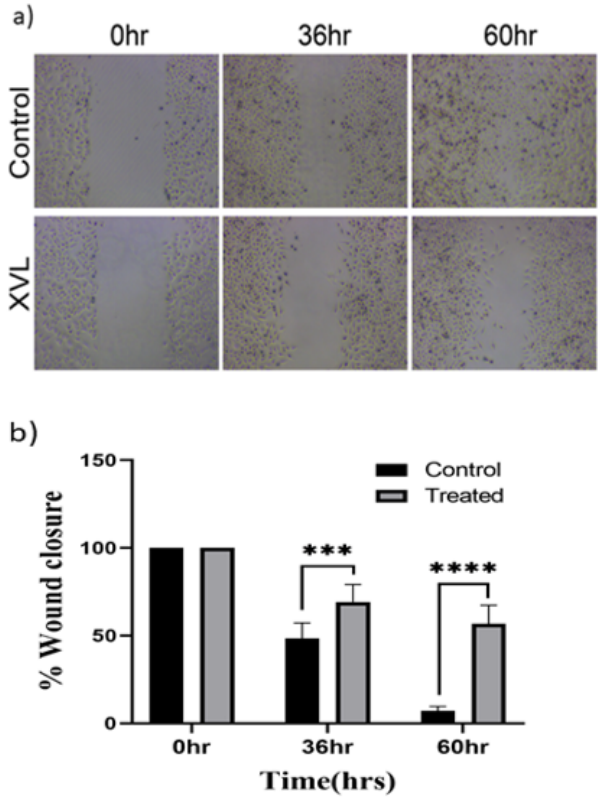

Fig. 3: Effect of XVL on A549 cell migration evaluated by wound healing assay

(a) Representative phase-contrast images captured at 0-, 36-, and 60-hours post-scratch. Cells treated with the IC_{50} concentration of XVL showed significantly reduced wound closure compared with the untreated control group. (b) Quantitative analysis of percentage wound closure over time. Data are expressed as mean ± SD of three independent experiments (n = 3); $p < 0.01$ vs. control.

XVL Activates Caspase-Mediated Apoptosis in A549 Cells

The activation of caspase activity is a distinctive characteristic of apoptotic cell death. We established that XVL prompted the activation of the proteolytic activities of caspase-3, caspase-8, and caspase-9 in A549 cells. Fig. 4 illustrates the activity of caspase-3, caspase-8, and caspase-9 in A549 cells treated with XVL at concentrations of IC_{25} (3.25 µg/100 µL), IC_{50} (6.508 µg/100 µL), and IC_{100} (13 µg/100 µL). Significant caspase-3 activity was seen at the IC_{100} concentration, indicating elevated expression of the executioner caspase. The substantial increase of Caspase-8 and Caspase-9 in the treated group substantiates the participation of both the extrinsic and intrinsic apoptotic pathways. The up-regulation of Caspase-3, 8, and 9 supports XVL's propensity to induce apoptosis via a caspase-mediated mechanism.

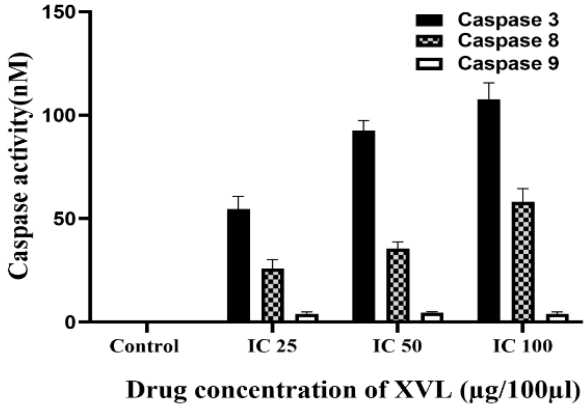

Fig. 4: Effect of *Xanthosoma violaceum* lectin (XVL) on caspase-3, caspase-8, and caspase-9 activities in A549 cells

Cells were treated with XVL at IC_{25} (3.25 µg/100 µL), IC_{50} (6.508 µg/100 µL), and IC_{100} (13 µg/100 µL) concentrations for 24 h. Caspase activities were measured using colorimetric assays. (a) Caspase-3 activity. (b) Caspase-8 activity. (c) Caspase-9 activity. Results are expressed as fold change relative to untreated control. Data represent the mean ± SD of three independent experiments. **$p < 0.05$, ***$p < 0.01$ vs. control.

