Med. J. Cairo Univ., Vol. 93, No. 3, Accepted 23/3/2025
DOI: 10.22608/MJCU. 1019-1026, September 2025
www.medicaljournalofcairouniversity.net

A Review Article:
Effect of Bioptron Light Therapy on Primary Dysmenorrhea

EMAN S. EL-DEEP, M.Sc.*; DOAA A. OSMAN, Ph.D.*; HAZEM S. ASHMAWY, Ph.D.** and MANAL A. EL-SHAFEI, Ph.D.*

The Department of Physical Therapy for Woman's Health, Faculty of Physical Therapy, Cairo University and Department of Obstetrics & Gynecology**, Faculty of Medicine, Cairo University*

Abstract

Primary Dysmenorrhea (PD) remains a significant health concern, particularly among adolescent females, and is known to adversely affect daily activities and quality of life. There are many therapeutic interventions for management of such-patients. The aim of this study was to evaluate the effects of Bioptron light therapy (BLT) in the management of PD. It was found that the BLT has significant effects in reducing the painful symptoms and improving quality of life for women with PD. This highlights the potential of BLT as a beneficial therapeutic intervention for those patients. So, BLT can be recommended as an alternative therapy for relieving PD.

Key Words: *Bioptron light therapy – Primary Dysmenorrhea.*

Introduction

DYSMENORRHEA is one of the most common problems of adolescents and mature women, whose prevalence ranges from 16.8% to 81%, with rates as high as 90% having been reported **[1].** The prevalence in married women is generally lower than that in unmarried women **[2]**, the prevalence in young women aged 17–24 years has been investigated and ranges from 67% to 90% and begins to decline after the age of 25 years **[3].**

Dysmenorrhea has been shown to affect women's quality of life, reflected mainly in school and work performance, including lack of energy for daily activities, higher level of stress, absenteeism and decreased efficiency, sports participation, and socialization, which causes a great deal of lost productivity **[4].**

Correspondence to: Dr. Eman S. El-Deep, The Department of Physical Therapy for Woman's Health*, Faculty of Physical Therapy, Cairo University

As menstrual health problems cause consequences for women's lives, the World Health Organization (WHO) stated that menstrual health to be recognized, framed, and addressed as a health and human rights issue, every month, those women must experience the torture of menstrual pain, which is why finding the right solution to overcome it is necessary **[5].** There are two types of dysmenorrhea: Primary, which occurs without any underlying pelvic conditions, and secondary, often linked to pelvic disorders like endometriosis or adenomyosis **[6].** The underlying mechanisms of dysmenorrhea are believed to involve an increase in the production of substances like prostaglandins (PGD) and leukotrienes in the uterus, which can lead to heightened uterine contractions and reduced blood flow **[7].**

Primary Dysmenorrhea

Primary dysmenorrhea (PD) is defined as pain occurring with menses in the absence of pelvic pathology. It leads to workplace and school absences, reduces one's quality of life and general wellbeing, and is associated with high health and social-economic costs **[8].** It is characterized by pain during menstruation without organic lesion in pelvis, caused by increased endometrial prostaglandin production. A wide variety of symptoms during menstruation have been noted, including lower abdominal pain, diarrhea, cramps, nausea, vomiting, fatigue, headache, irritability and depressive mood **[9].**

Its first manifestation usually appears 6 months after menarche because it occurs only during ovulatory cycles. The pain typically lasts from 8 to 72 hours and is most severe on the 1^{st} and 2^{nd} days of menstruation. It is associated with nausea, vom-

iting, diarrhea, low back pain, migraines, dizziness, fatigue, insomnia, and rarely, syncope and hyperthermia [10]. According to previous research, PD is considered as the outcome of abnormal prostaglandin (PGs) release which leads to myometrial hypercontractility, as well as insufficient oxygen supply on uterus muscles, in addition to unhealthy lifestyle such as dietary habit and other negative factors also contribute to outbreak of PD [11].

Epidemiology

The prevalence of PD is highest in the 16–25-year age group but is greatly underestimated as many women consider pain a normal part of the menstrual cycle and do not seek medical treatment, despite the considerable distress they experience [12]. Its prevalence of PD among adolescent's rangesvaries from 45 to 95 percent globally however only 15 percent of females seek medical advice for menstrual pain [9].

