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NATURAL OILS AND THEIR ANTI CARCINOGENIC PROPERTIES

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ABSTRACT: -

The constituents of essential oils are having an important characteristics constituent that can be used in cancer prevention and treatment. The Monoterpenes, sesquiterpenes, oxygenated monoterpenes, oxygenated sesquiterpenes, phenolics, and other essential oil constituents from aromatic herbs and dietary plants include monoterpenes, sesquiterpenes, oxygenated sesquiterpenes, phenolics, and others shows anticarcinogenic, antioxidant, antimutagenic, anti-proliferative, and enhancement of immunity are just a few of the mechanisms. Enzyme induction and detoxification enhancement, immune functions and surveillance multi-drug resistance modulation and synergistic mechanisms of volatile constituents are discussed. Their chemo-preventive properties are due to them. This review focuses on the most recent releases to summarize structural categories and molecular anticancer mechanisms in the literature aromatic herbs and dietary plants constituents.

KERWORD :- Essential Oil, Anticarcinogenic, Anti mutagenic, Chemo-preventive

INTRODUCTION

Herbs and herbal products are staging a comeback and herbal ‘renaissance’ is happening all over the globe. The herbal products today symbolize safety in contrast to the synthetics that are regarded as unsafe to human and environment.[1]

Essential oils of plant origin are one of the important products of agriculture-based industry. They are commonly used as flavouring agents in food products, drinks, perfumeries, pharmaceuticals and cosmetics.

Cancer is a worldwide public health problem, which involves uncontrolled growth of cells. The cells lose their interaction with each other, invade neighbouring tissues and finally spread to distant tissues of the body.

Aromatic plants have been used since ancient times for their medicinal properties. These properties can be partially or wholly attributed to their volatile oil fractions (essential oils).[4]

Volatile oils, also known as essential oils are lipophilic compounds containing volatile aroma compounds. The constituents of the oils are mainly monoterpenes and sesquiterpenes which are hydrocarbons with the general formula (C5H8)n.

1: -CANCER

1.1 CARCINOGEN

A carcinogen is a specific chemical or physical agent that can cause cancer in individuals exposed to that agent. Most human cancers result from exposure to environmental cancer-causing agents; those encompass each natural and artificial chemicals, radiation, and viruses. Carcinogens can be divided into numerous classes, (1) Genotoxic cancer-causing agents, if they react with nucleic acids. These may be without delay performing or number one cancer causing agents, if they are of such reactivity on the way to without delay influence cellular constituents. (2) Alternatively, they will be procarcinogens that require metabolic activation to result in carcinogenesis. (3) Epigenetic cancer-causing agents are those who aren't genotoxic. Molecular variety of the cancer-starting up compounds levels from metals to complicated natural chemicals (table. 1), and there may be big version in efficiency.

Table 1
Types of carcinogens (Timbrell, 2000)

Type	Example
1. Genotoxic carcinogen	
Primary, direct-acting alkylating agents	Dimethylsulfate, ethylene imine, β-propiolactonel
2. Procarcinogens	
Polycyclic aromatic hydrocarbons	Benzo[*a*]pyrene
Nitrosamines	Dimethylnitrosamine
Hydrazine	1,2-Dimethylhydrazine
Inorganic	Cadmium, plutonium
3. Epigenetic carcinogens	
Promotors	Phorbol esters, saccharin, bile acids
Solid state	Asbestos, plastic
Hormones	Estrogens
Immunosuppressants	Purine analogues
Cocarcinogens	Catechol
4. Unclassified	
Peroxisome proliferators	Clofibrate, phthalate esters

1.2) CELL CYCLE

The process of replicating DNA and dividing a cell can be described as a series of coordinated events that compose a ‘‘cell division cycle,’’ illustrated for mammalian cells in Fig. 1 (see legend for details).

At least two types of cell cycle control mechanisms are recognized: a cascade of protein phosphorylation’s that relay a cell from one stage to the next and a set of checkpoints that monitor completion of critical events and delay progression to the next stage if necessary.

