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# Chinese Cinnamon Essential Oil as a Potential Dental Additive: Antimicrobial Efficacy and Biocompatibility Assessment

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## Abstract

The objective of this study was to assess the antimicrobial potential and biosafe dose of Chinese cassia essential oil (CCEO) to be biocompatible for dental applications. CCEO extract was obtained by soaking Chinese cassia sticks in 3 solvents: petroleum ether, chloroform, and methyl alcohol. FTIR and GC-MS spectrometry investigations were used to study the chemical composition of CCEO extract. FTIR showed the presence of O-H functional group that is related to phenols and alcohols, C-H stretching for alkane, C =O bond for aldehyde of saturated fatty acids, C=C bond for alkenes, C-OH bending vibration of alcohols, and C-O-C for aromatic acid esters. The GC-MS results indicated the presence of 36 diverse compounds. Amongst these, E-cinnamaldehyde (38.08%), (7.99%) of carbohydrates (D-Talofuranose, D-Talopyranose, D-Mannopyranose and D-Glucopyranose), p-Coumaric alcohol (7.44%), Cinnamyl alcohol (6.15%) and Cinnamic acid (6.11%) were the most plentiful compounds. The antimicrobial action of the CCEO was tested against *Candida albicans*, methicillin-resistant *S. aureus* (MRSA), and *Enterobacter aerogenes* which displayed memorable antimicrobial activity against all tested microorganisms. CCEO revealed minimum inhibition concentration values of 15 µl/ml, 5 µl/ml, and 25 µl/ml against *C. albicans*, MRSA, and *E. aerogenes*, respectively. In addition, the biocompatibility of CCEO was evaluated on the oral cells. Biosafe dose analysis highlighted the necessity of carefully evaluating oil biocompatibility for dental applications, as excessive concentrations may compromise cell membrane integrity, induce oxidative stress, and trigger inflammatory responses. CCEO displayed to be a highly promising supplementary additive to improving antimicrobial and biosafe properties.

***Keywords***: Chinese cassia, antimicrobial, biocompatibility, dental, inflammatory, oral

## Introduction

Antimicrobial resistance (AMR) refers to the defense mechanisms that microorganisms have developed to fend against the effects of antibiotics or medications (Amaratunga *et al*.,

2016). Numerous microorganisms, including bacteria and yeasts, are capable of becoming so resistant to antimicrobial treatments that they lose their effectiveness. The various strategies by which microbes resist the available antibiotics include ribosome recycling and splitting, inactivating a drug, altering a drug target type, restricting drug uptake, and active drug efflux (Seely *et al*., 2024). AMR was responsible for about 3.57 million of the 4.95 million deaths that took place worldwide (Michael *et al*., 2014). AMR is estimated by the UN and WHO to have a far greater global impact than the 700,000 deaths per year that would lead to 10 million deaths per year by 2050 (WHO, 2023).

One of the most frequent infectious agent-related causes of morbidity and mortality in the globe is *Staphylococcus aureus.* These sphere-shaped, Gram-positive bacteria are the most dangerous of the many common staphylococcal bacteria. They can cause nosocomial bacteremia, cardiovascular infections, prosthetic joints, surgical sites, and acute skin infections that are the main cause of pneumonia and other respiratory tract infections (Gordon *et al*., 2021). It was estimated that 20,000 people died from *S. aureus* bacteremia each year in the United States. Because methicillin-resistant *S. aureus* (MRSA) is the most significant clinically, *S. aureus* infections are particularly worrisome because *S. aureus* isolates frequently exhibit drug resistance. (Fayed *et al*., 2024; Nicholas *et al*., 2019). *S. aureus* has acquired determinants by horizontal gene transfer of mobile genetic elements, which has led to the evolution of resistance to many drugs. Antibiotic manufacturers may have developed these determinants to protect themselves against potentially inhibiting compounds or their competitors. Mutations that alter the locations of drug binding on molecular targets and an increase in the production of endogenous efflux pumps can also result in resistance. In principle, a set of inhibitors that target different locations can reduce the development of resistance by mutation, or two or more mutations can be wanted for resistance to pass the MIC breakpoint (Timothy, 2017).

*Enterobacter aerogenes* belongs to the Enterobacteriaceae family which were originally divided into three genera: *Escherichia*, *Enterobacter*, and *Klebsiella*, where the *Enterobacter* genus included *E. aerogenes* and *E. cloacae* (Wesevich *et al*., 2020). *E. aerogenes* is a Gram-negative, electively anaerobic, rod-shaped bacillus, and it is motile by peritrichous flagella (Anne *et al*., 2019). It is found in the human gastrointestinal tract and does not cause disease in healthy people, but it hits neonates, immunocompromised patients, individuals that received antibiotic therapy, and old people (Abou-Dobara et al., 2024).

Another serious infection known as invasive candidiasis affects the eyes, heart, blood, bones, brain, and/or other bodily components and is brought on by the yeast, a type of fungus, *Candida* (El-Zahed et al., 2023). *Candida albicans* is a commensal fungus that colonizes the surface of the oral mucosa and that is normally harmless in healthy persons. Under particular conditions, this opportunistic microorganism can cause a superficial infection called candidiasis (Mohamed & El-Zahed, 2024). *C. albicans* have well-documented pathogenic features, involving its ability to adhere to epithelial cells, and the hyphae formation that infiltrates epithelial cells (O'Donnell *et al*., 2015). The most popular oral infection is denture candidiasis, which mainly derived from *C. albicans* in patients that wear polymethylmethacrylate (PMMA) dentures (Yeon *et al*., 2020). Oral candidiasis is characterized by pain when eating or swallowing and by the emergence of white plaques on red, inflamed mucosa (throat, inner cheeks, and tongue) (O'Donnell *et al*., 2015).

