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ANTIVIRAL AND ANTI-INFLAMMATORY PROPERTIES OF LEMON BALM (MELISSA OFFICINALIS L.)

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Abstract

Lemon balm, also known as melissa (Melissa officinalis L.), belongs to the mint family (Lamiaceae) and grows as a perennial herb. Native to Southern Europe, it now spans regions from North America to New Zealand [1]. Its name traces back to the Greek word “melissa”, meaning “honeybee”. Ancient writers already valued its medicinal uses: Dioscorides wrote about it in De Materia Medica, and Ibn Sina included information on the plant in The Canon of Medicine [2].

Essential oil, phenolic acids, flavonoids, and triterpenes accumulate in the leaves. Monoterpene aldehydes—specifically citral a, citral b, and citronellal—make up the main share of the essential oil [3]. Among phenolic compounds, rosmarinic acid stands out; World Health Organization monographs list it as a biological marker for checking plant quality [2]. Across published reviews, lemon balm is linked to antioxidant, anti-inflammatory, antimicrobial, spasmolytic, and sedative actions [4, 5, 6].

Antiviral activity warrants closer attention here. Because herpes viruses can develop resistance to standard antiviral drugs, researchers look to plant extracts and essential oils as potential anti-herpetic options [3, 7]. Anti-inflammatory properties matter just as much in chronic conditions driven by oxidative stress.

This review brings together foreign literature on the antiviral and anti-inflammatory effects of lemon balm, comparing findings on active constituents and their stages of action.

Keywords: “Melissa officinalis”, “lemon balm”, “antiviral activity”, “herpes simplex virus”, “anti-inflammatory” and “rosmarinic acid”.

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Introduction

METHODS. This work was structured as a literature review. Sources were gathered from open scientific databases, publisher websites, and university archives. Studies made the cut when lemon balm essential oil, extract, or an isolated compound was tested directly, the experimental model (cell culture, animals, or patients) was explicitly stated, and quantitative results were reported. In total, twelve English-language publications from 1999–2025 entered the analysis: 5 experimental studies, 1 clinical trial, and 6 review papers.

Findings were organized by preparation type (essential oil, aqueous extract, individual compound), experimental model, and observed outcome. Differences in study designs and measurement units ruled out a meta-analysis, so comparisons were made descriptively. Quantitative values were extracted from article abstracts and open full texts. The workflow appears in Figure 1.

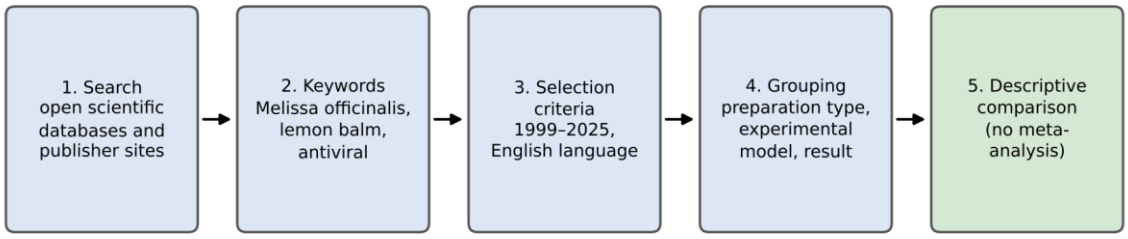


Figure 1. Literature search and analysis workflow

RESULTS

Essential oil. In HEp-2 cells, Allahverdiyev et al. reported no cytotoxicity from lemon balm essential oil up to 100 µg/ml, alongside suppression of herpes simplex virus type 2 (HSV-2) replication at those concentrations [8]. Schnitzler et al. used gas chromatography–mass spectrometry (GC-MS) to profile the oil and identified citral a, citral b, and citronellal as its principal constituents. Tested in monkey kidney cells, half-maximal plaque reduction (IC_{50}) occurred at 0.0004% for HSV-1 and 0.00008% for HSV-2. Activity appeared only when the oil was added prior to viral entry into host cells; once the virus had entered, no effect remained. Given its lipophilicity and capacity to cross skin, the authors suggest potential topical use against herpes [3].

Aqueous extract. Most studies evaluating the aqueous extract and its phenolic acids—caffeic, p-coumaric, and rosmarinic acids—focused on HSV-1. Once cells were already infected, the extract changed neither plaque formation nor expression of the viral ICP0 protein. Free viral particles, however, were inactivated directly, even at concentrations as low as 1.5 µg/ml. Dose-dependent inhibition of viral attachment to host cells was also observed. Reaching comparable inhibition with individual phenolic compounds required roughly 100-fold higher concentrations. The extract reached a selectivity index of 875—above those of the isolated phenolics—with rosmarinic acid contributing most [7]. Later work examined the extract against two acyclovir-resistant HSV strains. Against free virions, IC_{50} values reached 0.13 and 0.23 µg/ml, corresponding to selectivity indices of 2692 and 1522. Viral entry fell by 80% in drug-sensitive strains and by 96% in resistant strains [9]. Overall, these preparations act extracellularly during attachment and entry (Figure 2).

