Fibromyalgia Syndrome: It's more than just a mere syndrome

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Abstract
Fibromyalgia Syndrome (FMS) is a complex, chronic condition characterized by widespread musculoskeletal pain, fatigue, sleep disturbances, cognitive difficulties, and a variety of other symptoms. Despite being one of the most common chronic pain conditions, affecting millions worldwide, FMS remains poorly understood and frequently misdiagnosed. This paper aims to delve deeper into the multifaceted nature of fibromyalgia, challenging the perception that it is merely a syndrome. The pathophysiology of FMS involves a combination of genetic, neurobiological, and environmental factors. Central sensitization, where the central nervous system becomes hypersensitive to pain stimuli, plays a crucial role. Additionally, imbalances in neurotransmitters, such as serotonin and norepinephrine, contribute to the heightened pain perception and emotional symptoms observed in FMS patients. The syndrome also often coexists with other conditions like irritable bowel syndrome, migraine, and temporomandibular joint disorders, further complicating its diagnosis and management. Traditional diagnostic criteria for FMS, based primarily on tender points and symptom reports, have evolved. Understanding the underlying mechanisms and interactions within the neuroendocrine-immune system is crucial for developing more effective interventions. This paper underscores the necessity of recognizing fibromyalgia as more than just a syndrome. It is a multifactorial condition with profound implications for patients' physical, emotional, and social well-being. Enhanced awareness, comprehensive diagnostic criteria, and integrated treatment strategies are essential for improving the quality of life for patients with FMS.

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Introduction

The symptom of Fibromyalgia Syndrome (FMS) is chronic musculoskeletal pain, include low pain threshold, headache, anxiety, depression, exhaustion, and pain in many tender points in the morning [1]. Incidences of FMS range from 0.2 to 6.8% in women and from 2.4 to 6.8% in the general population [2]. Research indicates that women are nine times more likely than men to have FMS [3]. FMS refers to pain in the muscles and connective tissues. FMS is thought to be central-pain-processing condition which causes allodynia, or pain responses to non-painful response, as well as hyperalgesia, or heightened responses to painful stimuli. Therefore, rise in nociceptive neurotransmitters such glutamate and substance P may be the cause of the increase of transmission of pain. Debilitating fatigue, headaches, sleep disturbances, anxiety, melancholy, cold hands and feet, chemical sensitivities, decreased immunological function, and stiff joints are some of the other symptoms of familial multiple sclerosis.

FMS patients experience swallowing difficulties, irregularities in the bowel and bladder, tingling, numbness, and cognitive impairment (Figure 1). FMS impact the quality of life of the patients, making difficult to work and engage in daily activity. It can also negatively impact their relationships with friends, family, and employers. It places significant financial strain on both the sufferer and society. As of yet, FMS has no known treatment. Controlled clinical trials have demonstrated the efficacy of several treatments in mitigating symptoms, including as medication, behavioural therapy, educating patients, and physical exercise [4].

According to the American College of Rheumatology (ACR), definitional criteria for FMS in 1990, include having at least 11 tender sites out of a potential 18 and a history of discomfort lasting longer than three months. Patients with FMS experience widespread pain that affects all four quadrants of the body, including the right and left sides, and the area above and below the waist. There is no recognized cause of FMS. Nonetheless, a multitude of reasons may be accountable for the pathophysiology of familial myopathy, including the disruption of the hypothalamus-pituitary-adrenal axis (HPA). Low serotonin levels, sleep issues, cytokine malfunction, endocrine problems, and stress. The exact cause of FMS is unknown. Numerous factors, including problems with the neurological and autonomic nerve systems, psychological diseases, genetics, and the environment, are thought to contribute to FMS [5, 6]. FMS is a hereditary tendency, as evidenced by the presence of genetic polymorphism and familial aggregation. Certain genes that aided in analysing the amplitude of particular genetic variations were present in both healthy controls and FMS patients [7]. We discuss the latest FMS update in this review paper.

