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DNA degradation in diabetes mellitus patients and its impact on DNA quality for forensic analysis

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Abstract: This study investigates the effect of Diabetes Mellitus (DM) on DNA degradation and its quality in forensic analysis. DNA, as a key element in forensic investigations, is susceptible to degradation due to the altered physiological state and chronic hyperglycemia prevalent in DM patients. Blood samples from DM patients with fasting blood glucose (FBG) levels greater than 126 mg/dL and HbA1c ≥7% were analyzed. The results showed a significant decrease in DNA concentration, with the control group showing 1353.2 ng/µL, and reductions observed on day 7 (989.9 ng/µL), day 14 (750.4 ng/µL), and day 20 (354.2 ng/µL). DNA purity remained relatively stable, with a slight reduction from 1.83 on day 7 to 1.74 on day 20. Statistical analysis using ANOVA indicated significant differences between time points for both DNA concentration ($p < 0.05$) and purity ($p < 0.05$). Games-Howell post hoc tests confirmed a significant decrease in DNA concentration over time ($p < 0.05$). The significant decrease in DNA concentration reflects DNA degradation, which worsens over time, while DNA purity remains within an acceptable range. The findings suggest that DNA samples derived from DM patients may exhibit a higher susceptibility to progressive degradation, potentially impacting the reliability of subsequent forensic analyses. This study emphasizes the need for advanced forensic techniques capable of handling degraded DNA samples, enhancing the sensitivity of forensic DNA analysis and ensuring reliable forensic identification.

Keywords: DNA Degradation; DNA Quality; Diabetes Mellitus; Forensic Analysis; DNA Concentration.

INTRODUCTION

DNA analysis serves as the gold standard for forensic identification due to its inherent uniqueness and individuality, which allows for the definitive differentiation between individuals[1]. However, the quality of DNA recovered from crime scene samples significantly dictates the success of forensic analysis and the subsequent identification process[2,3].

A significant challenge occurs when the donor's chronic health conditions accelerate genetic degradation even prior to environmental exposure[4,5]. One medical condition of profound concern in modern forensic studies is Diabetes Mellitus (DM)[6,7]. As a metabolic disorder characterized by chronic hyperglycaemia, DM does not merely impair vascular systems and organs; it also compromises molecular structures[8,9].

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Chronic hyperglycemia in DM patients triggers aberrant metabolic pathways, leading to the excessive accumulation of Reactive Oxygen Species (ROS)[10]. Under homeostatic conditions, ROS facilitate cellular signaling; however, in diabetic patients, an imbalance occurs between free radical production and the body's antioxidant defense mechanisms[11]. According to literature, chronic hyperglycemia in DM patients is theoretically associated with metabolic disturbances that may increase cellular oxidative stress[12]. Previous studies have indicated that these conditions can lead to base modifications, such as the formations of 8-oxoguanine (8-oxoG), and subsequent DNA strand breaks[13,14].

In addition to base modification, literature suggests that ROS-induced attacks can result in DNA strand breaks, occurring as either single strand or double strand breaks[15] Hyperglycemia is theoretically understood to trigger the cleavage of the phosphodiester bonds that constitute the DNA backbone, which leads to severe DNA fragmentation in a forensic context [16].

While prior research has addressed mitochondrial DNA mutations within the oxidative phosphorylation complex regarding insulin secretion[17,18], this study introduces a novel dimension by focusing on how these hyperglycemic conditions baseline a technical constraint in forensic identification through the evaluation of macro-level DNA degradation metrics, specifically concentration and purity. By analyzing concentration and purity via UV spectrophotometry in patients with fasting blood glucose levels >126 mg/dL and HbA1c ≥7%.

This study aims to examine the effect of Diabetes Mellitus on DNA degradation and quality in blood samples from DM patients by analyzing DNA concentration and purity. The samples were obtained from DM patients with fasting blood glucose >126 mg/dL and HbA1c ≥7%, with DNA concentration and purity measured using UV spectrophotometry to assess degradation. This research is expected to provide a deeper understanding of DNA degradation dynamics associated with Diabetes Mellitus and its potential impact on DNA quality in forensic analysis.

MATERIALS AND METHOD

This study employs a Laboratory-based cross-sectional comparative design to observe the profile of DNA degradation. Due to sample limitations, each specific time point (Day 0,7,14,20) was represented by an individual subject (n = 1 per group) with specific metabolic characteristics, analyzed using technical replicates to ensure analytical reliability. The focus of this approach is to compare quantitative and qualitative DNA parameters specifically concentration and purity, across these distinct time cohorts. The primary objective of this framework is to model the rate of genetic material degradation as influenced by chronic hyperglycemic pathophysiological conditions within a molecular forensic context.

