TPM Vol. 32, No. S3, 2025
ISSN: 1972-6325
https://www.tpmap.org/

DEVELOPING A PERSONALIZED MEDICINE APPROACH FOR TREATING TYPE 2 DIABETES

[1]VENU ANAND DAS VAISHNAV, [2]SACHIN PRADHAN, [3]ISHWARI DATT SUYAL

[1]ASSISTANT PROFESSOR, DEPARTMENT OF PHARMACY, KALINGA UNIVERSITY, RAIPUR, India.ku.venuananddas@kalingauniversity.ac.in,0009-0005-3775-6156

[2]ASSISTANT PROFESSOR, DEPARTMENT OF PHARMACY, KALINGA UNIVERSITY, RAIPUR, India.ku.sachinpradhan@kalingauniversity.ac.in,0009-0009-4095-1496

[3]ASSISTANT PROFESSOR, NEW DELHI INSTITUTE OF MANAGEMENT, NEW DELHI, INDIA., E-mail: ishwari.suyal@ndimdelhi.org, https://orcid.org/0009-0009-8998-2154

Abstract

Every year, type 2 diabetes (T2D), a global public health concern, is becoming more prevalent in Asia, particularly Taiwan. Both genetic and environmental variables affect the likelihood of type 2 diabetes and its effects. Polymorphisms at particular loci may be useful in identifying those who are most at risk and who may react to oral antidiabetic medications, according to genetic association studies. Using our research sample as examples, this review examines the relationship between genetic profiling and type 2 diabetes and its consequences. The research will also look at the pharmacogenetics and pharmacogenomics of oral diabetes drugs. Personalized medicine, which involves discovering unique patient information and then suggesting a treatment suited to that patient's needs, is a new concept in the treatment of disease.

Keywords: antidiabetic, treating diseases, identify patients

1. INTRODUCTION

With the fastest-growing incidence of chronic disease in the twenty-first century, type 2 diabetes (T2D) is one of the most important health issues facing the globe today. By 2025, 300 million people worldwide will suffer from diabetes. A complex metabolic disease with a high morbidity and mortality rate is type 2 diabetes [2]. Individuals with diabetes are approximately twice as likely to die as those without the disease in the same age group [1]. These include, among other major public health concerns, a high risk of blindness, cardiovascular intervention, and renal replacement therapy. Because diabetes is becoming more common, T2D and its aftereffects will significantly lower patients' quality of life and increase healthcare expenses overall[11].

T2D is a group of metabolic diseases marked by anomalies in the metabolism of proteins, fats, and carbohydrates as well as consistently elevated blood sugar levels brought on by insufficiencies in the secretion or function of insulin. The pathophysiology and clinical manifestations of this disease are diverse, resulting from a multitude of interrelated aberrant pathways and molecular abnormalities that interact with both positive and negative environmental variables [10]. Despite significant heterogeneity between cases, these individuals are frequently treated similarly in modern medical care, with little attention paid to individual factors that may influence clinical prognosis and therapeutic response. Previous research has shown that heredity is important for both etiology and complications [3]. Based on the well-established idea that every person is born with distinct biological and genetic traits, personalized medicine is a relatively recent paradigm of evidence-based medicine. Since individual differences will impact the disease's risk, progression, and therapy, it is imperative to determine the most effective therapeutic method for each patient. It has been demonstrated that genetic variables significantly influence the pathophysiology and outcomes of diabetes. We concentrate on how genetic profiling impacts complications from diabetes and type 2 diabetes. Patients with the same illness differ greatly from one another. While some diabetics respond quickly to treatment, others do not. This variation is caused by changes in hundreds of genes' expression or coding sequences that increase vulnerability to disease [12]. Some of these genes are linked to the clinical response to treatment or to the etiology. Target therapy, preventative measures, and diagnostic accuracy should all be improved by analyzing genetic profiles for the existence of pharmacological targets and biomarkers.

A medicine may work effectively for some people but not for others. One possible explanation is a hereditary propensity to either react or not react to a medicine [4]. After evaluating each patient, the doctor must make an educated judgment as to which course of action will be most effective. Treatment plans may be customized for

each patient if the doctor had access to particular, individualized information about them, such as their genetic composition. Better results would follow from this strategy without wasting time on unsuccessful therapies[16]. Certain diseases and therapies have a 100% response rate when one treatment is used, a 0% response rate when another treatment is used, and a 100% effectiveness rate for other patients.

