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Myristica fragrans phytochemicals in inhibition of cancer cell adhesion - An *in silico* study

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Abstract:
Phytochemicals from Nutmeg are reported to have anticancer activity by multiple mechanisms; however, their effect on cell adhesion pathway is still not completely explored. Therefore, it is of interest to perform the *in silico* analysis of four major phytochemicals from Nutmeg against target enzymes of cell adhesion pathway. Ligands (Elemicin, Machilin A, Myristic and myricetin) were docked against targets (FAK, Src, ILK and ROCK1) where dasatinib was used as a positive control and their interaction was studied. Of the studied ligands, Myricetin showed the strongest and most consistent interaction with all four cancer-related kinases, while Machilin A showed moderate interaction and other two compounds showed relatively weak interactions. Thus, data shows that polyphenolic compounds from *Myristica fragrans* may exert anticancer activity through multi-target modulation of adhesion-related kinase signaling pathways involved in tumor progression and metastasis.

Keywords: *Myristica fragrans*, docking, adhesion pathway, myricetin, anti-cancer

Background:
Cancer progression is a multifactorial process involving dysregulated cell proliferation, evasion of apoptosis, metabolic reprogramming and enhanced migratory and invasive capabilities **[1].** Adhesion-associated kinases regulate the interaction between cells and the extracellular matrix and coordinate signaling pathways that control migration, invasion and survival of cancer cells. Dysregulation of these pathways contributes significantly to malignant transformation and metastatic dissemination **[2, 3].** One of the key mediators of cell adhesion signaling is Focal Adhesion Kinase (FAK), a non-receptor tyrosine kinase that localizes to focal adhesion complexes. FAK integrates signals from integrins and growth factor receptors to regulate cytoskeletal dynamics, cell survival and motility **[4]**. Overexpression or hyperactivation of FAK has been reported in multiple malignancies, including breast, colorectal, ovarian and pancreatic cancers and has been associated with enhanced tumor invasiveness and poor clinical prognosis **[5]**. Consequently, inhibition of FAK signaling has emerged as a promising therapeutic strategy for preventing tumor progression and metastasis **[6]**. Closely associated with FAK signaling is Proto-oncogene tyrosine-protein kinase Src (Src), a proto-oncogenic tyrosine kinase that participates in several oncogenic signaling cascades **[7].** Aberrant Src activation is a common feature in many cancers and contributes to tumor aggressiveness, resistance to apoptosis and enhanced invasive capacity **[8]**. Another critical component of integrin-mediated signaling is Integrin-linked kinase (ILK). ILK functions as an adaptor and signaling scaffold linking integrins to intracellular signaling pathways that regulate cell survival, proliferation and epithelial–mesenchymal transition (EMT) **[9]**. Dysregulation of ILK signaling has been implicated in tumorigenesis, particularly through its involvement in the PI3K or Akt pathway and regulation of β-catenin signaling **[10]**. In addition to these kinases, cytoskeletal tension and cellular contractility are strongly influenced by Rho-associated protein kinase 1 (ROCK1), a downstream effector of the Rho GTPase signaling pathway **[11]**. ROCK1 regulates actomyosin contraction, cell motility and focal adhesion dynamics. Inhibition of ROCK signaling has therefore been proposed as a strategy to reduce tumor invasion and metastasis **[12]**. Due to their central roles in regulating tumor cell adhesion, migration and survival, FAK, Src, ILK and ROCK1 represent important therapeutic targets in anticancer drug development. Several synthetic inhibitors targeting these kinases have been investigated; however, many of them are associated with toxicity, off-target effects, or limited clinical efficacy. Consequently, there is growing interest in identifying naturally derived compounds capable of modulating these signaling pathways with improved safety profiles **[13]**. Natural products have historically served as an important source of anticancer agents and numerous plant-derived phytochemicals exhibit anti-proliferative, anti-inflammatory and anti-metastatic activities. Among medicinal plants, *Myristica fragrans* has attracted considerable attention due to its diverse pharmacological properties **[14]**. Several phytochemicals isolated from *Myristica fragrans*, including Elemicin, Machilin A, Myricetin and Myristicin, have been reported to possess potential anticancer properties **[15]**. These compounds have shown inhibitory effects on cancer cell proliferation, induction of apoptosis and modulation of signaling pathways associated with tumor progression, in particular, polyphenolic compounds such as Myricetin are known to interact with multiple cellular targets and have been shown to interfere with kinase signaling pathways involved in cancer development **[16]**. Previous experimental studies have reported that *Myristica fragrans* has phytochemicals that have strong anti-cancer properties. Chirathaworn *et al.* (2007) has shown that methanolic extract of MF has inhibited Jurkat leukemia T cells by inducing apoptosis and reducing proliferation through downregulation of SIRT1 mRNA **[17]**. Akinboro *et al.* (2011) have shown antimutagenic

