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Antioxidant Potential, Selective Cytotoxicity, and Molecular Docking Insights of *Maresia nana* Methanol Extract against A549 Cancer Cells

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In this study, the antioxidant activity, phenolic content, and cytotoxicity of the above-ground parts of *Maresia nana* were evaluated using various assays. Antioxidant activity was assessed using the DPPH radical scavenging test, yielding an IC_{50} value of 90.55 ± 11.14 mg mL^{-1}. The total flavanol content of the extract was 0.41 ± 0.01 mg QE/g, the total flavonoid content was 29.26 ± 1.88 mg QE/g, and the total phenolic content was 29.76 ± 2.64 mg GAE/g, indicating significant antioxidant properties and richness in phytochemical compounds. Additionally, GC-MS analysis identified eight bioactive compounds in the methanol extract of *M. nana*. The extract demonstrated 58.50 ± 3.5% cytotoxicity in A549 cells at the highest dose, while it increased proliferation in HEK293 cells, indicating selective cytotoxicity toward cancer cells. Furthermore, the binding affinities and interactions of two small-molecule ligands, Acridin-1(2H)-one, 3,4-dihydro-3,3-dimethyl-9-propylamino- and Androsta-3,5-diene-3,17-diol diacetate, with the Ras protein were investigated. Acridin-1(2H)-one showed a binding energy of −5.1 kcal mol^{-1}, while Androsta-3,5-diene-3,17-diol diacetate demonstrated a stronger binding affinity with a binding energy of −5.5 kcal mol^{-1}. In conclusion, the *M. nana* extract's antioxidant and phenolic profiles support its potential health benefits, and its selective cytotoxic effects on cancer cells suggest its promise for cancer therapy. Additionally, the binding characteristics of the ligands provide valuable insights for future drug development strategies.

1. Introduction

Cancer is a multifaceted disease characterised by a complex molecular environment and altered cellular pathways leading to uncontrolled cell proliferation.[1] Among various cancer types, lung cancer remains a leading cause of cancer-related deaths worldwide, accounting for ≈1.8 million deaths annually. It represents 11.6% of all diagnosed cancer cases globally and 18.4% of all cancer-related deaths, affecting both sexes[2] Lung cancer is primarily classified into two main subtypes: small cell lung cancer (SCLC) and non-small cell lung cancer (NSCLC), with NSCLC accounting for more than 80% of all cases.[3]

While tobacco use is the primary risk factor for lung cancer, other factors such as genetic predisposition, diet, occupational exposures, and environmental pollutants also contribute to its development.[4] Despite the availability of conventional treatments, such as chemotherapy and targeted therapies, resistance to these drugs, combined with severe side effects, remains a major challenge in effectively treating lung cancer.[5]

In response to these limitations, there is growing interest in exploring plant-based compounds as potential therapeutic strategies. Plant-derived compounds are known for their target-specific actions and relatively lower cytotoxicity than conventional chemotherapeutic agents.[6] Several natural products have already been developed into effective chemotherapy drugs, yet the search for new natural treatments with fewer side effects continues. These compounds could offer alternative approaches to overcoming treatment resistance, making them valuable candidates in the development of lung cancer therapies.

One of the significant molecular drivers of lung cancer, particularly in NSCLC, involves mutations in the KRAS gene, part of the RAS oncogene family. Mutations in KRAS, NRAS, and HRAS genes are prevalent in various human cancers, including lung adenocarcinomas, which harbour KRAS mutations in ≈30% of cases.[7,8] Targeting RAS proteins, especially KRAS, has emerged as a potential strategy for inhibiting cancer cell growth and survival. Methods aimed at disrupting KRAS signalling, such as

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inhibiting its interaction with SOS1, are promising avenues for cancer therapy.[9]

Maresia nana (DC.) Batt. in J.A.Battandier & L.C.Trabut, Fl. Algérie, Dicot.: 68 (1888) belongs to the family Brassicaceae (Cruciferae) and represents one of the two species within the genus *Maresia* Pomel. Although only two species globally represent *Maresia*, *M. nana* is the sole species of this genus naturally occurring in Türkiye.[10–12] This annual herb typically grows to a height of 5–15 cm and exhibits a distinct ashy or pale white appearance due to the dense covering of large stellate trichomes. The stems are short and branched, while the leaves are ovate with entire or undulating dentate margins. Basal leaves are petiolate, whereas the stem leaves are nearly sessile. The flowers of *M. nana* may be either pedicellate or sessile, with sepals measuring ≈2.5 mm and featuring slightly saccate bases. The petals, ranging from pink to violet or lilac, are ≈5 mm long. The pedicels, which are very short during flowering, elongate to ≈5 mm during fruiting. The fruits are cylindrical, densely covered with stellate hairs, and reach up to 3 cm in length.[10]

Maresia nana has a distribution extending from the Mediterranean Basin to Afghanistan. In Türkiye, it is predominantly found in regions such as the Istranca Mountains, Southern Marmara, Western and Central Black Sea, Aegean, and Antalya subregions. The species thrives in sandy coastal habitats, often at low altitudes near sea level, and flowers between March and May. It is mainly observed in districts at elevations up to 10 meters above sea level. Its distribution also includes coastal and sandy habitats in southern Italy, North Africa, the Middle East, and Afghanistan.[12–14]

Taxonomic classification and current status of *Maresia nana* according to POWO, *M. nana* belongs to the family *Brassicaceae*. Based on the current taxonomic framework provided by Plants of the World Online,[12] its classification is as follows:

Kingdom: Plantae; Phylum: Streptophyta; Class: Equisetopsida; Subclass: Magnoliidae; Order: Brassicales; Family: Brassicaceae; Genus: *Maresia*; Species: *M. nana*. This classification aligns with contemporary phylogenetic analyses and reflects the most recent taxonomic consensus.

Plants from the Brassicaceae family have been noted for their antioxidant properties, with species such as watercress (*Nasturtium officinale*) and green mustard (*Brassica juncea*) recognised as the most active in terms of antioxidant activity.[15] Brassicaceae plants also produce phenolic compounds, tocopherols, and distinctive seed oils.[16] In vivo studies have identified Brassicaceae species as potent chemo-preventive agents against breast, lung, prostate, and gastrointestinal cancers.[17] *M. nana*, belonging to the Brassicaceae, has been shown to possess high antioxidant, anticancer, and phenolic content; however, no studies regarding this species are available in the literature. This study aims to conduct a phytochemical analysis, evaluate the antioxidant activity, and assess the cytotoxic effects of *M. nana*, thereby providing new insights into the cancer-related potential of this unexplored species and contributing to research on plant-based cancer therapies. Furthermore, the major compounds of the plant, including Androsta-3,5-diene-3,17-diol diacetate, and Acridin-1(2H)-one, 3,4-dihydro-3,3-dimethyl-9-propylamino, have been examined through in silico modelling, predicting that these compounds may have potential as pharmaceutical agents for cancer treatment. The selection of this plant material is based on the unexplored biological activities of *M. nana* and the overall potential of the Brassicaceae. This study aims to explore the phytochemical properties, antioxidant activities, and cancer therapeutic potential of *M. nana* in depth.

The limitation of the study is highlighted by the fact that the cytotoxicity evaluation was only conducted using the MTT assay, and molecular mechanisms or specific apoptotic pathways were not investigated. There is no existing research on *M. nana* in the literature, and this study stands out as an original work that fills this gap, focusing on phytochemical content analysis, antioxidant activity, chemical profiling, and cytotoxic effects. The initial findings from this study will significantly contribute to the literature, providing a solid foundation for future research. Additionally, the in-silico modelling presented in the study enables the prediction of *M. nana's* potential active compounds as drug candidates that could be used in cancer treatment. In conclusion, this study not only explores the biological activities of *M. nana* but also provides a solid starting point for future clinical and preclinical research.

