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Personalized Medicine Approach in the Treatment and Status of Autoimmune Diseases

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Abstract:

Precision medicine is accomplished by the process of categorising people and administering therapy that specifically targets their condition. Evidence demonstrates that the pathological circumstances of patients who are categorized or diagnosed with a singular disease exhibit significant diversity in nearly all autoimmune disorders. Hence, the use of precision medicine is crucial in the management of patients suffering from autoimmune disorders. At present, precision medicine is not available for any autoimmune disease. This article examines the use of precision medicine in the treatment of psoriasis, Alzheimer's disease and rheumatoid arthritis. Therefore, after a comprehensive understanding of these autoimmune disorders and their treatment strategies, we will use a personalized medicine approach in the management of these diseases.

Keywords: Personalized medicine, Autoimmune disease, Psoriasis, Alzheimer's disease, Rheumatoid arthritis

Introduction

Personalized medicine (PM) is a cutting-edge and captivating subject in health and medicine. The notion can revolutionize medical treatments by offering customized therapeutic methods that consider an individual's genetic, epigenomic, and proteomic profile while also considering the patient's circumstances (1). The efficacy of preventive measures is as significant as that of therapy in the realm of project management. Enhanced implementation of molecular stratification of patients, such as evaluating mutations that lead to resistance to certain medicines, would provide medical personnel with definitive data to establish treatment regimens for particular patients (2). This innovation will eliminate the need for risky, trial-and-error approaches to medication administration. At present, patients have the option to change medications if one is not working. In terms of adverse reactions, medication interactions, possible disease development, successful therapy delays, and patient discontent, the results for patients are worse when this strategy is based on trial and error (3).

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Boguski et al. introduced the term PM in 2009 and delineated its three fundamental characteristics: knowledge regarding the aetiology of diseases, capability to identify the presence of causal agents or factors, and efficacious treatment of the underlying causes (4). In 2011, the National Research Council of the US National Academies introduced a more precise definition in their paper titled 'Towards precision medicine'. The paper centred on the reclassification of diseases using genetic information and presented a blueprint for establishing new data platforms that would merge genetic information with clinical data from specific patients (5). The paper acknowledged the promise of genomics as well as other developing technologies in studying the molecular characteristics of diseases and creating a more precise classification system for disorders. Currently, there is a widespread belief that the diverse nature of numerous illness processes indicates that the approach to treating a person with a disease, as well as monitoring or preventing that disease, should be customized or 'personalized' based on the individual's distinct biochemical, physiological, environmental exposure, and behavioural features (6). Several commendable evaluations on personalized medicine have been published, such as an increasing number of textbooks specifically designed for medical learners and doctors. Moreover, it will aid in diminishing medical expenses and enhance the likelihood of achievement in the advancement of groundbreaking pharmaceuticals, rendering it a highly anticipated sector in the future (7).

Autoimmune diseases (ADs) are complicated medical issues resulting from a combination of hereditary and environmental factors. Currently, there have been over 80 distinct autoimmune illnesses identified, and this figure is constantly growing (8). While there is now no proven remedy for ADs, many therapy approaches may be used to achieve disease remission. In addition to alternative treatments for some autoimmune diseases, including autoimmune thyroid disease, the symptoms and progression of autoimmune diseases may be managed by the use of pharmaceuticals like as corticosteroids, immunosuppressive medications, and non-steroidal anti-inflammatory agents (9). Nevertheless, these alternatives are linked to significant adverse reactions and may not consistently provide desired outcomes for persons with treatment-resistant conditions. As a result, biologics have been developed to specifically target a certain signalling route. While these therapeutic alternatives are groundbreaking for ADs, individuals exhibit variability in their response to therapy and may encounter contradictory results. Given the significant role of genetic variables in the development of ADs, it is reasonable to anticipate that the effectiveness and potential harm of biological agents, as well as traditional immunosuppressant medicines, may be anticipated by examining the genetic profiles of patients (9, 10). When evaluating several therapy choices for a specific AD, identifying the optimal choice for patients may enhance efficacy and reduce toxicity. This paper aims to investigate the adoption of PM in illness areas outside cancer and uncommon illnesses, where PM is already well-established. The objective is to progress the implementation of PM in the future. The research study included the following three diseases: Rheumatoid arthritis, an inflammatory condition, is now being investigated for treatment with antibody drugs and JAK inhibitors. Psoriasis, another autoimmune disorder, has seen the discovery of *IL-17* antibodies, *PDE4* inhibitors, *TNFα* antibodies, and *IL-12/23* antibodies, broadening the range of available treatments. The focus on Alzheimer's disease is currently on understanding its mechanisms, developing diagnostic techniques, and creating medications.

