International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2025: 7.089

Drug-Class-Oriented Pharmacogenomics in Type 2 Diabetes: Implications for Personalised Therapy

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Abstract: ***Type 2 Diabetes Mellitus is characterized by insulin resistance and impaired insulin secretion, with considerable interindividual variability in response to antidiabetic therapy. In addition to clinical and lifestyle factors, genetic polymorphisms significantly influence drug efficacy and tolerability. This review summarizes current pharmacogenomic evidence related to major antidiabetic drug classes, including metformin, sulfonylureas, thiazolidinediones, incretin-based therapies, SGLT-2 inhibitors, and insulin. Particular emphasis is placed on key genes such as PPARG, TCF7L2, KCNJ11, ABCC8, and SLC22A1, which have demonstrated clinically relevant associations with drug response. By correlating genetic variability with therapeutic outcomes, this review highlights the potential of pharmacogenomics to advance personalized treatment strategies in Type 2 Diabetes Mellitus.***

Keywords: Type 2 Diabetes Mellitus (T2DM), Pharmacogenetics, Drug Response Variability, Precision medicine, OCT1

1. Introduction

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder that is defined by high blood glucose levels due to a combination of insulin resistance and impaired insulin secretion. Over the past few decades, the incidence of T2DM has risen substantially worldwide, and it has become a serious public health issue. Lifestyle factors such as a sedentary lifestyle, unhealthy diet, obesity, and genetic factors are known to contribute to the development and progression of T2DM. If T2DM is left untreated, it has been established that it can lead to serious long-term complications, including heart disease, renal disease, neuropathy, and retinopathy (Tinajero & Malik, 2021).

The process of managing T2DM requires the administration of different classes of drugs used in the management of T2DM, which in turn exert different effects in controlling blood sugar levels. The anti-diabetic drugs include metformin, sulfonylureas, thiazolidinediones, incretin, Sodium-glucose cotransporter-2 inhibitors, and insulin. However, even though these treatment modalities have been in practice, it has been observed that the response to these treatment regimens varies greatly in the patients. There are certain patients who have shown desirable effects, while in other individuals, low response to treatment or even adverse effects have been observed. It cannot be attributed to factors like clinical as well as lifestyle factors (Lu et al., 2024).

Pharmacogenomics has emerged as a promising field. The area of pharmacogenomics mainly focuses on the impact of genetic variations on drugs. When considering T2DM, specific genes have been identified that follow the pattern of insulin secretion, sensitivity to insulin, and transporting drugs, which then impact the response to drugs. Such knowledge can always help to make an informed decision (Florez, 2017).

This review takes a pharmacogenomic approach with a focus on drug classes to provide an overview of the current state of knowledge on the role of genetic factors in the response to antidiabetic medications. Rather than providing an overview of a large number of genes, this review provides an overview of a shortlist of genes that have been shown to play a role in the response to the main classes of antidiabetic medications, including PPARG, TCF7L2, KCNJ11, ABCC8, and SLC22A1

2. Methodology

This review summarizes published evidence on pharmacogenomic determinants of antidiabetic drug response in Type 2 Diabetes Mellitus (T2DM). A structured literature search was conducted using electronic databases including PubMed, Scopus, and Google Scholar to identify peer-reviewed articles relevant to gene–drug interactions in T2DM.

Search terms were developed to capture studies examining the impact of genetic polymorphisms on therapeutic efficacy and safety of major antidiabetic drug classes. Keywords included combinations of "Type 2 Diabetes Mellitus," "pharmacogenomics," "pharmacogenetics," "gene–drug interaction," "drug response variability," "PPARG," "TCF7L2," "KCNJ11," "ABCC8," "SLC22A1," "metformin," "sulfonylureas," "thiazolidinediones," "DPP-4 inhibitors," "GLP-1 receptor agonists," "SGLT-2 inhibitors," and "insulin."

Articles were considered eligible if they reported clinically relevant associations between genetic variants and variability in response to antidiabetic medications. Included study designs comprised clinical trials, pharmacogenetic association studies, systematic reviews, meta-analyses, and well-characterized observational studies. Only articles published in English between 2007 and 2024 were included to ensure representation of both foundational and contemporary evidence.

