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Research Article

Analysis of Genetic Variations in Diabetic Wistar Rats Induced with Alloxan Monohydrate Using Random Amplified Polymorphic DNA (RAPD) Polymerase Chain Reaction (PCR) Techniques

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Abstract

Background: Diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia due to impaired insulin secretion, action, or both. The genetic basis of diabetes has been widely studied in humans, but limited research has been conducted on animal models to understand genetic markers of diabetic expression. This study aims to analyze genetic variations in diabetic Wistar rats induced with alloxan using Random Amplified Polymorphic DNA (RAPD) polymerase chain reaction (PCR) techniques.

Materials and Methods: Six Wistar rats were obtained from Sure Amos Farm, Ikere, Ekiti State, Nigeria, and housed conditions at Afe Babalola University, Ado-Ekiti (ABUAD). After seven days of acclimatization, they were weighed, and blood samples were collected for the determination of diabetes indices which serve as baseline parameters. Diabetes was induced intraperitoneally with alloxan at a dose of 150 mg/kg. Six days after the inducement, the animals were weighed again, blood samples were collected via the retroorbital sinus method, and genomic DNA was extracted using the Quick-g DNATM Blood Miniprep kit. DNA amplification was performed using five RAPD primers (B06, T12, H04, T10, B08), and electrophoresis was conducted to assess polymorphic band patterns. Cluster analysis and phylogenetic relationships were determined using Jaccard's similarity coefficient and the unweighted pair-group method arithmetic (UPGMA) clustering.

Results: Alloxan administration significantly decreased body weight while increasing blood glucose levels in all six rats. Three primers (B06, H04, and B08) produced distinct and reproducible bands, demonstrating genetic variations between

control and alloxan-induced diabetic rats. PCR amplification revealed additional bands in diabetic rats, indicating genetic modifications associated with diabetes expression.

Conclusion: The RAPD-PCR analysis successfully identified genetic alterations in alloxan-induced diabetic Wistar rats. These findings suggest a potential genetic basis for diabetes expression, emphasizing the need for further studies to identify specific genetic markers associated with diabetes susceptibility.

Key Words: diabetes; rapd-pcr; genetic expression; blood glucose; phylogenetic analysis

Introduction

Diabetes mellitus (DM) represents a chronic metabolic disorder characterized by persistent hyperglycemia due to defects in insulin secretion, insulin action, or both [1,2]. This condition is a major public health concern worldwide because of its rising prevalence and the severe complications that accompany sustained high blood glucose levels. The complexity of diabetes is not only physiological but also genetic, as variations in genetic makeup can influence susceptibility, progression, and response to treatment [3]. Consequently, understanding the genetic underpinnings of diabetes has become a central focus in biomedical research.

Animal models remain indispensable tools in diabetes research. Among these, rodents (particularly Wistar rats) are extensively used due to their physiological similarity to humans, well-characterized genetics, and ease of handling in controlled experimental settings. Inducing diabetes in these animals using chemical agents allows researchers to mimic aspects of human diabetes, thereby enabling investigations into disease mechanisms and potential therapeutic interventions [4,5].

One of the most widely used chemical agents for experimental induction of diabetes is alloxan monohydrate. Alloxan selectively destroys insulin-producing β-cells in the pancreatic islets through the generation of reactive oxygen species (ROS) following uptake via the GLUT2 glucose transporter. This β-cell destruction leads to insulin deficiency and metabolic dysfunction that resembles type 1 diabetes in humans [6].

The induction of diabetes through alloxan not only results in characteristic clinical features of DM such as hyperglycemia, polyuria, polydipsia, and weight loss, but also triggers secondary changes at organ and tissue levels. These changes provide important physiological, biochemical, and histopathological insights into the progression of diabetic complications, including nephropathy and oxidative damage in various tissues [7].

While physiological and biochemical studies in diabetic rat models are well documented, understanding how diabetes affects the genome, or how different genetic profiles may influence susceptibility and severity, is equally important. These genetic variations can serve as markers for predisposition to disease states, phenotypic differences, and responses to treatments. One powerful molecular tool to investigate such genetic differences is Random Amplified Polymorphic DNA (RAPD) analysis.

RAPD is a polymerase chain reaction (PCR)-based technique that amplifies random segments of genomic DNA using short, arbitrary primer sequences. Unlike sequence-specific PCR methods, RAPD does not require prior knowledge of the DNA sequence of the organism. The technique amplifies multiple genomic regions simultaneously, generating a pattern of DNA fragments unique to a genetic profile. Differences in banding patterns reflect underlying genetic polymorphisms among individuals or experimental groups, making RAPD an efficient method for comparing genetic diversity and detecting genomic alterations without extensive sequencing efforts [8,9].

In rodent studies, RAPD-PCR has been used to distinguish between strains and to monitor genetic contamination, demonstrating that even closely related laboratory rat populations can exhibit notable genomic differences when analyzed with RAPD markers [RAPD has been applied to characterize genetic differences in rat strains]. The application of RAPD in diabetic research represents an important extension of traditional phenotype-focused studies, allowing researchers to investigate whether genetic changes accompany alloxan-induced diabetic states and how these may influence disease progression or experimental outcomes.

