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Cinnamaldehyde and Eugenol against Candida: an in vitro study

Dr. A. Colonnese[1], R. Panzironi[2]

1 Nutrition Biologist, Latina, Italy
2 Independent Researcher, Latina, Italy

1. ABSTRACT

A laboratory study compared five essential oil components against 18 strains of Candida. Cinnamaldehyde and eugenol were found to be the most active; together, they allowed for a reduction in the concentrations required to block growth. However, these findings concern fungal cultures, not people with candidiasis.The authors' summary describes an experiment on 18 vaginal strains of Candida: 15 Candida albicans, two Candida glabrata, and one Candida lusitaniae. The researchers evaluated whether five natural compounds would slow down or eliminate the yeast in culture. Cinnamaldehyde and eugenol inhibited all strains examined. Cinnamaldehyde showed an average minimum inhibitory concentration of 50.05 mg/L; eugenol showed 455.42 mg/L. The minimum inhibitory concentration, or MIC, is the lowest amount that prevents visible growth under laboratory conditions. In the time-kill assay, eugenol eliminated viable cells of the selected strain within one hour, and cinnamaldehyde within four hours. The combination achieved an FICI index of 0.625, indicative of an additive effect: the two molecules maintained activity at lower doses than those required individually. The mixture eliminated viable cells within 90 minutes in the tested model. These results indicate experimental antifungal potential, but they do not establish efficacy, dosage, safety, or therapeutic utility in human candidiasis.

VIDEO ARTICLE

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2. WHAT THIS RESEARCH ANALYZED

The 2022 study by Saracino and colleagues investigated whether pure components of essential oils could hinder the growth of *Candida* yeasts. In practical terms, it compared molecules found in various plants, rather than culinary cinnamon, spice powder, or a full extract. Cinnamaldehyde, eugenol, alpha-pinene, limonene, and eucalyptol were analyzed against clinical vaginal strains stored in a microbiological collection.The researchers used disk diffusion, microdilution, time-kill curves, and checkerboard assays. The latter compares multiple concentrations of two substances to determine whether their combination is synergistic, additive, indifferent, or antagonistic. They also verified the effect on *Lactobacillus acidophilus*, a bacterium used here as a model for a potentially protective microorganism. The next step was therefore to estimate not only the inhibition of growth but also the rate of fungal cell elimination.

3. WHAT RESULTS EMERGE FROM THE STUDY

The main finding is that cinnamaldehyde and eugenol were the most active compounds in the studied system. Both inhibited all 18 strains. For cinnamaldehyde, the average zone of inhibition around the disc was 69 mm, the average MIC was 50.05 mg/L, and the minimum fungicidal concentration, or MFC, was 109.26 mg/L. The MFC indicates the amount required to prevent fungal regrowth in the subsequent test. For eugenol, the average values were 35.2 mm, 455.42 mg/L, and 690.09 mg/L.In the *Candida albicans* strain selected for the time-kill assay, eugenol eliminated viable cells within one hour and cinnamaldehyde within four hours. Together, 20.48 mg/L of cinnamaldehyde and 41.40 mg/L of eugenol yielded an FICI of 0.625. Since an FICI between 0.5 and 1 indicates additivity, the data show that in this model, doses could be reduced while maintaining inhibition. However, it does not demonstrate synergy in the strict sense, which the authors define as an FICI equal to or less than 0.5. Alpha-pinene showed minor activity; limonene and eucalyptol did not provide comparable results.

4. WHY THIS STUDY IS RELEVANT

This research is relevant because it addresses a specific experimental question: can multiple aromatic components have different antifungal activity compared to isolated substances? Here, the answer is limited to cultures: the cinnamaldehyde-eugenol pair showed an additive effect against the strain used in the combination test. This supports the idea that the chemical composition of a mixture may matter, without turning it into proof of treatment.The data relates to a possible biological pathway: when the yeast membrane loses integrity or the availability of ergosterol—a molecule essential for the structure of the fungal membrane—changes, the cell may lose internal components and fail to proliferate. Independent studies have explored this pathway in *Candida* models [1]. However, it remains necessary to distinguish a signal measured in a test tube from an effect in human tissue, where absorption, metabolism, local irritation, and immune system response radically change the context.

5. STUDY LIMITATIONS AND WHAT IT DOES NOT DEMONSTRATE

This is an *in vitro* study, meaning it was conducted on microorganisms cultured in a laboratory. It does not include human subjects, does not compare a therapy against a placebo, and does not measure recovery, recurrence, symptoms, or clinical safety. Therefore, it does not allow for the conclusion that the mixture prevents or cures intestinal, vaginal, oral, or invasive candidiasis.The combination test was performed on the *Candida albicans* strain 15; it is therefore not a demonstration applicable to all species and strains. Furthermore, the tests were conducted in duplicate, a factor that limits the precision of the estimates. The absence of an effect on *Lactobacillus acidophilus* at the tested concentrations is a favorable preliminary signal in the model, not proof of selectivity within the human gut microbiota. Finally, the mixture was additive and not synergistic: using these terms as equivalents would make the result appear stronger than what was reported by the authors.

