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The Imperative of Implementing Precision Medicine in the Context of Diabetes and Treatment

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Abstract:
Precision medicine is a medical approach that involves customizing therapy for an individual by using extensive biological and external data. The advancements in the fields of molecular biology, genome sequencing, artificial intelligence, and other technologies have greatly enabled the use of precision medicine. This approach utilizes the wealth of comprehensive information obtained from these advancements to improve the decision-making process in clinical treatment for individuals, particularly in real-time scenarios during the progression of a disease. Diabetes is a prominent global health concern that necessitates the adoption of innovative approaches to improve patient outcomes. The efficacy of conventional treatment options that use a uniform approach has been shown to be limited in effectively addressing the heterogeneous character of the illness. In contemporary times, the concept of customized medicine has emerged as an innovative option, tailoring methods of therapy based on a person's medical characteristics, lifestyle preferences, and genetic background. This review emphasizes the significance of genetic evaluation in forecasting the vulnerability to diabetes and its response to treatment, while also emphasizing the potential of pharmacogenomics in improving the choice of drugs.

INTRODUCTION

Diabetes mellitus (DM), also known as diabetes, is an intricate and persistent metabolic disorder that has become a prominent global health issue. The issue at hand has a great worldwide impact due to its effects on broad populations and the high health concerns it presents. Consequently, it places a tremendous strain on health systems (1). Diabetes is a health disorder characterized by high levels of glucose in the blood, which occurs due to insufficient synthesis of insulin or reduced insulin efficacy. This condition has the capacity to have significant repercussions that affect several organ functions and greatly reduce the general condition of life for individuals affected by disease. Like we delve into the detailed features of diabetes and its global occurrence, the introduction of personalized therapy arises as a potentially beneficial possibility in the field of healthcare. The unique technique has significant promise for revolutionizing the management of diabetes and improving individual results. The prevalence of diabetes has risen to substantial magnitudes, becoming it a prominent worldwide health concern (2). The International Diabetes Federation (IDF) said that the worldwide incidence of diabetes in adulthood in 2019 was anticipated to exceed 463 million persons. It is anticipated that this figure will undergo a substantial rise, with an estimated 700 million persons being impacted by the year 2045 in the absence of prompt intervention. The rise in the prevalence of diabetes may be attributed to various reasons, including a lack of exercise, bad dietary patterns, escalating rates of obesity, and the advancing age of the worldwide population. Diabetes is a widespread medical disorder that affects persons of many age groups, races, and socioeconomic backgrounds (3). However, the impact of the illness is particularly significant among impoverished and middle-class people living in countries with limited healthcare, education, and resources, therefore worsening its serious consequences. Diabetes contributes to the continuation of health inequalities by worsening differences in healthcare results throughout various regions and demographic groups (4).

With the rising occurrence of diabetes, it is clear that the traditional strategy of employing a single

technique for managing the condition has shown its fundamental limitations. Diabetes is a complex health disorder that shows many clinical manifestations and has personal differences in response to therapy. The effectiveness of conventional treatment protocols may be restricted in meeting the unique requirements and distinguishing characteristics of individual patients, leading to suboptimal outcomes and difficulties in achieving optimal glucose management. The notion of precision medicine, sometimes referred to as customized medicine, has gained prominence as a very promising alternative in recent years (5). Individualized medicine is a therapeutic approach that involves customizing treatment tactics and procedures to match the distinct characteristics of each patient. The aforementioned attributes involve genetic composition, individual lifestyle choices, external environmental factors, and distinct health-related traits. By using a personalized strategy, healthcare practitioners may optimize treatment choices, therefore enhancing the effectiveness of interventions and promoting the overall well-being of patients (6). An essential aspect of personalized medicine in the area of diabetes therapy is in its ability to uncover the genetic factors that contribute to a person's vulnerability to the disease. Genetic analysis allows for the timely detection of individuals who may have an increased vulnerability to diabetes, therefore facilitating the implementation of targeted preventive measures and adjustments to their behavior (7). Moreover, the comprehension of genetics provides substantial aid to medical professionals in identifying the most appropriate pharmacological therapies for specific people. This intervention aims to mitigate the probability of negative consequences and the inefficacy of treatment (8). Personalized medicine involves the use of modern technology, such as continuously glucose measurement and portable devices, that allow people to actively participate in managing their diabetes. By using real-time data and offering personalized feedback, people are empowered to make educated decisions about their food choices, physical activity, and adherence to prescribed prescriptions (9). This method facilitates the development of empowerment and the assumption of responsibility for individual health (5).

