Advances in Bioresearch
Adv. Biores., Vol 16 (5) September 2025: 558-569

Print ISSN 0976-4585; Online ISSN 2277-1573
Journal's URL:http://www.soeagra.com/abr.html
CODEN: ABRDC3
DOI: 10.15515/abr.0976-4585.16.5.559569

Advances in Bioresearch

ORIGINAL ARTICLE

In Silico Investigation of Mirror Repeats Within Glucagon, Pyruvate Kinase (PKLR), Sox17 and GAPDH Diabetic Genes of *Rattus Norvegicus*

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ABSTRACT
Repetitive sequences are enormously distributed in the genomes of all viruses, bacteria, plants and animals. Repeats in coding and non-coding areas have been shown to have important effects on variation induction, gene expression regulation, and evolution. Many repeated sequences are capable of adopting non-B DNA conformations, which are thought to be essential for basic genetic processes. As a result, we were curious about the quantity and chromosomal distribution of repeated elements. Because of advances in bioinformatics, computational biology, and genomics, biological databases have grown rapidly during the last ten years. A computational method was used in this study to identify mirror repetitions in the Rattus norvegicus's Glucagon, pklr, sox17 and GAPDH genes. The Fast Parallel Complement Blast (FPCB) tool, a rapid and efficient bioinformatics method for identifying gene and genome repeats, was used to do this. 199 mirror repeats were discovered in glucagon, 167 in the pyruvate kinase (pklr), 135 in the sox17 and 116 in GAPDH genes during this study, whereas 5 mirror repeats were discovered in glucagon, 23 in the pyruvate kinase (pklr) and 16 in the sox17 and 16 in GAPDH genes when just the exonic areas were taken into account. Through this study, we can better understand the function of mirror repeats in the Rattus norvegicus's glucagon, pklr, sox17 and GAPDH genes which may be further linked to mirror repeats in the human genome.
Keywords*: Rattus norvegicus, Repetitive sequences, non-canonical secondary structures, Mirror repeat, FPCB, Diabetic genes.*

Received 24.05.2025 Revised 01.08.2025 Accepted 11.09.2025

How to cite this article:
Babli, Kavita S, Parveen K. *In Silico* Investigation of Mirror Repeats Within Glucagon, Pyruvate Kinase (PKLR), Sox17 and GAPDH Diabetic Genes Of Rattus Norvegicus. Adv. Biores. Vol 16 [5] September 2025. 558-569

INTRODUCTION

The repetitive sequences of the genome play a central role in the stability of the chromosome, the cell cycle and the regulation of gene expression, and they are also important substrates for genome evolution [1]. Variations in the number and kinds of repetitive sequences among animals may indicate how quickly an organism adapts to environmental changes [2]. They play an integral part in the cooperative molecular interactions that result in nucleoprotein complexes [3], and have also been related to cellular and molecular malfunctions that cause human diseases [4]. Some DNA sequence motifs can fold into secondary conformations that are different from the standard right-handed B-DNA helix with 10 bp per turn, at least for a portion of their length [5]. The length of these non-B DNA sequence motifs varies from a few dozen to a few hundred nucleotides, and their distribution throughout the genome is not random [6]. There are two repeat arms in each mirror, inverted, and direct repeat locus; these are often divided by a non-repetitive spacer. To construct H-, or triplex, DNA, homopurines and homopyrimidines can be arranged in mirror repeats with or without a less than or equal to100-nucleotide gap [7]. The patterns of nucleotide substitution inside repeat arms varied depending on the type of repeat [8]. The observed nucleotide substitution frequencies may be a result of processing by low-fidelity, specialized DNA polymerases [9], gene conversion, and increased DNA damage. The lower nucleotide substitution frequencies in the repeat arms of inverted and direct repeats may be explained by gene conversion, which

is frequent in cruciform and slipped-strand structures [10]. This is especially true for fixed nucleotide substitutions, where several rounds of gene conversion may have occurred. One significant explanation for the rise in mutagenesis at mirror repeats was suggested to be oxidative DNA damage [11]. It has been noted that Z-DNA and H-DNA-forming mirror repeats raise the frequency of mutations in their neighboring regions [12]. Nucleotide substitution frequencies at mirror, inverted, and direct repeats increase and decrease. Spacers had higher frequencies than repeat arms for each of the three repetition types. This is in line with other experimental research showing that spacers are more changeable in hairpin structures [13] and is suggestive of findings for cancer mutations) [6]. Approximately 13% of the human genome can fold into non-canonical (non-B) DNA structures (e.g. G-quadruplexes, Z-DNA, etc.) (Fig.1) [6], which have been implicated in vital cellular processes.