and antioxidant effects of MF methanolic extract **[18]**. Paul *et al.* (2013) have reviewed Macelignan and showed anticancer activity by multi-pathway modulation including oxidative stress and signaling pathways **[19]**. Suthisamphat *et al.* (2020) have used ethanolic mace extract on Kato III gastric cancer cells and found cytotoxicity and growth inhibition by anti-inflammatory and antioxidant mediated mechanisms **[20]**. Ginting *et al.* (2020) have used lignan from root extract on MCF-7 breast cancer cells and showed moderate cytotoxic activity associated with ROS-mediated oxidative stress **[21]**. Li *et al.* (2021) have used Dehydrodiisoeugenol on Colorectal cancer cells and showed Cell cycle arrest, autophagy, tumor growth inhibition **[22]**. Zhu *et al.* (2025) have shown that β-Sitosterol form MF has apoptotic action on T24 & 5637 bladder cancer cells **[23]**. Che *et al.* (2024) have used macelignan on colorectal cancer cells and found reduced metastasis and M2 polarization **[24]**. Piras *et al.* (2012) have showed that Nutmeg essential oil, myristicin inhibited growth of Caco-2 colon cancer cells **[25]**. Qian *et al.* (2025) have used Sabinene on HepG2 hepatocarcinoma and showed Apoptosis and tumor suppression **[26]**. Mickus *et al.* (2021) have used nutmeg essential oil constituents on novikoff hepatoma cells and reported reduced proliferation and colony formation **[27]**. Seneme *et al.* (2022) have used myristicin on NCI or ADR-RES ovarian MDR cells and have shown that it increased chemotherapy efficacy by inhibition of P-glycoprotein (MDR reversal) **[28]**. Despite increasing evidence supporting the anticancer potential of these phytochemicals, the molecular mechanisms underlying their activity remain incompletely understood **[29, 30].** Therefore, it is of interest to report the potential anticancer activity of selected phytochemicals derived from *Myristica fragrans* through molecular docking analysis against key adhesion-related kinases involved in tumor progression, namely FAK, Src, ILK and ROCK1.

Materials and Methods:

The active compounds that were reported to be present in the *Myristica fragrans* plant were carefully chosen based on their abundance and known anticancer activities. Hence only four compounds were selected, evaluated and compared by molecular docking analysis. The potential antitumor activity of phytochemicals derived from *Myristica fragrans*, molecular docking was performed against four key kinases involved in tumor progression and metastasis, viz. Focal Adhesion Kinase (FAK), Proto-oncogene tyrosine-protein kinase Src (Src), Integrin-linked kinase (ILK) and Rho-associated protein kinase 1 (ROCK1). Four phytochemicals were evaluated viz. Elemicin, Machilin A, Myricetin and Myristicin. **Figure 1** and **Figure 2** show the ligands and targets.

Protein preparation:

The structure (3D crystal) of adhesion targets and their pdb bumbers are shown in **Table 1.** They were downloaded from Protein Data Bank (www.rvcsb.org/pdb) they were cleared of non-essential water molecules, heteroatoms and the complexes attached to the receptor molecule. Then hydrogen atoms were supplied to the targeted molecule receptor.

Ligand preparation:

The identified crystallography structure of selected ligands was adopted from PubChem and was converted to PDB format using PyMOL software. They were cleaned of unwanted attached atoms and Gasteigar charges were added. Details of the ligands are shown in **Table 2.** At the binding location, the grid box size was set to 35 Å.