The first type of control involves a highly regulated kinase family (2). Kinase activation generally requires association with a second subunit that is transiently expressed at the appropriate period of the cell cycle; the periodic ‘‘cyclin’’ subunit associates with its partner ‘‘cyclin-dependent kinase’’ (CDK) to create an active complex with unique substrate specificity. Regulatory phosphorylation and dephosphorylation fine-tune the activity of CDK–cyclin complexes, ensuring well-delineated transitions between cell cycle stages. In the future, additional molecular definition of the cell cycle may lead to a more intricate progression than indicated in Fig. 1.

A second type of cell cycle regulation, checkpoint control, is more supervisory. It is not an essential part of the cycle progression machinery. Cell cycle checkpoints sense flaws in critical events such as DNA replication and chromosome segregation. When checkpoints are activated, for example by under replicated

or damaged DNA, signals are relayed to the cell cycle-progression machinery. These signals cause a delay in cycle progression, until the danger of mutation has been averted. Because checkpoint function is not required in every cell cycle, the extent of checkpoint function is not as obvious as that of components integral to the process, such as CDKs.

The first genetic alterations shown to contribute to cancer development were gain-of-function mutations.

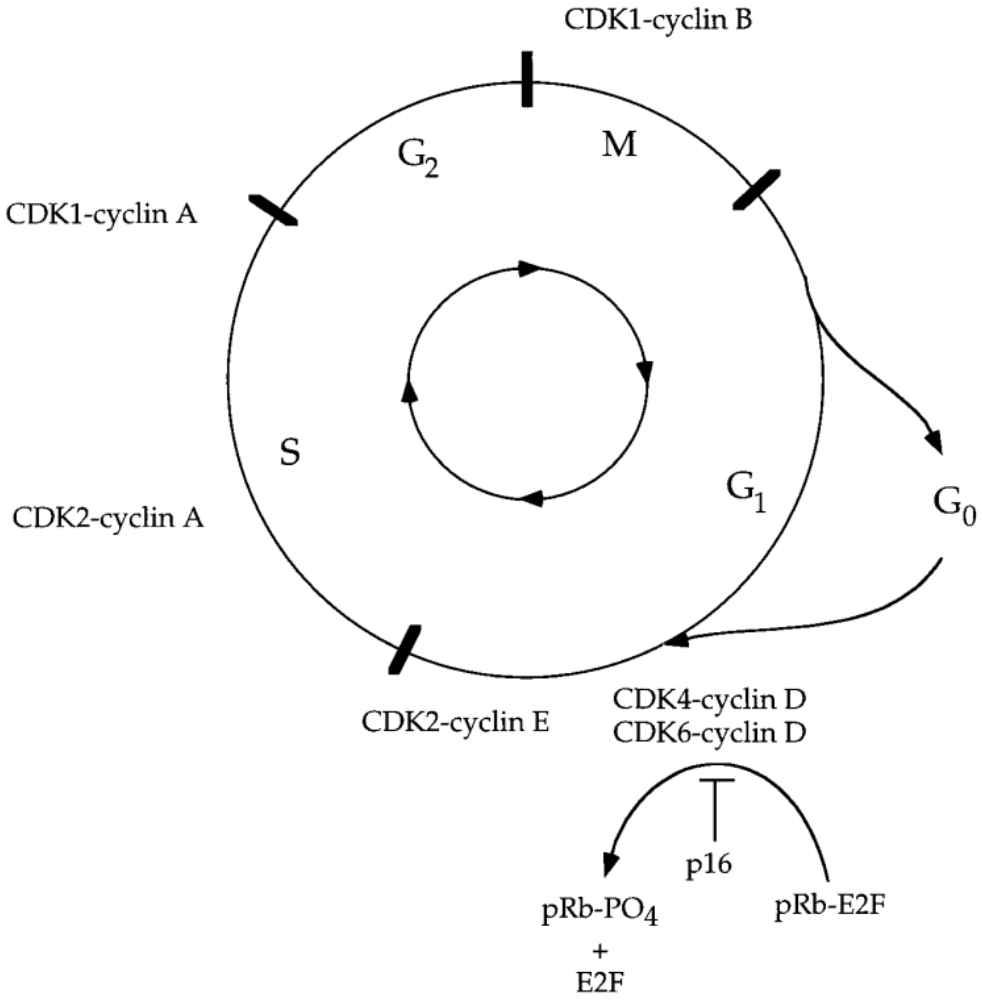


FIG. 1. A schematic representation of the mammalian cell cycle. In each cell division cycle, chromosomes are replicated once (DNA synthesis or S-phase) and segregated to create two genetically identical daughter cells (mitosis or M-phase). These events are spaced by intervals of growth and reorganization (gap phases G1 and G2). Cells can stop cycling after division, entering a state of quiescence (G0). Commitment to traverse an entire cycle is made in late G1. Progress through the cycle is accomplished in part by the regulated activity of numerous CDK–cyclin complexes, indicated here and described in the text.

These mutations define a set of ‘‘oncogenes’’ that are mutant versions of normal cellular ‘‘protooncogenes.’’ The products of protooncogenes function in signal transduction pathways that promote cell proliferation. However, transformation by individual oncogenes can be redundant (mutation of one of several genes will lead to transformation) or can be cell type-specific (mutations will transform some cells but have no effect on others). This suggests that multiple, distinct pathways of genetic alteration led to cancer, but that not all pathways have the same role in each cell type.