REVIEW

Introduction of diabetes mellitus

Diabetes mellitus (DM) is a persistent metabolic disorder defined by abnormally high amounts of glucose in the bloodstream. This condition may be caused by either inadequate synthesis of insulin by the body or impaired use of the insulin produced (10). Type 2 diabetes is the prevailing manifestation of diabetes, frequently observed in adulthood. It is characterized by the body's reduced sensitivity to insulin or insufficient production of insulin. Common symptoms are heightened urination rate, excessive thirst, and enhanced hunger. Uncontrolled diabetes may give rise to several complications (11). Acute consequences associated with diabetes may include diabetic ketoacidosis, hyperosmolar hyperglycemic condition, or mortality. The syndrome is linked to severe and long-lasting consequences such as cardiovascular illness, stroke, persistent renal illness, foot wounds, neuropathy, visual damage, and cognitive impairment (11).

Type 1 diabetes

Type 1 diabetes (T1D) is an autoimmune condition characterized by the immune system's erroneous assault and elimination of the pancreatic beta cells located in the islets of Langerhans. Patients confirmed to have type 1 diabetes have a significant decrease as well as complete absence of insulin production, necessitating lifelong administration of insulin on a daily basis (12). While autoantibodies targeting islet cell components are reliable indicators of the disease process, research suggests that the damage to β cells is mostly caused by the cytotoxicity of T cells and the production of cytokines, in conjunction with disease processes occurring inside the β cell. Hyperglycemia arises from a gradual decline in the body's ability to generate insulin, resulting in elevated amounts of glucose in the circulatory system. This condition may manifest at any stage of life, but the average age of diagnosis is about 12 years. The exact cause of T1D is not fully understood, but current knowledge indicates that it is likely to have a complex genesis involving a combination of genetic and environmental variables. Specific genetic variables have been shown to be associated with an increased susceptibility to T1D. Nevertheless, it is essential to acknowledge that the initiation of an autoimmune response may need an exogenous trigger, such as a viral infection (13). People with T1D have an autoimmune response in which their immune system mistakenly identifies pancreatic beta cells as foreign and proceeds to destroy them. The immunological reaction in issue involves the production of autoantibodies that specifically identify and attach to proteins situated on the external membrane of beta cells. The gradual reduction of beta cells results in a continuous decline in the manufacture of insulin, eventually causing insufficient regulation of blood glucose levels (14).

Manifestation of type 2 diabetes

Type 2 Diabetes Mellitus (T2DM) has traditionally been referred to as non-insulin dependent diabetes or adult-onset diabetes. Insulin resistance, that might ultimately progress to total resistance, is the defining

characteristic. However, in recent years, diminished β-cell function has been identified as a significant issue in T2DM (15). Undoubtedly, during the course of the last two decades, T2DM has surfaced as a novel and exceedingly consequential health concern, even within the pediatric population. The study done on young patients has shown the simultaneous occurrence of obesity, insulin resistance, and β-cell disorder, which is consistent with the findings seen in older persons with T2DM (15). T2DM is characterized by insulin resistance, a disease in which the organisms in the body do not respond properly to the physiologic effects of insulin. During the first phases, the pancreas demonstrates an increased secretion of insulin in response to immune system activity, therefore attempting to maintain blood glucose levels within an optimal range (16). Metformin is considered the first pharmacological treatment option for T2DM. In addition to its glucose-lowering properties, this intervention has insulin-sensitizing effects that impact several tissues, including the liver, skeletal muscle, endothelium, adipose tissue, and the ovary. A second medicine may be considered for addition if, after a period of three months, the levels of HbA1c exceed 7.0%. Unfortunately, metformin is associated with a range of adverse effects, varying in severity, which may lead to poor adherence. Consequently, it is considered to have the lowest compliance rate among oral therapies for diabetes. Nevertheless, it is vital to comprehend that the onset of this ailment is mostly shaped by lifestyle and environmental circumstances (17).