Integrating an RAPD-PCR approach into the analysis of alloxan-induced diabetic Wistar rats, therefore, serves a dual purpose. First, it provides insights into genetic variation that may occur as a result of pathological changes or as a predisposing genomic background. Second, it establishes a molecular fingerprint that can be compared across experimental conditions, potentially revealing new biomarkers or genetic signatures associated with diabetic states.

By analyzing genetic variation through RAPD-PCR, this study seeks to fill an important knowledge gap in diabetes research. Whereas physiological and metabolic aspects of alloxan-induced diabetes are well documented, the genomic characteristics of these models remain relatively underexplored. Understanding these genetic variations will enhance the utility of the rat model for translational studies and may point to genetic patterns that correlate with diabetic severity, organ-specific complications, or differential responses to therapeutic interventions.

Materials And Methods

Procurement of the Laboratory Animals and Housing

A total of six (6) Wistar rats were purchased from Mr Sure Amos Farm, Ikere, Ekiti State, Nigeria. The animals were housed in cages in the ABUAD Animal House at room temperature (27±0.5 oC with free access to clean water and standard feed, and they were acclimatized for 7 days.

Determination of the body weights

The body weight of the animals was determined and recorded using an analytical weighing scale (OHAUS Scientific Ltd). Also, blood samples were collected for the determination of diabetes indices, which serve as baseline parameters. Diabetes was induced using a single intraperitoneal administration of alloxan monohydrate at a dose of 150 mg/kg. Six days after the inducement, the animals were weighed again, and blood samples were collected from each rat for DNA extraction. Blood collection was done employing retroorbital sinus, a non-traumatic method of blood collection from a rat's eye blood vessel under light anaesthesia (halothane). The sample was immediately transferred into an ice pack for further analysis. This was done for genomic DNA extraction. Serum was

obtained from the whole blood by centrifuging at 2000 rpm for 10 minutes and then transferred into a new tube for the process of extraction.

DNA Extraction and Purification

1. Genomic DNA extraction from serum was carried out using Quick-gDNATM Blood Miniprep kit (Zymo Research Corporation, Irvine, U.S.A) according to the manufacturer's instructions.
2. 400 µl of Genomic Lysis Buffer was added to 100 µl of serum, vortexed for 4-6 seconds, then left to stand for 5- 10 minutes at room temperature.
3. The mixture was transferred to a Zymo-SpinTM column in a collection Tube and Centrifuged at 10,000 rpm for one minute.
4. The Zymo-SpinTM Column was transferred to a new collection Tube. 200 µl of DNA pre-wash buffer was added to the spin column and centrifuged at 10,000 rpm for one minute.
5. 500 µl of g-DNA buffer was added to the spin column and centrifuged at 10, 000 rpm for one minute
6. The spin column was transferred to a clean microcentrifuge tube. 50 µl DNA elution buffer was added to the spin column, incubated for 2-5 minutes at room temperature and then centrifuged at 10,000 rpm 10,000 rpm for 30 seconds to elude the DNA. The eluted DNA was stored at ≤ - 20 oC for further use.

Reproducibility of RAPD Bands

The RAPD-PCR marker assay was conducted at the Bioscience Laboratory Centre, International Institute of Tropical Agriculture (IITA), Ibadan, Oyo State. A total of five RAPD primers (Table 1), obtained from the Bioscience Centre were

1. Initially tested for primer variability and reproducibility through an optimization process.
2. The optimization process was conducted three times to find the optimum conditions of the PCR and the same bands were observed.
3. To represent wider optimization, three pooled genomic DNA were used. The pooled genomic DNA was obtained by selecting the first three albino rat genomic DNA extracts and mixing them to form the first pool and the last three to form the second pool which was then used to screen the five RAPD primers out of which the best three (B06, H04 and B04) were selected after testing their reproducibility and polymorphism (Table 2)

S/N	Primer	Sequence
1	B06	TGCTCTGCCC
2	T12	GGGTGTGTAG
3	H04	GGAAGTCGCC
4	T10	CCTTCGGAAG
5	B08	GTCCACACGG

Table 1: List of five RAPD Primers used for PCR screening of pooled rat genomic DNA

S/N	Primer	Sequence
1	B06	TGCTCTGCCC
2	H04	GGAAGTCGCC
3	B08	GTCCACACGG

Table 2: List of Selected three RAPD Primers used for PCR Genomic Analysis of 6 rats Genomic DNA

Detailed PCR protocol

The RAPD PCR amplification reaction was conducted with Masteral Thermal Cycler (Corbett Research Canada). The amplification was carried out in a 12.5 µl reaction mixture containing 2.5 µl of template DNA solution for PCR (10 µl of extracted DNA added to 90 µl deionized water) was added to 10 µl of a prepared cocktail. The cocktail was prepared by mixing 0.2 µl of DNA taq polymerase, 1.25 µl Taq buffer, 1.0 µl MgCl2, 1.25 µl diluted dATP, dGTP, dCTP, dTTP (deoxynucleotides were diluted with deionized water according to instruction on the manufacturer's manual), 1.25 µl Tween 20 (5%), 0.5 µl of each of diluted RAPD primer. All reagents were purchased from Inaba Biotech South Africa.