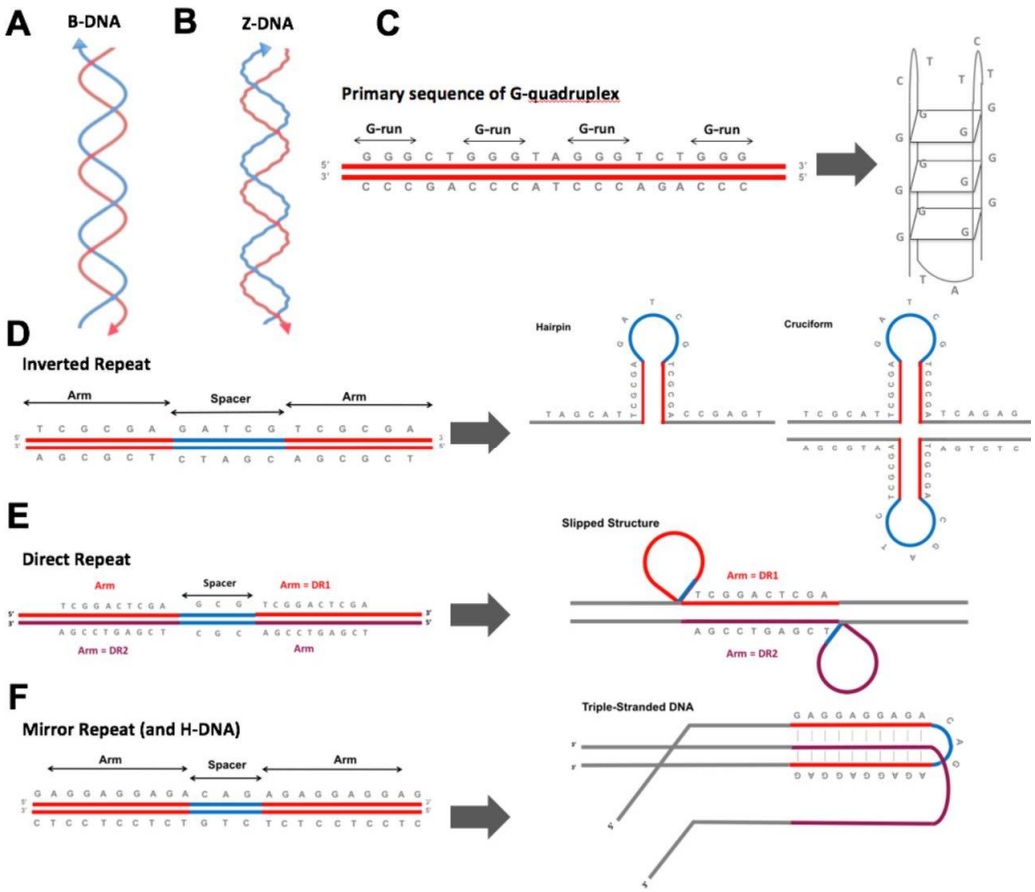


Fig 1. Repetitive sequences and non-canonical secondary structures.

All abundant repetitions' genomic distribution analysis revealed that they are essentially not found in coding sequences [14]. Repeated sequences can be divided into two major families- dispersed repeats and tandem repeats. Subfamilies can be formed within each of these two families. In contrast to tandem repeats, which are further classified into satellite DNA, minisatellites, and microsatellites, dispersed repeats contain transposons [15], tRNA genes, and gene paralogues [16].

Two main methods can be used to create animal models: genetic modification or disease induction. Both are important because they allow specific pathways associated with the disease to be analyzed, which is crucial for comprehending the pathophysiology and course of the disease and extrapolating to humans. About 25,000 genes make up the genome of Rattus norvegicus, and more than 90% of these genes match those in humans and mice. For sequence data, one may use DNA, RNA or Protein data depending on your interest. All the completed sequences can be retrieved from the NCBI public website: http://www.ncbi.nlm.nih.gov/. The database for the DNA sequence is called GenBank. According to earlier research, mirror repeats were implicated in karyotypic evolution, gene expression, chromosome structure, cell cycle, and chromosomal relocations [17]. The manual bioinformatics approach is used to identify mirror repeats where FPCB (FAST PARALLEL COMPLEMENT BLAST) is the most commonly used method for analyzing or locating mirror repeats in various prokaryotes and eukaryotes In addition, it was used to characterize maleless (mle) gene, a sex determination gene in *Drosophila melanogaster* [18].

Mirror-image nucleosides, as potential antiviral drugs, can inhibit virus DNA polymerase to prevent virus replication. Conversely, they may be inserted into the DNA strands during DNA replication or transcription processes, leading to mutations that affect genome stability [19].The existence of highly divergent repeats could be associated with the presence of a single-type triplet periodicity in various genes or with the packing of bacterial DNA into a nucleoid [20]. The objective of the current study is to identify the mirror repeats in *Rattus norvegicus*'s glucagon, pyruvate kinase (pklr), sox17 and GAPDH genes. This study can be used as a precursor to investigate the role and function of mirror repeats in these genes of *Rattus norvegicus*.