Recently, researchers have focused their research on discovering and developing alternative medicines from medicinal plants in order to prevent the serious side effects that are frequently connected to traditional pharmaceuticals (El Sadda *et al*., 2025; Fayed *et al*., 2025). One of these alternative medicines that has an important consideration is cinnamon, a frequently used cooking spice with a safety profile and remarkable features related to the regulations of blood sugar and its antimicrobial potential (El-Gohary *et al*., 2024; El Sadda *et al*. 2024). The dried bark of several species of the *Cinnamomum* genus, such as *C. zeylanicum, C. verum,* and *C. cassia*, is used to make cinnamon. Because different species of cinnamon in the genus *Cinnamomum* are produced under varied geographical and environmental conditions, there are notable differences in their chemical composition. These differences are mostly represented in the composition of the primary bioactive

components. The plant's branches, bark, and leaves are among the parts from which cinnamon oil can be produced; each part contributes a somewhat distinct chemical composition (Jiageng *et al*., 2024).

Mojtaba *et al*., (2022) documented that CCEO have powerful antibacterial and antifungal activity due to their major component cinnamaldehyde. The study documented their bactericidal action against *S. aureus* and *Escherichia coli*. Roberto *et al*. (2023) reported that *C. cassia*, called Chinese cassia or Chinese cinnamon, essential oil (CCEO) has displayed potent antimicrobial activity against *Candida* species. In addition, Chen *et al*., (2019) explained that CCEO shows anti-inflammatory properties by preventing the formation of inflammatory mediators. It has been shown that CCEO can decrease pro-inflammatory cytokine expression and thereby relieve inflammatory responses, suggesting that CCEO holds promise as a natural anti-inflammatory agent. Hesham *et al*., (2022) displayed the biocompatibility of, antibacterial formulations based on cinnamon oil that were efficient against *Culex pipiens* adults and pupae. Warunya *et al*. (2020) showed that cinnamon essential oil at each potent concentration did not display any toxicity when examined on normal human fibroblast cells. So, this essential oil could be a potential candidate for pharmaceutical and cosmetic and product. Thus, this study was aimed at extracting of CCEO, assessing its antimicrobial effect against three organisms (*C. albicans*, MRSA and *E. aerogenes*), and investigating its biocompatibility behavior on oral human cells.

## Materials and methods

*Sample collection*

Chinese cassia sticks (*C. cassia*, 100% organic, origin: India, Organic Way LLC, USA) were purchased from the market that in Damietta city, Damietta, Egypt, and taken to the Analytical Laboratory at the Faculty of Science, Damietta University, for extraction. They were oblong, rolled shapes, rough and thick sticks, 8-15 cm long, and had a dark brown-red color (Figure 1). The sticks were washed to eliminate any dirt, and any damaged sticks were discarded.

**Figure 1.** Chinese cassia sticks.

*Extraction of CCEO*

200 grams of Chinese cassia sticks were soaked for 3 weeks in the dark bottles at room temperature (25°C) with 330 ml of solvents (petroleum ether, chloroform, or methyl alcohol). Then, the extracted oils were placed in a rotary evaporator for an hour at a temperature of 55°C until the solvents evaporated from the extracted essential oils (EOs) (Singh et al., 2007).

*Chemical profile and constituents' identification of the extracted EOs*

GC–MS spectrometry (TSQ 9000 triple quadrupole mass spectrometer, Thermo Scientific™ TRACE™ 1310 Gas Chromatograph, Center for Excellence in Research of Advanced Agricultural Sciences (CERAAS), Damietta University, Egypt, and FTIR (FT/IR-4100type A, Central Laboratory, Faculty of Science, Damietta University, Egypt) were used to identify the phytochemicals in the extracted EOs.

*Biosafety assessment of CCEO on oral epithelial cells*

The oral epithelial cells (OECs, Nawah Scientific Inc., Mokattam, Egypt) and were used during the biosafety assessment of CCEO according to the method provided by Diaa *et al*. (2025). DMEM, which was enhanced with 10% heat-inactivated fetal bovine serum, 100 mg/ml streptomycin, and 100 units/ml penicillin, was used, and the incubation occurred in an incubator with 5% (v/v) $CO_2$ at 37°C. The

sulforhodamine B (SRB) test was used to assess cell viability.

*Antimicrobial activity of CCEO using agar well diffusion method*

The CCEO's antimicrobial activity was investigated based on the Clinical and Laboratory Standards Institute (CLSI) protocol (CLSI, 2017). 0.5 MacFarland ($1\text{-}2\times10^8$ CFU/ml) from *C. albicans* ATCC10231, MRSA ATCC 43300, and *E. aerogenes* ATCC13048 were kindly provided from the Microbiology Lab, Botany and Microbiology Department, Faculty of Science, Damietta University, prepared and added separately under aseptic conditions on the surface of Muller-Hinton agar (MHA) plates. CCEO and standard drugs (penicillin G and miconazole) were added into 5 mm wells. Plates were incubated at 37°C for 48 hours for bacterial strains or at 28°C for 48 hours for yeast. After the incubation, inhibition zone diameters (IZD) were recorded in millimeters.