Gestational diabetes

Gestational diabetes mellitus (GDM) is a medical disorder when glucose levels in the blood are higher than normal during gestation. It is diagnosed based on specific criteria outlined by the International Association of Diabetes and Pregnancy Study Groups (IADPSG) and the World Health Organization (WHO) (18). The criteria consist of fasting plasma glucose concentrations of 5.1 mmol/L or higher, 1-hour plasma glucose levels of 10 mmol/L or higher, and 2-hour plasma glucose levels of 8.5 mmol/L or higher. These values are evaluated through a 75 g the tolerance for oral glucose test. GDM is often identified for the first time during pregnancy (18). GDM is a prevalent medical problem associated with pregnancy, and insufficient management of this condition may result in significant detrimental health consequences for both the mother and the child. Gestational diabetes is a medical disorder that is distinguished by compromised insulin function resulting from the presence of placental hormones, leading to elevated levels of blood glucose. Generally, gestational diabetes tends to recover after giving delivery. However, women who have previously had this illness are more likely to acquire T2DM in the future (19). Various hormones, such as human placental lactogen and progesterone, may cause insulin resistance, hence reducing the ability of maternal cells to absorb glucose. Consequently, the levels of glucose in the bloodstream rise in order to deliver the necessary nutrients required to sustain the growth of the growing embryo. Normally, the mother's pancreas increases its secretion of insulin to overcome insulin resistance. Nevertheless, in certain women, this compensatory mechanism is inadequate, resulting in the occurrence of diabetes during pregnancy (19).

THE WORLDWIDE IMPACT OF DIABETES

Based on the latest edition of the International Diabetes Federation (IDF) Diabetes Atlas 2017, it is evident that the prevalence of diabetes is increasing globally. According to the specified criteria, the estimated global incidence of diabetes in 2017 was 8.8% (95% confidence interval (CI) 7.2–11.3%) among those aged 20 to 79 years (20). It is projected that this prevalence would rise to 9.9% (95% CI 7.5–12.7%) by the year 2045. The global population of individuals with diabetes in 2017 was estimated to be 424.9 million (95% CI 346.4–545.4 million), with a projected growth of 48% to reach 628.6 million people (95% CI 477.0–808.7 million) by the year 2045. The fact that a considerable percentage, over 50%, of diabetes patients remain undetected is cause for concern, as it underscores the profound effect of this condition (20). Diabetes significantly affects persons of all age groups, ethnicities, and socio-economic statuses. The incidence of diabetes has significantly risen in low- and middle-income nations, whereby individuals may have challenges in obtaining sufficient medical care and financial resources for managing diabetes. Diabetes has extensive ramifications that extend beyond individual well-being, imposing a substantial economic strain on healthcare institutions and the community at large. Notwithstanding substantial advancements in diabetes care, there exist many impediments which impede the efficacious therapy of this ailment (21). The primary cause of the rise in type 2 diabetes is mostly attributed to changes in habits, particularly the adoption of inactive behaviors and bad dietary patterns. The prevalence of obesity is a significant determinant in the onset of resistance to insulin and metabolic disorders. A significant proportion of patients with diabetes go undetected, leading to a lack of obtaining appropriate medical treatment and boosting the risk of adverse outcomes (22). The majority of individuals with diabetes are mostly affected by T2DM, which constitutes around 90% of the overall population. Those with this condition exhibit varying degrees of inadequate insulin production in connection to a broad range of

insulin resistance. Around 5% of persons diagnosed with diabetes fall into the monogenic diabetes group, which encompasses several subgroups of maturity-onset diabetes of the younger and other rare genetic illnesses. Another 5% of cases are classified as sub-forms of immune-mediated T1D, characterized by a significant, if not complete, long-term deficiency in insulin production (22).