Introduction of Rheumatoid Arthritis

Rheumatoid arthritis (RA) is a persistent inflammatory autoimmune disease characterized by significant deterioration of cartilage and underlying bone, resulting in widespread pain among individuals globally. Early diagnosis may avoid joint injury and result in better long-term results (11). Extensive research suggests that significant and lasting joint damage may occur during the first two years of the illness. Therefore, it is crucial to have effective therapy for rheumatoid arthritis within the first three to six months (12). Hence, there is a want for dependable biomarkers that can provide timely detection, precise prediction of the course of a disease, and enhanced disease control. One of the most important features of RA is the crucial involvement of immune cells infiltrating the joint, which subsequently leads to bone erosions. Regarding target antigens, many categories of auto-antibodies have been identified as distinctive features of rheumatoid arthritis (RA); two notable examples are rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPA) (13). Additionally, it is believed that genetic predisposition accounts for around 50 to 60% of the susceptibility to RA, making it the most influential factor. The *HLA* genes are the most influential genetic variations that increase the likelihood of having RA. Specifically, the *HLA-DRB1* gene, which belongs to the *HLA* class II histocompatibility antigen-*DRB1-beta* chain, has a conserved amino acid sequence that is shared by many risk alleles linked with RA. Indeed, the *HLA* locus has mostly been linked to seropositive RA and elevated levels of antibodies (Abs) against

citrullinated proteins in the blood (14).

As previously stated, the occurrence of auto-antibodies, including RF and ACPA, is a distinctive trait of RA. They occur before the onset of illness symptoms and indicate the likelihood of developing the typical seropositive form of rheumatoid arthritis (RA); this is why these antibodies are believed to have a significant impact on the development of RA (15). Given that RA is a complex illness, its onset is influenced not only by hereditary factors but also by serological changes and external factors. Considerable resources have been allocated to comprehending the possible impact of particular environmental variables, including cigarette usage, periodontitis, particular infections, insufficient sunlight, or processed meals. Moreover, air pollution is currently a significant and relevant matter. Recent case-crossover research has demonstrated a correlation between severe air pollution, an elevation in the inflammatory marker C-reactive Protein (CRP), and the likelihood of relapses in RA (16).

Diagnosis and treatment strategies

According to the criterion, if there is inflammation with swelling (synovitis) in one or more joints and no other illness is determined to be causing the inflammation, four additional items are assessed: The factors that are considered are (1) the count of joints showing symptoms, (2) the presence of RF or ACPA, (3) the levels of CRP or erythrocyte sedimentation rate (ESR), and (4) the length of time the symptoms have been present. If the cumulative score for each of these four items is equal to or more than six, a diagnosis of RA is made and therapy with anti-rheumatic medicines is initiated. Nevertheless, it is imperative to do a comprehensive examination to determine the presence of other diseases before assigning a score, as disorders other than RA can also result in a total score of six or higher (17).

