© 2026 Journal of Pharmacy & Pharmacognosy Research, 14 (5), 2688, 2026
ISSN 0719-4250
https://jppres.com

DOI: https://doi.org/10.56499/jppres_14.5.2688

Review

Antiviral and virucidal properties of essential oils against human viruses: A systematic review

[Propiedades antivirales y virucidas de los aceites esenciales contra virus humanos: una revisión sistemática]

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Abstract

Context: Essential oils (EOs) have been investigated as potential antiviral agents, particularly because of their extracellular virucidal activity.

Aims: This review synthesized evidence on whole EOs tested against viruses, with particular emphasis on human-virus evidence, and summarized reported mechanisms of action.

Methods: Web of Science, PubMed, EMBASE, Scopus, Google Scholar, CINAHL, and the Cochrane Library were searched from inception to February 2023. Human, animal, plant-model, and mechanistic studies of whole EOs were eligible. Isolated constituents, fractions, insufficiently identified oils, and most EO mixtures were excluded. Risk of bias was assessed with the OHAT tool and its *in vitro* extension.

Results: A total of 198 reports were included, yielding 203 design-specific experiments. Of these, 164 concerned human viruses or related surrogates. Most evidence was mechanistic. Overall, 123 experiments were rated Tier 1, 74 Tier 2, and 6 Tier 3. Activity was most frequently reported against herpes simplex viruses, influenza viruses, SARS-CoV-2, dengue virus, and coxsackieviruses. Several EOs showed favorable efficacy and selectivity in selected assays, and pre-adsorption mechanisms predominated.

Conclusions: Whole EOs showed promising experimental antiviral activity, but clinical applicability remains uncertain because human evidence is limited and study designs, EO characterization, cytotoxicity reporting, and outcome measures were heterogeneous.

Keywords: antiviral agents; cytotoxicity tests; herpesvirus 1, human; oils, volatile; virucidal agents.

Resumen

Contexto: Los aceites esenciales (AE) se han investigado como posibles agentes antivirales, en particular por su actividad virucida extracelular.

Objetivos: Esta revisión sintetizó la evidencia sobre AE completos evaluados frente a virus, especialmente humanos, y resumió los mecanismos de acción descritos.

Métodos: Se realizaron búsquedas en Web of Science, PubMed, EMBASE, Scopus, Google Scholar, CINAHL y Cochrane Library desde su inicio hasta febrero de 2023. Se incluyeron estudios en humanos, animales y modelos vegetales, así como estudios mecanísticos sobre AE completos. Se excluyeron componentes aislados, fracciones, aceites insuficientemente identificados y la mayoría de las mezclas de AE. El riesgo de sesgo se evaluó mediante la herramienta OHAT y su extensión *in vitro*.

Resultados: Se incluyeron 198 artículos, que aportaron 203 experimentos específicos por diseño. De ellos, 164 se referían a virus humanos o sustitutos relacionados. La mayoría de la evidencia fue mecanística. En total, 123 experimentos se clasificaron en el nivel 1, 74 en el nivel 2 y 6 en el nivel 3. La actividad se notificó con mayor frecuencia frente a los virus del herpes simple, de la gripe, del SARS-CoV-2, del dengue y de los coxsackievirus. Varios AE mostraron eficacia y selectividad favorables en ensayos seleccionados, y predominaron los mecanismos previos a la adsorción.

Conclusiones: Los AE completos mostraron una actividad antiviral experimental prometedora, pero su aplicabilidad clínica sigue siendo incierta debido a la escasez de evidencia en humanos y a la heterogeneidad en el diseño de los estudios, la caracterización de los AE, la notificación de citotoxicidad y las medidas de resultado.

Palabras Clave: aceites volátiles; agentes antivirales; agentes virucidas; herpesvirus humano 1; pruebas de citotoxicidad.

ARTICLE INFO
Received: November 28, 2025.
Accepted: September 19, 2026.
Available Online: October 15, 2026.

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Abbreviations: **CC_{50}**: 50% cytotoxic concentration; **EC_{50}**: 50% effective concentration; **EC_{90}**: 90% effective concentration; **EO**: essential oil; **HSV-1**: herpes simplex virus type 1; **HSV-2**: herpes simplex virus type 2; **IC_{50}**: 50% inhibitory concentration; **ID_{50}**: 50% inhibitory dose; **RoB**: risk of bias; **SI**: selectivity index.

INTRODUCTION

The COVID-19 pandemic renewed scientific interest in the development of novel antiviral agents, particularly compounds capable of acting through extracellular virucidal mechanisms. In parallel, the progressive emergence of antiviral resistance has sustained interest in agents that may complement conventional intracellular antivirals (Canini et al., 2014).

Essential oils (EOs) are complex volatile mixtures obtained from plant material by steam distillation, hydro-distillation, or mechanical expression. They are usually rich in terpenoids and oxygenated derivatives, although phenylpropanoids and other chemical classes may also contribute substantially to their composition (Sharifi-Rad et al., 2017). Experimental studies have suggested antiviral activity against several enveloped RNA and DNA viruses, especially herpes simplex viruses, influenza viruses, and coronaviruses, whereas evidence for non-enveloped human viruses is more limited (Schnitzler, 2019; Schnitzler et al., 2011).

Reported mechanisms are heterogeneous and incompletely characterized. Besides direct virucidal effects, EOs may interfere with viral adsorption, membrane fusion, intracellular replication, or release from infected cells. However, the literature is fragmented across virus families, assay systems, outcome measures, and levels of evidence, and EO characterization is often insufficiently standardized.

A systematic synthesis was therefore needed. Previous reviews were largely narrative and often focused on selected viruses or isolated EO constituents. The objective of this review was to identify, summarize, and critically appraise studies evaluating whole EOs against viruses, with detailed emphasis on human viruses and related surrogate models. Efficacy, selectivity, risk of bias, and reported mechanisms of action were examined in order to identify the most promising candidates and the main limitations of the current evidence base.

MATERIAL AND METHODS

Review methodology

This systematic literature search was conducted following the "Handbook for Conducting Systematic Reviews for Health Effects Evaluations" by the National Toxicology Program (OHAT, 2019). The review protocol was registered on the Open Science Framework on 6 October 2022, after the review had commenced but before its completion (Valussi et al., 2022).

See Supplementary Material for a PRISMA 2020 Checklist and a PRISMA-S Checklist (Search reporting).

Eligibility criteria

Eligible evidence included human studies, animal studies, experimental plant-model studies, and mechanistic *in vitro* studies evaluating whole EOs against viral infection, viral replication, or virus-induced effects.

There were no restrictions according to sex, life stage, route of administration, or cell and tissue model. Studies evaluating isolated compounds, EO fractions, mixtures of different EOs, mixtures of EOs with other extracts (except the three qualifying fixed clinical combinations described below), non-EO aromatic preparations (according to the ISO (International Organization for Standardization) definition of EOs [ISO 9235:2021] (ISO, 2021)) or insufficiently identified EOs were excluded.

No restriction was applied to the publication period, and no language filter was applied at the search stage; however, two retrieved reports could not be assessed because no translation was available (see below). Grey literature and unpublished studies were not actively sought. Clinical studies were eligible only when a comparator or reference condition—including placebo, no treatment, usual care, vehicle, or a within-participant baseline in a pre–post design—enabled an effect estimate; reports lacking such a reference condition were excluded from the synthesis.

Deviation from the registered protocol: fixed combinations of whole essential oils in clinical studies. The protocol (Valussi et al., 2022) restricted eligible exposures to single, whole EOs, excluding mixtures. The purpose of this restriction was to permit attribution of an observed antiviral effect to a defined, chemically characterised botanical material. That rationale applies with full force to mechanistic and preclinical studies, where the analytical object is the activity of a specific EO and its constituents, but it applies weakly to pragmatic clinical trials, whose outcome is a patient-level measure of symptom duration or severity rather than an attributable *in vitro* effect.

The eligibility criterion was therefore amended post hoc, before risk-of-bias assessment and before any efficacy synthesis, to admit human clinical studies evaluating fixed, fully specified combinations of whole EOs, provided that every component was an unmodified whole essential oil identified to species, that the composition and proportions were stated or subject to

pharmacopoeial specification, and that no isolated compounds, solvent extracts or undisclosed proprietary mixtures were involved. Combinations not meeting all of these conditions remained excluded.

Three human clinical trials met the amended criterion. Ben-Arye et al. (2011), Duijker et al. (2015), and Pfaar et al. (2023) evaluated fixed, fully specified combinations of whole EOs meeting the amended eligibility criterion. In each case, the preparation was sufficiently specified for inclusion under the amended clinical criterion, and no isolated compounds, solvent extracts, or undisclosed proprietary mixtures were involved. All three were assessed for risk of bias on the same terms as the single-oil clinical studies.

Reports were eligible irrespective of country of origin, provided they were published in a language accessible to the review team. Two reports published in Persian (Azizkhani & Tooryan, 2016; Khanavi et al., 2010) could not be assessed because no translation was available, and were excluded on this basis.

Information sources

The following databases were searched: Web of Science, Medline via PubMed, EMBASE, Scopus, Google Scholar, CINAHL, and the Cochrane Library. The original search was performed on all databases on 29 April 2020; the search was updated in two steps. On 13 February 2023, four databases (Web of Science, Medline via PubMed, Google Scholar, and the Cochrane Library) were searched, while the remaining three (CINAHL, EMBASE, Scopus) were searched on 17 February 2023 using the same database-specific strategies. Searches targeted viruses, EOs, *in vitro* activity, animal *in vivo* activity, *ex vivo* activity, and clinical activity.

Search strategy

Database-specific search strings combined the exposure and outcome elements of the PECO framework with Boolean operators (Morgan et al., 2018). Records were imported into EndNote and duplicates were removed manually after automated aggregation. Google Scholar results were screened by relevance order and limited to the first 1,150 records because of the very high redundancy and progressively negligible yield beyond that threshold. Backward and forward citation searching was not performed. The full search strategies are reported in Table 1.

Table 1. Search strategies for electronic databases

Database	Search string
PubMed/Medline	“((((essential[Title/Abstract] OR volatile[Title/Abstract] OR ethereal[Title/Abstract] OR herbal[Title/Abstract]) AND (oil[Title/Abstract] OR oils[Title/Abstract])) OR aromatherapy[Title/Abstract] OR distillate[Title/Abstract] OR aetheroleum[Title/Abstract] OR essence[Title/Abstract] OR terpenoid[Title/Abstract] OR terpenoids[Title/Abstract] OR terpene[Title/Abstract] OR terpenes[Title/Abstract] OR "Oils, Volatile"[MeSH Terms] OR "Aromatherapy"[MeSH Terms]) AND (virus[Title/Abstract] OR virucidal[Title/Abstract] OR antiviral[Title/Abstract] OR virustatic[Title/Abstract] OR antinfective[Title/Abstract] OR viral[Title/Abstract] OR virion[Title/Abstract] OR virological[Title/Abstract] OR "Viruses"[MeSH Terms] OR Disinfectant[Title/Abstract]))”
Web of Science	“TOPIC: (volatile oil OR volatile oils OR ethereal oil OR ethereal oils OR herbal oil OR herbal oils OR essential oil OR essential oils OR aromatherapy OR distillate OR aetheroleum OR aetherolium OR essence OR terpenoid OR terpenoids OR terpene OR terpenes) AND TOPIC: (virus OR virucidal OR antiviral OR virustatic OR virostatic OR antinfective OR antiinfective OR viral OR virion OR virological OR Viruses OR disinfectant)”
Scopus	“(TITLE-ABS-KEY ("volatile oil" OR "volatile oils" OR "ethereal oil" OR "ethereal oils" OR “herbal oil" OR "herbal oils" OR "essential oil" OR “essential oils" OR aromatherapy OR distillate OR aetheroleum OR essence OR terpenoid OR terpenoids OR terpene OR terpenes) AND TITLE-ABS-KEY (virus OR virucidal OR antiviral OR virustatic OR antinfective OR viral OR virion OR virological OR viruses))“
Google Scholar	“(volatile oil OR volatile oils OR ethereal oil OR ethereal oils OR herbal oil OR herbal oils OR essential oil OR essential oils OR aromatherapy) AND (virus OR virucidal OR antiviral OR virustatic OR antinfective)”
Cochrane	“(volatile oil OR volatile oils OR ethereal oil OR ethereal oils OR herbal oil OR herbal oils OR essential oil OR essential oils OR aromatherapy OR distillate OR aetheroleum OR essences OR terpenoid OR terpenoids OR terpene OR terpenes):ti,ab,kw AND (virus OR virucidal OR antiviral OR virustatic OR antiinfective OR viral OR virion OR virological OR Viruses):ti,ab,kw“
EMBASE	“('volatile oil':ab,ti OR 'volatile oils':ab,ti OR 'ethereal oil':ab,ti OR 'ethereal oils':ab,ti OR 'herbal oil':ab,ti OR 'herbal oils':ab,ti OR 'essential oil':ab,ti OR 'essential oils':ab,ti OR aromatherapy:ab,ti OR distillate:ab,ti OR aetheroleum:ab,ti OR essence:ab,ti OR terpenoid:ab,ti OR terpenoids:ab,ti OR terpene:ab,ti OR terpenes:ab,ti) AND (virus:ab,ti OR virucidal:ab,ti OR antiviral:ab,ti OR virustatic:ab,ti OR antinfective:ab,ti OR viral:ab,ti OR virion:ab,ti OR virological:ab,ti OR viruses:ab,ti)”
CINAHL	“TI (volatile oil OR volatile oils OR ethereal oil OR ethereal oils OR herbal oil OR herbal oils OR essential oil OR essential oils OR aromatherapy OR distillate OR aetheroleum OR essence OR terpenoid OR terpenoids OR terpene OR terpenes) AND TX (virus OR virucidal OR antiviral OR virustatic OR antinfective OR viral OR virion OR virological OR Viruses)”

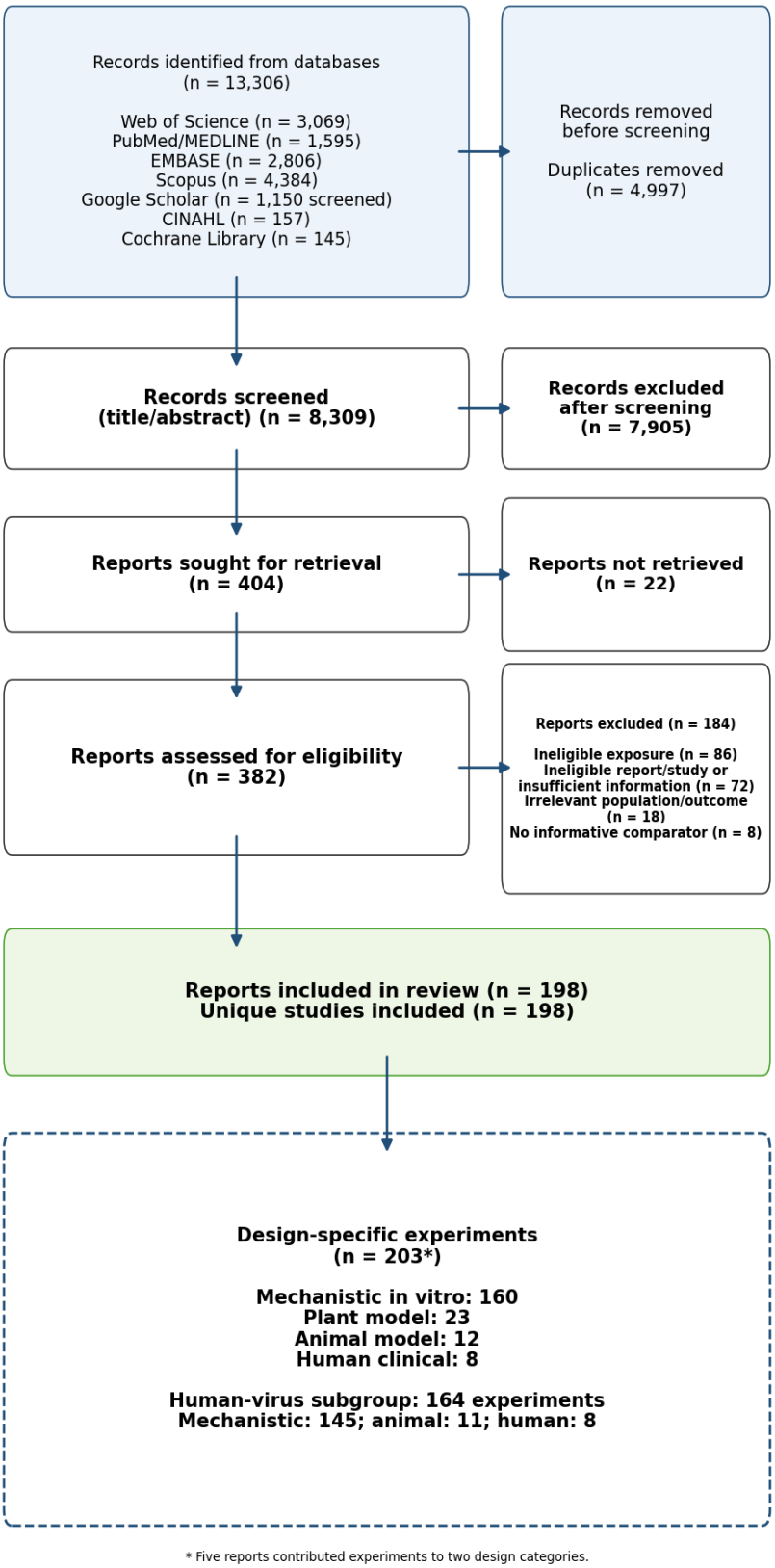


Figure 1. PRISMA 2020 flow diagram of study selection.

