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The Effect of Essential Oils on DNA

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Abstract

Essential oils are known to be substances with antimicrobial properties, inhibiting the growth of bacteria, fungi, some parasites, and even viruses. Their mechanisms of action have been described. They form pores in cell membranes, causing the release of cytoplasmic contents, the dissipation of the proton gradient, and a decrease in intracellular ATP levels. It has also been determined that essential oils can affect nucleic acids. Therefore, this work shows the effect of essential oils on DNA.

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Introduction

Essential oils are complex mixtures of volatile organic compounds, synthesized by plants as secondary metabolites, primarily obtained by hydrodistillation (Álvarez-Martínez *et al.,* 2021; Pezantes-Orellana *et al.,* 2024; Sadgrove *et al.,* 2022). Essential oils are composed of terpenes and phenylpropanoids. Monoterpenes and sesquiterpenes are the most abundant, and their oxygenated derivatives (alcohols, aldehydes, ketones, esters, ethers, phenols, oxides) are the primary mediators of their bioactive properties. For example, thymol and carvacrol (monoterpenic phenols) are characteristic of genera such as *Thymus* and *Origanum*. Eugenol (a phenylpropanoid) is abundant in *Syzygium aromaticum*, and limonene (a monoterpene hydrocarbon) is the major component in citrus fruits (Haro-Gonzalez *et al.,* 2021; Kosakowska *et al.,* 2024; Masyita *et al.,* 2022; Orchard and van Vuuren, 2017; Pezantes-Orellana *et al.,* 2024; Sadgrove *et al.,* 2022). Essential oils have demonstrated a wide range of antimicrobial, antioxidant, anti-inflammatory, analgesic, insecticidal, antiparasitic, and anticancer activities (Belete *et al.,* 2025; Dhifi *et al.,* 2016; Han and Parker, 2017; Haro-Gonzalez *et al.,* 2021; Leutcha *et al.,* 2025; Masyita *et al.,* 2022; Niksic *et al.,* 2021; Orchard and van Vuuren, 2017; Rajkowska *et al.,* 2016). The antimicrobial activity of essential oils is fundamentally due to their ability to interact with the microbial cell membrane, altering its permeability, dissipating the proton gradient, inhibiting ion pumping, and causing the leakage of cellular components, which ultimately leads to cell death (Álvarez-Martínez *et al.,* 2021; Flores-Encarnación *et al.,* 2026*a*; Flores-Encarnación *et al.,* 2026*b*; Lanzerstorfer *et al.,* 2021; Masyita *et al.,* 2022). This membrane-level mechanism of action is especially relevant against Gram-positive bacteria, whose cell wall is more accessible than the lipopolysaccharide outer membrane of Gram-negative bacteria (Masyita *et al.,* 2022; Sallam *et al.,* 2022). Furthermore, essential oils can act as antioxidant agents

by neutralizing free radicals (for example, through the hydroxyl groups of phenolic compounds) and chelating transition metals, thereby protecting cells from oxidative damage (Haro-Gonzalez *et al.,* 2021; Leutcha *et al.,* 2025; Orchard and van Vuuren, 2017). Therefore, this work shows the most relevant aspects of the effect of essential oils on nucleic acids, particularly on DNA.

Essential Oil Activities on Microorganisms

Antimicrobial properties.

The antimicrobial activity of these essential oils has been extensively documented, acting primarily at the cell membrane level (Ezzariga *et al.,* 2025; Flores-Encarnación *et al.,* 2026*b*; Gómez-Sequeda *et al.,* 2020; Masyita *et al.,* 2022; Orchard and van Vuuren, 2017; Selles *et al.,* 2024). However, recent studies have demonstrated that, at concentrations near the minimum inhibitory concentration (MIC), these compounds can also penetrate bacterial and eukaryotic cytoplasm and interact directly with genetic material (Sallam *et al.,* 2022; Thapa *et al.,* 2019). For example, in oregano essential oil, carvacrol induces DNA fragmentation in *Escherichia coli* and human colon cancer cells, an effect reversed by antioxidants such as N-acetylcysteine, suggesting a mechanism mediated by reactive oxygen species (ROS) (Kozics *et al.,* 2025; Sallam *et al.,* 2022; Thapa *et al.,* 2019). Similarly, thymol protects DNA from oxidative damage, but not from alkylation damage, indicating selectivity toward certain types of lesions (Thapa *et al.,* 2019). The first step in its antimicrobial property is the interaction of its lipophilic components with the lipids of the cytoplasmic membrane. Compounds such as carvacrol, thymol, eugenol, and cinnamaldehyde possess a high oil/water partition coefficient, allowing them to insert into the lipid bilayer.