Thus, unlike gain-of-function mutations, loss-of function tumour suppressor mutations can be carried in the gene pool with no direct deleterious consequence. However, individuals heterozygous for tumour suppressor mutations are more likely to develop cancer, because only one mutational event is required to prevent synthesis of any functional gene product.

Loss of function of the tumour suppressor gene product pRb, for example, would be predicted to liberate E2F transcriptional activators without requiring phosphorylation and thus bypass a normal negative regulation controlling entry into the cycle (Fig. 1). Loss of the tumour suppressor gene product p16 would have a similar consequence, liberating E2Fs by increasing pRb phosphorylation (Fig. 1). In addition, cell cycle progression can be halted at several points by the tumour suppressor gene product p53, activated in response to checkpoints sensing DNA and possibly also chromosome damage; loss of p53 would remove this brake to cycling.

2.Mechanisms of action of EO on carcinogenesis

The genes that control apoptosis have a major effect on malignancy through the disruption of the apoptotic process that leads to tumour initiation, progression, and metastasis.

The p53 protein, encoded by a tumour suppressor gene, mediates growth arrest or apoptosis in response to a variety of stresses. p53-Dependent apoptosis, occurring in several sensitive tissues after radiation or chemotherapy, is partially responsible for the side effects of cancer treatment, making p53 a potential target for therapeutic suppression. Hypoxic stress, such as DNA damage, induces p53 protein accumulation and p53-dependent apoptosis in ontogenically transformed cells.

2.1)Induction of Apoptosis. Apoptosis can occur due to effect on various signalling pathways, genetic material, and other cellular events like changes in the proteins at the intracellular level.

A study on human melanoma cells reported that treatment of EOs induces DNA damage in cancer cells which is an indicator of apoptosis. Apart from DNA damage, modification of various genes by the action of EOs is also responsible for apoptosis.

EOs were also demonstrated to change expression levels of Bcl-2 and Bax genes leading to release of cytochrome C into cytosol in KB human oral epidermoid carcinoma cells

This happens via activation of caspase-9 leading to caspase3 formation which in turn cleaves target that causes apoptosis and increased phosphorylation of extracellular signal regulated kinase (ERK), c-jun N-terminal kinase, and p38 MAPK

EO-induced apoptosis has been also suggested to be involving mitochondrial and MAPKs pathways

2.2)Cell Cycle Arrest.:- Mammalian cells have different cell cycle phases (G1, S, G2, and metaphase) to complete their life cycle. Fidelity of the cell cycle is lost due to the lack of response to the negative regulators of cell cycle progression in the cancer cells leading to uncontrolled cell division.