XVL Induces G_2/M Cell Cycle Arrest in A549 Cells

The antiproliferative action demonstrated by XVL led us to examine its impact on the distribution of cell populations across several phases of the cell cycle in A549 utilizing flow cytometry. Flow cytometric analysis of DNA content revealed that XVL exposure alters cell cycle progression in A549 cells. The distribution examination of untreated populations revealed the G_0/G_1 (resting/early growth) phase at 64.9%, succeeded by the G_2/M (mitotic preparation) phase at 26.35%, the S (DNA synthesis) phase at 6.16%, and the Sub (early apoptotic/hypo diploid) phase at 2.59%. After 24 hours of treatment with the IC_{50} concentration of XVL, the proportion of cells in the mitotic phase rose to 34.4%, while early apoptotic cells increased to 4.66%. A slight reduction to 55.05% was noted in the resting/early growing phase.

Nevertheless, the cellular population throughout the DNA synthesis phase remains constant (Fig. 5).

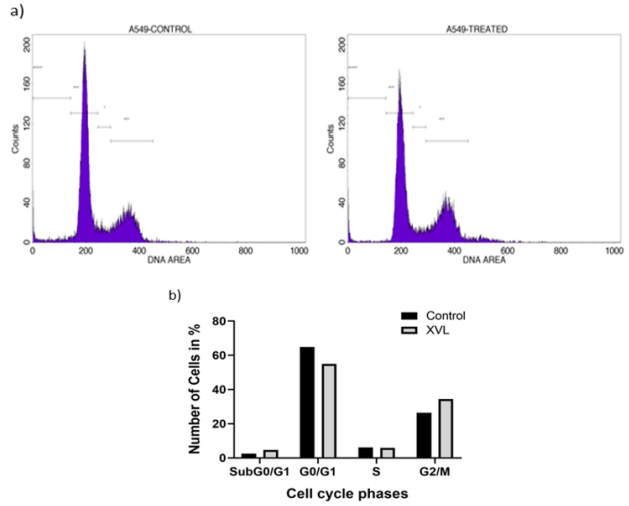

Fig. 5: Effect of XVL on cell cycle distribution in A549 cells

Cells were treated with IC_{50} XVL (6.508 µg/100 µL) for 24 h. (a) Representative flow cytometric histograms showing untreated control and XVL-treated cells. XVL treatment resulted in accumulation of cells in G_2/M and Sub G_0/G_1 phases, indicating cell cycle arrest and induction of apoptosis. (b) Bar graph showing the percentage of A549 cells in Sub G_0/G_1, G_0/G_1, S, and G_2/M phases. Data are expressed as mean ± SD of three independent experiments. **p < 0.05, **p < 0.01 vs. control.

XVL Induces DNA Damage in A549 Cells

Cells were treated with IC_{50} XVL (6.508 µg/100 µL) for 24 hours, subsequently removed using trypsinization, and analysed using the alkaline DNA comet assay.

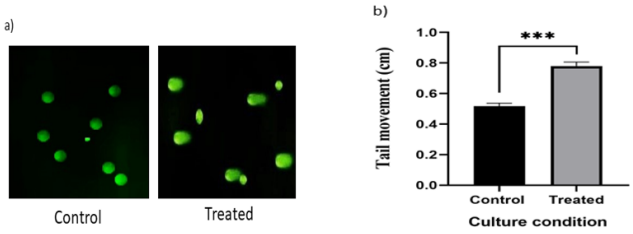

Fig. 6: Assessment of DNA damage in A549 cells using the alkaline comet assay

Cells were treated with IC_{50} XVL (6.508 µg/100 µL) for 24 h. (a) Representative fluorescent images of control (untreated) and XVL-treated cells showing comet formation. (b) Quantitative analysis of average comet tail length. Data are expressed as mean ± SD of three independent experiments (≥50 cells/slide). **p < 0.05 vs. control.

Fig. 6 illustrates that the cells exhibited discernible DNA comets in the presence of XVL, as evidenced by the enhanced comet tail length of 0.78 µm compared to the control cells, which measured 0.45 µm ($p < 0.05$). These data underscore the potential of XVL to induce DNA double-strand breaks in A549 cells, indicating a genotoxic effect of XVL on A549 cells *in vitro*.