After diagnosing a patient with RA, the primary goal of treatment is to achieve complete remission or substantially reduce disease progression within around 6 months. This is done to avoid joint deterioration, disability, and systemic symptoms of RA (18). The need for timely and focused treatment for RA is emphasized by the fact that 80% of patients who receive inadequate treatment will experience joint misalignment, while 40% of patients will become unable to work within a decade of the commencement of the disease. To accomplish the desired therapeutic objectives, it is crucial to promptly commence therapy and maintain a continuous approach, while regularly evaluating both the progression of the disease and the efficacy of the treatment plan being implemented (19). Before the early 1990s, the standard approach for treating RA involved a treatment pyramid that included bed rest, the use of non-steroidal anti-inflammatory medicines (NSAIDs), and if these methods were ineffective, disease-modifying anti-rheumatic drug (DMARD) therapy. Nevertheless, the effectiveness of this therapeutic approach was restricted, and over time, RA often led to the deterioration of joints, disability, the inability to work, and higher mortality rates [20].

The primary objective of treating rheumatoid arthritis (RA) is to achieve clinical remission, a state commonly referred to as “treatment to target (T2T).” This approach aims to halt the advancement of joint damage and optimise long-term physical function (20). Methotrexate is recommended as the initial treatment for RA following a diagnosis of RA. For over two decades, methotrexate has been the primary treatment for RA in the United States. It is recommended to first use low dosages of glucocorticoids in conjunction with it to effectively and promptly decrease joint inflammation degrees. Using this initial treatment schedule, around 30% to 50% of patients with early RA can achieve either remission or a state of low disease activity. Methotrexate is not only highly effective, but its safety and toxicity profile is also well-established (21). Additionally, the expenses of treatment with methotrexate are quite inexpensive when in comparison with targeted synthetic or biological DMARD treatment. Sulfasalazine, initially developed in Sweden during the 1930s, was incorporated into RA treatment due to its antibacterial properties and the perceived role of bacterial or viral infection in the development of RA (21). Sulfasalazine, a pro-drug, is metabolized by gut bacteria in vivo into its two active components: sulfapyridine and 5-aminosalicylic acid. It is effective in treating moderate RA. Although the precise way in which they work is not understood, both sulfapyridine and 5-aminosalicylic acid have been demonstrated to possess anti-inflammatory, immune-modulatory, and antibacterial characteristics. Nevertheless, sulfapyridine has been proposed as the primary medicinal ingredient of sulfasalazine (22, 23). Chloroquine and hydroxychloroquine, primarily utilised as antimalarial medications, also have anti-inflammatory properties and immunomodulatory agent characteristics that make them appropriate for treating moderate cases of inflammatory arthritic conditions. It is important to mention that hydroxychloroquine, when employed in the management of RA, has been found to have only a modest impact on the structural damage to the joints (24).

Nonsteroidal anti-inflammatory drugs (NSAIDs), such as aspirin, diclofenac, and ibuprofen, are useful in reducing pain and swelling and improving joint function. However, it is important to note

that they cannot modify the underlying condition and prevent further damage to the joints. The anti-inflammatory effects of NSAIDs can be primarily due to their inhibition of prostanoid production through a mechanistic process. Prostanoids, including prostaglandin (PG) E2, PGD2, PGF2α, thromboxane A2, and prostacyclin, are molecules that serve as second messengers. They bind to and activate G-protein coupled receptors on the cell surface, which in turn regulate many cellular processes. Although NSAIDs efficiently alleviate symptoms of RA, their use often leads to adverse effects on the kidneys, liver, gastrointestinal system, and cardiovascular system (25, 26).