Processes involved For PCR protocol

The amplification cycling parameters were 4 minutes of preheating, initial denaturation at 94 oC for 3 minutes and 35 cycles;

1. 1-minute primer annealing at 55 oC based on the primers used,
2. 2 minutes' extension at 72 oC and the final extension for 7 minutes at 72 oC .
3. The RAPD products were then stored at 4 oC until loading on a gel for electrophoresis.
4. The amplicons were separated by electrophoresis (1.4 % agarose gel electrophoresis).
5. Electrophoresis was done for 1.2 hours at a constant voltage of 100 V and recorded with the Alpha digital imager gel documentation system (Alpha Innotech, Canada) staining with G-green dye (0.5 µg/ml). All tests were done in duplicate.

Rationale For Choosing Rapd In Detecting Genetic Changes In Diabetic Rats

RAPD (Random Amplified Polymorphic DNA) is used to detect genetic changes in diabetic rats because it can reveal variations in DNA sequences that arise from mutations or other genetic alterations 9 [10]. By comparing RAPD profiles (DNA "fingerprints") of diabetic rats to those of control (non-diabetic) rats, researchers can identify specific DNA fragments that are present or absent, indicating genetic differences associated with diabetes. This technique is particularly useful because it doesn't require prior knowledge of the specific DNA sequences involved in diabetes.

Cluster analysis and phylogenetic relationship

Positions of unequivocally scorable PCR bands were transformed into a binary character matrix ("1" for the presence and "0" for the absence of a band at a particular position). Pairwise distance matrices were compiled by the NTSYS-pc 2.0 software using the Jaccard coefficient of similarity). A phylogenetic tree was created by the unweighted pair-group method arithmetic (UPGMA) average cluster analysis. The scored RAPD markers were converted into a binomial (0/1) matrix.

Quantification Of Polymophism

Pairwise distance matrices were compiled by the NTSYS-pc 2.0 software using the Jaccard coefficient of similarity. A phylogenetic tree was created by the unweighted pair-group method, arithmetic (UPGMA) average cluster analysis.The scored RAPD markers were converted into a binomial (0/1) matrix.

Different Phylogenetic Genotypes for Gel Photograph

The number of polymorphic bands generated by each primer was determined by initial visual examination of the gel photographs, taking into account the resolution and degree of amplification. Only bands of medium and strong intensity were included in the subsequent analysis. For RAPD analysis, the presence of the band was scored 1, whereas the absence of the band was scored 0. The scored RAPD markers were converted into a binomial (0/1) matrix. Based on the matrix, the genetic similarities between Wistar rats' genotypes were determined using Jaccard's coefficients and the dendrogram was constructed by applying an unweighted pair group.

Results

The administration of alloxan significantly impacted the body weight of the rats. As shown in Table 3, there was a marked reduction in body weight following alloxan treatment compared to the pre-administration weights. The weight reductions observed were significant, with values such as 5.3g, 6.6g, 8.9g, 9.02g, 4.52g, and 7.1g noted after treatment. This suggests that alloxan had a considerable effect on metabolic processes, potentially leading to weight loss. To investigate genetic variations, five RAPD primers (Table 4) were screened through PCR amplification to assess their variability and reproducibility. After an optimization process conducted three times, the three most suitable primers (B06, H04, B08) were selected based on their ability to produce clear, reproducible bands (Table 5). These primers were then utilized for DNA fingerprinting to assess genetic alterations following alloxan administration.The blood glucose levels of the rats also exhibited significant changes post-alloxan treatment. As indicated in Table 3, the glucose levels before administration were relatively stable (ranging around 7.0–8.9 mmol/dL). However, following treatment, glucose levels increased significantly, reaching values such as 12.1 mmol/dL and 12.2 mmol/dL. This confirms that alloxan effectively induced hyperglycemia in the rats.

PCR analysis using the three selected primers revealed genetic alterations induced by alloxan treatment. The gel electrophoresis images (Figures 2–8) demonstrated changes in the DNA fingerprinting patterns of the rats. A comparison of the bands before and after alloxan administration revealed differences, with the induced samples displaying the appearance of additional bands. This suggests genetic modifications due to alloxan exposure, further supported by the dendrogram analysis (Figure 3), which highlights shifts in genetic diversity among the treated rats.

Dendrogram interpretation

The Dendrogram in figures 3, 5, 7 and 8, revealed a clustering in genetic diversity among the treated rats. This reveals the truth that diabetes altered the genetic profile of the rats.