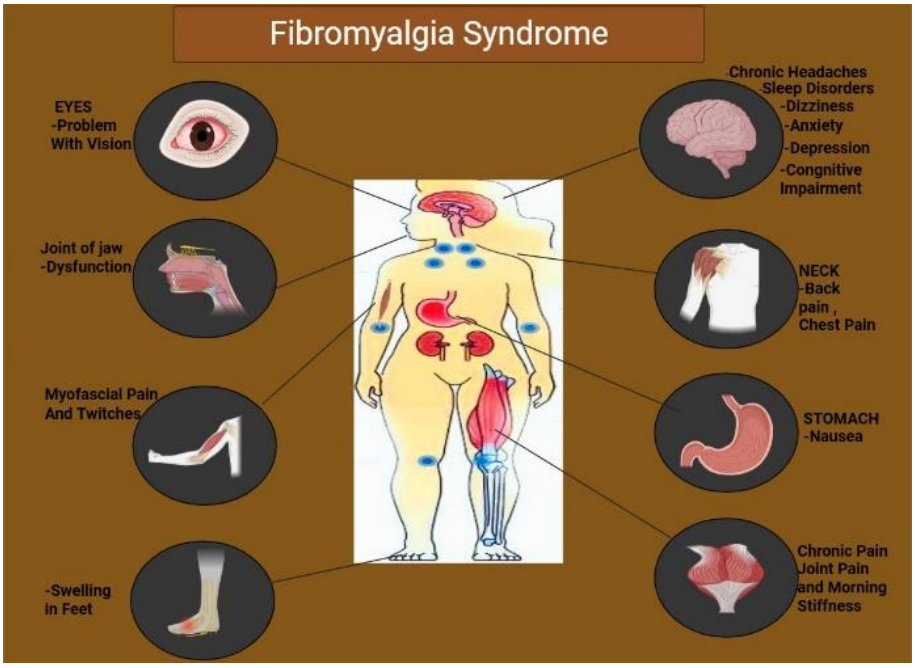

Fig 1: Symptoms of Fibromyagia Syndrome

Biomarkers: Possible future in Fibromyalgia diagnosis

FMS presents a significant diagnostic challenge due to the absence of universal diagnostic criteria. Digital pathology offers powerful tools for automated image analysis and pattern recognition. FMS is characterized by varying symptoms such as diffuse pain and cognitive impairments, are highly variable, digital tools could help to standardize the recognition of related tissue or neurological changes, leading to faster and more accurate diagnoses. With digital pathology, more objective, quantitative data can be derived, allowing for more precise analysis of tissue samples. This might be particularly useful in identifying potential biomarkers of FMS [8]. The complexity of FMS is underscored by its multifactorial nature, involving a combination of genetic predispositions and environmental factors such as trauma, stress responses, and sleep disturbances. These factors may contribute to the development and progression of the syndrome, as evidenced by numerous studies. Recent advancements in understanding the pathophysiology of FMS hold promise for transforming its diagnosis from a predominantly subjective process to a more objective. Identifying reliable biomarkers could play a crucial role in this transition, despite practical limitations such as the lack of specificity and the high costs associated with their implementation. These biomarkers can facilitate the identification of related conditions, therefore enhancing the diagnostic accuracy, even if their direct therapeutic applications remain limited. Genome studies have revealed that first-degree relatives of patients with FMS have a 13.6-fold increased risk of developing the condition [9]. This genetic linkage is further supported by the observation that family members of FMS patients often exhibit the pain-related conditions, such as irritable bowel syndrome and headaches. These findings underscore the potential role of genetic variations in influencing pain response mechanisms [10]. Research has identified several genes that could serve as reliable markers for FMS diagnosis. Polymorphisms in genes associated with the dopaminergic, serotoninergic, vitamin D receptor, and catecholaminergic systems have been implicated in the etiology of FMS. Specifically, variations in these genetic pathways may influence neurotransmitter activity, pain perception, and stress response, contributing to the development and severity of FMS symptoms [11-13]. Polymorphisms in genes related to serotonin receptors and

transporters may affect the FMS patient's response to pain [11]. Similarly, the serotoninergic system, which regulates mood, sleep, and pain perception, has linked to FMS through genetic variations that influence serotonin levels and receptor function [3]. Additionally, the vitamin D receptor gene has emerged as a potential factor in FMS pathophysiology. Vitamin D is crucial for musculoskeletal health, and polymorphisms in its receptor gene may impact pain sensitivity and immune function [5]. The catecholaminergic system, which involves neurotransmitters like norepinephrine and epinephrine, also plays a role in stress response and pain modulation. Variations in genes may affect the ability of the body to manage stress and pain, contributing to FMS symptoms [13].