Clinical Presentation

Regarding clinical presentation of PD, painingeneral hasa disabling nature and makes dysmenorrhea stress ful and it can become an important irritating factor in the life of many girls [13]. Usually associated with common symptoms, that can be categorized into two main dimensions: Physical and psychological symptoms. The commonly experienced physical symptoms are systemic, gastrointestinal, and elimination related. The systemic symptoms include headache, lethargy, fatigue, sleepiness/sleeplessness, tender breasts, heavy lower abdomen, backache, in addition to painful knees and inner thighs, myalgia, arthralgia, and swollen legs [14]. The gastrointestinal symptoms include an increase or decrease in appetite, nausea, vomiting, and bloating, while the elimination-related symptoms comprise constipation, diarrhea, frequent urination, and sweating [15].

Regarding the psychological symptoms, dysmenorrheic females may experience mood disturbances such as anxiety, depression, irritability, and nervousness, It was reported that depression, anxiety, and excess somatic symptoms were three-fold higher in females with dysmenorrheic pain [16]. The co-occurrence of dysmenorrhea along with psychological symptoms could suggest a neurological brain disorder that contributes to menstrual pain, whereas the hereditability of both PD and psychological symptoms could reflect a shared genetic variance. Some women (3 to 33%) have very severe pain, severe enough to render them incapacitated for 1 to 3 days each menstrual cycle, requiring absence from school or work [17].

Risk Factors

There are two types of risk factors for PD: non-modifiable and behavioral. Non-modifiable risk factors include: Family history of dysmenorrhea, age under 20 years (symptoms are more pronounced during adolescence), menarche before age 12 (due to early establishment of ovulatory cycles), menstrual flow lasting longer than 7 days and nulliparity [18]. Risk factors for PD include early age at menarche, heavy menstrual flow, nulliparity, family history of dysmenorrhea, and stress [19].

The association between multiparity and decreased risk of dysmenorrhea can be explained by several assumptions such as: Lower release of prostaglandins by the endometrium after term delivery, neuronal degeneration that occurs in the uterus after a term delivery and decreased uterine norepinephrine in the third trimester of pregnancy [3].

Behavioral risk factors include body mass index (BMI) <20 or >30, low intake of omega 3 (fish), smoking (nicotine induces vasoconstriction), caffeine consumption (also induces vasoconstriction), and psychosocial symptoms such as depression and anxiety. Also, a stressful relationship with parents may favor PD [20].

It is important to identify these behavioral factors as they are subject to intervention. Stress inhibits the release of luteinizing and follicle-stimulating hormones, which leads to impaired follicular development with alteration in progesterone synthesis and release that influences prostaglandin activity [21]. Besides, stress-related hormones, such as adrenaline and cortisol, also, influence prostaglandin synthesis and myometrial binding [3].

Some studies have shown an association between the occurrence of PD and other conditions that cause chronic pain, such as irritable bowel syndrome, migraines, and fibromyalgia. Women with PD are twice more likely to develop irritable bowel syndrome than women without dysmenorrhea. In addition, this condition may exacerbate symptoms of other diseases with the increased pain sensitivity [22]. A positive clinical correlation was found between vit. D deficiency (<12ng/mL) and dysmenorrheal pain scores as well as other menstrual symptoms of depression, fatigue, and headache in a study of Turkish women (sample size 683) [23].

Vitamin D has also been reported to reduce the production of prostaglandins. Many studies have reported a close relationship between Vit D deficiency and early dysmenorrhea due to the regulatory action of calciferol on prostaglandin levels [24]. Vit D re-

ceptors have been found in ovarian and endometrial tissue, and in epithelial cells of the fallopian tubes, decidua, and placenta [25].

Pathophysiology

While the pathophysiology of dysmenorrhea is not completely understood, current research suggests that prostaglandins play a major role [26].

During menses, endometrial sloughing releases prostaglandins, stimulating myometrial contraction and vasoconstriction. This subsequently causes uterine ischemia, anaerobic metabolite formation, and hypersensitization of nociceptive fibers. Interestingly, women with dysmenorrhea have higher levels of prostaglandin E2 and F2 in menstrual fluid, further supporting the role of prostaglandins in the pathophysiology of dysmenorrhea [27].

Prostaglandins are synthesized through the arachidonic acid cascade, mediated by the cyclooxygenase (COX) pathway. Arachidonic acid synthesis is regulated by the level of progesterone, through the activity of the lysosomal enzyme phospholipase A2. The progesterone level peaks during the middle of the luteal phase (the latter phase of the menstrual cycle) that occurs after ovulation. If conception does not occur, this results in degeneration of the corpus luteum and a decline in the circulating progesterone level. This rapid decline in the progesterone level is associated with endometrial sloughing, menstrual bleeding, and the release of lysosomal enzymes, leading to the generation of arachidonic acid, and therefore, the production of prostaglandins. A decrease in progesterone also contributes to the inflammatory response that leads to exfoliation of the endometrium and menstrual bleeding. The mechanisms that follow a decrease in progesterone concentration are complex reactions between the endocrine, vascular and immune systems [28].