2. REVIEW OF LITERATURE

Personalized medicine for diabetes for diabetes (PMFD) involves tailoring diabetes prevention, detection, treatment, or monitoring strategies according to an individual's genetic makeup. The PMFD method consists of four phases. The first step is to identify the genes and biomarkers for type 2 diabetes and obesity, which is the largest risk factor for the disease. The next step is choosing customized treatments for those who are impacted[5]. Choosing which medication to prescribe, how much to take, and what diet to recommend are all part of the choosing process. The medicine with the lowest risk of toxicity or adverse effects is also taken into consideration throughout the selecting process. The fourth step involves measuring diabetes biomarkers in the bloodstream to track how well treatment or prevention is working. Most people with type 2 diabetes have polygenetic versions of the disease, where each gene locus only slightly increases risk. Thus far, a number of loci have been identified, including transcription factor 7-like, peroxisome proliferator-activated receptor γ, calpain 10, potassium inwardly rectifying channel, subfamily J, member 11 (KCNJ11), and others. The organic cation transporter 1 (OCT1), which is the main route by which metformin enters enterocytes and hepatocytes, is another example of this. Individuals with OCT1 polymorphisms have poorer metformin responses. Improved genotyping techniques have accelerated the process of gene identification. More accurate genotyping suggests a growing role for a customized treatment strategy to diabetes in the future.

It is necessary to take into account the context of the genetic data utilized to implement customized therapy. The reaction to a specific medication is influenced by several factors. Success is not only determined by the patient's genetic makeup. Additional variables that may complicate the results of a genotype-specific tailored strategy include diet, competing comorbidities, interactions with other medications, environmental factors that may override the effects of a treatment, and genes other than the one or genes being evaluated. A combination of environmental variables, food, and multigene vulnerability frequently results in type 2 diabetes. The impact of a treatment based on a single gene's activity in this illness may be overshadowed by numerous additional complicating variables [6]. The customized medicine method may still be successful if a single gene provides a reliable indicator of how well a disease will respond to treatment[13]. Before the start of a chronic disease like diabetes, those who are at high risk for it typically go through a protracted period of asymptomatic living. Diabetes can be delayed or prevented by directing patients who are genetically tested to be at high risk for the condition to preventative interventions like lifestyle changes or medication. 6. Biomarkers and genetic tests can be used to track the progression of diabetes and anticipate the diagnosis. Finding genetically or nutritionally determined therapeutic targets in subgroups of diabetic patients can increase the efficiency of medication development. Variable drug reactions are caused by genetically determined polymorphisms in metabolizing enzymes, transporters, and receptors. Customized medication prescriptions are made possible by customized medicine, which reduces trial and error and the amount of time lost on ineffective or harmful responses.

3. MATERIALS AND METHODS

There are now five primary categories of personalized medicine research projects underway. First, the study of pharmacogenetics aims to explain why different people react differently to the same medication. Finding the genes that are essential to the illness state becomes much more difficult when gene products are measured across time. In the end, pharmacogenomics—which includes the utilization of cell systems or even living organisms—is able to clarify correlations that are more reliable than pharmacogenetics alone [7]. Third, the method known as nutrigenomics makes use of the identification of genetically mediated reactions to foods and the subsequent modification of the diet to capitalize on these reactions. Fourth, diseases can be predicted, diagnosed, or tracked using biomarkers. For example, autoantibodies can be used to predict type 1 diabetes, while adipokines can be used to predict type 2 diabetes. Last but not least, systems biology measures how the components of biological systems interact with one another and how these interactions affect the behavior and functionality of the system[14]. Diabetes prevention, diagnosis, treatment, and monitoring will all be impacted by personalized medicine. Advances in each of these treatment strategies for this illness will be facilitated by genetic knowledge. The medication that lowers blood sugar is a member of the insulin secretagogues, which speed up the synthesis and release of insulin. Tolbutamide, gliclazide, glipalamide, and glimepiride are common sulfonylureas that efficiently regulate blood sugar levels, but they come with a slight risk of weight gain and hypoglycemia. While most patients respond effectively to sulfonylurea, 10–20% of patients do not achieve adequate glycemic control,