Epigenetic Outlook

Previous studies have highlighted the significant impact of environmental factors and early life experiences on phenotypic and genome function through epigenetics, without altering the DNA sequence [14]. Epigenetic mechanisms, such as histone modifications, methylation changes, and the production of micro-ribonucleic acids (miRNAs), have been shown to mediate long-lasting changes in both the central and peripheral nervous systems [15]. Histone modifications can alter chromatin structure, thereby influencing gene expression. Methylation changes, particularly in DNA, can silence or activate genes, playing a crucial role in gene regulation. Additionally, miRNAs can regulate gene expression post-transcriptionally, affecting various cellular processes. These epigenetic modifications can be induced by various environmental factors; including stress, diet, and exposure to toxins, and can have lasting effects on the patients' health and disease susceptibility. Understanding these mechanisms provides valuable insights into how external factors can influence gene function and contribute to conditions such as fibromyalgia, where both genetic and environmental factors play a role. This knowledge opens avenues for potential therapeutic strategies targeting epigenetic modifications to manage or prevent such conditions.

Deoxyribonucleic Acid (DNA) Methylation

A genome-wide methylation pattern is the main focus of the first study considering into epigenetic modifications in FMS women. Ninety-one percent of the 69 differentially methylated sites that were identified in the study between the patients and controls were responsible for the higher frequency of micronuclei in FMS women [16]. The research identified the genes that were mapped on differentially methylated regions, suggesting potential roles for cell signalling pathways, skeletal and organ system development, and nervous system development in FMS [17]. Increased pain thresholds were associated with a substantial link between gate pain-related responses and low levels of DNA methylation in the promoter area of increased TRPA1 gene expression, especially in peripheral nociceptors [18]. In a different study, cortical excitability measures measuring both hemispheres revealed a different degree of methylation in the peripheral blood of FMS patients [19]. Based on encouraging findings from multiple research projects, DNA methylation may become a more useful biological marker for FMS diagnosis.

miRNAs

miRNAs, controls the levels of genes expression and essential modulators of processes for the establishment and maintenance of pain [20]. Nine miRNAs in cerebrospinal fluid of the brain and eight miRNAs in serum sample were differentially expressed in FMS cases according to a miRNA profile by Bjersing *et al*. [21, 22]. Masotti *et al*. performed the miRNA profiles in the serum and saliva of FMS patients and reported that six miRNAs were identified as connected to FMS [23]. To demonstrate the role of miRNA in FMS pathogenesis and facilitate diagnosis, more research is needed to validate these first results in larger cohorts [9].

Gene Expression

In the peripheral blood of 70 FMS patients and 70 healthy controls, Jones *et al*. performed a genome-wide expression profile. Of these, 421 genes were found to be differently expressed, many of which were connected to pathways involved in the processing of pain. The test has to be validated in larger patient populations to confirm its 95% sensitivity and 96% specificity for FMS in a subset of 10 probe sets [24]. Two of the 298 long non-coding ribonucleic acids (lncRNAs) that were recognised by Dolcino *et al*. identified in their recent investigation on the gene expression profiles in peripheral blood mononuclear cells from 10 patients and 10 healthy participants were found to target the most frequently occurring genes in FMS. These studies highlight the contribution of autoimmunity, epigenetics, and genetics on FMS pathophysiology and future diagnosis [25]. Biotechnology can contribute to better management and potential treatments in several ways such as gene therapy, biologic drugs, personalized medicine and biomarkers [26].

