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RESEARCH ARTICLE

Anti-Inflammatory Properties of Crude Extracts and Anti-Cancer Properties of Acetone Extract of Moringa Oleifera Lam. Flowers on Cell Line Models

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Abstract: In recent times, *Moringa oleifera* has earned great interest for its various physiological and therapeutic benefits. Acetone, ethanol, methanol, and aqueous extracts of *Moringa oleifera* flowers were investigated for anti-inflammatory and anti-cancer potential. Anti-inflammatory capacity was evaluated using the inhibition of nitric oxide production (LPS-induced) on RAW 264.7, and cell viability was appraised with the MTT assay. The potential of the acetone extract of Moringa oleifera flower (MOA) was evaluated on the C3A cell line treated with MOA for 48 h, with Melphalan as a positive control, as well as apoptotic markers (phosphatidylserine translocation, cell cycle arrest). Significant reduction in NO levels ($P<0.05$) was observed for extracts MFW and MFE at 200 µg/mL, with no cytotoxicity against macrophages, but gradual concentration-dependent inhibition of NO production by the methanol extract. MOA induced phosphatidylserine translocation resulting in apoptosis, cell cycle analysis revealed no significant cell-cycle arrest, while caspase-3 activation (MOA 125 µg/mL) showed a five-fold increase in cleaved caspase-3 ($P<0.05$). These findings imply for the first time that the acetone extract of *Moringa oleifera* has anti-inflammatory capacity and impedes hepatic cancer progression in C3A cells.

Keywords: Inflammatory, Apoptosis, Inhibition, Caspace-3, Cancer

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Introduction

Herbal remedies have been utilized by people for countless years to address a variety of health issues. There are over 500,000 distinct plant species on our planet, offering a valuable natural resource of plant compounds with healing benefits. Plants generate secondary metabolites as a way to protect themselves from harmful pathogens and environmental factors [1]. Medicinal plants are believed to be more safe, accessible and cheaper. Several medicinal plants have therapeutic capacity to treat conditions ranging from immune disorders, infections to cancer [2, 3].

Abnormal uncontrolled cell growth (cancer) is a lethal illness spreading quickly and emerging as a major health concern globally because of the absence of extensive and inclusive early detection techniques and appropriate medications [4].

Despite advances in biomedical science, cancer continues to be enigmatic from a therapeutic point of view and one of the most outstanding obstacles facing humanity and a frequent cause of mortality [5].

Inflammation and cancer are connected by the intrinsic and extrinsic pathways. Cancer development via the extrinsic pathway is triggered by inflammation, but the intrinsic pathway involves genetic mutations and cellular defects that activate signaling pathways, leading to increased inflammatory [6].

Medicinal plants are credited with natural products and bioactive compounds that could suppress cancer, or function as tumour inhibitors [4]. Many medicinal plants with the ability to inhibit proliferation of cells have paved the way for the design of targeted anti-cancer drugs. According to Kassas and Elbadry, the 6th most common cancer is liver cancer, which accounts for 8.3% of deaths of all cancers globally, third leading cause of cancer deaths and an estimated 900,000 cases annually worldwide [7, 8].

The use of plant-based medicines has a long history, dating back to ancient civilizations. Many cultures have relied on herbal remedies for treatment of diseases because of the perceived lower toxicity and side effects compared to synthetic/orthodox drugs [9]. In addition, their participation in complex cellular pathways is enhanced by the presence of multiple phytochemicals [10].

Moringa oleifera Lam. has a wide spread in Africa and Asia [11]. It is a significant vegetable of the Moringaceae family. All parts of the plant (except the flowers) has been used nutritionally and medicinally [12-14]. It has been reported to have significant potential for treatment of cancer, diabetes, oxidative stress and to induce apoptosis [15]. Several preclinical studies on seed, leaf, and bark of *M. oleifera* have reported its capacity to induce apoptosis in cancer cells [16-18]. The use of flowers in food and medicine though ancient, is making a come-back. The present study addressed an existing research deficit of virtually no reports on *M. oleifera* flowers usage. Anti-inflammatory and anti-cancer (evaluating Phosphatidylserine translocation, cell cycle analysis and caspace-activation/cleavage) effects of extracts from *Moringa oleifera* flowers were evaluated on macrophages and C3A cell lines respectively. This is the first study to our knowledge to assess the capacity of *Moringa oleifera* flowers acetone extract on anti-inflammatory and anti-cancer properties in cell line models.