Selection process

Study selection was performed in two stages and is summarized in the PRISMA flow diagram (Fig. 1). Two authors (MV and DP) independently screened records against prespecified eligibility criteria.

Stage 1 involved title and abstract screening. Records lacking enough information for confident exclusion were retained for full-text review. Disagreements were resolved by discussion and, when required, arbitration by a third author (PF). A formal inter-rater agreement statistic was not calculated and this is acknowledged as a limitation.

Stage 2 consisted of full-text review of all retained reports.

When a full text could not be retrieved through institutional or online resources, the corresponding author was contacted. This applied to 22 reports. No full text was obtained through these contacts; the 22 reports were classified as not retrieved and did not enter full-text eligibility assessment. Exclusions among retrieved full texts were assigned one mutually exclusive primary reason.

Inclusion/exclusion criteria

The operational eligibility criteria are summarized in Table 2.

Table 2. Operational PECO eligibility criteria applied to the studies.

A. Inclusion	
P (Population)	
Human studies	Human subjects with any type of viral infection without restriction based on sex or on life stage at exposure or outcome assessment
Animal models	Non-human primates, rodents, and any other experimental animal model in which viral infections may be induced
Mechanistic	All types of tissue cultures in which viruses can be cultured
Plant models	Experimental plants challenged with plant viruses or viral inocula
E (Exposure)	
Human studies	Single whole essential oils administered to humans by any reported route. A post hoc clinical exception admitted only the fixed, fully specified whole-EO combinations evaluated by Ben-Arye et al. (2011), Duijker et al. (2015), and Pfaar et al. (2023).
Animal models	Single whole essential oils are administered to animal models under any form of administration (per os, percutaneous, inhalation, i.v., i.p., etc.).
Mechanistic	Exposure of cells and tissue cultures to single, whole essential oils
Plant models	Single whole essential oils administered before, with, or after viral inoculation in experimental plant models
C (Control)	
Human studies	A comparator or reference condition enabling effect estimation: placebo, no treatment, treatment as usual, vehicle control, or a within-participant baseline in a pre–post design.
Animal models	Animals not exposed to essential oils or treated only with the vehicle control, or a different placebo or with standard treatment
Mechanistic	Cell-treatment or tissue-treatment group not exposed to essential oils or treated only with the vehicle control
Plant models	Plants or leaves not exposed to EOs, treated with vehicle, or challenged with virus under otherwise comparable conditions
O (outcomes)	
Human studies	Duration, severity, and symptoms of the viral infection; viral suppression; the measure of viral load
Animal models	Duration, severity, and signs of the viral infection; survival time; viral suppression; the measure of viral load
Mechanistic	In cell and tissue cultures. EC_{50} and EC_{90} values
Plant models	Local lesion counts, disease incidence or severity, symptom development, viral titre, or other direct plant-virus outcomes
Other.	
Publication types	Primary research reports meeting the operational PECO criteria.
B. Exclusion	
P (Population)	NA
E (Exposure)	Isolated compounds; aromatic extracts that do not meet ISO 9235:2021; mixtures of different essential oils or of essential oils with other extracts, except the three qualifying fixed clinical combinations identified above; and all non-clinical studies of mixtures.
C (Control)	Studies without a comparator or reference condition enabling effect estimation.
O (outcomes)	Lack of effect measure
Publication types	Reports without extractable primary numerical data, or with insufficient information to determine eligibility or interpret an effect estimate.

Table 3. Risk of bias answer format.

	RoB	Explanation
++	Definitely Low risk of bias	There is direct evidence of low risk-of-bias practices
+	Probably Low risk of bias	There is indirect evidence of low risk-of-bias practices **OR** it is deemed that deviations from low risk-of-bias practices for these criteria during the study would not appreciably bias results, including consideration of direction and magnitude of bias
-	Probably High risk of bias	There is indirect evidence of high risk-of-bias practices **OR** there is insufficient information (e.g., not reported or "NR") provided about relevant risk-of-bias practices
- -	Definitely High risk of bias	There is direct evidence of high risk-of-bias practices

Table 4. Operational OHAT-informed overall risk-of-bias tier rule.

Tier	Definition
1	Mechanistic, plant-model, and human: both key domains Low/Some concerns and a majority of other applicable domains Low/Some concerns. Animal: at least four Low domains and no High/Very high domain.
2	All combinations not classified as Tier 1 or Tier 3.
3	Mechanistic, plant-model, and human: both key domains High/Very high and a majority of other applicable domains High/Very high. Animal: at least three High/Very high domains.

Data collection process

Data extraction was performed independently by two authors (MV and DP) using a prespecified set of variables, and extracted data were checked for accuracy and completeness by two additional authors (DD and PF). Discrepancies were resolved by consensus.

Author contact was used only to attempt retrieval of the 22 unavailable full texts. Because no full text was obtained, these contacts did not change eligibility, data extraction, or risk-of-bias judgments.

Extracted items included first author and year; funding; botanical source and characterization of the EO; dose or concentration; duration and route of administration; study design; objectives; comparator; outcome measures; and adverse-event or cytotoxicity data when available.

For human studies, participant characteristics, clinical outcomes, virological outcomes, and adverse events were extracted. For animal studies, model characteristics and infection-related outcomes were extracted. For plant-model studies, host species, inoculation procedures, exposure timing, comparators, and lesion, symptom, or viral outcomes were extracted. For mechanistic studies, cell line or tissue model, virus, assay type, EO concentration, vehicle, exposure timing, CC_{50} or related toxicity measures, and effect estimates were extracted.

Risk of bias assessment

Risk of bias was assessed using the OHAT Risk of Bias Rating Tool for Human and Animal Studies together with the published OHAT extension for *in vitro* studies (OHAT, 2019). Initial domain-level risk-of-bias assessments were performed independently by MV, DP, and PF. Disagreements were discussed among the assessors and, when necessary, resolved through expert adjudication by MA and DD until consensus was reached.

The OHAT RoB framework covers selection, confounding, performance, attrition, detection, reporting, and other sources of bias. The corresponding questions and their applicability to the different study designs are summarized in Supplementary Table A1.

All applicable domains were considered during assessment. Because randomization, allocation concealment, and blinding were rarely reported in mechanistic and plant-model studies, these items were interpreted conservatively rather than being treated as proof of high-quality conduct; exposure characterization and outcome assessment remained the key domains driving overall judgment (see Table 3). Domains coded as not applicable were excluded from the applicable-domain denominator.

Overall RoB tiers were assigned using a prespecified OHAT-informed, design-specific categorical rule. Domain-level judgments were summarized using the design-specific criteria reported in Table 4, and the resulting Rule Tier was used for each experiment. The supplementary RoB workbook reports the domain judgments and Rule Tier for each experiment. For mechanistic, plant-model, and human experiments, Tier 1 required both key domains (D3 and D4) and a majority of other applicable domains to be Low or Some concerns; Tier 3 required both key domains and a majority of other applicable domains to be High or Very high; all other combinations were Tier 2. For animal studies, Tier 1 required at least four Low domains and no High or Very high domain, whereas Tier 3 required at least three High or Very high domains; all

other combinations were Tier 2. The tier was a categorical integration of domain judgments rather than a numerical score. Domain judgments and Rule Tiers are supplied in the RoB workbook.

Data synthesis

Because the included evidence was highly heterogeneous with respect to virus families, cell lines, models, formulations, assay endpoints, and effect measures, formal meta-analysis was not considered appropriate (for the list of evidence, see References A13.1 in Supplementary Materials). The results were therefore synthesized qualitatively and structured according to study design, virus group, effect size, selectivity, and reported mechanisms of action. The EC_{50} and SI categories used in the manuscript were intended as pragmatic descriptive rankings to facilitate comparison across highly heterogeneous experiments, not as validated pharmacological cutoffs. A formal certainty-of-evidence framework such as GRADE was not applied because the dataset was dominated by mechanistic studies with non-comparable outcomes; this is acknowledged as a limitation. Sensitivity analyses were conducted by restricting the synthesis to Tier 1 studies and by excluding studies admitted under the amended eligibility criterion.

Ethics statement

Not applicable – this study did not involve new data collection from human participants or animals.

RESULTS

Study selection

The original database search was performed in April 2020, and the update was completed in February 2023. In total, 13,306 records were retrieved (Cochrane: 145; PubMed: 1,595; Google Scholar: 1,150 screened; Web of Science: 3,069; Scopus: 4,384; EMBASE: 2,806; CINAHL: 157). After removal of 4,997 duplicates, 8,309 records underwent title and abstract screening. Of 404 reports sought for retrieval, 22 could not be retrieved after corresponding-author contact; 382 reports were assessed for eligibility.

Of the 382 reports assessed for eligibility, 184 were excluded using one primary reason per report: ineligible exposure (n = 86), ineligible report/study or insufficient information (n = 72), irrelevant population/outcome (n = 18), and no informative comparator (n = 8). The remaining 198 reports, representing 198 unique studies, were included in the qualitative synthesis.

The 198 included reports generated 203 design-specific experiments. Of these, 160 were mechanistic, 12 were animal, 23 were plant-model, and 8 were human clinical experiments. Five reports contributed experiments to more than one design category.

Details of the screening and selection process are shown in the PRISMA flow diagram (Fig. 1).

Characteristics of included studies

Among the 198 included reports, 155 were mechanistic-only studies, 21 were plant-model-only studies, 9 were animal-model-only studies, 8 were human-clinical-only studies, and 5 were mixed-design studies (3 animal model plus *in vitro* and 2 plant model plus *in vitro*).

The evidence base was dominated by mechanistic experiments. Human-virus-oriented evidence accounted for the largest subgroup, whereas plant-virus, bacteriophage, and animal-virus studies represented smaller subsets of the overall dataset.

Because some experiments assessed more than one virus category, virus-specific counts are not mutually exclusive.

Detailed extracted study characteristics are reported in Supplementary Table A2.

Time frame

Detailed extracted data are reported in Supplementary Table A2. Eligible studies were published between 1989 and 2023: 1 in the 1980s, 3 in the 1990s, 37 in the 2000s, 89 in the 2010s, and 68 from 2020 to early 2023 (10 in 2020, 20 in 2021, 27 in 2022, and 11 in early 2023) (Fig. 2).

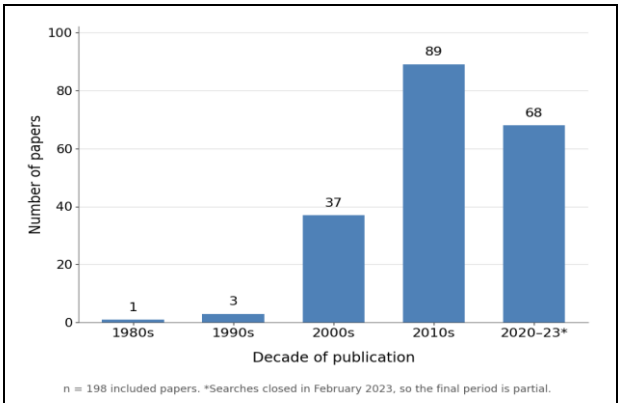


Figure 2. Time frame for all included studies.

Risk of bias assessment

Limited reporting of randomization, allocation concealment, and blinding was common in mechanistic studies. Rather than ignoring these shortcomings, the revised interpretation recognizes them as recurrent reporting limitations of the field, while still giving priority to exposure characterization and outcome assessment when assigning overall tiers.

Table 5. Overall risk-of-bias tiers (all experiments, n = 203).

Tier	Plant	Animal	Human	Mechanistic	Total
T1	9 (39.1%)	4 (33.3%)	2 (25.0%)	108 (67.5%)	123 (60.6%)
T2	14 (60.9%)	7 (58.3%)	4 (50.0%)	49 (30.6%)	74 (36.5%)
T3	0 (0.0%)	1 (8.3%)	2 (25.0%)	3 (1.9%)	6 (3.0%)
Total	23 (100%)	12 (100%)	8 (100%)	160 (100%)	203 (100%)

Percentages may not sum to 100 owing to rounding. Counts are design-specific experiments, not reports. Five reports contributed experiments to two design categories (three animal + *in vitro* and two plant + *in vitro*), so the total number of experiments (203) exceeds the number of included reports (198).

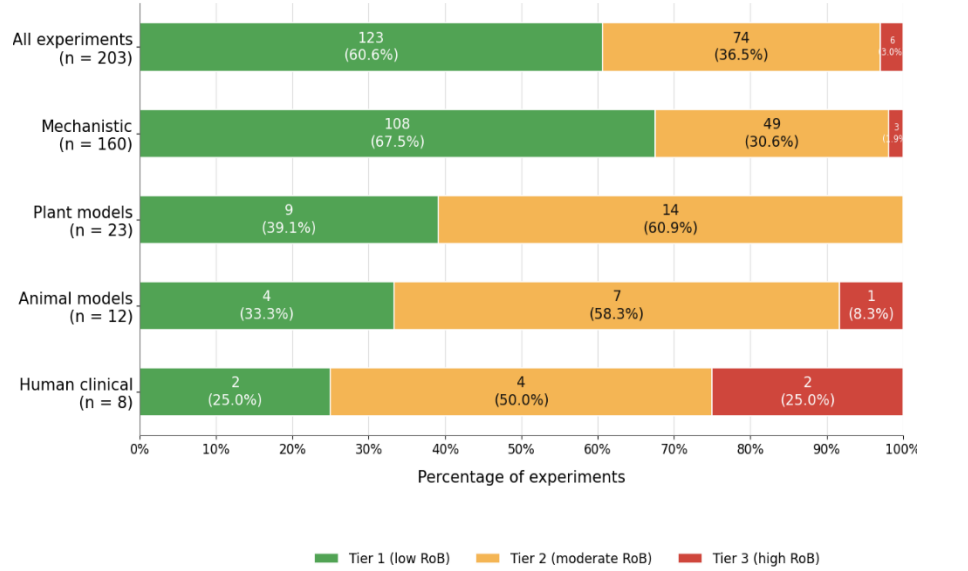


Figure 3. Overall RoB categorized by type of study.

Accordingly, absence of blinding or randomization reporting in mechanistic studies should not be interpreted as evidence of methodological robustness; instead, it was treated as a limitation of the evidence base and is considered in the interpretation of findings.

Exposure characterization emerged as the most important recurring methodological weakness.

Overall, 46 of 203 design-specific experiments (22.7%) were judged at High or Very high risk of bias for exposure characterization. The proportion of High or Very high concern in this domain was 21.3% in mechanistic assessments, 8.3% in animal studies, 26.1% in plant-model studies, and 62.5% in human clinical trials. Reporting remained inconsistent across publication years.

The complex natural composition of EOs makes accurate characterization particularly important. The main shortcomings observed were inadequate botanical identification, missing geographical provenance, absent production or batch information, and incomplete chemical analysis, either for commercial oils or for oils produced by the investigators themselves.

Across the 203 included experiments, 123 (60.6%) were Tier 1, 74 (36.5%) Tier 2, and 6 (3.0%) Tier 3 (Table 5; Fig. 3). Thus, most of the included literature fell within the Tier 1 or Tier 2 categories, but a small subset showed important methodological limitations.

Human-virus evidence base

A total of 164 design-specific experiments in the included evidence base were relevant to human viruses or closely related surrogate models and were therefore examined in detail in this article. This subgroup was dominated by mechanistic *in vitro* experiments (145), with fewer animal (11) and human clinical (8) studies (Table 6). For the references, see References A13.2 in Supplementary Materials. For a complete listing of the included studies, see Table A3 (in Supplementary Materials).

Tested viruses

Human-virus evidence was concentrated on herpes simplex viruses, SARS-CoV-2, influenza viruses, dengue virus, and coxsackieviruses. Because several experiments evaluated more than one viral target, virus-

specific test counts exceeded the number of experiments; detailed counts are reported in Table A4 (in Supplementary Materials).

