Dentistry 3000

Vol 14, No 2 (2026)
ISSN 2167-8677 (online)
DOI 10.5195/d3000/2026.1517

Effect of Lavender Essential Oil on Inflammatory Cell Response in Ligature-Induced Periodontitis in Rats

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Abstract

To evaluate the effect of systemically administered lavender essential oil on circulating inflammatory cell counts in a ligature-induced rat model of periodontitis. Periodontitis was induced in 18 rats using a 3-0 silk ligature placed around the maxillary right second molar the first use of this model in Iraq. Animals were randomly divided into three groups (n = 6 each): control (no ligation), ligated-only, and ligated plus daily oral systemic lavender essential oil (600 mg/kg). After 11 days, blood samples were collected via cardiac puncture and analyzed using an automated blood analyzer to quantify white blood cells, lymphocytes, monocytes, and granulocytes. Data were analyzed using one-way ANOVA with Tukey's HSD post-hoc test (SPSS v19). The control group showed no evidence of inflammation, while both ligated groups showed significant increases across all inflammatory cell types ($p < 0.001$). No statistically significant difference was found between the ligated-only and ligated-plus-lavender groups for WBC, granulocytes, or monocytes, indicating that lavender oil did not attenuate the systemic inflammatory response beyond ligation alone. Systemic administration of lavender essential oil (600 mg/kg) had no significant effect on inflammatory cell counts in ligature-induced periodontitis in rats. Future studies should investigate topical, rather than systemic, application.

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Citation: Al-Asadi AA et al. (2026) Effect of Lavander Essential Oil on Inflammatory Cell Response in Ligature-Induced Periodontitis in Rats. Dentistry 3000. 1:a001 doi:10.5195/d3000.2026.1517
Received: July 19, 2026
Accepted: July 24, 2026
Published: August 14, 2026

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Introduction

Periodontitis is a chronic inflammatory disease affecting the tooth-supporting structures the gingiva, periodontal ligament, cementum, and alveolar bone and remains one of the most prevalent oral diseases worldwide, affecting over a billion people and representing a leading cause of tooth loss in adults [1]. Its pathogenesis is driven by dysbiosis of the subgingival microbiome, in which keystone pathogens disrupt host–microbe homeostasis and trigger a dysregulated immune response [1,2]. Both innate immune cells (macrophages, neutrophils, monocytes) and adaptive immune cells (B and T lymphocytes) participate in this response, releasing cytokines, chemokines, and matrix metalloproteinases that ultimately degrade periodontal connective tissue and alveolar bone [2]. Because periodontitis does not occur spontaneously in rodents, ligature-induced models in which a suture is placed around a molar to promote local plaque accumulation remain the most widely used and reproducible experimental approach for studying periodontal inflammation and testing candidate therapeutic agents [3,4].

Lavender (Lavandula spp.) essential oil has long been used in traditional medicine for its sedative, antimicrobial, and anti-inflammatory properties [5]. More recent mechanistic work has shown that lavender essential oil and its major constituent, linalool, suppress lipopolysaccharide-induced production of pro-inflammatory cytokines including IL-6, IL-1β, IL-8, and TNF-α in human monocyte/macrophage cell models, an effect attributed at least in part to modulation of the NF-κB signaling pathway [5]. In vivo, lavender essential oil has also been shown to reduce acute inflammation induced by carrageenan and croton oil in animal models [6]. These findings support a plausible anti-inflammatory mechanism, but whether this effect extends to a chronic, bacterially driven inflammatory process such as periodontitis and whether it holds when the oil is delivered

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systemically rather than applied locally has not been established.

Given the established anti-inflammatory activity of lavender essential oil in acute and in vitro inflammation models, and the reproducibility of the ligature-induced periodontitis model in rats [3,4], the present study used this model established for the first time in Iraq to test whether daily systemic (oral) administration of lavender essential oil could attenuate the inflammatory cell response associated with ligature-induced periodontitis.