and 5–10% of patients who initially respond to sulfonylurea eventually lose the capacity to maintain an almost normal glucose level. This suggests a connection between treatment effectiveness and hereditary variables [9]. Sulfonylureas bind to the high-affinity plasma membrane receptor (SUR1) first, followed by an ATP-dependent K channel (KAPT) to release insulin from pancreatic beta cells. Recently, variations in how T2D patients react to sulfonylurea medications have been linked to polymorphisms in drug target genes, specifically ATP-binding cassette transporter subfamily C member 8 (ABCC8) and potassium inwardly-rectifying channel, subfamily J member. The pore-forming part of the ATP-sensitive potassium channel Kir6.2 in pancreatic β-cells is encoded by the KCNJ11 gene, which is found on the short arm of chromosome 11. KCNJ11 gain-of-function mutations cause the potassium channel to open and β-cell depolarization to stop, which prevents the production of insulin [8]. Cytochrome P450 (CYP) enzymes in the liver metabolize the majority of oral hypoglycemic medications; their impact on the response to sulfonylurea treatment was investigated. CYP2C9 variations are linked to decreased oral clearance of sulfonylureas and poor metabolism.

4. RESULT AND DISCUSSION

These peroxisome proliferators decrease the synthesis of glucose in the liver and increase insulin sensitivity in skeletal muscles via activating receptor γ (PPAR-γ). By controlling the transcriptional activity of several genes, is essential for adipogenesis and insulin sensitivity. TZDs increase leptin levels while drastically lowering the number of triglycerides in the liver, skeletal muscles, and adipose tissue [15]. When combined, these modifications lower the amount of free fatty acids (FFA) in the blood, which lessens the insulin resistance that FFA causes in skeletal muscles. Rosiglitazone, pioglitazone, and troglitazone may decrease the progression of β-cell failure and are known to improve glycemic control among TZDs. The majority of research focuses on PPAR-γ variations and the genes that harbor them in relation to the results of TZDs therapy.