Docking softwares:

Scientiflow tool (from scientiflow.com) was used to automate the entire process which used RDKit for ligand format conversion and standardization, Gypsum-DL for tautomer and enentiomer generation, Gnina for docking with deep learning based scoring and based on Autodock vina. Protein ligand interaction profiling was done using PLIP and generations of 2D interaction maps were done using PandaMapColor. For ADMET prediction SwissADME was used (**Table 5**).

Results:

Among these compounds (**Table 3**), Myricetin exhibited the strongest binding affinity toward all four targets, with binding energies ranging from -9.83 to -10.49 kcal/mol, suggesting strong interaction with the catalytic domains of these proteins. Machilin A also exhibited significant binding affinity, especially with Src (-9.17 kcal per mol) and ROCK1 (-8.89 kcal per mol), indicating potential kinase modulatory activity. However, Elemicin and Myristicin exhibited comparatively weaker interactions with all proteins. The best docking positions are shown in **Figure 3**. For comparison, dasatinib was docked against the targets and it was seen that phytochemicals said were at par with dasatinib. It can be seen that all the molecules used showed a maximum of 1 violation in the rule of lipinski, making them good drugs for therapy. With regard to Binding Affinity Analysis, the docking results showed substantial variation in binding affinity among the tested compounds. About ligand–protein interaction analysis (**Tables 3 and 4**), Myricetin exhibited the highest number of hydrogen bonds and interaction contacts with the kinase domains. The higher number of hydrogen bond donors and acceptors present in Myricetin has contributed significantly to its strong binding affinity. Its rigid molecular structure with minimal rotatable bonds has allowed efficient stabilization within the ATP-binding pockets of these kinases. Machilin A showed strong hydrophobic interactions with kinase residues, particularly within the Src binding pocket, where six hydrophobic interactions and a π-stacking interaction were observed. Therefore it may be suggested that Machilin A may stabilize within the hydrophobic regions of kinase catalytic pockets. Elemicin and Myristicin exhibited fewer interaction contacts and lower hydrogen bonding potential. Another possible explanation may be its relatively small molecular size and limited hydrogen bonding capacity likely contributed to weaker binding energies. Also higher structural flexibility may contribute to weaker interactions. The interactions observed were primarily hydrophobic and suggest their limited capacity to strongly modulate kinase activity.

Table 1: Details of proteins

Enzyme	Pathway Role	PDB ID
FAK (PTK2)	Central adhesion signaling hub	2AL6
Src	FAK partner, adhesion phosphorylation	2SRC
ILK	Integrin–actin linkage regulator	3KMW
ROCK1	Cytoskeletal tension & adhesion maturation	2ETR

Table 2: Characteristics of the ligands

Parameter	Elemicin	Machilin A	Myricetn	Myristicin
Heavy atoms	15	24	23	14
H-bond donors (HBD)	0	0	6	0
H-bond acceptors (HBA)	3	4	7	3
Aromatic rings	1	2	3	2
Rotatable bonds	5	5	1	3
Molecular weight (g/mol)	208.25	326.39	312.19	192.21
logP	2.44	4.2	4.32	2.15