Risk of bias assessment

In the human-virus-oriented subgroup, 102 of 164 experiments (62.2%) were Tier 1, 57 (34.8%) Tier 2, and 5 (3.0%) Tier 3 (Table 6; Fig. 4). Thus, the subgroup remained dominated by Tier 1 or Tier 2 evidence, but the scarcity of human studies and the predominance of mechanistic models limit direct clinical inference. For a complete listing of the RoB for single mechanistic studies, please refer to Fig. A1 (Supplementary Materials).

Efficacy and selectivity

Antiviral effect measures were heterogeneous and included EC_{50}, IC_{50}, percentage inhibition, and viral titer reduction. To make the qualitative synthesis more readable, experiments were grouped into pragmatic descriptive categories based on efficacy and SI. These categories were used only for comparative synthesis across a heterogeneous literature and should not be interpreted as validated clinical thresholds.

Efficacy alone does not adequately reflect translational relevance. Whenever available, SI was therefore considered together with antiviral effect size, because it provides an *in vitro* indication of the relationship between antiviral activity and cytotoxicity. However, SI does not establish pharmacokinetic suitability, formulation stability, mucosal tolerability, or clinical safety (Table 7). For the references, see References A13.3 in Supplementary Materials.

The complete lists of essential oils reaching the highest efficacy categories are provided in the Supplementary Materials, both without a reported selectivity index (Table A5) and with good-to-very-good selectivity (Table A6).

Table 6. Risk-of-bias tiers, human-virus subgroup (n = 164).

Tier	Animal	Human	Mechanistic	Total
T1	4 (36.4%)	2 (25.0%)	96 (66.2%)	102 (62.2%)
T2	6 (54.5%)	4 (50.0%)	47 (32.4%)	57 (34.8%)
T3	1 (9.1%)	2 (25.0%)	2 (1.4%)	5 (3.0%)
Total	11 (100%)	8 (100%)	145 (100%)	164 (100%)

Percentages may not sum to 100 owing to rounding.

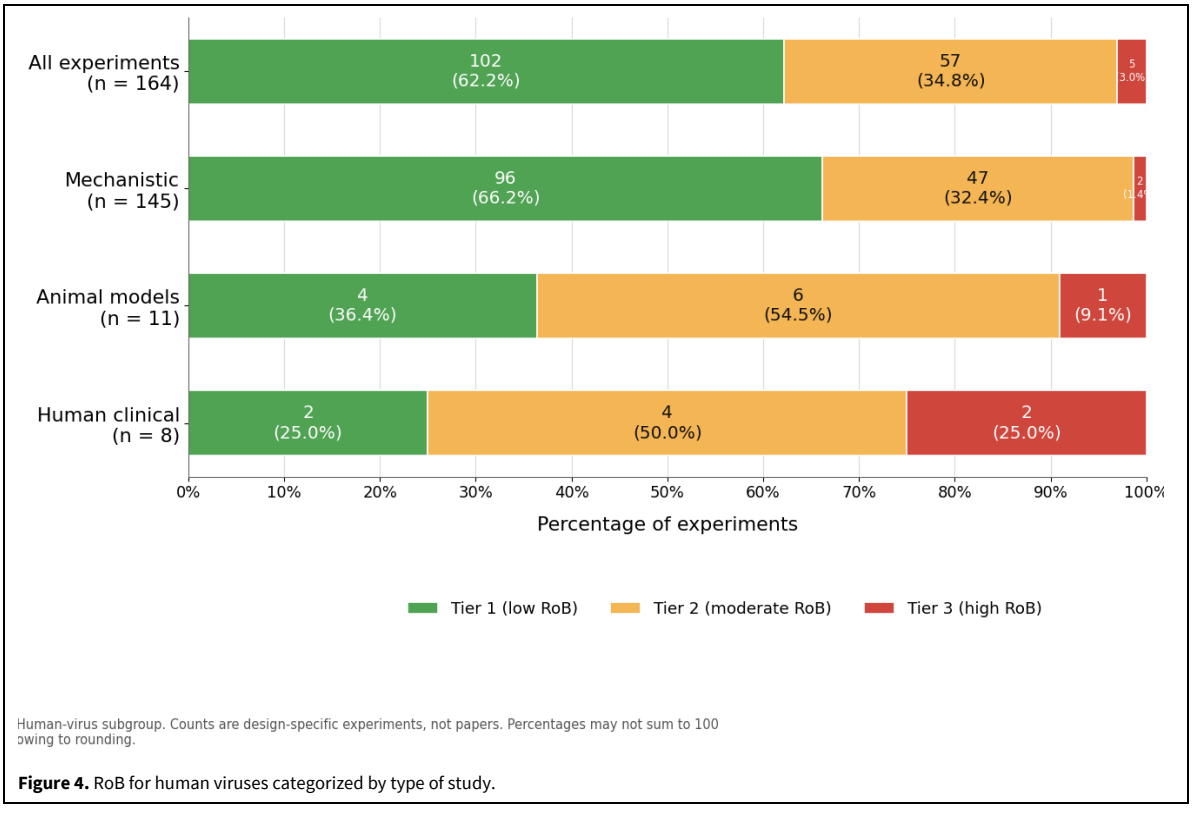


Figure 4. RoB for human viruses categorized by type of study.

Table 7. Efficacy and selectivity index scores.

Parameters	Insufficient (-)	Sufficient (+)	Good (++)	Very good (+++)
EC_{50} (µg/mL)	> 100	100-10	10-1	≤ 1
SI (CC_{50}/EC_{50})	< 4	4-10	10-100	≥100
Viral inhibition %	< 10%	10% ≤ 30%	30% ≤ 70%	≥70%
Viral inhibition log	< 1 log10	1 log10 ≤ 2 log10	2 log10 ≤ 10 log10	≥10 log10

When efficacy and SI were considered jointly, only a limited subset of EOs showed consistently favorable profiles across selected experiments. Many apparently highly active results could not be interpreted confidently because CC_{50} or SI values were not reported.

The most convincing combined profiles were observed for *Lavandula angustifolia, Matricaria recutita, Lippia alba, Salvia officinalis, Santolina insularis, Eucalyptus camaldulensis, Leptospermum scoparium,* and *Illicium verum*. These oils should be regarded as priorities for confirmatory testing rather than as clinically established candidates.

Experiments reporting very low EC_{50} values but lacking selectivity data remain hypothesis-generating only. Their apparent potency requires confirmation in assays that report EC_{50}, EC_{90}, CC_{50}, SI, and positive controls within the same experimental system.

Analysis of data relative to selected viruses

A detailed analysis of all the data according to tested viruses or tested EOs is beyond the scope of this article. However, by selecting the five viruses that received the greatest number of tests, an overview could be provided of the most effective interventions. The five most frequently tested virus groups were herpes simplex viruses, influenza viruses, SARS-related coronaviruses, dengue viruses, and coxsackieviruses.

Herpes simplex viruses (comprising herpes simplex virus type 1 and type 2)

The review encompasses 67 reports, contributing 211 HSV oil–virus assessments. Of these assessments, 151 (71.6%) were Tier 1, 60 (28.4%) Tier 2, and none were Tier 3. A single design-specific experiment could contribute more than one oil–virus assessment.

This subsection focuses on experiments with the most favorable efficacy profiles. Several highly active HSV results originated from a limited number of studies, which makes external validation important. In addition, many reports still lacked full cytotoxicity or characterization data, restricting realistic interpretation.

Amongst the results with a sufficiently good SI, notable instances include *Matricaria recutita* virucidal activity against HSV-1 (ACV-resistant) at 0.3 µg/mL with an SI of 100 (Koch et al., 2008a); *Leptospermum scoparium* J.R.Forst. & G.Forst. virucidal activity or inhibition of viral adsorption against HSV-2 strain HG52 at 0.58 µg/mL with an SI of 66.6 (Reichling et al., 2005); *Eucalyptus camaldulensis* effectiveness against HSV-1 at 0.85 µg/mL with an SI of 117 (El-Baz et al., 2015); *Santolina insularis* virucidal activity with potential intracellular mechanisms against HSV-1 F at 0.88 µg/mL with an SI of 127 (Valenti et al., 2001); *Illicium verum* effectiveness against HSV-1 ACV-sensitive at 1.0 µg/mL with an SI of 160 (Astani et al., 2011); *Hyssopus officinalis* L. virucidal action against HSV-1 ACV-resistant at 1.0 µg/mL with an SI of 75 (Schnitzler et al., 2007); and *Zingiber officinale* Roscoe virucidal activity against HSV-2 strain HG52 at 1.0 µg/mL with an SI of 40 (Koch et al., 2008b). Notably, the primary mechanisms of action for the most effective EOs appear to involve pre-adsorption, either through virucidal action or inhibition of viral adsorption, as supported by data across numerous studies.

Specifically, in 18 papers, all active mechanisms were pre-adsorption (Astani et al., 2010; Brand et al., 2016; Heidary Navid et al., 2013; Koch et al., 2008a; 2008b; Reichling et al., 2005; Romeilah et al., 2016; Saddi et al., 2007; Salah-Fatnassi et al., 2010; Samra et al., 2020; Saralieva et al., 2022; Schnitzler et al., 2007; 2008a; Schuhmacher et al., 2003; Sinico et al., 2005; Torky et al., 2021; Toujani et al., 2018; Valenti et al., 2001). Post-adsorption effects were reported less frequently and were generally supported by heterogeneous or indirect mechanistic evidence. Table 8 below presents selected studies with EC_{50} ≤ 1.00 µg/mL, with selectivity-index values reported when available. For a comprehensive list of tested EOs (Almeida et al., 2018; Astani et al., 2010; 2011; De Logu et al., 2000; El-Baz et al., 2015; Koch et al., 2008a; 2008b; Orhan et al., 2012; Reichling et al., 2005; Schnitzler et al., 2007; 2008a; Valenti et al., 2001), refer to Table A7 (Supplementary Materials).

Table 8. Herpes simplex viruses.

EO	Virus	Cell/model	Assay	EC_{50} (µg/mL)	SI	RoB	Reference
Matricaria recutita	HSV-1 ACV-resistant	RC-37 cells, Vero cells, MDCK cells	Plaque reduction assay	0.3	100	T1	Koch et al., 2008a
Cymbopogon citratus	HSV-1	Vero cells	Viral titer reduction	0.32	>4.67	T1	Almeida et al., 2018
Cymbopogon citratus	HSV-2	Vero cells	Viral titer reduction	0.33	>4.50	T1	Almeida et al., 2018
Leptospermum scoparium	HSV-2 strain HG52	RC-37 cells	Plaque reduction assay	0.58	66.6	T1	Reichling et al., 2005
Melissa officinalis	HSV-2 strain HG52	Vero cells	Plaque reduction assay	0.80	37.5	T1	Schnitzler et al., 2008a
Eucalyptus camaldulensis	HSV-1	Vero cells	Plaque reduction assay	0.85	117	T1	El-Baz et al., 2015
Santolina insularis	HSV-1 F	Vero cells	Plaque reduction assay	0.88	127	T1	Valenti et al., 2001
Leptospermum scoparium	HSV-1	RC-37 cells	Plaque reduction assay	0.96	40	T1	Reichling et al., 2005
Zingiber officinale	HSV-2 strain HG52	RC-37 cells	Plaque reduction assay	1.00	40	T1	Koch et al., 2008b
Hyssopus officinalis	HSV-1 ACV-resistant	RC-37 cells	Plaque reduction assay	1.00	75	T2	Schnitzler et al., 2007

Table 9. Influenza viruses.

EO	Virus	Cell/model	Assay	EC_{50}	SI	RoB	Reference
Melaleuca alternifolia	H11N9	MDCK cells	Plaque reduction assay	0.0005 µg/mL (100%), 0.017 µg/mL (70%)	NA	T2	Usachev et al., 2013
Eucalyptus polybractea	H11N9	MDCK cells	Viral titer reduction	0.042 µg/mL (70%)	NA	T2	Usachev et al., 2013
Lavandula angustifolia	H5N1	MDCK cells	Viral titer reduction	0.11 µg/mL	445.40	T1	Abou Baker et al., 2021
Salvia officinalis	H5N1	MDCK cells	Plaque reduction assay	0.41 µg/mL	365.80	T1	Abou Baker et al., 2021

Influenza viruses (comprising influenza virus A and B)

The review encompasses 25 reports, contributing 97 influenza oil–virus assessments. Of these assessments, 56 (57.7%) were Tier 1, 41 (42.3%) Tier 2, and none were Tier 3. A single design-specific experiment could contribute more than one oil–virus assessment.

In influenza models, the most favorable results were confined to a limited number of studies and virus strains. *Melaleuca alternifolia* and *Eucalyptus polybractea* showed marked activity against H11N9, whereas *Lavandula angustifolia* and *Salvia officinalis* showed the most favorable SI values against H5N1.

Considering SI, *Lavandula angustifolia* demonstrated notable efficacy at 0.11 µg/mL against H5N1, with an SI of 445.4, whilst *Salvia officinalis* exhibited activity at 0.41 µg/mL against H5N1, with an SI of 365.8 (Abou Baker et al., 2021). Mechanistic data are limited, presenting an ambivalent picture with instances of both pre-adsorption mechanisms, such as virucidal action of *Citrus medica*, *Limonia acidissima* (El Hawary et al., 2021), *Citrus limon* (L.) Osbeck (Bergamot group) (syn.: *Citrus bergamia*), *Eucalyptus globulus* Labill., *Melaleuca alternifolia* (Madia et al., 2022), and *Thuja plicata* Donn ex D.Don (Selvarani & James, 2013); mixed mechanisms (masking the cell surface, interference with intracellular steps, structural damage to the virion) for *Melissa officinalis* L. (Pourghanbari et al., 2016), as well as post-adsorption mechanisms like that observed with *Melaleuca alternifolia*, interfering with acidification of the intralysosomal compartment, potentially inhibiting viral uncoating (Garozzo et al., 2011).

Table 9 below presents selected studies with $EC_{50} \leq 6.00$ µg/mL, with selectivity-index values reported when available. For a comprehensive list of high-efficacy EOs (Abou Baker et al., 2021; El Hawary et al., 2021; Hassan et al., 2016; Najar et al., 2022; Pyankov et al., 2012; Usachev et al., 2013), refer to Table A8 (Supplementary Materials).

SARS-CoV-2

SARS-CoV-2 evidence was based almost entirely on mechanistic assays. The most promising results were reported for thyme oil, *Illicium verum*, *Agathis robusta*, *Syzygium aromaticum*, *Citrus limon*, and *Pelargonium graveolens*, but direct comparison across studies is limited by differences in assay design, units, and completeness of cytotoxicity reporting.

Table 10. SARS-CoV-2.

EO	Virus	Cell/model	Assay	EC_{50}	SI	RoB	Reference
Syzygium aromaticum	SARS-CoV-2 delta pseudovirus	HeLa cells	Cell-based pseudoviral entry assay	0.09 µg/mL	4.44	T1	Torres Neto et al., 2022
Citrus limon	SARS-CoV-2 delta pseudovirus	HeLa cells	Cell-based pseudoviral entry assay	0.15 µg/mL	8.67	T1	Torres Neto et al., 2022
Pelargonium graveolens	SARS-CoV-2 delta pseudovirus	HeLa cells	Cell-based pseudoviral entry assay	0.2 µg/mL	8.5	T1	Torres Neto et al., 2022
Agathis robusta	SARS-CoV-2	Vero cells	Plaque infectivity assay	0.324 µL/mL (ca. 0.29 µg/mL)	17.5	T1	Mohamed et al., 2022
Illicium verum	SARS-CoV-2 delta pseudovirus	HeLa cells	Cell-based pseudoviral entry assay	0.5 µg/mL	60.00	T1	Torres Neto et al., 2022

ACE2-target assays were reported for EOs including *Mentha* spp. (Demirci et al., 2021), *Salvia* spp. (Karadağ et al., 2022), *Eucalyptus* spp. (Ak Sakallı et al., 2022), *Salvia rosmarinus* Spenn. (syn.: *Rosmarinus officinalis*) (Demirci et al., 2022), and *Senecio massaicus* (Maire) Maire (Kebbi et al., 2021). These cell-free results do not establish inhibition of viral adsorption or infection and were excluded from antiviral mechanism frequencies and efficacy rankings. Some virus-containing studies reported post-adsorption effects involving viral main protease (Mpro), RNA-dependent RNA polymerase (Mohamed et al., 2022), or chymotrypsin-like protease 3CLpro (Bahgat et al., 2023).

Table 10 below shows a selection of the studies, according to effect size (only studies with $EC_{50} \leq 1.00$ µg/mL) and selectivity index (only studies with SI ≥ 4). For a comprehensive list of high-efficacy EOs (Mohamed et al., 2022; Sakalar & Ertürk, 2023; Torres Neto et al., 2022), refer to Table A9 (Supplementary Materials).

Dengue viruses (comprising dengue virus types 1, 2 and 3)

Dengue (DENV) evidence remained limited to a small number of studies. *Lippia alba* and *Aloysia citrodora* showed the most favorable reported profiles, but the evidence was narrow and depended heavily on a limited number of research groups.