This insertion disrupts fatty acids and phospholipids (Lanzerstorfer *et al.,* 2021; Masyita *et al.,* 2022); structural alteration causes an increase in membrane fluidity and permeability, leading to the leakage of essential components such as ions (K^+, H^+, PO_4^{3-}), which results in the loss of the proton gradient and the collapse of the proton motive force necessary for ATP synthesis (Lanzerstorfer *et al.,* 2021; Masyita *et al.,* 2022). In Gram-negative bacteria, the outer membrane constitutes an additional barrier due to the presence of lipopolysaccharides; however, certain phenolic compounds (especially carvacrol) are capable of destabilizing this outer layer, releasing lipopolysaccharide (LPS) and increasing the permeability of the inner membrane, which facilitates the entry of other antimicrobial compounds (Lanzerstorfer *et al.,* 2021; Masyita *et al.,* 2022). In Gram-positive bacteria, the thick peptidoglycan cell wall does not prevent the access of essential oils to the cytoplasmic membrane, which explains the greater susceptibility of these bacteria to them (Lanzerstorfer *et al.,* 2021; Masyita *et al.,* 2022). Antimicrobial activity has been quantified by MIC, which for the most active compounds (carvacrol, thymol, eugenol, cinnamaldehyde) is typically in the range of 0.25–1 mg/mL against clinically important microorganisms (Masyita *et al.,* 2022; Sallam *et al.,* 2022). For example, oregano oil (carvacrol) has an MIC of 0.25–0.5 mg/mL against *Staphylococcus aureus* and *E. coli*, whereas thyme oil (thymol) shows an MIC of 0.5–1 mg/mL against *C. albicans* (Masyita *et al.,* 2022; Sallam *et al.,* 2022). Synergism has been described between different essential oil components (e.g., carvacrol with cinnamaldehyde) and also between essential oils, allowing for a reduction in the effective concentration and, therefore, the potential toxicity (Masyita *et al.,* 2022). This synergism is due to the fact that one compound can facilitate the entry of another by altering the membrane, or they may act on different regions of the cell envelope (Lanzerstorfer *et al.,* 2021; Masyita *et al.,* 2022).

Changes in cellular integrity and morphology

Electron microscopy studies have demonstrated that exposure of bacteria (e.g., *E. coli* or *S. aureus*) to inhibitory concentrations of essential oils such as oregano or thyme produces drastic morphological changes: cell shortening, formation of blebs (protrusions) on the surface, cytoplasmic disorganization, and cell lysis (Masyita *et al.,* 2022; Sallam *et al.,* 2022).

In yeasts such as *Candida albicans*, clove essential oil (eugenol) and thyme essential oil (thymol) induce loss of plasma membrane integrity, leakage of cytoplasmic material, and formation of anomalous pseudo-hyphae (Masyita *et al.,* 2022; Rajkowska *et al.,* 2016; Sallam *et al.,* 2022).

These effects do not require the specific inhibition of any enzyme; rather, they are a direct consequence of structural membrane damage. In fact, the addition of osmoprotectants (such as polyethylene glycol or sucrose) does not reverse the bactericidal activity of the essential oils, indicating that cell death is not solely due to osmotic shock, but rather to irreversible membrane disorganization (Lanzerstorfer *et al.,* 2021; Masyita *et al.,* 2022).