Material and Methods

Ethical approval and animal husbandry

This study was conducted in accordance with institutional and international standards for the ethical conduct and reporting of animal research, following the ARRIVE 2.0 guidelines [7]. Ethical approval was obtained from the Babylon University's College of Sciences institutional committee (Project No. 2230901) in 1-9-2023 [8-10]. Eighteen albino (New Zealand) rats of both sexes (females confirmed non-pregnant), aged 2–3 months, were obtained from the Animal House at Babylon University's College of Sciences. Males and females were housed separately from 10 days before the experiment through its completion to prevent unplanned breeding, and all animals were maintained under a 12-hour light/12-hour dark cycle with standardized diet and environmental conditions. Only animals free of visible disease and exhibiting normal activity were included.

Experimental design

Animals were randomly allocated into three groups (n = 6 per group): (1) control (untreated, non-ligated); (2) ligated-only (sham ligation, untreated); and (3) ligated plus systemic lavender essential oil treatment. Group size was determined based on prior methodology for experimental periodontitis models in rats [11]. The maxillary right second molar was selected as the ligation site owing to the accessibility provided by adjacent teeth, consistent with established ligature-induced periodontitis protocols [3,4].

Periodontitis induction

Following a 7-day acclimatization period, animals in the ligated groups underwent placement of a 3-0 silk ligature around the maxillary second molar under anesthesia (ketamine 100 mg/kg and xylazine 60 mg/kg, administered per Institutional Animal Care and Use Committee protocol). The ligature was secured with two knots (palatal and buccal) and adapted passively to the tooth surface. All animals including controls received the same anesthetic protocol to standardize experimental conditions. To promote plaque accumulation, all groups received a daily supplementary diet of cheese and sweet cake alongside standard chow. Animals were monitored daily for general health, body weight, and ligature stability. Periodontitis was allowed to develop over 11 days, consistent with previously validated ligature-induced models [3,4].

Lavender essential oil administration

Lavender essential oil (J.L.P.L. Panadora, Sri Lanka) was characterized by single-quadrupole GC/MS using an Agilent J&W HP-5ms Ultra Inert column at the Baghdad University College of Science and Technology. The compound profile is presented in Table 1. Group 3 animals received 600 mg/kg lavender essential oil determined by multiplying the animal's weight, administered orally via disposable syringe and stomach tube once daily for ten days prior to sacrifice, consistent with dosing used in prior anti-inflammatory studies of lavender essential oil [6].

Sample collection and analysis

On day 11, animals were euthanized under ketamine–xylazine anesthesia. Blood was collected via cardiac puncture using a 5-cc syringe; 1 mL was placed into an EDTA tube to prevent coagulation and transported to the laboratory within one hour for analysis. Inflammatory cell counts (white blood cells, lymphocytes, monocytes, and granulocytes) were quantified using an automated blood analyzer.

Statistical analysis

Data were analyzed using SPSS version 19. Normality of distribution was assessed using the Shapiro-Wilk test; all inflammatory cell variables showed $p > 0.05$ and were treated as normally distributed. Descriptive statistics (mean, standard deviation, minimum, maximum) were calculated for each group. Between-group differences were assessed using one-way ANOVA, with Tukey's HSD post-hoc test used to identify pairwise differences between groups.

Results

Identification of an oil sample characterization of lavender essential oil

The chemical composition of the lavender essential oil was determined by single-quadrupole gas chromatography–mass spectrometry (GC/MS). Eight major constituents were identified (Table 1), with linalyl acetate (44.01%) and linalool (39.61%) representing the predominant components the oil.

Distribution of inflammatory cell data

The distribution of inflammatory cell variables was assessed using the Shapiro–Wilk test. All variables demonstrated normal distribution (all $p > 0.05$), supporting the use of parametric statistical analyses (Table 2).

Inflammatory cell counts

Descriptive statistics for total white blood cells (WBCs), lymphocytes, monocytes, and granulocytes are summarized in Table 3.

Compared with the control group, ligature-induced periodontitis resulted in marked increases in total WBC, lymphocyte, and granulocyte counts. Animals receiving systemic lavender essential oil also exhibited elevated inflammatory cell counts relative to controls. Among all evaluated parameters, lymphocyte counts were highest in the lavender-treated group, whereas granulocyte counts were comparable between the ligated-only and lavender-treated groups. Monocyte counts showed relatively small variation across groups, although the highest mean value was observed following lavender administration (Figure 1).