The optimal management of blood sugar levels relies on several variables, including medication adherence, dietary choices, and engagement in physical activity. The inter-individual variances among individuals with diabetes exhibit significant variety in terms of their clinical manifestations and reactions to treatment. The conventional methodology, which operates on the assumption that a single solution can sufficiently cater to the requirements and obstacles of all patients, may be insufficient in effectively addressing the different array of wants and issues faced by individuals. The significance of individualized medicine is underscored by this statement. The rationale for using personalized strategies in the treatment of diabetes arises from the recognition of individual variations and influential variables that impact the course and response to the disease. Individualized medicine refers to a therapeutic strategy that customizes treatment choices based on an individual's unique genetic makeup, behavioral factors, and clinical traits. The aim of this strategy is to improve the efficacy and accuracy of medicines (23, 24).

DIABETES PHENOTYPE DIAGNOSIS

Historically, phenotypic features have been used to ascertain the specific kind of diabetes in a person, as per the categorization above. The attributes mentioned above include the age at which symptoms first manifest, physical body composition, reliance on insulin, and past occurrences of ketoacidosis. Relying just on the phenotypic technique may not enough to distinguish all cases of T1D from those of T2DM, since people belonging to both groups might display any extremity of several phenotype pairings (25). As an example, those diagnosed with autoimmune diabetes may also display obesity, whereas those with type 2 diabetes may show symptoms of ketoacidosis. The primary goal of using the precision medicine paradigm within the realm of diabetes is to augment comprehension of an individual's condition beyond mere clinical characteristics. This is accomplished by the utilization of laboratory examinations, including assessments of inherited, immunological, and metabolic processes indicators, alongside other pertinent data. The objective of this study is twofold: firstly, to provide valuable insights for treatment making choices, and secondly, to predict the trajectory of the illness and anticipate clinical consequences in various scenarios (25).

GENETICS AND DIABETES SUSCEPTIBILITY

DM is a metabolic disorder characterized by elevated levels of sugar in the bloodstream, caused by insufficient manufacture or reduced efficiency of insulin. The influence of our lifestyle variables on the development and progression of diabetes is generally acknowledged. However, it is vital to comprehend that genetics additionally have a substantial role in determining a person's vulnerability to this condition. Currently, significant advancements in genetic studies possess provided valuable knowledge about the genetic variables that increase the likelihood of developing diabetes (26). T1D is a medical condition characterized by the inability of the human system to produce insulin. T1D is largely recognized as an autoimmune illness, in which genetic variables play a crucial role in its onset. There is a significant correlation among certain human leukocyte antigen (HLA) genes, namely HLA-DR3 and HLA-DR4, and a heightened susceptibility to the development of T1D. These genes play a crucial role in controlling the immunological response and are accountable for identifying both self and non-self antigens. Genetic analysis of polymorphisms in the HLA genes has significantly influenced an individual's immune reaction, making them more susceptible to autoimmunity that specifically attacks pancreas beta cells (27). T2DM is a multifaceted condition that is shaped by a confluence of hereditary and environmental elements. Genome-wide association studies (GWAS) have discovered genetic regions that are linked to a higher vulnerability to type 2 diabetes. It is noteworthy that genes associated with the control of beta-cell function, including TCF7L2, KCNJ11, and HNF1A, as well as genes related to insulin resistance, such as PPARG, IRS1, and GCKR, have been implicated in this particular situation (28).