The study population comprises Diabetes Mellitus patients undergoing clinical management at the Griya Luka Pringsewu Clinic. Subjects were selected via purposive sampling based on stringent inclusion criteria to ensure the homogeneity of the biological samples. The inclusion criteria restricted subjects to an age range of 35 to 75 years with standardized clinical profiles, defined by a fasting blood glucose (FBG) level >126 (mg/dL) and a glycated hemoglobin (HbA1c) level >7%. Standardizing these glycemic profiles is critical to minimizing inter-individual biological variability, which could otherwise confound the observed DNA degradation patterns.

The collected biological samples were allocated into four distinct observation groups based on the temporal dimension of storage or exposure. Each specific time point was represented by an individual subject (n =1) for each group possessing identical clinical criteria. The experimental groups were designated as follows: Control Group (Day 0): Establishes the baseline data to determine the initial DNA integrity prior to subsequent degradation, Experimental Group I (Day 7): Designed to evaluate the early phase of DNA degradation, Experimental Group

II (Day 14): Formulated to observe the progression of degradation during the intermediate phase, Experimental Group III (Day 20): Intended to analyze the detection limits and the extent of structural DNA damage during the advanced phase. To enhance internal validity and instrumental reliability, technical replications were performed in quintuplicate 5 times for each analytical measurement within every experimental group. This replication strategy was implemented to mitigate random experimental errors and to ensure that any observed data fluctuations accurately reflected the actual degradation phenomena within the samples, rather than procedural artifacts or pipetting variations.

Research Stages

Stage 1: Screening Diabetes Mellitus Patients

Screening involves testing fasting blood glucose (levels >126 mg/dL) and HbA1c (≥7%). Patients confirmed to have Diabetes Mellitus will proceed to the next stage.

Stage 2: Blood Sample Collection

Blood samples were taken from confirmed DM patients using a 3 mL EDTA vacutainer tube.

Stage 3: Blood Sample Droplet on Filter Paper

The blood samples were dropped onto filter paper, divided into four sections. Each section received one drop of blood, which was then left at room temperature. The four time groups are: control, day 7, day 14, and day 20.

Stage 4: DNA Extraction and Isolation

DNA isolation from blood began by pipetting 0.5 cc of blood into a centrifuge tube. DNAzol reagent (1 cc) was added and vortexed for 15 minutes to ensure thorough mixing. After adding chloroform (0.2 cc), the mixture was vortexed and incubated for 10 minutes. The next step involved centrifugation at 8,000 rpm for 10 minutes to separate the components. The supernatant was carefully transferred to a new Eppendorf tube, and isopropanol (0.5 cc) was added to precipitate DNA, incubated for 15 minutes, followed by centrifugation at 12,000 rpm for 10 minutes. The pellet was washed with 70% ethanol and centrifuged again. The pellet was air-dried for 5 minutes, and 50 µL of sterile water was added [19].

Stage 5: DNA Concentration and Purity Measurement

DNA concentration and purity were determined by pipetting 10 µL of the isolated DNA into an Eppendorf tube and adding 690 µL of water. Spectrophotometry was used to measure absorbance at wavelengths of 260 nm (λ260) and 280 nm (λ280). DNA concentration was calculated using the formula OD λ260 multiplied by the dilution factor and 50 ng/µL. DNA purity was calculated by comparing absorbance values at λ260 and λ280 (OD λ260 / OD λ280). DNA concentration and purity measurements were repeated five times for the samples on days 7, 14, and 20[20].

Stage 6: Data Analysis

Data obtained were analyzed using SPSS software for normality testing (Shapiro-Wilk test) and parametric statistical analysis using One-Way ANOVA to compare variations between time groups for DNA concentration and purity. The correlation between FBG and DNA quality parameters (concentration and purity) was established via Pearson correlation analysis. The calculation was mathematically derived from the paired datasets of individual glycemic measurements and the corresponding mean values of five-fold technical instrument repetitions. underscore a robust time-dependent linear trend between elevated glucose and DNA degradation during storage, despite the recognized limitations concerning the number of independent biological observations[21].

RESULTS AND DISCUSSION

Table 1 shows the characteristics of the samples and the measurement results for DNA concentration and purity at various time points. The control sample showed the highest Mean DNA concentration (1353.2 ng/µL) and DNA purity (1.71) compared to samples stored on days 7, 14, and 20. The DNA concentration decreased over time, from 1353.2 ng/µL in the control group to 354.2 ng/µL on day 20, indicating DNA degradation during storage[19]. Meanwhile, DNA purity remained relatively stable, slightly decreasing from 1.83 on day 7 to 1.74 on day 20.

Table 1. Baseline Characteristics of Participants with Diabetes Mellitus and DNA Quality Measurements Across Storage Time Points

Participant	Sex	Storage Time	Age (Years)	FBG (mg/dL)	HbA1c (%)	DNA Concentration (ng/µL), Mean	DNA Purity (λ260/λ280), Mean
A1	F	Control	65	252	7.3	1353.2	1.71
A2	F	Day 7	56	273	8.1	989.9	1.83
A3	F	Day 14	70	264	7.8	750.4	1.81
A4	F	Day 20	50	230	7.2	354.2	1.74

Note: FBG, fasting blood glucose; HbA1c, glycated hemoglobin. DNA concentration and purity values are presented as the mean of five technical replicates for each participant.