The selected literature was evaluated based on its relevance to gene–drug interactions and organized according to major antidiabetic drug classes. Evidence was synthesized

qualitatively to provide a drug-class-oriented pharmacogenomic perspective highlighting implications for personalized therapy in T2DM.

3. Overview of Antidiabetic Drug Classes

3.1 Metformin

Metformin is the first-line medication for the treatment of Type 2 Diabetes Mellitus. The mechanism of its action is mostly through the reduction of glucose levels in the liver and the increase in the sensitivity of body tissues to insulin. This method effectively lowers blood sugar levels without causing hypoglycemia. In contrast, the digestive system's side effects and how well it works can vary from person to person (Foretz et al., 2019).

3.2 Sulphonylureas

Sulphonylureas act as insulin secretagogues, which means they help the pancreas release more insulin. They do this by closing potassium channels, which then causes the pancreas to release more insulin.

However, this type of medication is effective but associated with hypoglycemia and weight gain. In addition, differences have been observed in the effect of the medication among different patients, mainly due to the function of beta cells (Thulé & Umpierrez, 2014).

3.3 Thiazolidinedions

Thiazolidinediones work by increasing glycemic control through increased insulin sensitivity in peripheral tissues, especially adipose tissue and skeletal muscle. They work by activating the peroxisome proliferator-activated receptor gamma (PPARγ), a nuclear receptor that regulates glucose and lipid metabolism. Although they are effective in the management of insulin resistance, they are associated with side effects such as weight gain, edema, and cardiovascular events. There are also interindividual differences in response to thiazolidinediones (Soccio et al., 2014).

3.4 DPP-4 Inhibitors

Dipeptidyl peptidase-4 (DPP-4) inhibitors work by enhancing the activity of the body's own incretin system by preventing the breakdown of GLP-1. This leads to insulin secretion depending on glucose levels and the inhibition of glucagon secretion. DPP-4 inhibitors are safe and associated with a low risk of hypoglycemia; nonetheless, the glycemic effects may vary among different individuals (Makrilakis, 2019).

3.5 SGLT-2 Inhibitors

Sodium glucose cotransporter-2 (SGLT-2) inhibitors lower blood glucose levels by inhibiting renal glucose reabsorption, resulting in increased excretion of glucose in the urine. This action is insulin-independent and is seen as advantageous in diabetic patients with insulin resistance. This action is independent of insulin and shows therapeutic effects in patients with insulin resistance. The SGLT2 inhibitors have also exhibited therapeutic effects regarding cardiovascular and renal outcomes. Adverse reactions such as genitourinary infections and differences in response have also been observed (Gomez-Peralta et al., 2017).

3.6 GLP-1 Receptor Agonists

These drugs act like the body's own incretin hormones, which stimulate insulin secretion in a glucose-dependent manner. They also delay gastric emptying and enhance satiety, which leads to weight reduction. GLP-1 receptor agonists have proved to be very effective in lowering glucose and cardiovascular risk. However, gastrointestinal side effects and interindividual variability may restrict their use (Nauck et al., 2021).

3.7 Insulin

Insulin therapy is employed in patients with Type 2 Diabetes Mellitus if oral and injectable non-insulin therapies are ineffective in providing sufficient glycemic control. Insulin therapy works directly by replacing or supplementing body insulin. Despite its efficacy, insulin therapy also has its possible side effects, which include hypoglycemia and weight gain. The dosage efficacy of insulin therapy varies greatly among different people (Home et al., 2014).

4. Pharmacogenomics in Type 2 Diabetes

Pharmacogenomics investigates the impact of genetic variations on individual drug responses. In Type 2 Diabetes Mellitus (T2DM), the effectiveness of treatments and the occurrence of side effects can vary significantly among patients, even when they are given the same medications.

Although clinical factors such as age, body mass index, disease duration, and comorbidities influence this variability, they do not entirely explain the observed differences (Srinivasan et al., 2018).