2. Results and Discussion

2.1. Antioxidant Activity and Phenolic Content of *Maresia Nana* Methanol Extract: Evaluation of DPPH Radical Scavenging, Flavonoid, and Phenolic Compounds

In our study, we analysed the antioxidant activity and phenolic content of the above-ground parts of *M. nana* using various assays. The findings are summarised in Table 1. The antioxidant activity of *M. nana* methanol extract was assessed using the DPPH radical scavenging assay, yielding an IC_{50} value of 90.55 ± 11.14 mg mL^{-1}. This value indicates that *M. nana* exhibits good antioxidant activity compared to the positive control, EDTA (0.23 ± 0.01 mg mL^{-1}). The total flavanol content of the extract was measured at 0.41 ± 0.01 mg QE/g, the total flavonoid content was 29.26 ± 1.88 mg QE/g and the total phenolic content was 29.76 ± 2.64 mg GAE/g. These results indicate that the extract has a rich phytochemical content.

To understand the biological activity of a medicinal plant, it is important to know its chemical constituents. Plants produce secondary metabolites in response to environmental stimuli, and these compounds, which are independent of primary metabolism, hold significant therapeutic potential. In recent years, concerns about the safety of synthetic antioxidants have increased, leading to a renewed interest in discovering new and safer antioxidants from plants. This study evaluated the antioxidant activity (DPPH test) and total bioactive components of *M. nana*.[18]

There are no studies in the literature regarding antioxidant activity, phytochemical content analysis, chemical profile, and cytotoxic effects of *M. nana*. *M. nana*, which belongs to the Brassicaceae family, has been discussed comparatively with other species from this family.

Table 1. Diphenyl-1-picrylhydrazyl (DPPH) radical scavenging activities and phytochemical content of *M. nana* extracts.

Plant Name	DPPH (IC_{50} mg/mL)	Total Flavanol Compound (mg QE/g extract)	Total Flavonoid Compound (mg QE/g extract)	Total Phenolic Compound (mg GAE/g extract)
M. nana	90.55 ± 11.14	0.41 ± 0.01	29.26 ± 1.88	29.76 ± 2.64
BHT (positive control)	0.23 ± 0.01			

*Values expressed are means ± S.D. of three parallel measurements. GAE: Gallic acid equivalent; QE: Quercetin equivalent.
*Values expressed are means ± S.D. of three parallel measurements. BHT: Butylated HydroxyToluene.

The IC_{50} value of the ethanol extract from *Brassica juncea* seeds was measured at 103 µg mL^{-1}.[19] The *B. juncea* seed ethanol extract shows higher antioxidant activity than the *M. nana* methanol extract. In another study related to *B. juncea*, the IC_{50} value of the ethanol-water extract was determined to be 170 µg mL^{-1}.[20] The *B. juncea* ethanol-water extract shows higher antioxidant activity than the *M. nana* methanol extract. The IC_{50} value of the *Brassica tournefortii* diethyl ether essential oil extract was measured at 75.28 µg mL^{-1}.[21] The antioxidant activity of *B. tournefortii* diethyl ether oil extract is higher than that of *M. nana* methanol extract. The total phenol content of *Brassica nigra* was reported to be 6.67 mg g^{-1}, and the total flavonoid content was found to be 2.04 mg g^{-1} QE. Additionally, it was noted that the IC_{50} value of the *B. nigra* extract was 63.09 µg mL^{-1}, indicating its antioxidant activity.[22] The *B. nigra* extract, when compared to the *M. nana* extract, has lower total flavonoid and phenolic content, yet its antioxidant activity is much higher.

The total phenolic content of cauliflower extract was 5.83 ± 0.133 mg GAE/g, and the flavonoid content was 1.69 ± 0.522 mg CAE/g. Additionally, the extract's antioxidant activity was reported with an IC_{50} value of 917.81 ± 24.114 mg. The total phenolic content in broccoli extract was 12.85 ± 0.195 mg GAE/g, and the flavonoid content was 6.71 ± 0.467 mg CAE/g. The antioxidant activity of broccoli extract was found to have an IC_{50} value of 81.45 ± 1.919 mg. The total phenolic content of *Brussels sprouts* extract was 8.10 ± 0.219 mg GAE/g, and the flavonoid content was 3.07 ± 0.262 mg CAE/g. The antioxidant activity of *B. sprouts* extract was reported with an IC_{50} value of 339.57 ± 22.848 mg.[23] The total phenolic and flavonoid contents of cauliflower, broccoli, and *B. sprouts* ethanol extracts are lower than the *M. nana* methanol extract. However, the antioxidant activity of cauliflower and *B. sprouts* ethanol extracts is lower than that of the *M. nana* extract. In contrast, the antioxidant activity of broccoli ethanol extract is higher than that of the *M. nana* extract.

The total phenolic content was found to be greater in the *Brassica incana* leaf extract than in the flowering top extract, with values of 37.20 ± 0.93 mg GAE/g extract and 27.98 ± 0.32 mg GAE/g extract, respectively. The antioxidant activity of *B. incana* leaf extract and flowering top extract, with IC_{50} values of 1.306 ± 0.049 mg/mL and 2.077 ± 0.011 mg/mL, respectively.[24] Compared to *M. nana* above-ground extract, *B. incana* leaf extract has a higher total phenolic content, while the flower extract has a lower content. The antioxidant activity of *B. incana* leaf and flower extracts is 45 and 90 times higher than that of *M. nana* above-ground extract.

The total polyphenol content of *Brassica oleracea* acephala extract is reported to be 6.37 mM GAE/g, while the total polyphenol content of *Brassica oleracea* convar. *botrytis* var. *cymosa* extract is 2.24 mM GAE/g. The IC_{50} values for the antioxidant activity of *B.oleracea* var. *acephala* and *B.oleracea* convar. *botrytis* var. *cymosa* extracts are determined to be 1.53 and 6.51 mg, respectively.[25] The *M. nana* extract, when compared to both plant extracts, has a high phenolic content but exhibits lower antioxidant activity.

For *Cardaria draba*, the methanol extract had a total phenolic content of 18.41 ± 0.92 mg GAE/g and a total flavonoid content of 22.21 ± 1.28 mg RE/g. In *Descurainia sophia*, the methanol extract contained 11.48 ± 1.01 mg GAE/g of total phenolic content and 27.67 ± 0.27 mg RE/g of total flavonoid content. The methanol extract of *C. draba* shows antioxidant activity with a DPPH value of 48.33 ± 0.66 mg TE/g. In contrast, the methanol extract of *D. sophia* demonstrates higher antioxidant activity with a DPPH value of 63.00 ± 2.20 mg TE/g.[18] The methanol extract of *C. draba* and the methanol extract of *D. sophia* have lower total phenolic and flavonoid content compared to the methanol extract of *M. nana*. *M. nana* has an antioxidant content as high as both plant extracts.

The total phenolic content of *Brassica fruticulose* hydroalcoholic leaf extract has been reported as 32.63 ± 1.11 mg GAE/g extract. The IC_{50} value of *B. fruticulosa* hydroalcoholic leaf extract is 1.65 ± 0.08 mg mL^{-1}.[26] The *B. fruticulosa* hydroalcoholic leaf extract has a higher total phenolic content than the *M. nana* above-ground methanol extract. Additionally, the *B. fruticulosa* hydroalcoholic leaf extract exhibited higher antioxidant activity.

The total phenolic and flavonoid content of *B. raimondoi* methanol extract was 38.12 ± 0.50 mg GAE/g and 8.45 ± 0.60 mg QE/g extract, respectively. The IC_{50} value of *B. raimondoi* methanol extract in the DPPH test was determined to be 1.33 ± 0.02 mg mL^{-1}, showing antioxidant activity.[27] *B. raimondoi* methanol extract has a higher total phenolic content than *M. nana* methanol extract, but its flavonoid content is lower. However, the activity of *B. raimondoi* methanol extract is higher than that of *M. nana* methanol extract.

