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Personalized Medicine Approaches in the Management of Chronic Pain: From Genomics to Targeted Therapy

Hossein Fazli[1,*], Mehdi Rezaee[1]

[1]**Assistant professor of anesthesiology, school of Medicine, Alborz University of Medical Science, Karaj, Iran.**

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Corresponding author:
Hosein Fazli
Email:drhf_md@yahoo.com

ABSTRACT

A common and incapacitating condition, chronic pain offers a difficult field of work because of its variability and response to conventional treatments. Genomic and proteomic-based personalized medicine including epigenetic and biomarker information could help to lower treatment variability, so improving diagnosis, phenotypic classification, and individualized approaches to treatment. Recent developments in genetics and pharmacogenetics of pain, pain phenotyping techniques, and the development of focused therapies including epigenetic modulators, peptides, biologics and nanomedicine are underlined in this review. Personalized medicine seeks to match every patient's individual genetic makeup to their course of treatment. It is increasingly accepted that pain chronology involves epigenetic processes, including DNA methylation and histone modifications. Furthermore discussed are the value of biomarkers in evaluating therapy response and prognosis as well as ethical, financial, and data availability-related issues. Finally, future directions involve the use of artificial intelligence mixed with multi-omics data for tailored optimal pain management. Adopting these changes can help patients to have less chronic pain and improve the therapeutic outcomes.

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INTRODUCTION

Background information commonly defined as pain lasting more than three months, chronic pain affects 20–30% of the worldwide population and ranks as a major healthcare issue in both developed and underdeveloped areas of the world (1, 2). It is accompanied by major psychological suffering, handicap, lower productivity, and healthcare use. A reflection of this complexity and variability is neuropathic pain, inflammatory disease (e.g., rheumatoid arthritis), and musculoskeletal syndrome (e.g., fibromyalgia, chronic low back pain) (3). Generic approaches to pain management include widely used drugs, opiates, NSAIDs, antidepressants, and physiotherapy for pain. However, a variety of side effects, such as tolerance, stomach pain, and cognitive problems, are associated with these treatments, and their effectiveness varies widely among individuals. For many patients, the "one-size-fits-all" approach has been inadequate; hence, the urgent search for more individualized treatments is still necessary (4).

Personalized medicine is one of the new paradigms suggested to enhance the diagnosis, classification, and treatment of chronic pain in the past years. Personalized, or precision, medicine is the application of information about a patient's genes, proteins, epigenomes, environments, lifestyle, and preferences to prevent, diagnose, and treat diseases (5). Regarding chronic pain, it would seek to identify the biological

processes behind pain in every patient, identify predictive biomarkers of response to treatment, and select the most appropriate treatment to maximize benefit and minimize harm (6).

Genomic data has helped to clarify, for instance, genetic polymorphisms affecting drug metabolism (e.g., CYP2D6 variants impacting opioid efficacy and safety), pain perception (e.g., SCN9A mutations), and risk for chronic pain problems (7). It is increasingly accepted that pain chronology involves epigenetic processes, including DNA methylation and histone modifications. These systems could serve as both diagnostic markers and therapeutic targets. Thanks to the identification of biomarkers associated with inflammatory and neuropathic pain states by proteome and metabolomics studies, more thorough phenotyping of pain syndromes is now feasible (8). Furthermore, sophisticated tools such as functional imaging, machine learning algorithms, and multi-omics integration are beginning to enable real-time, patient-specific decision-making. These developments allow patients to be stratified into clinically significant subgroups based on molecular signatures instead of symptom clusters by themselves (9).

Personalized medicine's promise notwithstanding, it is not without difficulties in terms of chronic pain management. These include high costs, the need for standardized protocols for biomarker validation, limited access to genomic technologies in regular clinical practice, ethical questions regarding genetic testing, and the need for multidisciplinary cooperation among clinicians, researchers, and data scientists (10).

This review aims to provide a comprehensive overview of the current state of individualized medicine for chronic pain. We investigate its uses across several pain types neuropathic, inflammatory, and musculoskeletal highlight recent discoveries in genomics and molecular profiling, assess the clinical value of these approaches, and address continuing implementation challenges. With an eye toward precision therapeutics, digital health integration, and patient-centered care models, we last discuss the future paths of this developing discipline.