Obstacles in the field of PM

Although biologics have superior efficacy in treating rheumatoid arthritis (RA) compared to conventional drugs, the response to treatment varies across individuals. Not all patients attain a cure after the initiation of treatment. Although remission induction may be attainable, maintaining relapse proves to be a formidable task for all individuals (27). Furthermore, only a restricted minority of patients can sustain remission even after undergoing remission and subsequently discontinuing the medication. Immunity and inflammation are influenced by multiple factors, including a variety of immunocompetent cells and chemicals, like cytokines. Currently, there is no established approach for personalized therapy in this area. Regarding biologics, numerous studies have examined the genetic correlation between therapeutic impacts of drugs, although no genetic mutations or polymorphisms have been identified as definitive indicators (28). No indication indicators have been detected in research evaluating genetic biomarkers for TNFα inhibitors. RA includes a hereditary component, and it is classified as a multifactorial disease. This means that the disease is caused by the combined effects of several environmental and genetic variables. Among the genes linked to disease susceptibility that have been discovered so far, HLA is the most consistently connected across different populations. While the relationship between the shared epitope of HLA-DRB1 and the susceptibility, severity, and clinical manifestation of RA has been established, the specific molecular mechanisms behind this connection are still unknown. Moreover, numerous studies indicate a correlation between HLA-DRB1 and ACPA-positive individuals, making it a highly sought-after clinical biomarker.

Introduction of psoriasis

Psoriasis is a persistent skin condition that results from an over-activated immune system, characterized by inflammation. It is linked to several health conditions like psoriatic arthritis, mental health issues, heart disease, and liver problems. In 2014, the World Health Organisation officially acknowledged psoriasis as a significant non-communicable disease (29). They emphasised the emotional suffering caused by misdiagnosis, insufficient treatment, and the social stigma associated with this condition. In 2016, the Global Burden of Disease Study found that psoriasis caused 5.6 million disability-adjusted life-

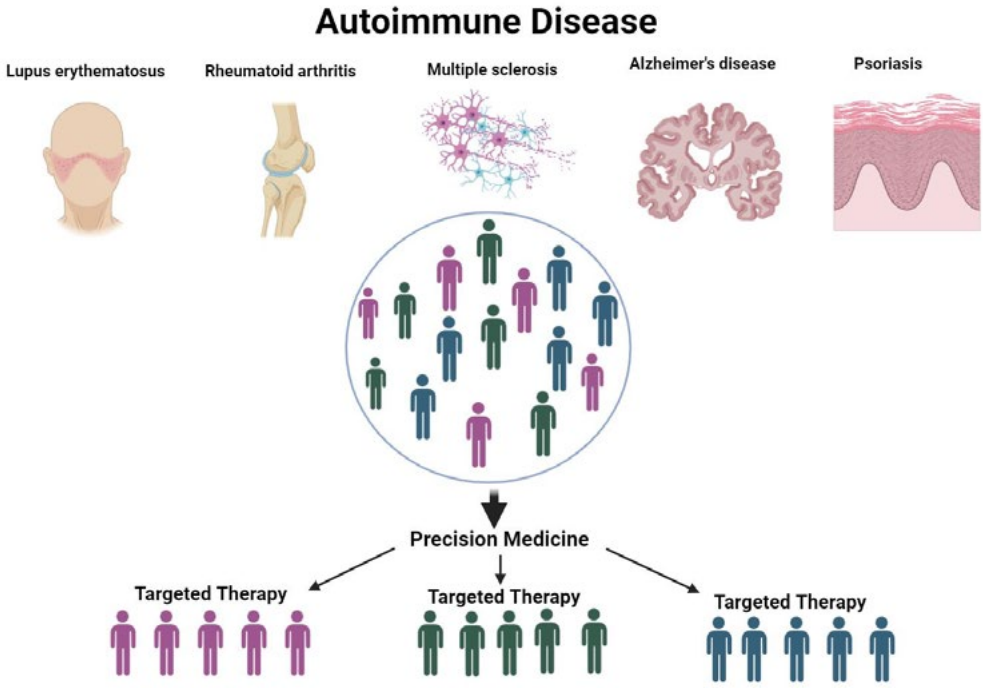

Fig1. Using a personalized medicine approach in the targeted treatment of autoimmune diseases

years (DALYs) across all age groups. This is at least three times more than the number of DALYs caused by inflammatory bowel disease. Psoriasis impacts individuals of both genders, with females and those with a familial background experiencing an earlier development of the condition. The age at which it often begins has a bimodal distribution, with the highest number of cases occurring between the ages of 30 and 39, and between the ages of 60 and 69 in men. In women, the onset tends to occur around 10 years earlier (30).