Females with regular menstrual cycles have elevated endometrial prostaglandin levels during the late luteal phase. However, several studies that measured prostaglandin concentrations in the luteal phase, through endometrial biopsies and menstrual fluids, revealed that dysmenorrheic females have higher levels of prostaglandins than eumenorrheic females [28].

PD results from an increase in the production of prostanoids, in particular prostaglandin F2α (PGF2α), by stimulation of the cyclooxygenase pathway by phospholipase A2 secreted at the end of the luteal phase when the level of progesterone declines [29].

Studies have shown that metabolism and absorption of vitamins and minerals may play a major role in the development and treatment of menstrual disorders [30]. Primary dysmenorrhea is related to both insufficient calcium consumption and, hence, low blood calcium and low vit. D levels One 25(OH) D intervention altered calcium homeostasis, which modulates the pathogenesis of PD [31].

There is a significant relationship between increased parathormone levels and severity of dysmenorrhea. Some studies have suggested a physiological effect of calcium on muscle contraction and expansion, since calcium homeostasis is mediated by the functions of calcitonin, parathyroid hormone, and 25-hydroxy Vit D3, it can be expected that these 3 hormones may play a role in the pathophysiology of PD [32].

When serum Vit D levels are reduced, intestinal calcium absorption is significantly reduced. Thereafter, the calcium in the extracellular fluid decreases and the secretion of parathyroid hormone increases. In turn, the parathyroid hormone increases renal reabsorption of calcium and intestinal absorption of calcium and phosphate and hence, it appears that elevated parathyroid hormone levels may affect the severity of dysmenorrhea [33].

Low calcium absorption through food increases uterine cramps and pain in women with PD as calcium influx plays a role in the modulation of smooth muscle contraction/relaxation, low blood calcium levels lead to the contraction of the smooth uterine muscle [34].

As it is hormone-like, vit. D has receptors throughout the body, with its action mediated by insulin-like growth factor-1. Moreover, 1-α hydroxylase, an enzyme, is responsible for vit. D metabolism, and it is expressed in the myometrium; this points to the critical role of vit. D in dysmenorrhea and its related symptoms [24].

Additionally, other factors such as lysosomes, progesterone, arginine vasopressin, pro-inflammatory cytokines, and even menstrual bleeding itself have been described as possible contributors [35]. Moreover, some studies have focused on central neuroplastic changes in areas associated with pain processing [36].

Diagnosis

Primary dysmenorrhea may be diagnosed based on the typical history alone [37]. Pain begins just before or at the start of menstrual bleeding. It is cyclic and begins with the onset of ovulatory cycles, usually within 2 years of menarche. Pain peaks at 23 to

48 hours after the onset of bleeding and usually lasts no more than 72 hours [38]. The focused medical history to be obtained includes, but is not limited to, the following information: age at menarche; regularity and duration of menstrual bleeding; abnormal vaginal discharge; onset and duration of symptoms relevant to the age of menarche; menstrual cycle; location of pain; and associated systemic symptoms [10].

Furthermore, patients should be asked about their sexual activity and history of sexually transmitted diseases. Since females with typical symptoms of PD can be diagnosed solely based on their medical information, without any physical or pelvic examination, empiric treatment, including nonsteroidal anti-inflammatory drugs (NSAIDs) and/or oral contraceptives should be initiated [39]. A pelvic examination is important for evaluating dysmenorrhea if the history of onset and duration of lower abdominal pain suggests secondary dysmenorrhoea or if the dysmenorrhea is not responding to medical treatment [40].

The use of ultrasound in the evaluation of PD has little benefit. However, ultrasound can be useful in differentiating the cause of secondary dysmenorrhea, including endometriosis, leiomyomas, Mullerian anomalies, and adenomyosis. Patients who are at risk of sexually transmitted infections (STIs) or when pelvic inflammatory disease (PID) are suspected may need endocervical or vaginal swabs [41]. If indicated, cervical cytology samples and/or HPV testing may be considered to rule out a suspected cervical malignancy. Magnetic resonance imaging (MRI) or Doppler ultrasonography may be useful if torsion of the adnexa, adenomyosis, or deep pelvic endometriosis is suspected or if there are inconclusive findings on ultrasound. Laparoscopy is usually reserved for women who desire fertility and have suspected endometriosis as a cause of secondary dysmenorrhea [42].