Although data on possible mechanisms are limited, there are indications of events during cell adsorption (Silva-Trujillo et al., 2022) or post-adsorption, such as observed with *Buddleja cordobensis*, likely acting on glycoproteins inserted in the envelope (Duschatzky et al., 2005), or *Saccocalyx saturejoides* Coss. & Durieu targeting DENV polymerase (Laouer et al., 2006). For a comprehensive list of high-efficacy EOs (Ocazionez et al., 2010), refer to Table A10 (Supplementary Materials).

Coxsackieviruses (comprising coxsackievirus B3 and B4)

Coxsackievirus evidence was modest and methodologically heterogeneous. *Osmunda regalis* showed the most favorable SI, whereas *Haplophyllum tuberculatum* showed activity that appeared to depend strongly on timing of exposure.

Among these studies, three EOs exhibited high efficacy: *Haplophyllum tuberculatum* (Forssk.) A.Juss., *Bontia daphnoides* L., and *Osmunda regalis* L. *Osmunda regalis* EO showed activity against CoxB4, with an EC_{50} of 2.24 µg/mL and an SI of 789.84 (Bouazzi et al., 2018).

Haplophyllum tuberculatum EO, obtained from both leaves and stems, demonstrated activity against CoxB3 and CoxB4, with the highest efficacy observed when applied before cell inoculation, with EC_{50} values of 1.7 µg/mL and 1.3 µg/mL, and SIs of 2.8 and 3.6, respectively. Additionally, the EO obtained solely from leaves showed activity against CoxB4, with an EC_{50} of 1.5 µg/mL and an SI of 2.8, while that obtained solely from stems showed activity against both CoxB3 and CoxB4, with EC_{50} values of 3.0 µg/mL and 2.5 µg/mL, and SIs of 1.9 and 2.2, respectively (Hamdi et al., 2021). The EO of *Bontia daphnoides* fruit exhibited activity against CoxB4, causing 61.75% inhibition of the virus at a dose of 1.56 µg/mL, although no SI was provided (Thabet et al., 2022).

Information on the mechanisms of action against coxsackieviruses is limited. Hamdi et al. (2021) suggested that *Haplophyllum tuberculatum* EO may act by masking binding structures on the cell surface, making adsorption difficult, especially given its increased efficacy in cell pretreatment. Similarly, Samra et al. (2020) proposed that the EO of *Cyperus rotundus* L. might act as a direct virucidal agent or by masking binding structures.

For a comprehensive list of high-efficacy EOs, refer to Table A11 (Supplementary Materials).

Table 11. Antiviral evidence for essential oils by viral category (assessments).

Virus group	Assessments	Distinct source-reported EO labels	RoB Tier 1/2/3	High-efficacy	Most active EOs (by *in vitro* SI)
HSV-1	178	157	125 / 53 / 0	47	*Illicium verum* (160.0); *Santolina insularis* (127.3); *Eucalyptus camaldulensis* (117.0)
HSV-2	33	31	26 / 7 / 0	15	*Thymus bovei* (98.6); *Leptospermum scoparium* (66.6)
Influenza A/B	97	72	56 / 41 / 0	27	*Lavandula angustifolia* (445.4); *Salvia officinalis* (365.8); *Melaleuca alternifolia* (41.0)
Dengue (DENV)	61	43	61 / 0 / 0	12	*Lippia alba* (349.0); *Lippia citriodora* (30.3); *Turnera diffusa* (7.7)
Coxsackievirus	45	42	18 / 27 / 0	8	*Osmunda regalis L.* (789.8); *Dysphania ambrosioides* (74.3)
HIV	26	22	25 / 1 / 0	4	All SI ≤7; no strong selectivity. *Ridolfia segetum* (6.4); *Rosmarinus officinalis* (3.6); *Hornstedtia bella* (3.0)
Hepatitis A (HAV)	15	15	7 / 8 / 0	2	*Chrysanthemum morifolium* (38.2); *Chrysanthemum indicum* (37.0); *Acacia nilotica fruit* (3.8)
Adenovirus	11	11	3 / 8 / 0	1	*Melaleuca alternifolia* (1.0)
SARS-CoV (classic, 2003)	8	7	8 / 0 / 0	0	*Satureja thymbra* (4.5); *Laurus nobilis* (4.2); *Thuja orientalis* (3.8)
SARS-CoV-2 (live + pseudovirus)	63	59	43 / 11 / 9	10	*Illicium verum* (60.0); *Syzygium aromaticum* (26.3); *Agathis robusta* (17.5)
Other human coronaviruses (HCoV-229E, HCoV-OC43)	4	4	3 / 1 / 0	0	*Cestrum diurnum* (37.1)
Animal coronavirus surrogates (FCoV, IBV, BCoV)	6	6	2 / 4 / 0	2	—
Non-coronavirus surrogates (MNV, feline calicivirus, bacteriophage)	28	22	21 / 7 / 0	0	*Rosmarinus officinalis* (6.5); *Thymus vulgaris* (6.2); *Lippia graveolens* (5.8)
SARS-CoV-2 ACE2 / 5-LOX enzyme (no virus)	40	40	40 / 0 / 0	17	—

Qualitative synthesis

In the absence of a defensible quantitative synthesis, the evidence is summarized qualitatively by virus family and by essential-oil (EO) type. Efficacy is reported using the descriptive categories defined in the Methods ("+++" to "−"); "high efficacy" denotes a "++" or "+++" rating. Selectivity index (SI) values are *in vitro* ratios of cytotoxicity to antiviral activity and are not clinical parameters. Counts are oil–virus assessments, not unique papers or design-specific experiments, and virus-specific assessments are not mutually exclusive. The distinct-label column in Table 11 reports case-insensitive, whitespace-normalized source-reported EO labels; these counts are descriptive and do not define reports or design-specific experiments.

Evidence by virus family

SARS-CoV-2 counts (63) exclude the 40 ACE2 / 5-lipoxygenase enzyme-inhibition assays, which contain no virus and are reported separately in Table A4; they are shown on the final line for reference only and are not part of the antiviral evidence. SI values are illustrative *in vitro* maxima and are not comparable across viruses because of differing assays, cell lines, and exposure metrics; potency and efficacy do not always coincide (e.g., *Eucalyptus globulus* shows SI ≈ 9.6 against SARS-CoV-2, while no single efficacy score was derivable from the reported values). SARS-CoV-2 high-efficacy signals derived mainly from spike-pseudotyped assays (8 of 10); 2 occurred in live-virus assessments. The high SI values in the other-human-coronavirus row come from a single study of *Cestrum diurnum* against HCoV-229E (Bahgat et al., 2023).

Evidence by essential-oil type

Across the assessed literature, a small number of oils recur across multiple virus families or account for most of the high-efficacy signals. Breadth of testing and potency do not always coincide: several broadly tested oils show no high-efficacy assessment (Table 12).

Table 12. Most frequently studied or broadest-spectrum essential oils.

Essential oil	Assessments	Virus groups	High-efficacy	Profile
Melaleuca alternifolia (tea tree)	18	11	6	Coronavirus, coxsackie, HSV-1, HSV-2, influenza, phage, plant, polio, ECHO-9, HADV-2, MCV
Cymbopogon citratus (lemongrass)	17	7	8	Coronavirus, HIV, HSV-1, HSV-2, plant, RSV, RRV
Thymus vulgaris	14	9	8	Coronavirus, HIV, HSV-1, HSV-2, NDV, plant, CyHV-3, FCV, HADV
Rosmarinus officinalis	13	8	3	Coronavirus, HIV, HSV-1, HSV-2, plant, AIBV, FCV, PIV-3
Foeniculum vulgare Mill.	12	11	3	Coxsackie, HIV, HSV-1, plant, polio, RSV, VV, YFV, BVDV, VSV, WNV
Hornstedtia bella	13	10	0	Coxsackie, HIV, HSV-1, polio, RSV, VV, YFV, BVDV, EV-A71, REO-1
Eucalyptus globulus Labill.	12	12	0	Coxsackie, DENV, HIV, HSV-1, phage, polio, RSV, VV, YFV, BVDV, VSV, WNV
Helichrysum italicum	11	11	0	Coxsackie, DENV, HIV, HSV-1, polio, RSV, VV, YFV, BVDV, VSV, WNV
Salvia officinalis L.	10	10	1	Coronavirus, coxsackie, HIV, HSV-1, phage, RSV, YFV, BVDV, VSV, WNV
Atalantia sessiflora	10	10	0	Coxsackie, HIV, HSV-1, polio, RSV, VV, YFV, BVDV, VSV, WNV
Limnocitrus littoralis	10	10	0	Coxsackie, HIV, HSV-1, polio, RSV, VV, YFV, BVDV, VSV, REO-1
Paramignya trimera	10	10	0	Coxsackie, HIV, HSV-1, polio, RSV, VV, YFV, BVDV, VSV, REO-1
Laurus nobilis L.	9	9	0	DENV, HIV, polio, RSV, VV, YFV, BVDV, VSV, WNV
Pistacia lentiscus L.	9	9	0	Coxsackie, HIV, HSV-1, polio, RSV, VV, YFV, BVDV, WNV

"Virus groups" counts distinct viral categories in which the oil was tested. Breadth of testing does not imply higher potency.

Narrative synthesis

Herpes simplex viruses

Anti-herpes activity forms the largest and most consistent body of evidence (HSV-1: 178 assessments across 157 source-reported EO labels; HSV-2: 33 across 31 labels). It is dominated by low-risk-of-bias assessments (Tier 1 in 125/178 HSV-1 and 26/33 HSV-2), and it contains many of the stronger antiviral signals. *Santolina insularis, Cymbopogon citratus*, peppermint, basil, and *Artemisia arborescens* recur among the more active oils, with *in vitro* SI values in the tens to low hundreds.

Dengue and influenza

For dengue (61 assessments) and influenza A/B (97 assessments), the evidence includes isolated strong signals: *Lippia alba* and *L. citriodora* for dengue and *Melaleuca alternifolia* for influenza. Most dengue assessments are low-risk-of-bias, whereas influenza includes a larger Tier 2 fraction.

Coronaviruses

Coronavirus evidence is heterogeneous and must be read with care, because the records span several distinct viruses and one non-viral assay type. For SARS-CoV-2, there are 63 oil–virus assessments using live or spike-pseudotyped virus, of which 43 are low-risk-of-bias (Tier 1). Ten of these assessments were rated as high efficacy ("++"/"+++"). Among the 44 live-virus assessments, 10 were rated "−", 13 had no efficacy data, 19 yielded no derivable score, and 2 were rated "++"/"+++". A further 40 records are ACE2 or 5-lipoxygenase enzyme-inhibition assays; these contain no virus, measure target-related activity rather than infection, and are reported separately (Table A4) rather than as antiviral evidence. The remaining coronavirus data are limited: eight SARS-CoV assessments (Loizzo et al., 2008b), all Tier 1 and none reaching the high-efficacy category; four assessments of the common-cold human coronaviruses HCoV-229E and HCoV-OC43 (Bahgat et al., 2023; Romeo et al., 2022); and a small set of animal-coronavirus surrogates (feline coronavirus, avian infectious bronchitis virus, and bovine coronavirus). No controlled human evidence is specific to any coronavirus.

Other human viruses

Coxsackievirus, HIV, hepatitis A, and adenovirus are each supported by 45, 26, 15 and 11 assessments, respectively, and show few or no high-efficacy results; HIV assessments in particular report all SI ≤7. These groups should be described as exploratory.

Across oils

A few oils are tested broadly across virus families (tea tree, *Thymus vulgaris*, and several Vietnamese oils), but breadth does not track with potency: some broadly tested oils yield no high-efficacy assessment, while the strongest signals come from oils studied against only one or two viruses (e.g., *Santolina insularis* for HSV, *Lippia* spp. for dengue).

Table 13. Sensitivity analysis of mechanistic outcome-level assessments with an explicit overall efficacy score.

Tier	High efficacy (++/+++)	Total assessments	High efficacy (%)
Tier 1 (low RoB)	125	512	24.4%
Tier 2	69	208	33.2%
Tier 3 (high RoB)	0	8	0.0%
Total	194	728	26.6%

This pattern reinforces that current evidence identifies candidate oil–virus pairs rather than broad-spectrum antivirals, and that apparent potency must be interpreted against the heterogeneity of assays, cell lines, and exposure metrics and against the predominantly *in vitro*, mechanistic nature of the evidence.

Sensitivity analysis

Among 728 mechanistic outcome-level assessments with an explicit Overall efficacy score, high efficacy ("++" or "+++") was recorded in 125/512 Tier 1 assessments (24.4%), 69/208 Tier 2 assessments (33.2%), and 0/8 Tier 3 assessments (0.0%), for 194/728 assessments overall (26.6%). Restricting the analysis to Tier 1 assessments yielded high efficacy in 125/512 assessments (24.4%), compared with 194/728 (26.6%) in the unrestricted dataset. These descriptive results do not show a simple monotonic relationship between RoB tier and high efficacy, and the small Tier 3 denominator limits inference (Table 13).

Because the admission of fixed combinations was a post hoc amendment, the synthesis was repeated with all three affected studies excluded. This reduced the evidence base to 195 reports and 200 design-specific experiments, with five human clinical trials, one of which was Tier 1. The overall risk-of-bias distribution changed only slightly: 122 (61.0%) Tier 1, 72 (36.0%) Tier 2, and 6 (3.0%) Tier 3, compared with 123 (60.6%), 74 (36.5%), and 6 (3.0%) in the main analysis. The broad descriptive interpretation was not materially altered, but the small human subgroup precludes strong claims of robustness.

Mechanisms of action

Mechanistic information was available for a substantial proportion of experiments. Across these reports, pre-adsorption effects, including direct virucidal activity and inhibition of viral adsorption, were the most frequently described mechanisms. Post-adsorption effects and mixed-stage activity were reported less often.

However, mechanistic interpretation should remain cautious because time-of-addition designs, solvent controls, volatility control, and orthogonal confirmation assays were inconsistently reported. Some apparent virucidal effects may reflect nonspecific membrane or assay interference rather than specific antiviral action.

When a stage of action could be assigned, pre-adsorption activity was more frequent than intracellular or immediate post-adsorption effects. Detailed mechanism-specific data are provided in Table A12. For the references, see References A13.4 in Supplementary Materials.

DISCUSSION

Unlike previous narrative overviews focused on selected viruses or isolated compounds, the present review applied explicit eligibility criteria and RoB assessment to whole-EO studies across multiple virus groups. Overall, the evidence base showed a mixed but not uniformly weak quality profile: 60.6% of experiments were classified as Tier 1, 36.5% as Tier 2, and 3.0% as Tier 3.

A central issue concerned exposure characterization. Reporting of randomization, allocation concealment, and blinding was also sparse in mechanistic studies, and this should be interpreted as a limitation of the field rather than as evidence that these domains are irrelevant. For this reason, the revised interpretation emphasizes exposure characterization and outcome assessment as the most informative domains while acknowledging the broader reporting weaknesses of the included literature.

Accurate EO identification is particularly important because chemical composition varies according to species, chemotype, plant part, geography, harvest conditions, extraction method, oxidation state, and storage. Inadequate botanical authentication or missing GC-MS or GC-FID characterization can therefore generate exposure misclassification and seriously limit reproducibility.

These standardization problems are not merely editorial. They affect internal validity, comparability across studies, and the feasibility of future clinical translation. Future EO antiviral studies should report voucher specimens, plant part, harvest provenance, extraction procedure, batch identification, storage conditions, and full chemical characterization whenever possible, as listed in the "Transparent Reporting for Essential oil and Aroma Therapeutic Studies" (TREATS)

checklist (Reven et al., 2024), which provides a valuable framework for assessing reporting quality in EO studies.

In the human-virus-oriented subgroup, 62.2% of experiments were Tier 1, 34.8% Tier 2, and 3.0% Tier 3. This profile still indicates a predominance of Tier 1 or Tier 2 evidence, but it also underscores that clinically relevant inference is constrained by the overwhelming dominance of mechanistic studies over animal and human data. Specifically, 145 of 164 experiments (88.4%) were mechanistic, whereas only eight were human clinical studies and only two of those eight were Tier 1. Thus, the proportion of Tier 1 experiments should not be interpreted as stronger clinical certainty.

The outcome-level sensitivity analysis also showed no monotonic quality gradient: high-efficacy ratings occurred in 24.4% of Tier 1 and 33.2% of Tier 2 assessments, whereas the Tier 3 estimate was based on only 8 assessments. This pattern suggests that the overall signal is not explained solely by studies in the highest RoB tier, but it does not validate efficacy because the comparison is descriptive and vulnerable to outcome and design heterogeneity.

Another important limitation is incomplete toxicological reporting. Many experiments reporting apparently strong antiviral effects did not provide CC_{50} or SI values, making it impossible to judge whether antiviral activity occurred within a plausible safety window.