It is commonly recognised that polyphenols are strong antioxidants that can prevent ROS from forming and stop them from doing so after they have. According to the "biochemical scavenger theory," polyphenolic substances neutralise or scavenge free radicals by generating stable chemical complexes, limiting their reactions. This theory provides a general explanation for the advantageous features of polyphenols.[28,29]

According to our findings, the *M. nana* extract has strong antioxidant properties.

2.2. Identification and Quantification of Bioactive Compounds in *Maresia Nana* Methanol Extract: GC-MS Analysis

Various bioactive compounds were identified in the above-ground parts of the methanol extract of *M. nana*. The extract contains 8 bioactive phytochemical compounds with their retention time (RT), concentration (% area), and chemical structure presented in Table 2. The GC-MS chromatogram has been added based on the reference from the article[30] (Figure SM1).

Semioxamazide constituted 3.30% of the sample, indicating its moderate presence. Cycloheptasiloxane, tetradecamethyl- was detected with an area percentage of 2.21%, while Cyclohexasiloxane, dodecamethyl- made up 3.20% of the sample. The compound 6,8-Difluoro-2,2,4,4,6,7,7,8,9,9-decamethyl-[1,3,5,2,4,6,7,8,9] trioxahexasilonane was present at 2.22%. Cyclononasiloxane, octadecamethyl- accounted for 3.31% of the sample. Notably, 1,2-Benzenedicarboxylic acid, bis(2-methylpropyl) ester was a significant component, comprising 8.43% of the total area. The two most abundant compounds were Androsta-3,5-diene-3,17-diol diacetate, and Acridin-1(2H)-one, 3,4-dihydro-3,3-dimethyl-9-propylamino-, which constituted 35.77% and 38.08% of the sample, respectively. These results highlight the predominant presence of the latter two compounds in the analysed sample.

2.3. Cytotoxic Activities of *Maresia Nana* Extract Against A549 Lung Cancer Cells: In Vitro Assays

In this study, the cytotoxicity levels of *M. nana* extract were evaluated using the A-549 cell line with the MTT assay. IC_{50} values for the treatment of A-549 cells with *M. nana* extract were calculated. The in vitro cytotoxic activity of *M. nana* extract against A-549 cancer cells was evaluated, revealing an IC_{50} value of 1.3 ± 14.86 mg mL^{-1} after 48 hours of treatment. This result indicates the extract's potential anti-cancer activity against lung adenocarcinoma cells. Compared to the controls, A-549 cells demonstrated a 58.50 ± 3.5 percent reduction following 48 hours of treatment with the highest dose of *M. nana* extract (Figure 1). On the other hand, this extract did not show any inhibition in normal cells (HEK-293); instead, it significantly increased their proliferation ($p < 0.01$). This indicates that the extract is selectively cytotoxic to cancer cells and does not affect normal cells (Figure 2).

Natural agents in cancer chemo-prevention emerge as a significant area in cancer treatments by the ability to halt, slow, or reverse the transformation of neoplastic cells into malignant cancer or the initiation stage of carcinogenesis. The potential of plant-based compounds such as polyphenols, flavonoids, and alkaloids to be used in this process, either alone or in combination, is supported by the biological effects of these agents.[31]

It has been reported that *Brassica nigra* extract inhibits the proliferation of A-549 cells in a concentration-dependent

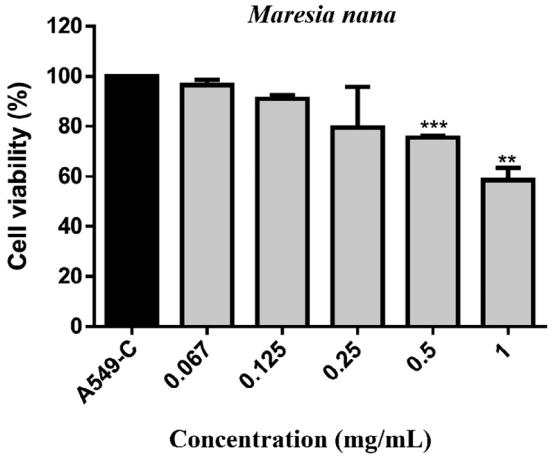


Figure 1. In vitro cytotoxic activity % (mean ± SD) of *M. nana* extract against A549cancer cells, 48-h post-treatment as compared with (HEK293) a normal cell line.

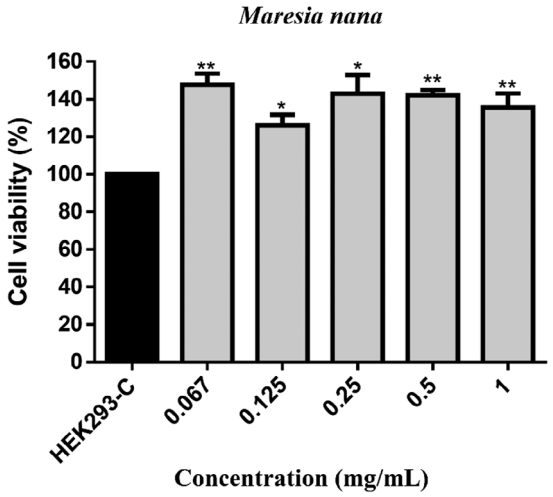
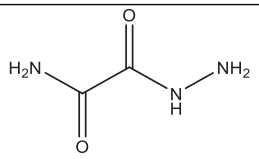
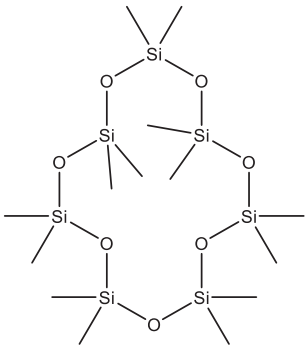
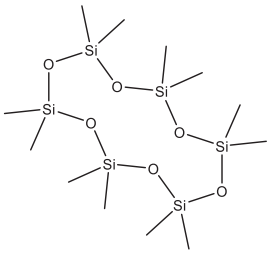
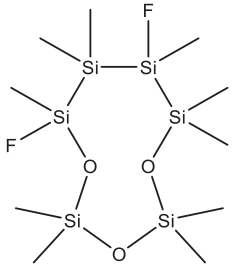
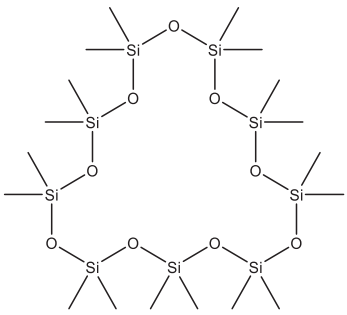


Figure 2. In vitro, cytotoxic activity % (mean ± SD) of *M. nana* extract against (HEK293) a normal cell line, 48-h post-treatment.

manner.[32] The cytotoxic effect of *B. nigra* extract on A-549 cells was observed with an IC_{50} value of 25.38 µg mL^{-1}. Compared to *M. nana* extract, *B. nigra* extract exhibits significantly higher cytotoxicity. The IC_{50} value for A-549 cells treated with *B. juncea* extract was determined to be 121.6 µg mL^{-1}.[33] *B. juncea* extract exhibits significantly higher cytotoxicity compared to *M. nana* extract. It has been shown that *Brassica rapa* significantly reduces the viability of A-549 cells in a dose- and time-dependent manner. The IC_{50} value for A-549 cells treated with *B. rapa* was reported to be 483.2 µg mL^{-1}.[34] *B. rapa* extract exhibits significantly higher cytotoxicity compared to *M. nana* extract.