The development of psoriasis involves multiple factors, with genetics playing a significant role, particularly in individuals who develop plaque psoriasis before the age of 40. This was evidenced by twin, family-based, and large-scale population-level studies, where heritability has been calculated to vary from sixty per cent to ninety per cent (31). The probable causative genes implicated in this study are associated with several biological processes, including antigen presentation (*HLA-C* and *ERAP1*), NF-kappa B signalling (*TNIP1*), type 1 interferon pathway (*RNF113* and *IFIH1*), interleukin (*IL)-23/Th17* axis (*IL23R, IL12B,* and *TYK2*), and skin barrier function (*LCE3*) (31). The pathogenesis of psoriasis involves an intricate interaction between T cells, dendritic cells, and keratinocytes. The *IL-23/Th17* axis plays a major role in activating the immune system, causing persistent inflammation, and promoting the development of keratinocytes. Psoriasis can be worsened by various environmental factors, including being overweight, anxiety, beta-blockers, cigarette smoking, and lithium (32).

Psoriasis is characterised by persistent inflammation that causes uncontrolled growth of keratinocytes and impaired differentiation. The histology of the psoriatic plaque reveals acanthosis, which is an excessive growth of the outer layer of the skin (epidermis) (33). This excessive growth is accompanied by an accumulation of inflammatory cells in the deeper layer of the skin (dermis), including dendritic cells, macrophages, T cells, and neutrophils. Neovascularization is a major characteristic as well. The inflammatory pathways present in plaque psoriasis and other clinical variations have similarities but also exhibit distinct variances that contribute to the varying characteristics and treatment responses (33).

Diagnosis and treatment strategies

The identification of psoriasis is mostly based on clinical observations. Psoriasis has various clinical forms, with the most prevalent being chronic plaque psoriasis, which affects 80% to 90% of individuals with psoriasis. Classic plaque psoriasis is characterized by distinct, balanced, and red plaques with a layer of silvery scale on top. Plaques are commonly found on the scalp, trunk, buttocks, and extremities, however, they can appear on any part of the body. Patients may exhibit nail involvement, which may happen without accompanying plaques (34). Active lesions may cause pruritus or discomfort. Psoriasis can sometimes manifest as an isomorphic reaction, characterized by the development of new lesions in previously unaffected skin that has experienced stress or injury. The disease's severity is a useful factor in determining how to manage it, and it is categorised as mild, moderate, or severe (35).

The primary remedies for psoriasis are topical therapy with ointments, phototherapy with ultraviolet irradiation, and systemic therapy with small molecules and biologics. Granulocyte and monocyte adsorption depletion therapy may be employed as a treatment for pustular psoriasis (35). The management of psoriasis follows a systematic approach, starting with the use of topical medications such as corticosteroids and vitamin D analogues for mild to moderate signs. For moderate to severe signs, treatment options include phototherapy, oral medications containing small molecules, and biologics. Oral therapy for this condition involves the administration of immunosuppressants like cyclosporine and methotrexate, as well as *PDE4* inhibitors like apremilast. Biologics, on the other hand, utilise antibodies including *TNFα*, *IL-12/23, IL-17,* and *IL-23* inhibitors for therapy. These biologics are administered based on the patient's observations. Data suggest that *TNFα* and *IL-17* antibodies have a stronger efficacy against psoriatic arthritis compared to *IL-12/23* antibodies. Additionally, *IL-17* antibodies may worsen the efficacy of inflammatory illnesses (IBDs). The biologics were found to exhibit a significant therapeutic efficacy, however, approximately 20-30% of patients did not experience any discernible impact (36).