Materials and Methods

Plant Materials

Moringa oleifera flowers were obtained and collected between June-July 2022 from Lefakong Farm, North West Province of South Africa. Professor Cupido of the Botany Department, University of Fort Hare, identified and validated the flowers and a voucher specimen (MAP/004/2019) was archived at Giffen Herbarium of the university.

Preparation of Extracts

Flowers of *Moringa oleifera* were air-dried at room temperature ground finely, then extracted in solvents. For this study, solvents were chosen because of their capacity to dissolve a wide range of compounds with varying polarities, making them versatile for extracting the various bioactive compounds in the flowers. Methanol is particularly effective for phytochemical analysis and pharmaceutical extractions and for its ability to isolate various bioactive polar compounds like flavonoids, tannins and glycosides, while acetone is excellent at extracting non-polar compounds like certain lipids, carotenoids and some alkaloids [19, 20]. It is also relatively inexpensive and can be easily removed from the extract, making it practical for several applications. Distilled water at room temperature and 100 °C apart from its natural extractive power, was used to simulate the traditional ways of using *Moringa oleifera* flowers.

Each of the ground plant materials (60 g) was extracted separately (on an orbital shaker) in distilled water, hot water boiled at 100 °C (tea), methanol, or acetone for 48 h. Each of the extracts was filtered (Buckner funnel and Whatman No. 1 filter paper), filtrates concentrated to dryness using a rotavapor for solvent extracts and a freeze dryer (48 h) was used for the aqueous extracts and samples stored at 4 °C till needed.

Reagents and Cells

Unless otherwise stated, all reagents and chemicals used including Sulfanilamide, Lipopolysaccharide (LPS), aminoguanidine andN-1-napthylethylenediamine dihydrochloride (NED) were supplied by Sigma-Aldrich (St. Louis, MO, USA). DMEM (Low glucose), NEAA and PBS with and without Ca^{2+} and Mg^{2+}, RPMI 1640 culture medium and foetal bovine

serum (FBS) were products of Cytiva (Marlborough, MA, USA), while penicillin/streptomycin were products of Biowest (Nuaillè, France). RAW 264.7 mouse macrophages were purchased from Cellonex (South Africa). Human hepatoma-derived C3A hepatocytes were purchased from the American Type Culture Collection.

Cell Line Maintenance

Dulbecco's Modified Eagle's Medium (DMEM) with Fetal Bovine Serum (FBS) and 1% penicillin-streptomycin and maintained in a 5% CO_2 incubator at 37 °C was used to culture the RAW 264.7 cells. Human hepatoma-derived C3A hepatocytes was maintained in complete medium in a humidified atmosphere with 5% CO_2 at 37°C.

Sample Preparation

Samples were dissolved in DMSO (100 mg/mL); Aminoguanidine (100 µM) was the positive control for anti-inflammatory activity, while Melphalan was the standard for apoptotic assays.

Anti-Inflammatory Assay on RAW 264.7 Cells

This assay was done as described previously [21] (Abifarin et al., 2020). Prepared cell suspension was dispensed into a 96-well microtiter plate (1 x 10^5 cells per well), followed by overnight incubation in RPMI medium to facilitate adherence. Test samples (aqueous, methanol, or acetone) were used to replace spent medium. Medium containing 1µg/ml lipopolysaccharide (LPS) was infused, then cultivated in a humidified incubator for 18 h at 37°C. Spent culture medium (50 µl) was transferred to new 96-well plate, followed by the addition of 50 µL of sulfanilamide solution (1% sulfanilamide in 5% phosphoric acid) was added to all wells (Griess reagent), allowed to stand for 10 min in the dark at room temperature, after which absorbance was measured at 540 nm (BioTek ® PowerWave XS spectrophotometer (Winooski, VT, USA). Concentration of nitrate released into the culture medium was calculated from the standard curve constructed using sodium nitrate (100 µM in complete medium). Subsequently, the spent medium was eliminated, 100 µL of MTT (0.5 mg/mL in complete medium) was added to the remaining cells and incubated for 1 h at 37°C after which MTT was removed and 100 µl DMSO added to the cells to enhance dissolution of MTT crystals, followed by measurement of absorbance at 560 nm to assess cell viability.