Several EOs nevertheless emerged as comparatively stronger candidates within the available *in vitro* literature, especially *Lavandula angustifolia*, *Matricaria recutita*, *Lippia alba*, *Salvia officinalis*, *Santolina insularis*, *Eucalyptus camaldulensis*, and *Leptospermum scoparium*. These findings justify confirmatory research, but they do not imply optimized pharmacokinetics or established clinical utility.

EOs showing high efficacy but unknown SI should be interpreted even more cautiously. They are best considered priorities for re-testing in standardized systems rather than reliable leads for translation.

Across individual virus groups, the most consistent signals were observed for enveloped viruses, especially HSV, influenza viruses, and SARS-CoV-2. This pattern is biologically plausible because EO lipophilicity may favor interactions with viral envelopes and early entry steps, but the current data remain too heterogeneous for strong cross-virus ranking.

For SARS-CoV-2 in particular, the literature is recent and methodologically diverse. Differences in cell systems, pseudovirus versus live-virus assays, exposure timing, and expression units complicate direct comparison and argue against overinterpretation of apparently very low EC_{50} values. In interpreting the coronavirus data, two distinctions are material. First, ACE2 and 5-lipoxygenase inhibition assays measure target-related activity rather than inhibition of viral infection; they are mechanistically informative but are not antiviral tests, and are therefore reported separately from experiments involving live or pseudotyped virus and excluded from the count of antiviral experiments against SARS-CoV-2. Second, the coronavirus records span several distinct entities that should not be pooled: SARS-CoV-2 itself, the 2003 SARS-CoV-1, the common-cold coronaviruses HCoV-229E and HCoV-OC43, animal coronaviruses used as surrogates (feline coronavirus, avian infectious bronchitis virus and bovine coronavirus), and non-coronaviral surrogates, including feline calicivirus and bacteriophages. Table A4 disaggregates the data on these lines.

Read this way, the direct SARS-CoV-2 evidence is more limited than the overall number of coronavirus-indexed records implies. It rests on a modest set of live-virus and spike-pseudotyped assays, with the larger share of coronavirus records deriving from enzyme-inhibition or surrogate systems whose relationship to infection *in vivo* is indirect. This pattern is consistent with the review's wider finding that the antiviral evidence for essential oils remains predominantly mechanistic and preclinical.

The same caution applies to HSV, DENV, and coxsackievirus studies. A few experiments reported excellent SI values, but independent replication, batch-level chemical characterization, standardized positive controls, and physiologically relevant models remain limited.

Mechanistically, pre-adsorption activity predominated across the reviewed literature. However, this category likely encompasses both specific antiviral effects and less specific phenomena, such as membrane perturbation or assay interference, especially when solvent effects, volatility, and concentration stability were not rigorously controlled.

A smaller subset of studies suggested mixed pre- and post-adsorption activity, indicating that some EOs or EO components may act at multiple stages of the viral cycle. Where post-entry effects were claimed, direct intracellular confirmation was often limited.

Taken together, the mechanistic evidence supports experimental antiviral potential, not therapeutic efficacy.

Limitations and future reflections

This review has important limitations. It included whole EOs and excluded isolated EO constituents, fractions, and blends, apart from the three qualifying fixed combinations of whole EOs evaluated in clinical

studies. Grey literature and unpublished studies were not actively sought. In addition, no formal meta-analysis or GRADE assessment was performed because the evidence base was dominated by heterogeneous mechanistic designs with non-comparable outcomes. The search strategy was not formally peer-reviewed by an information specialist; however, it was reviewed by the authors with the most experience in systematic reviews (D.D. and M.A.) until consensus was reached. Backward and forward citation searching was not performed, which may have reduced retrieval of additional eligible reports. The search was last updated in February 2023; consequently, studies published subsequently fall outside the evidence window of the present synthesis and may modify the current evidence base.

Translational barriers also remain substantial. EO volatility, hydrophobicity, formulation instability, batch variability, mucosal tolerability, inhalation safety, uncertain bioavailability, and regulatory standardization all complicate extrapolation from cell-culture assays to clinical use.

The eligibility criterion for EO mixtures was amended after screening had begun. Although the amendment was applied by a stated rule rather than case by case, and although a sensitivity analysis excluding the affected studies did not materially alter the broad descriptive interpretation, post hoc modification of eligibility criteria is a recognised source of selection bias and should be weighed when interpreting the clinical stratum.

Future work should therefore prioritize authenticated and chemically characterized oils, standardized dose-response designs, mechanism-specific assays, physiologically relevant models, and, only thereafter, well-designed animal and clinical studies.

A language restriction was applied: two reports published in Persian were excluded because no translation could be obtained. Language-restricted eligibility is a recognised source of selection bias; the potential effect of these exclusions cannot be quantified from the available data.

CONCLUSION

Whole EOs demonstrated promising antiviral and virucidal activity in experimental studies, particularly against enveloped viruses such as HSV, influenza viruses, and SARS-CoV-2. The strongest signals were observed in mechanistic *in vitro* assays and were most frequently associated with pre-adsorption effects, including direct virucidal activity and inhibition of viral adsorption.

However, the current evidence base remains limited by methodological heterogeneity, incomplete EO characterization, inconsistent cytotoxicity and SI reporting, and the scarcity of animal and human studies.

Accordingly, the present findings should be interpreted as evidence of experimental antiviral potential rather than established therapeutic applicability. Standardized chemical characterization, validated dose-response designs, appropriate positive controls, physiologically relevant models, and controlled *in vivo* and clinical studies are needed before therapeutic recommendations can be proposed.

On current evidence, selected EOs may be considered candidate adjunctive agents for further investigation, not clinical alternatives to established antiviral therapies.

CONFLICT OF INTEREST

The research was conducted entirely free from any involvement in commercial, financial, or personal matters that might be perceived as a possible conflict of interest or that could have influenced the outcomes or interpretation of the study.

ACKNOWLEDGMENTS

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

GENERATIVE ARTIFICIAL INTELLIGENCE (AI)

The authors used ChatGPT (OpenAI) to improve the language and readability of the manuscript. The authors reviewed and edited the content and take full responsibility for its accuracy. No images were generated or manipulated using AI tools.

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AUTHOR CONTRIBUTION:

Contribution	Valussi M	Pesoni D	Frennet P	Antonelli M	Donelli D
Concepts or ideas	x			x	x
Design	x			x	x
Definition of intellectual content					
Literature search	x	x	x		
Data acquisition	x	x	x		
Data analysis	x	x		x	x
Manuscript preparation	x	x	x		
Manuscript editing	x		x		x
Manuscript review	x	x	x	x	x

Citation Format: Valussi M, Pesoni D, Frennet P, Antonelli M, Donelli D (2026) Antiviral and virucidal properties of essential oils on human viruses: A systematic review. J Pharm Pharmacogn Res 14(5): 2688. https://doi.org/10.56499/jppres_14.5.2688

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Supplementary data

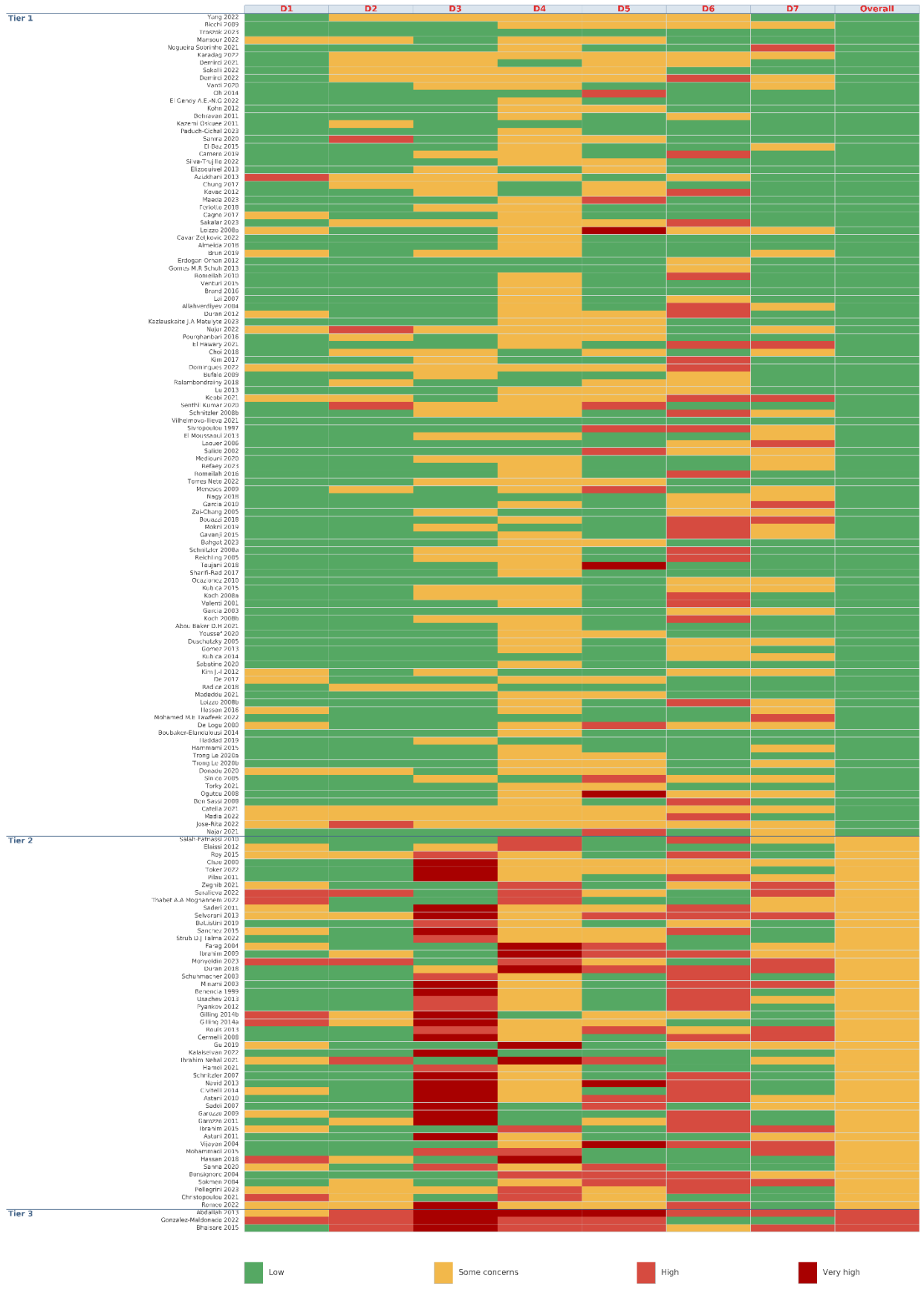


Figure A1. Domain-level risk-of-bias matrix for the 160 mechanistic experiments.

Table A1. OHAT Risk-of-Bias tool. Bias domains and risk-of-bias questions applied during the systematic review

Bias Domains	Risk-of-Bias Question	1 *In vitro*	2 Experimental animal	3 Human controlled exposure	4 Cohort	5 Case-control	6 Cross-sectional	7 Case series
Selection Bias	1. Was administered dose or exposure level adequately randomized?	—	X	X	—	—	—	—
	2. Was allocation to study groups adequately concealed?	—	X	X	—	—	—	—
	3. Did selection of study participants result in the appropriate comparison groups?	—	—	—	X	X	X	—
Confounding Bias	4. Did study design or analysis account for important confounding and modifying variables?	—	—	—	X	X	X	X
Performance Bias	5. Were experimental conditions identical across study groups?	X	X	—	—	—	—	—
	6. Were research personnel blinded to the study group during the study?	—	X	X	—	—	—	—
Attrition/Exclusion Bias	7. Were outcome data complete without attrition or exclusion from analysis?	X	X	X	X	X	X	—
Detection Bias	8. Can we be confident in the exposure characterization?	X	X	X	X	X	X	X
	9. Can we be confident in the outcome assessment (including blinding of assessors)?	X	X	X	X	X	X	X
Selective Reporting Bias	10. Were all measured outcomes reported?	X	X	X	X	X	X	X
Other Sources of Bias	11. Were there no other potential threats to internal validity	X	X	X	X	X	X	X

Legend. X = applicable; — = not applicable. The seven columns identify the study designs covered by the OHAT applicability matrix: 1, *in vitro* exposure; 2, experimental animal; 3, human controlled exposure; 4, cohort; 5, case-control; 6, cross-sectional; and 7, case series.

Mapping of the consolidated mechanistic matrix to OHAT questions: D1 = question 5; D2 = question 7; D3 = question 8 (key domain); D4 = question 9 (key domain); D5 = question 10; D6 and D7 partition question 11 into funding/conflicts of interest and other threats to internal validity. Questions 1–4 and 6 were not operationalized in the consolidated *in vitro* matrix because they were not applicable to the extracted mechanistic designs.

For mechanistic, plant-model, and human experiments, Tier 1 required both key domains (D3 and D4) and a majority of other applicable domains to be Low or Some concerns; Tier 3 required both key domains and a majority of other applicable domains to be High or Very high; all other combinations were Tier 2. For animal studies, Tier 1 required at least four Low domains and no High or Very high domain, whereas Tier 3 required at least three High or Very high domains; all other combinations were Tier 2. The design-specific criteria were applied directly to the domain-level assessments to derive the reported Rule Tier.

Table A2. Characteristics of included studies.

See Table A2 here

Table A3. Essential oils tested against human viruses and surrogates.

See Table A2 here

Table A4. Human-virus, surrogate, and target-assay categories.

Virus ID	Number of assessment rows (not papers)
HSV (herpes simplex virus)	211
SARS-CoV-2-related subtotal (live/spike-pseudovirus + target-only)	103 (63 + 40; non-additive subtotal)
SARS-CoV-2 (live + spike-pseudovirus)	63
SARS-CoV-1 (classic, 2003)	8
Other human coronaviruses (HCoV-229E, HCoV-OC43)	4
Animal coronavirus surrogates (FCoV-II, IBV, BCoV)	6
Non-coronavirus surrogates (feline calicivirus, murine norovirus, bacteriophages)	28
SARS-CoV-2 target-only ACE2/5-LOX assays (no virus)	40
IV (influenza virus)	97
DENV (dengue virus)	61
Cox B (coxsackie virus B)	45
HIV (human immunodeficiency virus)	26
JUNV (Junin virus)	23
PV-1s (poliovirus)	21
YFV (yellow fever virus)	21
MNV (murine norovirus) (surrogate)	16
PIV (parainfluenza virus)	15
HEV (hepatitis A virus)	15
hRSV (human respiratory syncytial virus)	14
NDV (Newcastle disease virus)	14 (animal and human)
VV (vaccinia virus)	15
FCV (feline calicivirus) (surrogate)	10
WNV (West Nile virus)	11
hADV (human adenovirus)	11
SARS-CoV-2 spike-pseudovirus subset (within live + pseudovirus row)	19
Reo (reovirus)	4
MHV (mouse hepatitis virus) (surrogate)	3
RRV (Ross River virus)	3
HCV (hepatitis C virus)	2
EVA71 (enterovirus A71)	3
MCV (molluscum contagiosum virus)	2
RhinoV (rhinovirus)	2
hRV (human rotavirus)	2
ECHO (enteric cytopathic human orphan virus)	4
JEV (Japanese encephalitis virus)	2
ZIKV (Zika virus)	2
MV (measles virus)	1
IBV (infectious bronchitis virus)	4
HEV (hepatitis E virus)	1
FCoV (feline enteric coronavirus) surrogate	2
ORFV (ORF virus)	1 (animal and human virus)

Counts are assessment rows, not unique experiments or papers; one experiment may contribute more than one assessment. Broad categories and explicitly labelled subset rows overlap and therefore must not be added together. Target-only ACE2/5-LOX assays contain no virus and are reported separately from antiviral evidence.