Oxidative stress in FMS

Oxidative stress is a significant factor in the pathology of FMS [27]. Heavy metals, such as mercury, lead, and cadmium, induce the production of free radicals, which cause oxidative stress. Toxic metals like mercury and lead can trigger the activation of microglial cells, which are immune cells in the brain. Once activated, microglia release pro-inflammatory cytokines that contribute to pain hypersensitivity and cognitive dysfunction [28]. Oxidative stress arises when there is an imbalance between the production of reactive oxygen species (ROS) and the ability of body to neutralize these free radicals with antioxidants. This imbalance leads to cellular damage and inflammation, which are central to the symptoms of FMS. In FMS, oxidative stress contributes to the pathophysiology by promoting inflammation and altering pain perception. Elevated levels of ROS can damage cellular structures such as lipids, proteins, and DNA, exacerbating inflammation and potentially leading to an increase in pain sensitivity. This damage may also impair cellular function and energy production, contributing to the fatigue and muscle pain commonly experienced by FMS patients. Several studies have indicated that FMS patients exhibit increased oxidative stress markers and decreased antioxidant levels compared to healthy controls. The role of oxidative stress in FMS may also be linked to mitochondrial dysfunction, which is involved in energy production and can further exacerbate fatigue and pain [27]. Management strategies for oxidative stress in FMS often include dietary interventions rich in antioxidants, such as vitamins C and E, and lifestyle changes that minimize

oxidative damage, such as reducing exposure to environmental toxins and engaging in regular physical activity. Additionally, targeted treatments to address mitochondrial dysfunction and enhance antioxidant defences may provide relief and improve overall symptom management [29]. Peptides with antioxidant properties, such as those from cyanobacteria, may help to reduce oxidative stress [30]. Understanding and mitigating oxidative stress could be crucial in developing more effective therapies for FMS

Circadian Rhythm and FMS

FMS is characterized by widespread musculoskeletal pain, fatigue, and sleep disturbances. Research has highlighted that circadian rhythm disruptions are significant in FMS patients. The circadian rhythm is the body's internal clock which regulates sleep-wake cycles and various physiological processes over a 24-hour period. In FMS, this rhythm can be impaired, leading to poor sleep quality, altered hormone levels, and exacerbated symptoms. Studies suggest that FMS patients often experience irregularities in their sleep-wake patterns, including difficulty falling asleep, frequent awakenings, and non-restorative sleep [31]. This disruption can further aggravate pain sensitivity and fatigue. Additionally, disturbances in circadian rhythm may affect the release of melatonin, hormone that regulates sleep and pain-modulating properties. Strategies may include establishing a regular sleep schedule, exposure to natural light, and maintaining sleep routine. Pharmacological treatments, like melatonin supplements, can also be considered. Understanding and managing these circadian rhythm disruptions are crucial for alleviating some of the symptoms associated with FMS and improving overall quality of life for those affected [32]. Disruptions in circadian rhythms is caused by shift work, jet lag, or genetic mutations may increase the risk of cardiovascular diseases, including MI. Altered expression or dysfunction of circadian clock genes has associated with higher risks of heart disease, as these genes regulate the timing of inflammation, oxidative stress, and vascular function [33]. The PER3 gene as a potential predictive marker for FMS pathogenesis and underscores the importance of genetic factors in disease assessment and management [31].

Postacute Coronavirus (COVID-19) Syndrome (Long COVID-19) and FMS

COVID-19 induces oxidative stress as part of the body response to inflammation. The virus triggers immune cells to produce ROS, which can damage tissues and organs, contributing to the severity of symptoms, especially in severe cases [34]. Recent studies and clinical observations suggest that there may be an overlap between PACS and FMS. Both conditions involve chronic pain and fatigue, and some individuals with PACS have reported developing symptoms that closely resemble FMS after recovering from COVID-19 [35]. Some researchers have reported that serum cortisol concentration indicates the severity of the underlying condition [36]. Corticosteroids or other immune-modulating medications may increase the risk of mucormycosis. FM patients recovering from COVID-19 should regularly monitor their health, as stress, inflammation, and medications can all have an impact on symptom management. Similarly, patients who develop signs of mucormycosis (such as nasal pain, swelling, blackened tissue, or difficulty breathing) following a COVID-19 infection should seek rapid medical assistance [37]. Many people with Long COVID develop chronic respiratory symptoms such as coughing, dyspnea, and chest discomfort. Steam inhalation may provide temporary relief from these symptoms. Patients requires to consult the healthcare practitioners for comprehensive management [38]. Patients with FMS who experience respiratory problems or acquire COVID-19 may benefit from nebulization [39]. Vitamin D supplementation is recommended for bone health [40]. The multifaceted nature of FMS requires a comprehensive approach to diagnosis and management. While the identification of genetic and biochemical markers holds promise for improving diagnostic accuracy, ongoing research is essential to fully understand the complex interactions underlying FMS. Enhanced diagnostic tools and personalized treatment strategies will be critical in addressing the diverse needs of individuals affected by this challenging condition [41, 42].