Differential Diagnosis

Differential diagnosis of dysmenorrhea is broad, and it can be listed as gynaecological conditions and non-gynaecological conditions. Gynecological conditions (Secondary dysmenorrhea) include the following. Endometriosis that is characterized by the presence of endometrial tissue outside the uterus, endometriosis can cause severe, chronic pelvic pain and dysmenorrhea. Diagnostic laparoscopy is often required for confirmation [43]. Uterine fibroids that benign tumors can cause heavy menstrual bleeding and pain, particularly in the presence of submucosal fibroids [44]. Adenomyosis that is the invasion of endometrial tissue into the myometrium can lead to dysmenorrhea, often associated with a bulky, tender uterus on palpation [45]. Pelvic inflammatory disease which is an inflammation of the reproductive organs can result in pain that mimics dysmenorrhea, especially in the context of recent sexual activity or a history of sexually transmitted infections [46].

Bioptron Light Therapy

Phototherapy is a relatively recent, noninvasive, pain-free, and low-cost interventional treatment method for numerous disorders. Polarized phototherapy includes low-level laser therapy and bioptron light therapy (BLT). The BLT differs from laser light, in that BLT is a polychromic, incoherent, and low-energy light. The light emitted by BLT is characterized as polarized (its waves oscillate on parallel planes), polychromatic (wavelength: 480–3400 nm), incoherent (out of phase light, unlike laser light) and low energy light. Polychromacy means bioptron contains not only one wavelength (like laser light) but a wide range, including visible light and a portion of the infrared range. Unlike laser light, Bioptron Light is incoherent, or out–of phase light. This means that the light waves are not synchronized. Bioptron Light has low energy density. This energy density has bio–stimulative effects [47,48].

Bioptron light therapy produces polarized light where the light waves pass in a parallel plane, generating a narrow-focused beam. Unlike conventional light, where the waves oscillate in all directions, polarized light refracts standard light through specific laminated mirrors to be transmitted using a photo filter system. The efficacy of this new light therapy is dependent on several parameters that can significantly influence treatment outcomes [49].

The polarized light released has a broad wavelength range (480–3400 nm); this spectrum includes visible light and a part of infrared radiation but excludes ultraviolet radiation [48]. The specific power density for Bioptron light is about 40 mW/cm^2 for treatment at 10 cm; this is equivalent to an energy density average of 2.4 J/cm^2. These characteristics of Bioptron light enable it to penetrate the skin's surface with minimal heating, no skin injury, and no known negative effects [50]. The most common indication for utilizing polarized light therapy is tissue repair [51].

The biological effects of polarized light are well known. It increases the activities of the cell membrane, accelerates the mitochondrial synthesis of adenosine triphosphate, restores normal cell membrane potential that was impaired, and activates

regenerative mechanisms. In addition, it speeds up fibroblast proliferation and collagen deposition **[52].**

Bioptron light therapy efficacy as either a monotherapy or an adjunct treatment for pain management in several indications, including orthopedic physical therapy as osteoarthritis, rheumatoid arthritis, and conditions such as low back pain (LBP), shoulder and neck pain syndrome, and issues related to scar and muscle tissues **[53]**. This pain reduction could potentially be attributed to the enhancement of local blood circulation and improved tissue oxygenation within the affected region **[54].** Moreover, physiotherapy with polarized polychromatic non-coherent low-energy light can alleviate pain, increase mobility in the lower back, and enhance the quality of life of patients with LBP **[55].**

Stasinopoulos et al. **[56]** stated that patients suffering from acute ankle sprains who had cryotherapy along with BLT therapy for five days experienced statistically significant reductions in pain intensity, edema, as well as ankle range of motion.

Bioptron can be used to relief pain and inflammation of various musculoskeletal disorder, light with rated power of halogen = 90 W; light wavelength = 480–3400 nm; degree of polarization = 95%; specific power density = 40mW/cm^2; and energy density = 2.4 J/cm^2 led to significant functional and pain improvement in patients with lateral epicondylitis **[57].**

Polarized light in the red as well as near-infrared rays may provide a feeling of warmth in the treated region. A direct mechanism on the free nerve endings or nerve trunk which supplies the affected area triggers the production of histamine and prostaglandins, which in turn increase vasodilation, adjust enzyme activity as well as metabolic rate, and increase pain threshold **[58].**