Table A5. Best efficacy with no SI. EOs showing relevant efficacy against human viruses but for which no SI was reported in the source studies

EO	Virus	EC_{50}/IC_{50}/efficacy measure	RoB	Reference
Citrus medica leaves	IV-A/H5N1	0.25 µg/µL = 90% inhibition; 0.5 µg/µL = 95% inhibition	Tier 1	El Hawary et al., 2021
Limonia acidissima leaves	IV-A/H5N1	0.25 µg/µL = 88% inhibition; 0.5 µg/µL = 92% inhibition	Tier 1	El Hawary et al., 2021
Origanum vulgaris	HSV-1	0.2- 0.4 µg/mL	Tier 1	Orhan et al., 2012
Mentha piperita	HSV-1	0.2- 0.4 µg/mL	Tier 1	Orhan et al., 2012
Mentha spicata	HSV-1	0.2- 0.4 µg/mL	Tier 1	Orhan et al., 2012
Origanum munitiflorum	HSV-1	0.2- 0.4 µg/mL	Tier 1	Orhan et al., 2012
Satureja cuneifolia	HSV-1	0.2- 0.4 µg/mL	Tier 1	Orhan et al., 2012
Foeniculum vulgare (fully mature)	HSV-1	0.2- 0.4 µg/mL	Tier 1	Orhan et al., 2012
Mentha piperita	PIV-3	0.4 - 0.8 µg/mL	Tier 1	Orhan et al., 2012
Mentha spicata	PIV-3	0.4 - 0.8 µg/mL	Tier 1	Orhan et al., 2012
Origanum munitiflorum	PIV-3	0.4 - 0.8 µg/mL	Tier 1	Orhan et al., 2012
Origanum vulgaris	PIV-3	0.4 - 0.8 µg/mL	Tier 1	Orhan et al., 2012
Satureja cuneifolia	PIV-3	0.4 - 0.8 µg/mL	Tier 1	Orhan et al., 2012
Anethum graveolens	PIV-3	0.4 - 0.8 µg/mL	Tier 1	Orhan et al., 2012
Foeniculum vulgare (fully mature)	PIV-3	0.4 - 0.8 µg/mL	Tier 1	Orhan et al., 2012
Citrus medica fruits	IV-A/H5N1	0.504 µg/µL = 50% inhibition; 0.252 µg/µL = 20.8% inhibition	Tier 1	El Hawary et al., 2021
Foeniculum vulgare (flowering)	HSV-1	0.8 µg/mL	Tier 1	Orhan et al., 2012
Lavandula officinalis	HSV-1	0.8 µg/mL	Tier 1	Orhan et al., 2012
Ocimum basilicum (green variety)	HSV-1	0.8 µg/mL	Tier 1	Orhan et al., 2012
Ocimum basilicum (purple variety)	HSV-1	0.8 µg/mL	Tier 1	Orhan et al., 2012
Origanum majorana	HSV-1	0.8 µg/mL	Tier 1	Orhan et al., 2012
Origanum onites	HSV-1	0.8 µg/mL	Tier 1	Orhan et al., 2012
Rosmarinus officinalis	HSV-1	0.8 µg/mL	Tier 1	Orhan et al., 2012
Salvia officinalis	HSV-1	0.8 µg/mL	Tier 1	Orhan et al., 2012
Thyme (*Thymus* spp.?)	SARS-CoV-2	0.0013 µL/mL = 99.99% inhibition	Tier 1	Sakalar & Ertürk, 2023

Table A6. Best efficacy with good to very good SI. EOs with high efficacy and a reported selectivity index considered good to very good.

Essential oil	Virus ID	EC_{50}/IC_{50}/efficacy measure	SI	RoB	Reference
Lavandula angustifolia	IV-A/H5N1	0.11 µg/mL	445.4	Tier 1	Abou Baker et al., 2021
Calamintha nepeta	HSV-1	0.12–0.44 [0.063–0.46] µg/mL (range); 0.22 [0.2] µg/mL (average)	2.50–34.57	Tier 1	Sabatino et al., 2020
Matricaria recutita	HSV-1 Angelotti (ACV-resistant) + 2 clinical isolates	0.3 µg/mL	100	Tier 1	Koch et al., 2008a
Agathis robusta	SARS-CoV-2	0.324 µL/mL	17.5	Tier 2	Mohamed et al., 2022
Lippia alba	DENV-2	0.4 µg/mL	349	Tier 1	Ocazionez et al., 2010
Salvia officinalis	IV-A/H5N1	0.41 µg/mL	365.8	Tier 1	Abou Baker et al., 2021
Illicium verum	SARS-CoV-2 Delta	0.5 µg/mL	60.00	Tier 1	Torres Neto et al., 2022
Leptospermum scoparium	HSV-2 strain HG52	0.58 µg/mL	66.6	Tier 1	Reichling et al., 2005
Santolina insularis	HSV-2 strain G	0.7 µg/mL; IC_{80}: 1.25 µg/mL	160	Tier 1	De Logu et al., 2000
Melissa officinalis	HSV-2 strain HG52	0.8 µg/mL	37.5	Tier 1	Schnitzler et al., 2008a

Table A6. Best efficacy with good to very good SI. EOs with high efficacy and a reported selectivity index considered good to very good (continued...)

Essential oil	Virus ID	EC_{50}/IC_{50}/efficacy measure	SI	RoB	Reference
Eucalyptus camaldulensis	HSV-1	0.85 µg/mL	117	Tier 1	El-Baz et al., 2015
Santolina insularis (FEO)	HSV-1 F	0.88 µg/mL	127.3	Tier 1	Valenti et al., 2001
Santolina insularis	HSV-1 F	0.88 µg/mL; IC_{80}: 1.87 µg/mL	127	Tier 1	De Logu et al., 2000
Leptospermum scoparium	HSV-1 ACV- sensitive KOS	0.96 µg/mL	40	Tier 1	Reichling et al., 2005
Artemisia arborescens	HSV-1 F	1.14-2.4 µg/mL	55	Tier 2	Saddi et al., 2007
Zanthoxylum myriacanthum var. *pubescens*	IV-A H1N1, PR/8-R292K	1.87 µg/mL	85.13	Tier 1	Yang et al., 2022
Illicium verum? (star anise)	HSV-1 ACV- sensitive KOS	1.0 µg/mL	160	Tier 2	Astani et al., 2011
Zingiber officinale	HSV-2 strain HG52	1.0 µg/mL	40	Tier 1	Koch et al., 2008b
Hyssopus officinalis	HSV-1 ACV-resistant	1.0 µg/mL	75	Tier 2	Schnitzler et al., 2007
Matricaria recutita	HSV-2 strain HG52	1.5 µg/mL	20	Tier 1	Koch et al., 2008b
Foeniculum vulgare var. *azoricum* leaves	HCV NS5B polymerase	1.8 µg/mL	34.66	Tier 2	Ibrahim & Moussa, 2021
Lippia citriodora	DENV-1	1.9 µg/mL	30.3	Tier 1	Ocazionez et al., 2010
Zingiber officinale	HSV-1 ACV-resistant	2.0 µg/mL	20	Tier 2	Schnitzler et al., 2007
Thymus bovei	HSV-2	2.13 µg/mL	> 98.59	Tier 2	Hassan et al., 2018
Santolina insularis (SUV)	HSV-1 F	2.2 µg/mL	50.9	Tier 1	Valenti et al., 2001
Osmunda regalis L.	Cox-B4	2.244 µg/mL	789.84	Tier 1	Bouazzi et al., 2018
Lippia citriodora	DENV-3	2.6 µg/mL	22.15	Tier 1	Ocazionez et al., 2010
Lippia citriodora	DENV-2	2.9 µg/mL	19.86	Tier 1	Ocazionez et al., 2010
Chrysanthemum indicum	VSV	3.14 µg/mL	40	Tier 1	Youssef et al., 2020
Thymus capitata	BHV-1 Cooper-1	3.36 µg/mL	14.49	Tier 1	Boubaker et al., 2014
Chrysanthemum indicum	HAV	3.38 µg/mL	37	Tier 1	Youssef et al., 2020
Chrysanthemum indicum	HSV-1	3.51 µg/mL	35.8	Tier 1	Youssef et al., 2020
Chrysanthemum morifolium	VSV	3.69 µg/mL	39.4	Tier 1	Youssef et al., 2020
Lippia origanoides	YFV	3.7 µg/mL	26.4	Tier 1	Nogueira Sobrinho et al., 2021
Origanum vulgare	YFV	3.7 µg/mL	26.5	Tier 1	Nogueira Sobrinho et al., 2021
Lippia alba	YFV	3.7 µg/mL MIC (50%)	22.9	Tier 1	Nogueira Sobrinho et al., 2021
Chrysanthemum morifolium	HSV-1	3.73 µg/mL	38.96	Tier 1	Youssef et al., 2020
Chrysanthemum morifolium	HAV	3.80 µg/mL	38.25	Tier 1	Youssef et al., 2020
Foeniculum vulgare var. *azoricum* bulb	HCV NS5B polymerase	3.94 µg/mL	33.68	Tier 2	Ibrahim & Moussa, 2021
Acacia nilotica fruit	HSV-2	3.99 µg/mL	16	Tier 1	El Gendy et al., 2022
Artemisia arborescens	HSV-2 strain G	4.1 µg/mL	32.2	Tier 2	Saddi et al., 2007
Santolina insularis (MLV)	HSV-1 F	4.6 µg/mL	24.3	Tier 1	Valenti et al., 2001
Lippia alba	YFV 17 DD	4.9 µg/mL	28.46	Tier 1	Ocazionez et al., 2010
Mentha suaveolens	HSV-1	5.1 µg/mL	67	Tier 2	Civitelli et al., 2014
Thymus vulgaris leaves	CyHV-3	5.826 µg/mL	14.49-18.47	Tier 1	Troszok & Roszko, 2023
Melaleuca alternifolia	IV-A/H1N1	6 µg/mL	41.66	Tier 2	Garozzo et al., 2009
Hyssopus officinalis	HSV-2 strain HG52	6 µg/mL	12.5	Tier 1	Koch et al., 2008b
Cymbopogon citratus (FEO)	HSV-2	6.69 µg/mL	17.32	Tier 1	Almeida et al., 2018
Fortunella margarita, fruit	IV-A/H5N1	6.77 µg/mL	35.38	Tier 2	Ibrahim & Moussa, 2021
Thymus vulgaris	HSV-2 strain HG52	7.0 µg/mL	10	Tier 1	Koch et al., 2008b
Mentha piperita	HSV-2 strain HG52	8.0 µg/mL	17.5	Tier 2	Schuhmacher et al., 2003

Table A7. EOs active on herpes simplex viruses (HSV) with high efficacy. Subset of EOs with high efficacy against herpes simplex viruses.

Essential oil	EC_{50}/IC_{50}/efficacy measure	Virus ID	RoB	SI	Reference
Anethum graveolens	0.025-0.8 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Foeniculum vulgare (fully mature)	0.2-0.4 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Mentha piperita	0.2-0.4 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Mentha spicata	0.2-0.4 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Origanum minutiflorum	0.2-0.4 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Origanum vulgare	0.2-0.4 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Satureja cuneifolia	0.2-0.4 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Matricaria recutita	0.3 µg/mL	HSV-1 ACV-resistant	Tier 1	100	Koch et al., 2008a
Cymbopogon citratus (HNPVO)	0.32 µg/mL	HSV-1	Tier 1	>4.67	Almeida et al., 2018
Cymbopogon citratus (HNPVO)	0.33 µg/mL	HSV-2	Tier 1	> 4.50	Almeida et al., 2018
Leptospermum scoparium	0.58 µg/mL	HSV-2 strain HG52	Tier 1	66.6	Reichling et al., 2005
Cymbopogon citratus (HVO)	0.71 µg/mL	HSV-2	Tier 1	>2.31	Almeida et al., 2018
Santolina insularis	0.7 µg/mL	HSV-2 strain G	Tier 1	160	De Logu et al., 2000
Melissa officinalis	0.8 µg/mL	HSV-2 strain HG52	Tier 1	37.5	Schnitzler et al., 2008a
Foeniculum vulgare (flowering)	0.8 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Lavandula officinalis	0.8 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Ocimum basilicum (green variety)	0.8 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Ocimum basilicum (purple variety)	0.8 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Origanum majorana	0.8 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Origanum onites	0.8 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Rosmarinus officinalis	0.8 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Salvia officinalis	0.8 µg/mL	HSV-1	Tier 1	NA	Orhan et al., 2012
Eucalyptus camaldulensis	0.85 µg/mL	HSV-1	Tier 1	117	El-Baz et al., 2015
Santolina insularis (FEO)	0.88 µg/mL	HSV-1 F	Tier 1	127.3	Valenti et al., 2001
Santolina insularis	0.88 µg/mL; IC_{80}: 1.87 µg/mL	HSV-1 F	Tier 1	127	De Logu et al., 2000
Leptospermum scoparium	0.96 µg/mL	HSV-1 ACV-sensitive	Tier 1	40	Reichling et al., 2005
Illicium verum? (star anise)	1.0 µg/mL	HSV-1 ACV-sensitive	Tier 2	160	Astani et al., 2011
Hyssopus officinalis	1.0 µg/mL	HSV-1 ACV-resistant	Tier 2	75	Schnitzler et al., 2007
Zingiber officinale	1.0 µg/mL	HSV-2 strain HG52	Tier 1	40	Koch et al., 2008b
Cymbopogon citratus (NPVO)	1.32 µg/mL	HSV-1	Tier 1	>9.83	Almeida et al., 2018
Cymbopogon citratus (NPVO)	1.34 µg/mL	HSV-2	Tier 1	>9.67	Almeida et al., 2018
Matricaria recutita	1.5 µg/mL	HSV-2	Tier 1	20	Koch et al., 2008b
Melaleuca alternifolia	2.0 µg/mL	HSV-1	Tier 1	60	Astani et al., 2010

Table A8. EOs active on IV with high efficacy.

Essential oil	EC_{50}/IC_{50}/efficacy measure	Virus ID	RoB	SI	Reference
Origanum acutidens	> 0.08 µg/mL	IV-A/H3N2 (Aichi/2/68)	Tier 2	< 1	Sökmen et al., 2004
Melaleuca alternifolia	0.0005 µL/mL; 96.66% inhibition	IV-A/H11N9 strain NWS/G70C	Tier 2	NA	Pyankov et al., 2012
Melaleuca alternifolia	0.017 µg/mL; 64.6% inhibition	IV-A/H11N9	Tier 2	NA	Usachev et al., 2013
Eucalyptus polybractea	0.042 µg/mL; 68.8% inhibition	IV-A/H11N9	Tier 2	NA	Usachev et al., 2013
Lavandula angustifolia	0.11 µg/mL	IV-A/H5N1	Tier 1	445.4	Abou Baker et al., 2021
Nepeta deflersiana	0.156 µg/mL	IV-A/H5N1	Tier 2	2	Hassan et al., 2016
Salvia officinalis	0.41 µg/mL	IV-A/H5N1	Tier 1	365.8	Abou Baker et al., 2021
Limonia acidissima leaves	0.5 µg/µL = 92% inhibition 0.25 µg/µL = 88% inhibition	IV-A/H5N1	Tier 1	NA	El Hawary et al., 2021
Citrus medica leaves	0.5 µg/µL = 95% inhibition 0.25 µg/µL = 90% inhibition	IV-A/H5N1	Tier 1	NA	El Hawary et al., 2021
Citrus medica fruits	0.504 µg/µL	IV-A/H5N1	Tier 1	NA	El Hawary et al., 2021
Helichrysum araxinum	1.0 µg/mL > 70% inhibition	IV-A H1N1	Tier 1	NA	Najar et al., 2022
Helichrysum foetidum	1.0 µg/mL > 70% inhibition	IV-A H1N1	Tier 1	NA	Najar et al., 2022
Helichrysum italicum	1.0 µg/mL > 70% inhibition	IV-A H1N1	Tier 1	NA	Najar et al., 2022

Table A9. EOs active on SARS-CoV-2 with high efficacy.

Essential oil	EC_{50}/IC_{50}/efficacy measure	Virus ID	RoB	SI	Reference
Thyme oil	0.0013 µL/mL; 99.99% inhibition	SARS-CoV-2	Tier 1	NA	Sakalar & Ertürk, 2023
Syzygium aromaticum	0.09 µg/mL	SARS-CoV-2 Delta PsV	Tier 1	4.44	Torres Neto et al., 2022
Cymbopogon citratus	0.11 µg/mL	SARS-CoV-2 Delta PsV	Tier 1	2.00	Torres Neto et al., 2022
Citrus limon	0.15 µg/mL	SARS-CoV-2 Delta PsV	Tier 1	8.67	Torres Neto et al., 2022
Pelargonium graveolens	0.20 µg/mL	SARS-CoV-2 Delta PsV	Tier 1	8.50	Torres Neto et al., 2022
Agathis robusta	0.324 µL/mL	SARS-CoV-2	Tier 2	17.5	Mohamed et al., 2022
Origanum vulgare	0.36 µg/mL	SARS-CoV-2 Delta PsV	Tier 1	1.00	Torres Neto et al., 2022
Illicium verum	0.5 µg/mL	SARS-CoV-2 Delta PsV	Tier 1	60.00	Torres Neto et al., 2022
Matricaria recutita	1.1 µg/mL	SARS-CoV-2 Delta PsV	Tier 1	1.09	Torres Neto et al., 2022
Thymus vulgaris	3.1 µg/mL	SARS-CoV-2 Delta PsV	Tier 1	0.67	Torres Neto et al., 2022

Table A10. EOs active on DENV with high efficacy.