Table 3: Results of the molecular docking

Compounds	Enzyme	Binding Free energy Kcal/mol	Inhibition constant Ki µM	Amino Acid Residue interactions
Elemicin	FAK (PTK2)	-5.84491491	52	LEU99, GLU226, ASP124, ARG95
Machilin A	FAK (PTK2)	-7.6393795	2.51	PRO296, LYS345, GLN333, LYS289, GLU350, LEU346, THR271
Myricetin	FAK (PTK2)	-9.83408737	0.062	ARG95, CYS227, LEU99, ASP224, TYR317, ASP124, GLU128
Myristicin	FAK (PTK2)	-5.90495968	47.1	ARG97, GLU226, ARG95, ASP124
Elemicin	Src	-5.71626568	64.7	VAL281, MET341, LEU273
Machilin A	Src	-9.17219734	0.19	LYS295, THR338, PHE405, ILE336, VAL323, LEU393
Myricetin	Src	-10.4955339	0.02	THR338, ALA403, VAL281, LEU273, ASP404, MET341, LEU393, VAL323
Myristicin	Src	-6.14841938	31.4	LEU393, VAL281, ASP404, LEU273
Elemicin	ILK	-5.31857538	127	LEU199, VAL218, TRP271, ASN279, MET272, SER276
Machilin A	ILK	-8.42164993	0.67	VAL218, SER276, SER336, ASN279
Myricetin	ILK	-9.88888073	0.057	LYS220, ASN202, ASP339, ALA338, THR269, LEU207, MET272, VAL218, ASN200, SER336
Myristicin	ILK	-5.68254519	68.4	LEU207, LEU199, MET272, TRP271
Elemicin	ROCK1	-5.75066042	61.2	THR219, SER118, PHE120
Machilin A	ROCK1	-8.88844204	0.31	LYS105, THR219, PHE87, SER118, LEU107, PHE120
Myricetin	ROCK1	-9.8904171	0.057	ALA86, ILE82, ARG84, LEU106, VAL90, LYS105, PHE87
Myristicin	ROCK1	-6.11376286	33.3	ASP146, TYR39, GLN391, PHE43

Table 4: Binding affinity

Compound	FAK (2AL6)	Src(2SRC)	ILK (3KMW)	ROCK1 (2ETR)
Elemicin	-5.84	-5.72	-5.32	-5.75
Machilin A	-7.64	-9.17	-8.42	-8.89
Myricetin	-9.83	-10.49	-9.88	-9.89
Myristicin	-5.90	-6.15	-5.68	-6.11
Dasatinib	-7.799	-8.527	-8.704	-7.902

Table 5: ADMET prediction

Molecule	Canonical SMILES	MR	TPSA	iLOGP	XLOGP3	WLOGP	MLOGP	Silicos-IT Log P	Consensus Log P	ESOL Log S
Elemicin	C=CCc1cc(OC)c(c(c1)OC)OC	60.02	27.69	2.89	2.53	2.44	1.97	3.06	2.58	-2.69
Machilin A	C[C@H]([C@@H](Cc1ccc2c(c1)OCO2)C)Cc1ccc2c(c1)OCO2	92.06	36.92	3.78	5.35	4.2	3.62	5.16	4.42	-5.27
Myricetin	Oc1cc(O)c2c(c1)oc(c(c2=O)O)c1cc(O)c(c(c1)O)O	80.06	151.59	1.08	1.18	1.69	-1.08	1.06	0.79	-3.01
Myristicin	C=CCc1cc(OC)c2c(c1)OCO2	53.1	27.69	2.67	2.94	2.15	1.7	2.99	2.49	-3
Molecule	ESOL Solubility (mg/ml)	ESOL Solubility (mol/l)	ESOL Class	Ali Log S	Ali Solubility (mg/ml)	Ali Solubility (mol/l)	Ali Class	Silicos-IT LogSw	Silicos-IT Solubility (mg/ml)	Silicos-IT Solubility (mol/l)
Elemicin	0.424	0.00204	Soluble	-2.76	0.364	0.00175	Soluble	-3.64	0.0479	0.00023

Machilin A	0.00174	0.00000532	Moderately soluble	-5.88	0.000432	0.00000132	Moderately soluble	-6.08	0.000274	0.000000839
Myricetin	0.314	0.000988	Soluble	-3.96	0.035	0.00011	Soluble	-2.66	0.698	0.00219
Myristicin	0.191	0.000993	Soluble	-3.18	0.126	0.000655	Soluble	-3.13	0.142	0.000738
Molecule	**Silicos-IT class**	GI absorption	BBB permeant	Pgp substrate	CYP1A2 inhibitor	CYP2C19 inhibitor	CYP2C9 inhibitor	CYP2D6 inhibitor	CYP3A4 inhibitor	log Kp (cm/s)
Elemicin	Soluble	High	Yes	No	Yes	No	No	No	No	-5.77
Machilin A	Poorly soluble	High	Yes	No	No	Yes	Yes	Yes	Yes	-4.49
Myricetin	Soluble	Low	No	No	Yes	No	No	No	Yes	-7.4
Myristicin	Soluble	High	Yes	No	Yes	No	No	No	No	-5.39
Molecule	Lipinski #violations	Ghose #violations	Veber #violations	Egan #violations	Muegge #violations	Bioavailability Score	PAINS #alerts	Brenk #alerts	Leadlikeness #violations	Synthetic Accessibility
Elemicin	0	0	0	0	0	0.55	0	1	1	2
Machilin A	0	0	0	0	1	0.55	0	0	1	3.5
Myricetin	1	0	1	1	2	0.55	1	1	0	3.27
Myristicin	0	0	0	0	1	0.55	0	1	1	2.4

