

# A RECENT REVIEW OF THE IMPACT OF LAVANDER ESSENTIAL OIL ON MALE AND FEMALE SEX HORMONES

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## Introduction

Lavender (*Lavandula angustifolia* Mill.) essential oil is widely used in cosmetics, aromatherapy, and traditional medicine. Concerns were raised after case reports suggested a possible association between dermal exposure to lavender-containing products and prepubertal gynecomastia or premature thelarche in children [1]. Early in vitro studies indicated weak estrogen receptor activation and anti-androgenic effects of its main constituents, linalool and linalyl acetate, leading to hypotheses about endocrine-disrupting potential. [2]

## Suggested mechanisms of the prior views on the effect [1, 2]

1. Estrogen receptor (ER) activation – Some in vitro studies and yeast assays suggested that linalool, linalyl acetate, or lavender oil extracts could weakly bind to and activate estrogen receptors, mimicking estrogen-like effects.
2. Androgen receptor (AR) antagonism – The same compounds were also reported to inhibit androgen receptor activity in cell-based assays, reducing androgen signaling.
3. Combined effect – This dual action (weak estrogenic + anti-androgenic activity) was thought to shift the hormonal balance toward a more estrogenic state, potentially explaining the case reports of prepubertal gynecomastia in boys and premature thelarche in girls.

## Recent evidence overview

### • Guideline-Compliant In Vitro & In Vivo Studies (2023–2024)

A comprehensive follow-up study published in Archives of Toxicology found no consistent estrogenic (ER) or androgenic (AR) activity for linalool (Lin) or linalyl acetate (LinAc) in both recombinant yeast screening assays and mammalian cell reporter tests. Although an initial anti-androgenic signal was noted in yeast assays, it was not confirmed in validated mammalian systems. [3]

In vivo studies—including Uterotrophic and Hershberger assays—showed no significant dose-related effects on hormone-sensitive organ weights, reproductive behavior, or developmental outcomes in rodents.

### • Epidemiological Evidence in Pediatric Populations

A cross-sectional study involving 556 children (aged 2–15) revealed no cases of prepubertal gynecomastia or other endocrine disorders linked to lavender or tea tree oil exposure. The prevalence of hormone-related issues in exposed children was comparable to those unexposed (risk ratio not statistically significant). [4]

### • Systematic Reviews Undermining Causal Links

A systematic review of available case reports found them methodologically weak, with only four case-series and low potential for causality. Conclusions state there is little to no evidence supporting a causal relationship between lavender essential oil and pediatric endocrine disruption. [2]

## Conclusion

More recent regulatory in vitro and in vivo studies have not confirmed these effects, highlighting the need for a critical evaluation of the evidence.

The current body of evidence does not support lavender essential oil or its main constituents as clinically relevant endocrine disruptors. Concerns raised by case reports and isolated in vitro findings are not substantiated by validated in vitro assays, computational models, or in vivo studies. These results emphasize the importance of integrating multiple lines of evidence before proposing causal links between exposure to natural products and endocrine-related outcomes. Further research should focus on potential confounding components in cosmetic formulations and employ standardized protocols to resolve remaining uncertainties.

## References:

- [1] Henley D. V., Lipson N., Korach K. S., Bloch C. A., (2007). Prepubertal gynecomastia linked to lavender and tea tree oils, *New England Journal of Medicine*, 356, 479–485.
- [2] Linklater A., Hewitt J. K., (2015). Premature thelarche in the setting of high lavender oil exposure, *Journal of Paediatrics and Child Health*, 51, 235.
- [3] Hareng L., Kollé S. N., Gomes C., Schneider S., Wahl M., (2024). Critical assessment of the endocrine potential of linalool and linalyl acetate: proactive testing strategy assessing estrogenic and androgenic activity of lavender oil main components, *Archives of Toxicology*, 98(1), 347–361.
- [4] Hawkins J., Hires C., Dunne E., Keenan L., (2022). Prevalence of endocrine disorders among children exposed to lavender essential oil and tea tree essential oils, *International Journal of Pediatrics and Adolescent Medicine*, 9(2), 117–124.