DIABETES MELLITUS WITH A SINGLE GENE: MODERN CUSTOMIZED HEALTHCARE IMPLEMENTATION

Certain types of monogenic diabetes now provide the potential for the use of tailored treatment. Maturity-onset diabetes of the young (MODY) is well recognized and extensively researched as the predominant and extensively investigated kind of monogenic diabetes. Currently, there exist a total of 13 distinct types of MODY, which are categorized based on the specific gene mutation responsible for the resulting phenotype. MODY is often seen in individuals with a low body mass index before to the age of 25. It develops in an autosomal dominant manner, and affected individuals continue to display signs of pancreatic cell functioning. Epidemiological research undertaken after the first definition of MODY indicates that the primary criteria fail to include all

instances (29). The prevalence of MODY in diabetes mellitus sufferers is estimated to be around 1-2%. Nevertheless, due to the presence of features associated with both type 1 diabetes (such as early start and lean body type) and T2DM (such as a family history of the condition and preserved pancreatic cell function), this particular presentation is often subject to misdiagnosis. The issue of misdiagnosis is particularly concerning because the most common forms of MODY have distinct pharmacogenetic guidelines determined by the underlying genetic causes (30).

In the extensively studied United Kingdom, MODY3 stands out as the greatest often seen variant of the illness, representing around 52% of all reported cases. Nevertheless, the incidence of MODY3 exhibits variations based on factors such as ethnicity and geographic location. A genetic alteration occurring in the transcription factor hepatic nuclear factor 1-α (HNF1-α), which is encoded by the HNF1A gene, leads to the activation of many genes linked to the processes of release of insulin, metabolism of glucose, and insulin synthesis. HNF1-α is 55% similar in amino acid sequence to hepatic nuclear factor 4-α (HNF4-α), an orphan protein with a mutation in MODY1. The interaction between HNF1-α and HNF4-α has been shown to occur in an epistatic way (31). Identifying MODY1 or MODY3 is crucial to providing appropriate treatment since these individuals have been seen to have heightened sensitivity to sulfonylureas. The observed hypersensitivity might perhaps be ascribed to a reduction in the transcriptional activity of genes under the regulation of HNF1 and HNF4 in the hepatic tissue (32). The decline in gene expression results in a reduction in the absorption of sulfonylureas, eventually leading to a sustained increase in the levels of these medications in the bloodstream.

Consequently, individuals diagnosed with MODY1 and MODY3 need a significantly reduced dosage of sulfonylurea medication, subject to inter-individual variability. Sulfonylureas have a notable degree of sensitivity, making them a preferred first therapeutic approach for individuals diagnosed with MODY1 and MODY3. Individuals diagnosed with both kinds of MODY retain their sensitivity to insulin due to the genetic etiology that leads to malfunction in the pancreatic cells. Prior studies have shown that the introduction of genetic diagnosis for MODY1, along with a shift in treatment from insulin to sulfonylureas, improved glycemic oversight, as indicated by the measure of %HbA1c (33).

Neonatal diabetes mellitus (NDM) is an additional kind of monogenic diabetes that is characterized by its potential for practical intervention. Neonatal diabetes mellitus (NDM) is typically identified during the initial six months of an infant's life, manifesting either as a temporary or permanent condition. The underlying causes of NDM can be attributed to various genetic factors, including KCNJ11, ABCC8, GCK, INS, ZFP57, and paternal duplication or hypomethylation of chromosome 6q24. Additionally, several other congenital abnormalities, such as EIF2AK3 and PTF1A, result in syndromic forms of NDM (34). NDM is mainly attributed to activating mutations in KCNJ11 or ABCC8, the two genes responsible for producing the subunits of the ATP-sensitive potassium channel found in pancreatic cells. These mutations hinder the process of membrane depolarization when the ATP: ADP ratio decreases, leading to a reduction in insulin production. A considerable proportion of individuals manifesting these genetic alterations may be efficiently managed with high-dose sulfonylureas for a viable substitute for insulin, which conventionally serves as the customary therapeutic approach for NDM (35). This alternative treatment option is more cost-effective and less invasive and demonstrates higher efficacy. Moreover, it reduces the likelihood of hypoglycemic episodes in individuals with these mutations. Sulfonylureas act by inhibiting the same channels that are continuously activated due to the presence of NDM mutations. These instances exemplify the significance of accurately diagnosing monogenic types of diabetes (35).