Comparison of inflammatory cell counts among experimental groups

One-way ANOVA demonstrated statistically significant differences among the three experimental groups for all inflammatory cell parameters, including total WBCs ($F = 42.48$, $p < 0.001$), lymphocytes ($F = 97.76$, $p < 0.001$), monocytes ($F = 13.71$, $p < 0.001$), and granulocytes ($F = 38.32$, $p < 0.001$) (Table 4).

Post hoc analysis using Tukey's HSD test identified significant increases in total WBC and granulocyte counts in both ligated groups compared with the control group (all $p < 0.001$), whereas no significant differences were observed between the ligated-only and lavender-treated groups ($p = 0.09$ and $p = 0.992$, respectively).

For lymphocytes, significant differences were detected between all experimental groups. Lymphocyte counts were significantly higher in the ligated group than in controls ($p = 0.012$) and were further increased in the lavender-treated group compared with both the control and ligated-only groups (both $p < 0.001$).

Monocyte counts did not differ significantly between the control and ligated-only groups ($p = 0.138$). However, the lavender-treated group demonstrated significantly higher monocyte counts than both the control group ($p = 0.017$) and the ligated-only group ($p < 0.001$) (Table 5). Overall, systemic administration of lavender essential oil did not reduce total leukocyte or granulocyte responses associated with ligature-induced periodontitis. Instead, lymphocyte and monocyte counts were significantly higher in lavender-treated animals than in untreated ligated animals.

Discussion

The present study evaluated the effect of systemically administered lavender essential oil on circulating inflammatory cell counts in a ligature-induced rat model of periodontitis. The ligature model successfully induced a systemic inflammatory response, as demonstrated by

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significant elevations in total white blood cell (WBC), lymphocyte, and granulocyte counts compared with the non-ligated control group. However, daily oral administration of lavender essential oil (600 mg/kg) did not significantly reduce total WBC or granulocyte counts compared with untreated ligated animals. Furthermore, lavender-treated animals exhibited significantly higher lymphocyte and monocyte counts than the ligated-only group. Collectively, these findings suggest that under the experimental conditions employed in the present study, systemic administration of lavender essential oil failed to attenuate the systemic inflammatory response associated with ligature-induced periodontitis.

The significant elevation of circulating inflammatory cells observed following ligature placement confirms the successful establishment of experimental periodontitis. The ligature-induced model remains one of the most reproducible animal models for investigating periodontal inflammation because the retention of dental plaque promotes microbial dysbiosis and persistent activation of the host immune response, ultimately resulting in inflammatory cell recruitment, connective tissue destruction, and alveolar bone loss [12–14]. The increased total leukocyte and granulocyte counts observed in the present study are therefore consistent with the acute inflammatory reaction that accompanies bacterial accumulation around the ligated tooth and reflect systemic activation of innate immune mechanisms.

Neutrophils constitute the predominant granulocyte population involved in the early stages of periodontal inflammation and represent the first line of defense against periodontal pathogens. During disease progression, excessive neutrophil activation contributes to collateral tissue destruction through the release of reactive oxygen species, proteolytic enzymes, and neutrophil extracellular traps, thereby amplifying periodontal tissue injury [15,16]. The marked increase in granulocyte counts observed in both ligated groups supports previous reports demonstrating that ligature-induced periodontitis is associated with enhanced innate immune activation and systemic inflammatory responses extending beyond the periodontal tissues [13,15].

Lavender (*Lavandula angustifolia*) essential oil has attracted considerable scientific interest because of its documented antimicrobial, antioxidant, analgesic, and anti-inflammatory properties. Experimental studies have demonstrated that its principal constituents, particularly linalool and linalyl acetate, suppress the production of pro-inflammatory mediators including tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), interleukin-6 (IL-6), nitric oxide, and prostaglandin E2 through inhibition of the nuclear factor-kappa B (NF-κB) signaling pathway and modulation of macrophage activation [17–20]. These biological activities formed the rationale for investigating lavender essential oil as a potential adjunctive therapy for inflammatory diseases, including periodontitis.

Contrary to our initial hypothesis, systemic lavender essential oil administration did not significantly reduce total leukocyte or granulocyte counts. These findings differ from previous experimental investigations in which lavender essential oil reduced inflammatory cytokine production and inflammatory edema in acute inflammation models [18–20]. Several explanations may account for these discrepancies. First, most previous investigations evaluated acute chemically induced inflammation or in vitro cell cultures, whereas ligature-induced periodontitis represents a chronic, polymicrobial inflammatory disease characterized by continuous bacterial stimulation and complex interactions between microbial biofilms and host immune responses. Consequently, anti-inflammatory effects observed under acute experimental conditions may not necessarily translate to chronic periodontal inflammation [18,20].