*Minimum inhibitory concentration (MIC)*

The MICs of CCEO, penicillin, and miconazole were studied according to the broth microdilution method (CLSI, 2000; 2012). Serial dilutions (0–100 µl/ml) of the antimicrobial agents were prepared and tested against 0.5 McFarland standard of each microbial strain that was sub-cultured in Mueller-Hinton broth medium (MHB). After the incubation at 37°C for 48 hours for bacterial strains or at 28°C for 48 hours for yeast, microbial growth was assessed spectrophotometrically at 600 nm.

*Statistical analysis*

Statistical analysis of the data was administered using SPSS version 18 software. Experimental values are demonstrated as the mean ± standard deviation (SD) for each outcome were given (O'connor, 2000). The ANOVA test was used for the analysis at a significance threshold of 0.05. Three iterations of the experiments were conducted.

**Results**

FTIR spectra (Figure 2) were employed to identify the obvious functional groups of the CCEO samples (crude and fractions A & B). Consecutive absorption bands at 3389.28 $cm^{-1}$, 3406.64 $cm^{-1}$ and 3416.28 $cm^{-1}$ assigned to (O-H), H- bonded for phenol and alcohol. Peaks at 2941.88 $cm^{-1}$, 2957.3 $cm^{-1}$, 2835.81 $cm^{-1}$, 2840.63 $cm^{-1}$ and 2841.6 $cm^{-1}$ assigned to (C-H) vibration for alkane. 1719.23 $cm^{-1}$ is due to (C =O) bond for aldehyde of saturated fatty acids. Absorption bands at 1670.05 $cm^{-1}$, 1656.55 $cm^{-1}$and 1630.52 $cm^{-1}$ are implies stretching of alkenes (C=C) . Bands at 1455.99 $cm^{-1}$-1400.7 $cm^{-1}$ are corresponds to bending vibration of alcohol (C-OH). 1302.68 $cm^{-1}$ is attributed to bending (=C-H) and ($CH_2$) alkanes. Absorption bands at 1266.04 $cm^{-1}$, 1259.29 $cm^{-1}$ and 1250.61 $cm^{-1}$ indicated to the stretching vibration of phenols (C-OH) and symmetric expansion of the aromatic acid ester (C-O-C). Other peaks at 975.804 $cm^{-1}$-679.785 $cm^{-1}$ showed the existence of aromatic (C=C) (Lixourgioti *et al*., 2022).

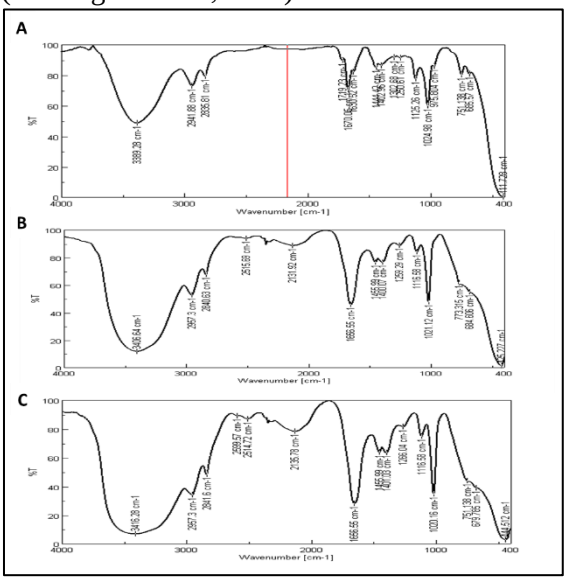


Figure **2.** FTIR spectra of CCEO crude; (A), fraction A; (B), and fraction B; (C).

*Chemical composition of CCEO using GC-MS analysis*

Table 1 and Figure 3 show 36 different compounds, including E-cinnamaldehyde (38.08%), 7.99% of carbohydrates (D-talofuranose, D-talopyranose, D-mannopyranose, and D-glucopyranose), p-coumaric alcohol (7.44%), cinnamyl alcohol (6.15%) and cinnamic acid (6.11%)

were the main constituents of the cinnamon EOs.

*Biosafety dose of CCEO for oral cells*

Figure 4 illustrates the impact of CCEO on normal oral epithelial cells. In the control group (no oil), cells appear intact and densely packed, indicating healthy growth without any signs of cytotoxicity. At a low oil concentration (0.01 µl/ml), a slight reduction in cell density is observed, though the cells remain largely intact, suggesting minimal effects on viability. However, at a higher concentration (100 µl/ml), clear changes happen, such as cells getting smaller, coming off their surfaces, and showing less color when stained, which means they are being harmed significantly. This suggests that higher oil concentrations may have a more detrimental effect compared to lower concentrations. Figure 5 presents the $IC_{50}$ value of CCEO, which was determined to be 87.7 µl/ml.