The leaf and flower extracts of *Brassica incana* have been reported to show no toxic effects on human dermal fibroblast cells.[24] Similarly, no harmful effects were observed on HEK-293 normal cells with the *M. nana* above-ground extract. The *B. incana* leaf extract did not reduce the viability of MCF-7 cells after a 48-hour exposure. On the other hand, the *B. incana* flower extract exhibited a significant cytotoxic effect on MCF-7

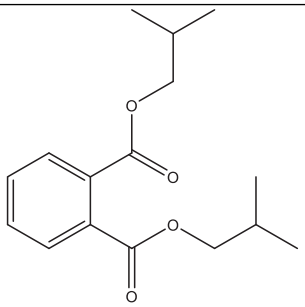
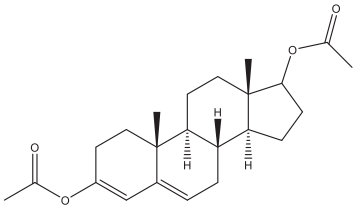
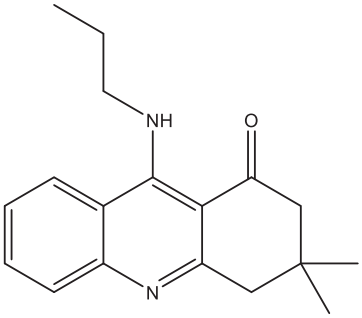
Table 2. Gas Chromatography-Mass Spectrometry analysis results of *M. nana*.

No	Retention Time (minutes)	RI	Compound Name	Area (%)	Molecular Structure
1	9.699	907	Semioxamazide	3.30	
2	32.282	1418	Cycloheptasiloxane, tetradecamethyl-	2.21	
3	35.379	1492	Cyclohexasiloxane, dodecamethyl	3.20	
4	38.246	1555	6,8-Difluoro-2,2,4,4,6,7,7,8,9,9-decamethyl-[1,3,5,2,4,6,7,8,9]trioxahexasilonane	2.22	
5	45.674	1820	Cyclononasiloxane, octadecamethyl-	3.31	

cells, showing about a 30% reduction in viability after 72 hours of exposure.[24] In the *M. nana* extract study, A-549 cells showed a 58.50 ± 3.5 percent reduction in viability after 48 hours of treatment. Furthermore, the IC_{50} value for the cytotoxic effect of the flower extract on CaCo-2 cells was determined to be 1.25 ± 0.037 mg mL^{-1} after 48 hours of treatment.[24] The IC_{50} value of the *M. nana* extract for A-549 cancer cells after 48 hours of treatment was determined to be 1.3 ± 14.86 µg/mL. When comparing the IC_{50} values of *M. nana* above-ground extract and *B. incana* flower extract, both extracts exhibited similar cytotoxic effects.

It has been reported that *B. raimondoi* extract did not affect cell viability in healthy normal HIF cells. In HepG-2 cells, however, the extract affected cell viability starting from a 500 µg mL^{-1}

Table 2. (Continued)

No	Retention Time (minutes)	RI	Compound Name	Area (%)	Molecular Structure
6	46.191	1840	1,2-Benzenedicarboxylic acid, bis(2-methylpropyl) ester	8.43	
7	53.052	2433	Androsta-3,5-diene-3,17-diol diacetate	35.77	
8	55.727	2726	Acridin-1(2H)-one, 3,4-dihydro-3,3-dimethyl-9-propylamino-	38.08	

concentration. Exposure of CaCo-2 cells to *B. raimondoi* extract for 24 hours resulted in cytotoxic effects, while no effect was observed in DU-145 cells at any of the tested concentrations.[27] *B. raimondoi* extract shows potential anti-cancer activity against HepG-2 and CaCo-2 cells, while *M. nana* extract demonstrates similar activity against A-549 cells. *M. nana* extract, like *B. raimondoi* extract, does not exhibit cytotoxicity against normal HEK-293 cells.

The extracts of *B. oleracea* and *B. incana* have shown antiproliferative effects on HeLa cells, but they did not affect the proliferation of normal human skin fibroblasts.[35] Similarly, the *M. nana* extract showed antiproliferative effects on A-549 cells, while it did not exhibit cytotoxic effects on normal HEK-293 cells.

The IC_{50} values of *B. nigra* extract applied to A-549 and H-1299 lung cancer cells for 72 hours were determined as 32.02 and 25.38 µg mL^{-1}, respectively.[36] The IC_{50} value of *M. nana* extract applied to A-549 cancer cells for 48 hours was determined as 14.86 ± 1.3 mg mL^{-1}. *B. nigra* extract exhibits a higher cytotoxic effect against cancer cells compared to *M. nana* extract.

It has been reported that the highest dose of polar extracts obtained from the basal leaves of *Isatis tinctoria* causes a reduction of ≈85% in the cell viability of CAL-62 cells.[37] The highest dose of *M. nana* extract resulted in a 58.50 ± 3.5% reduction in A-549 cells, while *I. tinctoria* basal leaf extract caused a more significant decrease in cell viability in CAL-62 cells.

L. libyca seed hydrolysate inhibited the growth of colorectal HCT-116, hepatic HUH-7, and breast MCF-7 cells, with IC_{50} values reported as 0.31, 2.25, and 37 µg mL^{-1}, respectively. The methanol extract of *M. nana* above-ground parts exhibited cytotoxic effects on A-549 cells at high doses. In comparison, it showed lower activity than *L. libyca* seed hydrolysate.

Furdak et al.[38] demonstrated a correlation between the polyphenol concentration of various plant extracts, including white cabbage extracts, and their cytotoxic effects. Additionally, flavonoids have been identified, particularly those reported as powerful antioxidants and anticarcinogenic phytochemicals. These polyphenols may be partly responsible for the antiproliferative effects.[35] Further metabolic research and screening of potentially beneficial compounds, such as polyphenols, in Brassicaceae species are necessary, in line with Mandrich and Caputo's claim that *Brassica* metabolites are emerging as new weapons for anticancer therapeutics.[39] The *M. nana* extract's potential anticancer activity is reported for the first time in this family.

In the study by El-fayoumy et al.,[40] the anticancer activities of tetradecamethyl cycloheptasiloxane have been reported. In the study by Kumar et al.,[41] it was reported that

1,2-benzenedicarboxylic acid, bis(2-methylpropyl) ester exhibited anti-proliferative activity, and its effects on cancer cells were further investigated. 5-En-androstene-3β, 17β-diol inhibits the growth of MCF-7 breast cancer cells when estrogen receptors are blocked by estradiol.[42] Furthermore, intratumoral treatment with 5-androstene-3β,17α-diol reduces tumor size and lung metastasis in a triple-negative experimental model of breast cancer.[43] In both studies, it has been reported that 5-androstene-3β, 17α-diol exhibits anticancer activity. Androsta-3,5-diene-3,17-diol diacetate has been shown to possess potential anticancer agent properties against A549 cancer cells, as demonstrated through molecular docking interactions. In human colorectal HCT-116 cells, acridine chalcone 1C ((2E)-3-(acridin-9-yl)-1-(2,6-dimethoxyphenyl) prop-2-en-1-one) plays a role in its antiproliferative effect.[44] Acridine, thiourea, gold, and acridinone derivatives exhibit cytotoxic effects on cell lines such as MDA-MB-231, SK-BR-3, and MCF-7.[44] Acridine derivatives have drawn attention due to their anticancer effects. Compounds such as acridine-1(2H)-one and 3,4-dihydro-3,3-dimethyl-9-propylamine have been shown to possess potential anticancer agent properties against A549 cancer cells, as demonstrated through molecular docking interactions. The effects of these compounds on A549 cells could contribute to the development of new approaches to cancer treatment.