Second, differences in the route of administration may have influenced the therapeutic response. The present study employed systemic oral administration, whereas several studies reporting beneficial anti-inflammatory effects have investigated either topical application or direct local administration of essential oils [17,19]. Systemically administered lavender oil undergoes gastrointestinal absorption, hepatic metabolism, and extensive biotransformation before reaching periodontal tissues, potentially reducing the bioavailability of active compounds at the site of inflammation. Local drug delivery systems have been shown to achieve substantially higher concentrations within periodontal pockets while minimizing systemic metabolism, suggesting that topical formulations may provide greater therapeutic efficacy in periodontal therapy [21,22].

Another factor that may explain the limited therapeutic response is the complexity of the host immune response during periodontitis. Although lavender essential oil has demonstrated inhibitory effects on selected inflammatory signaling pathways, periodontal disease involves numerous interconnected immune mechanisms, including activation of neutrophils, macrophages, dendritic cells, T lymphocytes, B lymphocytes, osteoclasts, complement pathways, and multiple cytokine networks [14,23]. Modulation of a limited number of inflammatory mediators may therefore be insufficient to suppress the overall inflammatory cascade generated by persistent bacterial challenge.

An interesting finding of the present study was the significant increase in circulating lymphocyte counts following lavender treatment compared with untreated ligated animals. To our knowledge, few studies have evaluated the influence of systemic lavender essential oil on lymphocyte responses in experimental periodontitis, making direct comparison difficult. Although lavender has generally been regarded as an anti-inflammatory agent, several natural products possess immunomodulatory rather than purely immunosuppressive properties, and their effects may vary according to dose, disease model, duration of treatment, and immune cell subtype [18]. Consequently, the observed increase in lymphocyte numbers should not necessarily be interpreted as evidence of increased tissue inflammation but may instead reflect altered immune regulation or redistribution of circulating lymphocyte populations. Confirmation of this hypothesis would require characterization of lymphocyte subsets and cytokine profiles using flow cytometry and immunological assays.

Similarly, monocyte counts were significantly higher in lavender-treated animals than in untreated ligated rats. Monocytes are circulating precursors of macrophages and osteoclasts and play a pivotal role in periodontal tissue destruction through production of inflammatory cytokines and differentiation into osteoclasts responsible for alveolar bone resorption [23]. However, monocytes also contribute to tissue repair and inflammation resolution depending on their phenotypic polarization. The present study quantified total circulating monocytes but did not distinguish between pro-inflammatory (M1-associated) and anti-inflammatory (M2-associated) phenotypes. Consequently, the biological significance of the observed increase remains uncertain and warrants further investigation using immunophenotypic characterization and molecular biomarkers.

It is noteworthy that although statistically significant differences were detected in lymphocyte and monocyte counts, no corresponding reduction was observed in total leukocyte or granulocyte responses. Taken together, these findings indicate that systemic lavender essential oil did not produce a clinically meaningful attenuation of systemic inflammation in this experimental model. Rather than demonstrating a protective anti-inflammatory effect, the results suggest that the selected dose and route of administration were insufficient to modify the systemic inflammatory response induced by ligature-associated periodontal infection.

The present findings should also be interpreted considering several methodological factors.

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Essential oil composition varies according to plant origin, extraction technique, storage conditions, and chemical profile, all of which may influence biological activity [17]. Although GC/MS analysis confirmed that the investigated oil contained high proportions of linalool and linalyl acetate, variability in minor constituents may contribute to differences between studies. In addition, only a single dosage and treatment duration were evaluated, precluding assessment of dose-response relationships. Future studies should investigate multiple doses, longer observation periods, and different delivery methods to determine whether therapeutic efficacy can be improved.

This study possesses several strengths. The ligature-induced model employed is well established and highly reproducible for investigating periodontal inflammation. Random allocation of animals, standardized induction of periodontitis, chemical characterization of the lavender essential oil, and objective quantification of inflammatory cells using an automated hematology analyzer enhance the internal validity of the findings. Furthermore, this study represents one of the first investigations evaluating the systemic effects of orally administered lavender essential oil on circulating inflammatory cells in experimental periodontitis and, to our knowledge, the first application of this ligature-induced rat model in Iraq.