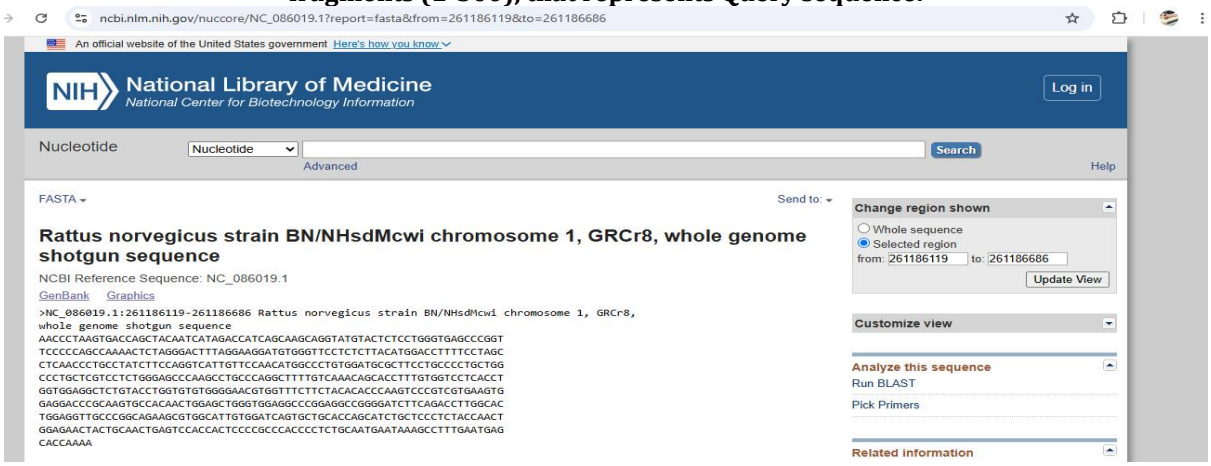
Mammalian Glucagon (Gcg) is primarily expressed in the alpha cells of the pancreas, L-cells of the intestine, and some neuronsof the brainstem [21].In diabetic rats, glucagon gene expression is augmented and is accompanied by hyperglucagonemia in conditions of hyperglycemia and insulin deficiency. Pancreatic loss of Sox17 resulted in trafficking defects of proinsulin through the ER, dilated and distended secretory organelles, and a trend of increased secretion of proinsulin, all of which are hallmarks of prediabetes [22]. Sox17 is expressed at high levels in the human fetal pancreas and at lower levels in adult islets [23]. Pyruvate kinase is a key glycolytic enzyme. Isoforms that are expressed in the red cell, liver, pancreatic beta-cells, small intestine, and proximal renal tubule are encoded by the 12 exons of the PKLR gene, which maps to chromosome 1q23. The PK-L isoform is expressed in pancreatic β-cells, small intestine, and proximal renal tubule [24]. The gene is upregulated by glucose, perhaps acting through the carbohydrate response element in the L-type promoter region [24] and is among the downstream targets of hepatocyte nuclear factor (HNF)-1α [25].

Embryos from diabetic rats and embryos cultured in high glucose concentrations [26] showed decreased activity of GAPDH (by 40–60%) and severe dysmorphogenesis on gestational days 10.5 and 11.5. A possible consequence of excess ROS generation in the mitochondria would be increased ROS leakage and inhibition of the cytosolic glycolytic enzyme glyceraldehyde-3-phosphate dehydrogenase (GAPDH). This enzyme has displayed sensitivity to ROS in several different conditions of oxidative stress [27].

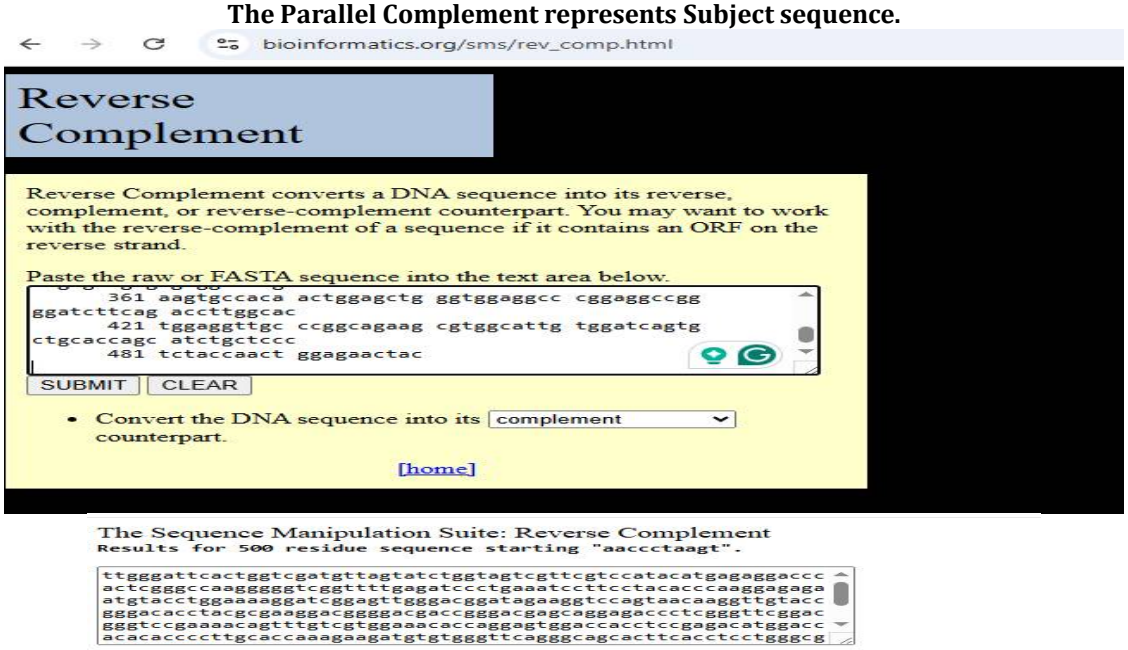
METHODS

The coding sequence of the glucagon, pklr, sox17 and GAPDH genes of *Rattus norvegicus* was downloaded from the NCBI website (link: https://www.ncbi.nlm.nih.gov/). The nucleotide base pair sequence was divided into 500 base pair regions. This sequence represents the query sequence. Using the Reverse complementary tool (https://www.bioinformatics.org/sms/rev_co mp.html), the parallel complement of each region of the query sequence was retrieved. This parallel complement sequence represents the subject sequence. Using the BLAST tool, both the coding sequence (query sequence) and its parallel complement (subject sequence) were aligned for the BLAST homology search (https://blast.ncbi.nlm.nih.gov/Blast.cgi?PAGE_TYPE=BlastSearch). Here within the result of the hit, if the position number is precisely reversed in query and subject, it will be a mirror repeat. Similarly, mirror repeats were analyzed within different exons of genes. The algorithm parameters were fixed for the word size 7, the threshold value at which maximum no. of hits were observed and was used to identify mirror repeats in all genes. The identified mirror repeats were classified as perfect mirror repeats, perfect mirror repeats with one spacer and imperfect mirror repeats (Fig. 2) [30].