Conclusion

FMS is difficult to diagnose but the newer approaches emphasize a broader understanding of symptoms. Comprehensive assessments, including patient history, physical examination, and questionnaires, are recommended to understand the full spectrum of FMS manifestations. Management of FMS requires a multidisciplinary approach, integrating pharmacological and non-pharmacological treatments. Medications such as antidepressants, anticonvulsants, and muscle relaxants are commonly prescribed to alleviate symptoms. Non-pharmacological strategies, including cognitive-behavioural therapy, physical exercise, and stress management techniques, are equally important. Emerging therapies, like neuromodulation and novel pharmacological agents, show promise in enhancing treatment efficacy. Research continues to uncover the complexities of FMS, highlighting the need for personalized treatment plans tailored to individual patient profiles.

Conflict of Interest

No conflict of interest.

Abbreviations

Fibromyalgia Syndrome (FMS)
American College of Rheumatology (ACR)
Hypothalamus-pituitary-adrenal axis (HPA)
Osteoarthritis (OA)
Suprachiasmatic nucleus (SCN)
Retino-hypothalamic-tract (RHT)
Non-coding ribonucleic acids (lncRNAs)

References

1. Koca TT, Seyithanoglu M, Sagiroglu S, Berk E, Dagli H. Frequency of audiological complaints in patients with fibromyalgia syndrome and its relationship with oxidative stress. Nigerian Journal of Clinical Practice. 2018 Oct;21(10):1271-1277.
2. Ablin JN, Buskila D. Update on the genetics of the fibromyalgia syndrome. Best Practice & Research Clinical Rheumatology. 2015;29:20-8. doi:10.1016/j.berh.2015.04.018.
3. Parvez S, Fatima G, Das SK, Ahmad I. Serotonin role in fibromyalgia. Era's Journal of Medical Research. 2021 Jan 1;8(1). doi:10.24041/ejmr2021.10.
4. Buskila D, Sarzi-Puttini P. Biology and therapy of