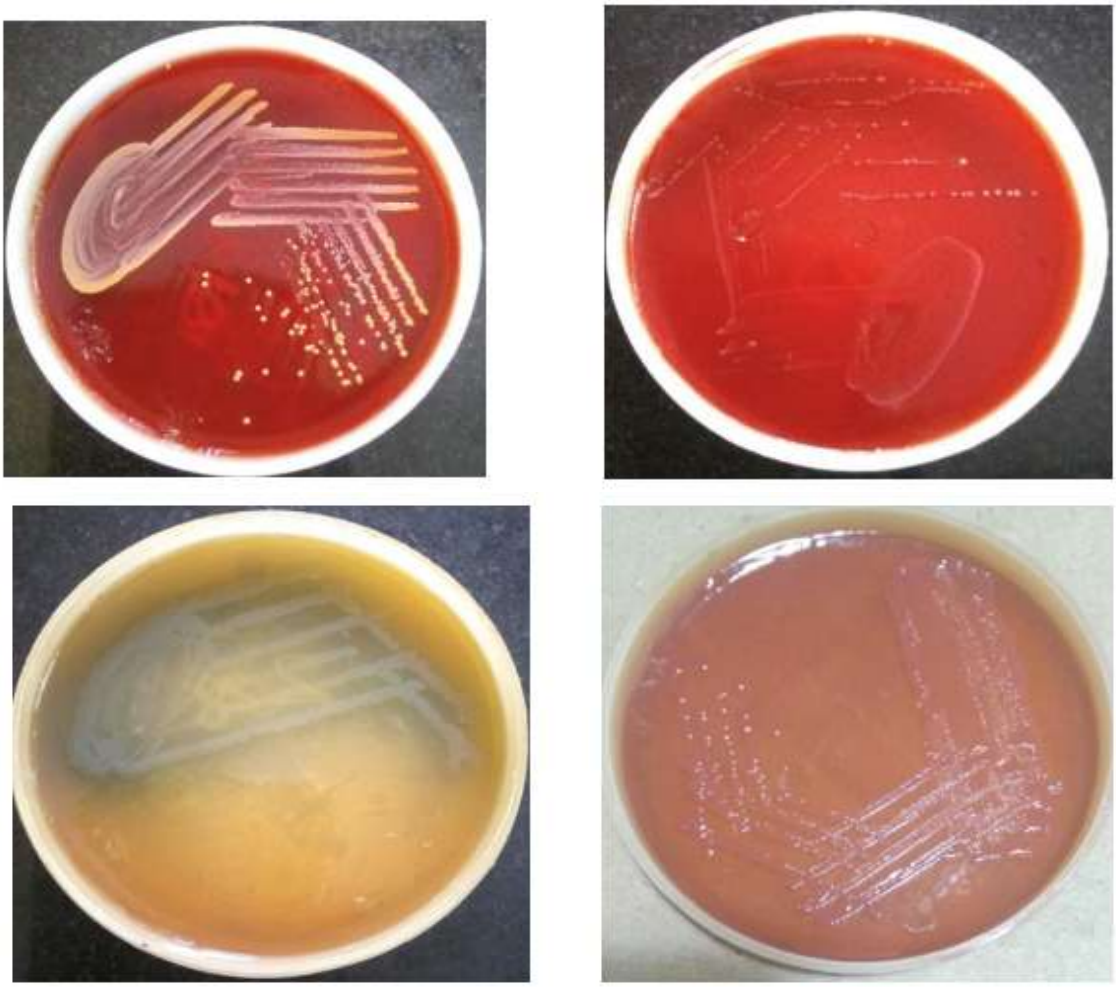

Figure 1: Growth of species

There is no assurance that diabetic patients will respond similarly to a particular medication, even if they share the same age, body mass index, duration of the condition, and hemoglobin A1c (HbA1c) levels. Each patient

must be evaluated by the healthcare provider in order to identify the most likely effective course of treatment. This can be a challenging task for the doctor. Group-level average results from clinical studies indicate the specific proportion of individuals who will react to a certain medication.

Figure 2: Growth of a) Escherichia coli b) Proteus species c) Klebsiella species

These results, meanwhile, might not always apply to a particular patient. The issue is that doctors are unsure of the best course of action for a certain patient. At the moment, medical practitioners prioritize the treatment that has the best possibility of working for the most number of patients, even though some people won't benefit from it.

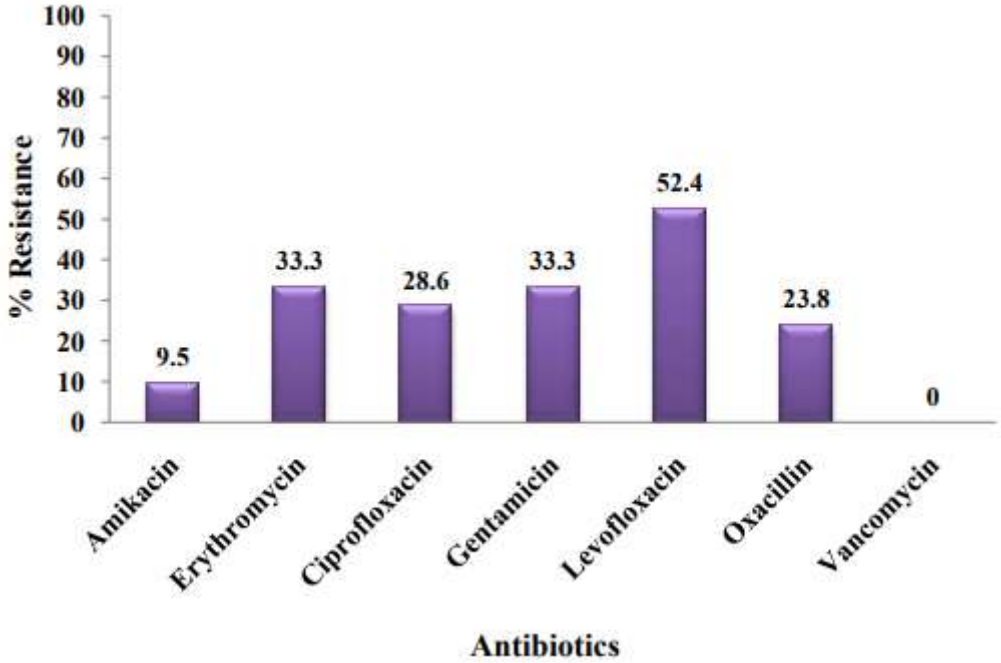

Figure 3: Antibiogram of Gram-positive cocci

Comorbidity is the first variable that could be used to tailor treatment for diabetes. Medical comorbidities such as obesity, hypertension, liver illness, diabetes kidney disease (DKD), cardiovascular disease, and others can affect the doctor's prescription choices. Current diabetes risk engines may also incorporate family history, age, gender, smoking status, blood pressure, glycemia, dyslipidaemia, and other significant diabetes-related characteristics to predict cardiovascular risk. Additional parameters and kidney function may be incorporated into more sophisticated instruments.