2.4. In Silico ADMET Studies and Molecular Docking of Bioactive Compounds in *Maresia Nana* Methanol Extract

The pharmacokinetic properties of the compounds presented in Table 3 provide crucial insights into the biological activities and potential interactions of these compounds. In terms of gastrointestinal absorption, some compounds exhibit high absorption (e.g., Cycloheptasiloxane, tetradecamethyl and Cyclohexasiloxane, dodecamethyl), while others show low absorption (e.g., Semioxamazide and Cyclononasiloxane, octadecamethyl). This variation can influence the rate at which compounds enter the systemic circulation and their bioavailability. Regarding the ability to cross the blood-brain barrier (BBB), most compounds can cross the BBB (e.g., Cycloheptasiloxane, tetradecamethyl and Cyclohexasiloxane, dodecamethyl). In contrast, some compounds, such as Semioxamazide and Cyclononasiloxane, octadecamethyl, cannot pass through the BBB. This characteristic is significant in determining whether the compounds might exert neurological effects. Evaluations of P-glycoprotein (P-gp) substrates show that most compounds do not interact with P-gp, although some compounds (e.g., 1,2-Benzenedicarboxylic acid, bis(2-methylpropyl) ester) fall into this category and may have their movement within the body limited as a result. Additionally, the potential for inhibiting cytochrome P450 enzymes is a critical parameter. For instance, Androsta-3,5-diene-3,17-diol diacetate inhibits CYP1A2, CYP2C19, CYP2C9, and CYP3A4 enzymes, while other compounds show no such effect. These inhibitory actions can influence the pharmacokinetic interactions of the compounds and their potential interactions with other drugs. Finally, when examining the ability to permeate the skin (Log Kp), compounds like Cycloheptasiloxane and tetradecamethyl

Table 3. Pharmacokinetic properties and biological interactions of selected compounds.

	Semioxamazide	Cycloheptasiloxane, Tetradecamethyl-	Cyclohexasiloxane, Dodecamethyl	6,8-Difluoro-2,2,4,4,6,7,7,8,9,9-decamethyl-[1,3,5,2,4,6,7,8,9]trioxahexasilonane	Cyclononasiloxane, octadecamethyl	1,2-Benzenedicarboxylic acid, bis(2-methylpropyl) ester	Androsta-3,5-diene-3,17-diol diacetate	Acridin-1(2H)-one, 3,4-dihydro-3,3-dimethyl-9-propylamino-
GI absorption	Low	High	High	High	Low	High	High	High
BBB permeant	No	Yes	Yes	Yes	No	Yes	Yes	Yes
P-gp substrate	No	No	No	No	Yes	No	No	No
CYP1A2 inhibitor	No	No	No	No	No	Yes	No	Yes
CYP2C19 inhibitor	No	No	No	No	No	Yes	No	Yes
CYP2C9 inhibitor	No	No	No	No	No	No	Yes	No
CYP2D6 inhibitor	No	No	No	No	No	No	No	Yes
CYP3A4 inhibitor	No	No	No	No	No	No	Yes	Yes
Log Kp (skin permeation)	−7,89 cm/sn	−4.47 cm/s	−4.73 cm/s	−4.23 cm/s	−3.95 cm/s	−5.08 cm/s	−5.41 cm/s	−4.76 cm/s

Table 4. Docking scores and report of predicted interactions of docked conformations of Small-molecule ligands bind to a distinct pocket in Ras and inhibit SOS-mediated nucleotide exchange activity (4DSN).

Ligand	Binding Energy (kcal/mol)	Amino acid	Interacting	Distance
Acridin-1(2H)-one, 3,4-dihydro-3,3-dimethyl-9-propylamino	−5.1	A: ARG97:HH22 -: O1	Conventional Hydrogen Bond	2.46
		A: VAL109:HN -: O1	Conventional Hydrogen Bond	2.31
		H8 – A: TYR137:O	Conventional Hydrogen Bond	2.22
		A:ASP108: HA -: O1	Carbon Hydrogen Bond	2.31
		H6 – A: ASP108:OD1	Carbon Hydrogen Bond	3.08
		H7 – A: GLU107:O	Carbon Hydrogen Bond	2.27
		H7: O1	Carbon Hydrogen Bond	2.97
		A: ASP108:OD1	Pi-Anion	4.02
		C17 – A:PRO110	Alkyl	3.69
		A: HIS166: C17	Pi-Alkyl	4.52
Androsta-3,5-diene-3,17-diol diacetate	−5.5	A: ARG97:HH11 -: O1	Conventional Hydrogen Bond	2.25
		A: ARG97:HH22 -: O1	Conventional Hydrogen Bond	2,25
		A: MET111:HN -: O2	Conventional Hydrogen Bond	3,01
		A:PRO110	Alkyl	5,41
		A: LYS165	Alkyl	4,82
		A: LYS165	Alkyl	4,41
		A: LYS165	Alkyl	5,01
		A: LYS169	Alkyl	3,80
		C19 – A: LYS165	Alkyl	4,08

show higher Log Kp values, indicating easier skin permeation. In contrast, Semioxamazide, with a Log Kp of −7.89 cm s^{-1}, exhibits significantly lower skin permeability, suggesting difficulty crossing the skin barrier. These data are crucial for understanding the compounds' potential for systemic absorption via topical applications. These pharmacokinetic properties are critical in determining the compounds' potential uses in pharmaceutical formulations and their biological effectiveness.

This study investigates the binding affinity and interactions of two different small-molecule ligands with the Ras protein (Table 4 and Figures 3 and 4). The binding energies and interactions with amino acids were evaluated in detail. The binding energy of the ligand Acridin-1(2H)-one, 3,4-dihydro-3,3-dimethyl-9-propylamino- to the 4DSN protein was determined to be −5.1 kcal mol^{-1}. This ligand interacts with the protein through various hydrogen bonds, carbon-hydrogen bonds, Pi-anion, and alkyl interactions. For instance, conventional hydrogen bonds are formed between A: ARG97 and A: VAL109 with O1 at distances of 2.46 and 2.31 Å, respectively. A hydrogen bond between H8 and A: TYR137 at 2.22 Å, and carbon-hydrogen bonds between A:ASP108: HA and H7: O1 at 2.31 and 2.97 Å, respectively, are present. Other interactions include a carbon-hydrogen bond between A:ASP108 and H6 at 3.08 Å, a carbon-hydrogen bond between H7 and A: GLU107 at 2.27 Å, and a Pi-anion interaction with A:ASP108 at 4.02 Å. Furthermore, an alkyl interaction between C17 and A at 3.69 Å and a Pi-alkyl interaction between A: HIS166 and C17 at 4.52 Å were observed. These interactions indicate that the ligand binds strongly to the protein, contributing to its stabilisation. On the other hand, the ligand Androsta-3,5-diene-3,17-diol diacetate exhibited a binding energy of −5.5 kcal mol^{-1}, indicating a strong binding affinity to the protein through hydrogen bonds and alkyl interactions. Conventional hydrogen bonds are formed between A: ARG97 and A: ARG97 with O1 at distances of 2.25 and 2.25 Å, respectively, and a hydrogen bond between A: MET111 and O2 at 3.01 Å. Additionally, various alkyl interactions with A: PRO110, A: LYS165, and A: LYS169 were observed at distances ranging from 3.80 to 5.41 Å. This ligand's binding energy and interactions also indicate a strong binding affinity to the protein, contributing to its stabilisation. The low binding energies, strong binding affinities, and diverse interactions of both ligands with the protein are significant findings of this study. Such studies are crucial for understanding ligands' binding mechanisms and biochemical effects on target proteins, which can guide future drug design and development processes.