Fig.1 *Lavandula angustifolia* 'Sevtopolis', one of the frequently cultivated genotypes in Eastern Europe, inflorescence (owner: Ioana C., 2022)

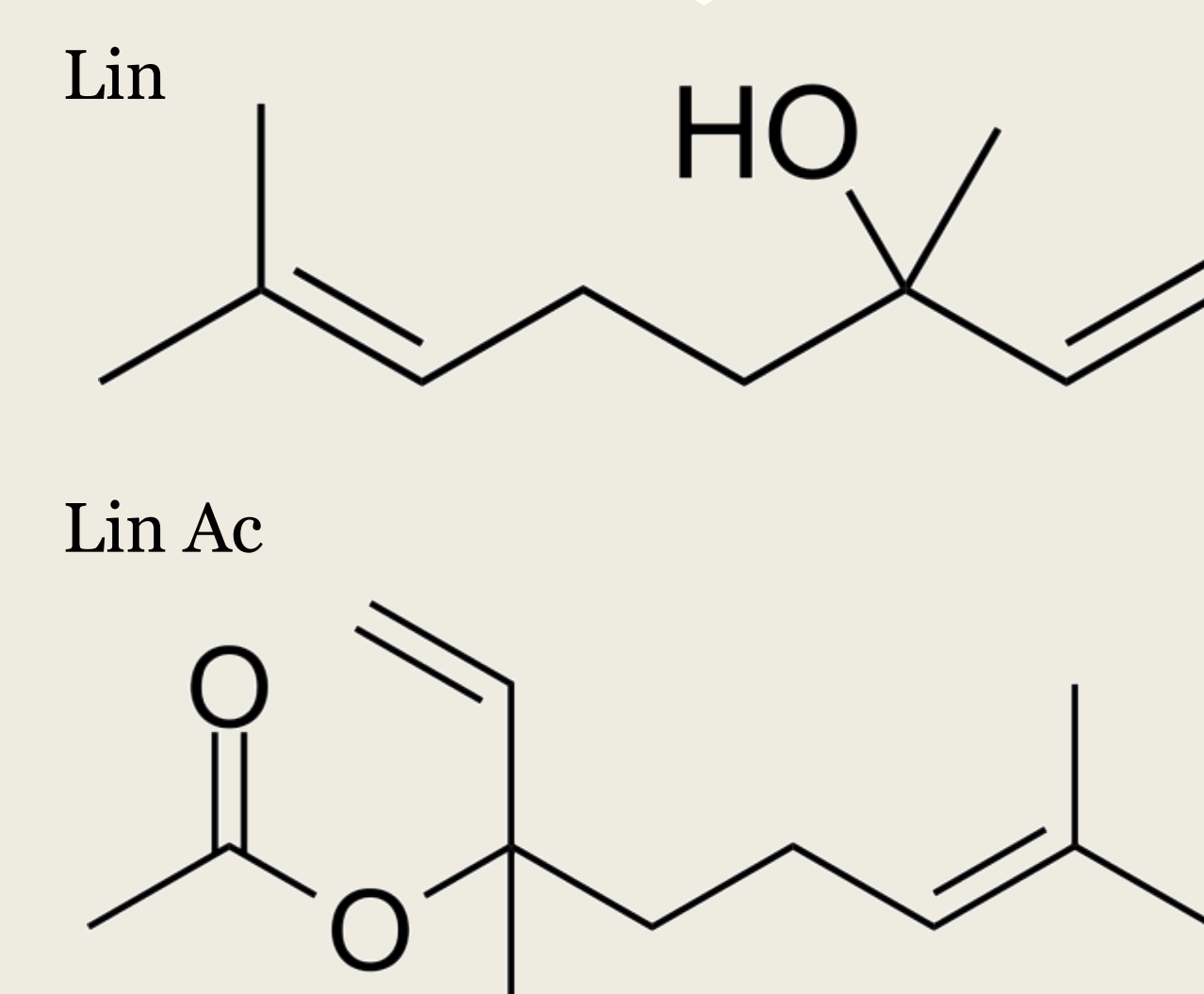


Fig.2 Lavender essential oil and its main components, linalool (Lin) and linalyl acetate (LinAc) (owner: MedChemExpress)