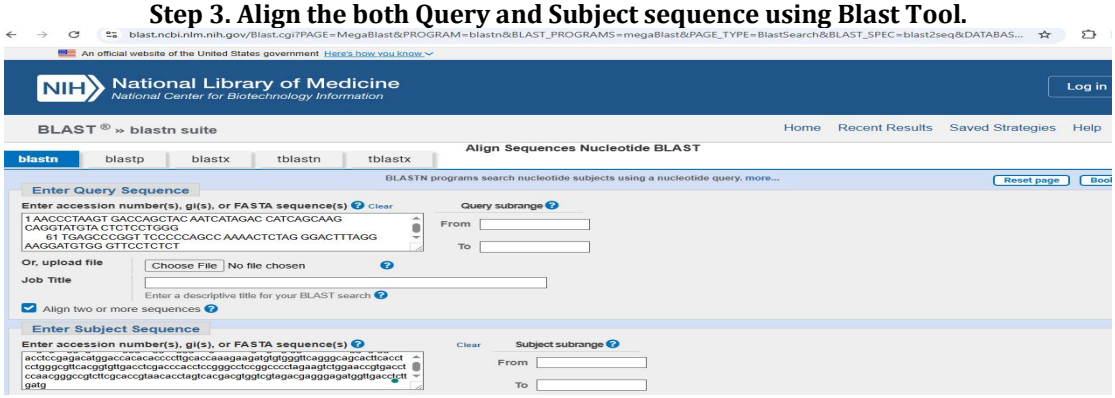
Step 1. Retrieve the original nucleotide sequence from NCBI, divide the whole sequence into fragments (1-500), that represents Query sequence.



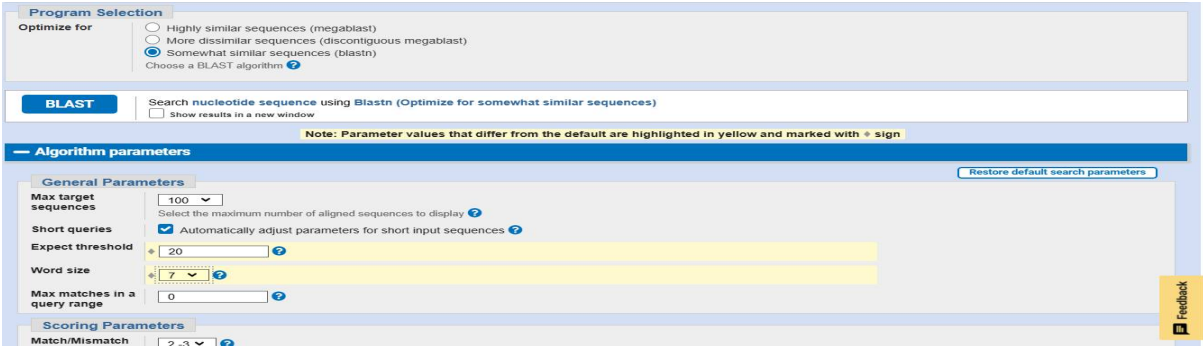
Step 2. Using the Reverse Complement Tool, make the Parallel Complement of Query sequence. The Parallel Complement represents Subject sequence.



Step 3. Align the both Query and Subject sequence using Blast Tool.



Set program selection – Somewhat similar sequences, Algorithm parameters – expected threshold 20 and word size limit 7.



Step 4. Mirror repeats can be detected by examining the nucleotide which are reversed in Query and Subject sequence.

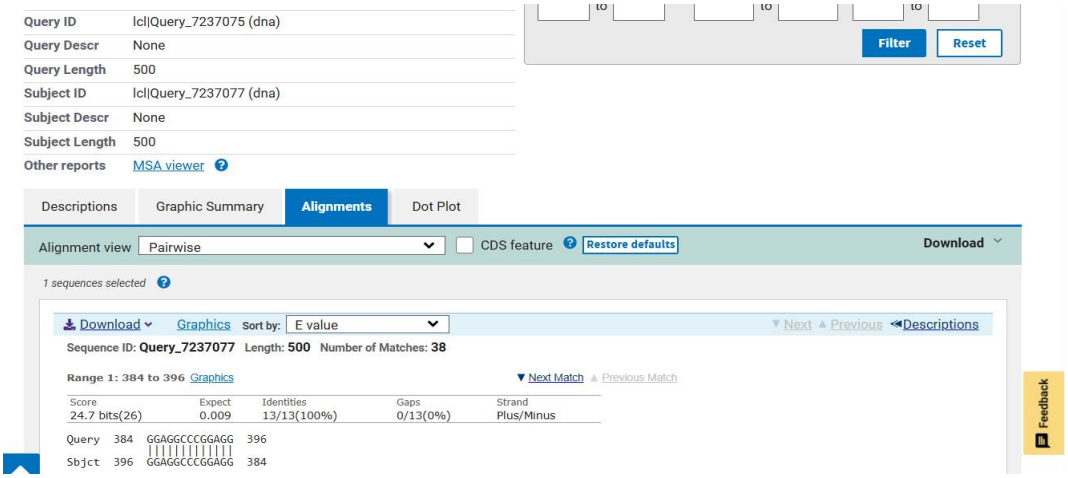


Figure 2. FPCB approach to find out mirror repeats

RESULTS

In the present study, a simple bioinformatics approach was used to identify mirror repeats in the glucagon, pklr, sox17 and GAPDH genes of *Rattus norvegicus*.