Regulation of the genes involved in this process is also hampered. Thus, halting any cell cycle event in the cancer cell leads to prevention of their growth and division, a widely employed therapeutic strategy

Many, EO has been reported to upregulate p21 expression and suppress cyclin D1 and cyclin-dependent kinase 4 (CDK4) expression in colorectal cancer cells with increase in dose

As p21 is negative regulator of G1 phase transition, increased expression of this protein by the action of patchouli alcohol is indicative of cell cycle inhibition

2.3)Effect on Detoxification Enzymes. Genotoxins lead to alteration of the internal antioxidants and antioxidant enzymes like superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and glutathione reductase (GR) along with alteration of various important body functions resulting in damage to tissues and membranes. Phase I and phase II detoxification enzymes are responsible for the degradation of the harmful compounds.

Certain compounds of the Eos act as an inducer of those detoxification enzymes and thus stop the induced-toxicity and even cancer within the cell line models.

2.4)Modulation of Deoxyribonucleic Acid damage and Repair signalling by EOs. enhanced ROS production (as mentioned above) results in DNA damage and can cause the death.

EOs have shown the induced damage at the deoxyribonucleic acid level that drives the cancer cells towards death. This activity is very harmful in cancer cells, whereas no such harm is encountered in the normal cells; this provides additional advantage of using these EOs.

Targeting DNA repair pathways is an effective treatment method currently in use in the cancer to encounter the high proliferation rate in the cancer cells.

One of the peculiar properties of the EOs is that though being cytotoxic to cancer cells, these induce proliferation of the normal cells [88].

Effects of EOs on various types of cancer

Prostate cancer

Apoptosis is an important cell method in the homeostasis of multicellular organisms and its regulation has been involved in several human tumours, as well as prostate cancer [41]. Recently, it's been reported that caryophyllene oxide selectively affected cancer cells [42] and synergistically potentiated the paclitaxel anticancer activities in the DU-145 human prostate cancer cell line [43]. Antitumor activities of caryophyllene oxide could also be exerted through suppression of cellular growth and induction of apoptosis [44]. In particular, the oxygenated sesquiterpene suppressed PC-3 prostate cancer cell proliferation in a dose-dependent manner. Moreover, it induced oxygen species generation, Mitogen-activated protein kinase (MAPK) activation, and inhibition of the PI3K/AKT/mTOR/S6K1 signal pathway in these cells, a pathway that is important in cell survival, proliferation, and angiogenesis of the tumour [45]. Moreover, equivalent authors found that it considerably reduced levels of pro-cancer proteins, those concerned with proliferation, cyclin D1, metastasis Cox (cyclooxygenase 2), growing VEGF (vascular epithelial growth factor), and caspase-mediated cell death inhibitors bcl-2, bcl-xL, IAP-1, IAP-2 (inhibitor of caspase-mediated cell death apoptosis and 2), and surviving.

Glioblastoma

Adult glioblastoma (GBM) is one of the most deadly and recalcitrant of all malignant solid tumours. Despite considerable effort, little progress has been made toward prolonged survival in GBM, with much of the perceived improvement coming from the recognition of two prognostic biomarkers: mutations in isocitrate dehydrogenase (IDH) and O6-methylguanine-methyltransferase (MGMT) promoter methylation.1850

The activation of caspase-9 indicated that the effects Essential oils on GBM cells are mediated by the intrinsic (mitochondrial) pathway of apoptosis. . In addition to the release of proapoptotic proteins, the generation of reactive oxygen species (ROS) is used by the intrinsic pathway to induce apoptosis in tumours cells. Due to the high content of ROS, it has been proposed that insults leading to further ROS generation would turn cancer cells very susceptible to ROS damage.(1)