Genetic and Pharmacogenetics Basis of Pain

Pain is a sensory experience; environmental, psychological, and genetic elements all profoundly influence it (3). Genetic variation significantly influences personal responses to pain sensation and analgesic treatments. The main objective of personalized medicine is pain management transformation; knowledge of the genetic bases of pain helps enable focused, patient-specific treatments (11). Personalized medicine seeks to match every patient's individual genetic makeup to their course of treatment. Combining genetic knowledge about pain management strategies helps doctors make analgesics more effective and enhance patient outcomes, thus facilitating a more compassionate and effective way of treating pain (12).

Genetic Modulators of Pain Perception

Candidate gene studies and genome-wide association studies (GWAS) have found several genetic polymorphisms notably affecting pain sensitivity and susceptibility to chronic pain disorders (13). Among the genes under the most research are

COMT (Catechol-O-methyltransferase)

This enzyme breaks down important modulators of pain and stress reactions, catechol amines, including dopamine, epinephrine, and norepinephrine. Changing enzymatic activity, the COMT Val158Met polymorphism has been linked to variations in pain sensitivity. Reduced COMT activity and higher catecholamine levels cause those with the Met/Met genotype to often exhibit more pain sensitivity (14).

OPRM1 (Mu-Opioid Receptor Gene)

The A118G polymorphism (rs17999) causes changes in receptor binding and expression, thus influencing both side effect risk and opioid efficacy. Target of endogenous opioids and morphine, the OPRM1 gene codes the mu-opioid receptor. For efficient analgesia, carriers of the G allele might need more opioids, and their treatment results could be more variable (15).

SCN9A (Sodium Voltage-Gated Channel Alpha Subunit 9)

Found in peripheral neurons, this gene codes the Nav1.7 sodium channel and is fundamental for nociceptive signal propagation. From congenital insensitivity to pain to inherited erythromelalgia a disorder of extreme burning pain mutations in SCN9A have been linked to a spectrum of pain phenotypes (16). Common differences in SCN9A could also play a part in the predisposition for persistent pain. These findings underline the complex and polygenic nature of pain and suggest that the interaction of several genetic loci forms an individual's pain experience (17).

Pharmacogenetics of Pain Management

Given chronic pain, where inter-individual variability in treatment efficacy and side effect profiles is a major clinical challenge, pharmacogenetics the study of how genetic variations affect drug response is especially pertinent (18).

Opioid Analgesics

Apart from OPRM1 polymorphisms, genetic differences in drug-metabolizing enzymes, such as CYP2D6, are also quite crucial. Among the many opioids CYP2D6 breaks down are codeine, tramadol,

and oxycodone. While ultra-rapid metabolizers may be at risk of opioid toxicity, those with poor metabolizer phenotypes e.g., those resulting from loss-of-function alleles may have inadequate analgesia. Genotype-guided prescribing can thus enhance both efficacy and safety (19).

NSAIDs and COX Inhibitors

Variations in CYP2C9 and UGT enzymes affect NSAID metabolism, including those of ibuprofen and celecoxib. Some polymorphisms may increase the risk of gastrointestinal toxicity or cardiovascular events, which calls for customised dosage or drug choice (20).

Antidepressants and Anticonvulsants

Often prescribed for the treatment of neuropathic pain, drugs including amitriptyline, duloxetine, and gabapentin also affect genes. Variations in CYP2D6 and CYP1A2, for instance, could change plasma concentrations of tricyclic antidepressants, so affecting the therapeutic value of these drugs as well as the risk of sedation or cardiac side effects (19). Medications routinely prescribed for the treatment of neuropathic pain, including amitriptyline, duloxetine, and gabapentin, can also be affected by genetic factors. Variations in CYP2D6 and CYP1A2 can affect the plasma concentrations of tricyclic antidepressants, for example, influencing the therapeutic benefit as well as the risk of sedation or cardiac side effects during treatment (21).

Clinical Applications and Future Directions

Pain pharmacogenomics is still in its early years of clinical use, despite some fascinating studies. Some hospitals and pain clinics have started regularly including pharmacogenetics testing into daily operations for especially challenging or treatment-resistant pain conditions (22). Commercial pharmacogenomics panels today provide useful data for customizing analgesic regimens, even if access, cost, and provider familiarity remain challenges. Combining genetic testing into electronic health records (EHRs) with decision-support tools will enable future genotype-based, real-time prescription writing (23). Like the All of Us program run by the NIH, large-scale biobanks and precision medicine projects are expected to concurrently speed the identification of new pain-related genetic markers and improve phenotype-genotype correlations. Reducing trial-and-error prescription writing, minimizing adverse events, and improving patient outcomes depend, especially, on the integration of genetic and pharmacogenetics data in pain management (24).