Complete gene mirror repeats in the Glucagon gene:

With the help of the BLAST tool and using a simple manual strategy, we have identified mirror repeats within the glucagon of *Rattus norvegicus*. The nucleotide sequence of the gene was divided into 500 base pair regions to identify the maximum number of mirror repeats. During the present investigation, we noticed that if we increase the threshold value (E), the number of hits changes, so firstly, we selected the threshold value at which there is no change in number of hits. The maximum number of hits were observed at threshold value 20 for different regions and threshold value 10 for exons of glucagon gene, so we identify mirror repeat at these values. A mirror repeat with identical sequences around the center axis of symmetry is termed a perfect mirror repeat. A perfect mirror repeats have one spacer element is termed a perfect mirror repeat with one spacer. Mirror repeats that have mismatch sequences around the center axis are termed imperfect mirror repeats. Further, the 538 mirror repeats were identified in the Pax6 gene at the expected threshold 20 (Fig. 3).

In Glucagon gene, out of 199 mirror repeats, 122 were perfect with one spacer, 50 were perfect and 27 were imperfect repeats. A maximum number of hits 60 observed in fragment 4001-4500 and the corresponding mirror repeats were 13 with 6 perfect, 1 imperfect and 6 perfect with one spacer type as shown in Table 1.

Table 1. Number of mirror repeats of Glucagon gene and their type at expected threshold E-valve 20.

Complete gene sequence	Expected threshold	No. of Hits	No. of mirror repeats	Perfect mirror repeats	Perfect mirror repeats with one spacer	Imperfect mirror repeats
1-500	20	51	11	2	7	2
501-1000	20	38	10	1	9	-
1001-1500	20	60	7	3	2	2
1501-2000	20	51	17	5	9	3
2001-2500	20	28	14	2	12	-
2501-3000	20	42	10	1	8	1
3001-3500	20	27	11	7	4	-
3501-4000	20	42	11	1	3	7
4001-4500	20	60	13	6	6	1
4501-5000	20	44	3	1	-	2
5001-5500	20	43	10	-	10	-
5501-6000	20	43	18	3	15	-
6001-6500	20	32	9	4	4	1
6501-7000	20	30	9	4	3	2
7001-7500	20	30	9	1	7	1
7501-8000	20	37	11	2	9	-
8001-8500	20	59	12	3	4	5
8501-9000	20	50	12	4	8	-
9001-9045	20	2	2	-	2	-

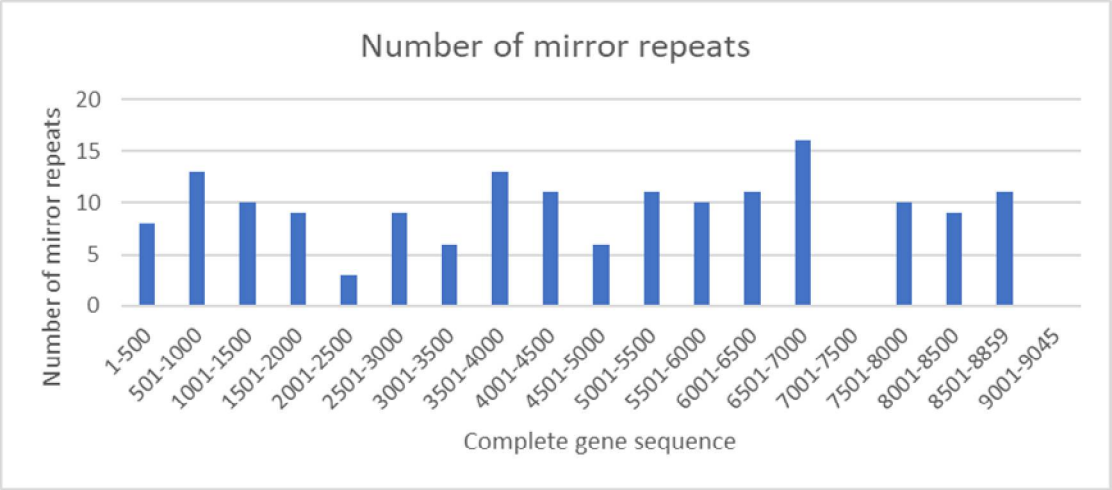


Figure 3. Distribution of MR's (mirror repeats) in Glucagon gene.

Exon Mirror Repeats in the Glucagon gene:

Further, a similar study was performed on the exonic region of the glucagon gene. 5 mirror repeats were found in all three exons of the gene as shown in table2. In exons, out of 5 mirror repeats 3 are perfect mirror repeats with one spacer (POS) and 2 are imperfect mirror repeats (IP). The largest mirror repeat within the exons of the gene is 13 bp (IP) while the smallest mirror repeat is 7bp (POS). We could not detect the small-sized mirror repeat of less than 7 as the minimum word size limit of the BLAST tool is 7 as shown in **Table 2.**

Table 2. Glucagon exon mirror repeats and their types at expected threshold 10.