Biological meaning of clustering

In biology, clustering refers to the process of grouping together similar biological entities, such as genes, proteins, or cells, based on shared characteristics or functions. This technique is widely used in systems biology and bioinformatics to analyze large datasets, identify patterns, and understand underlying biological mechanisms.

Determination of Genetic Similarity

The genetic similarities between albino rats genotypes were determined using Jaccard's coefficients, and a dendrogram was constructed by applying the unweighted pair group

Difference Between the Banding Pattern of Control and Diabetic Rat

A comparison of the bands before (control) and after alloxan administration (Diabetic) revealed differences, with the induced samples displaying the appearance of additional bands. This suggests genetic modifications due to alloxan exposure, and the primers which amplified unique bands include the following: (B06, H04, B08) primers.

Diabetic Animal Loci

In the diabetic animals, specific genetic loci associated with an increased risk of developing diabetes were identified. Among these loci, were - Iddm1, Iddm2, and Iddm4 and Iddm24, on chromosome 8

S/N	Animals	Treatment	Body Weight	Blood Glucose
1	Rat BI – 1	Pre-	170.5g	8.9mol/dl
2	Rat AI – 1	Post- 150mg/kg	165.2g	12.1mol/dl
3	Rat BI – 2	Pre-	172.1g	7mol/dl
4	Rat AI – 2	Post -150mg/kg	165.5g	12.1mol/dl
5	Rat BI – 3	Pre-	169.2g	7mol/dl
6	Rat AI – 3	Post- 150mg/kg	160.3g	11.9mol/dl
7	Ra tBI – 4	Pre-	203.78g	7mol/dl
8	Rat AI – 4	Post- 150mg/kg	194.76g	11.9mol/dl
9	Rat BI – 5	Pre-	202.1g	7mol/dl
10	Rat AI – 5	Post- 150mg/kg	197.58g	12.1mol/dl
11	Rat BI – 6	Pre-	182.1g	7mol/dl
12	Rat AI – 6	Post- 150mg/kg	175g	12.2mol/dl

Table 3: Rats' Weight Response and Blood Glucose Level before and after Alloxan Exposure

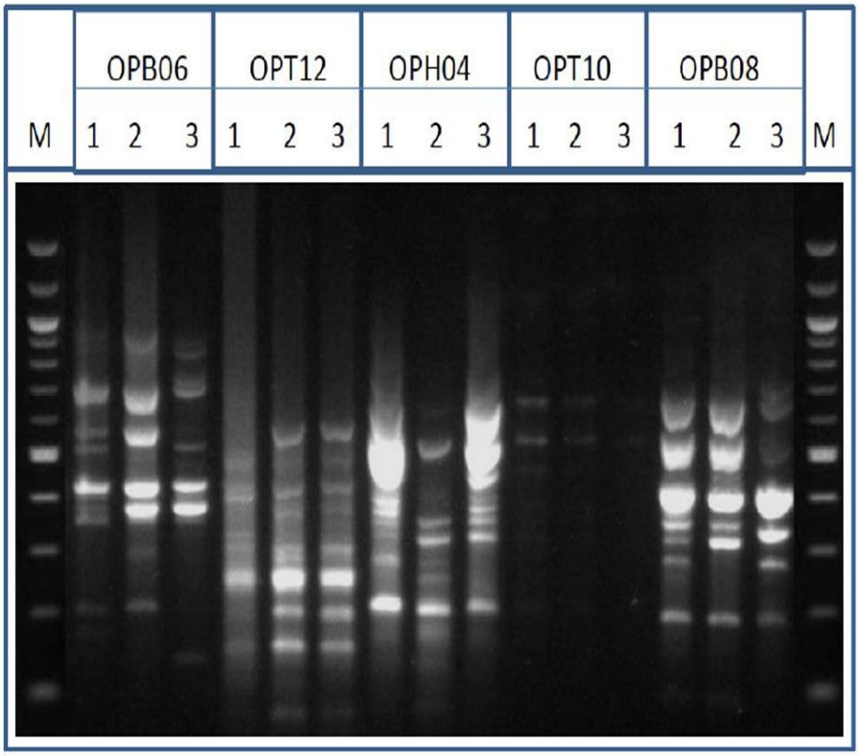

Figure 1: PCR Profile of 5 RAPD Primers used to screen three pooled rats' genomic DNA