It was found that 630 nm LED modulate inflammatory process and increase the vascularity **[59]**. However, despite its long–standing presence in the medical field, the clinical application of PBM has remained relatively limited. One of the primary obstacles lies in achieving an adequate irradiance of light for effective PBM therapy, that is, the balance between effectiveness and the tolerance of human epidermal tissue to temperature **[60].**

A previous study demonstrated the effect of Pulsed 630 nm LED Photobiomodulation Therapy for Anti–Primary Dysmenorrhea. The pulse light parameters were designed according to the transmittance of red light. 46 women with PD were included and randomly assigned to either pulsed 630 nm light therapy or white light sham control therapy. The intervention lasted for 20 min per day and was administered for 7 consecutive days before and during menstruation. The differences in pain intensity and global assessment of pain relief evaluated through pain relief scores indicated that PBM was significantly superior to white light placebo. Moreover, participants reported an improved their quality of life during the active treatment phase and generally preferred it as a more effective method for relieving PD **[61].**

Conclusion:

The BLT has significant effects in reducing the painful symptoms and improving quality of life for women with PD. This highlights the potential of BLT as a beneficial therapeutic intervention for those patients. So, BLT can be recommended as an alternative therapy for relieving PD.

References

1- GUTMAN G., NUNEZ A.T. and FISHER M.: Dysmenorrhea in adolescents. Current Problems in Pediatric and Adolescent Health Care, May 1; 52 (5): 101186, 2022.

2- HU Z., TANG L., CHEN L., KAMINGA A.C. and XU H.: Prevalence and risk factors associated with primary dysmenorrhea among Chinese female university students: A cross-sectional study. Journal of pediatric and adolescent gynecology, Feb 1; 33 (1): 15-22, 2020.

3- JU H., JONES M. and MISHRA G.: The prevalence and risk factors of dysmenorrhea. Epidemiologic reviews, Jan 1; 36 (1): 104-13, 2014.

4- SCHOEP M.E., ADANG E.M., MAAS J.W., DE BIE B., AARTS J.W. and NIEBOER T.E.: Productivity loss due to menstruation-related symptoms: A nationwide cross-sectional survey among 32 748 women. BMJ open, Jun 1; 9 (6): e026186, 2019.

5- NUHA K., RUSMIL K., GANIEM A.R., PERMADI W. and DIAHHERAWATI D.M.: Single-Blind Randomized Controlled Trial: Comparative Efficacy of Dark Chocolate, Coconut Water, and Ibuprofen in Managing Primary Dysmenorrhea. International journal of environmental research and public health, Aug 21; 20 (16): 6619, 2023.

6- ABDI F., AMJADI M.A., ZAHERI F. and RAHNEMAEI F.A.: Role of vitamin D and calcium in the relief of primary dysmenorrhea: A systematic review. Obstetrics & Gynecology science. Jan 7; 64 (1): 13-26, 2021.

7- DONAYEVA A., AMANZHOLKYZY A., NURGALIYEVA R., GUBASHEVA G., ABDELAZIM I. and SAMAHA I.I.: The relation between primary dysmenorrhea in adolescents and body mass index. Menopause Review/Przegląd-Menopauzalny. Sep 20; 22 (3): 126-9, 2023.

8- ÇELIK A.S. and APAY S.E.: Effect of progressive relaxation exercises on primary dysmenorrhea in Turkish students: A randomized prospective controlled trial. Complementary Therapies in Clinical Practice, Feb 1; 42: 101280, 2021.

9- VLACHOU E., OWENS D.A., LAVDANITI M., KALEMIKERAKIS J., EVAGELOU E., MARGARI N., FASOI G., EVANGELIDOU E., GOVINA O. and TSARTSALIS A.N.: Prevalence, wellbeing, and symptoms of dysmenorrhea among university nursing students in Greece. Diseases, Jan 8; 7 (1): 5, 2019.

10- ACOG committee opinion no. 760. Dysmenorrhea and endometriosis in adolescents. Obstet. Gynecol., 132 (69), 2248, 2018.

11- IWATA M., OIKAWA Y., SHIMIZU Y., SAKASHITA N., SHOJI A., IGARASHI A. and OSUGA Y.: Efficacy of Low-Dose Estrogen–Progestins and Progestins in Japanese Women with Dysmenorrhea: A Systematic Review and Network Meta-analysis. Advances in Therapy, Nov. 39 (11): 4892-909, 2022.

12- RAMOS-PICHARDO J.D., ORTEGA-GALÁN Á.M., IGLESIAS-LÓPEZ M.T., ABREU-SÁNCHEZ A. and FERNÁNDEZ-MARTÍNEZ E.: Why do some Spanish nursing students with menstrual pain fail to consult healthcare professionals?. International Journal of Environmental Research and Public Health, Nov. 17 (21): 8173, 2020.