Exon	Expected Threshold	No. of hits	No. of mirror repeats	Mirror repeats	Position of mirror repeats	Type
3030-3134	10	1	1	1. TGTTTGT	29-35	Perfect with one spacer
4886-5047	10	10	2	1. CCCAGACAGAACC 2. CACCAGTGACTAC	15-27 82-94	Imperfect Imperfect
7724-7867	10	4	2	1. CAAGAAG 2.CAGAGAC	8-14 100-106	Perfect with one spacer Perfect with one spacer

Complete gene mirror repeats in the PKLR gene:

A total of 167 mirror repeats were found in different regions of the pklr gene (Fig. 4). Out of 167, 131 mirror repeats were perfect with one spacer, 22 were perfect and 16 were imperfect repeats. A maximum number of hits 47 was observed in fragment 3501-4000 and the corresponding mirror repeats were 13 with 3 perfect, no imperfect and 10 perfect with one spacer type as shown in Table 3.

Table 3. Number of mirror repeats of PKLR gene and their type at expected threshold E-valve 20.

Complete gene sequence	Expected threshold	No. of Hits	No. of mirror repeats	Perfect mirror repeats	Perfect mirror repeats with one spacer	Imperfect mirror repeats
1-500	20	39	8	2	5	1
501-1000	20	32	13	1	11	2
1001-1500	20	20	10	1	8	1
1501-2000	20	33	9	1	7	1
2001-2500	20	29	3	-	2	1
2501-3000	20	27	9	3	6	-
3001-3500	20	25	6	1	4	1
3501-4000	20	47	13	3	10	-
4001-4500	20	33	11	1	10	-
4501-5000	20	32	6	-	3	3
5001-5500	20	36	11	1	11	1
5501-6000	20	27	10	-	8	2

6001-6500	20	38	11	2	8	1
6501-7000	20	34	16	2	13	1
7001-7500	20	-	-	-	-	-
7501-8000	20	28	10	-	9	1
8001-8500	20	37	9	1	8	-
8501-8859	20	29	11	3	8	-

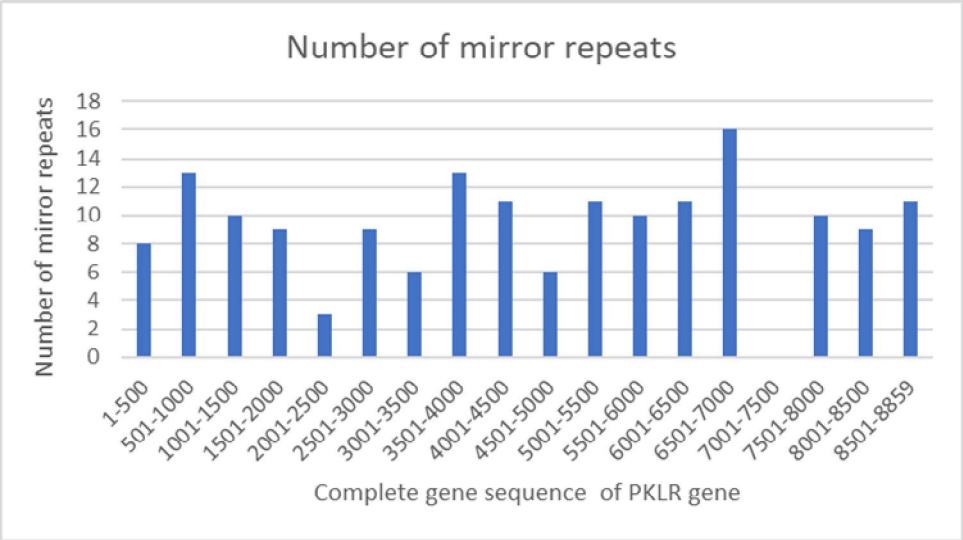


Figure 4. Distribution of MR's (mirror repeats) in PKLR gene.

Exon Mirror Repeats in the PKLR gene:

23 mirror repeats were found in all 9 exons of the gene. In exons, out of 23 mirror repeats 20 are perfect mirror repeats with one spacer (POS), 2 perfect mirror repeat (P) and 1 imperfect mirror repeat (IP). The largest mirror repeat within the exons of the gene is 29 bp (IP) while the smallest mirror repeat is 7bp (POS) as shown in Table 4.

Table 4. PKLR exon mirror repeats and their types at expected threshold 10.

Exon	Expected Threshold	No. of hits	No. of mirror repeats	Mirror repeats	Position of mirror repeats	Type
1092-1274	10	7	3	1. CCTTCTTCC 2. GACCCAG 3. TGCTCGT	61-69 41-47 152-158	Perfect with one spacer Perfect with one spacer Perfect with one spacer
3472-3603	10	5	1	1. TGAGAGT	39-45	Perfect with one spacer
3738-3924	10	7	3	1. AGGTGGA 2.GTGGGTA 3. CATCTAC	14-20 93-99 141-147	Perfect with one spacer Perfect with one spacer Perfect with one spacer
4241-4511	10	8	2	1. AGGTGGA 2. CAGTGAC	79-85 179-185	Perfect with one spacer Perfect with one spacer
4624-4774	10	1	1	1. GGCACGG	40-46	Perfect with one spacer
4987-5139	10	2	2	1. ATCACTA 2. ACCGCCA	16-22 106-112	Perfect with one spacer Perfect with one spacer
5233-5399	10	4	4	1. CTTCCTTC 2. ACCGCCA 3. GTCACTG 4. TCGTGCT	116- 123 32-38 91-97 146-152	Perfect Perfect with one spacer Perfect with one spacer Perfect with one spacer

6620-6801	10	8	6	1. TCTACCGTGAG-CCTCCAGAG-GCCATCT 2. GTCCACCTG 3. TTCCCCTT 4.TCTATCT 5. CCTGTCC 6. TGAAAGT	111-137 80-88 101-108 13-19 85-91 175-181	Imperfect Perfect with one spacer Perfect Perfect with one spacer Perfect with one spacer Perfect with one spacer
7478-8859	10	1	1	1. GTGGGTG	24-30	Perfect with one spacer

Complete gene mirror repeats in the Sox17 gene:
A total of 135 mirror repeats were found in different regions of the Sox17 gene (Fig. 5). Out of 135, 103mirror repeats were perfect with one spacer, 18were perfect and 16were imperfect repeats. A maximum number of hits 84 was observed in fragment 5001-5500 and the corresponding mirror repeats were 13 with 2 perfect,3 imperfect and 8 perfect with one spacer type as shown in Table 5.