**Table 1.** Chemical composition of CCEO using GC-MS.

| Peak | Retention time | Components | Contents (%) | Molecular formula | Molecular weight |
|---|---|---|---|---|---|
| 1 | 8.773 | Glycerol, 3TMS derivative | 0.42 | $C_{12}H_{32}O_3Si_3$ | **308.64** |
| 2 | 9.100 | E-Cinnamaldehyde | 34.53 | $C_9H_8O$ | 132 |
| 3 | 9.557 | Copaene | 2.97 | $C_{15}H_{24}$ | 204 |
| 4 | 9.649 | E-Cinnamaldehyde | 3.55 | $C_9H_8O$ | 132 |
| 5 | 9.929 | Cinnamyl alcohol, TMS derivative | 6.15 | $C_9H_{10}O$ | 134 |
| 6 | 10.399 | *a*- Muurolene | 5.21 | $C_{15}H_{24}$ | 204 |
| 7 | 10.536 | Naphthalene, 1,2,3,5,6,8a-hexahydro-4,7-dimethyl-1-(1-methylethyl)-, (1S-cis)- | 4.01 | $C_{15}H_{24}$ | 204 |
| 8 | 10.708 | Cinnamic acid, (E)-, TMS derivative | 6.11 | $C_{12}H_{16}O_2Si$ | 220 |
| 9 | 11.085 | p-Coumaric alcohol, 2TMS derivative | 7.44 | $C_{15}H_{24}O_3Si_2$ | 308.5 |
| 10 | 11.491 | Coumarin | 1.76 | $C_9H_6O_2$ | 146 |
| 11 | 11.715 | Coumarin | 0.79 | $C_9H_6O_2$ | 146 |
| 12 | 11.789 | Epizonarene | 1.27 | $C_9H_{13}NO_3$ | 183 |
| 13 | 12.046 | D-(-)Tagatofuranose, pentakis(trimethylsilyl) ether (isomer 1) | 2.08 | $C_{21}H_{52}O_6Si_5$ | 541 |
| 14 | 12.241 | D-(+)-Talofuranose, pentakis(trimethylsilyl) ether (isomer 2) | 0.82 | $C_{21}H_{52}O_6Si_5$ | 541 |
| 15 | 12.498 | *a*.-D(+)Talopyranose, 5TMS derivative | 1.33 | $C_{21}H_{52}O_6Si_5$ | 541 |
| 16 | 12.773 | 1H-Inden-1-one, 2,3-dihydro-3-methyl | 0.60 | $C_{10}H_{10}O$ | 146 |
| 17 | 12.928 | *a*-D-Mannopyranose, 5TMS derivative | 0.88 | $C_{21}H_{52}O_6Si_5$ | 541 |
| 18 | 13.157 | Palmitic Acid, TMS derivative | 1.15 | $C_{19}H_{40}O_2Si$ | 328.6 |
| 19 | 13.483 | Ferulic acid, 2TMS derivative | 0.28 | $C_{16}H_{26}O_4Si_2$ | 338.5 |
| 20 | 13.923 | 9,12-Octadecadienoic acid (Z,Z)- , TMS derivative | 1.74 | $C_{21}H_{40}O_2Si$ | 352.6 |
| 21 | 14.232 | 1H-Inden-1-one, 2,3-dihydro-2-methyl | 0.09 | $C_{10}H_{12}O$ | 148 |
| 22 | 15.016 | Silane, diethylnonyloxypentadecyloxy | 2.96 | $H_4Si$ | 32 |
| 23 | 15.285 | Succinic acid, butyl 3,4-dimethylphenyl ester | 1.70 | $C_{17}H_{24}O_4$ | 292 |
| 24 | 15.640 | 1-Ethyl-4-phosphorinanone thiosemicarbazone | 3.40 | $C_8H_{16}N_3PS$ | 217.27 |
| 25 | 15.840 | *a*.-D-Glucopyranose, 5TMS derivative | 1.14 | $C_{21}H_{52}O_6Si_5$ | 541 |
| 26 | 15.995 | 1-Monooleoylglycerol, 2TMS derivative | 1.40 | $C_{27}H_{56}O_4Si_2$ | 500.9 |
| 27 | 16.349 | 3-Chloro-4-methylphenol | 1.53 | $C_7H_7ClO$ | 142.5 |
| 28 | 16.515 | 6-Hydroxynicotinic acid, 2TBDMS derivative | 1.59 | $C_{18}H_{33}NO_3Si_2$ | 367.6 |
| 29 | 17.007 | Imidazole, 2-bromo-4-methyl-5-nitro- | 0.28 | $C_4H_4BRN_3O_2$ | 206 |
| 30 | 17.128 | Fenoprop, TMS derivative | 0.29 | $C_{12}H_{15}Cl_3O_3Si$ | 154 |
| 31 | 17.585 | Erythritol, 4TMS derivative | 0.32 | $C_{16}H_{42}O_4Si_4$ | 410.8 |
| 32 | 18.415 | Cholesterol, TMS derivative | 0.20 | $C_{30}H_{54}OSi$ | 458.8 |
| 33 | 18.587 | 1-Monolinolein, 2TMS derivative | 0.12 | $C_{27}H_{54}O_4Si_2$ | 498.9 |
| 34 | 18.970 | Stigmast-5-ene, 3.beta.-(trimethylsiloxy)-, (24S)- | 1.35 | $C_{32}H_{58}OSi$ | 486.8 |
| 35 | 21.104 | 1-Monooleoylglycerol, 2TMS derivative | 0.38 | $C_{27}H_{56}O_4Si_2$ | 500.9 |
| 36 | 21.482 | Triethylenemelamine | 0.15 | $C_9H_{12}N_6$ | 204 |