Pain Phenotyping for Treatment Stratification

The treatment of chronic pain is usually based on symptom-based classifications and subjective pain ratings. Still, these conventional methods occasionally fail to adequately depict the biological variation of chronic pain syndromes. Many of them thus receive either inadequate or non-responsive treatment (25). Recent attempts in pain phenotyping seek to classify patients based on objective biological, psychological, and neurophysiological criteria to overcome this restriction. Fundamental concepts of personalised medicine: this stratification enables doctors to customize treatments to the fundamental processes generating a patient's suffering (26).

Multidimensional Pain Assessment Tools

Chronic pain is a complex and multifaceted experience that affects emotional, cognitive, functional, and physical aspects, as well as physical sensation. Given this complexity, researchers have developed multidimensional pain assessment tools that offer a thorough evaluation across basic intensity ranges (27). These devices enable doctors to customise treatments and help them to understand the whole spectrum of a patient's pain experience by capturing many elements of pain, including sensory characteristics, emotional impact, interference with daily activities, and psychological factors (28). Using descriptive words falling into sensory, affective, and evaluative categories, the McGill Pain Questionnaire (MPQ) ranks the qualitative aspects of pain among the most often used instruments. This questionnaire detects several types of pain (e.g., sharp, burning, throbbing) and catches emotional reactions connected with pain, such as fear or anger (29). The depth of the MPQ guarantees a perceptive study of the basic causes of pain and guides mechanism-based treatments. The Brief Pain Inventory (BPI) is another helpful tool for determining pain degree and the degree of influence it has on many spheres of life, including mood, job, sleep, and social events (30). The BPI can find patients who might require multidisciplinary approaches combining pharmacologic and rehabilitative treatments by means of pain estimation. Particularly evaluating functional disability resulting from pain is made possible by tools such as the Pain Disability Index (PDI), which gauges how pain limits participation in daily activities, including employment, family responsibilities, and leisure activities (31). This assessment helps one to grasp the more general consequences of chronic pain on quality of life and guides occupational therapy and rehabilitation. Apart from the physical and functional spheres, psychological and cognitive aspects are rather important for pain experience and chronicity (32). Often accompanied by multidimensional pain assessments are patient-reported outcome measurements (PROMs) assessing anxiety, depression, pain catastrophising, and coping strategies, including the Pain Catastrophising

Scale (PCS) and Beck Depression Inventory (BDI). Among other psychological or behavioral therapies, these tools help to identify individuals who might benefit from mindfulness-based interventions or cognitive behavioural therapy (CBT) (33). Modern clinical practice emphasizes more and more the need to include these multidimensional tools in regular evaluations so that the patient's pain can be totally understood. Using more accurate phenotyping of pain types and contributing factors, this all-encompassing approach helps doctor's design tailored treatment plans that not only control the physical sensation but also address the emotional and functional effects of pain (34). Furthermore, multidimensional tests help to monitor treatment efficacy constantly by guiding suitable modifications in therapeutic strategies and identifying differences among several pain areas. These tools provide consistent, validated approaches in research to evaluate outcomes and evaluate the effectiveness of new treatments among several patient groups (35). Good long-term pain management usually depends on multidimensional pain assessment tools. By accepting the complexity of pain and including sensory, emotional, cognitive, and functional evaluations, these instruments help doctors to deliver patient-centered, mechanism-based treatment that maximizes clinical outcomes and improves quality of life (27). Chronic pain is a complex, multifarious experience that influences emotional, cognitive, functional, and physical aspects as well as physical sensation. Understanding this complexity, multidimensional pain assessment instruments have been created to offer an all-encompassing assessment going beyond basic intensity ranges (36). These tools help doctors to understand the whole spectrum of a patient's pain experience by allowing them to customise treatments by capturing many aspects of pain, including sensory characteristics, emotional impact, interference with daily activities, and psychological factors (28, 37).

The McGill Pain Questionnaire (MPQ) is among the most often used instruments since it evaluates the qualitative features of pain using descriptive words falling into sensory, affective, and evaluative categories. This questionnaire distinguishes pain types (e.g., sharp, burning, throbbing) and catches emotional reactions like fear or anger linked with pain. The depth of the MPQ guarantees perceptive study of the basic causes of pain and guides mechanism-based treatments (38).