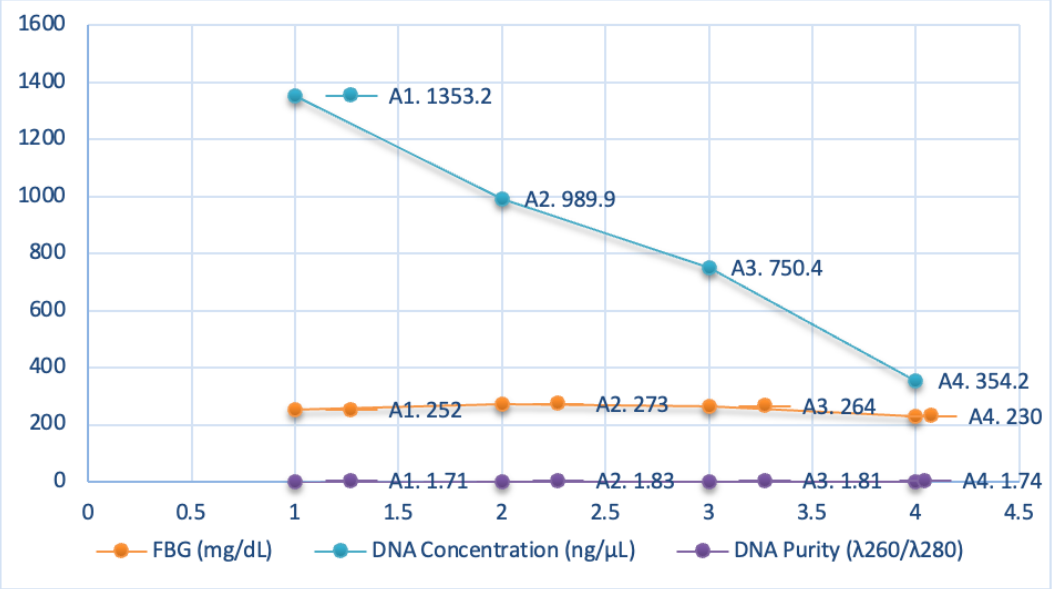

Figure 1. FBG with DNA Concentration and Purity

Figure 1. shows the changes in FBG (mg/dL), DNA concentration (ng/µL), and DNA purity (λ260/λ280) across the various time points (Control, Day 7, Day 14, and Day 20). The graph illustrates that while FBG remained stable, DNA concentration exhibited a significant decline over time. DNA purity showed a minor decrease on day 20, though it remained within acceptable limits for forensic analysis. The generally accepted threshold for DNA purity in forensic applications is between 1.8 and 2.0[22].

Figure 2 and 3 illustrates the variations in DNA concentration (ng/µL) and DNA purity (λ260/λ280) at four different time intervals: Control, Day 7, Day 14, and Day 20. The graph demonstrates a marked decrease in DNA concentration with increased storage time, while DNA purity remained relatively constant, with only a slight decline by day 20.