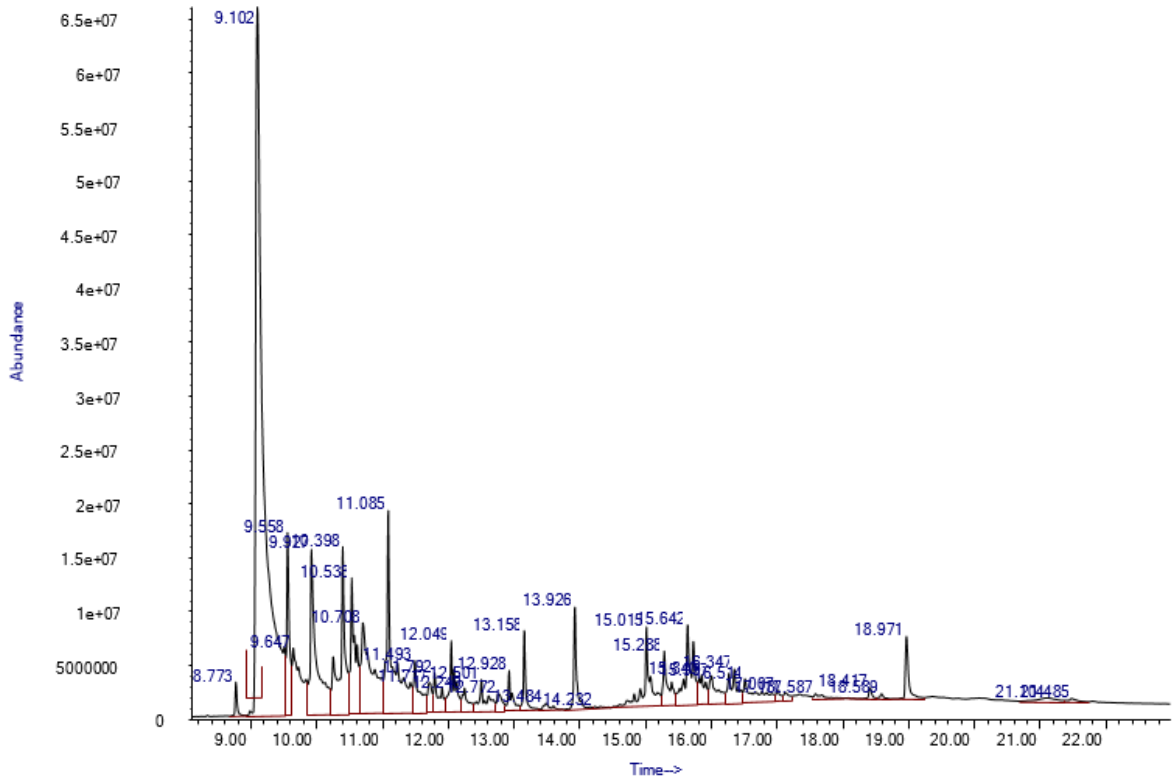


**Figure 3.** Compounds and their % ratios determined using GCMS analysis of the CCEO.

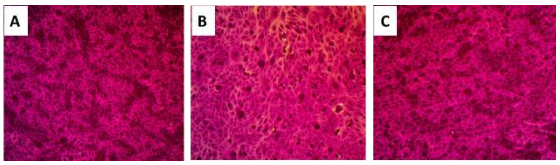


**Figure 4.** Effect of CCEO on normal dental oral cells. Morphological changes at different concentrations (low concentration (0.1 µl/ml); (A), and high concentration (100 µl/ml); (B)) using SRB stain compared to the oral epithelial cells control; (C).

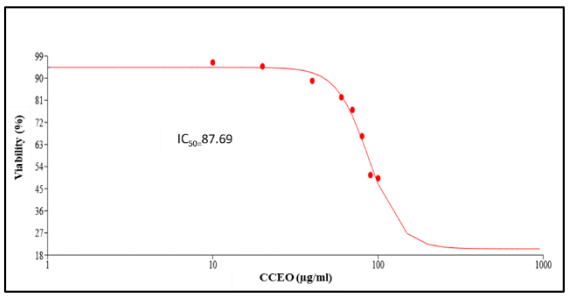


Figure 5. Dose response curve for CCEO on normal dental oral cells viability.

*Antimicrobial activity of CCEO*

The antimicrobial activity of the CCEO was evaluated against various organisms (*C. albicans*, MRSA, and *E. aerogenes*), as shown in Figure 6 and Table 2. CCEO demonstrated greater antifungal activity against *C. albicans*, exhibiting an IZD of 50 mm compared with miconazole with an IZD of 18 mm. Also, CCEO showed strong antibacterial action against MRSA and *E. aerogenes* with an IZD of 20 mm for both compared with penicillin G, which showed no inhibition against MRSA, and an IZD of 15 mm for *E. aerogenes*.