Personalized therapeutic approach for psoriasis based on individual biological factors

The effectiveness of anti-*TNF* treatment was assessed by examining the expression of various genes, including *HLA*: Cw6, the *TNF* cytokine, and its receptor. The *HLA*: C:06:02 gene was historically linked to positive results, however, subsequent investigations have raised doubts about this association. These disparities may be explained by the wide variety of genetic investigations and the variations in allele frequencies among different ethnic communities (37). The study examined various polymorphisms (specifically single nucleotide polymorphisms [SNPs]) in the *TNF* gene, including *rs1800629*, *rs1799964, rs1799724*, and rs361520. The results of the investigation yielded conflicting outcomes. Various research has examined the involvement of *TNF* receptor superfamily member

1B (*TNFRSF1B*) in connection to different anti-TNF drugs. The *TNFRSF1B rs1061622-TT* variant was found to be linked to a more favourable response to all anti-*TNF* treatments, and particularly to etanercept, with statistical significance. In contrast, the *rs1061622-G* polymorphism was linked to a more unfavourable outcome (38).

Ustekinumab has been extensively researched in terms of pharmacogenetics, perhaps because it has been on the market for a long time and is widely prescribed globally. The impact of *HLA*: *C:06:02* status on the response to ustekinumab has been extensively studied. Multiple studies have conclusively demonstrated that this specific allele is strongly linked to a favourable clinical outcome and a rapid response to treatment, regardless of a person's ethnicity. Consistent findings were derived from many investigations conducted on Caucasian, American, and Chinese participants. However, a subsequent meta-analysis raised doubts about the significance of the findings, as both patient groups, namely those who tested positive (*HLA: C:06POS*) and negative (*HLA: C:06NEG*), showed high response rates, albeit with modest variations. Therefore, it is advisable not to just rely on this allele when making treatment decisions. Instead, a combination of biomarkers could provide a more dependable assessment. The study evaluated polymorphisms on the *IL17* isoforms, *IL12*, and *IL23R* genes, but did not find any statistically significant results (38, 39).

Introduction of Alzheimer's Disease

Alzheimer's disease (AD) is a degenerative neurological disorder that primarily affects older individuals. The majority of patients exhibit initial memory impairment, and as the condition progresses, they will also experience language impairments, disorientation, and anxiety-related behaviours. Late-stage patients exhibit anomalous mental activity, including cognition, emotion, and behaviour, as well as a gradual loss of body functioning (40). Due to societal development and shifts in the human environment, the prevalence of AD has been steadily increasing throughout the years. The Alzheimer's Association conducted an epidemiological survey in the United States in 2017, revealing that the number of AD patients surpassed 5.5 million. Additionally, individuals aged 65 and above were shown to have double the likelihood of experiencing AD. Nevertheless, the underlying cause of AD is still not fully understood, and there are now no medications available that may effectively eradicate AD or alleviate its symptoms (40).

AD is caused by both physical and functional harm to the central nervous system (CNS), which involves the abnormal accumulation of proteins in the nervous system and the deterioration of nerve cells. Two distinct types of abnormalities have been observed in AD: amyloid plaques consisting of beta-amyloid peptides (Aβ), which gather abnormally exterior nerve cells, and neurofibrillary tangles (NFT) caused by the excessive phosphorylation of tau protein, which aggregates within neurons. AD can be viewed as a gradual series of biochemical, neurophysiological, neuroanatomical, and cognitive dysfunctions. The early aggregation of soluble Aβ in the brain leads to specific impairments in dendrites, axonal processes, and synapses (41).

In the past few decades, scientists have demonstrated an increasing fascination with neurological signs and behavioural disorders, including psychotic symptoms, sadness, apathy, aggressiveness, and sleep abnormalities. In 1996, the International Psychogeriatric Association introduced the concept of behavioural and psychological symptoms of dementia (BPSD) to describe the common symptoms of perception, thinking content, mood, and behaviour disturbances that often occur in individuals with neurocognitive disorders (ND). AD can be understood as a series of chemical, physiological, and structural alterations in the brain that can be detected several years before the appearance of clinically observable cognitive-behavioural symptoms (CBS) (42).