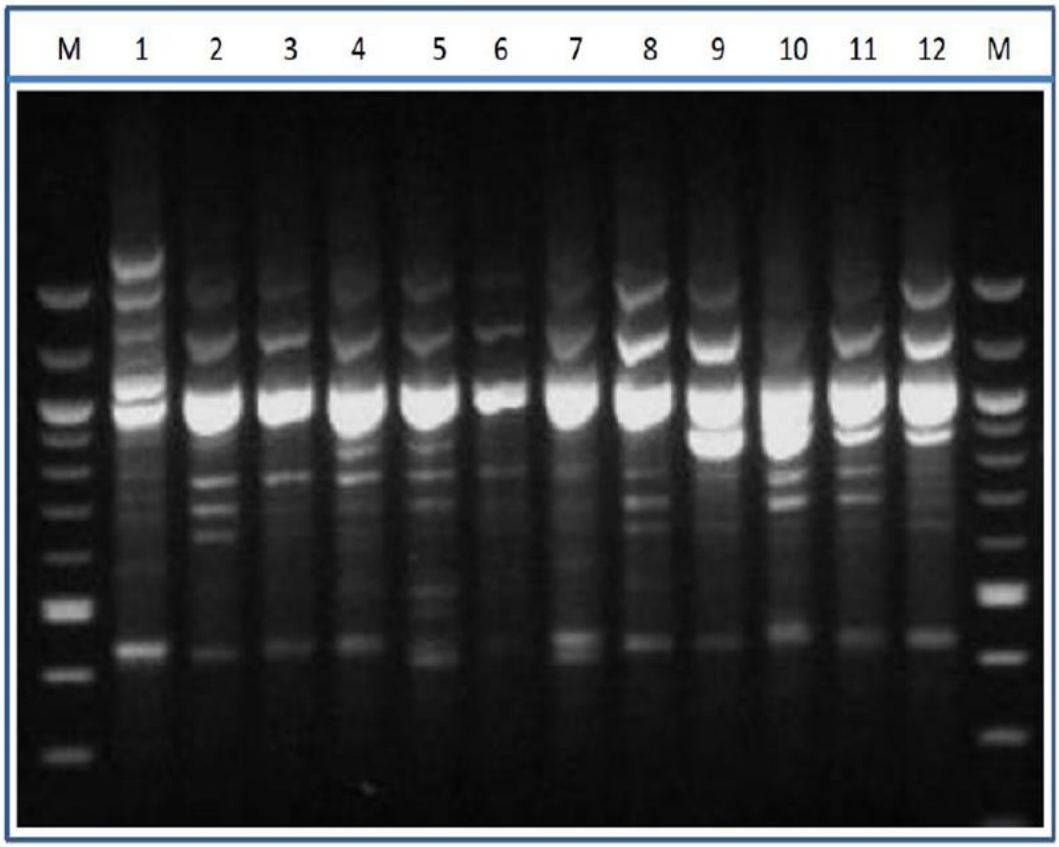

Figure 2: DNA Fingerprint of Rats as Revealed by B06 RAPD PCR analysis

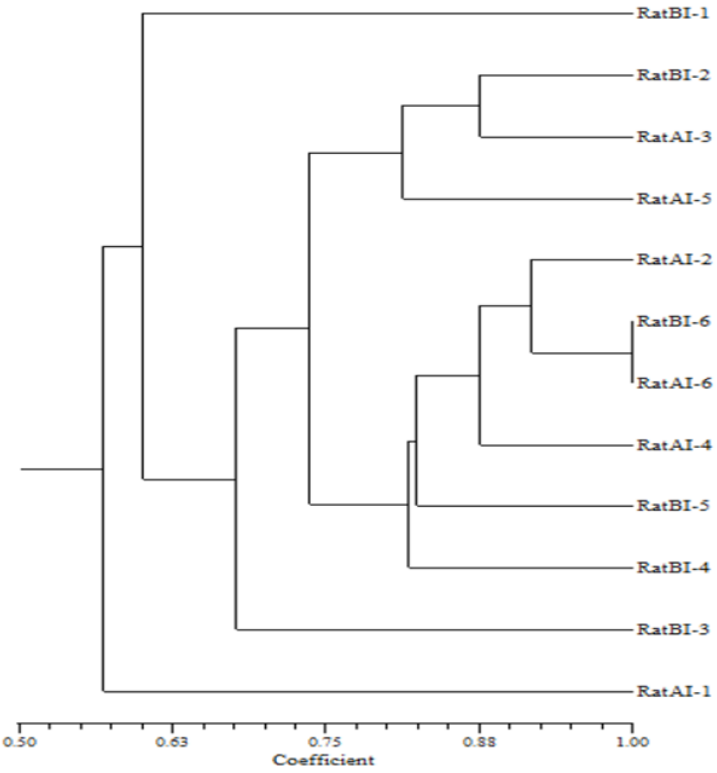

Figure 3: Dendrogram showing change in genetic diversity among 6 Rats after Alloxan treatment as revealed by B06 RAPD PCR analysis