Another useful instrument for assessing pain level and the degree to which it affects many spheres of life, including mood, employment, sleep, and social events, is the Brief Pain Inventory (BPI). By means of pain estimation, the BPI can identify patients who might need multidisciplinary approaches combining pharmacologic and rehabilitative therapies (39).

Tools such as the Pain Disability Index (PDI), which measures how pain limits participation in daily roles including employment, family responsibilities, and leisure activities, enable the specific evaluation of functional disability resulting from pain. This evaluation guides occupational treatments and rehabilitation and helps one to understand the more general effects of chronic pain on quality of life (40).

In addition to physical and functional aspects, psychological and cognitive factors play a significant role in the experience of pain and its chronicity. Often accompanied by multidimensional pain assessments are patient-reported outcome measures (PROMs) assessing anxiety, depression, pain catastrophising, and coping strategies, including the Pain Catastrophising Scale (PCS) and Beck Depression Inventory (BDI) (41). These instruments enable the identification of patients who might benefit from mindfulness-based interventions or cognitive behavioural therapy (CBT), among other psychological or behavioural treatments (42).

Modern clinical practice is increasingly emphasizing the importance of incorporating these multidimensional tools into regular assessments to fully understand the patient's pain (43). This all-encompassing approach, which uses more accurate phenotyping of pain types and contributing elements, helps doctors develop customised treatment plans that manage not only the physical sensation but also the emotional and functional consequences of pain (44). Moreover, multidimensional tests help to support continuous monitoring of treatment efficacy by guiding appropriate changes in therapeutic approaches by catching changes across many pain areas. These instruments offer consistent, validated methods in research to assess results and compare the efficacy of new treatments among several patient groups (45). Effective long-term pain treatment depends on general multidimensional pain assessment instruments. These tools enable doctors to offer patient-centered, mechanism-based treatment that optimizes clinical outcomes and enhances quality of life by accepting the complexity of pain and including sensory, emotional, cognitive, and functional evaluations (46).

Functional Neuroimaging

A new tool showing central nervous system pain processing in a useful perspective is functional brain imaging. Functional magnetic resonance imaging (fMRI), positron emission tomography (PET), and magnetoencephalography (MEG), among other methods, reveal brain regions engaged in pain experience and modulation (47).

Studies have revealed different patterns of brain activity produced by different pain disorders. Often including changed activity in the thalamus

and somatosensory cortex, neuropathic pain is Hyperactivity in pain-related regions, including the anterior cingulate cortex and insula has been linked to fibromyalgia (48).

In the salience networks and default mode, chronic low back pain can result from maladaptive connectivity. These results support the hypothesis of central sensitization a condition of increased neural responsiveness in chronic pain and help distinguish between several kinds of pain. Apart from diagnosis, functional neuroimaging can help to predict response to treatments including pharmacotherapy, neuromodulation, or cognitive behavioural therapy (CBT) (49).

Biomarkers of Inflammation and Pain Mechanisms

Essential tools in comprehending the complicated biological processes underlying chronic pain and in differentiating several pain mechanisms are now molecular biomarkers. These biomarkers help doctors to identify particular pathways causing pain, so allowing them to more precisely target treatments and forecast treatment responses (47). Among the mostwell-investigated are inflammatory cytokines, neurotrophic factors, and metabolic products, which offer a window into the dynamic interactions between the peripheral nervous system, immune response, and central nervous system modulation of pain (50).

Rheumatoid arthritis, osteoarthritis, and complex regional pain syndrome (CRPS) are among the inflammatory and autoimmune diseases linked with chronic pain that routinely show elevated levels of pro-inflammatory cytokines, including tumour necrosis factor-alpha (TNF-α), C-reactive protein (CRP), and interleukin-6 (IL-6) (51). At both peripheral and central levels, these molecules help to sensitize nociceptors and amplify pain signalling. These markers are helpful not only for diagnosis but also for monitoring therapeutic efficacy since elevated levels of them usually correspond with disease activity and pain intensity (52).

A key mediator of central sensitization, a phenomena whereby the central nervous system shows increased responsiveness to pain stimuli, brain-derived neurotrophic factor (BDNF) has attracted interest. In pain pathways, BDNF affects synaptic plasticity and neuronal excitability; higher BDNF expression has been associated with the onset and maintenance of chronic pain states (53). Thus, BDNF could be a useful biomarker for determining patients who run the danger of pain chronology and for evaluating the effects of therapies meant to reduce central sensitivity (54).