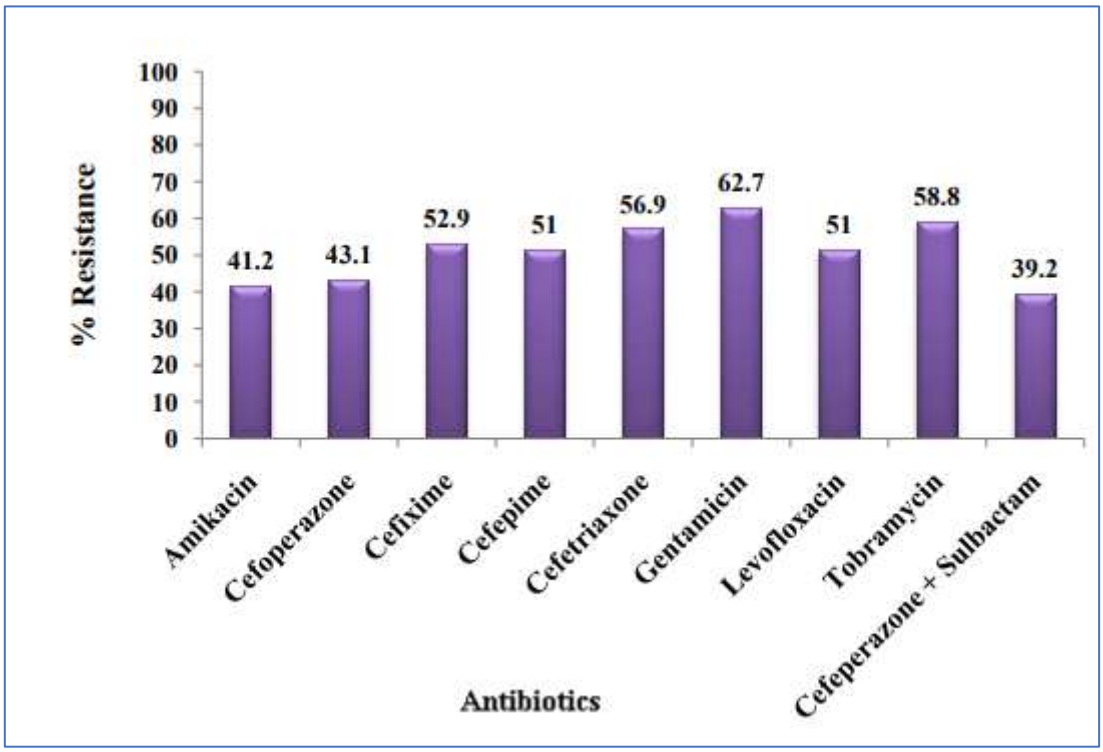

Figure 4: Antibiogram of Enterobacteriaceae

There is a great chance to increase the effectiveness of diabetes management with recent advancements in healthcare delivery technologies, including smartphone applications, telemedicine, mHealth, device connectivity (e.g., Continuous Glucose Monitoring (CGM) devices to continuously measure glucose values), machine learning, and artificial intelligence. Furthermore, they facilitate increased patient involvement in diabetes self-management, which could ultimately result in reduced costs for diabetes-related medical care.