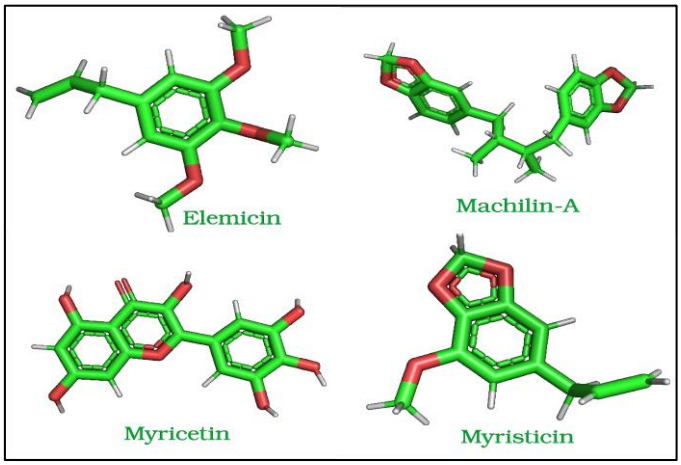


Figure 1: 3D images of ligands

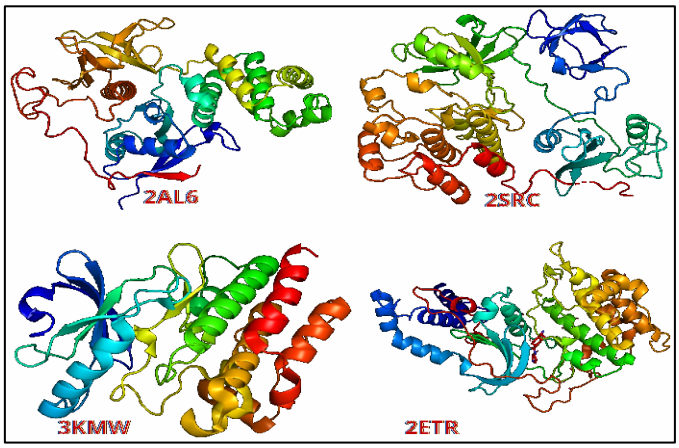


Figure 2: 3D Images of the targets used in the study

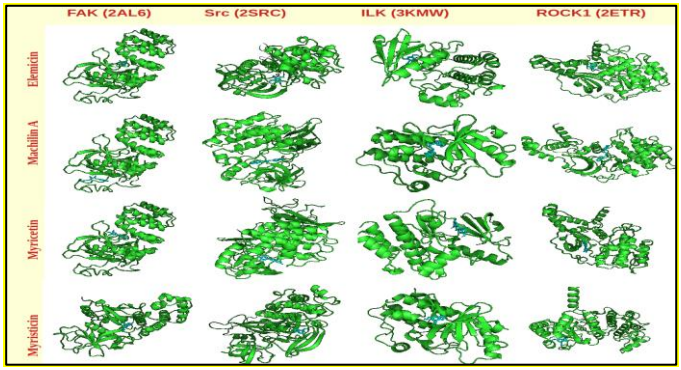


Figure 3: Best docking positions of the ligands

Discussion:

In the literature, the screening of phytochemicals has been performed against several targets for ascertaining their therapeutic potential **[31, 32]**. The current molecular docking study evaluated the interaction of phytochemicals derived from *Myristica fragrans* with major adhesion-related kinases that are involved in tumor progression and metastasis. The results of the present study showed that among the evaluated phytochemicals evaluated, Myricetin had the strongest binding affinity across all the protein targets used. This was followed by Machilin A, while Elemicin and Myristicin had comparatively weaker interactions. The high binding affinity seen in Myricetin may be suggestive of this compound acting as a potential multi-target modulator of the studied adhesion signaling pathway. The binding energies ranging from -9.83 to -10.49 kcal/mol across the kinases, added with multiple hydrogen bond interactions with catalytic site residues may support this interpretation. These observations are consistent with previous studies reporting polyphenol compounds like Myricetin having strong anticancer properties.

