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Evaluation The Effect of Natural Phytotherapy on Level of Leptin Hormone in Serum With Chronic Periodontitis

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ABSTRACT:

For a considerable amount of time, periodontal diseases have been recognized as serious health issues in various populations. It refers to a class of locally acquired microbially generated infections that affect the gingiva and the tissues that support the teeth. The two most common kinds of periodontal disorders are gingivitis and chronic periodontitis. It is acknowledged that the primary causative factor for the development and advancement of periodontitis is bacterial plaque. When untreated, chronic periodontitis begins as an inflammatory illness of the gingiva and can eventually spread to other sections of the periodontal attachment mechanism, resulting in damage and collapse. Natural agents with antibacterial and anti-inflammatory properties may be able to manage inflammatory illnesses like periodontitis. Natural plant chemicals have the capacity to regulate the immune system's inflammatory response.

Key words: Leptin, IL-6, E. Coli, Moringa oleifera, and periodontal inflammation.

Objective :

The current study aims to assess the impact of natural phytotherapy, specifically Moringa Oleifera Extract, on serum levels of IL-6 and leptin that are high in serum with chronic periodontitis.

Introduction:

A collection of inflammatory oral illnesses known as periodontal disease are brought on by oral pathogens and manifest as complex biolayers on the tooth's surface. The degree of this infectious disease varies from moderate and reversible gingiva inflammation (gingivitis) to persistent destruction of connective tissues due to an imbalance between the microbial biofilm that accumulates on teeth and the host immunological inflammatory response.¹, The gum tissues separate from the tooth as a result of this process, resulting in the loss of bone support and a periodontal pocket. Consequently, deep gaps between teeth and gums that cause

teeth to loosen and eventually fall out, gingival recession, and gum swelling and bleeding during probing are all signs of chronic periodontitis (CP) ².

More than 500 bacterial species invade, and advanced periodontal diseases are highly correlated with red complex. *P. gingivalis* and *Treponema denticola* are two of the red complex bacteria seen in periodontal diseases. ³ One of the primary substances found in the membrane of Gram-negative bacteria is lipopolysaccharide (LPS). It has the capacity to trigger immunological reactions by activating periodontal tissue-resident cells, which results in the release of several inflammatory mediators like as adhesion molecules, chemokines, and interleukins. ⁴,

In the acute and early chronic phases of inflammation, resident cells such fibroblasts and epithelial cells, as well as phagocytes (macrophages and neutrophils) and immune cells (lymphocytes) in established and advanced lesions, release the cytokines of innate immunity. ⁵ Tumor necrosis factor alpha, interleukin-1beta, and interleukin-6 are examples of innate response cytokines that are involved in the pathophysiology pathways of periodontal disease, initially emerge following the identification and presentation of bacteria to the proper cells. ⁶ Two hallmark innate cytokines, IL-1 beta and IL-6, have been linked to osteoclastogenesis and inflammatory cell migration. ⁷

Leptin (ob) is a 16 kDa non-glycosylated polypeptide hormone that is primarily produced by adipocytes and released into the bloodstream. It regulates numerous endocrine axes, consumes and stores energy, regulates thermoregulation, and regulates bone metabolism. ⁸ Leptin's biological actions are aided by its contact with the unique cell surface leptin receptor (Ob-R). ⁹ In addition to being present in salivary gland acinar cells and oral mucosa, Ob and Ob-R have been identified in a number of tissues and organs, including the hypothalamus, stomach, and intestinal mucosa. ¹⁰ The salivary glands and oral mucosa contain Ob and Ob-R, indicating that leptin activity has a role in oral disorders. ¹¹

Microorganisms and the increased frequency of oral disorders are intimately linked, and this is because some common antibacterial medications have toxic and dangerous side effects. as well as the growing antibiotic bacterial resistance, there is a need for safe, effective, and reasonably priced alternative treatment options and therapies, such as herbal therapies. ¹² In the purest definition of the word, phytochemicals are substances made by plants that may have an effect on health. Numerous phytochemicals are present in *Moringa oleifera*, some of which are particularly interesting due to their potential medical benefits. ¹³