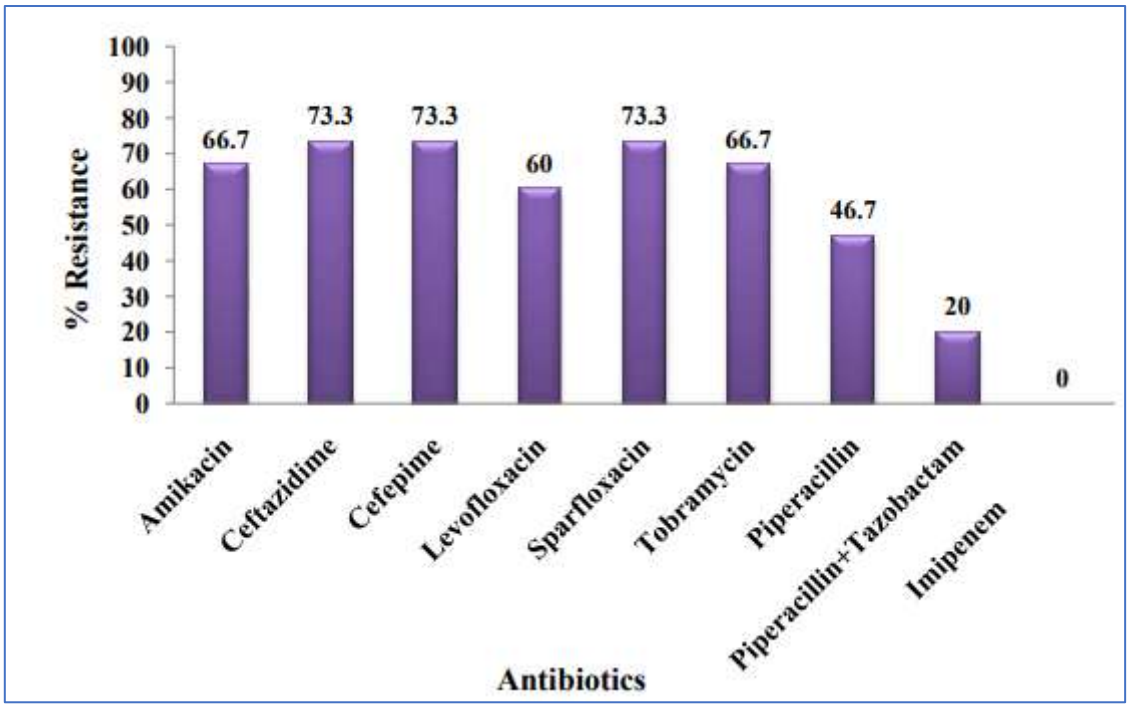

Figure 5: Antibiogram of Pseudomonas aeruginosa

Researcher, doctor, diabetes educator, geneticist, legislator, patient advocate, clinical laboratory, pharmaceutical and diagnostic companies, IT managers, payers, and government regulators are among the stakeholders impacted by the growing use of personalized medicine in diabetes treatment.