Nevertheless, several limitations should be acknowledged. The relatively small sample size may have limited the statistical power to detect subtle treatment effects. Only circulating inflammatory cell counts were evaluated, whereas local periodontal parameters—including alveolar bone loss, histopathological inflammatory infiltrate, osteoclast activity, and cytokine expression—were not assessed. Similarly, inflammatory mediators such as TNF-α, IL-1β, IL-6, receptor activator of nuclear factor kappa-B ligand (RANKL), osteoprotegerin (OPG), oxidative stress biomarkers, and macrophage polarization markers were not measured. These biomarkers would have provided a more comprehensive understanding of the mechanisms underlying the observed immune responses. Finally, because only systemic administration was investigated, the potential therapeutic benefits of local periodontal delivery of lavender essential oil remain unknown.

Future investigations should combine systemic and local inflammatory assessments, evaluate cytokine expression and oxidative stress biomarkers, characterize immune cell phenotypes using immunohistochemistry or flow cytometry, and quantify alveolar bone loss using micro-computed tomography or histomorphometric analysis. In addition, topical formulations, nanoencapsulation systems, biodegradable local drug delivery devices, and combination therapy with conventional periodontal treatment should be explored to optimize the therapeutic potential of lavender essential oil in periodontal disease.

Overall, the present findings indicate that systemic administration of lavender essential oil at a dose of 600 mg/kg did not attenuate the systemic inflammatory response associated with ligature-induced periodontitis in rats. Although lavender essential oil possesses well-documented anti-inflammatory properties in other experimental models, these effects were not reproduced under the chronic inflammatory conditions of ligature-induced periodontal disease. The significant increases observed in lymphocyte and monocyte counts following treatment suggest that lavender essential oil may exert complex immunomodulatory effects rather than simple suppression of inflammation. Further mechanistic studies incorporating local periodontal outcomes, molecular inflammatory markers, and alternative routes of administration are required before systemic lavender essential oil can be considered a potential adjunctive therapy for periodontal disease.

Conclusion

Within the limitations of this experimental study, systemic administration of lavender (*Lavandula angustifolia*) essential oil at a dose of 600 mg/kg did not attenuate the systemic inflammatory response induced by ligature-induced periodontitis in rats. Although lavender essential oil has been widely reported to possess anti-inflammatory and immunomodulatory properties, no significant reductions were observed in total white blood cell or granulocyte counts compared with untreated ligated animals. Conversely, lymphocyte and monocyte counts were significantly higher following lavender treatment, suggesting that lavender essential oil may exert complex immunomodulatory effects rather than simply suppressing inflammation under chronic periodontal conditions.

These findings indicate that systemic administration of lavender essential oil, using the dosage and treatment protocol employed in the present study, cannot be considered an effective stand-alone approach for reducing systemic inflammatory cell responses associated with experimental periodontitis. The discrepancy between the present findings and previously reported anti-inflammatory effects observed in other experimental models highlights the complexity of host–microbial interactions during chronic periodontal disease and underscores the importance of disease-specific evaluation of phytotherapeutic agents.

Further well-designed experimental studies incorporating histopathological assessment, alveolar bone loss analysis, inflammatory cytokine profiling, oxidative stress biomarkers, and macrophage phenotyping are warranted to elucidate the underlying mechanisms of lavender essential oil in periodontal inflammation. Future investigations should also evaluate different dosages, treatment durations, and local drug delivery systems to determine whether optimized formulations may enhance the therapeutic potential of lavender essential oil as an adjunctive therapy for periodontal disease.