Known as the "miracle tree" or "Mother" Best Friend, *Moringa Oleifera* (MO) is the most well-known and extensively distributed species in the *moringaceae* family. It has a high nutritional value and an astounding range of medical uses worldwide. ¹⁴ The leaves and blossoms of the moringa plant are used as a significant source of vitamins and minerals. ¹⁵ The calcium oxalate crystals found in the leaves and stems of *Moringa oleifera* are widely known for containing a significant amount

of calcium. It has more potassium than bananas, more iron than spinach, more calcium than milk, more than oranges in vitamin C and greater than carrots in vitamin A.¹⁶

Materials and Methods :

36 albino rats in good health, weighing between 150 and 200 grams, were utilized in this investigation. They were housed in Egypt's Nile Center for Trial Research. Rats were housed in conditions that were appropriate in terms of humidity and temperature. **Grouping of animals:**

Three primary categories were identified for the animals. (12 rats for each) :

➤ Group A (Control group)

➤ The animals in this group were allowed to behave as totally calm control animals without any intervention.

➤ Group B (LPS group)

Animals of this group were injected by 30µg of lipopolysaccharide into the rat mandibular gingiva of the first molar, thrice every week for ten days of induction of periodontal disease¹⁷

➤ Group C (Libopolysaccharide LPS and MO group)

As group B, this group manipulated. Additionally, during the trial period, it was administered MO at a dose of 300 mg/kg once day.¹⁸

Next, serum levels of IL-6 were assessed in each group to confirm that groups B and C had developed periodontitis. The MO leaves were bought from an Egyptian herbal store. The pharmacognosy department of the Faculty of Pharmacy's Liver Research Lab handled the extraction, purification, and extract preparation. Preparation of aqueous extract of MO, MO leaves were air-dried under room temperature. The dried leaves were powdered using a grinder to produce a fine uniform powder. The powder was subjected to extraction by percolation at room temperature using 70% Methanol. The powder was extracted several times till exhaustion to produce over 8 liters of alcoholic extracts. The alcoholic extracts were dried using rotary evaporator under vacuum at 45 oC to produce a semi solid green residue. The produced residue was kept in the refrigerator till use.

The needed dose was prepared according to the protocol (300mg/kg). The dose was calculated/rat (assuming rat's weight 200 grams) i.e 60 mgs/rat, which was found in 1.2 mL (1.2 mL contains 60 mgs of extract per rat) administered orally by gastric tube started the evening before starting of LPS injections.¹⁸

Induction of Periodontitis:

General anesthesia was established with intraperitoneal doses of ketamine hydrochloride (50 mg/ Kg) and xylazine hydrochloride (5 mg/ Kg), then were positioned on his back. PBS was used to dissolve 30 µg of E. Coli LPS (Strain

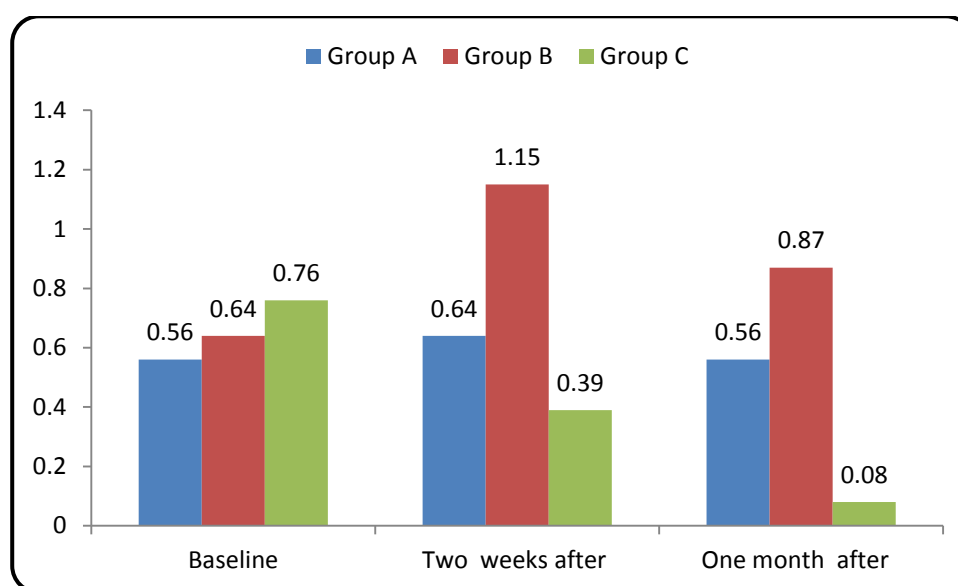
055:B5 – Sigma Chem Co., St. Louis, MO, USA), then injected bilaterally into the mandibular gingiva at the distobuccal aspect of the first molar (30 µg/site For ten days, three times a week, a total of five injections and 150 µg of LPS per site were administered. The injections were performed gradually and the needle was kept in position for a few seconds after injection to make sure the needle track did not lose any LPS.