Table 5. Number of mirror repeats of Sox17 gene and their type at expected threshold E-valve 20.

Complete gene sequence	Expected threshold	No. of Hits	No. of mirror repeats	Perfect mirror repeats	Perfect mirror repeats with one spacer	Imperfect mirror repeats
1-500	20	34	14	3	11	1
501-1000	20	43	12	2	9	1
1001-1500	20	53	8	-	8	-
1501-2000	20	37	13	2	10	1
2001-2500	20	42	15	3	12	-
2501-3000	20	52	12	-	11	1
3001-3500	20	55	23	3	16	5
3501-4000	20	48	14	1	10	3
4001-4500	20	30	4	2	1	1
4501-5000	20	36	7	-	7	-
5001-5500	20	84	13	2	8	3
5501-5515	20	-	-	-	-	-

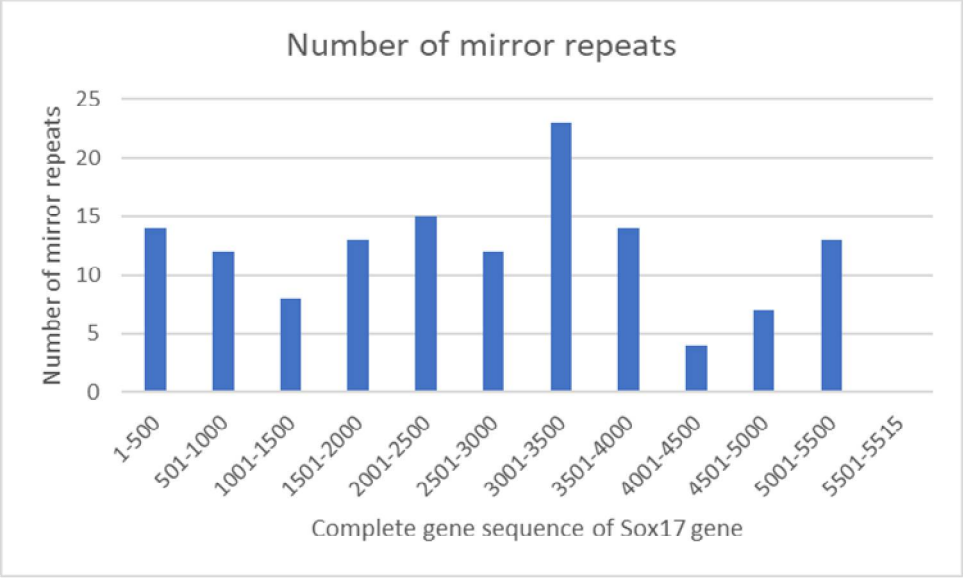


Figure 5. Distribution of MR's (mirror repeats) in Sox17 gene.

Exon Mirror Repeats in the Sox17 gene:
16 mirror repeats were found in all two exons of the gene. In exons, out of 16 mirror repeats 11 are perfect mirror repeats with one spacer (POS), 3 perfect mirror repeats (P) and 4 imperfect mirror repeats

(IP).The largest mirror repeat within the exons of the gene is20 bp (IP) while the smallest mirror repeat is 7 bp (POS) as shown in Table 6.

Table 6. Sox17 exon mirror repeats and their types at expected threshold 10:

Exon	Expected Threshold	No. of hits	No. of mirror repeats	Mirror repeats	Position of mirror repeats	Type
2968-3358	10	28	12	1.GTCCCTGGGCCGAGTCCCTG 2. CGAGTCCCTGAGC 3. CCGGGTTGGGCC 4.GGGCCCCTGTCCCTGGG 5.GAGAGGTGGTGGCGAG 6. CGAACGCAAGC 7. GCTCTCTCG 8. CCGGCGGCC 9. GCGGGGCG 10. CAGTGAC 11. GGAGAGG 12. GGTGTGG	89-108 99-111 74-85 81-97 137-152 243-253 110-118 204-212 155-162 27-33 136-142 228-234	Imperfect Perfect with one spacer Perfect Imperfect Imperfect Perfect with one spacer Perfect with one spacer Perfect with one spacer Perfect Perfect with one spacer Perfect with one spacer Perfect with one spacer
3796-5513	10	24	6	1. CGCGGCGGCGC 2. CCGGCCGGCC 3.GGAGGGAGG 4.CGTCTCGC 5.CCGGCGGCC 6. CTCCACCTC	109-119 258-267 146-154 164-172 429-437 721-729	Perfect with one spacer Perfect Perfect with one spacer Imperfect Perfect with one spacer Perfect with one spacer

Complete gene mirror repeats in the GAPDH gene:

A total of 116 mirror repeats were found in different regions of the glucagon gene (Fig. 6). Out of 116, 70mirror repeats were perfect with one spacer, 29 were perfect and 17 were imperfect repeats. A maximum number of hits 52 was observed in fragment 1501-2000 and the corresponding mirror repeats were 14 with 2 perfect, 2 imperfect and 10 perfect with one spacer type as shown in Table 7.