References

1. Hashim NT, Babiker R, Padmanabhan V, Ahmed AT, Chaitanya NCSK, Mohammed R, et al. The global burden of periodontal disease: a narrative review on unveiling socioeconomic and health challenges. Int J Environ Res Public Health. 2025;22(4):624. doi:10.3390/ijerph22040624

2. López-Valverde N, Quispe-López N, Blanco Rueda JA. Inflammation and immune response in the development of periodontal disease: a narrative review. Front Cell Infect Microbiol. 2024;14:1493818. doi:10.3389/fcimb.2024.1493818

3. Zhang X, Xu M, Xue Q, He Y. A modified method for constructing experimental rat periodontitis model. Front Bioeng Biotechnol. 2023;10:1098015. doi:10.3389/fbioe.2022.1098015

4. Chatzaki N, Stavropoulos A, Denes B, Cancela J, Kiliaridis S, Giannopoulou C. Induced periodontitis in rats with three ligature types: an exploratory study. Clin Exp Dent Res. 2024;10(4):e946. doi:10.1002/cre2.946

5. Pandur E, Balatinácz A, Micalizzi G, Mondello L, Horváth A, Sipos K, et al. Anti-inflammatory effect of lavender (Lavandula angustifolia Mill.) essential oil prepared during different plant phenophases on THP-1 macrophages. BMC Complement Med Ther. 2021;21:287. doi:10.1186/s12906-021-03461-5

6. Da Silva GL, Luft C, Lunardelli A, Amaral RH, Melo DAdaS, Donadio MVF, et al. Antioxidant, analgesic and anti-inflammatory effects of lavender essential oil. An Acad Bras Cienc. 2015;87(2 Suppl):1397–408.

7. Percie du Sert N, Hurst V, Ahluwalia A, Alam S, Avey MT, Baker M, et al. The ARRIVE guidelines 2.0: updated guidelines for reporting animal research. PLoS Biol. 2020;18(7):e3000410. doi:10.1371/journal.pbio.3000410

8. Al-Kufi, H.M., Alhumadi, A., Huthiafa, S.M. A comparative evaluation of postoperative re-pigmentation following gingival depigmentation using a 940-nm diode laser and abrasion method: “method: a split-mouth study” Lasers in Dental ScienceThis link is disabled., 2024, 8(1), 3

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9. Al-Khafaji TJ, Agha B, Alhumadi A, Alhamadi WW, Mills D, Davis GR, Cresswell-Boyes AJ, Fleming PS. An assessment of mineral concentration of dental enamel neighbouring hypothetical orthodontic brackets using X-ray microtomography. Journal of Dentistry. 2022 Nov 1;126:104306.
10. Alhumadi A. Gender-related response to different wavelength low-level laser therapy in orthodontic mandibular canine movement: a split mouth study. Endodontics Today. 2026;24(1):58–66. https://doi.org/10.36377/ET-0159
11. Sae Kwang Ku, Hyung Rae Cho, Yun Sub Sung, Su Jin Kang, Young Joon Lee. Effects of Calcium Gluconate on Experimental Periodontitis and Alveolar Bone Loss in Rats. BCPT 2010 ; 108 [4]: 241–250.
12. Chatzaki N, Stavropoulos A, Denes B, Cancela J, Kiliaridis S, Giannopoulou C. Induced periodontitis in rats with three ligature types: an exploratory study. Clin Exp Dent Res. 2024;10(4):e946.
13. Zhang X, Xu M, Xue Q, He Y. A modified method for constructing experimental rat periodontitis model. Front Bioeng Biotechnol. 2023;10:1098015.
14. López-Valverde N, Quispe-López N, Blanco Rueda JA. Inflammation and immune response in the development of periodontal disease: a narrative review. Front Cell Infect Microbiol. 2024;14:1493818.
15. Hashim NT, Babiker R, Padmanabhan V, Ahmed AT, Chaitanya NCSK, Mohammed R, et al. The global burden of periodontal disease: a narrative review on unveiling socioeconomic and health challenges. Int J Environ Res Public Health. 2025;22(4):624.
16. Zhang Y, Kuang Z, Wang Y, et al. Immunomodulation in the treatment of periodontitis: progress and perspectives. Front Immunol. 2021;12:781378.
17. Pandur E, Balatinácz A, Micalizzi G, Mondello L, Horváth A, Sipos K, et al. Anti-inflammatory effect of lavender (*Lavandula angustifolia* Mill.) essential oil prepared during different plant phenophases on THP-1 macrophages. BMC Complement Med Ther. 2021;21:287.
18. Kajjari S, Joshi RS, Hugar SM, Gokhale N, Meharwade P, Uppin C. The effects of lavender essential oil and its clinical implications in dentistry: a review. Int J Clin Pediatr Dent. 2022;15(3):385–388.
19. Sabaoui Z, Lakhdar L. Essential oils in periodontics: what is the interest? Integr J Med Sci. 2021;8:499.
20. Castellino G, Mesa F, Cappello F, Benavides-Reyes C, Malfa GA, Cabello I, et al. Effects of essential oils and selected compounds from Lamiaceae family as adjutants on the treatment of subjects with periodontitis and cardiovascular risk. Appl Sci (Basel). 2021;11(20):9563.
21. de Sousa ET, de Araújo JSM, Pires AC, dos Santos EJL. Local delivery natural products to treat periodontitis: a systematic review and meta-analysis. Clin Oral Investig. 2021;25(7):4599–4619.
22. Li J, Wang Y, Tang M, Zhang C, Fei Y, Li M, et al. New insights into nanotherapeutics for periodontitis: a triple concerto of antimicrobial activity, immunomodulation and periodontium regeneration. J Nanobiotechnology. 2024;22:9.
23. Blancas-Luciano BE, Zamora-Chimal J, da Silva-de Rosenzweig PG, Ramos-Mares M, Fernández-Presas AM. Macrophages immunomodulation induced by Porphyromonas gingivalis and oral antimicrobial peptides. Odontology. 2023 Oct;111(4):778-792.