Blood sampling collection and measuring cytokines :

All rats had blood obtained from their eyes on the first day, and on the fifteenth and thirtieth days, respectively, additional blood samples were taken from the hearts of half of each group and the other half. entering micro-capillaries coated with heparin while under ketamine/xylazine anesthesia. To separate the serum, the samples were centrifuged for 15 minutes at 3000 RPM. Using a Pasteur pipette, The serum was meticulously removed and placed in sterile, dry Wasserman tubes. It was then frozen until investigation at a temperature of -20 oC. Next, using a rat standard ELISA kit, the concentrations of IL6 and leptin in serum were determined on the first (baseline), 15th, and 30th days.

Results:

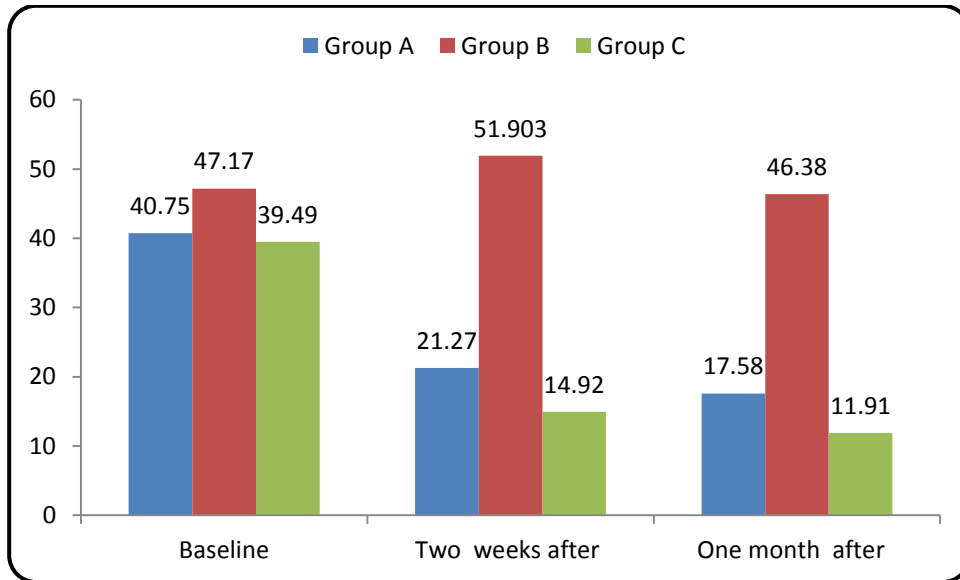
Table (1) Comparison of the changes in leptin levels between the study groups at baseline, after 2 weeks, and after a month into the investigation.



shows that the difference is statistically significant between groups B and C in terms of the change in leptin levels from the study's baseline to one month later. Leptin level decreased from baseline, after 2 weeks & after a month of the study (0.76 ± 0.47 , 0.39 ± 0.17 & 0.08 ± 0.03 , respectively) in group C and increased level (0.64 ± 0.39 , 1.15 ± 0.53 & 0.87 ± 0.47 , respectively) for group B. In group A, there is a minor increase followed by a reduction in leptin levels from baseline after two

weeks and one month (0.56 ± 0.31 , 0.64 ± 0.48 , and 0.56 ± 0.35 , respectively), but there is no statistically significant difference.