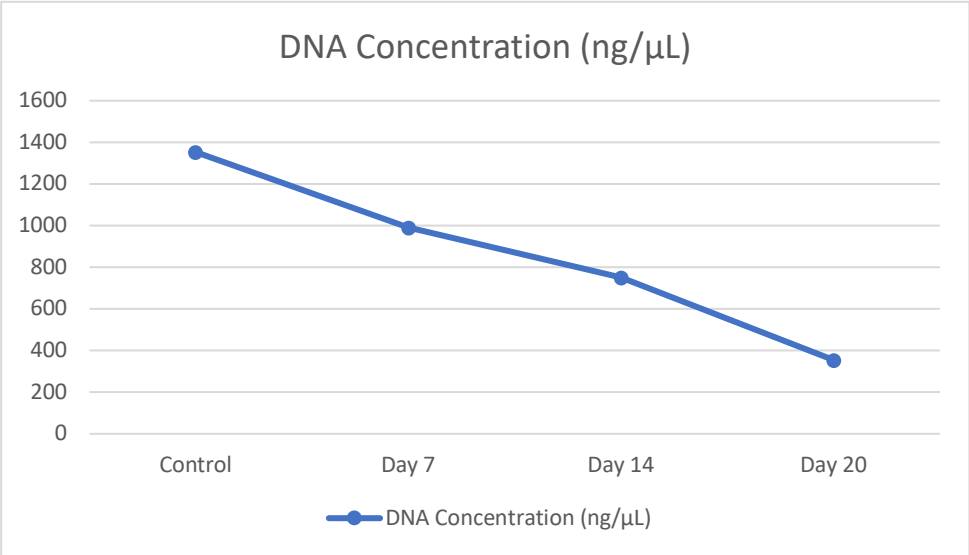

Figure 2. Time Variation with DNA Concentration

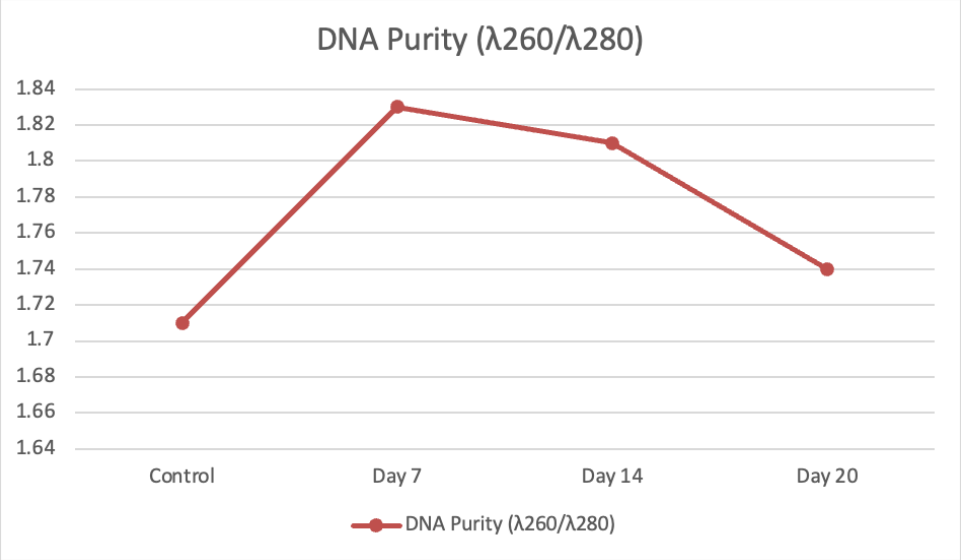

Figure 3. Time Variation with DNA Purity

Descriptive statistics and the results of the Shapiro–Wilk normality test are presented in Table 2**.** DNA concentration showed a progressive decrease across the predefined storage time points, from 1353.2 ± 37.35 ng/µL at Day 0 (Control) to 354.2 ± 9.96 ng/µL at Day 20. In contrast, DNA purity exhibited only minor fluctuations throughout the storage period, with mean A260/A280 ratios ranging from 1.71 ± 0.040 to 1.83 ± 0.043. The Shapiro–Wilk test, performed because of the small sample size (n = 5 technical replicates per group), indicated that all DNA concentration and DNA purity datasets were normally distributed, as all p-values exceeded the significance threshold of 0.05. Therefore, the assumption of normality required for parametric analysis was satisfied.

Variance homogeneity was subsequently evaluated using Levene's test. The results indicated homogeneous variances for DNA purity ($p > 0.05$), whereas DNA concentration violated the homogeneity assumption ($p = 0.034$). Consequently, one-way ANOVA was performed for both variables, and the Games–Howell post hoc test was selected for pairwise comparisons because it is robust to unequal variances.

Table 2. Results of the Shapiro–Wilk Normality Test for DNA Concentration and DNA Purity Across Storage Time Points

Parameter	Storage Time	n*	Mean ± SD	Shapiro–Wilk (p-value)
DNA Concentration (ng/µL)	Day 0 (Control)	5	1353.2 ± 37.35	0.173
	Day 7	5	989.9 ± 3.99	0.704
	Day 14	5	750.4 ± 6.79	0.157
	Day 20	5	354.2 ± 9.96	0.767
DNA Purity (A260/A280)	Day 0 (Control)	5	1.71 ± 0.040	0.314
	Day 7	5	1.83 ± 0.043	0.107
	Day 14	5	1.81 ± 0.038	0.613
	Day 20	5	1.74 ± 0.030	0.777

Note: *Shapiro–Wilk test was used to assess the normality of the data prior to parametric analysis. A p-value > 0.05 indicates that the data do not significantly deviate from a normal distribution.* * n = 5 represents technical replicates for each participant at each storage time point.