Table 1. The compounds that consisted of the oil sample.

peak	Retention time	Compound name	Area	CAS number
1	5.738	Alpha-pinene	0.51	000080-56-8
2	10.63	D-limonene	0.45	005989-27-5
3	10.748	Ecalyptol	6.63	00470-82-6
4	16.86	1,6-Ocatadien-3-ol, 3,7-Dimethyl	39.61	000078-70-6
5	19.242	Camphor	6.1	000076-22-2
5	19.242	(+)-Bornanone	6.1	000464-49-3
6	20.881	Endo-borneol	0.57	000507-70-0
6	20.881	Bicyclo [2.2.1}heptan-2-ol, 1,7,7-trimethyl-, (1S-endo) -	0.57	000464-45-9
7	25.976	1,6-octadien-3-ol, 3,7-dymethyl-,2-amenobenzoate	44.01	007149-26-0
7	25.976	Linalyl acetate	44.01	000115-95-7
7	25.976	1,5-dimethyl-1-vinyl-4-hexenyl butyrate	44.01	000078-36-4
8	31.485	Caryophyllene	2.13	000087-44-5

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Table 2. The distribution of the inflammatory cells.

Groups	cells	Shapiro-Wilk test	d.f.	p-value
Control	WBC	0.818	6	0.084
	LYM	0.894	6	0.341
	MON	0.831	6	0.111
	GRA	0.961	6	0.83
Ligated	WBC	0.882	6	0.28
	LYM	0.96	6	0.819
	MON	0.828	6	0.102
	GRA	0.996	6	0.999
Ligated and lavender	WBC	0.972	6	0.907
	LYM	0.945	6	0.703
	MON	0.866	6	0.212
	GRA	0.97	6	0.891

Table 3. Descriptive statistics of inflammatory cells in different groups.

Inflammatory cells	Groups	No.	Mean	S.D.	Min.	Max.
WBCtotal	Control	6	11.517	2.214	9.7	15.6
	Ligated	6	18.667	2.088	16.6	22.2
	Ligated and lavender	6	21.133	1.145	19.6	22.6
LYM	Control	6	8.417	0.553	7.8	9.4
	Ligated	6	10.017	0.945	8.8	11.3
	Ligated and lavender	6	14.833	0.924	13.6	16
MON	Control	6	2.267	0.308	2	2.7
	Ligated	6	1.967	0.28	1.7	2.5
	Ligated and lavender	6	2.733	0.151	2.5	2.9
GRA	Control	6	1.383	0.172	1.1	1.6
	Ligated	6	4.567	0.924	3.3	5.9
	Ligated and lavender	6	4.517	0.823	3.3	5.8