Essential oil	EC_{50}/IC_{50}/efficacy measure	Virus ID	RoB	SI	Reference
Lippia alba	0.4 µg/mL	DENV-2	Tier 1	349	Ocazionez et al., 2010
Lippia citriodora	1.9 µg/mL	DENV-1	Tier 1	30.3	Ocazionez et al., 2010
Lippia citriodora	2.6 µg/mL	DENV-3	Tier 1	22.15	Ocazionez et al., 2010
Lippia citriodora	2.9 µg/mL	DENV-2	Tier 1	19.86	Ocazionez et al., 2010
Lippia alba	10.1 µg/mL	DENV-1	Tier 1	13.8	Ocazionez et al., 2010

Table A11. EOs active on Cox with high efficacy.

Essential oil	Virus ID	EC_{50}/IC_{50}/efficacy measure	SI	RoB	Reference
Haplophyllum tuberculatum leaves + stems	CoxB4	1.3 µg/mL (before) 7.7 µg/mL (together) 7.5 µg/mL (after)	3.6 (before) 0.6 (together) 0.6 (after)	Tier 2	Hamdi et al., 2021
Haplophyllum tuberculatum leaves	CoxB4	1.5 µg/mL (before) 7.0 µg/mL (together) 7.5 µg/mL (after)	2.8 (before) 0.6 (together) 0.6 (after)	Tier 2	Hamdi et al., 2021
Bontia daphnoides fruits	CoxB4	1.56 µg/mL (inhibition = 61.75%)	NA	Tier 2	Thabet et al., 2022
Haplophyllum tuberculatum leaves + stems	CoxB3	1.7 µg/mL (before) 3.4 µg/mL (together) 3.6 µg/mL (after)	2.8 (before) 1.4 (together) 1.3 (after)	Tier 2	Hamdi et al., 2021
Osmunda regalis	Cox-B4	2.24 µg/mL	789.84	Tier 1	Bouazzi et al., 2018
Haplophyllum tuberculatum stems	CoxB3	3.0 µg/mL (before) 5.7 µg/mL (together) 9.0 µg/mL (after)	1.9 (before) 1.0 (together) 0.6 (after)	Tier 2	Hamdi et al., 2021
Haplophyllum tuberculatum stems	CoxB4	2.5 µg/mL (before) 18.1 µg/mL (together) 24.7 µg/mL (after)	2.2 (before) 0.3 (together) 0.2 (after)	Tier 2	Hamdi et al., 2021

Table A12. Mechanisms of action.

EO	Summary	Reference
Pre-adsorption mechanisms		
Angelica archangelica	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Strub et al., 2022
Artemisia dubia	**V.** Direct virucidal activity	Kim, 2012
Artemisia vulgaris	**V.** Direct virucidal activity. Possible active molecules: carvacrol (44%), thymol (15%), γ-terpinene (10%)	Meneses et al., 2009
Callistemon viminalis	**V. (AP/PA).** Significant virucidal effect, minor effect in masking viral compounds, slight effect on post-infection stages	Romeilah et al., 2016
Carlina acanthifolia radix	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Saralieva et al., 2022
Carum copticum	**V.** Direct virucidal activity	Kazemi Oskuee et al., 2011
Cinnamomum zeylanicum	**V.** Direct virucidal activity	Refaey et al., 2023
Citrus bergamia	**V.** Direct virucidal activity	Madia et al., 2022
Citrus medica fruits	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	El Hawary et al., 2021
Citrus medica leaves	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	El Hawary et al., 2021
Cupressus sempervirens	**V.** Direct virucidal activity	Ibrahim et al., 2009
Eucalyptus citriodora	**Target-only ACE2 assay; this cell-free result does not establish inhibition of viral adsorption or infection and is excluded from antiviral mechanism frequencies**	Ak Sakallı et al., 2022
Eucalyptus globulus	**V.** Direct virucidal activity	Madia et al., 2022
Eucalyptus globulus.	**Target-only ACE2 assay; this cell-free result does not establish inhibition of viral adsorption or infection and is excluded from antiviral mechanism frequencies**	Ak Sakallı et al., 2022
Eucalyptus polybractea	**V.** Direct virucidal activity	Pyankov et al., 2012
Haplophyllum tuberculatum	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Hamdi et al., 2021
Hyssopus officinale	**V.** Significant virucidal effects. Minimal or no effects for cell pretreatment, during adsorption, and after adsorption	Schnitzler et al., 2007
Hyssopus officinalis	**V.** Virucidal action, limited effect on virus replication in cells and on cell-to-cell spread	Koch et al., 2008b
Illicium verum	**V.** Virucidal action, limited effect on virus replication in cells and on cell-to-cell spread	Koch et al., 2008b
Illicium verum	**V.** Direct virucidal activity	Koch et al., 2008a
Illicium verum	**V.** Direct virucidal activity	Madia et al., 2022

Table A12. Mechanisms of action (continued…)

EO	Summary	Reference
Limonia acidissima leaves	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	El Hawary et al., 2021
Lippia alba	**V.** Direct virucidal activity	Ocazionez et al., 2010
Lippia alba	**V.** Direct virucidal activity	Nogueira Sobrinho et al., 2021
Lippia alba	**V.** Direct virucidal activity. Possible active molecules: carvacrol (44%), thymol (15%), γ-terpinene (10%)	Nogueira Sobrinho et al., 2021
Lippia alba CT citrals	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Silva-Trujillo et al., 2022
Lippia citriodora	**V.** Direct virucidal activity	Ocazionez et al., 2010
Lippia origanoides CT carvacrol	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Silva-Trujillo et al., 2022
Lippia origanoides CT phellandrene.	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Silva-Trujillo et al., 2022
Lippia origanoides CT thymol	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Silva-Trujillo et al., 2022
Lippia origanoides CT thymol-carvacrol	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Silva-Trujillo et al., 2022
Lippia origanoides,	**V.** Direct virucidal activity. Possible active molecules: carvacrol (44%), thymol (15%), γ-terpinene (10%)	Meneses et al., 2009
Lippia multiflora	**V.** Direct virucidal activity	Bouagnon et al., 2022
Matricaria recutita	**V.** Virucidal action, limited effect on virus replication in cells and on cell-to-cell spread	Koch et al., 2008b
Matricaria recutita	**V.** Direct virucidal activity	Koch et al., 2008a
Melaleuca alternifolia	**V.** Direct virucidal activity	Pyankov et al., 2012
Melaleuca alternifolia	**V.** Direct virucidal activity	Madia et al., 2022
Mentha arvensis	**Target-only ACE2 assay; this cell-free result does not establish inhibition of viral adsorption or infection and is excluded from antiviral mechanism frequencies**	Demirci et al., 2021
Mentha citrata	**Target-only ACE2 assay; this cell-free result does not establish inhibition of viral adsorption or infection and is excluded from antiviral mechanism frequencies**	Demirci et al., 2021
Mentha spicata	**Target-only ACE2 assay; this cell-free result does not establish inhibition of viral adsorption or infection and is excluded from antiviral mechanism frequencies**	Demirci et al., 2021
Myristica fragrans seed	**V.** Direct virucidal activity	Kazlauskaite et al., 2023
Ocimum basilicum CT linalool	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Silva-Trujillo et al., 2022
Origanum vulgare	**V.** Direct virucidal activity	Sánchez & Aznar, 2015
Origanum vulgare	**V.** Direct virucidal activity. Possible active molecules: carvacrol (44%), thymol (15%), γ-terpinene (10%)	Meneses et al., 2009
Phlomis aurea	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Torky et al., 2021
Picea glehnii	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Maeda et al., 2023
Pinus mugo	**V.** Direct virucidal activity	Koch et al., 2008a
Piper aduncum CT piperitone	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Silva-Trujillo et al., 2022
Rosmarinus officinalis L.	**Target-only ACE2 assay; this cell-free result does not establish inhibition of viral adsorption or infection and is excluded from antiviral mechanism frequencies**	Demirci et al., 2022
Salvia fructicosa	**V.** Possible active molecules: thujone, camphor	Sivropoulou et al., 1997
Salvia officinalis L.	**Target-only ACE2 assay; this cell-free result does not establish inhibition of viral adsorption or infection and is excluded from antiviral mechanism frequencies**	Karadağ et al., 2022
Salvia sclarea L.	**Target-only ACE2 assay; this cell-free result does not establish inhibition of viral adsorption or infection and is excluded from antiviral mechanism frequencies**	Karadağ et al., 2022
Salvia triloba L.	**Target-only ACE2 assay; this cell-free result does not establish inhibition of viral adsorption or infection and is excluded from antiviral mechanism frequencies**	Karadağ et al., 2022
Santalum album	**V.** Virucidal action, limited effect on virus replication in cells and on cell-to-cell spread	Koch et al., 2008b
Santalum album	**V.** Significant virucidal effects. Minimal or no effects for cell pretreatment, during adsorption, and after adsorption	Schnitzler et al., 2007
Santalum spicatum	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Strub et al., 2022

Table A12. Mechanisms of action (continued…)

EO	Summary	Reference
Schinus molle	**V. (AP/PA).** Significant virucidal effect, minor effect in masking viral compounds, slight effect on post-infection stages	Romeilah et al., 2016
Senecio massaicus	**Target-only ACE2 assay; this cell-free result does not establish inhibition of viral adsorption or infection and is excluded from antiviral mechanism frequencies**	Kebbi et al., 2021
Teucrium pseudochamaepitys	**V.** Direct virucidal activity	Hammami et al., 2015
Thyme oil	**V.** Direct virucidal activity	Sakalar & Ertürk, 2023
Thymus vulgaris	**V.** Virucidal action, limited effect on virus replication in cells and on cell-to-cell spread	Koch et al., 2008b
Thymus vulgaris	**V.** Significant virucidal effects. Minimal or no effects for cell pretreatment, during adsorption, and after adsorption	Schnitzler et al., 2007
Thymus capitatus	**V.** Direct virucidal activity	Toujani et al., 2018
Thymus vulgaris leaves	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Troszok & Roszko, 2023
Turnera diffusa	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Silva-Trujillo et al., 2022
Verronia curassavica	**AP.** Inhibiting viral attachment or adsorption by effects on superficial structures	Silva-Trujillo et al., 2022
Zataria multiflora	**V.** Direct virucidal activity	Sánchez & Aznar, 2015
Zingiber officinale	**V.** Virucidal action, limited effect on virus replication in cells and on cell-to-cell spread	Koch et al., 2008b
Zingiber officinale	**V.** Direct virucidal activity	Camero et al., 2019
Zingiber officinale	**V.** Significant virucidal effects. Minimal or no effects for cell pretreatment, during adsorption, and after adsorption	Schnitzler et al., 2007
Zingiber officinalis	**V.** Direct virucidal activity	Bouagnon et al., 2022
Pre- and post-adsorption, or ambiguous, mechanisms		
Aloysia triphylla	**V/AP.** Virucidal or inhibiting viral attachment or adsorption	Duschatzky et al., 2005
Anethum graveolens	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Aniba rosaeodora	**V / I.** Suggested action during viral replication	Kohn et al., 2012
Artemisia arborescens	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Sinico et al., 2005
Artemisia arborescens	**V/CC.** Direct virucidal activity and inhibition of cell-to-cell transmission	Saddi et al., 2007
Artemisia mendozana	**V/AP.** Virucidal or inhibiting viral attachment or adsorption	Duschatzky et al., 2005
Artemisia princeps var. orientalis	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption. Possible active molecule: alpha-thujone	Chung, 2017
Ayapana triplinervis	**AP or PA.** The assays showed viral entry (internalisation phase) inhibition. Possible active molecule: thymohydroquinone dimethyl ether	Taglienti et al., 2023
Baccharis dracunculifolia	**AP or PA, plus I.** Best activity in the test during virus adsorption, where it might block partially the viral entry, or lead to RNA degradation before the virus entry, but also some activity on the steps of viral cycle replication, or RNA degradation after their release to the supernatant	Búfalo et al., 2009
Buddleja cordobensis	**V/AP./PA** Virucidal or inhibiting viral attachment or adsorption, but also post-adsorption events	Duschatzky et al., 2005
Citrus limon	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Battistini et al., 2019
Citrus paradisi	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Battistini et al., 2019
Citrus sinensis	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Battistini et al., 2019
Cymbopogon citratus (lemongrass)	**V/AP and I.** EO very effective virucidal but also in inhibiting viral attachment or adsorption to host cells. It also suppresses genome replication in host cells	Kim et al., 2017
Cymbopogon citratus (lemongrass)	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Gilling et al., 2014b
Cyperus rotundus	**V/AP.** Virucidal or inhibiting viral attachment or adsorption	Samra et al., 2020
Eucalyptus astringens	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Elaissi et al., 2012
Eucalyptus bicostata	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Elaissi et al., 2012
Eucalyptus cinerea	**V/AP./PA** Virucidal or inhibiting viral attachment or adsorption, but also post-adsorption events	Elaissi et al., 2012
Eucalyptus globulus	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Cermelli et al., 2008

Table A12. Mechanisms of action (continued…)

EO	Summary	Reference
Eucalyptus globulus? (eucalyptus)	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Astani et al., 2010
Eucalyptus maidenii	**V/AP./PA** Virucidal or inhibiting viral attachment or adsorption, but also post-adsorption events	Elaissi et al., 2012
Eucalyptus polybractea	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Usachev et al., 2013
Foeniculum vulgare	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Gaillardia megapotamica	**V/AP.** Virucidal or inhibiting viral attachment or adsorption	Duschatzky et al., 2005
Heterothalamus alienus	**V/AP./PA** Virucidal or inhibiting viral attachment or adsorption, but also post-adsorption event	Duschatzky et al., 2005
Hyptis mutabilis	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Brand et al., 2016
Illicium verum (star anise)	**V/AP.** Acts either directly as a virucide or by blocking viral adsorption, during and after viral infections. Thymol likely active	Astani et al., 2011
Jungia polita	**V/AP.** Virucidal or inhibiting viral attachment or adsorption	Duschatzky et al., 2005
Lallemantia royleana	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Sharifi-Rad et al., 2017
Lavandula officinalis	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Lepechinia salvifolia	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Brand et al., 2016
Lepechinia vulcanicola	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Brand et al., 2016
Leptospermum scoparium	**V/AP.** Significant virucidal effect with interference with virion envelopes or masking viral compounds necessary for adsorption. Possible active molecules: flavesone and leptospermone	Reichling et al., 2005
Lippia alba	**V/AP/PA.** EO more active before adsorption than after	Gómez et al., 2013
Lippia citriodora	**V/AP/PA.** EO more active before adsorption than after	Gómez et al., 2013
Lippia graveolens	**V/PA.** Direct virucidal activity and effects after adsorption	Kubiča et al., 2015
Lippia turbinata	**V/AP.** Virucidal or inhibiting viral attachment or adsorption	Duschatzky et al., 2005
Maytenus ilicifolia	**V / I.** Suggested action during viral replication	Kohn et al., 2012
Melaleuca alternifolia	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Brun et al., 2019
Melaleuca alternifolia	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Usachev et al., 2013
Melaleuca alternifolia? (tea tree)	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Astani et al., 2010
Melissa officinalis	**V/AP.** Significant virucidal effects, some adsorption effects	Schnitzler et al., 2008a
Melissa officinalis	**V/AP/I.** It appears active in different steps of replication cycle: 1. interfere with the cell surface proteins such as masking the cell surface. 2. interfere with intracellular step of the virus replication. 3. antiviral activity before adsorption, via structural damage to the virion	Pourghanbari et al., 2016
Mentha piperita	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Mentha spicata	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Mentha suaveolens	**V/AP.** Slightly virucidal or inhibiting viral attachment or adsorption. Piperitone oxide appears to act intracellularly	Civitelli et al., 2014
Mentha suaveolens	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	El Moussaoui et al., 2013
Mentha x piperita	**V/AP.** Significant virucidal effects, slight efficacy in adsorption	Schuhmacher et al., 2003
Minthostachys mollis	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Brand et al., 2016
Ocimum basilicum	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Ocimum basilicum	**V/PA.** Direct virucidal activity and effects after adsorption	Kubiča et al., 2014
Ocimum campechianum	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Brand et al., 2016
Ocotea quixos	**V to I.** Apparently active on all steps from virion to intracellular mechanisms, but antiviral activity more important	Radice et al., 2019
Olbas mix	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Heidary Navid et al., 2013
Origanum elongatum	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	El Moussaoui et al., 2013
Origanum majorana	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Origanum minutiflorum	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012

Table A12. Mechanisms of action (continued…)