6. INDEPENDENT CONFIRMATORY EVIDENCE

A first key point concerns the fungal membrane. Khan and colleagues studied a strain of *Candida albicans* in vitro using electron microscopy, flow cytometry, and biochemical tests. Cinnamaldehyde and eugenol damaged the cell wall and membrane and, at half of their respective MIC, reduced ergosterol biosynthesis by 67.41% and 76.23%. This result supports the transition from molecular exposure to loss of cellular integrity hypothesized in Saracino's study; the confirmation is mechanistic and moderate, but based on a single strain and not on a human organism [1].A second study examined two strains of *Candida albicans*, including pre-formed biofilms, which are adherent microbial communities that are more difficult to control. Eugenol and cinnamaldehyde reduced the biofilm and showed favorable interactions with fluconazole; FICI against biofilms ranged up to 0.14 for eugenol and 0.312 for cinnamaldehyde in specific comparisons. This does not verify the same mixture as the main study, but it confirms that both molecules can remain active in a more complex experimental condition. The limitation is the absence of clinical data [2].Alves and colleagues then evaluated cinnamaldehyde against multiple *Candida* species, multispecies biofilms, and human cells in culture. They observed fungicidal and anti-biofilm activity; computer simulations suggested possible interactions with proteins involved in the membrane. This evidence is indirect regarding the association with eugenol: molecular docking only indicates that a molecule could fit into a protein zone; it does not prove that the binding occurs in the organism. The level of confirmation therefore remains experimental [3].

7. CONFLICTING EVIDENCE

Results are not uniform across every species, combination, and model. De Sousa and colleagues examined 24 strains of *Candida auris* in vitro. Eugenol and cinnamaldehyde had a fungicidal effect, but the combination of each with itraconazole proved to be antagonistic or showed no detectable interaction. Furthermore, cinnamaldehyde was cytotoxic to Vero cell lines at concentrations active against the yeast, whereas eugenol was not in the same system. This does not refute the additive effect observed by Saracino and colleagues, but it shows that the response depends on the associated compound, the fungal species, and the cellular model [4].In a subsequent study on *Candidozyma auris* biofilms, eugenol and cinnamaldehyde reduced biomass in most clinical isolates, but metabolic activity increased in the majority of isolates during treatment. The authors proposed, without definitively proving it, the existence of heteroresistance, i.e., the presence of cell subgroups with different sensitivities. This finding suggests that one should not equate biomass reduction with complete biofilm inactivation. No directly conflicting evidence sufficiently relevant to the same cinnamaldehyde-eugenol mixture against the same strains has been identified; this does not prove the absence of contrary results [5].

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

Independent studies useful for contextualizing the experimental results of the main research.

Quadro comparativo

Publication	Model and main result	Relation to the main study	Interpretative limitation
Khan et al., 2013 [1]	A Candida albicans strain; reduction of ergosterol and membrane damage.	Confirms the possible cellular mechanism of the antifungal action.	Only one strain, no clinical data.
Khan and Ahmad, 2012 [2]	Two Candida albicans strains in biofilm; antibiofilm activity and favorable interactions with fluconazole.	Confirms the experimental activity of the two compounds in a more complex model.	Does not test the same cinnamaldehyde-eugenol mixture.
Alves et al., 2020 [3]	Candida spp., biofilm and cultured human cells; fungicidal and antibiofilm activity of cinnamaldehyde.	Supports the activity of cinnamaldehyde and hypotheses regarding the membrane.	Eugenol not evaluated as a component of the same mixture.
De Sousa et al., 2025 [4]	Twenty-four Candida auris strains; antagonistic or absent interactions with itraconazole.	Shows that the outcome changes with the species and the associated drug.	Does not examine the cinnamaldehyde-eugenol combination.
De Sousa et al., 2026 [5]	Candidozyma auris biofilm; reduced biomass but often increased metabolic activity.	Re-evaluates the interpretation of biofilm data alone.	Number of isolates not reported in the consulted summary; no clinical data.

9. SINTESI EDITORIALE E CONCLUSIONI

EDITORIAL CONCLUSION

The 2022 study by Saracino and colleagues shows that cinnamaldehyde and eugenol can inhibit *Candida* strains in culture and that, in the strain selected for the combination test, the association has an additive effect. This means that the substances maintained activity at lower concentrations than those required individually; however, this does not mean they have demonstrated a cure in humans. Independent evidence makes an involvement of the fungal membrane and ergosterol plausible and also confirms activity in some biofilm models. At the same time, studies on *Candida auris* indicate that the outcome can change depending on the species, biofilm, associated drug, and cell type used to evaluate toxicity. The most rigorous conclusion is therefore that the mixture deserves further preclinical research, with defined formulations and studies on safety and clinical efficacy, but it cannot replace established diagnoses or antifungal therapies.

Aggiornato al 05/11/2026. Questo contenuto riflette una sintesi divulgativa delle evidenze scientifiche al momento della pubblicazione e non sostituisce il parere medico.

10. TRANSPARENCY AND SCIENTIFIC RESPONSIBILITY

Artificial Intelligence was used solely as a tool for editorial and documentary support. All content, interpretations, bibliographic citations and conclusions have been verified, reviewed and approved by the author, who assumes full scientific responsibility for them.

The manuscript was also subjected to independent peer review prior to publication.

To ensure the greatest transparency, the complete history of the article, including the dates of submission, revision, acceptance and publication, as well as information on the peer review process, is available on the dedicated Article History page.

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