PS Translocation and Cell Cycle Analysis

The method described by Swanepoel et al. was used for this assay [22]. Cells were seeded in 96 well plates at 4000 cells/well in 100 µL aliquots, allowed to adhere overnight, then treated with the 62.5 µg/mL and 125 µg/mL MOA, while Melphalan (15 µM and 30 µM) was control. Cells were stained using Annexin V-FITC/PI Kit protocol (MACS Miltenyi Biotec) with modifications to include the Hoechst 33342 nuclear dye. The cells were treated with the diluted binding buffer (at final concentrations of 2 and 5 µg/mL respectively).

Thereafter, treatments were aspirated from all wells, 100 µL of dyes added and cultivated for at room temperature 20 min.

Cells were imaged for cell cycle analysis (ImageXpress Micro XLS Widefield Microscope; Molecular Devices), fitted with a 10x Plan Fluor objective, and DAPI and FITC filter cubes for Hoechst 33342 and Annexin V-FITC, respectively. Nine image sites representing 70% of the surface area were acquired in each well, followed by addition of 10 µL of Propidium Iodide (PI) prepared in binding buffer (100 µg/mL). Images of the cells were taken as soon as PI was added, as described previously and the images analysed for PS translocation to determine viability and death (live, apoptotic, necrotic, and late apoptotic/necrotic).

Caspase -3 Activation

Cultured and treated cells were fixed and permeabilized using the IntraPrep kit (Beckman Coulter) for detection of intracellular antigens which create holes or openings in the cell membrane. Cleaved caspase -3 (Asp 175) monoclonal antibody was used to determine the presence of the activated caspase enzyme, PBS containing 0.5% BSA was initially used to block the cells, antibody incubation followed, (1:250) for 1 h at 37°C, washing and then incubation (Alexa 647 conjugated secondary antibody (1:1000)) for 30 min at 37°C in the dark. Additionally, 5 µg/mL Hoechst for staining, then imaged as described previously.

Data Quantification

The ImageXpress MicroXLS Widefield was employed for quantifying live and dead cells, while. The DAPI, FITC and Texas Red filter cubes were used for PS translocation, caspase -3 activation and cell cycle analysis experiments. The MetaXpress

software and Multi-Wavelength Cell Scoring Application Module were used to analyse images and EXCEL spreadsheet used for data processing and analysis.

Results

Anti-Inflammatory Properties of *Moringa oleifera* Flower Extracts

The capacity of Moringa oleifera flower extracts to prevent inflammation are presented in Figure 1. All the extracts (MFW, MFM and MFE) significantly ($P<0.05$) suppressed NO production in a concentration dependent manner. A reduction in NO levels was observed for extracts MFW, MFM, and MFE especially at 200 µg/mL (highest tested concentration) compared to standard.