Genetic polymorphisms in drug targets, transporters, metabolic enzymes, and signaling pathways significantly influence pharmacokinetic and pharmacodynamic responses to glucose-lowering therapies. Genetic variations that influence insulin secretion, its efficacy, hepatic glucose production, and renal glucose handling can modulate individual responses to pharmacological agents such as metformin, sulfonylureas, thiazolidinediones, incretin-based therapies, and insulin (Nasykhova et al., 2020; Baye et al., 2021).

5. Key Genes for Affecting Antidiabetic Drug Response

There will also be a brief explanation of the pharmacogenomic significance of specific genes, some of which have consistently been linked to variations in response to different anti-diabetic drugs. Genes that will be discussed include those that mediate insulin sensitivity, insulin release, and drug transport.

5.1 Peroxisome Proliferator-Activated Receptor Gamma (PPARG)

PPARG, or peroxisome proliferator-activated receptor gamma (PPARG), is a nuclear transcription factor that controls how fat cells develop, how lipids are broken down, and how sensitive insulin is. It is also a key part of the pathophysiology of insulin resistance in Type 2 Diabetes Mellitus (Zhou et al., 2016). Thiazolidinediones (TZDs), such as pioglitazone and rosiglitazone, primarily function by activating PPARG.

The Pro12Ala polymorphism (rs1801282) is the most thoroughly investigated variant, linked to modified insulin sensitivity and varied responses to TZD therapy. Individuals possessing the Ala allele have demonstrated an increased glycemic response in specific populations (Della-Morte et al., 2014; Zeng et al., 2020). Moreover, PPARG variants may exert an influence on the overall metabolic phenotype, potentially affecting the effectiveness of glucose-lowering pharmacotherapies. Notwithstanding these considerations, PPARG genotyping is still rarely utilized in clinical practice.

5.2 Transcription Factor 7 Like 2 (TCF7L2)

Transcription factor 7-like 2 (TCF7L2) is a crucial regulator in the Wnt signaling pathway, significantly influencing pancreatic β-cell function and insulin secretion. Variations in TCF7L2 constitute significant genetic risk factors for Type 2 Diabetes Mellitus.

The rs7903146 polymorphism has consistently been linked to diminished insulin secretion and heightened susceptibility to diabetes (Cropano et al., 2017). Due to its impact on β-cell functionality and incretin signaling, TCF7L2 variants may alter the therapeutic response to incretin-based therapies and insulin secretagogues. Research indicates that individuals with risk alleles may demonstrate modified responses to glucagon-like peptide-1 receptor agonists and dipeptidyl peptidase-4 inhibitors (Tkáč & Gotthardová, 2016). These findings underscore pharmacogenomic potential. However, routine clinical application is still restricted.

5.3 Potassium Inwardly Rectifying Channel Subfamily J Member 11 (KCNJ11)

Potassium inwardly rectifying channel subfamily J member 11 (KCNJ11) encodes the Kir6.2 subunit of the ATP-sensitive potassium (K_ATP) channel in pancreatic β-cells, which regulates glucose-stimulated insulin secretion.

The E23K polymorphism (rs5219) has been linked to changes in β-cell function and a higher risk of getting Type 2 Diabetes Mellitus (Souza et al., 2017). Genetic variation in KCNJ11 can significantly affect the response to sulfonylurea therapy, as sulfonylureas work by closing the K_ATP channel (Song et al., 2017). Even though there are known links, KCNJ11 genotyping is still not widely used in clinical settings.

5.4 ATP Binding Cassette Subfamily C Member 8 (ABCC8)

The sulfonylurea receptor 1 (SUR1), a key component of the ATP-sensitive potassium (K_ATP) channel, is located within pancreatic β-cells. The ATP binding cassette subfamily C member 8 (ABCC8) is responsible for synthesizing this receptor. ABCC8, working with Kir6.2, links glucose metabolism to the process of insulin secretion.

Genetic polymorphisms in ABCC8 have been linked to modified β-cell function and dysregulated insulin secretion, influencing the variability in susceptibility to Type 2 Diabetes Mellitus (Stefanski et al., 2007). Sulfonylureas directly bind to the SUR1 subunit to stimulate insulin secretion, so changes in ABCC8 may affect how well the treatment works and how likely it is to cause low blood sugar. Even though pharmacogenomic relevance has been shown, its use in everyday clinical practice is still limited.