fibromyalgia. Genetic aspects of fibromyalgia syndrome. Arthritis Research & Therapy. 2006;8:218. doi:10.1186/ar2005.
5. Parvez S, Fatima G, Das SK, Ahmad I. Positive and negative association of vitamin D with fibromyalgia syndrome. Era's Journal of Medical Research. 2020;7(1):126-33. doi:10.24041/ejmr2020.21.
6. Park DJ, Lee SS. New insights into the genetics of fibromyalgia. Korean Journal of Internal Medicine. 2017;32:984-95. doi:10.3904/kjim.2016.207.
7. Ablin JN, Buskila D. Update on the genetics of the fibromyalgia syndrome. Best Practice & Research Clinical Rheumatology. 2015;29:20-8. doi:10.1016/j.berh.2015.04.018.
8. Fatima G, Alhmadi H, Mahdi AA, Hadi N, Fedacko J, Magomedova A, Parvez S, Raza AM. Transforming diagnostics: A comprehensive review of advances in digital pathology. Cureus. 2024 Oct 19;16(10):e71890. doi:10.7759/cureus.71890.
9. Silioni I, Russell IJ, Stewart JM, Gleason RM, Theoharides TC. Neuropeptides CRH, SP, HK-1, and inflammatory cytokines IL-6 and TNF are increased in serum of patients with fibromyalgia syndrome, implicating mast cells. Journal of Pharmacology and Experimental Therapeutics. 2016 Mar;356(3):664-72.
10. D'Agnelli S, Arendt-Nielsen L, Gerra MC, Zatorri K, Boggiani L, Baciarello M, Bignami E. Fibromyalgia: Genetics and epigenetics insights may provide the basis for the development of diagnostic biomarkers. Molecular Pain. 2019 Jan;15:1744806918819944.
11. Parvez S, Fatima G, Mahdi F, Hadi NR, Fedacko J. Assessment of the association of serotonin transporter gene (5-HTTVNTR & 5-HTTLPR) polymorphism in patients with fibromyalgia syndrome. Wiadomosci Lekarskie (Warsaw, Poland: 1960). 2023 Jan 1;76(6):1378-84. doi:10.36740/WLek202306108.
12. Parvez S, Fatima G, Mehdi F, Hadi NR, Fedacko J, Hadi N, Fedacko J. Relationship between vitamin D receptor gene BsmI polymorphism and fibromyalgia syndrome. Cureus. 2022 Jul 21;14(7). doi:10.7759/cureus.27113.
13. Parvez S, Fatima G, Mahdi F, Fedacko J, Hadi NR. Unraveling the clinico-genetic association of catechol-O-methyltransferase-rs4680 G>A gene polymorphism in women with fibromyalgia syndrome. Wiadomosci Lekarskie. 2022 Jan 1;75(10):2439-44. doi:10.36740/WLek202210123.
14. Tiffon C. The impact of nutrition and environmental epigenetics on human health and disease. International Journal of Molecular Sciences. 2018 Nov 1;19(11):3425.
15. González-Flores D, López-Pingarrón L, Castaño MY, Gómez MÁ, Rodríguez AB, García JJ, Garrido M. Melatonin as a coadjuvant in the treatment of patients with fibromyalgia. Biomedicines. 2023 Jul 12;11(7):1964.
16. Kim TO, Park DI, Han YK, *et al*. Genome-wide analysis of the DNA methylation profile identifies the fragile histidine triad (FHIT) gene as a new promising biomarker of Crohn's disease. Journal of Clinical Medicine. 2020 May 4;9(5):1338.
17. Menzies V, Lyon DE, Archer KJ, *et al*. Epigenetic alterations and an increased frequency of micronuclei in women with fibromyalgia. Nursing Research and Practice. 2013;2013:795784. doi:10.1155/2013/795784.
18. Fajardo O, Meseguer V, Belmonte C, Viana F. TRPA1 channels mediate cold temperature sensing in mammalian vagal sensory neurons: pharmacological and genetic evidence. Journal of Neuroscience. 2008;28:7863-75. doi:10.1523/JNEUROSCI.1696-08.2008.
19. Ciampi de Andrade D, Maschietto M, Galhardoni R, *et al*. Epigenetics insights into chronic pain: DNA hypomethylation in fibromyalgia—a controlled pilot-study. Pain. 2017;158:1473-80. doi:10.1097/j.pain.0000000000000932.
20. Andersen HH, Duroux M, Gazerani P. MicroRNAs as modulators and biomarkers of inflammatory and neuropathic pain conditions. Neurobiology of Disease. 2014;71:159-68. doi:10.1016/j.nbd.2014.08.003.
21. Bjersing JL, Lundborg C, Bokarewa MI, Mannerkorpi K. Profile of cerebrospinal microRNAs in fibromyalgia. PLoS One. 2013;8:e78762. doi:10.1371/journal.pone.0078762.
22. Bjersing JL, Bokarewa MI, Mannerkorpi K. Profile of circulating microRNAs in fibromyalgia and their relation to symptom severity: an exploratory study. Rheumatology International. 2015;35:635-42. doi:10.1007/s00296-014-3139-3.
23. Masotti A, Baldassarre A, Guzzo MP, Iannuccelli C, Barbato C, Di Franco M. Circulating microRNA profiles as liquid biopsies for the characterization and diagnosis of fibromyalgia syndrome. Molecular Neurobiology. 2017;54:7129-36. doi:10.1007/s12035-016-0235-2.
24. Jones KD, Gelbart T, Whisenant TC, *et al*. Genome-wide expression profiling in the peripheral blood of patients with fibromyalgia. Clinical and Experimental Rheumatology. 2016;34:S89-98.
25. Dolcino M, Tinazzi E, Puccetti A, Lunardi C. Gene expression profiling in fibromyalgia indicates an autoimmune origin of the disease and opens new avenues for targeted therapy. Journal of Clinical Medicine. 2020;9:10.3390/jcm9061814.
26. Fatima G, Magomedova A, Parvez S. Biotechnology and sustainable development. Shineeks Publishers; 2024 Apr 14.
27. Assavarittirong C, Samborski W, Grygiel-Górniak B. Oxidative stress in fibromyalgia: from pathology to treatment. Oxidative Medicine and Cellular Longevity. 2022;2022(1):1582432.
28. Tiwari B, Fatima G, Hadi N, Fedacko J, Magomedova A, Raza AM, Alharis N, Qassam H, Alhmadi HB, Parvez S. Metal toxicity: significant health assessment. Kufa Medical Journal. 2024 Dec 1;20(2). doi:10.36330/kmj.v20i2.17957.
29. Pagliai G, Giangrandi I, Dinu M, Sofi F, Colombini B. Nutritional interventions in the management of fibromyalgia syndrome. Nutrients. 2020 Aug 20;12(9):2525.
30. Ahmad IZ, Parvez S, Tabassum H. Cyanobacterial peptides with respect to anticancer activity: structural and functional perspective. Studies in Natural Products Chemistry. 2020 Jan 1;67:345-88. doi:10.1016/B978-0-12-819483-6.00010-2.
31. Parvez S, Dzupina A, Fatima G, Fedacko J, Magomedova A, Mehdi AA. Unveiling the role of human PER3 gene polymorphism (rs57875989) as a potential risk factor in fibromyalgia syndrome