13- PITANGUI A.C., GOMES M.R., LIMA A.S., SCHWINGEL P.A., ALBUQUERQUE A.P. and DE ARAÚJO R.C.: Menstruation disturbances: Prevalence, characteristics, and effects on the activities of daily living among adolescent girls from Brazil. Journal of pediatric and adolescent gynecology, Jun 1; 26 (3): 148-52, 2013.

14- CALIS K.A., DANG D.K., KALANTARIDOU S.N. and EROGUL M.: Dysmenorrhea: Practice essentials, background, pathophysiology, 2019.

15- ITANI R., SOUBRA L., KAROUT S., RAHME D., KAROUT L. and KHOJAH H.M.: Primary dysmenorrhea: Pathophysiology, diagnosis, and treatment updates. Korean journal of family medicine, Mar 17; 43 (2): 101, 2022.

16- MATTHEWMAN G., LEE A., KAUR J.G. and DALEY A.J.: Physical activity for primary dysmenorrhea: A systematic review and meta-analysis of randomized controlled trials. American journal of obstetrics and gynecology, Sep 1; 219 (3): 255-e1, 2018.

17- ZANNONI L., GIORGI M., SPAGNOLO E., MONTANARI G., VILLA G. and SERACCHIOLI R.: Dysmenorrhea, absenteeism from school, and symptoms suspicious for endometriosis in adolescents. Journal of pediatric and adolescent gynecology, Oct 1; 27 (5): 258-65, 2014.

18- YU A.: Complementary and alternative treatments for primary dysmenorrhea in adolescents. The Nurse Practitioner, Nov 16; 39 (11): 1-2, 2014.

19- AKSHATHA K. and SHILPA D.: Dysmenorrhoea. Obst. Gynaecol. Reprod Med., 29: 286-91, 2019.

20- XU K., CHEN L., FU L., XU S., FAN H., GAO Q., XU Y. and WANG W.: Stressful parental-bonding exaggerates the functional and emotional disturbances of primary dysmenorrhea. International journal of behavioral medicine, Aug. 23: 458-63, 2016.

21. RYAN S.A.: The Treatment of Dysmenorrhea. Pediatric Clinics of North America, Apr 1; 64 (2): 331-42, 2017.

22- CHEN C.X., KWEKKEBOOM K.L. and WARD S.E.: Self-report pain and symptom measures for primary dysmenorrhoea: A critical review. European Journal of Pain, Mar. 19 (3): 377-91. 2015.

23- KARACIN O., MUTLU I., KOSE M., CELIK F., KANAT-PEKTAS M. and YILMAZER M.: Serum vitamin D concentrations in young Turkish women with primary dysmenorrhea: A randomized controlled study. Taiwanese Journal of Obstetrics and Gynecology, Feb 1; 57 (1): 58-63, 2018.

24- AMZAJERDI A., KESHAVARZ M., GHORBALI E., PEZARO S. and SARVI F.: The effect of vitamin D on the severity of dysmenorrhea and menstrual blood loss: A randomized clinical trial. BMC women's health, Mar. 27; 23 (1): 138, 2023.

25- MATSAS A., SACHINIDIS A., LAMPRINOU M., STAMOULA E. and CHRISTOPOULOS P.: Vitamin effects in primary dysmenorrhea. Life, Jun 1; 13 (6): 1308, 2023.

26- GUIMARÃES I. and PÓVOA A.M.: Primary dysmenorrhea: Assessment and treatment. RevistaBrasileira de Ginecologia e Obstetrícia, Sep 25; 42: 501-7, 2020.

27- KIRSCH E., RAHMAN S., KEROLUS K., HASAN R., KOWALSKA D.B., DESAI A. and BERGESE S.D.: Dysmenorrhea, a narrative review of therapeutic options. Journal of Pain Research, Dec. 31: 2657-66, 2024.

28- BARCIKOWSKA Z., RAJKOWSKA-LABON E., GRZYBOWSKA M.E., HANSDORFER-KORZON R. and ZORENA K.: Inflammatory markers in dysmenorrhea and therapeutic options. International journal of environmental research and public health, Feb. 17 (4): 1191, 2020.

29- GUY M., FOUCHER C., JUHEL C., RIGAUDIER F., MAYEUX G. and LEVESQUE A.: Transcutaneous electrical neurostimulation relieves primary dysmenorrhea: A randomized, double-blind clinical study versus placebo. Progrèsenurologie, Jul 1; 32 (7): 487-97, 2022.