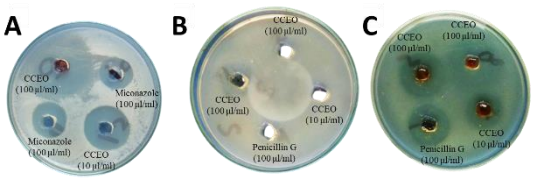


**Figure 6.** Agar well diffusion test of CCEO against *C. albicans*; (A), MRSA; (B), and *E. aerogenes*; (C).

The MIC of CCEO extract against *C. albicans*, MRSA, and *E. aerogenes* was determined (Figure 7). CCEO completely inhibited *C. albicans* at 15 µl/ml, MRSA at 5 µl/ml, and *E. aerogenes* at 25 µl/ml.

**Table 2.** Inhibition zone in mm of CCEO against the tested microorganisms.

| Antimicrobial agent (100 µl/ml) | Inhibition zones (mm±SD) | | |
|---|---|---|---|
| | *C. albicans* | MRSA | *E. aerogenes* |
| CCEO | $50 \pm 0^{c*}$ | $20 \pm 0^{b}$ | $20 \pm 0^{b}$ |
| Penicillin G | - | -ve | $16 \pm 0.14^{a}$ |
| Miconazole | $18 \pm 0.03^{a}$ | - | - |

*Means with common letters are not significantly different according to Duncan's multiple range test ($P < 0.05$, n = 3).

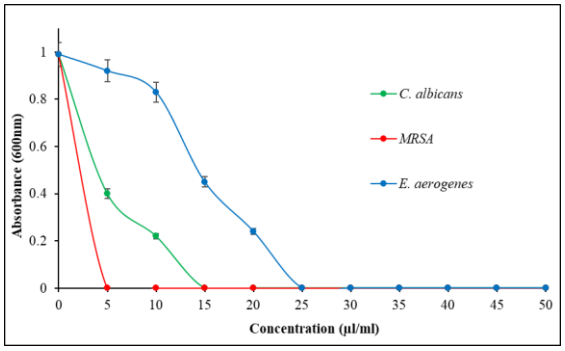


Figure 7. The MIC of CCEO against *C. albicans*, MRSA and *E. aerogenes*.

## Discussion

Cinnamon oil is an aromatic liquid acquired from the bark, leaves, and twigs of *C. zeylanicum* (Melyssa *et al*., 2014). The obtained results showed that CCEO contains various bioactive compounds that share in antimicrobial activity. Similarly, several bioactive compounds such as cinnamaldehyde, cumarin and coumaric alcohol, cinnamyl alcohol, and cinnamic acid were previously documented (Raluca *et al*., 2024). The main identified components in GC-MS analysis are E-cinnamaldehyde (CA) (38.08%), which is in agreement with (Katarzyna *et al*., 2016). The chemical formula for CA, an aromatic aldehyde, is $C_9H_8O$. A benzene ring ($C_6H_5$) connected to a propenyl group (-CH=$CH_2$) and an aldehyde functional group (-CHO) at the alpha position make up this compound. CA has powerful antioxidant action, making it helpful for medicinal objectives, and is also known for its capability to inhibit bacteria and fungi growth (Jiageng *et al*., 2024).

Numerous pharmacological studies have also documented the anti-inflammatory, hypoglycemic, antitumor, antibacterial, and other pharmacological properties of CA (Saifudin *et al*., 2013). Since cinnamon oil contains cinnamaldehyde, coumaric alcohol, cinnamyl alcohol, and cinnamic acid, numerous studies have documented its anti-inflammatory and antibacterial qualities. These attributes demonstrate that cinnamon oil exerts immunomodulatory activity by inhibiting the generation of important inflammatory mediators (Aftab *et al*. 2023; Yi *et al*. 2024). Low concentrations of CCEO have little effect on normal oral cells; higher concentrations may cause irritation and inflammation. This is consistent with prior research on the biocompatibility and cytotoxic effects of various oils on oral cells. Numerous studies have investigated the role of oils in dental applications, such as medicated mouth rinses and essential oil-based formulations. Some plant-derived oils, including coconut oil and tea tree oil, have shown antimicrobial activity while remaining biocompatible at lower concentrations (Shekar *et al*., 2015). However, at elevated concentrations, certain oils may compromise cell membrane stability, trigger oxidative stress, and interfere with metabolic functions, ultimately reducing cell viability (Ali *et al*., 2023). The cytotoxic effects seen at higher oil concentrations may be linked to disturbances in cell membrane integrity and mitochondrial activity. Lipid-based compounds present in oils can interact with cellular lipids, altering membrane permeability, increasing the production of reactive oxygen species (ROS), and eventually leading to apoptosis or necrosis. Additionally, cytotoxicity may arise from bioactive components within the oil that provoke inflammatory responses or inhibit critical cellular enzymes (Ali *et al*., 2023).

Under ordinary immune function, *C. albicans* exists safely with the host, but with weakness in the immune system, it can become pathogenic, causing grave systemic infections when it enters the bloodstream (Baka *et al*., 2024a; Clarissa & Alexander, 2015). The anti-candidal activity of CCEO oil against planktonic and biofilm cultures of *C. albicans* has been documented by Yaru *et al*. (2014). The current study displayed a good antifungal action of the CCEO against *C. albicans*, achieving a 50 mm inhibition zone with MIC at 15 µl/ml. The result of the present study agrees with Tran et al. (2020) who showed that IZD of bark CCEO against two strains of *C. albicans* were 53 mm and 56.68 mm, and MICs for both strains were below 0.03% (v/v). In addition, the current result is in agreement with Rym *et al*. (2017), who reported that CCEO inhibits