The one-way ANOVA results (Table 3) demonstrated that storage duration had a statistically significant effect on DNA concentration, $F(3,16) = 2260.539$, $p < 0.001$. The between-group variation (SS = 2,639,927.898; MS = 879,975.966) was substantially greater than the within-group variation (SS = 6,228.432; MS = 389.277), indicating a marked decline in DNA concentration across the storage time points. Similarly, storage duration significantly affected DNA purity, $\mathbf{F}(3,16) = 11.724$, $p < 0.001$. The between-group mean square (MS = 0.017) exceeded the within-group mean square (MS = 0.001), indicating significant differences in DNA purity among the storage time groups. Overall, these findings indicate that storage duration significantly influenced both DNA concentration and DNA purity, supporting the presence of measurable changes in DNA quality across the predefined storage intervals.[21,23]

Table 3. One-Way ANOVA Results for DNA Concentration and DNA Purity Across Storage Time Points

Parameter	Source of Variation	SS	df	MS	F	p-value
DNA Concentration	Between groups	2,639,927.898	3	879,975.966		
	Within groups	6,228.432	16	389.277	2260.539	<0.001
	Total	2,646,156.330	19			
DNA Purity	Between groups	0.052	3	0.017		
	Within groups	0.024	16	0.001	11.724	<0.001
	Total	0.076	19			

Note*:* SS = Sum of Squares; MS = Mean Square; df = degrees of freedom. One-way ANOVA was performed to compare DNA concentration and DNA purity across storage time points

Table 4. Effect of Storage Duration on DNA Concentration and DNA Purity Using Games-Howell Post Hoc Test

Storage Duration	n	DNA Concentration Mean ± SD	Post hoc Notation	DNA Purity Mean ± SD	Post hoc Notation
Day 0 (Control)	20	1353.2 ± 37.35	a	1.71 ± 0.040	a
Day 7	20	989.9 ± 3.99	b	1.83 ± 0.043	b
Day 14	20	750.4 ± 6.79	c	1.81 ± 0.038	c
Day 20	20	354.2 ± 9.96	d	1.74 ± 0.030	d

Note : Data are expressed as Mean ± Standard Deviation (SD). Different superscript letters (a, b, c, d) within the same column indicate statistically significant differences between storage duration groups based on Games-Howell post hoc test ($p<0.05$).

The effect of storage duration on genomic parameters was evaluated using a One-Way ANOVA. Due to the violation of the homogeneity of variances assumption (Levene's test, $p < 0.05$), Games-Howell post hoc test was employed to determine specific pairwise differences, as summarized in Table 4. The analysis revealed that storage duration significantly impacted both DNA concentration and DNA purity across all examined time points ($p < 0.05$).

For DNA concentration, a severe and continuous degradation trend was observed. The baseline concentration of 1353.2±37.35 ng/µL in the control group (Day 0) significantly dropped to 989.9±3.99 ng/µL on Day 7, followed by a further decline to 750.4±6.79 ng/µL on Day 14, and reached its lowest yield on Day 20 at 354.2±9.96 ng/µL. Each storage interval demonstrated distinct, non-overlapping

post hoc notations (*a, b, c, d*), confirming that the reduction in DNA quantity at each progressive time point was statistically significant under unequal variances.

Concurrently, DNA purity also exhibited statistically significant fluctuations throughout the storage period ($p < 0.05$). The purity ratio altered significantly from the initial control baseline (1.71±0.040, notation *a*) to Day 7 (1.83±0.043, notation *b*) and Day 14 (1.81±0.038, notation *c*). By Day 20, the purity value shifted again to 1.74±0.030 (notation *d*). The distinct post hoc notation for each group underscores that every examined storage duration uniquely and significantly influenced the resulting DNA purity values.

Table 5. Shapiro–Wilk Normality Test for FBG with DNA Concentration and DNA Purity

Parameter	FBG (mg/dL)	Shapiro–Wilk Statistic (W)	n*	p-value
DNA Purity	252	0.967	5	0.853
	230	0.902	5	0.421
	264	0.961	5	0.814
	273	0.881	5	0.314
DNA Concentration	252	0.964	5	0.837
	230	0.961	5	0.814
	264	0.817	5	0.111
	273	0.881	5	0.314

Table 5 presents the results of the Shapiro-Wilk normality test with FBG as a category, showing that the data for both DNA purity and DNA concentration are normally distributed at all FBG levels tested, as all p-values were greater than 0.05.[21]

Table 6. Pearson Correlation Analysis Between Fasting Blood Glucose (FBG) and DNA Quality Parameters

Variable 1	Variable 2	Pearson's *r*	p-value	n*
FBG	DNA Purity	0.093	0.683	20
FBG	DNA Concentration	-0.996	<0.001	20

Note: Pearson's correlation coefficient (r) was used to evaluate the linear relationship between fasting blood glucose (FBG) and DNA quality parameters. * n = 20 represents technical replicate measurements and should not be interpreted as independent biological observations.
Because the correlation analysis was performed using technical replicate measurements, the results primarily reflect measurement consistency within the experimental dataset and should not be interpreted as population-level biological associations.

The Pearson correlation analysis (Table 6) demonstrated statistical associations between fasting blood glucose (FBG) and the measured DNA quality parameters. FBG levels showed a negligible correlation with DNA purity ($r = 0.093$, $p = 0.683$) and strong correlation with DNA concentration ($r = -0.996$, $p < 0.001$). These findings indicate that FBG levels have a selective, powerful linear impact primarily on DNA concentration rather than purity; however, the results should be interpreted cautiously because the correlation analysis was based on technical replicate measurements rather than independent biological observations.