Table (2) Comparison of the research groups' alterations in IL-6 levels at the beginning, after two weeks, and after a month.



demonstrates that group C had the only statistically significant difference in IL 6 level change from baseline to one month following the research. After two weeks and one month of the trial, the IL6 level dropped from baseline (39.49 ± 10.39 , 14.92 ± 0 & 11.91 ± 4.5 , respectively). Group B exhibits a rise in IL 6 following two weeks, followed by a decline after one month (51.903 ± 26.9 & 46.38 ± 17.09 , respectively). There is no statistically significant difference observed between baseline and the two weeks ($p=0.063$) or between the baseline and the month ($p=0.36$).

The results: - After two weeks and one month, group B with chronic periodontitis showed statistically significant elevated total levels of leptin and IL-6 in serum, and group C with chronic periodontitis treated with *Moringa oleifera* showed highly statistically significant reduction in leptin and IL-6 in serum. LPS injection caused a significant increase in gingival and periodontal inflammation.

Discussion

Many diseases, such as skin conditions, respiratory issues, ear and tooth infections, hypertension, diabetes, anemia, and cancer, are treated with the plant in traditional medicine. It also has many other therapeutic uses.¹⁹ Moringa leaves are a rich source of minerals and proteins, and they include all eight of the essential amino acids.. Moringa has argenine and histidine in it. Particularly for newborns who

cannot produce enough protein to meet their growing needs, amino acids are crucial. It has calcium, K, and vitamin A in it.²⁰

The current study's results showed that there were no statistically significant differences in the study groups' baseline blood levels of leptin; nevertheless, following two weeks of comparison, group A and group C showed a very statistically significant difference in leptin levels. No discernible difference existed between groups A and B or B and C. These findings from the research done by **Johnson and Shari 2001** found that the gingiva in healthy individuals had the highest levels of leptin, which declined as the severity of PD increased. This fluctuation was ascribed to the increased serum leptin level and leptin clearance from gingival tissue induced by the expanded microvasculature present in periodontitis.²¹

After a month, there was no statistically significant difference between group A and B, however there was a high significant difference between group B and C and between group A and C, respectively. These results in accordance to **Karthikeyan and Pradeep 2007** who carried out the study found that the degree of periodontal damage raised serum levels of leptin and lowered gingival crevicular fluid levels.²²

In the current investigation, group B's serum IL-6 level was marginally higher at baseline than it was for groups A and C. Furthermore, after two weeks, group B's serum IL-6 level was noticeably greater than group C's. The inflammatory responses to the microorganisms that were observed in group B could account for the elevated levels of IL-6. These outcomes were confirmed by **Monea et al., 2014** that found higher levels of IL-6 in the serum of patients with chronic periodontitis than in healthy periodontal control individuals.²³ **Choi et al., 2014** who revealed that prevotella intermedia LPS stimulated IL-6 production.²⁴

The elevated IL-6 levels observed in the research groups were explained by **Noh et al., 2013** They clarified that IL-6 is expressed in a range of circumstances pertaining to inflammatory responses to bacterial lipopolysaccharides (LPS) and host immunological responses.²⁵ Periodontal tissue is destroyed when IL-6 activates gingival fibroblasts to create collagenolytic enzymes.²⁶ Visfatin expression can be markedly elevated by elevated proinflammatory cytokines, such as IL-6. Visfatin increases the expression of the inflammatory and adhesion proteins, intercellular adhesion molecule 1 (ICAM-1), Vascular cell adhesion protein1 (VCAM-1), As periodontal disease severity grows, so do leukocyte endothelial adhesion molecules (E-Selectin), which function in an autocrine and paracrine way to induce PGE2 production, GCF, and serum Visfatin concentrations.²⁷

When compared to the baseline level, the group C's serum level of IL-6 showed a substantial decrease following Moringa Oleifera administration. These outcomes were

consistent with **Kardesler et al., 2010** and **Kocak et al. 2016** They found that because periodontal therapy controls inflammation, it can lower levels of circulating inflammatory mediators like IL-6.²⁸⁻²⁹

In the current study about leptin and IL-6 our results in agreement with **Shi et al., 2015** who reported the In patients with aggressive periodontitis, elevated plasma leptin concentration may be linked to higher systemic levels of inflammatory markers.³⁰ **Shimada et al., 2010** examined the relationship between patients' serum levels of CRP, IL-6, and leptin and chronic periodontitis. According to their findings, serum leptin, IL-6, and CRP levels rose as periodontal disease worsened and sharply declined following non-surgical periodontal therapy, suggesting that elevated serum leptin may increase the risk of cardiovascular disease..³¹