5.5 Solute Carrier Family 22 Member 1 (SLC22A1)

The solute carrier family 22 member 1 (SLC22A1) gene codes for the organic cation transporter 1 (OCT1). This transporter is important in the liver, helping metformin enter cells.

The ability of metformin to reduce glucose production in the liver depends on the proper function of OCT1.

Reduced-function variants in SLC22A1 have been associated with diminished intracellular metformin accumulation and a less robust therapeutic effect (Bokelmann et al., 2018). Clinical investigations have demonstrated correlations between specific polymorphisms and variations in patient responses to metformin treatment (Chan et al., 2018). Furthermore, studies involving populations have shown that certain variations in the SLC22A1 gene are linked to reduced effectiveness of treatments (Umamaheswaran et al., 2015).

6. Challenges and Future Perspective

In spite of the evident links being established between pharmacogenomics and the guidance of anti-diabetic therapy, the application of pharmacogenomics within the clinic appears to be limited. Thus, despite the established links between the activity of pharmacogenomics and genes such as PPARG, TCF7L2, KCNJ11, ABCC8, and SLC22A1, the standardization of such therapy is still in its infancy (Venkatachalapathy et al., 2021).

One of the major challenges with pharmacogenomics-assisted therapy for Type 2 Diabetes Mellitus is that the effect of genetics differs between individuals. This variability arises because of the genetic variability between different ethnic groups, variability in the rate of metabolism, and the lack of consistency in pharmacogenomics studies. Moreover, there have been issues with the lack of samples used in some studies, as well as with the design or validation of other studies.

Another significant barrier to pharmacogenomics involves the intricacy of Type 2 Diabetes Mellitus, as this condition has been found to be associated with various polygenic, environmental, and lifestyle factors. Pharmacogenomics response does not rely on only a single gene variant, and gene-gene as well as gene-environment interactions act as major complicating factors for pharmacogenomics markers. Additional challenges include the high cost of

pharmacogenomics and the lack of accessibility as well as clinicians' lack of awareness of pharmacogenomics (Gloyn & Drucker, 2018).

Future studies should therefore be designed as large, multiethnic, prospective studies to validate pharmacogenomic biomarkers and integrate them with clinical and metabolic parameters. Bioinformatics, systems biology, and precision medicine approaches may further help develop clinically actionable decision-support tools. In conclusion, pharmacogenomics has immense potential in diabetes management for increasing therapeutic efficacy and reducing adverse drug reactions and will advance personalized treatment approaches in Type 2 Diabetes Mellitus (Li & Florez, 2022).

7. Conclusion

Pharmacogenomics has been recognized as a significant approach to explain the variability of drug response across patients suffering from Type 2 Diabetes Mellitus. Current clinical evidence has revealed that genetic variations in important genes such as drug targets, insulin, and transport mechanisms have significant implications for the efficacy and tolerability of existing antidiabetics commonly used in clinical settings.

This review aims to highlight the key impact of major pharmacogenetic variants, such as PPARG, TCF7L2, KCNJ11, ABCC8, and SLC22A1, in determining the response to thiazolidinediones, incretin-based agents, sulphonylureas, and metformin. These genetic factors are known to affect glycaemic control through their influence on insulin sensitivity, β-cell function, and liver drug uptake, respectively. The extent of influence of these genetic factors plays an important role in providing valuable insights into the etiology of treatment heterogeneity in Type 2 Diabetes Mellitus.

Clinical use of pharmacogenomics in the management of diabetes is still greatly limited due to population-specific genetic variability, polygenic complexity, and inadequate prospective validation. The potential of emerging technologies in genomics, bioinformatics, and precision medicine offers a promising way to overcome these challenges. By combining pharmacogenomic data with other important clinical and metabolic information, we could develop therapies that are personalized, effective, and safe.

Concluding, pharmacogenomics reflects one crucial step toward personalized diabetes care. Further research and efforts for clinical translation are yet to be done to achieve the full potential for the optimization of antidiabetic therapy and improving long-term outcomes in patients with Type 2 Diabetes Mellitus.

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