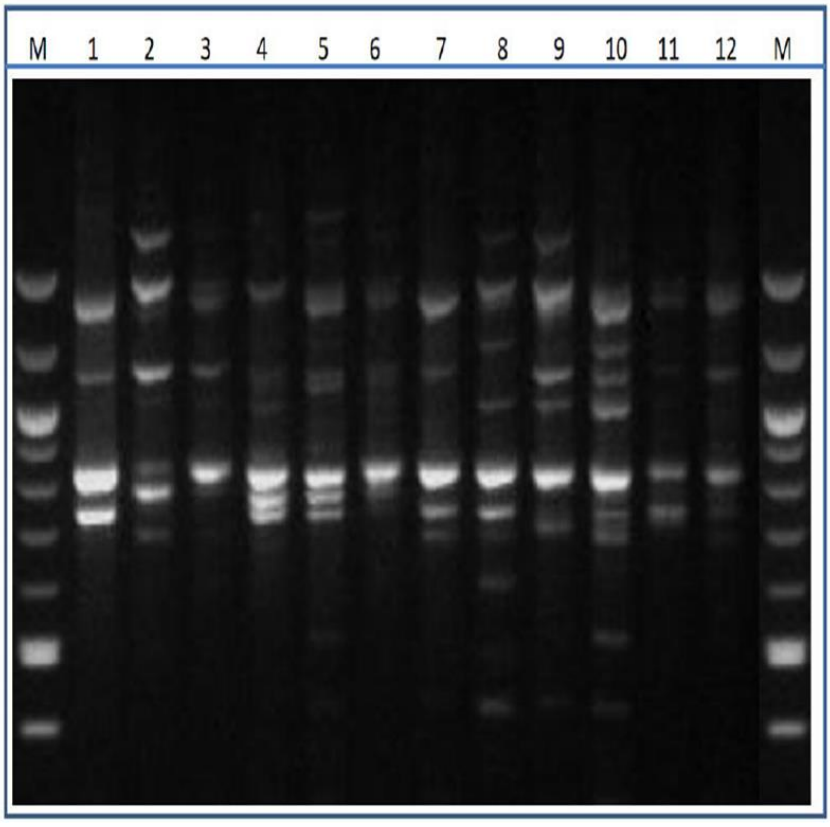

Figure 4: DNA fingerprint of Rats as revealed by H04 RAPD Analysis

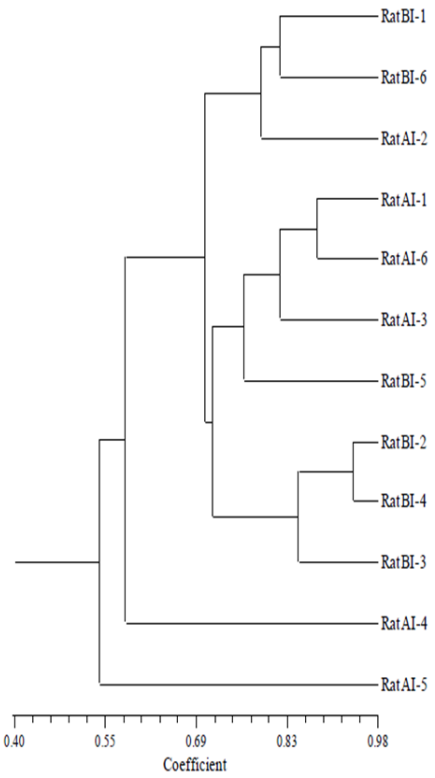

Figure 5: Dendrogram showing change in genetic diversity among 6 Rats after alloxan treatment as revealed by H04 RAPD PCR analysis