The findings from the SEARCH for Diabetes in Youth study indicate that a subset of study participants who lacked type 1-related antibodies and displayed intrinsic generation of insulin, as determined by C-peptide levels, exhibited alterations in one of the three most commonly observed MODY genes. Specifically, a total of 47 individuals, accounting for 8.0% of the sample, were identified with such mutations. It is worth noting that only three of these individuals had received a MODY diagnosis before the commencement of the study (36). As a result, it was found that a significant proportion (79%) of patients diagnosed with MODY were receiving therapy that did not align with the recommended course of action. The emergence and progress of next-generation sequencing methodologies have presented the potential for precisely identifying these hereditary conditions. Numerous studies have provided evidence for the efficacy of various sequencing systems in reliably identifying pathogenic mutations. Prior to the emergence of next-generation sequencing, the major focus of research on monogenic diabetes was directed on investigating the frequency and distinctive characteristics of MODY1, MODY2, and MODY3. Although this method adequately covers almost all of cases of monogenic diabetes, it does not include the rate or distinctive characteristics of fewer kinds of monogenic diabetes (37).

PHARMACOGENETICS

Pharmacogenetics studies how genetic variability influences several aspects of medication response, including the pharmacokinetic and pharmacodynamic profiles, the impact of polymorphisms in drug targets on therapeutic results, and adverse events. Genetic diversity in diabetes medicines may be associated with factors such as glycemic response, side effects, cardiovascular risk reduction, and progressive decrease of microvascular disease (38). The field of pharmacogenetics is primarily concerned with identifying individuals who are most likely to experience therapeutic benefits from a particular medicine and those who are most likely to avoid adverse side effects. There are two approaches to consider when examining the hereditary factors that might indicate how someone will respond to medication: One potential strategy is acquiring a thorough comprehension of the natural progression of diabetes and distinguishing the unique pathophysiological features that distinguish one group of patients from another in terms of their illness and the fundamental origins of their diabetes (39). This knowledge can then inform the selection of the most suitable and productive drug for the specific pathophysiology exhibited by each subgroup. The second method involves the identification of genotypes or other markers linked to changes in drug transport or drug metabolism. These changes can impact the exposure and effectiveness of drugs. By identifying individuals who have genotypes linked to modified drug responses, it becomes feasible to deliver drugs that are more likely to be efficacious and secure for these people (40). In 2018, Udler et al. conducted a cluster analysis on 14,183 individuals. The research examined 94 genetic variants of type 2 diabetes (T2D) and 47 metabolic characteristics linked to diabetes (41). The data used for the analysis were obtained from publically accessible genome-wide association study (GWAS) databases and biobanks. The researchers found five unique clusters of type 2 diabetes variations, which have significant biological relevance and reflect separate mechanistic pathways. Two groups are associated with the function of pancreatic β-cells, and an additional three clusters are associated with insulin resistance pathways. It was discovered that around 30% of the whole population had a genetic load that positioned them inside the uppermost 10% of one of these clusters (41). The researchers indicated that the subsequent phase of this analysis would involve investigating whether individuals who belong to one of the clusters would exhibit varying responses to medications that impact the disrupted pathway. Additionally, they would explore whether these individuals would demonstrate different rates of disease progression and the development of complications. Regarding the second method, it is worth noting that so far, most genes linked to an elevated susceptibility to diabetes have shown little correlation with varying reactions to different medications. The majority of research on the identification of varied responses to a diabetic medicine has been focused on the uptake and tolerance of metformin. However, a limited number of genes that possess cardioprotective qualities in the presence of glucagon-peptide 1 receptor agonists and sulfonylureas have been discovered (42).