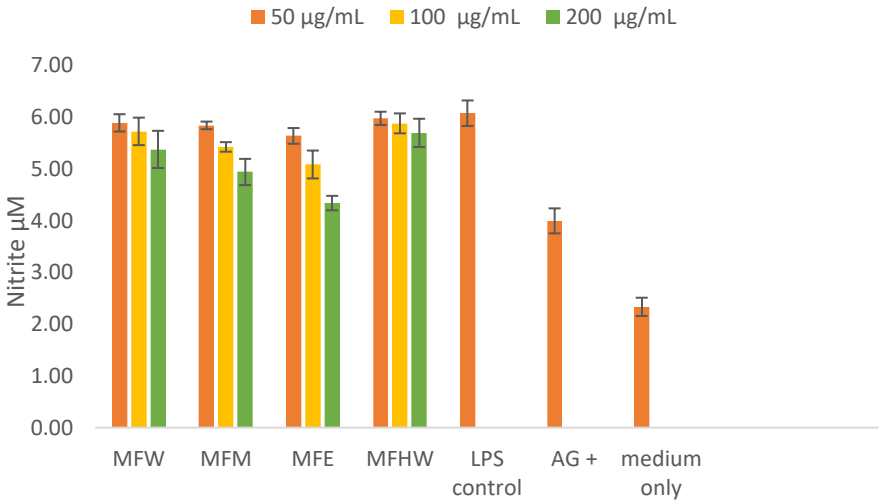

Fig. 1: Effect of aqueous (MFW), methanol (MFM), ethanol (MFE) and boiled aqueous (tea) (MFHW) extracts of *M. oleifera* flower on nitric oxide (NO) production. Experiments were in triplicates, results presented as mean ± SD

To rule out cytotoxicity as a contributing factor to reduction in NO levels, the MTT assay was performed (Figure 2) for cell viability and to verify that the observed inhibitions of nitric oxide production was caused by activity of the test compounds. MTT evaluation revealed that the extracts were not toxic and observed inhibitory action was not due to cell damage.

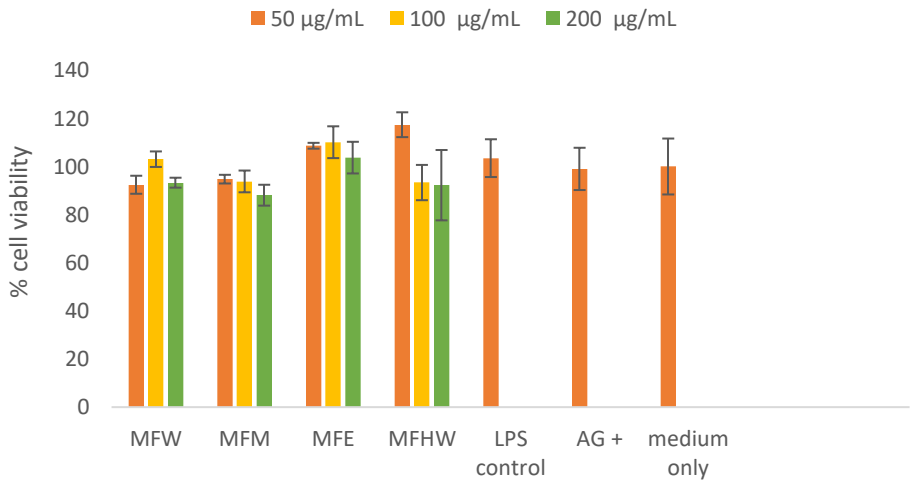

Fig. 2: Cell viability (%) of macrophages after 24 h exposure to aqueous (MFW), methanol (MFM), ethanol (MFE) and boiled aqueous (tea)(MFHW) extracts of *M. oleifera* flower at different concentrations. Data represents the mean ± standard deviation for three replicate wells, LPS was used as positive control. Two-tailed student t-test: * $p < 0.05$ and # $p < 0.005$ was used to compare significance of treated to untreated control

Anti-Cancer Effect of Acetone Extract from *Moringa oleifera* Flower

Anti-cancer properties of MO flower acetone extract (MOA) were evaluated by observing changes in phosphatidylserine translocation, cell cycle analysis and caspase-3 activation in C3A cells.

Phosphatidylserine (PS) Translocation

Apoptosis induction effect of *M. oleifera* acetone extract (MOA) using Annexin V-FITC and PI revealed significant ($P<0.05$) concentration dependent decrease in number of live cells in treated compared to untreated. Results showed that as concentration increased the number of apoptotic cells also increased while late apoptotic was undetected.