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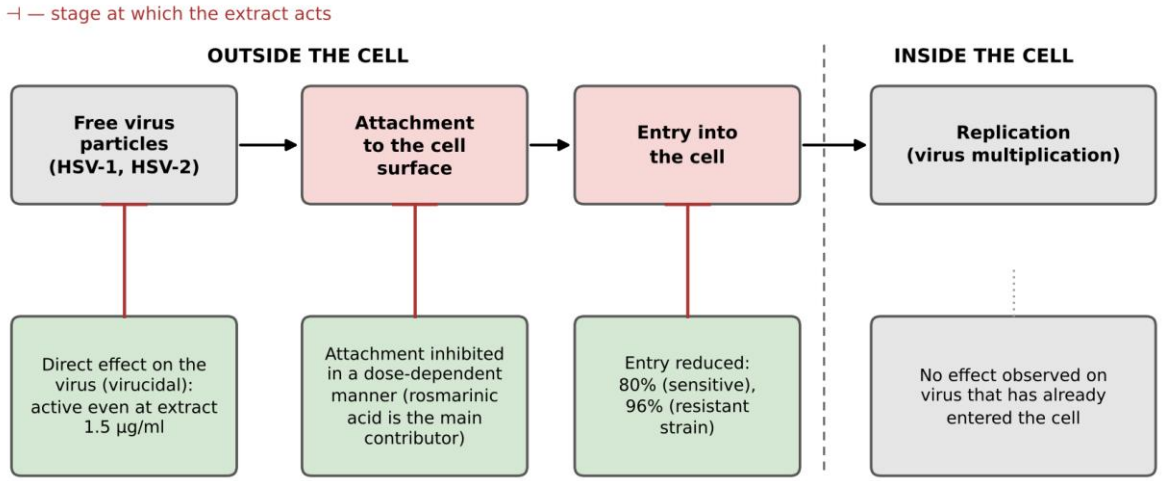


Figure 2. Action of lemon balm extract on the stages of herpes simplex virus entry into the cell (based on data from [7, 9])

Anti-inflammatory activity. Rosmarinic acid, flavonoids, and essential oil are the main drivers cited for anti-inflammatory effects [4, 6]. Leaf essential oil given orally at 200 or 400 mg/kg was evaluated against 10 mg/kg indomethacin in a rat paw edema model [4]. Three extract types—ethanolic maceration, aqueous extract, and ethanolic extract—were further compared using the carrageenan model. Rosmarinic acid led in content across all extracts, while flavonoids, notably quercetin, were higher in the aqueous preparation. Six hours after carrageenan injection, edema reduction peaked at 63.89% with ethanolic maceration (200 mg/kg) and 69.44% with the aqueous extract (100 mg/kg), compared with 76.67% for indomethacin. Across all three extracts, hydrogen peroxide and TBARS dropped while glutathione (GSH) rose [10] (Figure 3).

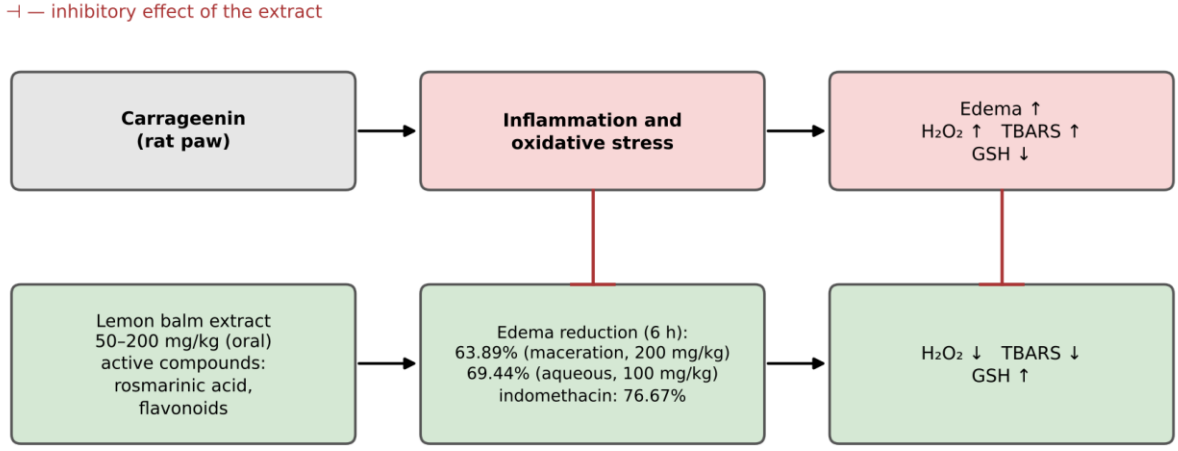


Figure 3. Scheme of the action of lemon balm extract in the carrageenan model (based on data from [10])

CONCLUSION

Essential oil and aqueous extract from lemon balm show activity against herpes simplex virus, including acyclovir-resistant strains. Their action centers on viral attachment and entry, with

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rosmarinic acid playing a central role in the extract. In animal models, extracts eased inflammation and improved oxidative stress markers. Human trials remain scarce and small, so clinical efficacy still lacks firm proof. Even so, standardized preparations and larger clinical studies offer a clear path forward for stronger analytical and clinical evidence.

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