The progressive decline in DNA concentration observed over time suggests a compromised molecular stability in samples from Diabetes Mellitus (DM) patients. This phenomenon can be explained by established biochemical models, where chronic hyperglycemia is understood to exacerbate metabolic stress, potentially increasing free radical production and leading to subsequent cellular damage that affects DNA integrity[24]. The findings of this study show that DNA concentration in the control group (1353.2 ng/µL) was higher than in samples taken on day 7 (989.9 ng/µL), day 14 (750.4 ng/µL), and day 20 (354.2 ng/µL). This significant decrease indicates that DNA degradation occurs over time, triggered by internal factors such as chronic inflammation and metabolic disturbances[24,25]. The temporal parameters employed in this study (Day 0 to Day 20) served as a functional stress test to assess the inherent stability of DNA derived from Diabetes Mellitus (DM) patients. The use of different individuals with uniform clinical criteria (HbA1c and FBG) is an

effort to observe representative degradation patterns in the DM patient population, so that the research results are not biased by one individual's genetic profile alone. Rather than merely observing storage induced decay, the time dependent degradation observed highlights the compromised molecular integrity of the samples. These findings suggest that the altered metabolic environment in individual DM patients is suggested to have a potential role in predisposing DNA to fragmentation. Consequently, the rapid decline in DNA quality over time serves as a proxy for the reduced biological stability of the genome in individuals with DM, which has critical implications for the reliability of forensic genetic profiling. DNA purity also decreased, although it remained somewhat more stable than DNA concentration, from 1.92 in the control group to 1.73 on day 20. This suggests increasing contamination or DNA damage over time. Literature suggest that a major factor contributing to DNA degradation in diabetes patients is oxidative damage, which is driven by increased free radicals due to hyperglycemia[26,27]. Correlation results found no significant difference in purity between days 7 and 14 ($p > 0.05$), indicating that DNA purity may remain relatively stable despite a decrease in DNA concentration, especially during shorter storage periods (e.g., between days 7 and 14). The significant decrease in DNA concentration directly impacts the quality of forensic analysis[28]. To evaluate the linear relationship between metabolic and genomic parameters, a Pearson correlation analysis was executed specifically to determine the strength and direction of the association between fasting blood glucose (FBG) levels and downstream DNA metrics, namely concentration and purity (λ260 / λ280). It is critical to clarify that this correlation matrix was constructed by treating the mean values derived from the five technical replicates of each biological time-point as individual data points, rather than pooling independent biological subjects. The resulting correlation coefficients ($r = -0.996$ for concentration and $r = 0.097$ for purity) should be interpreted as a reflection of technical trend consistency rather than generalized population wide biological covariation. Degraded DNA will not yield accurate results for techniques such as PCR, which require high quality DNA[29,30]. This distinction is highly relevant for downstream molecular applications. While degraded or low concentration DNA can severely compromise the accuracy of techniques such as PCR which inherently require high quality and sufficient template DNA the stability of DNA purity observed here suggests that the metabolic state primarily threatens DNA yield. This study demonstrates that elevated FBG levels adversely affect DNA concentration in DM patients, potentially influencing the availability of amplifiable material for applications like forensic identification. Therefore, understanding how diabetes selectively impacts DNA metrics is essential for optimizing DNA analysis sensitivity, particularly when adopting advanced technologies designed to work with limited or compromised samples[31].

Limitation of the study, The authors acknowledge several limitations in this study. First, due to practical constraints in sample procurement, temporal degradation was not observed longitudinally from the same individual; rather, each time point was represented by distinct subjects with variations in age, HbA1c, and FBG levels. Consequently, the observed changes reflect inter-individual variations rather than true temporal degradation within a single subject. Second, the small sample size (n=4) and the absence of a healthy control group limit the generalizability of the findings. Lastly, the mechanistic pathway linking hyperglycemia to DNA damage was not validated through specific oxidative stress biomarkers, such as ROS assays. Future studies with larger, longitudinal cohorts and standard control groups are warranted to confirm these preliminary observations.

CONCLUSION

The results of this study show that DNA concentration in Diabetes Mellitus (DM) patients with high FBG levels significantly impacts DNA quality, where DNA concentration decreases over time, indicating DNA degradation potentially by hyperglycemia. While DNA purity remained relatively stable on days 7 and 14, a more significant decline occurred on day 20, suggesting that extended storage duration cohorts exhibit lower DNA quality parameters, highlighting a potential vulnerability of genetic material under these specific metabolic conditions, which affects DNA concentration more significantly than DNA purity, potentially influencing the accuracy of forensic and genetic analyses. Overall, longer storage durations and higher FBG levels may be associated with reduced DNA quality.