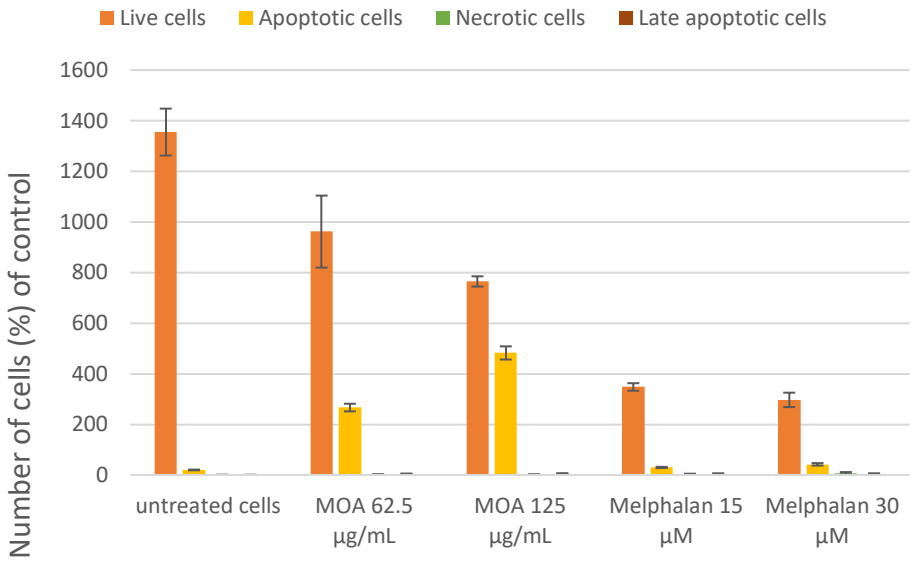

Fig. 3: Percentage (%) of live, apoptotic, necrotic and late apoptotic C3A cells after 48 h exposure to MOA and Melphalan. Bars represent mean of quadruplicate experiment. SD is defined as error bars

Cell Cycle Analysis

The phase in which cell cycle arrest occurred was identified using DNA cell cycle analysis (Figure 4). This information could indicate the possible mechanism of cell death. As the concentration increased, the percentage apoptotic cells treated with MOA extract increased, an indication that apoptosis by acetone extract was concentration-responsive. Although no convincing effect on the cell cycle attributable to MOA was indicated.

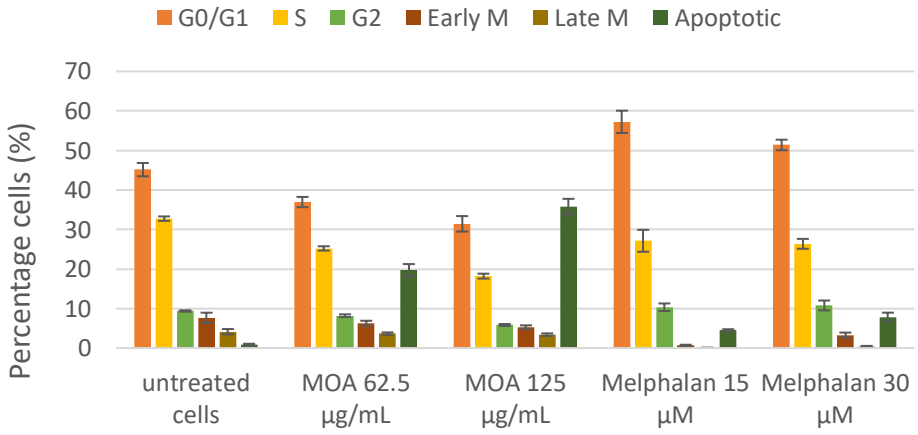

Fig. 4: Cell cycle analysis of C3A cells after 48 h exposure to MOA and Melphalan. Bars are means of individual experiment performed in quadruplicate. Significance was determined using the two-tailed student t-test

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Caspase -3 Activation

Figure 5 presents Caspase-3 activation in C3A cells after 48 h exposure to treatments and reveals a significant ($P<0.05$) five-fold dose-responsive increase of cleaved caspase-3, on administration of MOA compared to untreated cells, an indication that MOA extract has the capacity to induce cleavage of caspase-3.