ergosterol biosynthesis in *Candida* species and influence fungal membrane integrity by permeabilizing the cell. According to Wijesinghe *et al*. (2021), CCEO inhibits fungal growth by disrupting the cell wall, which results in intracellular leakage. The MIC of CCEO was determined to be 1.0 mg/ml. According to a different study, CCEO may cause *C. albicans* to accumulate reactive oxygen species (ROS), which would ultimately lead to cell death. Chen and associates (2019). Additionally, the antifungal properties of cinnamon bark essential oil were described by Marie & Daniel (2019), who also found that the oil's MIC values ranged from 0.039% to 0.078% (v/v). It was also shown that CCEO permeabilized the cell membrane of *C. albicans*, which resulted in increased uptake of SYTOX Green dye and ultimately cell death. Additionally, CCEO demonstrated anti-inflammatory qualities by lowering the release of pro-inflammatory cytokines IL-6 and IL-8 from human oral epithelial cells stimulated with TNF-α. The secretion of IL-6 and IL-8 was reduced by 29% and 57%, respectively, by CCEO at a concentration of 62.5 µl/ml.

The current findings are explained by the high concentration of CA, which has inherent antimicrobial qualities and a special antifungal effect. It might inhibit microbial growth through the cell wall that can harm the fungal cell walls' structural and functional integrity. The hydrophilic aldehyde group in CA's structure makes it easy for hydrophilic radicals on the fungal surface to absorb it, which breaks down the polysaccharide structure of the wall to enter the cell wall, affecting cell biosynthesis and preventing growth and reproduction (Fugo *et al*., 2014). Furthermore, Jie *et al*. (2022) noted that by CA, the cell wall's surface was minus, the hyphae and external layer of the cell wall of *C. albicans* disintegrated and dropped off, and the cell wall barely deteriorated. The CA caused necrosis, disintegration, denaturation, and cell edema by directly penetrating the cell membrane and harming the nucleus and organelles, ultimately resulting in cell death. With its potent fungicidal action, CA can eradicate harmful fungi, improve the recovery rate from invasive *Candida* infections, and lower mortality rates. In another work, Mohd *et al*. (2013) found that CA decreased β-1-glucan synthase, which in turn caused *C. albicans* cell walls to thicken. CA has been shown to have antibacterial efficacy against a range of fungi in another study (Hongbo *et al*., 2015).

The current study showed good antibacterial action of the CCEO against *E. aerogenes,* achieving a 20 mm with MIC at 25 µl/ml, and displayed good antibacterial activity of the CCEO against MRSA, achieving 20 mm with MIC at 5 µl/ml. Current results are in agreement with Rana & Jayashankar (2022), as they concluded that the inhibition zone of the cinnamon oil was 23 mm for *E. aerogenes* and 27 mm for MRSA. According to Chengjie *et al*. (2024), cinnamon oil and its constituents have varying degrees of antibacterial activity against both Gram-negative bacteria, including *Salmonella* sp., *E. coli*, and *Pseudomonas aeruginosa*, and Gram-positive bacteria like *S. aureus*, as well as yeast such as *C. albicans*.

Positive findings have also been found in studies on the antifungal properties of plant extracts, particularly against *Candida* species and other harmful fungus, including *Aspergillus niger* and *A. fumigatus* (Baka *et al*., 2024b; Elazab *et al*., 2024). The major reason for inhibition attributed to the growth of bacteria is the active component in cinnamon oil, which contains CA as the main component responsible for its antibacterial, anti-inflammatory, and anti-cancer properties (Raluca *et al*. 2024). The MIC results in the current study demonstrated that MRSA (Gram-positive bacterium) 5 µl/ml was more sensitive to CCEO than *E. aerogenes* (Gram-negative bacterium) 25 µl/ml. This result agreed with Shareef (2001), who reported that CCEO gave MIC against some pathogenic bacteria such as *(S. aureus, K. pneumonia, E. coli, P. aeruginosa, Proteus* spp., and *Brucella* spp.), and showed that Gram-positive bacteria were more sensitive than Gram-negative to CCEO. The obtained study that evaluated the antibacterial properties of cinnamon oil, particularly from Chinese cassia, against *S. aureus* was also supported by Yunbin *et al*. (2016). They specified a MIC of 1.0 mg/ml and an inhibition zone of 28.7 mm. The bacteria were immediately cultured in a culture medium that was identical to a progression concentration of cinnamon oil. In addition, the current study agreed with Mahfuzul *et al*. (2008), who recorded that CCEO displayed high antibacterial activity against *S. aureus.* Furthermore, Lidaiane *et al*. (2022) examined the antibacterial action of cinnamon oil against *S. aureus*, which showed antibacterial potential with an MIC of 1.6 mg/ml. Also, Vinicius &