There is an increasing interest in statins due to their potential anti-cancer effects. Natural inhibitory agents exhibit antioxidant, anti-cancer, antiproliferative, pro-apoptotic, and anti-invasive properties. These agents inhibit the mevalonate pathway, leading to critical alterations in various cellular functions, particularly related to the post-translational modifications of oncogenic proteins. Notably, RAS activation requires the covalent binding of non-sterol isoprenoids such as farnesyl pyrophosphate (FPP) or geranylgeranyl pyrophosphate (GGPP). By inhibiting 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, the rate-limiting enzyme in the mevalonate pathway, statins also impede the synthesis of downstream mevalonate derivatives, including FPP and GGPP.[45] Acridin-1(2H)-one, 3,4-dihydro-3,3-dimethyl-9-

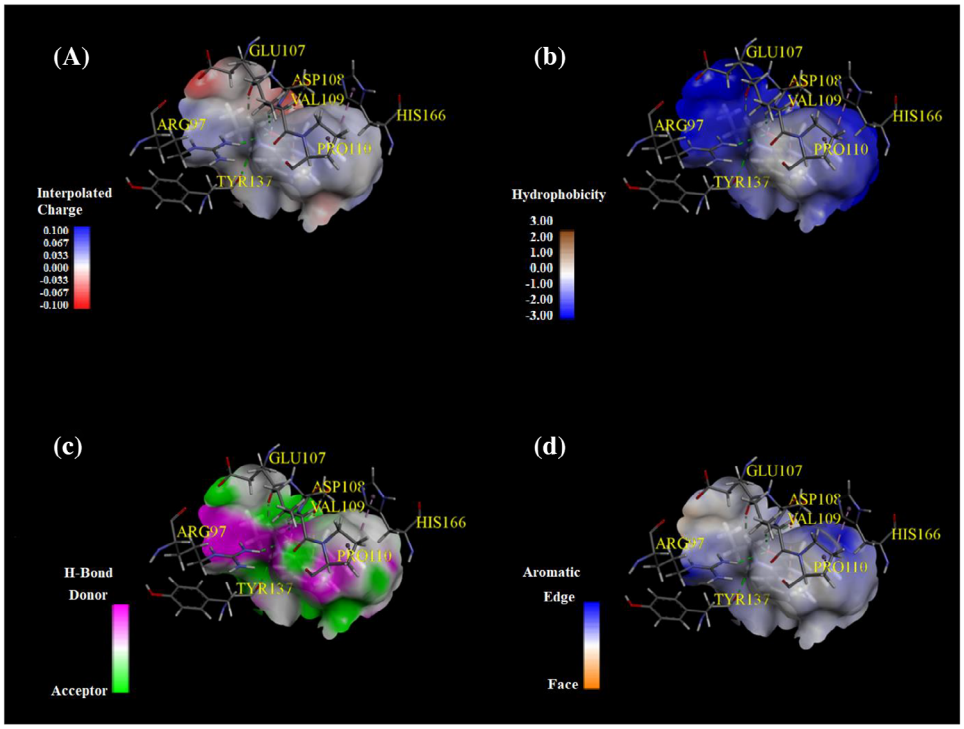


Figure 3. Interaction diagram of Acridin-1(2H)-one, 3,4-dihydro-3,3-dimethyl-9-propylamino-with 4DSN proteins in terms of a) interpolated, b) hydrophobic C) H-bond, and d) aromatic.

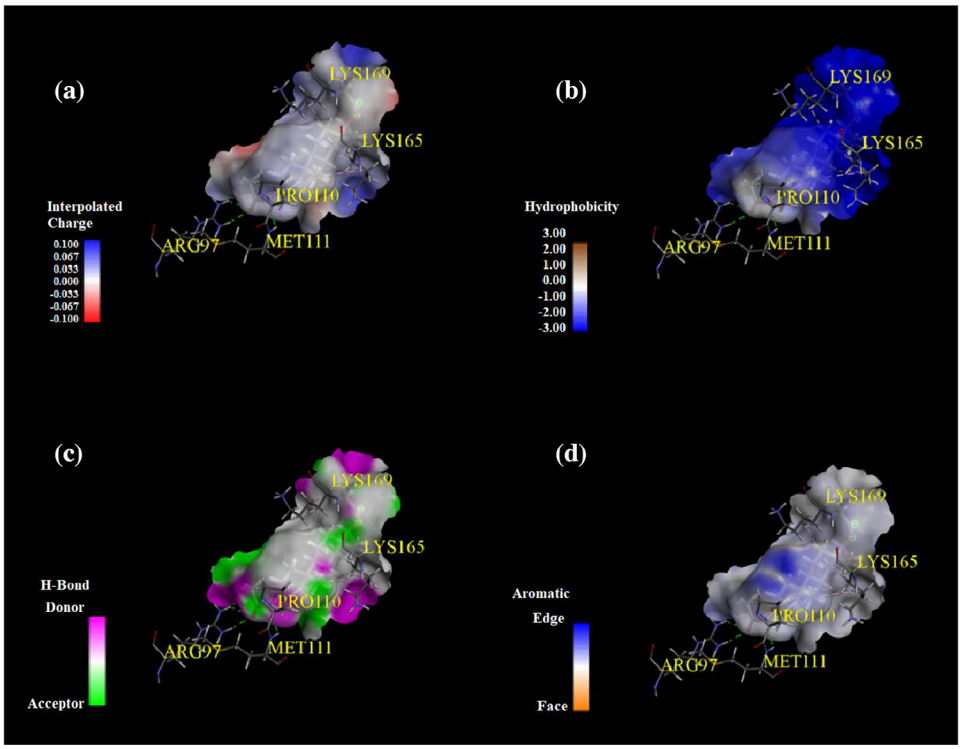


Figure 4. Interaction diagram of Androsta-3,5-diene-3,17-diol diacetate with 4DSN proteins in terms of a) interpolated, b) hydrophobic c) H-bond, and d) aromatic.

Figure 5. The morphological appearance of *Maresia nana*.

propylamino- and Androsta-3,5-diene-3,17-diol diacetate binds to the RAS protein with moderate affinity, inhibiting the mevalonate pathway, and this mechanism may lead to the inhibition of proliferation and invasion in cancer cells.

3. Materials and Methods

3.1. Plant Collections

Plant material of *Maresia nana* was collected during April from dense populations inhabiting the coastal dunes of Samsun Province. Fieldwork revealed that the populations of this species are notably homogeneous and dense in areas of the Kızılırmak and Yeşilırmak delta plains that are undisturbed by human or animal activity. The specimens were identified by Dr. Alper Durmaz, and their current taxonomic status was verified using Plants of the World Online and other authoritative databases.[12] A herbarium voucher of *M. nana* has been deposited at the Herbarium of the Faculty of Science, Department of Biology, Ondokuz Mayıs University, under accession number OMUB-7678. The morphological appearance and distribution map of *M. nana* are presented in Figures 5 and 6, respectively

3.2. Plant Extraction

The freshly harvested *M. nana* above-ground parts were washed with distilled water, cleaned, and dried in an oven at 40 °C for two days. After drying, the plant's above-ground parts were ground into a fine powder using a blender. 200 grams of dried samples were transferred into separate containers, and 2 litres of methanol were added to each. These solutions were kept in the dark for 72 hours (3 days). After this period, the methanol-extracted solutions were separated by filtration through filter paper. Subsequently, the solvents were evaporated using a rotary evaporator (Heidolph, Germany) at 40 °C under reduced pressure and stored at 4 °C until further use.[46] Methanol is a perfect solvent for extracting various substances, such as polyphenols and other compounds with biological activity, because it dissolves polar and moderately polar molecules.[47]