DISCUSSION

The predominant causes of mortality globally have shifted from unintentional incidents and infectious diseases to cancer. Despite the significant anticancer efficacy of numerous therapeutic techniques, the toxicity of these conventional treatments to normal cells remains unavoidable. Currently, specific innovative anticancer medicines that are effective yet less hazardous to normal cells are very intriguing. Lung cancer, or lung carcinoma, affects both males and females. It is regarded as the primary cause of mortality globally. Lung cancer is categorized into small cell and non-small cell lung cancer based on histological analysis. An advanced form of non-small cell lung cancer is typically associated with a poor prognosis; thus, innovative strategies are urgently required to enhance patient survival rates[1, 2]. Lectins are a category of proteins prevalent in various living beings that have garnered interest as potential inducers of cell death in tumour tissues by initiating apoptotic signalling pathways. Lectins are recognized for their cytotoxic and/or anti-proliferative effects on cultured cells. This study indicated that XVL inhibits population growth of human lung cancer cells in a dose- and time-dependent manner. Our investigation further shown that XVL treatment resulted in the activation of initiator caspases-8 and -9, as well as effector caspase-3, culminating in the induction of apoptosis. The activation of both caspase-8 and -9 indicates potential participation of both extrinsic and intrinsic mechanisms in the generation of apoptosis, aligning with previous findings from other plant-derived lectins[5, 6]. The antiproliferative activity of XVL on A549 was assessed. XVL induced growth inhibition in lung cancer cells in a dose-dependent manner, with an IC_{50} of 6.508 µg/100 µL. XVL exhibited no harmful effect, even when incubated at a higher dosage (10µg/100 µL) against HEK293 cells, underscoring the potential of lectins as viable options for biopharmaceuticals. The selective growth inhibitory effect may be attributed to the differential expression of glycans on the cell surface. These findings are significant, as numerous conventional chemotherapeutic agents demonstrate considerable toxicity towards normal cells, resulting in adverse side effects and consequently restricting their clinical application. Consequently, there is a distinct necessity for novel medicines with alternative modes of action for the direct therapy of these disorders. The induction of apoptosis by XVL was evidenced by the production of caspases and an increase in the hypo diploid cell population. Numerous lectins are recognized for their ability to induce cell cycle arrest at the G0/G1, S, or G2/M phases, or in combination, to trigger apoptosis. Compared to other tuber lectins, XVL elicited apoptosis in 85% of

treated cells, demonstrating its significant efficacy in causing apoptosis. Programmed cell death is a significant process that involves various caspases, which can activate extrinsic and intrinsic pathways[12]. The XVL therapy activated caspases-8, -3, and -9, indicating potential engagement of both intrinsic and extrinsic caspase-dependent pathways.

The clonogenic assay results corroborated the sustained antiproliferative action of XVL, demonstrating a substantial decrease in colony formation in treated A549 cells. The clonogenic survival of cancer cells, which signifies their capacity to preserve reproductive integrity, suggests that XVL compromises both immediate viability and long-term proliferative capability[9].

Cell migration is a pivotal element in tumour advancement and metastasis[10, 11]. The wound-healing assay indicated that XVL markedly impeded A549 cell migration, implying an anti-migratory action. This inhibition may stem from changes in cytoskeletal architecture or signalling pathways involved to migration, however additional molecular investigations are required to clarify the exact mechanism. Considering that metastasis significantly impacts lung cancer mortality, XVL's capacity to impede cell migration amplifies its therapeutic relevance[20]. XVL administration induced cell cycle arrest at the G_2/M phase, along with an elevation in the Sub-G_0/G_1 population. G_2/M arrest is frequently linked to DNA damage and disrupted mitotic progression, ultimately resulting in apoptosis[15-18]. The comet assay demonstrated substantial DNA strand breaks, showing genotoxic stress and implying that DNA damage leads to cell cycle arrest and subsequent caspase-dependent death[14-16]. These findings corroborate earlier research indicating that plant lectins might induce DNA damage and alter the cell cycle in cancer cells[17, 18].

Collectively, our data indicate that XVL inhibits lung cancer cell growth via complementary mechanisms, including the induction of apoptosis, cell cycle arrest, DNA damage, and migration inhibition, while preserving normal cells. The noted selective action underscores the promise of XVL as a phytogenic anticancer drug. However, since this work was confined to *in vitro* models, additional research incorporating in vivo validation, toxicity assessment, and molecular target identification is necessary to comprehensively evaluate its therapeutic potential[5, 6, 19].

CONCLUSION

The current study illustrates that *Xanthosoma violaceum* lectin (XVL) displays targeted anticancer efficacy against human lung adenocarcinoma A549 cells, while exhibiting no cytotoxicity towards normal HEK293 cells. XVL stopped A549 cells from growing, lowered their clonogenic survival, and stopped them from moving. The anticancer effects of XVL were mediated by caspase-dependent apoptosis, induction of G_2/M phase cell cycle arrest, and DNA damage, underscoring its complex mechanism of action. Overall, these results imply that XVL is a promising anticancer drug that comes from plants and can target specific tumours. Additional research, including comprehensive molecular pathway analysis and in vivo studies, is necessary to confirm its therapeutic efficacy and safety, as well as to investigate its potential applicability in lung cancer treatment.

DISCLOSURE

Funding:

None.

Conflict of Interest:

The authors declare no conflict of interest.

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