The Effect of Essential Oils on DNA

DNA binding

It is important to note that although the primary mechanism of essential oils is at the membrane level, sublethal concentrations or prolonged exposures allow some lipophilic components to penetrate the cytoplasm and access the nucleus (or bacterial nucleoid), where they can interact directly with DNA (Masyita *et al.,* 2022; Thapa *et al.,* 2019). This phenomenon is especially relevant in eukaryotic cells (such as enterocytes or hepatocytes) and tumor cells, where the nuclear membrane is not an absolute barrier for small apolar molecules (Masyita *et al.,* 2022; Thapa *et al.,* 2019). Therefore, the study of the effects on DNA is fundamental for understanding both the toxicity and the possible therapeutic applications of these compounds. Essential oil components can bind to DNA through three main modes: 1. Intercalation between base pairs. 2. Binding in the minor or major groove of the DNA double hélix. 3. Electrostatic binding to the phosphodiester bonds (Leutcha *et al.,* 2025; Liu *et al.,* 2021; Pezantes-Orellana *et al.,* 2024). Intercalation is characteristic of compounds with planar or nearly planar aromatic systems, such as flavonoids (apigenin, luteolin) and some phenylpropanoids (eugenol, safrole). These compounds insert between two adjacent base pairs, distorting the DNA helix and impeding transcription and replication (Leutcha *et al.,* 2025; Shahmoradi *et al.,* 2023; Thapa *et al.,* 2019). Minor groove binding is mediated by van der Waals interactions and hydrogen bonds, and is typical of smaller molecules such as thymol and carvacrol, which can establish weak bonds with the edges of nitrogenous bases (Masyita *et al.,* 2022; Thapa *et al.,* 2019; Yin *et al.,* 2024). Electrostatic binding, on the other hand, is nonspecific and depends on the positive charge of some compounds (e.g., volatile alkaloids or amines) that are attracted to the negatively charged phosphodiester backbone (Leutcha *et al.,* 2025).

DNA oxidative damage

This is one of the most studied mechanisms by which essential oils affect DNA. Phenolic compounds such as carvacrol, thymol, and eugenol can, under certain concentrations and conditions, generate reactive oxygen species (ROS, such as superoxide anion, hydrogen peroxide, hydroxyl radical), either through auto-oxidation, enzymatic metabolism (such as cytochrome P_{450}), or alteration of the mitochondrial respiratory chain (Masyita *et al.,* 2022; Sallam *et al.,* 2022; Thapa *et al.,* 2019; Wróblewski *et al.,* 2025). ROS attack nitrogenous bases, with guanine being the most susceptible due to its low oxidation potential. The main product is 8-oxo-7,8-dihydroguanine (8-oxoguanine, 8-oxoG), a mutagenic lesion that mispairs with adenine during replication, producing G:C to T:A transversions (Masyita *et al.,* 2022; Thapa *et al.,* 2019). In studies with human colon cells HT-29, exposure to carvacrol from oregano essential oil increased 8-oxoguanine (8-oxoG) levels up to 5-fold above the control, an effect reversed by N-acetylcysteine (an antioxidant) (Sallam *et al.,* 2022; Thapa *et al.,* 2019). 8-oxoG is the most abundant DNA lesion caused by oxidative stress, occurring when reactive oxygen species modify the guanine base. Similarly, thymol from thyme oil showed a biphasic effect: at low concentrations (50 µg/mL) it reduced oxidative damage induced by H_2O_2 (genoprotective effect), whereas at high concentrations (200 µg/mL) it increased 8-oxoG formation, probably due to ROS overload (Sallam *et al.,* 2022; Thapa *et al.,* 2019). In cells treated with eugenol and cinnamaldehyde were detected other oxidized bases, include 8-oxoadenine, 5-hydroxyuracil, thymidine glycol, and FapyG (2,6-diamino-4-hydroxy-5-formamidopyrimidine), which indicated that essential oils can induce global oxidative stress not limited to guanine (Masyita *et al.,* 2022; Thapa *et al.,* 2019).