Melanoma

Melanoma is a malignancy of melanocytes, which are pigment-producing cells of neuroectodermal origin that can be found throughout the body (including in the skin, iris and rectum). Sun (UV) exposure is the major risk factor for cutaneous melanoma and leads to a genetic signature that is characteristic of melanoma.(2)

Several studies have found that p38 is a key intracellular signalling molecule for pigmentation and that activation of the p38 MAPK pathway has been reported to be related to an increase in melanin synthesis. Moreover, JNK1/2 and ERK1/2 are related to the regulation of melanogenesis through MITF and tyrosinase activities. M-EO decreases the production of melanin through the downregulation of tyrosinase activity and this effect is associated with the regulation of the JNK and ERK signalling pathways in a-MSH stimulated B16 cells.(3)

Breast cancer

Breast cancer is categorized into 3 major subtypes based on the presence or absence of molecular markers for estrogen or progesterone receptors and human epidermal growth factor 2 (ERBB2; formerly HER2): hormone receptor positive/ERBB2 negative (70% of patients), ERBB2 positive (15%-20%), and triple-negative (tumors lacking all 3 standard molecular markers; 15%).(4)

γ-bisabolene was shown to activate p53-mediated apoptosis through protein phosphatase1/histone deacetylase 2 (HDAC2) and extracellular signal-regulated kinase signalling. It is possible that the higher levels of HDAC2 observed in breast cancers5 play an essential role in mitigating the pro-apoptotic effects of p53, making breast cancer cells more susceptible to inhibition of HDAC2 by bisabolene.(5)

ANTI CANCER ACTIVITY OF OILS

Garlic oil

Garlic (Allium sativum L.) is amongst the oldest plants cultivated for its utilization as food and medicine.

Chemical Constituents of Garlic: chemical Constituents of Garlic: There are more than two hundred chemical compounds in the Garlic bulb, of which, volatile oil with sulphur containing compounds like Ajoene (4,5,9-trithiadodeca-1,6,11-triene-9-oxide), Alliin and Allicin, enzymes like peroxidase, alliinase, myrosinase and other compounds like -phellandrene.(6)

One of the major active constituents of garlic is Diallyl sulphide (DAS), which is the major factor for growth reversion of in vitro cancer cells and advances in vivo immune responses in trial settings. The study involved investigations of the anti-cancer effects of DAS in Hela human cervical cancer cells and to examine the fundamental mechanisms in vitro. Apoptosis and cytotoxicity in human cervical cancers were investigated by viability assay, morphological changes, comet assay, DAPI staining, confocal microscopy examination and Western blotting. Therefore, the study revealed that DAS considerably retrain the growth and instigates apoptosis of Hela human cervical cancer cells in vitro.(7)

The studies by various researchers confirmed the inhibitory activities of different chemical constituents of garlic against various carcinogens and mutagens, which revealed that garlic played a major role in cancer control and elimination (Table 1). These garlic compounds cause inhabitation against various carcinogenic compounds and control various cancers like buccal pouch, colon, skin, liver, forestomach and lung cancers in model animals like rat, mouse and hamster etc (Table 1).

Table 1: List of chemicals from garlic, experimental inhibition of cancer by garlic and antimutagenic activity of garlic constituents.