There are several different therapeutic approaches to eliminate or lessen PD. These include of using prescription drugs, home cures, and mechanical plaque reduction.³² The chemical approach, which is predicated on systemic antibiotic therapy, has a danger of bacterial resistance, while the mechanical method is uncomfortable and painful. But the potential of natural plant compounds to control the immune system's inflammatory response has raised interest in them as potential treatments for a number of illnesses, including periodontitis. Thus, natural substances with antibacterial and anti-inflammatory properties may be able to manage inflammatory conditions like periodontitis.³³

That was aggremented with **Swathi et al., 2016 2016** investigated to evaluate the effects of Moringa Oleifera extract on periodontopathogens like Aggregatebacter actinomycetemcomitans (Aa), Porphyromonas gingivalis(Pg), Subgingival plaque samples were obtained from patients suffering from chronic periodontitis, and Prevotella intermedia (Pi) and Fusobacterium nucleatum (Fn) were grown and incubated anaerobically in accordance with normal protocol.. The produced extracts of Moringa oliefera are used to examine the subcultured strains of Aa, Pg, Pi, and Fn. Their findings provided evidence in favor of Moringa oliefera's application as a natural antibacterial agent in periodontal therapy.³⁴

Because moringa oleifera leaves contain a variety of antioxidant components, including flavonoids, phenolics, ascorbic acid, and essential amino acids including lysine, cystine, tryptophan, and methionine, they function as a good natural source of antioxidants. 35 in line with our findings, **Nagashree et al., 2011** revealed that MOE (200 mg/kg bw/day) includes compounds that function as antioxidants and shield the body from the harm caused by arsenite, resulting in a (substantially) decrease in albino rats' Hb and blood cell counts..³⁶

Conclusions

1. Rats can be given an easily generated, repeatable experimental model of periodontal inflammation via intragingival injection of LPS, which replicates the characteristics of human disease..
2. Serum levels of leptin and IL-6 may be utilized as possible diagnostic markers for periodontal disease activity.
3. systemic administration of *Moringa Oleifera* in the treatment of periodontal diseases was effective in improving the condition of the periodontitis in animals.
4. In the end, I recommend a study and researching in evaluation the effect of *Moringa Oleifera* in the treatment of human periodontal diseases act as an alternative treatment in Periodontal Diseases.

References

1. Kah Yan How, Keang Peng Song and Kok Gan Chan. *Porphyromonas gingivalis*: An Overview of Periodontopathic Pathogen below the Gum Line. *Front Microbiol.* 2016; 7: 53.
2. Pihlstrom BL, Michalowicz BS and Johnson NW. Periodontal diseases. *Lancet* 2005; 366: 1809–1820.
3. Mysak J, Podzimek S, Sommerova P, Lyuya-Mi Y, Bartova J, Janatova T et al., *Porphyromonas gingivalis*: Major periodontopathic pathogen overview. *J Immunol Res* 2014; 2014: 476068.
4. Gorąca A, Huk-Kolega H, Kleniewska P, Piechota-Polańczyk A and Skibska B. Effects of lipoic acid on spleen oxidative stress after LPS administration. *Pharmacol Rep* 2013; 65: 179–186.
5. Ara T, Kurata K, Hirai K, Uchihashi T, Uematsu T, Imamura Y, et al., Human gingival fibroblasts are critical in sustaining inflammation in periodontal disease. *J Periodontal Res* 2009; 44: 21–27.
6. Garlet GP. Destructive and protective roles of cytokines in periodontitis: a re-appraisal from host defense and tissue destruction viewpoints. *J Dent Res* 2010; 89: 1349–1363.
7. Fonseca JE, Santos MJ and Canhao H. Choy E. Interleukin-6 as a key player in systemic inflammation and joint destruction. *Autoimmun Rev* 2009; 8: 538–542.
8. Zhang Y, Proenca R, Maffei M, Barone M, Leopold L and Friedman JM. Positional cloning of the mouse obese gene and its human homologue. *Nature.* 1994; 372: 425–432.
9. Lord GM, Matarese G, Howard JK, Baker RJ, Bloom SR and Lechler RI. Leptin modulates the T-cell immune response and reverses starvation-induced immunosuppression. *Nature.* 1998; 394: 897–901.
10. Groschl M, Rauh M, Wagner R, Neuhuber W, Metzler M, Tamguney G, et al., Identification of leptin in human saliva. *J Clin Endocrinol Metab.* 2001; 86: 5234–5239.
11. Karthikeyan BV and Pradeep AR. Leptin levels in gingival crevicular fluid in periodontal health and disease. *J Periodontal Res.* 2007; 42: 300–304.