DNA single and double-strand breaks

ROS produced by essential oils can also directly attack the deoxyribose backbone, causing single-strand breaks (SSBs) through the abstraction of hydrogen atoms at positions C1', C2', or C4' of deoxyribose, followed by ring fragmentation (Masyita *et al.,* 2022; Thapa *et al.,* 2019). SSBs are the most frequent lesions induced by low doses of essential oils. In the comet assay, cells treated with oregano essential oils (50–100 µg/mL) showed a significant increase in comet tail, indicative of SSBs (Sallam *et al.,* 2022; Thapa *et al.,* 2019). SSBs can be efficiently repaired by the base excision repair (BER) machinery, but if the dose is high or exposure is prolonged, SSBs become double-strand breaks (DSBs), either due to the proximity of two SSBs on opposite strands or the collapse of the replication fork upon encountering an unrepaired SSB (Masyita *et al.,* 2022; Thapa *et al.,* 2019). Double-strand lesions are lethal if not repaired correctly. It has been demonstrated that eugenol from clove essential oil induces DSBs in HeLa cells at concentrations ≥75 µM (Haro-Gonzalez *et al.,* 2021; Thapa *et al.,* 2019). In prostate cancer cells (PC-3 line), thymol produced dose-dependent DSBs, associated

with activation of the non-homologous end joining (NHEJ) repair pathway and, eventually, apoptosis (Elbe *et al.,* 2020; Sallam *et al.,* 2022).

DNA fragmentation

When damage is massive and strand breaks are numerous, DNA fragments into pieces of variable size, visualized on electrophoresis gels as a "ladder" or as a diffuse band (smear) (Masyita *et al.,* 2022; Thapa *et al.,* 2019). Internucleosomal fragmentation (fragments of approximately 180–200 bp) is characteristic of apoptosis, whereas random fragmentation (smear) suggests necrosis (Thapa *et al.,* 2019). Several studies have demonstrated that essential oils rich in thymol or carvacrol induce internucleosomal DNA fragmentation in tumor cells (MOLT-4 leukemia, MCF-7 breast carcinoma), accompanied by activation of caspases-3 and -9, confirming an apoptotic mechanism (Elbe *et al.,* 2020; Sallam *et al.,* 2022). In contrast, in healthy cells (human dermal fibroblasts, primary hepatocytes), DNA fragmentation is only observed at concentrations much higher than therapeutic ones (>300 µg/mL), indicating a certain margin of safety (Masyita *et al.,* 2022; Thapa *et al.,* 2019). In bacteria (*E. coli*, *S. aureus*), DNA fragmentation induced by essential oils is associated with the release of bacterial endonucleases activated by membrane damage, rather than a direct effect on the nucleoid (Masyita *et al.,* 2022).

DNA methylation

DNA methylation is a key epigenetic mechanism that regulates gene expression. Changes in methylation patterns are associated with cancer, aging, and inflammatory diseases. Some essential oil components can modulate the activity of DNA methyltransferases (DNMTs) or alter the availability of S-adenosylmethionine (methyl donor). Recent studies have demonstrated that eugenol and rosmarinic acid (present in rosemary and also in *J. mimosifolia* in the form of glucoiridoids) inhibit DNMT activity (Leutcha *et al.,* 2025; Thapa *et al.,* 2019). Likewise, carvacrol at high doses induces hypermethylation of certain promoters in tumor cells (Thapa *et al.,* 2019).

Adduct formation with DNA

Adducts are products of covalent addition of a chemical compound (electrophile) to nitrogenous bases or the deoxyribose backbone. Some essential oil components, especially those possessing an aldehyde group (cinnamaldehyde, citral) or an epoxy group (caryophyllene oxide), can form stable adducts with DNA (Masyita *et al.,* 2022; Thapa *et al.,* 2019). Cinnamaldehyde reacts with the exocyclic amine of guanine (N2 position) and with adenine (N6), forming cyclic adducts of the 1,N2-propanoguanine and 1,N6-propaneadenine type (Masyita *et al.,* 2022; Thapa *et al.,* 2019). These adducts block RNA polymerase and DNA polymerase, inducing transcriptional pauses and mutations (Thapa *et al.,* 2019). Eugenol, after hepatic metabolization to eugenol-2',3'-oxide (by cytochrome P450), can form adducts with guanine and adenine, which would explain its genotoxicity observed in micronucleus studies (Haro-Gonzalez *et al.,* 2021; Thapa *et al.,* 2019). However, adduct formation by essential oils does not always lead to mutation. Nucleotide excision repair (NER) efficiently removes these adducts in healthy cells, but if repair capacity is saturated or deficient (as in cancer cells with NER mutations), adducts persist and contribute to genomic instability (Masyita *et al.,* 2022; Thapa *et al.,* 2019).