Chemicals	Carcinogen and Mutagens	Organ/Species
Fresh garlic extract	DMBA (7,12dimethylbenz(a) anthracene)	Buccal pouch/Hamster
Garlic oil	DMBA/PMA (phorbol myristate acetate)	Skin/Mouse
Diallyl sulfide (DAS)	DMH (1,2-dimethyl hydrazine),	Esophagus, Colon /Rat
	NMBA (N-nitrosomethyl benzylamine),	Lungs, Forestomach
	BP (benzo[a] pyrene), DMBA	Skin/Mouse
Allyl methyl sulfide	BP	Forestomach/Mouse
Diallyl trisulfide (DATS)	BP	Forestomach/Mouse
Allyl methyl trisulfide	BP	Forestomach/Mouse
Methanol extract of garlic	AFB1 (aflatoxin B1)	Liver/Rat
Fresh garlic powder	DEN (diethyl nitrosamine)	Liver/Rat
S-methyl cysteine (SMC)	DEN	Liver/Rat
Methanolic garlic extract	AFB1 (aflatoxin B1)	*Salmonella* TA98
Aqueous garlic extract	AFB1	*Salmonella* TA100 88
Aqueous garlic extract	4-NQO (4-nitroquinoline -1-oxide)	*E. coli*
Aqueous garlic extract	Gamma rays, Hydrogen peroxide	
	Cumene hydroperoxide, t-butyl hydroperoxide	*Salmonella* TA102
S-allyl cysteine (SAC)	DMBA	Buccal pouch/Hamster
Diallyl sulfide	PhIP (2-amino-1-methyl-6-phenylimidazo [4,5-b] pyridine)	Colon/ Rat
Garlic powder	PhIP	Colon/ Rat

Thyme oil :-

Chemical composition:- thymol 46.2% γ terpinene (14.1%), P-cymene (9.9%), linalool (4.0%), myrcene (93.5%), α-Pinene (3.%) and α -thujene (2.8%). Also ά- thujene ,ά- pinene, βpinene , octan-1-en-0-o1, myrcene, ά – phellandrene, ά – terpinene, p-cymene, limonene, 1,8-cineole, γ-terpinene, cis-sabinen hydrate, terpinolene, linalool, terpinen-4-ol, thymol methyl ether, thymol, carvacrol, terpinyl acetate, eugenol, β- bourbonene, β- elemene, methyl eugenol, β- caryophyllene, β- copaene, ά humulene, germacrene D, β- bisabolene, δ- cadinene, caryophyllene oxide.(8)

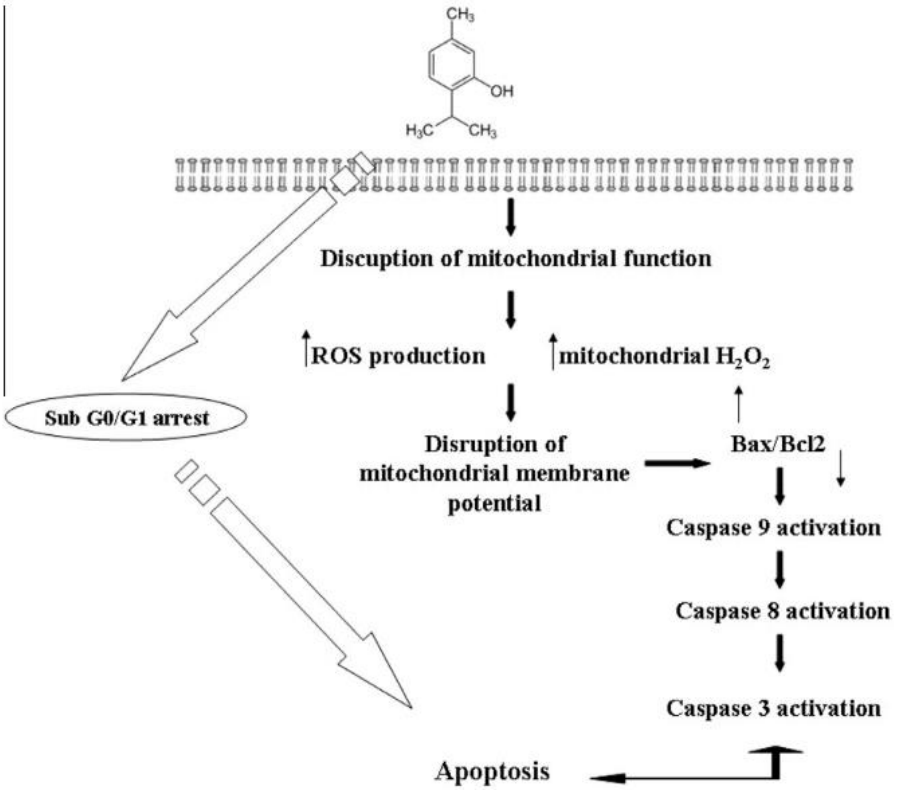
Thymol has been reported to exert anti-carcinogenic activity through a different mechanism of action on the cancer cells lines. These include oxidative stress and cancer cell death, apoptotic cancer cell death and antiproliferative effects on cancer cells. Contrary, antioxidant activity, protective effects, antiapoptotic effects, anti-inflammatory immunomodulatory effects and antigenotoxic effects may be the key mechanisms of thymol's anti-carcinogenic activity in normal cells.