patients. Cureus. 2024 Dec 6;16(12):e75210. doi:10.7759/cureus.75210.
32. González-Flores D, López-Pingarrón L, Castaño MY, Gómez MÁ, Rodríguez AB, García JJ, Garrido M. Melatonin as a coadjuvant in the treatment of patients with fibromyalgia. Biomedicines. 2023 Jul 12;11(7):1964.
33. Fatima G, Parvez S, Tuomainen P, Fedacko J, Kazmi DH, Elkilany GE. Amalgamation of circadian clock gene with incidence of myocardial infarction. Indian Journal of Cardiovascular Disease in Women. 2024:1-7. doi:10.25259/IJCDW_69_2023.
34. Parvez S, Fatima G. Unveiling the link between long COVID-19 and fibromyalgia. International Journal of Medical Research and General Education. 2025;6(1):1189-1192. doi:10.54660/.IJMRGE.2025.6.1.1189-1192.
35. Khoja O, Mulvey M, Astill S, Tan AL, Sivan M. New-onset chronic musculoskeletal pain following COVID-19 infection fulfils the fibromyalgia clinical syndrome criteria: a preliminary study. Biomedicines. 2024 Aug 23;12(9):1940.
36. Parvez S, Fatima G. Mini review: association between serum cortisol levels and COVID-19 disease. Era's Journal of Medical Research. 2021 Jul 1;8(2). doi:10.24041/ejmr2021.35.
37. Fatima G, Parvez S, Raza AM, Fedacko J, Hadi NR. COVID-19 and mucormycosis: a short report from India. Era's Journal of Medical Research. 2021;8(2). doi:10.24041/ejmr2021.38.
38. Parvez S, Fatima G, Hadi NR, Jha H. Early steam inhalation: the first crucial step in combating evil—the COVID-19. Era's Journal of Medical Research. 2021;8(1):82-4. doi:10.24041/ejmr2021.15.
39. Singh RB, Fatima G, Parvez S, Halabi G, Hadi N. Safety and efficacy of nebulization for reducing the intensity of viral load and clinical manifestations in patients with COVID-19. In: Features, Transmission, Detection, and Case Studies in COVID-19. Academic Press; 2024 Jan 1. p. 507-514. doi:10.1016/B978-0-323-95646-8.00054-8.
40. Parvez S, Fatima G, Al-Awaida W, Hussien YA, Tamam SA, Hadi NR. Vitamin D: implications in COVID-19. Latin American Journal of Pharmacy. 2021 Apr 1;40:23-6.
41. Fatima G, Siddiqui Z, Parvez S. AI and precision medicine: paving the way for future treatment. Preprints. 2024;2024120036. doi:10.20944/preprints202412.0036.v1.
42. Bajwa J, Munir U, Nori A, Williams B. Artificial intelligence in healthcare: transforming the practice of medicine. Future Healthcare Journal. 2021;8(2):188-194.