Edeltrudes (2009) evaluated the antimicrobial activity of 11 essential oils, including cinnamon, against 10 strains of Gram-positive and Gram-negative bacteria; the results concluded that CCEO showed a greater inhibitory effect. Antibacterial activity of CCEO occurred by cell membrane degeneration by damaging the force of protons, flowing of electrons, active transport, and cell component coagulation. The active components in the CCEO, such as cinnamaldehyde, are responsible for the bacterial cell wall destruction and have the ability to interfere with the bacterial enzymes' synthesis (Matan *et al*., 2006). Using the disk diffusion method, Shinta *et al*. (2021) examined the antibacterial activity of cinnamon oil that was steam-distilled from *C. burmannii* against MRSA. Cinnamon oil was made in 1%, 2%, 4%, and 8% concentrations for the study, and its efficacy against five MRSA isolates that were obtained from cow's milk was evaluated. The results showed that 4% cinnamon oil effectively inhibited all tested MRSA strains, with inhibition zones exceeding 8 mm, while 8% cinnamon oil produced an average inhibition zone diameter of 20 mm. The potent antimicrobial action of CCEO might be due to the presence of E-cinnamaldehyde (38.08%) and cinnamic acid (6.11%), with a total existence ratio reaching to 44.19%, as recorded by the GC-MS results compared to previous studies (Katarzyna et al., 2016; Raluca *et al*., 2024). In an effort to clarify the mechanism of action, numerous studies and reviews have been conducted. The findings indicate that CCEO breaks down bacterial cell membranes, increasing membrane permeability and ultimately causing cell death because of the presence of the aforementioned bioactive compounds. Bacterial cell enlargement and laceration are two major morphological alterations that might result from high CCEO concentrations. Furthermore, intracellular potassium ion ($K^+$) release signifies membrane damage, and CCEO's lipophilicity makes it easier for it to penetrate bacterial membranes, altering their characteristics and enhancing permeability (Vasconcelos *et al*., 2018).

## Conclusions

CCEO displayed no harmful action on human cells, proposing its potential as a good alternative for treating microbial infections. A thorough evaluation of oil biocompatibility is essential, especially for dental applications. The detected cytotoxic effects could stem from membrane damage, oxidative stress, or inflammatory reactions. Understanding the safety profile and mechanisms of oil-induced cytotoxicity is key to ensuring its effective and safe use in oral applications. CCEO showed anti-candidal properties due to the presence of CA that might help in the prevention of its biofilm formation and damage the structure of the cell wall of the fungal cell. Also, CCEO showed antibacterial action against MRSA and *E. aerogenes*. Future work is required to study the antimicrobial mechanisms of CCEO against different microbes.

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## الملخص العربي

### عنوان البحث: زيت القرفة الصينية العطري كمكون إضافي قوي لمواد الأسنان: دراسات التوافق الحيوي والتقييم الفعال له كمضاد للميكروبات

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هدفت هذه الدراسة إلى تقييم النشاط المضاد للميكروبات والجرعة الآمنة بيولوجيًا لزيت القرفة العطري لضمان توافقه الحيوي مع تطبيقات طب الأسنان. تم الحصول على مستخلص زيت القرفة العطري عن طريق نقع أعواد القرفة في ثلاث مذيبات: بتروليوم إيثر، والكلوروفورم، والكحول الميثانولي. استُخدمت دراسات طيف الأشعة تحت الحمراء، وكروماتوغرافيا الغاز المزود بمطياف الكتلة لدراسة التركيب الكيميائي لمستخلص زيت القرفة العطري. أظهر طيف الأشعة تحت الحمراء وجود المجموعة الوظيفية O-H المرتبطة بالفينولات والكحولات، وتمدد C-H للألكانات، ورابطة C = O للألدهيدات في الأحماض الدهنية المشبعة، ورابطة C = C للألكينات، وكذلك انحناء C-OH للكحولات، وC-O-C لإستر الأحماض العطرية. أشارت نتائج كروماتوغرافيا الغاز المزود بمطياف الكتلة إلى وجود ٣٦ مركبًا متنوعًا. من بين هذه المركبات، كانت E-cinnamaldehyde (٣٨،٠٨%)، و(٧،٩٩%) من الكربوهيدرات (D-Talofuranose، وD-Talopyranose، وD-Mannopyranose، وD-Glucopyranose)، والكحول p-Coumaric (٧،٤٤%)، وكحول Cinnamyl (٦،١٥%)، وحمض Cinnamic (٦،١١%) هي المركبات الأكثر شيوعا. تم اختبار التأثير المضاد للميكروبات لمستخلص زيت القرفة العطري ضد كانديدا ألبيكانس، وبكتيريا مارسا، والإنتروباكتر إيروجينيس، والتي أظهرت نشاطًا مضادًا للميكروبات ملحوظًا ضد جميع الكائنات الدقيقة المختبرة. أظهر مستخلص زيت القرفة العطري قيم تركيز تثبيط أدنى تبلغ ١٥ ميكرولتر/مل، و٥ ميكرولتر/مل، و٢٥ ميكرولتر/مل ضد كانديدا ألبيكانس، وبكتيريا مارسا، والإنتروباكتر إيروجينيس، على التوالي. بالإضافة إلى ذلك، تم تقييم التوافق الحيوي لمستخلص زيت القرفة العطري على الخلايا الفموية. أبرز تحليل الجرعة الآمنة بيولوجيًا ضرورة التقييم الدقيق للتوافق الحيوي للزيوت في تطبيقات طب الأسنان، إذ قد تُضعف التركيزات الزائدة سلامة غشاء الخلية، وتُسبب الإجهاد التأكسدي، وتُحفز الاستجابات الالتهابية. وقد ثبت أن مستخلص زيت القرفة العطري مادة تكميلية واعدة للغاية لتحسين خصائصها المضادة للميكروبات والسلامة بيولوجيًا.