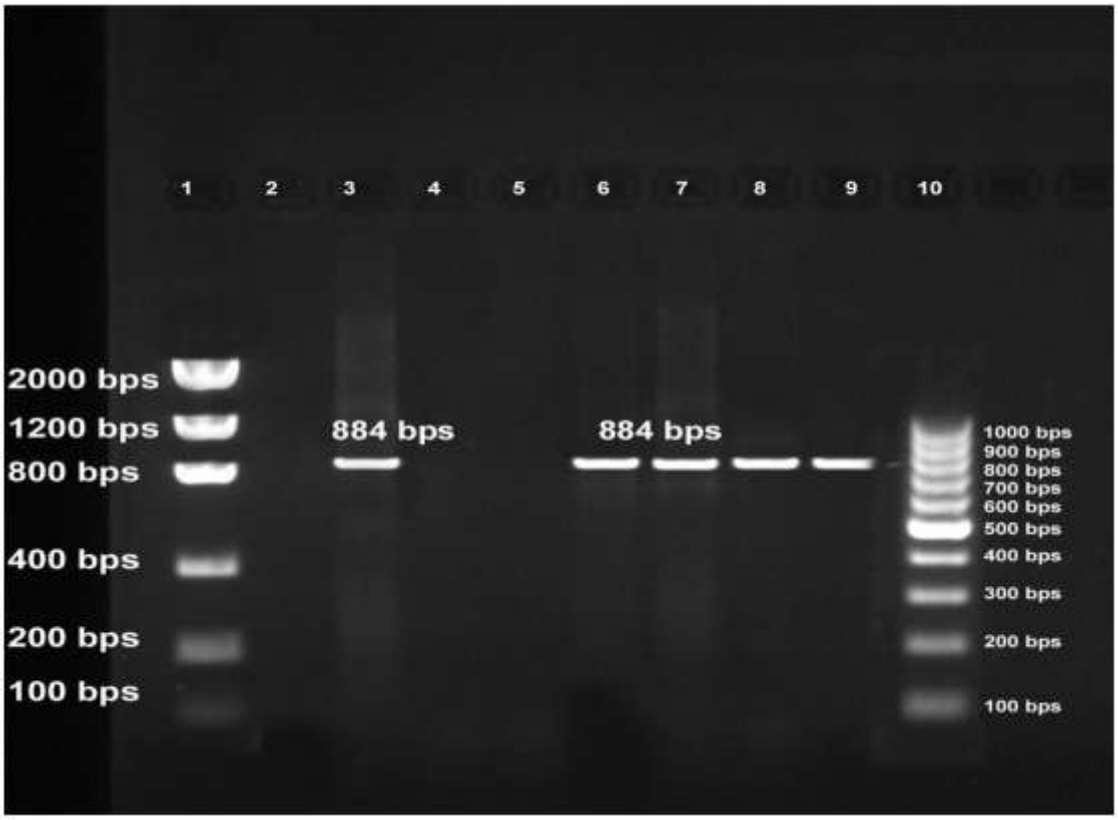

Figure 6: Gel image showing specific amplification

To control tailored treatment for diabetes, members of these organizations will need to collaborate. Currently, there is very little regulation of these two aspects of the logistics of managing personalized medicine initiatives. The establishment of a Genome Commons might speed up the creation of novel drugs for individualized diabetes

care. This database would serve as a resource for understanding human genomes and a storehouse of common human inheritance.

5. CONCLUSION

From the viewpoints of medical professionals, this study sought to understand whether and how personalized medicine concepts are now included into Dutch diabetes type 2 secondary care. The study also aimed to determine the elements that influence implementation, both facilitating and inhibiting. According to the study, secondary care diabetic healthcare providers in the Netherlands incorporate personalized medicine to some extent in their treatment of patients with the disease. While some aspects of the tailored approach, such as medication use, diabetes phenotype, comorbidities, and personal traits, are already being actively utilized, other aspects, such as genetic variables, biomarkers, and healthcare resources, are not being fully utilized. Personalized medicine will be highly helpful in treating diabetes. This approach won't be extensively used unless effective medication is paired with the discovery of risk variables using biomarkers or genotyping. Additionally, with individualized care, diabetes will be avoided before it shows symptoms. In the fight against diabetes, individualized medication treatment will be more important. Genomic markers for type 2 diabetes are still not widely used in clinical settings. Large cohort study results must, however, include basic information that can be utilized to identify new therapeutic targets and profile risk variables. Personalized treatment will also take Pharmacoeconomics into account. Based on information gathered from the study's interviews, it is evident that medical practitioners tailor treatment to each patient's current prescription regimen in order to prevent any potential drug interactions.

REFERENCES

1. Venkatachalapathy, Poongothai, SruthiPadhilahouse, Mohan Sellappan, Tharunika Subramanian, Shilia Jacob Kurian, Sonal Sekhar Miraj, Mahadev Rao et al. "Pharmacogenomics and personalized medicine in type 2 diabetes mellitus: potential implications for clinical practice." Pharmacogenomics and Personalized Medicine (2021): 1441-1455.
2. Saidova, K., Abdullayeva, S., Yakubova, D., Gudalov, M., Abdurahmonova, K., Khudoykulova, H., Mukhammadova, G., &Zokirov, K. (2024). Assessing the Economic Benefits of Climate Change Mitigation and Adoption Strategies for Aquatic Ecosystem. International Journal of Aquatic Research and Environmental Studies, 4(S1), 20-26. https://doi.org/10.70102/IJARES/V4S1/4
3. Spiegel, Allen M., and Meredith Hawkins. "'Personalized medicine'to identify genetic risks for type 2 diabetes and focus prevention: can it fulfill its promise?." Health Affairs 31, no. 1 (2012): 43-49.
4. Vranješ, B., Vajkić, M., Figun, L., Adamović, D., & Jovanović, E. (2024). Analysis of Occupational Injuries in an Iron Ore Mine in Bosnia and Herzegovina in the Period from 2002 to 2021. Archives for Technical Sciences, 1(30), 33-44. https://doi.org/10.59456/afts.2024.1630.033V
5. Reddy, S. Sethu K. "Evolving to personalized medicine for type 2 diabetes." Endocrinology and Metabolism Clinics 45, no. 4 (2016): 1011-1020.
6. Sundara Bala Murugan, P., Ganesan, A., Paranthaman, P., & Aruna, V. (2024). Feasibility Design and Analysis of Process-aware Accounting Information System for Business Management. Indian Journal of Information Sources and Services, 14(2), 56–62. https://doi.org/10.51983/ijiss-2024.14.2.09
7. Galiero, Raffaele, Alfredo Caturano, Erica Vetrano, MarcellinoMonda, RaffaeleMarfella, CelestinoSardu, Teresa Salvatore, Luca Rinaldi, and Ferdinando Carlo Sasso. "Precision Medicine in Type 2 Diabetes Mellitus: Utility and Limitations." Diabetes, Metabolic Syndrome and Obesity (2023): 3669-3689.
8. Flores-Fernandez, G. A., Jimenez-Carrion, M., Gutierrez, F., & Sanchez-Ancajima, R. A. (2024). Genetic Algorithm and LSTM Artificial Neural Network for Investment Portfolio Optimization. Journal of Wireless Mobile Networks, Ubiquitous Computing, and Dependable Applications, 15(2), 27-46. https://doi.org/10.58346/JOWUA.2024.I2.003
9. Raciti, Gregory Alexander, Cecilia Nigro, Michele Longo, Luca Parrillo, Claudia Miele, PietroFormisano, and Francesco Béguinot. "Personalized medicine and type 2 diabetes: lesson from epigenetics." Epigenomics 6, no. 2 (2014): 229-238.
10. Jiménez-Carrión, M., Flores-Fernandez, G. A., & Jiménez-Panta, A. B. (2023). Efficient Transit Network Design, Frequency Adjustment, and Fleet Calculation Using Genetic Algorithms. Journal of Internet Services and Information Security, 13(4), 26-49. https://doi.org/10.58346/JISIS.2023.I4.003