Thymol's tumor selective growth inhibitory effect. It is evident from our result that thymol showed cell proliferation inhibition which is highly related to activation of variety of intracellular signaling pathways to arrest cell cycle in various phases.

Results indicated that treatment of thymol led to increase in cell accumulation at G0/G1 phase in a dose dependent manner with decreasing cell population in the S and G2/M phases. Damage caused to sub-G0/G1 phase arrest is irreversible and thus leads the cell to undergo apoptosis.

Apoptosis induced by thymol occurred in a mitochondrial relevant manner. We observed that thymol led to a sequential disruption of **ΔΨ**m with decrease in the Bcl-2 protein and increase in Bax protein expression levels and also activated caspases. Such changes in Bax/Bcl- 2 ratios have been known to induce apoptosis in cells.(9) and (10).

Studies also revealed that Bcl-2 opposes cell death by regulating cellular redox status.(11) Thymol showed a significant increase in mitochondrial H2O2 production and also disruption of **ΔΨ**m. Thus, it may be hypothesized that the increase in intracellular H2O2 would have promoted mitochondrial outer membrane permeabilization and released pro-apoptotic proteins such as AIF and increase in Bax level, which had led, the cell to undergo apoptosis.(12)



Scheme 1. The schematic drawing represents the proposed mechanism by which thymol caused apoptosis in HL-60 cells. This diagrammatic representation compiles the result and conclusion of this report.

CLOVE OIL:-

Chemical composition:-

Various studies have been carried out to find various constituents of S. aromaticum [10-12]. Clove buds contain 15–20% essential oil, which is dominated by eugenol (70–85%), eugenyl acetate (15%) and β-caryophyllene (5–12%). Other essential oil ingredients of clove oil are vanillin, crategolic acid, tannins, gallotannic acid, methyl salicylate, flavonoids eugenin, kaempferol, rhamnetin, eugenitin and triterpenoids like oleanolic acid. The constituents of the oil also include methyl amyl ketone, methyl salicylate, α and β-humulene, benzaldehyde, β-ylangene and chavicol. (13)

Clove essential oil has also been reported to show anticarcinogenic.(14) and antimutagenic potential because of its strong free radical scavenging activity.(15). Several Preliminary studies suggested chemo preventive role of clove oil, particularly in cases of lung, skin and digestive cancers.(13) Ethyl acetate extract of clove inhibits tumor growth and promotes cell cycle arrest and apoptosis. Oleanolic acid one of the components of ethyl acetate extract of clove was found to be responsible for its antitumor activity. Its mechanism was attributed to the promotion of Go/G1 cell cycle arrest and induction of apoptosis in a dose-dependent manner.(13) Eugenol acts as a potential molecule that can interfere with several cell lsignaling pathways, specifically the NF-κB. In an another study, eugenol was found to suppress growth of malignant melanoma WM1205Lu of both anchorage-dependent and anchorage independent growth, decreased size of tumors and inhibited melanoma invasion and metastasis by the inhibition of two transition factors of the E2F family. (16) Hussain et al. studied the effect of eugenol combined with gemcitabine on cervical carcinoma and found that the combination of eugenol and gemcitabine can inhibit cancer cell growth, even in low concentrations . Studies on related gene also found that eugenol can reduce the possibility of apoptosis of B-cell lymphoma-2 (Bcl-2), Cyclooxygenase-2 (COX-2), and interleukin-1β (IL-1β), reduce inflammation, and increase the treatment efficacy of gemcitabine. Moreover, Eugenol showed better curative effects in skin cancer and melanoma.(13)