30- ABDI F., AMJADI M.A., ZAHERI F. and RAHNEMAEI F.A.: Role of vitamin D and calcium in the relief of primary dysmenorrhea: A systematic review. Obstetrics & Gynecology science, Jan 7; 64 (1): 13-26, 2021.

31- FLEET J.C.: Vitamin D-mediated regulation of intestinal calcium absorption. Nutrients, Aug 16; 14 (16): 3351, 2022.

32- NAZ M.S., KIANI Z., FAKARI F.R., GHASEMI V., ABED M. and OZGOLI G.: The effect of micronutrients on pain management of primary dysmenorrhea: A systematic review and meta-analysis. Journal of caring sciences, Mar 1; 9 (1): 47, 2020.

33- ALKHATATBEH M.J., KHWAILEH H.N. and ABDUL-RAZZAK K.K.: High prevalence of low dairy calcium intake and association with insomnia, anxiety, depression and musculoskeletal pain in university students from Jordan. Public health nutrition, May 24 (7): 1778-86, 2021.

34- CHIANG Y.F., HUNG H.C., CHEN H.Y., HUANG K.C., LIN P.H., CHANG J.Y., HUANG T.C. and HSIA S.M.: The inhibitory effect of extra virgin olive oil and its active compound oleocanthal on prostaglandin-induced uterine hypercontraction and pain—ex vivo and in vivo study. Nutrients, Sep 30; 12 (10): 3012, 2020.

35- IACOVIDES S., AVIDON I. and BAKER F.C.: What we know about primary dysmenorrhea today: A critical review. Human reproduction update, Nov 1; 21 (6): 762-78, 2015.

36- LOW I., WEI S.Y., LEE P.S., LI W.C., LEE L.C., HSIEH J.C. and CHEN L.F.: Neuroimaging studies of primary dysmenorrhea. Advances in pain research: Mechanisms and modulation of chronic pain, 179-99, 2018.

37- KHO K.A. and SHIELDS J.K.: Diagnosis and management of primary dysmenorrhea. JAMA, Jan 21; 323 (3): 268-9, 2020.

38- FERRIES-ROWE E., COREY E. and ARCHER J.S.: Primary dysmenorrhea: Diagnosis and therapy. Obstetrics & Gynecology, Nov 1; 136 (5): 1047-58, 2020.

39- BURNETT M. and LEMYRE M.: No. 345-primary dysmenorrhea consensus guideline. Journal of Obstetrics and Gynaecology Canada, Jul 1; 39 (7): 585-95, 2017.

40- OSAYANDE A.S. and MEHULIC S.: Diagnosis and initial management of dysmenorrhea. American family physician, Mar 1; 89 (5): 341-6, 2014.

41- NAGY H., CARLSON K. and KHAN M.A.B.: Dysmenorrhea. [Updated 2023 Nov 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing, 2025.

42- ZHAO Y., WANG Y. and LI Y.: Advances in the diagnosis of endometriosis. Journal of Reproductive Medicine, 59 (1), 12-20, 2024.

43- HUANG X., ZHANG J. and LIU S.: Endometriosis: Diagnosis and management update. Obstetrics and Gynecology Reviews, 22 (3): 45-56, 2024.

44- MILLER P. and JONES M.: Secondary Dysmenorrhea: Beyond the Basics. Obstetrics & Gynecology Clinics of North America, 48 (3): 475-487, 2021.

45- AHN H., KIM M. and PARK H.: Endometriosis and Other Pathological Causes of Dysmenorrhea: Diagnostic Approach. International Journal of Women's Health, 14: 153-160, 2022.

46- CIBILS L. and MASTRONARDI A.: Primary Dysmenorrhea: New Insights into Pathophysiology and Management. Journal of Obstetrics and Gynecology Research, 49 (6): 1621-1629, 2023.

47- NOBUTA S., SATO K., NAKAGAWA T., HATORI M. and ITOI E.: Effects of wrist splinting for carpal tunnel syndrome and motor nerve conduction measurements. Upsala journal of medical sciences, Jan 1; 113 (2): 181-92, 2008.

48- REDDY M., GILL S.S., KALKAR S.R., WU W., ANDERSON P.J. and ROCHON P.A.: Treatment of pressure ulcers: A systematic review. JAMA, Dec 10; 300 (22): 2647-62, 2008.