Current treatments strategies

Existing pharmacological interventions for AD mostly focus on managing the symptoms rather than providing a cure. The aim is to slow down the advancement of cognitive symptoms and address the behavioural and psychosocial symptoms of dementia (BPSD). There are four medications (donepezil, memantine, galantamine, rivastigmine) that have been approved for use and they can be classified into two categories: anticholinesterase blockers and anti-glutaminergics. These therapies are administered orally or through the skin (43). Anticholinesterase inhibitors are specifically engineered compounds that enhance the concentration of acetylcholine in the brain. Acetylcholine is a crucial chemical involved in transmitting data between specific neurons and is also involved in memory processes. These therapies aim to rectify the shortage of acetylcholine exhibited in the central nervous system of individuals with Alzheimer's disease. Anti-glutaminergics are employed to modulate glutamate levels by exerting a noncompetitive antagonist impact on N-methyl-D-aspartate (NMDA) receptors. Glutamate is a neurotransmitter that plays a crucial role in cognitive processes such as learning and memory (44). Non-pharmacological therapies, alongside medication, offer an additional approach to treating

neurodegenerative illnesses. Numerous research and worldwide experiments have been conducted or are currently underway to study the multidomain intervention in AD. This strategy involves the use of many activities. Studies have demonstrated a clear link between higher levels of physical exercise, cognitive training, better nutrition, and a reduction in cognitive and functional decline, as well as the severity of BPSD (45).

A personalized medicine approach in the management of AD

Aside from traditional clinical indicators, which aid in distinguishing between distinct conditions and evaluating the likelihood of concurrent illnesses, the most valuable biomarkers for predicting or confirming a diagnosis of Alzheimer's disease before death include genomic markers, epigenetic biomarkers, neurotransmitters, and levels of Aβ/tau in the brain in bodily fluids. Innovative biomarker studies aim to uncover distinct diagnostic, prognostic, and predictive biomarker traits, similar to the strategy used in oncology (46). This will be done in combination with SB (system biology), with the goal of customising therapy for each patient. Furthermore, biomarker-guided precision medicine eliminates the conventional method of trial and error in pharmacological therapies. This has significant medical implications for patients and healthcare institutions. The concept of PM aims to tailor medical treatment to the individual patient's distinct genetic, physiological, and clinical characteristics of the condition. It aims to tailor sickness prevention and therapy to the specific biological composition of each individual (customised treatment), which stands in stark contrast to the present "one size fits all" strategy (47). Due to the highly intricate nature of AD, it is highly improbable to discover a solitary medicine that can effectively treat every patient. Additionally, other disciplines including oncology and cardiology are also impacted. An effective PM strategy for AD therapy necessitates the comprehensive utilisation of genetics to provide personalised guidance. AD is related to about 600 human genes. Amyloid precursor protein (APP) mutations, with over 50 distinct variations, as well as presenilin 1 (*PSEN1*) mutations, with over 300 variations, and presenilin-2 (*PSEN2*) mutations, with over 40 variations, are found in a percentage of AD patients (5-10%) (48, 49). These mutations cause the development of brain amyloidopathy. The presence of over 100 mutations in the microtubule-associated protein tau (*MAPT*) gene, which are also found in certain patients with AD, can lead to brain tauopathies such as frontotemporal dementia and Pick's disease. Amyloidopathy and tauopathy are the two main pathogenesis hypotheses in Alzheimer's disease. Genetic factors significantly contribute to the development of AD, accounting for around 60 to 80% of cases (50). It is believed that multiple genetic factors, known as polygenic factors, are associated with the onset and progression of AD. The E4 allele of the Apolipoprotein E gene (*APOE*) on chromosome 19 is recognised as the most significant genetic risk factor for sporadic Alzheimer's disease (AD). Apolipoprotein E is one of the molecules responsible for the formation and aggregation of amyloid beta peptides. The APOE genes consist of three primary variants, namely ε (epsilon) 2, ε3, and ε4, which are present in pairs. When studying the correlation between the presence or absence of ε4 and the start of AD, individuals with one or two ε4 genotypes have a risk of getting AD that is around 3 to 12 times higher than those with no ε4 genotype (51). Nevertheless, numerous more genetic risk factors remain unidentified. Overall, genotype-specific techniques can benefit patients by utilising specific methodologies and focused methods that have been proven to be very beneficial for individuals with comparable genotypes. Further inquiry is necessary to uncover the impact of APOE on different physical activities, nutritional preferences, and lifestyle modifications as the personalised medicine approach to Alzheimer's disease prevention progresses (51).