These findings are indicative of the interfering potential of Myricetin with Src catalytic activity and consequent disruption of downstream oncogenic signaling cascades. Same observation is true for ILK and ROCK1. It is suggestive of role of Myricetin in disrupting integrin-mediated signaling networks. The strong binding affinity of Myricetin toward ROCK1 may indicate inhibition of ROCK-mediated cytoskeletal remodeling and tumor cell migration. Therefore Myricetin may exert synergistic anticancer effect via disruption of interconnected signaling pathways. With regard to Machilin A, with strong affinity towards Src and ROCK1, along with its extensive hydrophobic interactions with catalytic site residues, it may be suggested that it may stabilize within kinase binding pockets and lead to effects similar to Myricetin. Elemicin and Myristicin had weaker binding interactions across all targets, ruling out their effect via the adhesion kinase pathway. Summarily, the results of this study show that phytochemicals from *Myristica fragrans*, especially Myricetin, may be a potential candidate for multi-target based cancer inhibition and metastasis. This ability of a single compound to involve with multiple signaling proteins is an essential advantage in anticancer drugs. This significant finding has provided insights and future directions for in vitro and clinical verification for validating the role of these phytochemicals in tumor treatment. From the literature, the most frequently reported mechanisms were Apoptosis induction, oxidative stress modulation, PI3K/AKT pathway inhibition, ER stress–induced autophagy and Multidrug resistance reversal **[33]**. Essentially these are downstream of FAK-Src signalling pathways. This study has analyzed the phytochemicals of *Myristica fragrans* for kinase related inhibitory effect, which are a novel and more relevant pathway for anti-cancer activity. In conclusion, the findings of the present study highlight Myricetin as a promising multi-target inhibitor of adhesion-related kinases involved in cancer metastasis. By targeting key components of the FAK–Src–ILK–ROCK signaling axis, this compound may interfere with critical processes such as tumor cell migration, invasion and survival. These results provide a mechanistic basis for further investigation of *Myristica fragrans* phytochemicals as potential anticancer agents. Future studies may have to focus on using **molecular dynamics simulations** to assess the stability of ligand–protein complexes over time. Similar to current study, Okselni *et al.,* (2026), have analysed the anti-tumor potential of components from mace essential oil on breast cancer cells and found above 70% inhibition. There *in silico* analysis also corroborate with the experimental results **[34]**. ***In vitro* kinase inhibition assays** and animal studies need to be performed for clinical translation of these findings. Drugs may be developed from derivatives of Myricetin and Machilin A for use in cancer therapy. De silva *et al.,* (2026) have reviewed about antimicrobial properties of spices and their potential to disrupt the biofilms. In this context they have observed that phytochemicals analysed in this study, namely myristicin and elemicin have been shown to possess good antimicrobial activity. Also, a concern on toxicity of myristicin is also pointed out **[35]**. Similar to current work, Marulasiddaswamy *et al.,* (2021) has performed *in silico* studies on anti-inflammatory potential of Fragransol B from *Myristica dactyloides* and found its multi target nature. In current study, similar multitarget activity is seen for these molecules analysed **[36]**.

Conclusion:

The molecular docking study results suggest that Myricetin shows the strongest and most consistent interaction with all four cancer-related kinases. Machilin A shows moderate to strong binding, particularly toward Src and ROCK1. Elemicin and Myristicin display relatively weak interactions. Thus, data shows that polyphenolic compounds from *Myristica fragrans* may exert anticancer activity through multi-target modulation of adhesion-related kinase signaling pathways involved in tumor progression and metastasis.

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