49- HAMBLIN M.R., NELSON S.T. and STRAHAN J.R.: Photobiomodulation and cancer: What is the truth?. Photomedicine and laser surgery, May 1; 36 (5): 241-5, 2018.

50- BEGIC-RAHIC J. and VRANIC S.: The application of bioptron light therapy in dermatology and wound healing. Eur. Dermatol., 5: 57–60, 2010.

51- MIHAJLOVIĆ V.: Physical Therapy. Rijeka Crnojevića: ObodskoSlovo, 2002. [Serbian].

52. MEDRADO A.R., PUGLIESE L.S., REIS S.R. and ANDRADE Z.A.: Influence of low level laser therapy on wound healing and its biological action upon myofibroblasts. Lasers Surg Med., 32 (3): 239-44, 2003.

53- NICOLAOU V., STASINOPOULOS D. and LAMNISOS D.: The Effectiveness of Polarized Light in Musculoskeletal, Skin Problems and Burns. American Journal of Biomedical Science & Research, 10 (2): 159-67, 2020.

54- CHAUDARY S., KARNER L., WEIDINGER A., MEIXNER B., RIEGER S., METZGER M., ZIPPERLE J. and DUNGEL P.: In vitro effects of 635 nm photobiomodulation under hypoxia/reoxygenation culture conditions. Journal of Photochemistry and Photobiology B: Biology, Aug 1; 209: 111935, 2020.

55- MIHAYLOVA M., RUSEVA Z. and FILKOVA S.: The effect of polarized polychromatic non-coherent light (Bioptron) therapy on patients with lower back pain. ScriptaScientificaSalutisPublicae, Apr 13; 3 (1): 23-7, 2017.

56- STASINOPOULOS D., PAPADOPOULOS C., LAMNISOS D. and STASINOPOULOS I.: The use of Bioptron light (polarized, polychromatic, non-coherent) therapy for the treatment of acute ankle sprains. Disability and rehabilitation, Feb 27; 39 (5): 450-7, 2017.

57- STASINOPOULOS D., STASINOPOULOS I. and JOHNSON M.I.: Treatment of carpal tunnel syndrome with polarized polychromatic noncoherent light (Bioptron light): A preliminary, prospective, open clinical trial. Photomedicine and Laser Therapy, Apr 1; 23 (2): 225-8, 2005.

58- SHIRYAN G.T., AMIN F.S. and EMBABY E.A.: Effectiveness of polarized polychromatic light therapy on myofascial trigger points in chronic non-specific low back pain: A single blinded randomized controlled trial. Bulletin of Faculty of Physical Therapy, Dec. 27 (1): 33, 2022.

59- BUSANELLO-COSTA M., RENNO A.C., DE GOES SANTOS C.P., QUINTANA H.T., MARTIGNAGO C.C., TIM C.R. and ASSIS L.: Red LED light therapy associated with epidermal growth factor on wound repair process in rats. Lasers in Medical Science, Jan 10;38 (1): 36, 2023.

60- BAROLET A.C., LITVINOV I.V. and BAROLET D.: Light-induced nitric oxide release in the skin beyond UVA and blue light: Red & near-infrared wavelengths. Nitric Oxide, Dec 1; 117: 16-25, 2021.

61- FU Q., JIANG H., YANG J., LI Y., FEI H., HUANG J., LI Y. and LIU M.: Bypassing the Heat Risk and Efficacy limitations of pulsed 630 nm LED photobiomodulation therapy for Anti-primary Dysmenorrhea: A prospective Randomized Cross-over Trial. InPhotonics, Jan 31 (Vol. 11, No. 2, p. 136), 2024. MDPI.

تأثيرالبيوبترون علي عسرالطمث الأولى: مقالة مرجعية

لايزال عسرالطمث الأولى يُشكل مصدر قلق صحى كبير، لاسيما بين المراهقات ،ومن المعروف أنه يؤثرسلبًا على الأنشطة اليومية وجودة الحياة لديهن. هدفت هذه الدراسة إلى تقييم آثار العلاج بالضوء البيوبتروني فى علاج عسر الطمث الأولى. وقد وُجد أن العلاج بالضوء البيوبترونى يُظهر تأثيرات ملحوظة فى تخفيف الأعراض المؤلمة وتحسين جودة الحياة لدى النساء المصابات بعسرالطمث الأولى. وهذا يُبرز إمكانات العلاج بالضوء البيوبترونى كتدخل علاجى مفيد لهؤلاء المريضات. لذا، يُمكن التوصية بالعلاج بالضوء البيوبترونى كعلاج بديل لتخفيف عسر الطمث الأولى.