Conclusion and Future Perspectives

Advancements in personalised medicine using genomic panel diagnosis are anticipated to be increasingly utilised in the field of oncology. Despite these diagnoses, there remains a subset of patients, approximately 10-20%, who are eligible for targeted medication selection and ongoing participation in clinical trials. Among these individuals, about half are expected to experience clinical benefits. This approach aims to promptly administer drugs that are predicted to be more efficacious. Nevertheless, there is a scarcity of evidence that unequivocally demonstrates the enhancement of patient's quality of life in real-world clinical settings. We are eagerly anticipating additional research and development of novel treatment medicines that align with the advancements in genetic analysis and diagnosis. This review examines the advancements in precision medicine and emphasises the significance of personalised medicine in diseases such as systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and Alzheimer's disease (AD). Precision medicine is achieved by categorising patients according to specified attributes and providing treatment that is precisely customised to meet their requirements. When both of these characteristics are effectively executed, patients are more inclined to experience favourable outcomes. The prospect of precision medicine in Alzheimer's disease is

highly promising, as ongoing research consistently uncovers novel biomarkers and focused therapeutic approaches. Utilising PM can enhance the precision of diagnoses, resulting in prompt intervention and potentially superior results. An area of research that shows promise is the utilisation of genetic biomarkers to categorise different subtypes of AD, enabling more customized treatment strategies. The aetiology of RA and psoriasis remains elusive, and the primary approach to treatment involves disease-modifying medicines that seek to ameliorate symptoms and impede disease progression. We are eagerly awaiting a more detailed understanding of how genetic backgrounds, environmental variables, and the immune system contribute to the development of these illnesses. To further our understanding of diseases, improve classification systems, and with proceed the study and creation of new medications, we hope for future progress in the fields of genetic analysis, complete analysis of gene and protein expression, analysis of epigenetic factors, genomic analysis of the gut microbiome, and the research and development of new biomarkers including miRNA.

Treating autoimmune disease with personalized medicine is more complicated than treating cancer using this method for at least three reasons. Historically, progress in the development of targeted therapies for autoimmune disorders has been slower than that of drugs for malignant tumors. Nevertheless, the authors are optimistic about the potential for change, noting the continued progress of several specific treatments. In addition, obtaining tissue samples from people with autoimmune diseases poses more logistical challenges compared to taking samples from people with cancer. Biopsies are commonly conducted in the management of malignant tumours to definitively diagnose the pathology and provide accessible samples for laboratory testing. Unlike joint biopsies, numerous autoimmune disorders, like rheumatoid arthritis, can be managed without the need for a joint biopsy. This complicates the task of identifying the genetic and metabolic variations that contribute to the disease in specific patients. Furthermore, it is worth noting that certain genetic mutations have been identified in many types of cancer. These mutations have been confirmed through the analysis of patient DNA and/or RNA expression. Consequently, they serve as highly advantageous targets for precision medicine strategies.

In contrast, the process is more intricate in autoimmune disorders since, in many instances, there is no singular dominant mutation. Consequently, it is challenging to select a viable target for therapy.

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Authors' Contribution

Yasaman Vojgani and Niusha Zeynalniya Toosi were involved in the conceptualization, design, and support of the study. All authors read and confirmed the final manuscript.

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Competing interests

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