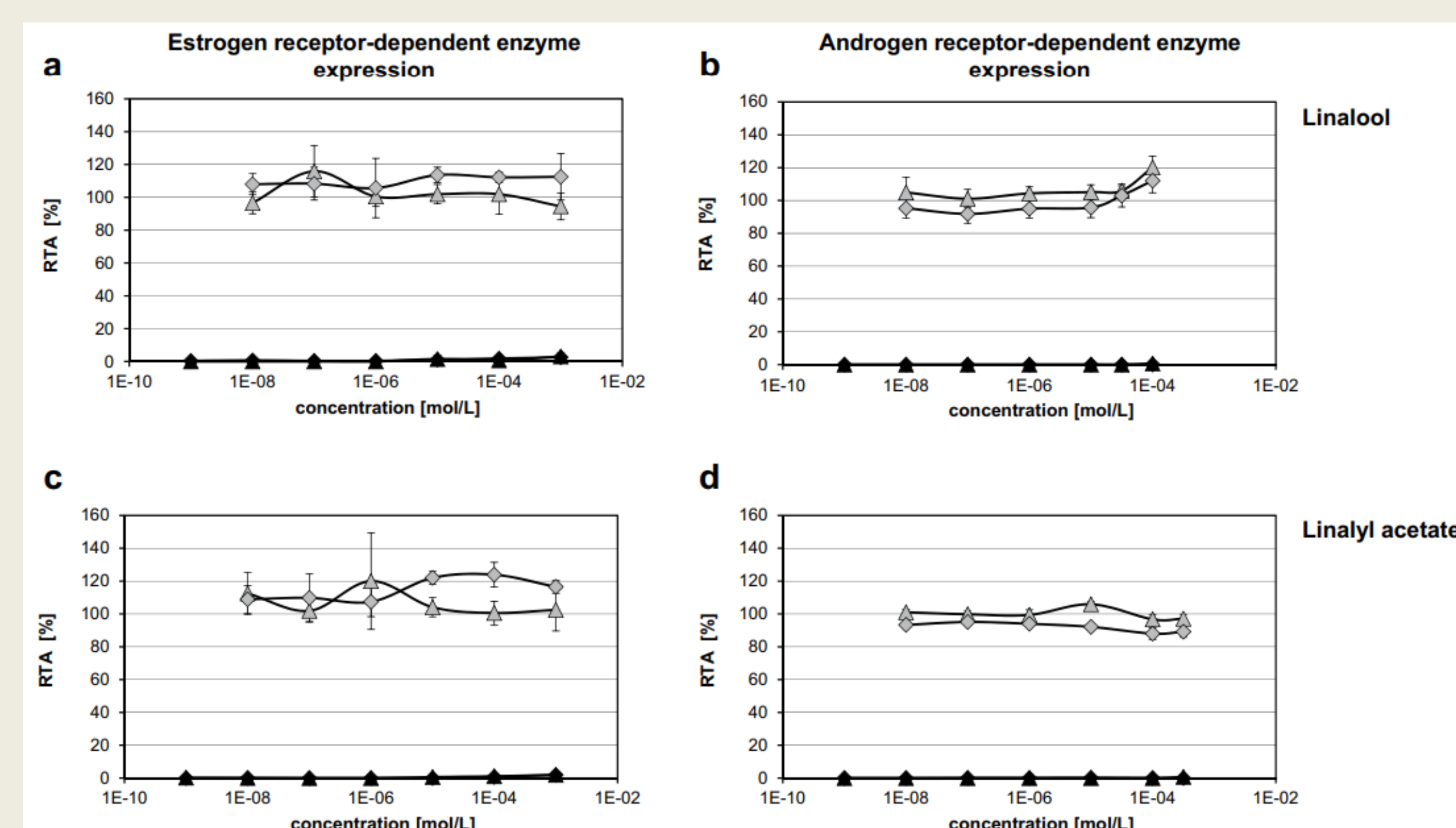


Fig 3. Estrogen (ERTA) and androgen (ARTA) receptor reporter assays. Luciferase activity was measured after 20–24 h incubation with linalool (a/b) or linalyl acetate (c/d) at  $10^{-9}$ – $10^{-3}$  M, in the absence (solid) or presence (open grey) of 25 pM E2 (anti-ER) or 500 pM DHT (anti-AR). Relative transcriptional activity (RTA) was normalized to positive controls (1 nM E2 or 10 nM DHT = 100%) and spike-in controls (25 pM E2 or 500 pM DHT = 100%). Results are % mean RTA  $\pm$  SD from two independent experiments (triangles/diamonds, n=3 wells/condition). [3]

| Endocrine disorder        | Frequency | Percent (%) | t     |
|---------------------------|-----------|-------------|-------|
| Prepubertal gynecomastia  | 0         | 0           | –     |
| Precocious puberty        | 2         | 0.49        | 1.36  |
| Delayed puberty           | 1         | 1.39        | –0.80 |
| Growth hormone deficiency | 1         | 0.49        | 1.35  |
| Hypothyroidism            | 4         | 0.73        | 1.41  |

Fig. 5 The table summarizes the frequency and prevalence of endocrine disorders observed among 556 children exposed to lavender and tea tree essential oils. Out of nine total cases, the most common disorder identified was hypothyroidism (0.73%, t=1.41), followed by precocious puberty (0.49%, t=1.36), growth hormone deficiency (0.49%, t=1.35), and delayed puberty (1.39%, t=–0.8). No cases of prepubertal gynecomastia were reported. Statistical comparisons (one-sample t-tests) showed that the prevalence rates in this exposed group were not significantly different from established population norms. [4]



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