AUTHORS' CONTRIBUTIONS

Egita Windrianatama Puspa: Conceptualization, Writing - Original Draft, Funding acquisition. **Gustiadi Saputra:** Methodology, Resources, Project administration. **Nuri Muliani:** Supervision, Investigation, Data Curation. **Santika Dwi Lesmana:** Software, Validation, Writing - Review & Editing. **Susi Susanti:** Formal analysis, Visualization, Output.

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DATA AVAILABILITY STATEMENT

The minimal datasets generated, analyzed, and utilized to support the empirical findings and contribute to the outcomes of this investigation are available from the corresponding author on reasonable request.

DISCLOSURE STATEMENT

The views and opinions expressed in this article are those of the authors and do not necessarily reflect the official policy or position of any affiliated agency of the authors. The data is the result of the author's research and has never been published in other journals.

REFERENCES

1. Sahajpal V, Bhandari D. DNA profiling in India: Addressing issues of sample preservation, databasing, marker selection, & statistical approaches. *Science & Justice*. 2024;64(4):389-396. doi:10.1016/j.scijus.2024.05.003

2. Panneerchelvam S, Mohd Nor N. DNA Profiling in Human Identification: From Past to Present. *Malaysian Journal of Medical Sciences*. 2023;30(6):5-21. doi:10.21315/mjms2023.30.6.2
3. Singh OG. Crime Scene Investigation. In: *Current Innovations in Medicine and Medical Science Vol. 5*. Book Publisher International (a part of SCIENCEDOMAIN International); 2022:11-18. doi:10.9734/bpi/cimms/v5/3386C
4. Alketbi Salem K. The role of DNA in forensic science: A comprehensive review. *International Journal of Science and Research Archive*. 2023;9(2):814-829. doi:10.30574/ijsra.2023.9.2.0624
5. Wang X, Yang W, Zhu Y, et al. Genomic DNA Methylation in Diabetic Chronic Complications in Patients With Type 2 Diabetes Mellitus. *Front Endocrinol (Lausanne)*. 2022;13. doi:10.3389/fendo.2022.896511
6. Szczechla M, Balewska A, Naskręt D, Zozulińska-Ziółkiewicz D, Uruska A. Molecular Changes in Cells of Patients with Type 2 Diabetes Mellitus Depending on Changes in Glycemia Level in the Context of Lifestyle—An Overview of the Latest Scientific Discoveries. *Curr Issues Mol Biol*. 2023;45(3):1961-1981. doi:10.3390/cimb45030126
7. Elshad Abuzar Novruzov, Dilara Magsood Aliyeva, Sabina Anvar Mashadiyeva-Bayramova, Adil Allahyar Bayramov. Diabetes mellitus and anti-oxidative stress. *International Journal of Science and Research Archive*. 2023;9(1):390-393. doi:10.30574/ijsra.2023.9.1.0425
8. Yang T, Qi F, Guo F, et al. An update on chronic complications of diabetes mellitus: from molecular mechanisms to therapeutic strategies with a focus on metabolic memory. *Molecular Medicine*. 2024;30(1):71. doi:10.1186/s10020-024-00824-9
9. Zhang Q, Wu C, Liu Y, et al. Chronic Inflammation Plays a Role of a Bridge Between Cardiovascular Disease and Hyperglycemia. *Metab Syndr Relat Disord*. 2023;21(8):468-474. doi:10.1089/met.2023.0086
10. Thaina De Santos Silva L, Paula De Sousa Figueiredo M. Inflammatory Markers In Chronic Diabetic Complications. *Health and Society*. 2023;3(03):294-314. doi:10.51249/hs.v3i03.1415
11. Wani ZA, Sharma AK, Muzamil S, et al. Molecular pathways of oxidative stress in diabetes: redox imbalance and insulin pathway dysregulation. *Mol Biol Rep*. 2026;53(1):222. doi:10.1007/s11033-025-11389-z
12. Hong X, Hu Y, Yuan Z, et al. Oxidatively Damaged Nucleic Acid: Linking Diabetes and Cancer. *Antioxid Redox Signal*. 2022;37(16-18):1153-1167. doi:10.1089/ars.2022.0096
13. Bennici G, Almahasheer H, Alghrably M, et al. Mitigating diabetes associated with reactive oxygen species (ROS) and protein aggregation through pharmacological interventions. *RSC Adv*. 2024;14(25):17448-17460. doi:10.1039/D4RA02349H
14. Al-dulaimi DHA. Evaluation of 8-Oxoguanine DNA Glycosylase-1(OGG1) Serum Levels in Patients with Type2 Diabetes Mellitus. *Wasit Journal for Pure sciences*. 2024;3(4):156-163. doi:10.31185/wjps.543
15. Mahmoud HM, Ali AF, Hassan WM, Ahmed IH, Altimimi DJ. Evaluation of Oxidative DNA Damage among Type 2 Diabetes Mellitus Patients and Healthy Individuals in Duhok, Iraq: A Case-control Study. *Journal Of Clinical And Diagnostic Research*. Published online 2023. doi:10.7860/JCDR/2023/53508.17344
16. Agus Saputra IPB, Ni Made Wiasty Sukanty, Suci Fitriana Pramudya, Putu Kermawan, Irna Olvaliani Aimang. Peran NRF2 Dan NF-KB Dalam Patogenesis Diabetes Melitus: Narrative Review. *Indonesian Journal of Health Research Innovation*. 2026;3(2):273-285. doi:10.64094/7hf4mg65