3.3. Phytochemical Studies

3.3.1. Total Phenolic Content

The total phenolic content (TPC) of *M. nana* above-ground parts was evaluated using the Folin-Ciocalteu reagent technique. Plant extracts were diluted to a concentration of 1 mg mL^{-1}. 0.5 mL was taken from these solutions and mixed with 2.5 mL of Folin-Ciocalteu reagent and 2 mL of 7.5% $NaHCO_3$. The Folin-Ciocalteu reagent had been previously diluted tenfold with distilled water. The mixture was incubated at 45 °C for 15 minutes. After the incubation period, the absorbance was measured at 765 nm using a UV spectrophotometer. A calibration curve was prepared using gallic acid solutions at concentrations of 1000, 500, 250, 125, 62.5, 31.25, 15.625, 7.8125, 3.90625, and 1.953125 µg mL^{-1}, obtained by serially diluting the gallic acid stock solution by half. Each standard solution was treated with the same reagents and under the same conditions as the samples. The absorbance values of the standards were plotted to create a calibration curve, which was then used to calculate the phenolic content of the samples. Each

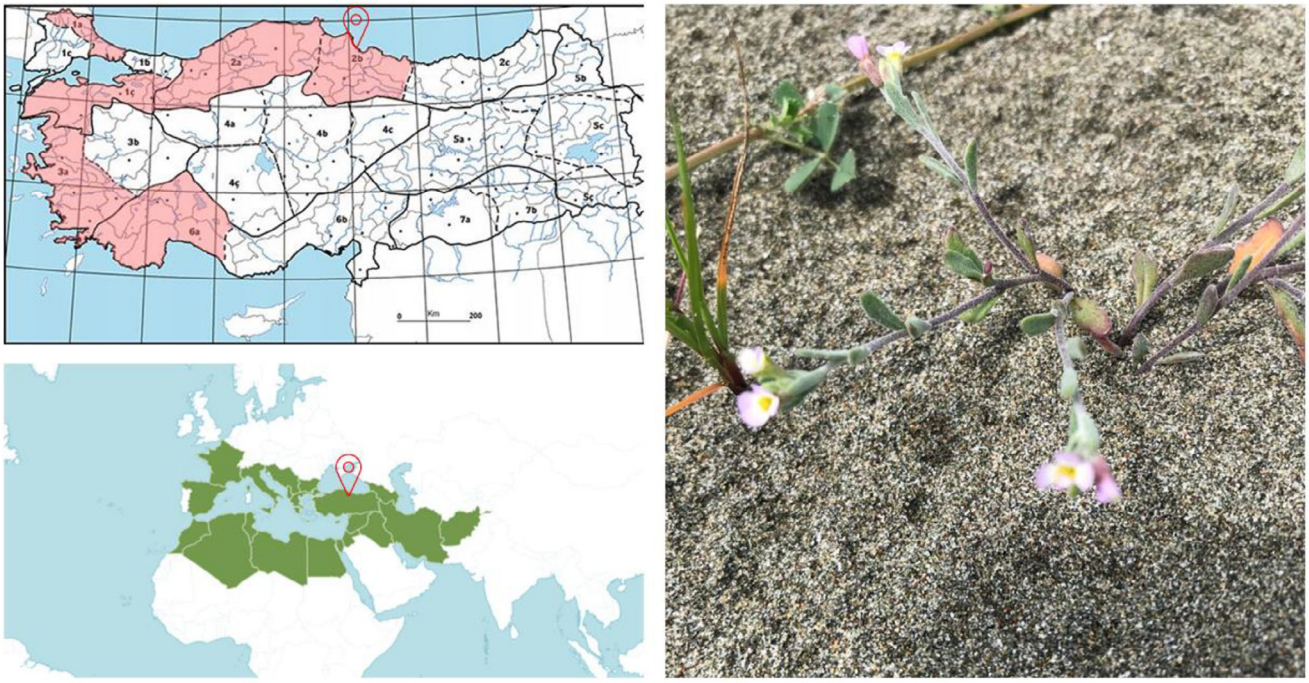


Figure 6. Global distribution, distribution in Turkey, and collection site of *Maresia nana*.

experiment was carried out in triplicate, and the results were expressed as gallic acid equivalents of the extract (mg GAE/g extract).[48]

3.3.2. Total Flavonoid Content

The total flavonoid (TFC) content was measured following a previously reported spectrophotometric method by Dewanto et al.[49] The procedure was as follows: Extracts of each plant material (1 mL containing 0.1 mg mL^{-1}) were diluted with water (4 mL) in a 10 mL volumetric flask. Initially, 5% $NaNO_2$ solution (0.3 mL) was added to each volumetric flask. At the 5th minute, 10% $AlCl_3$ (0.3 mL) was added. At the 6th minute, 1.0 M NaOH (2 mL) was added. Water (2.4 mL) was added to the reaction flask and mixed well. The absorbance of the reaction mixture was read at 510 nm using a UV spectrophotometer. A calibration curve was prepared using quercetin solutions at concentrations of 1000, 500, 250, 125, 62.5, 31.25, 15.625, 7.8125, 3.90625, and 1.953125 µg mL^{-1}, obtained by serially diluting the quercetin stock solution by half. Each standard solution was treated with the same reagents and under the same conditions as the samples. The absorbance values of the standards were plotted to create a calibration curve, which was then used to calculate the flavonoid content of the samples. Each experiment was carried out in triplicate, and the results were expressed as the extract's quercetin equivalents (mg QE/g).

3.3.3. Total Flavanols Content

The total flavonoid (TFlC) content was measured following a previously reported spectrophotometric method by Mahmoudi et al.[50] Briefly, 1 mL of the extracts was mixed with $AlCl_3$. Subsequently, 3 mL of sodium acetate solution was added. The mixture was then kept at room temperature in a dark environment for 30 minutes. After the incubation period, the absorbance of the sample was measured at 415 nm using quercetin as the standard. A calibration curve was prepared using quercetin solutions at concentrations of 1000, 500, 250, 125, 62.5, 31.25, 15.625, 7.8125, 3.90625, and 1.953125 µg mL^{-1}, obtained by serially diluting the quercetin stock solution by half. Each standard solution was treated with the same reagents and under the same conditions as the samples. The absorbance values of the standards were plotted to create a calibration curve, which was then used to calculate the flavonoid content of the samples. Each experiment was carried out in triplicate, and the results were expressed as quercetin equivalents of the extract (mg QE/g extract)

3.4. Antioxidant Studies

3.4.1. Diphenyl-1-picrylhydrazyl (DPPH) Radical Scavenging Activity of the M. nana Extract

The methanol extract of *M. nana* above-ground parts was subjected to radical scavenging activity using the DPPH radical scavenging method, as illustrated in the study by Elbestawy et al.[51] Various concentrations of *M.nana* extract were prepared (1, 0.5, 0.25, 0.125, 0.625, 0.3125 mg mL^{-1}). An 80 µg mL^{-1} DPPH ethanol solution was prepared (8 mg of DPPH was dissolved in 100 ml of methanol). 2 mL of DPPH solution was added to each extract solution, and the mixture was thoroughly shaken. Butylated HydroxyToluene (BHT) was used as the standard control. A stock solution of BHT was prepared at a concentration of 500 µg mL^{-1} and serially diluted by half to obtain the

following concentrations: 500, 250, 125, 62.5, 31.25, 15.625, 7.8125, 3.90625, 1.953125, and 0.9765625 μg mL^{-1}. The reaction mixtures were allowed to rest for 30 min at 27 °C. After the incubation period, the absorbance of the samples and standards was measured at 517 nm. The radical scavenging activity was calculated as a percentage of inhibition, and IC_{50} values were determined for both the extract and the standard control (Figure S2). The reaction mixtures were allowed to rest for 30 min at 27 °C. After the incubation period, the absorbance of the samples was measured at 517 nm. The IC_{50} values for BHT and *M. nana* extract, indicating the concentration required to reduce the initial DPPH concentration by 50%, were determined. The antioxidant activity of the *M. nana* extract was evaluated using the following formula:

$$DPPH\ scavenging\ activity\ (\%inhibition) = \left[(A\ control - A\ sample)\ /A\ control\right] x100$$

3.5. Gas Chromatography-Mass Spectrometry Analysis

The optimisation structures were analysed using the Gas Chromatography-Mass Spectrometry (GC-MS) analysis method.[46] The solid extract of *M. nana*, obtained through rotary evaporation, was dissolved in methanol, and centrifuged at 3500 rpm for 10 minutes. The resulting supernatant was utilised for GC-MS analysis. The analysis was conducted using a SHIMADZU GCMS-QP2010 Mass Spectrometer equipped with an AOC-5000 Auto-Injector. An Rxi-5MS column (30 m × 0.25 mm × 0.25 μm) was employed, with a scanning range of 30–450 Da.