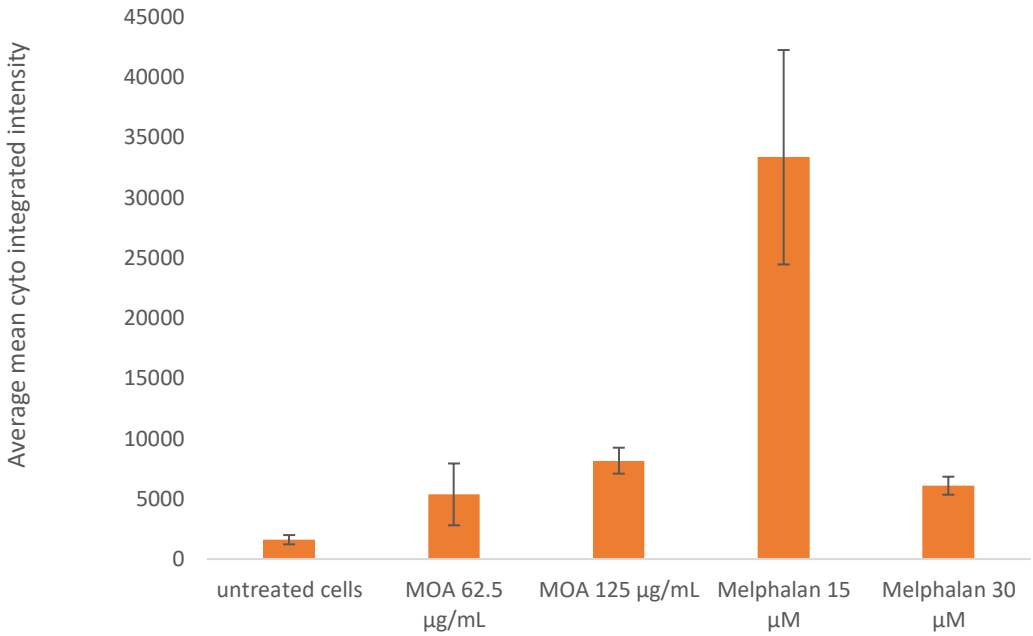

Fig. 5: Cleaved caspase -3 in C3A cells after 48 h exposure to MOA and Melphalan. Bars represent individual experiment performed in quadruplicate. Significance was determined using the two-tailed student t-test

Table 1: Summary of the results

Assays	MOA 62.5 µg/mL	MOA 125 µg/mL	Melphalan 15 µM	Melphalan 30 µM
Phosphatidylserine (PS) translocation	Apoptotic cell death	Cell death as a result of apoptosis	2/3 cell viability lost	2/3 cell viability lost
Cell cycle analysis	Increase in % apoptotic cells	Increase in % apoptotic cells	G0/G1 arrest	G0/G1 arrest
Caspase -3 activation	3-fold increase in cleaved caspase -3	5-fold increase in cleaved caspase -3	Great increase in cleaved caspase -3	4-fold increase in cleaved caspase -3

Discussion

Anti-inflammatory and anti-cancer potentials of extracts from *Moringa oleifera* flowers with focus on the acetone extract which exhibited cytotoxicity compared to the other extracts were investigated. Exposure to LPS caused concomitant NO formation and expression of inducible nitric oxide synthase (iNOS). MTT assay confirmed non-toxicity of the extracts.

Reduction in NO levels for extracts of aqueous, methanol, tea and ethanol at 200 µg/mL indicates their concentration-dependent anti-inflammatory potential. Lee et al. reported that moringa fruit extract inhibited LPS-stimulated macrophages compared to moringa root and leaf investigated [23]. Xu et al. also reported that *Moringa oleifera* leaf and seed extracts (11.1–100 µg/mL) from Kenya inhibited NO production an indication of antioxidant and anti-inflammatory capacity, correlated with their flavonoids content [24]. On the contrary, higher NO production than aspirin was observed with ethanol extract of moringa seeds at the same concentration; while another report showed that 80% hydro-ethanol extract of *Moringa oleifera* flower extract had a good inhibitory effect on nitrite production [25]. When nitric oxide (a key inflammatory mediator) is produced in excess within biological systems, various diseases including cardiovascular diseases, cancer and atherosclerosis can result

[26]. Most diseases are caused by disorder in homeostasis and excess NO, therefore reducing NO s production could be of healing advantage in diseases caused by high levels of NO [27]. Exogenous or endogenous aggressions characterised by vascular and cellular events can trigger inflammation [28].