12. Palombo EA. Traditional medicinal plant extracts and natural products with activity against oral bacteria: potential application in the prevention and treatment of oral diseases. *Evid-Based Compl. AIT.* 2011; 2011: 15.
13. Bose CK. Possible role of Moringa Oleifera Lam. Root in epithelial ovarian cancer. *MED Gen MED.* 2007; 9: 26.
14. Farooq F, Rai M, Tiwari A, Khan AA and Farooq S . Medicinal properties of Moringa oleifera: An overview of promising healer. *J of Medicinal Plants Research.* 2012; 6: 4368-4374.
15. Khalafalla MM, Abdellatef E, Dafalla MM, Nassrallah AA, Aboul-Enein KM, Lightfoot DA, et al., Active principle from Moringa oleifera Lam leaves effective against two leukemias and a hepatocarcinoma. *Afr. J. Biotechnol.* 2010; 9: 8467-8471.
16. Singh N and Gilca M. Herbal Medicine: Science embraces tradition: A new insight into ancient Ayurveda. Lambert Academic Publishing AG and Co KG Dudweiler Landstr 99,66123. Saarbrücken, Germany: ISBN 978-3-8383-2145-5.
17. Cowan MM. Plant products as antimicrobial agents. *Clin. Microbiol. Rev.* 1999; 12: 564–582.
18. Sinha M, Das DK, Bhattacharya S, Majumdar S and Dey S. Leaf extract of Moringa oleifera prevents ionizing radiation-induced oxidative stress in mice. *J MED food.* 2011; 14: 1167-1172.
19. Mukunzi D, Nsor-Atindana J, Zhang XM, Gahungu A, Karangwa E and Mukamurezi G. Comparison of volatile profile of Moringa oleifera leaves from Rwanda and China using HS-SPME. *Pak J Nutr* 2011; 10: 602-608.
20. Tiloke C, Phulukdaree A and Chaturgoon AA. The antiproliferative effect of Moringa oleifera crude aqueous leaf extract on cancerous human alveolar epithelial cells. *BMC complementary and alternative medicine.* 2013; 13: 226.
21. Johnson RB and Serio FG. Leptin within healthy and diseased human gingiva. *J Periodontol.* 2001; 72: 1254–1257.
22. Karthikeyan BV and Pradeep AR. Gingival crevicular fluid and serum leptin :their relationship to periodontal health and disease. *J Clin Periodontol* 2007; 34: 467-472.
23. Monea A, Gruber R, Elod N , Bereşescu G , Moldovan C and Monea M. Saliva and serum of TNF- α and IL- 6 in a sample of Romanian adult subjects with type 2 diabetes mellitus and periodontal disease. *European Scientific Journal* , 2014 ; 10: 1857 -7881 .
24. Choi EY, Jin JY, Choi JI, Choi IS and Kim SJ. DHA suppresses Prevotella intermedia lipopolysaccharide-induced production of proinflammatory mediators in murine macrophages. *Br J Nutr*, 2014; 111: 1221–1230.
25. Noh MK, Jung M, Kim SH, Lee SR, Park KH, Kim DH, et al., Assessment of IL-6, IL-8 and TNF- α levels in the gingival tissue of patients with periodontitis. *Experimental And Therapeutic Medicine*, 2013; 6: 847-851.
26. Takashiba S, Naruishi K and Murayama Y. Perspective of cytokine regulation for periodontal treatment: fibroblast biology. *Journal of periodontology* ,2003; 74: 103-110.
27. Raghavendra NM, Pradeep AR, Kathariya R, Sharma A, Rao NS and Naik SB. Effect of non surgical periodontal therapy on gingival crevicular fluid and serum