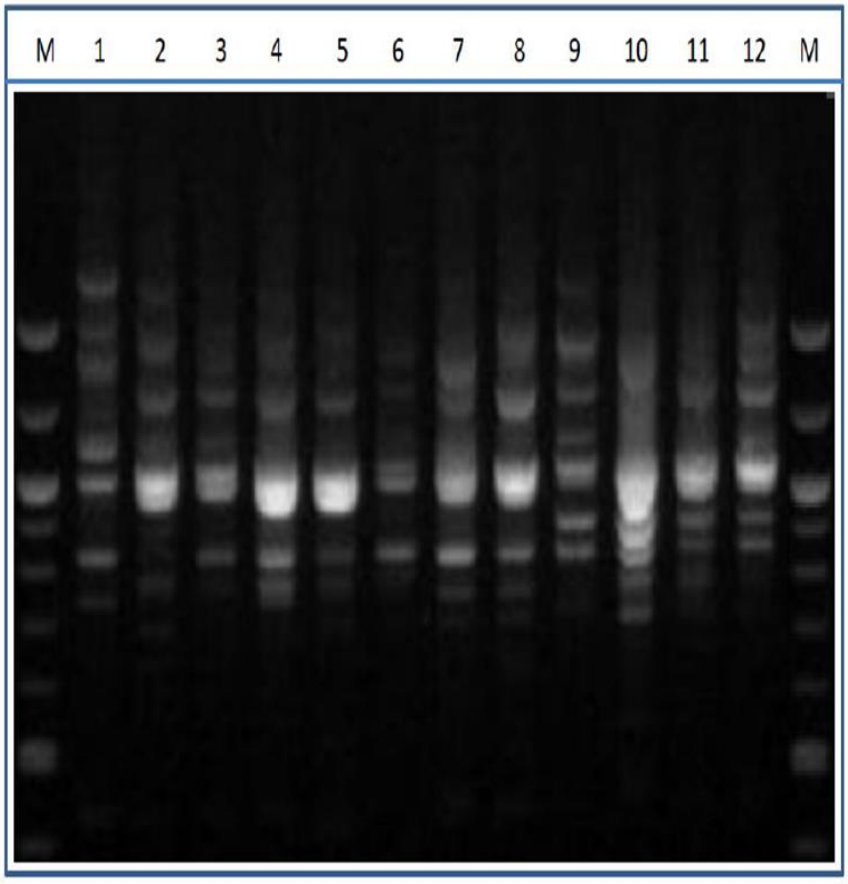

Figure 6: DNA fingerprint of Rat as revealed by B08 RAPD PCR Analysis

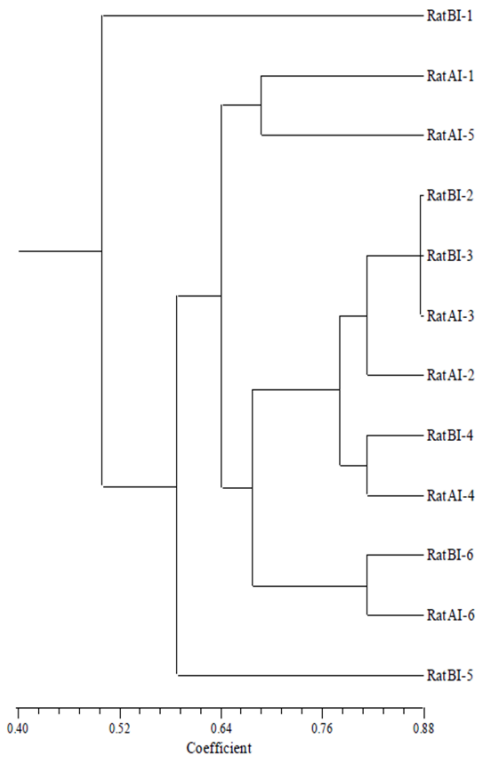

Figure 7: Dendrogram showing change in genetic diversity among 6 Rat after alloxan treatment revealed by B08 RAPD PCR analysis

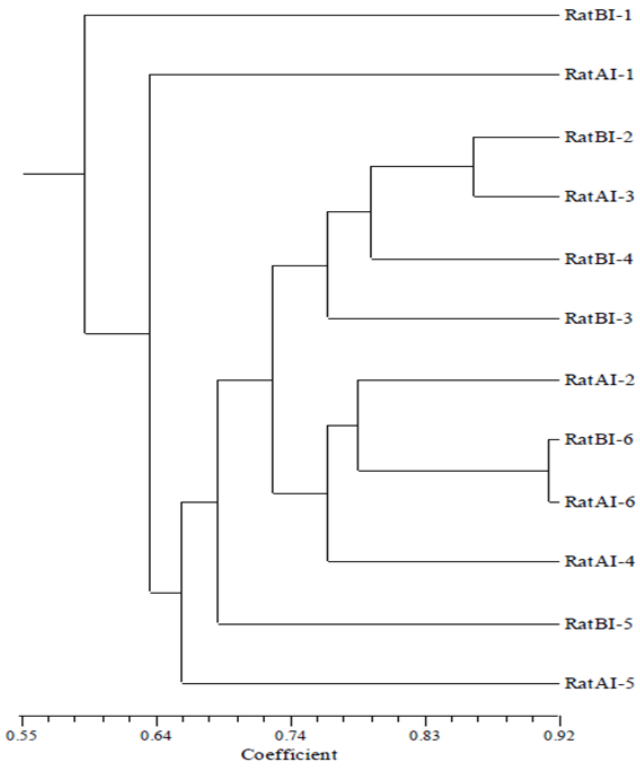

Figure 8: Dendrogram showing the change in genetic diversity among 6 Rats after alloxan treatment as revealed by 57 RAPD PCR markers

Discussion

Diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia due to impaired insulin secretion, action, or both [11]. The genetic basis of diabetes has been widely studied in humans, but limited research has been conducted on animal models to understand genetic markers of diabetic expression. This study aims to analyze genetic variations in diabetic Wistar rats induced with alloxan, using Random

Amplified Polymorphic DNA (RAPD) polymerase chain reaction (PCR) techniques. Administration of alloxan at a dose of 150 mg/kg resulted in a significant decrease in body weight among the treated rats. The weight reductions observed were 5.3g, 6.6g, 8.9g, 9.02g, 4.52g, and 7.1g, respectively, when compared to their pre-administration weights. This study indicates weight loss as a common consequence of alloxan-induced diabetes. For instance, a study by Yin et al. [12], reported significant weight loss in alloxan-induced diabetic rats, attributing this to increased muscle wasting and loss of tissue proteins due to insulin deficiency. In a related study on Sprague Dawley rats, Airaodion et al. [13] reported significant weight loss following alloxan administration, attributing it to the catabolic effects of insulin deficiency and hyperglycemia. Following alloxan administration, there was a significant increase in blood glucose levels in the treated rats. The elevations recorded were 3.2 mmol/L, 5.1 mmol/L, 4.9 mmol/L, 4.9 mmol/L, 5.1 mmol/L, and 5.2 mmol/L, respectively, compared to their baseline levels. This hyperglycemic effect is consistent with the known action of alloxan, which selectively destroys insulin-producing beta cells in the pancreas, leading to decreased insulin secretion and subsequent elevation in blood glucose levels. These findings are consistent with established research demonstrating that alloxan selectively destroys pancreatic beta cells, leading to insulin deficiency and subsequent hyperglycemia. Another study by Lenzen [14] corroborates this mechanism, highlighting that alloxan induces diabetes by generating reactive oxygen species (ROS) that damage pancreatic beta cells.