The body's stress response is mostly dependent on the hypothalamic-pituitary-adrenal (HPA) axis; thus, dysregulation of this axis has been linked to the pathophysiology of several pain conditions. Together with other HPA-axis markers, cortisol, the main glucocorticoid hormone produced by the adrenal glands in response to stress, reflects the complicated bidirectional link between psychological stress and pain (55). Conditions including fibromyalgia and post-traumatic stress disorder (PTSD), where chronic stress aggravates pain perception and symptom severity, have seen elevated or blunted cortisol responses (56).

Advances in high-throughput technologies have recently enabled the investigation of omics-based biomarkers, including transcriptomics, proteomics, and metabolomics to reflect the multidimensional character of pain biology (57)University of Porto, 4200-319 Porto, Portugal.Transforming Clinical Research: The Power of High-Throughput Omics Integration. While metabolomics studies reveal changes in biochemical pathways in pain states, transcriptomic profiling lets one identify gene expression patterns linked with particular pain phenotypes. These methods have great potential to create more complex pain stratification systems and allow tailored therapeutic interventions addressing the molecular heterogeneity of chronic pain (58). Together, the identification and validation of inflammation- and pain-related biomarkers present a transforming chance to improve diagnostic accuracy, track disease progression, and direct individualized treatment plans, so improving outcomes for patients with chronic pain (59).

Psychosocial Phenotyping

The experience of chronic pain is much influenced by psychological and social factors, so it affects not only the general therapy response but also long-term consequences and pain perception (60). The biopsychosocial model of pain acknowledges that, rather than only a biological phenomenon, it is a complex interaction between biological, psychological, and social elements. Therefore, a thorough understanding of the pain experience of the patient and the design of particular, effective treatment plans depend on careful pain assessment, including the evaluation of psychological aspects (61). By evaluating catastrophic thinking about pain, the Pain Catastrophising Scale (PCS) enables doctors to identify those who might be prone to chronic disabling pain or unsatisfactory treatment results. Likewise, the Beck Depression Inventory (BDI) is widely used to assess the degree of depressed symptoms, which usually accompany chronic pain and demand coordinated treatment plans (62). Knowing psychological aspects helps one to apply focused treatments different from pharmacological ones. For those who show significant degrees of catastrophising or depressed symptoms, cognitive behavioural therapy (CBT) helps them to change maladaptive thought patterns and increase coping mechanisms. Furthermore, shown to improve

psychological well-being and pain perception are integrative therapies, including acceptance and commitment therapy, biofeedback, and mindfulness-based stress reduction (63).

Targeted Therapies in Personalized Pain Medicine

Individualized pain management techniques are a range of focused treatments meant to specifically target the biological pathways causing a person to suffer. Using histone deacetylase (HDAC) inhibitors and other epigenetic treatments shows promise in changing the patterns of gene expression linked with neuropathic pain, so offering new paths for pain reduction in circumstances when traditional painkillers are not working (64). By their specific targeting of ion channels and receptors regulating neuronal excitability, treatments based on peptides, which may have fewer side effects, are able to precisely modulate pain signals. Monoclonal antibodies against key pro-inflammatory cytokines, including TNF-α and interleukins, have changed the course of treatment for inflammatory pain syndromes (65). This is so since biologics directly neutralize the molecular causes of pain and inflammation. Furthermore, developments in nanomedicine allow one to design customised drug delivery systems aiming at therapeutic agents, especially in damaged or inflammatory tissues (66). This reduces system exposure and toxicity at the same time as raising efficiency. Taken as a whole, these creative ideas show the ways in which tailored, mechanical pain management can be applied to precisely enhance the results of treatment for patients (67). Personalised treatment plans aiming at multiple objectives consist of the following elements:

Epigenetic therapies: HDAC inhibitors for neuropathic pain

Development and continuation of chronic pain depend on epigenetic control, which is reached by altering gene expression without altering the underlying DNA sequence. Especially histone deacetylases (HDACs), damage-induced modifications in chromatin remodelling enzymes can suppress genes required for neuropathic pain, anti-inflammatory signalling, neural repair, and inhibition of neurotransmission (68). Emerging as a possible class of therapy able to reverse maladaptive epigenetic changes in glial cells and sensory neurons are HDAC inhibitors HDAC, is combining trichostatin A and suberoylanilide hydroxamic acid (SAHA), has been shown in preclinical studies in animal models of neuropathic pain to lower mechanical allodynia and thermal hyperalgesia. Utilizing better isoform-selective HDAC inhibitors, constant research seeks to maximize therapeutic efficacy and reduce systemic side effects (69).