Olive Oil

Chemical constituent:- (z)-3-hexanol,(E)-2-Hexenol, Į –pinene, ù – pinene, (Z)-3-Hexenyl acetate, 2-Methoxy-3-isopropylpyrazine, (E,Z)-2,6-Nonadienal, (E,Z)-2,6-Nonadienol, 1,4-Dimethoxybenzene, Decanal, 2,6-D(17)imethyloctane, 2,4-Dimethyldodecane, 2,6,11-Trimethyldodecane, Į-ionone, Į-humulene, 1-Dodecanol, ù -Ionone, hexadecanoicacid, Oleic acid, Docosane, Tetracosane .(17)

The apoptotic mechanism triggered by JFE treatment was also studied through the phosphorylation level of Akt (protein kinase B) and Erk (extracellular signal-regulated kinases) as well as the expression of p53. Indeed, Akt, one of the major signaling enzymes involved in cell survival against oxidative stress, was shown to suppress apoptosis and promote cell survival.(18) Hence, the influence of JFE on Akt phosphorylation was determined using an anti-phospho-Akt monoclonal antibody. Moreover, Erk belonging to the MAPK subfamilies have been shown to be activated in response to oxidant injury, thus contributing to apoptosis.(19) Apoptosis is also considered to be regulated by p53 protein which is inactive in normal cells unless cells are exposed to stress signals. (20)

Fennel oil:-

Chemical constituents:- α-Ethyl-p-methoxybenzyl alcohol, p-Anisaldehyde, Carvone, 1-Phenylpenta-2,4-diyne, Fenchyl butanoate, Neomenthol, (2E)-Dodecenal, β-Ethyl-p-methoxybenzyl alcohol, trans-Thujone, Fenchone, Carvacrol, Linalyl acetate, (E)-Chrysanthenyl acetate, Thymol, Fenchyl isobutanoate, (E)-β-Terpineol, Linalool, cis-Thujone, (E)-Dihydrocarvone, Geranial, Myrtenyl acetate, exo-Fenchyl acetate, Penta-1,3-diynylbenzene, Dill ether, Methylchavicol (=estragole), Caryophyllene oxide, Camphor, iso-Menthone, 1-Hexadecene, Terpene hydrocarbons, Oxygenated terpenoids, The main three compounds (trans-anethole, p-anisaldehyde, α-ethyl-p-methoxybenzyl alcohol) are both ethers, having methoxy functional groups.(21)

The cytotoxicity of F. vulgare oil is most likely due to the lipophilic properties of essential oil and alkylating properties of the major components trans-anethole and p-anisaldehyde. Caco-2 and CEM/ADR5000 overexpress the ABC transporter p-gp which can actively pump out any lipophilic compound that has entered the cell by free diffusion.(22) Thus, both cell lines are rather insensitive towards lipophilic cytotoxic agents. In contrast, the parent cell line CCRF-CEM should be sensitive. We also suspect that some components of the essential oil are may be substrates for p-gp, as IC50 values were higher in CEM/ADR5000 cells.(23)

Conclusion:-

According to the World Health Organization , incidence and mortality of cancer is increasing worlwide. The various treatments require a careful selection of one or more of existing modalities such as surgery, radiotherapy and systemic therapy.(24)

Essential oils have been shown to positively effect the immune system on a chemical level despite their direct effect on tumour cell. For removing foreign material and microbes from the body, essential oil enhance the activity of white blood cells making them more efficient.(25)

This review presents evidence that agents derived from plants used in Ayurvedic medicine can be used not only to prevent cancer, but also to treat cancer. Because of their pharmacological safety, these agents can be used alone or as adjuncts to current chemotherapeutic agents to enhance therapeutic effects and minimize chemotherapy induced toxicity.(26)

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