In this study, five primers were initially tested for variability and reproducibility through optimization and screening processes. The optimization was conducted three times under consistent conditions, yielding reproducible bands. Three primers (B06, H04, B08) were selected based on their reproducibility and polymorphism as revealed by agarose gel electrophoresis. These primers produced visible and consistent bands during the DNA fingerprinting analysis, indicating their suitability for detecting genetic variations.

The RAPD-PCR fingerprinting analysis using primers B06, H04, and B08 revealed distinct differences in band patterns between control and alloxan-induced rats. Specifically, there was an appearance of additional bands in the induced samples compared to the non-induced ones. This suggests that alloxan administration may lead to genetic alterations detectable by these primers. Similar findings were reported by Yarson et al. [15]. who observed changes in DNA band patterns in diabetic rats, indicating genetic instability associated with diabetes-induced oxidative stress. These findings are also in line with previous studies that have utilized RAPD-PCR to detect genetic variations in diabetic models, highlighting its utility in assessing the genetic impacts of diabetic conditions [16]. Nearly all RAPD markers are dominant, i.e. it is not possible to distinguish whether a DNA segment is amplified from a locus that is heterozygous (1 copy) or homozygous (2 copies). Co-dominant RAPD markers, observed as different-sized DNA segments amplified from the same locus, are detected only rarely. PCR is an enzymatic reaction, the quality and concentration of template DNA, concentrations of PCR components, and the PCR cycling conditions may greatly influence the outcome. Thus, the RAPD technique is notoriously laboratory dependent and needs carefully developed laboratory protocols to be reproducible. Mismatches between the primer and the template may result in the total absence of PCR product as well as in a merely decreased amount of the product. Thus, the RAPD results can be difficult to interpret. The qpcr and genomic sequencing are more robust methods of PCR analysis. qPCR adds two elements to the standard PCR process, that is, Fluorescent dye and Fluorometer. These two elements turn qPCR from a processing step in another procedure – to a measurement technique in its own right. Thermal cyclers meant for use with qPCR include a fluorometer to detect that fluorescence in real time as the thermal cycler runs, giving readings throughout the amplification process of the PCR. The Genomic sequencing method on the other hand is the process of determining the order of the chemical building blocks (nucleotides) in an organism's DNA or RNA. It involves sequencing the entire genome or specific regions of it to understand the genetic makeup and identify variations that may be associated with diseases or other traits [17]. The observed polymophism is random polymorphism.

Genetic variations, including mutations and polymorphisms, can significantly influence the progression of various diseases. These variations can affect a person's susceptibility to a disease, the rate at which it develops, and the severity of its symptoms. For instance, mutations in genes like HNF4A, GCK, HNF1A and mutations in KCNJ11 and ABCC8 genes, which affect potassium channels in pancreatic beta cells are implicated in the diagnosis of diabetes including neonatal diabetes [18]. Mutations in the APP, PSEN1, and PSEN2 genes are implicated in the diagnosis of early-onset of Alzheimer disease, while the APOE gene is a major risk factor for late-onset.

Conclusion

The genetic analysis of diabetic expression in Wistar rats using RAPD-PCR successfully demonstrated the impact of alloxan induction on body weight, blood glucose levels, and DNA fingerprinting. The study revealed a significant increase in blood glucose levels and a corresponding decrease in body weight following alloxan administration. The RAPD-PCR analysis further confirmed genetic variations induced by diabetes, as evidenced by the presence and absence of specific DNA bands in the gel electrophoresis patterns. These findings highlight the genetic alterations associated with diabetic conditions, reinforcing the potential use of RAPD-PCR in identifying genetic markers for diabetes susceptibility and progression. Hence, future studies should incorporate gene expression profiling to identify specific genes upregulated or downregulated due to alloxan-induced diabetes. Conducting a time-course analysis of genetic variations over different stages of diabetes progression would provide a deeper understanding of the molecular mechanisms involved. Investigating genetic variations in other animal models of diabetes could offer comparative insights into species-specific diabetic responses. And translating these findings to human studies through genomic approaches may aid in identifying genetic markers for early diabetes detection and targeted therapy. Assessing the effects of potential antidiabetic agents on the genetic expression of diabetes could provide insights into new therapeutic approaches.

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Conflict of Interest

There is none to declare by the Authors.

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