EO	Summary	Reference
Origanum onites	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Origanum vulgaris	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Origanum vulgaris? (oregano)	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Gilling et al., 2014a
Pectis odorata	**V/AP.** Virucidal or inhibiting viral attachment or adsorption	Duschatzky et al., 2005
Pimenta dioica? (allspice)	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Gilling et al., 2014b
Piper aduncum	**V to I.** Apparently active on all steps from virion to intracellular mechanisms, but antiviral activity more important	Radice et al., 2019
Pulicaria vulgaris	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Sharifi-Rad et al., 2017
Rosmarinus officinalis	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Rosmarinus officinalis	**V/PA.** Direct virucidal activity and effects after adsorption	Kubiča et al., 2015
Rosmarinus officinalis CT 1,8-cineole	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Battistini et al., 2019
Salvia officinalis	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Santolina insularis	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	De Logu et al., 2000
Santolina insularis	**V/I.** Virucidal action before adsorption, with possible involvement of intracellular mechanism	Valenti et al., 2001
Satureja cuneifolia	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Orhan et al., 2012
Sinapis arvensis	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Sharifi-Rad et al., 2017
Thuja plicata	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Selvarani & James, 2013
Thymus capitata	**V/AP/PA.** Virucidal or blocking viral adsorption effect, also during and after viral infections. Thymol likely active	Boubaker et al., 2014
Thymus capitatus	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Salah-Fatnassi et al., 2010
Thymus capitatus	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	El Moussaoui et al., 2013
Thymus transcaspicus	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption. Possible active molecules: thymol (56.4%), γ-terpinene (7.7%), carvacrol (7.6%) and p-cymene (6.3%)	Behravan et al., 2011
Thymus vulgaris	**V/PA.** Direct virucidal activity and effects after adsorption	Kubiča et al., 2015
Thymus vulgaris (thyme)	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Astani et al., 2010
Trachyspermum ammi	**V/AP.** Direct virucidal activity and effects on structures preventing adsorption	Roy et al., 2015
Zataria multiflora	**AP or PA.** The pretreatment assay showed no activity; the test during virus adsorption (EO applied simultaneously with virus) showed a marked inhibitory effect; the post-infection test showed no considerable effect. Possible active molecules: carvacrol (30.6%) and thymol (29.5%)	Mohammadi et al., 2015
Post-adsorption mechanisms		
Acacia nilotica bark	**PA/I.** Effects after adsorption, possibly inhibits viral replication by action on 3C proteases	El Gendy et al., 2022
Agathis robusta	**AP/PA/I.** Inhibiting viral attachment or adsorption, effects after adsorption **and** activity on viral main protease (Mpro) and RNA-dependent RNA polymerase (RdRp)	Mohamed et al., 2022
Backhousia citriodora	**CC.** Inhibition of cell-to-cell transmission	Burke et al., 2004
Bulnesia sarmientoi	**AP/PA/I.** Inhibiting viral attachment or adsorption, effects after adsorption **and** activity on proteases	Strub et al., 2022
Callitris intratropica	**AP/PA/I.** Inhibiting viral attachment or adsorption, effects after adsorption **and** activity on proteases	Strub et al., 2022
Cedrus libani	**PA/I.** The EO inhibits a step of viral replicative cycle after virus attachment and entry	Loizzo et al., 2008a
Cestrum diurnum	**PA/I.** Effects after adsorption, possibly inhibits viral replication by action on 3C proteases	Bahgat et al., 2023
Chrysopogon zizanioides	**AP/PA/I.** Inhibiting viral attachment or adsorption, effects after adsorption **and** activity on proteases	Strub et al., 2022
Citrus reticulata Blanco	**AP/PA/I.** Inhibiting viral attachment or adsorption, effects after adsorption **and** activity on proteases	Strub et al., 2022

Table A12. Mechanisms of action (continued…)

EO	Summary	Reference
Cymbopogon citratus	**PA.** Effects after adsorption	Kim et al., 2017
Cymbopogon citratus	**PA/I.** Effects after adsorption, possibly inhibit a step of viral replicative cycle (Potential)	Feriotto et al., 2018
Fokienia hodginsii	**AP/PA/I.** Inhibiting viral attachment or adsorption, effects after adsorption **and** activity on proteases	Strub et al., 2022
Glechon marifolia	**PA.** Effects after adsorption	Venturi et al., 2015
Glechon spathulata	**PA.** Effects after adsorption	Venturi et al., 2015
Hornstedtia bella	**PA.** Inhibiting a step of its replicative cycle that occurs during the early phase of viral infection and probably during entry	Sanna et al., 2021
Illicium verum? (star anise)	**PA.** Effects after adsorption	Abdallah et al., 2013
Juniperus phoenicea	**AP/PA/I.** Inhibiting viral attachment or adsorption, effects after adsorption **and** activity on proteases	Strub et al., 2022
Levisticum officinale	**AP/PA/I.** Inhibiting viral attachment or adsorption, effects after adsorption **and** activity on proteases	Strub et al., 2022
Lippia graveolens	**I.** EO slightly more efficient when applied after viral inoculation. Possible activity not on genome replication but on other steps of virus replication	Pilau et al., 2011
Melaleuca alternifolia	**I.** Activity on the steps of viral cycle replication (Potential)	Garozzo et al., 2011
Melaleuca alternifolia	**PA.** Interference with acidification of the intralysosomal compartment that could inhibit viral uncoating. Possible active molecules: terpinen-4-ol, terpinolene, alpha-terpineol	Garozzo et al., 2009
Melissa officinalis	**I.** Possible “inhibition of protein synthesis in cells”	Allahverdiyev et al., 2004
Myrocarpus fastigiatus	**AP/PA/I.** Inhibiting viral attachment or adsorption, effects after adsorption **and** activity on proteases	Strub et al., 2022
Oenanthe crocata	**I.** Activity on the steps of viral cycle replication (Potential)	Bicchi et al., 2009
Pelargonium graveolens	**PA/I.** Effects after adsorption, possibly inhibits a step of viral replicative cycle	Ralambondrainy et al., 2018
Ridolfia segetum	**I.** Activity on the steps of viral cycle replication (Potential)	Bicchi et al., 2009
Rosmarinus officinalis	**PA/I.** Effects after adsorption, possibly inhibits a step of viral replicative cycle **(**Potential)	Feriotto et al., 2018
Saccocalyx satureioides	**I.** Activity on the steps of viral cycle replication (Potential)	Laouer et al., 2006
Salvia desoleana	**PA/I.** The EO inhibits a step of viral replicative cycle after virus attachment and entry. Possible active molecules: β-caryophyllene, less likely germacrene D	Cagno et al., 2017
Santalum album	**I.** Activity on the steps of viral cycle replication	Benencia & Courrèges, 1999
Santalum austro-caledonicum	**AP/PA/I.** Inhibiting viral attachment or adsorption, effects after adsorption **and** activity on proteases	Strub et al., 2022
Thymus bovei	**I.** Activity on the steps of viral cycle replication	Hassan et al., 2018
Thymus vulgaris	**I.** EO inhibits the replication cycle but does not affect the resistance of cells	Saderi & Abassi, 2011
Thymus vulgaris	**PA/I.** Effects after adsorption, possibly inhibits a step of viral replicative cycle (Potential)	Feriotto et al., 2018

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PRISMA 2020 Checklist

Checklist adapted from Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, Shamseer L, Tetzlaff JM, Akl EA, Brennan SE, Chou R, Glanville J, Grimshaw JM, Hróbjartsson A, Lalu MM, Li T, Loder EW, Mayo-Wilson E, McDonald S, McGuinness LA, Stewart LA, Thomas J, Tricco AC, Welch VA, Whiting P, Moher D (2021) The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 372: n71. https://doi.org/10.1136/bmj.n71

This systematic review was conducted according to the OHAT approach for health-effects evaluations and is reported in accordance with PRISMA 2020. The review protocol was registered on the Open Science Framework on 6 October 2022, after the review had commenced but before its completion (DOI: https://doi.org/10.17605/OSF.IO/YRP9S).

Section	Topic	Item #	Checklist item	Location in manuscript
TITLE	**Title**	1	Identify the report as a systematic review.	Title page
ABSTRACT	**Abstract**	2	See the PRISMA 2020 for Abstracts checklist.	Abstract
INTRODUCTION	**Rationale**	3	Describe the rationale for the review in the context of existing knowledge.	Introduction, paras 1–3
	Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Introduction, final para
METHODS	**Eligibility criteria**	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Methods — Eligibility criteria; Table 2, section A
	Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Methods — Information sources
	Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Methods — Search strategy; Table 1
	Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Methods — Selection process; Figure 1
	Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	Methods — Data collection process
	Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Methods — Data collection process
		10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Methods — Data collection process
	Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Methods — Risk of bias assessment; Tables 3 and 4; Supplementary Table A1; RoB workbook
	Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	Methods — Data synthesis; Table 7

Section	Topic	Item #	Checklist item	Location in manuscript
	Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis).	Methods — Data synthesis
		13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics or data conversions.	Methods — Data synthesis (residual unit heterogeneity not converted; see Discussion)
		13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	Methods — Data synthesis; Tables 5–13; Figures 2–4
		13d	Describe any methods used to synthesise results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	Methods — Data synthesis (no meta-analysis; rationale given)
		13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	*Methods — Data synthesis (structured subgroup summaries by virus family and by essential-oil type; no meta-regression)*
		13f	Describe any sensitivity analyses conducted to assess robustness of the synthesised results.	*Methods — Data synthesis (synthesis restricted to Tier 1 studies; studies admitted under the amended eligibility criterion excluded)*
	Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Methods — Data synthesis (not formally assessed); Discussion — Limitations
	Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	Methods — Data synthesis (GRADE not applied; rationale given); Discussion — Limitations
RESULTS	**Study selection**	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	Results — Study selection; Figure 1
		16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Results — Study selection; Figure 1
	Study characteristics	17	Cite each included study and present its characteristics.	Results — Characteristics of Included Studies; Supplementary Tables A2 and A3
	Risk of bias in studies	18	Present assessments of risk of bias for each included study.	Results — Risk of Bias Assessment; Tables 5 and 6; Figures 3 and 4; Supplementary Figure A1;
	Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	Results — Efficacy and selectivity; Tables 8–10; Supplementary Tables A5–A11
	Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	Results — Human-virus evidence base; Tables 6 and 8–10
		20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	Results — Efficacy and selectivity (narrative synthesis; no pooled estimates)

Section	Topic	Item #	Checklist item	Location in manuscript
		20c	Present results of all investigations of possible causes of heterogeneity among study results.	*Results — Qualitative synthesis (Evidence by virus family; Evidence by essential-oil type); Tables 11 and 12*
		20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesised results.	*Results — Sensitivity analysis; Table 13*
	Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	Discussion — Limitations and future reflections
	Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	Discussion — Limitations and future reflections
DISCUSSION	**Discussion**	23a	Provide a general interpretation of the results in the context of other evidence.	Discussion, paras 1–10
		23b	Discuss any limitations of the evidence included in the review.	Discussion — Limitations and future reflections
		23c	Discuss any limitations of the review processes used.	Discussion — Limitations and future reflections
		23d	Discuss implications of the results for practice, policy, and future research.	Discussion — future directions; Conclusions
OTHER	**Registration and protocol**	24a	Provide registration information for the review, including the register name and registration number, or state that the review was not registered.	Methods — Review methodology (OSF: https://doi.org/10.17605/OSF.IO/YRP9S)
		24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	Methods — Review methodology (OSF registration)
		24c	Describe and explain any amendments to information provided at registration or in the protocol.	Methods — Eligibility criteria; Discussion — Limitations
	Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	*Funding (no specific grant received; funders played no role in the review)*
	Competing interests	26	Declare any competing interests of review authors.	Conflict of Interest
	Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytical code; any other materials used in the review.	Supplementary Tables A1–A12 and Reference lists A13.1–A13.4; accompanying A2, A3 and RoB workbooks.

PRISMA-S Checklist (Search reporting)

Checklist adapted from Rethlefsen ML, Kirtley S, Waffenschmidt S, Ayala AP, Moher D, Page MJ, Koffel JB; PRISMA-S Group (2021) PRISMA-S: an extension to the PRISMA Statement for Reporting Literature Searches in Systematic Reviews. Syst Rev 10(1): 39. https://doi.org/10.1186/s13643-020-01542-z

Section	Element	Item #	PRISMA-S item	How it is reported in this review	Location in manuscript
INFORMATION SOURCES AND METHODS	**Database name**	1	Name each individual database searched, stating the platform for each.	Seven databases were searched: Web of Science (Clarivate), PubMed/MEDLINE (NCBI), EMBASE (Elsevier), Scopus (Elsevier), Google Scholar (Google), CINAHL (EBSCO), and the Cochrane Library (Wiley).	Methods — Information sources
	Multi-database searching	2	If databases were searched simultaneously on a single platform, state the name of the platform, listing all of the databases searched.	Databases were searched individually on their respective native platforms; no multi-database aggregator was used.	Methods — Information sources
	Study registries	3	List any study registries searched.	No clinical-study registries (e.g. ClinicalTrials.gov, ICTRP) were searched. Eligible clinical trials were captured via the bibliographic databases listed above.	*Methods — Information sources (no registry searched)*
	Online resources and browsing	4	Describe any online or print source purposefully searched or browsed (e.g., tables of contents, print conference proceedings, web sites), and how this was done.	No purposeful browsing of journal tables of contents or print sources was performed.	*Methods — Search strategy (not performed)*
	Citation searching	5	Indicate whether cited references or citing references were examined, and describe any methods used for locating cited/citing references (e.g., browsing reference lists, using a citation index, setting up email alerts for references citing included studies).	Backward and forward citation searching was not performed. This is acknowledged as a limitation in the Methods and Discussion.	Methods — Search strategy; Discussion — Limitations
	Contacts	6	Indicate whether additional studies or data were sought by contacting authors, experts, manufacturers or others.	Corresponding authors were contacted for 22 reports that could not be retrieved through institutional or online resources. No full text was obtained; the reports were classified as not retrieved and did not enter eligibility assessment. No eligibility, extraction, or risk-of-bias judgment was changed through these contacts.	Methods — Selection process; Results — Study selection; Figure 1
	Other methods	7	Describe any additional information sources or search methods used.	No additional search methods were used (no grey-literature search, no preprint server search, no conference proceedings search). These exclusions are acknowledged as limitations.	Methods — Eligibility criteria; Discussion — Limitations
SEARCH STRATEGIES	**Full search strategies**	8	Include the search strategies for each database and information source, copied and pasted exactly as run.	Database-specific search strategies are reproduced in Table 1 of the manuscript, exactly as executed.	Methods — Search strategy; Table 1
	Limits and restrictions	9	Specify that no limits were used, or describe any limits or restrictions applied to a search (e.g., date or time period, language, study design) and provide justification for their use.	No language filter was applied at the search stage. Two retrieved reports published in Persian could not be assessed because no translation was available and were excluded on that basis; this de facto language restriction is declared in the Methods and in the Limitations. Google	Methods — Eligibility criteria; Methods — Search strategy;

Section	Element	Item #	PRISMA-S item	How it is reported in this review	Location in manuscript
				Scholar results were screened in relevance order and capped at the first 1,150 records because of high redundancy and negligible yield beyond that threshold. Unpublished studies were not actively sought.	Discussion — Limitations
	Search filters	10	Indicate whether published search filters were used (as originally designed or modified), and if so, cite the filter(s) used.	No published methodological search filters (e.g., Cochrane Highly Sensitive Search Strategy) were applied. PECO terms were combined with Boolean operators in each database's native syntax.	Methods — Search strategy; Table 1
	Prior work	11	Indicate when search strategies from other literature reviews were adapted or reused for a substantive part or all of the search, citing the previous review(s).	Search strategies were developed de novo for this review and not adapted from a previous published review.	Methods — Search strategy
	Updates	12	Report the methods used to update the search(es) (e.g., rerunning searches, email alerts).	An initial search was performed in April 2020 and re-run in February 2023 using identical database-specific strategies. No automated email alerts were used between the two searches.	Methods — Information sources
	Dates of searches	13	For each search strategy, provide the date when the last search occurred.	The original search was performed on all the databases on the 29th of April 2020. Web of Science, Medline via PubMed, Google Scholar, and the Cochrane Library were last searched on the 13th of February 2023, while the remaining three (CINAHL, EMBASE, Scopus) were searched on the 17th of February 2023.	Methods — Information sources
PEER REVIEW	**Peer review**	14	Describe any search peer review process.	The search strategies were not formally peer-reviewed by an information specialist (for example, by PRESS). They were internally reviewed by D.D. and M.A., the authors with the most systematic-review experience, until consensus; the absence of formal information-specialist review is acknowledged as a limitation.	*Discussion — Limitations*
MANAGING RECORDS	**Total records**	15	Document the total number of records identified from each database and other information sources.	Records retrieved per source: Cochrane Library 145; PubMed 1,595; Google Scholar 1,150 (screened, see item 9 regarding the Google Scholar cap); Web of Science 3,069; Scopus 4,384; EMBASE 2,806; CINAHL 157. Total: 13,306 records. See PRISMA 2020 flow diagram (Figure 1).	Results — Study selection; Figure 1 (PRISMA flow diagram)
	Deduplication	16	Describe the processes and any software used to deduplicate records from multiple database searches and other information sources.	Records were imported into EndNote and duplicates were removed manually after automated aggregation. A total of 4,997 duplicates were removed, leaving 8,309 records for title and abstract screening.	Methods — Search strategy; Results — Study selection; Figure 1