Peptide-based therapies: Targeting specific ion channels

Whether synthetic or natural, the selectiveness of peptides determines the evolution of treatments aimed at molecules involved in nociceptive signalling. One important use is aiming at voltage-gated ion channels

also known as calcium, sodium, or potassium channels which regulate the excitability of the nervous system and the beginning of pain (70). For intrathecal injection in the treatment of severe chronic pain, for example, the Food and Drug Administration (FDA) has licensed the peptide ziconotide, derived from cone snail venom. It also reduces in dorsal horn neurons the activity of N-type voltage-gated calcium channels (Cav2.2) (71). Further peptides linked to inflammatory pain and thermosensation are under development now. Either target themselves or target the inhibition of the change of transient receptor potential (TRP). Their relative specificity to their target allows these peptides to generate analgesia free from the broad receptor activation linked with conventional medications (72). Even though we still need to improve their stability, distribution, and penetration across biological barriers, peptide treatments hold great potential for individualized pain management (73).

Biologics: Monoclonal antibodies against key cytokines

Sometimes dysregulated immune responses marked by elevated levels of pro-inflammatory cytokines define chronic pain syndromes. This is particularly true in disorders including an inflammatory or autoimmune component. Monoclonal antibodies (mAbs) are a specifically targeted therapy since they can neutralize these cytokines or their receptors (74). Targeting tumour necrosis factor-alpha (TNF-α), agents including infliximab, adalimumab, interleukin-1 beta (IL-1β), or interleukin-6 (IL-6) have shown their efficacy in reducing pain and inflammation in a range of diseases, including psoriatic arthritis, ankylosing spondylitis, and rheumatoid arthritis (75). These biologics have the potential to change the processes causing central and peripheral pain by sensitizing nociceptive neurons driven by cytokines. Younger monoclonal antibodies are under investigation for their possible use in the treatment of non-autoimmune chronic pain syndromes, including fibromyalgia and chronic migraine, both of which are thought to be typified by strong neuroimmune interactions (76). The application of biologics is most reasonable in well-defined patient subgroups created using exact diagnosis techniques. This is so because parenteral treatment is much sought after, and biologics are sometimes costly (77).

Future Perspectives

Combining advanced artificial intelligence and deep learning techniques with multi-omics data, including genomics, proteomics, and metabolomics,

will define personalized pain medicine's future. From this mix, one can create comprehensive, unique pain profiles reflecting the complex genetic, molecular, environmental, and psychological factors influencing pain (78). Better biomarker discovery, patient stratification, and treatment response prediction made feasible by artificial intelligence-driven models will produce more customised treatments from which to draw (79). Wearable sensors and digital health apps will also enable dynamic treatment changes and real-time monitoring, thus improving customised treatment. While validating these strategies by means of large-scale clinical studies still poses difficulties, they guarantee cost-effectiveness, address ethical and privacy issues, and foster cooperation among several professionals (80). Supported regulatory and reimbursement systems should enable the regular clinical practice to bring customised pain management (81).

CONCLUSION

Chronic pain is a complex condition with great challenges for appropriate treatment since it reacts differently to conventional drugs. Customized medicine offers each patient's unique biology and clinical profile a personalized pain management solution. Reaching this is made possible by developments in genes, proteomics, biomarkers, and targeted medications. Integration of multi-omics data with artificial intelligence and machine learning has the potential to bring about a positive change, even if there are still main challenges to overcome, such as high costs, limited access to comprehensive molecular data, and ethical questions about genetic information. Apart from customized treatment plans that maximize efficacy while simultaneously reducing side effects, these technologies could enable exact pain phenotyping. Future research stressing large-scale clinical trials and real-world studies assessing the safety, efficacy, and cost-effectiveness of tailored treatments will enable us to fully meet this promise. At last, tailored pain management can improve quality of life, alter the course of treatment for millions of people worldwide, and increase patient outcomes.

Author's Contribution

Hosein Fazli and Mehdi Rezaee were involved in the conceptualization, design and writing of the manuscript draft. The authors read and confirmed the final manuscript.

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