17. Praveen Kumar, Jyothi MN, Prashant A. Mitochondrial DNA variants in the pathogenesis and metabolic alterations of diabetes mellitus. *Mol Genet Metab Rep*. 2025;42:101183. doi:10.1016/j.ymgmr.2024.101183
18. Berezina TA, Kopytsya MP, Petyunina O V., et al. Lower Circulating Cell-Free Mitochondrial DNA Is Associated with Heart Failure in Type 2 Diabetes Mellitus Patients. *Cardiogenetics*. 2023;13(1):15-30. doi:10.3390/cardiogenetics13010003
19. Puspa EW, B.Tena HA, Setiawan F, Ruth MSA, Yudianto A. Analysis of time exposure to DNA touch quality on face shield using STR CODIS – TH01 and D18S51. *Jurnal Teknologi Laboratorium*. 2022;10(2):68-74. doi:10.29238/teknolabjournal.v10i2.287
20. Yudianto A, Kurniawan A, Setiawan F, Puspa EW, Youngest R. 42 Sibling Pair Analysis In The Identification Process Of The Madurese Population Using STR CODIS Loci. *J Infect Dis*. 2022;16(2):42-54. doi:10.21010/Ajid
21. Sianturi R. Uji Normalitas Sebagai Syarat Pengujian Hipotesis. *Jurnal Pembelajaran Dan Matematika Sigma (JPMS)*. 2025;11(1):1-14. doi:10.36987/jpms.v11i1.7091
22. Yudianto A, Setiawan F. The Effectiveness of Mini Primer STR CODIS in DNA Degradation as the Effect of High-Temperature Exposure. *Analytical Cellular Pathology*. 2020;2020(2007):1-6. doi:10.1155/2020/2417693
23. Juarros-Basterretxea J, Aonso-Diego G, Postigo Á, Montes-Álvarez P, Menéndez-Aller Á, García-Cueto E. Post-hoc tests in one-way ANOVA: The case for normal distribution. *Methodology*. 2024;20(2):84-99. doi:10.5964/meth.11721
24. González P, Lozano P, Ros G, Solano F. Hyperglycemia and Oxidative Stress: An Integral, Updated and Critical Overview of Their Metabolic Interconnections. *Int J Mol Sci*. 2023;24(11):9352. doi:10.3390/ijms24119352
25. Zhao Y, Simon M, Seluanov A, Gorbunova V. DNA damage and repair in age-related inflammation. *Nat Rev Immunol*. 2023;23(2):75-89. doi:10.1038/s41577-022-00751-y
26. ElSayed NA, Aleppo G, Aroda VR, et al. Addendum. 2. Classification and Diagnosis of Diabetes: *Standards of Care in Diabetes—2023* . Diabetes Care 2023;46(Suppl. 1):S19–S40. *Diabetes Care*. 2023;46(9):1715-1715. doi:10.2337/dc23-ad08
27. Singh A, Kukreti R, Saso L, Kukreti S. Mechanistic Insight into Oxidative Stress-Triggered Signaling Pathways and Type 2 Diabetes. *Molecules*. 2022;27(3):950. doi:10.3390/molecules27030950
28. Poethe SS, Holtel J, Biermann JP, et al. Cost-Effective Next Generation Sequencing-Based STR Typing with Improved Analysis of Minor, Degraded and Inhibitor-Containing DNA Samples. *Int J Mol Sci*. 2023;24(4):3382. doi:10.3390/ijms24043382
29. Fattorini P, Previderè C, Livieri T, Zupanc T, Pajnič IZ. SNP analysis of challenging bone DNA samples using the HID-Ion AmpliSeq™ Identity Panel: facts and artefacts. *Int J Legal Med*. 2023;137(4):981-993. doi:10.1007/s00414-023-03019-9
30. Djelić N, Borozan S, Dimitrijević-Srećković V, et al. Oxidative Stress and DNA Damage in Peripheral Blood Mononuclear Cells from Normal, Obese, Prediabetic and Diabetic Persons Exposed to Thyroid Hormone In Vitro. *Int J Mol Sci*. 2022;23(16):9072. doi:10.3390/ijms23169072
31. Brawerman G, Pipella J, Thompson PJ. DNA damage to β cells in culture recapitulates features of senescent β cells that accumulate in type 1 diabetes. *Mol Metab*. 2022;62:101524. doi:10.1016/j.molmet.2022.101524