This study agree with other reports that Moringa flowers contain anti-inflammatory principles mediated via inhibition of the inflammatory cascade in LPS-challenged macrophages [29]. The observed anti-inflammatory action could be ascribed to bioactive compounds especially flavonoids (e.g., quercetin), isothiocyanates, ascorbic acid, carotenoids and polyphenols, which have the capacity to regulate and reduce inflammation [30].

Tian et al. and Al-Khayri et al. reported that flavonoids and polyphenols such as luteolin, kaempferol, apigenin and quercetin present in vegetables, herbs, fruits, seeds, stems, roots and flowers of plants possess anti-inflammatory and antioxidant properties [31, 32]. These bioactives-flavonoids, polyphenols and quinic acid (also present in *Moringa oleifera* flowers), have the potential for use as adjuvant treatment for inflammatory diseases and oxidative stress.

Apoptosis is significant in the development and progression of cancer, making it a critical focus for several established clinical cancer treatment strategies. [33]. Unlike the damaging, uncontrolled cell lysis seen in necrosis, apoptosis is a regulated programmed cell death involving a precise sequence of intracellular events ending in cellular destruction where the final cellular debris is encapsulated in apoptotic bodies, a process that effectively mitigates the inflammatory response in surrounding normal tissues associated with necrotic cell rupture [34]. Therefore, apoptosis is used as a favorable pharmacodynamic end point for evaluating anti-cancer drug action [35, 36].

Apoptosis is important for preventing accumulation or survival of damaged or mutated cells that could lead to cancer. The early stage of apoptosis is characterized by condensation of chromatin, nuclear fragmentation, pyknosis, cells round up and retraction of pseudopods, while blebbing, shrinkage, exposure of internal phosphatidylserine to the outer surface of the cell and apoptotic bodies formation are features of late apoptosis [18, 37, 38]. This confirms the apoptotic effect of MOA on C3A cells through phosphatidylserine translocation, a characteristic feature of apoptosis. MOA treatment significantly increased ($P<0.05$) apoptotic cell populations compared with untreated controls (Figure 2).

Presence of activated caspases is indicated by increase in mean integrated fluorescence intensity. After 48 h treatment, presence of cleaved caspase-3 was observed, indicating that cytotoxicity was induced via caspase-3 activation. The observed significant increase ($P<0.05$) confirms that MOA induced cytotoxicity and apoptosis via caspase -3 activation.

From the summary (Table 1) of *in vitro* biochemical assays on C3A hepatocarcinoma cells, it can be concluded that the mechanism of cell death by MOA was through induced caspase-dependent apoptosis as evident from the PS translocation and five-fold increase in caspase-3.

Several plants (flower, leaves, fruits, seeds, bark, roots, etc.) and marine-derived crude extracts and their compounds such as xanthones, polyphenols, alkaloids, terpenes, *Viscum album*, *Azadricta indica*, *Inula racemosa*, *Momordica cohinchinensis*, *Oceanapia sagittaria*, *Moringa oleifera, Musa paradisiaca, Cucurbita ficifolia, Tulbaghia violacea* and many more, have shown anti-cancer capacities by inducing apoptosis mediated through caspase-induced apoptosis *in vitro* [39-44].

Findings from this study agree with several reports that *Moringa oleifera* possess anti-cancer properties demonstrated in various cell-line models, which is often mediated through caspace-dependent apoptosis attributed to bioactives like palmitic acid, β-sitosterol, flavonoids, anthocyanin and isothiocyanates, among others [19, 45-50].

Conclusion

Moringa oleifera flowers' anti-inflammatory action is correlated with the presence of bioactives especially flavonoids, isothiocyanates, ascorbic acid, quinic acid, carotenoids and polyphenols, which have the capacity to regulate and reduce inflammation as well as apoptosis. These findings imply for the first time that acetone extract of *Moringa oleifera* has anti-inflammatory action and impedes hepatic cancer progression in C3A cells. Therefore, acetone extracts from *Moringa oleifera flowers* could be a promising source of anticancer agents. Further research into the specific compounds and mechanisms involved could lead to new therapeutic strategies for acetone extracts of Moringa oleifera flowers as cancer chemoprevention, adjuvant or therapeutic target for cancer.

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Conflict of Interest

The Authors declared no conflict of interest.

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