Genoprotective or Genotoxic Effect of Essential Oils

Essential oils can exert opposite effects on DNA depending on concentration, exposure time, cell type, and redox context. At low concentrations (MIC/10), many phenolic compounds act as genoprotectors since they induce the expression of antioxidant enzymes (Nrf2, HO-1, SOD, catalase) and scavenge free radicals, reducing basal oxidative damage (Masyita *et al.,* 2022; Thapa *et al.,* 2019). In contrast, at higher concentrations (MIC), the same compounds generate ROS, form adducts, break DNA, and induce apoptosis or necrosis (Elbe *et al.,* 2020; Sallam *et al.,* 2022; Thapa *et al.,* 2019). Melušová *et al.,* (2014) reported that treatment with carvacrol and rosemary oil induced genomic damage and programmed cell death; the data showed a notable increase in the frequency of micronuclei and HepG2 cells in apoptotic state after 24 h of treatment. This duality, where a clear genotoxic damage is observed at certain doses, is fundamental for the possible use of essential oils as chemopreventive agents (at low doses) or as antineoplastic agents at high doses as was suggested by cell mortality and genotoxicity trends (Masyita *et al.,* 2022; Thapa *et al.,* 2019). Furthermore, the chemical composition of essential oils modulates the net effect on DNA. For example, thyme essential oil, rich in thymol and *p*-cymene, is more genoprotective than oregano oil, rich in carvacrol, due to the ability of *p*-cymene to induce phase II enzymes without generating excess ROS (Masyita *et al.,* 2022; Thapa *et al.,* 2019).

Once the main mechanisms by which essential oils can damage genetic material have been described, it is possible to integrate this information into a comparative format. Essential oils constitute a diverse and promising source of bioactive compounds with documented effects on nucleic acids, particularly on DNA, since their major components (flavonoids, monoterpenic phenols, phenylpropanoids) can interact with DNA through multiple mechanisms such as intercalation, groove binding, oxidative damage with 8-oxoguanine formation, single- and double-strand breaks, internucleosomal fragmentation, modulation of DNA methylation, and covalent adduct formation (Leutcha *et al.,* 2025; Masyita *et al.,* 2022; Sallam *et al.,* 2022; Thapa *et al.,* 2019). The final DNA response (genoprotection versus genotoxicity) depends on the balance between essential oil concentration, exposure time, cell type, and the redox state of the environment (Masyita *et al.,* 2022; Sallam *et al.,* 2022; Thapa *et al.,* 2019).

However, current evidence presents important limitations. Most studies have been conducted in vitro in immortalized cell lines, with concentrations that often do not reflect those achievable in human tissues after realistic administration (Masyita *et al.,* 2022; Thapa *et al.,* 2019). Furthermore, the compositional variability of essential oils (due to geographic origin, phenology, extraction method) hinders comparison between studies and their standardization (Sadgrove *et al.,* 2022; Thapa *et al.,* 2019). Adduct formation and effects on DNA methylation are areas still with few detailed mechanistic studies (Masyita *et al.,* 2022; Thapa *et al.,* 2019). Future perspectives require investigations addressing in vivo studies in animal models to evaluate the bioavailability, tissue distribution, and systemic genotoxicity of essential oils.

In conclusión, essential oils and their major components exert a dual influence on DNA: at low doses they act as genoprotectors by neutralizing free radicals, whereas at higher concentrations they induce oxidative damage, strand breaks, fragmentation, epigenetic alterations, and adduct formation. This biphasic response depends on cell type, dose, and the chemical composition of the oil. Understanding these mechanisms is fundamental to harnessing their antiproliferative properties in cancer and minimizing their toxicological risks in clinical and food applications.

Conflict of interest

The authors declare that they have no conflict of interest.

Declaration of competing interest

The authors declare that they have no potential competing financial interests.

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