- visfatin concentration in periodontal health and disease. *Disease Markers*, 2012; 32: 383–388 .
- 28.Kardesler L, Buduneli N, Cetinkalp S & Kinane D F** . "Adipokines and Inflammatory Mediators after Initial Periodontal Treatment in Patients with Type 2 Diabetes and Chronic Periodontitis," *Journal of Periodontology*, 2010; 81: 24-33.
- 29.Kocak E, Sağlam, Kayış SA et al.** Nonsurgical periodontal therapy with/without diode laser modulates metabolic control of type 2 diabetics with periodontitis: a randomized clinical trial, 2016; 31: 343-353.
- 30.Shi D, Liu YY, Li W, Zhang X, Sun XJ, Li Xu, Zhang L, Chen ZB, and Meng HN.** Association between Plasma Leptin Level and Systemic Inflammatory Markers in Patients with Aggressive Periodontitis. *Chin Med J*, 2015; 128: 528–532.
- 31.Shimada Y, Komatsu Y, Ikezawa-Suzuki I, Tai H, Sugita N and Yoshie H.** The effect of periodontal treatment on serum leptin, interleukin-6, and C-reactive protein. *J Periodontol.* 2010; 81: 1118–1123.
- 32.Bansal S, Rastogi S and Meenakshi Bajpai M.** Mechanical, chemical and herbal aspects of periodontitis.*IJPSR.* 2012; 3: 1260–1267.
- 33.Chainy GB, Manna SK, Chaturvedi MM and Aggarwal BB.** Anethole blocks both early and late cellular responses transduced by tumor necrosis factor: effect on NF- κ B, AP-1, JNK, MAPKK and apoptosis.*Oncogene.* 2000; 19: 2943–2950.
- 34.Swathi K, Savita AM, Pallavi Nanaiah. K, Abhilash N, Vaijinathrao SS and Abdul H.** ANTIMICROBIAL EFFECTS OF PHYLLANTHUS EMBLICA EXTRACT AND MORINGA OLIEFERA EXTRACT ON SPECIFIC PERIODONTOPATHOGENS - AN IN VITRO STUDY, *International Journal of Analytical, pharmaceutical and biomedical sciences.* 2016; 5: 2278-0246.
- 35.Anwar F, Ashraf M, Latif S and Gilani AH.** Moringa Oleifera: A Food Plant with Multiple Medicinal Uses.*Phytother. Res.* 2007; 21: 17–25.
- 36.Nagasheree R, Latha R and Karthikeyan V.** Effect of leaves of Moringa Oleifera on biochemical and physiological parameters in rats. *Journal of natural remedies* . 2011; 11: 1.

تقييم تأثير العلاج بالنباتات الطبيعية على مستوى هرمون اللبتين في مصل مرضى التهاب اللثة المزمن

عبدالعزیز عمر الشويرف

الخلاصة:

تعتبر أمراض اللثة من المشاكل الصحية الرئيسية في مختلف السكان لفترة طويلة . وهي تمثل مجموعة من العدوي الموضعية التي تسببها الميكروبات والتي تشمل اللثة والانسجة الداعمة للأسنان. التهاب اللثة والتهاب دواعم الأسنان المزمن هما أكثر أنواع أمراض اللثة شيوعا . يتم التعرف على اللويحة البكتيرية كعامل مسبب رئيسي في بدء وتطور التهاب اللثة. يبدأ التهاب دواعم السن المزمن كمرض التهابي في اللثة والذي إذا ترك دون علاج بمرور الوقت قد يتطور الي إحداث تلف وانهيار لأجزاء أخرى من جهاز التعلق اللثوي، ومركبات النباتات الطبيعية لديها القدرة علي تعديل الاستجابة الالتهابية المناعية، والعوامل الطبيعية لها مضادات الميكروبات ومضادات الالتهابات قد تكون لها القدرة علي التحكم في الأمراض الالتهابية مثل التهاب اللثة.

الكلمات المفتاحية: هرمون اللبتين، انتر لوكين- 6، بكتيريا إشريشيا كولاي ، المورينقا، والتهاب اللثة.