11. Williams, David M., Hannah Jones, and Jeffrey W. Stephens. "Personalized type 2 diabetes management: an update on recent advances and recommendations." Diabetes, metabolic syndrome and obesity: targets and therapy (2022): 281-295.
12. Dinesh, R (2024). Evaluation of Fuel Consumption and Exhaust Emissions in a Single Cylinder Four-Stroke Diesel Engine Using Biodiesel Derived from Chicken Waste with Additives. Natural and Engineering Sciences, 9(2), 326-334. https://doi.org/10.28978/nesciences.1574462
13. Fitipaldi, Hugo, Mark I. McCarthy, Jose C. Florez, and Paul W. Franks. "A global overview of precision medicine in type 2 diabetes." Diabetes 67, no. 10 (2018): 1911-1922.
14. Meybodi, Hamid Reza Aghaei, MandanaHasanzad, and BagherLarijani. "Path to personalized medicine for type 2 diabetes mellitus: reality and hope." ActaMedicaIranica (2017): 166-174.
15. Dennis, John M. "Precision medicine in type 2 diabetes: using individualized prediction models to optimize selection of treatment." Diabetes 69, no. 10 (2020): 2075-2085.
16. Liao, Wen-Ling, and Fuu-Jen Tsai. "Personalized medicine in type 2 diabetes." BioMedicine 4 (2014): 1-8.
17. Velliangiri, A. (2025). Reinforcement Learning-Based Adaptive Load Forecasting for Decentralized Smart Grids. National Journal of Intelligent Power Systems and Technology, 1(1), 21-28.
18. Uvarajan, K. P. (2025). Advanced Thermal Energy Storage Materials for Concentrated Solar Power (CSP) Plants. National Journal of Renewable Energy Systems and Innovation, 38-46.
19. Sadulla, S. (2025). Energy-Efficient Motor Control Algorithms for Variable Load Industrial Processes. National Journal of Electric Drives and Control Systems, 32-39.
20. Kavitha, M. (2025). Design and Optimization of High-Speed Synchronous Reluctance Machines for Industrial Drives. National Journal of Electrical Machines & Power Conversion, 1-10.
21. Karthika, J. (2025). Power Converter Design for Next-Generation Wind Energy Systems Using GAN Devices. Transactions on Power Electronics and Renewable Energy Systems, 1-12.
22. Veerappan, S. (2025). Integration of Hydrogen Storage with PV Systems for Off-Grid Power Supply. Transactions on Energy Storage Systems and Innovation, 1(1), 41-49.
23. Ogbonnaya, E., &Wai, Y. M. (2024). Design and optimization of energy harvesting circuits for ultra-low power wearable electronics. Electronics, Communications, and Computing Summit, 2(1), 123–130.