The operational conditions were as follows: electron ionisation mode at 70 eV, helium as the carrier gas at a constant flow rate of 1 mL min^{-1}, an injection volume of 1.5 μL with a split ratio of 10:1, injector temperature set to 250 °C, and ion source temperature at 200 °C. The oven temperature program began with an initial hold at 70 °C for 10 minutes, followed by a ramp of 3 °C min^{-1} to 150 °C, maintained for 5 minutes. The temperature then increased at 10 °C min^{-1} to 250 °C and held for an additional 5 minutes. The solvent delay was set from 0 to 2 minutes, and the total GC-MS runtime was 56.67 minutes. Plant extracts prepared in methanol were diluted 100-fold and transferred into 1.5 mL vials for analysis. Identification of compounds was performed using the NIST Standard Reference database.

3.6. Preparation of Extract

The *M. nana* extract was dissolved in methanol to achieve a 100 mg mL^{-1} concentration. The stock solutions were then diluted with Dulbecco's Modified Eagle's Medium (DMEM) to prepare five different concentrations: 0.0675, 0.125, 0.25, 0.5, and 1 mg mL^{-1}. These varying concentrations were used for subsequent experiments to assess the effects of the extract on cultured cells.

3.7. Cell Culture

The adenocarcinomic human alveolar basal epithelial cells (A549) and Human Embryonic Kidney 293 (HEK293) cells were obtained from the American Type Culture Collection (ATCC). The cells were cultured DMEM (Gibco, USA) supplemented with 10% fetal bovine serum (FBS) (Gibco, USA) and 1% penicillin/streptomycin (Gibco, USA) in a humidified 5% CO_2 incubator at 37 °C. After reaching 80–90% confluence, the cells were detached using trypsin-EDTA (Sigma–Aldrich) and plated in 96-well plates (1 × 10^4 cells/well) for experiments. The cultured A549 cells were treated with *M. nana* extracts at 0.0675, 0.125, 0,25, 0.5, and 1 mg mL^{-1}, respectively. HEK293 cells, used as a healthy control cell line, were treated with all extracts under the same conditions to evaluate the effects on non-cancerous cells. This approach allowed for assessing cytotoxic effects and potential therapeutic benefits of the plant extracts on both cancerous and non-cancerous cell lines. The same protocol was followed for negative controls (methanol). In the cell culture experiments, the methanol concentration in which the extract was dissolved did not exceed 1%.

3.8. Assessment of Cell Toxicity Using the MTT Assay

MTT (4,5-dimethyl diphenyl tetrazolium bromide) biochemical in-vitro assay was conducted to determine the extract's effect on the cells' viability. For the MTT assay, 1 × 104 cells were seeded in each well of a 96-well plate, followed by a 24-h incubation. Plant extracts were applied to the wells, and the plate was incubated for 48 hours under the same conditions. At the end of incubation, 20 μL of 5 mg mL^{-1} MTT solution was added to the wells, followed by an immediate incubation at 37 °C for the next three hours. Finally, DMSO (Dimethyl Sulfoxide) was added after removing the MTT solution to dissolve the developed crystals. The absorption was found at 570 nm using an ELISA reader to determine the percentage cell viability by using the following formulae:

$$\text{Cellviability}\ (\%) = \frac{\text{OD of sample}}{\text{OD of control}} \times 100$$

3.9. Determination of IC_{50} Values

The results from the MTT assay were then used to calculate the IC_{50} values. The absorbance data were input into an IC_{50} calculation tool, which generated the IC_{50} values for each extract. These values represent the extract concentration required to inhibit cell viability by 50%. The IC_{50} values were determined for each cell line to assess the cytotoxic effects of the extracts.

3.10. In Silico SwissADME Studies

The toxicity ADMET properties of selected phytocompounds were evaluated to assess their toxicity characteristics. The 3D

structures of 8 phytocompounds were saved in SMILES format and uploaded to the PROTOX-II (http://www.swissadme.ch/) web servers (Charite University of Medicine, Institute for Physiology, Structural Bioinformatics Group, Berlin, Germany[52–54] for ADMET screening. SWISSADMET is an online tool used to predict a molecule's ADMET and pharmacokinetic and physicochemical properties, which are critical determinants for clinical trials.[55,56]

3.11. Molecular Docking Studies

For molecular docking studies, protein receptors were sourced from the Protein Data Bank (PDB). The crystal structures of the target receptors were prepared by removing ions, water molecules, and any bound ligands using Molegro software. To optimise the receptors for docking, hydrogen atoms were added with the aid of Autodock Vina, and the structures were saved in pdbqt format for compatibility with the docking software. Ligands were initially minimised in ChemDraw and converted to mol2 format, then to pdb format using Molegro. The ligand structures were then further transformed into pdbqt format using AutoDock tools. AutoGrid was used to create ligand-centered grid maps with a grid size of 90 Å × 90 Å × 90 Å, while other parameters were kept at default settings.[57] The docking process was executed using AutoDock Vina and AutoGrid to generate the grid maps, and Discovery Studio 4.5 was employed to visualise and analyse.[58] 2D interactions between ligands and target receptors. This study utilised the Small-molecule ligands bind to a distinct pocket in Ras and inhibit SOS-mediated nucleotide exchange activity (PDB ID: 4DSN) at a resolution of 2.03 Å. This approach enabled an in-depth understanding of ligand-receptor binding interactions.

3.12. Statistical Analyses

Correlation coefficients (R) were calculated using the CORREL statistical function in MS Excel software to determine the relationship between two variables. Data are expressed as mean ± SD obtained from three separate observations. The analysis of the data was conducted using SPSS 21.

4. Conclusion

The antioxidant activity of *M. nana* methanol extract has demonstrated effective antioxidant activity when compared to the EDTA positive control. Additionally, it has been concluded that the *M. nana* extract is rich in total phenolic and flavonoid content. The *M. nana* extract exhibits cytotoxic effects against A549 lung adenocarcinoma cells. The lack of inhibitory effects on normal cells and the enhancement of cell proliferation highlights the minimal impact of the extract on healthy tissues. These findings suggest that *M. nana* is a potential anti-cancer agent. Moreover, chemical compounds analysed from this plant using GC-MS are likely to be a valuable source for pharmaceutical applications. These results may contribute to future drug development processes. The interactions of the major chemical compounds in *M. nana* with the Ras protein have been revealed through in-silico modelling, showing moderate binding affinities. These findings suggest that these ligands may potentially modulate the function and stability of the Ras protein, which could be a critical factor in developing new therapeutic strategies targeting Ras-related pathways in cancer. In conclusion, these findings provide a foundation for future research aimed at developing antioxidant and anti-cancer agents derived from plants.

Acknowledgements

The authors have nothing to report.

Conflict of Interests

The authors declare no conflict of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Keywords: A549 cells · Anti-cancer · GC-MS analysis · Maresia nana · Ras protein

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