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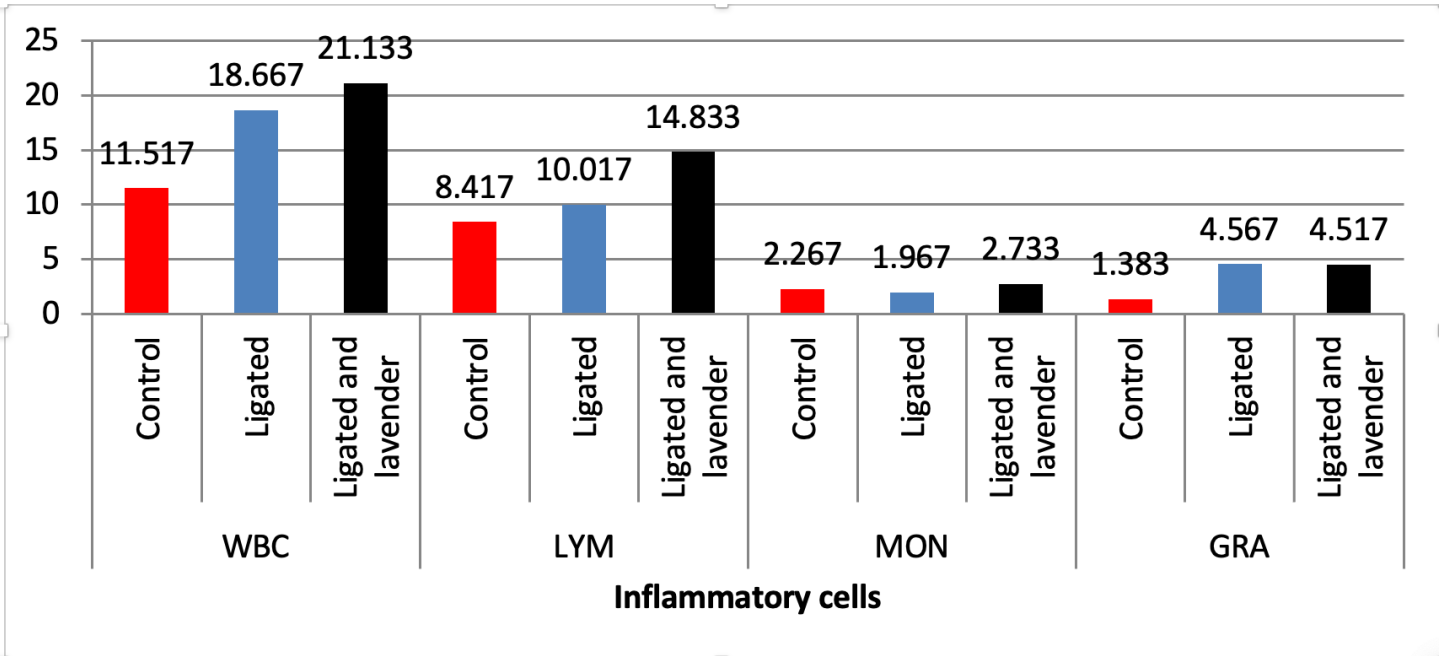

Figure 1. Bar chart showing the mean values of all inflammatory cells in the three groups.

Table 4. Comparison the Inflammatory cells among the groups.

Inflammatory cells	ANOVA	Sum of Squares	d.f.	Mean Square	F-test	p-value
WBC	Between Groups	299.374	2	149.687	42.481	0.000
	Within Groups	52.855	15	3.524		
	Total	352.229	17			
LYM	Between Groups	133.868	2	66.934	97.761	0.000
	Within Groups	10.27	15	0.685		
	Total	144.138	17			
MON	Between Groups	1.791	2	0.896	13.707	0.000
	Within Groups	0.98	15	0.065		
	Total	2.771	17			
GRA	Between Groups	39.908	2	19.954	38.324	0.000
	Within Groups	7.81	15	0.521		
	Total	47.718	17			

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Table 5. The Tukey's HSD test for intergroup comparison of mean difference.

cells	Groups		Mean Difference	p-value
WBC	Control	Ligated	-7.15	0.000
		Ligated and lavender	-9.617	0.000
	Ligated	Ligated and lavender	-2.467	0.09
LYM	Control	Ligated	-1.6	0.012
		Ligated and lavender	-6.417	0.000
	Ligated	Ligated and lavender	-4.817	0.000
MON	Control	Ligated	0.3	0.138
		Ligated and lavender	-0.467	0.017
	Ligated	Ligated and lavender	-0.767	0.000
GRA	Control	Ligated	-3.183	0.000
		Ligated and lavender	-3.133	0.000
	Ligated	Ligated and lavender	0.05	0.992