STATE OF PRECISION MEDICINE CURRENTLY

The categorization of patients with diabetes based on the etiology, pathophysiology, and progression of the disease may provide potential advantages in terms of identifying the most effective treatment strategies. The categorization of diabetes is predominantly determined by various widely employed factors, such as the age of onset, the degree of islet cell dysfunction, the level of resistance to insulin, the existence of diabetes-related antibodies, and the presence of particular genetic variations (43). These criteria are used for the purpose of classifying individuals into one of the five established forms of diabetes, as previously delineated. However, it is important to acknowledge the potential for misclassification in patients, since the presence of many subgroups may not perfectly coincide with the diagnostic parameters for the five main categories of diabetes. Additionally, individuals who meet the defining standards may exhibit significant variations in subtypes (44).

Currently, there is a substantial amount of information accessible to enhance the categorization of diabetes kinds, in addition to the five conventional parameters used for classification. This information may be obtained from several sources. The data collection methods employed in this study encompassed four main areas (45). Initially, a series of patient questionnaires were administered in order to collect data pertaining to the inherent course of the condition. This included several elements, including family genealogy, ethnicity, psychological state, drugs, and lifestyle. Additionally, anthropometric measurements of bodily features were collected, using either conventional paper instruments or more sophisticated computerized technologies to enhance productivity. Thirdly, measurements of molecules or cells in the bloodstream or urine were obtained using conventional laboratory tests and biomarkers (44). This also included information on continuous glucose concentrations. Finally, behavioral measures were conducted in order to evaluate behaviors such as food consumption and physical activity, which were made available via the use of sensors. The categorization of patients often relies on the use of phenotypic and

diagnostic data. Nevertheless, the notion of precision medicine proposes that a more substantial depiction may be attained by including a wider array of assessments that comprehensively investigate crucial biological aspects. The domains that possess the capacity to engage with the environment encompass a range of aspects, including inherited variation (genomics), the characteristics that govern the activation or inhibition of genes in specific tissues (epigenomics), the degree of gene expression (transcriptomics), the proteins synthesized by specific genes (proteomics), the small molecules created through enzymatic processes (metabolomics), and the assemblage of microbial organisms that exist with the human organism (the metagenome) (45). Currently, the use of genetic markers in determining appropriate diabetes treatment is limited since they often lack the necessary level of specificity. However, it is essential to note that exceptions exist in the form of MODY and neonatal diabetes, which are distinguished by identifiable genetic alterations. Various professional groups are now trying to develop diabetic recommendations that may assist in making therapy choices and facilitate the customization of diabetes treatment. The options are determined mainly by the patient's condition and the potential good or negative consequences associated with various accessible drugs (46).

SIGNIFICANT INITIATIVES IN PRECISION MEDICINE

Since 2005, many precision diabetes projects have been introduced in the United States, Europe, Asia, and Australia. Public-private coalitions have often provided the funding for these initiatives. The Nordic Precision Medicine Initiative, which was launched in 2015, seeks to collect genomic and additional biological information from a population of over one million people in the Nordic region. This data is maintained in biobanks specific to each person. The Precision therapy in Diabetes Initiative, established by the American Diabetes Association (ADA), seeks to provide a comprehensive agreement document on precision diabetes therapy within a five-year timeframe. Additionally, this initiative will initiate supplementary endeavors in support of its objectives (47).

CONCLUSION

This literature review focuses on using personalized medicine in treating diabetes, providing insights into the possible benefits and challenges connected with this approach. The potential benefits of implementing customized treatment include improved glycemic control, increased patient involvement, and optimized medication selection. This approach involves tailoring healthcare programs to individuals by considering their unique genetic, lifestyle, and clinical characteristics. The importance of precision medicine in the realm of diabetes cannot be overstated, since it plays a pivotal role in guiding treatment decisions. Precision medicine utilizes a significant amount of omic and other information to create methods for disease care and improve treatment results. By prioritizing these particular areas, it is feasible to support a profound shift in the approach to diabetes treatment, leading to improved individual outcomes and a precise and efficient management plan.

Consent for publication

The authors of this paper affirm their agreement for its publication.

Conflict of interest

The writers assert that they possess no conflicts of interest.

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