Table 7. Number of mirror repeats of GAPDH gene and their type at expected threshold E-valve 20.

Complete gene sequence	Expected threshold	No. of Hits	No. of mirror repeats	Perfect mirror repeats	Perfect mirror repeats with one spacer	Imperfect mirror repeats
1-500	20	46	9	3	6	-
501-1000	20	46	14	3	10	1
1001-1500	20	50	14	3	7	4
1501-2000	20	52	14	2	10	2
2001-2500	20	45	13	4	7	2
2501-3000	20	38	14	3	6	5
3001-3500	20	18	4	1	2	1
3501-4000	20	36	12	2	8	2
4001-4500	20	39	13	5	8	-
4501-4845	20	9	9	3	6	-

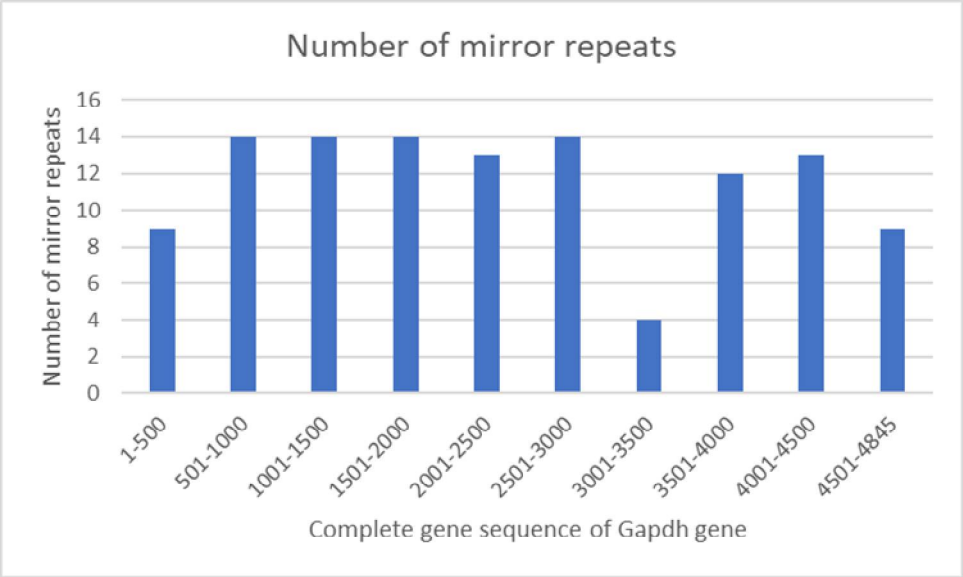


Figure 6. Distribution of MR's (mirror repeats) in GAPDH gene.

Exon Mirror Repeats in the GAPDH gene:

16 mirror repeats were found in all eight exons of the gene. In exons, out of 16 mirror repeats 10 are perfect mirror repeats with one spacer, 4 perfect mirror repeats without a spacer, and 2 are imperfect mirror repeats. The largest mirror repeat within the exons of the gene is 20 bp (IP) while the smallest mirror repeat is of 7bp (POS) as shown in Table 8.

Table 8.GAPDH exon mirror repeats and their types at expected threshold 10.

Exon	Expected Threshold	No. of hits	No. of mirror repeats	Mirror repeats	Position of mirror repeats	Type
3181-3387	10	9	3	1.CACGGCAAGTTCAACGGCAC 2. CAGTATGAC 3. TTCTCTT	128-147 113-121 38-44	Imperfect Perfect with one spacer Perfect with one spacer
3468-3558	10	2	2	1.GGTGCTGAGTATGTCGTGG 2.ACCACCA	35-53 71-77	Imperfect Perfect with one spacer
3655-3852	10	4	4	1.AACTCCCTCAA 2.GGGTGGG 3.TGTTTGT 4.ACCACCA	97-107 12-18 62-68 130-136	Perfect with one spacer Perfect with one spacer Perfect with one spacer Perfect with one spacer
3965-4195	10	7	3	1.TCAGAAGACT 2.GCCTTCCG 3.ACCCCCA	27-36 169-176 184-190	Perfect Perfect Perfect with one spacer
4285-4466	10	9	3	1.TTCCACCTT 2.GGACCAGG 3.AGTATGA	120-128 78-85 5-11	Perfect with one spacer Perfect Perfect with one spacer
4577-4845	10	1	1	1. GGTGGTGG	31-88	Perfect

DISCUSSION

In this study, we find that mirror repeats are frequently distributed in the *Rattus norvegicus*. Amongst these mirror repeats, perfect with one spacer mirror repeats were found more repeatedly in glucagon, pklr, sox17 and GAPDH and exon as compared to perfect and imperfect mirror repeats. A total of 199

mirror repeats were discovered in glucagon, 167 in the pyruvate kinase (pklr), 135 in the sox17 and 116 in GAPDH in complete gene sequence during this study, whereas 5 mirror repeats were discovered in glucagon, 23 in the pyruvate kinase (pklr),16 in the sox17 and 16 in GAPDH gene when just the exonic areas were taken into account.It was also concluded here that frequency and distribution of shorter mirror repeats is maximum.

CONCLUSION

This study applies the bioinformatics tool to identify mirror repeats in the glucagon, pklr, sox17 and GAPDH genes which are involved in the diabetic studies of Rattus norvegicus. Diabetic genes are analyzed using a computational-based tool, BLAST. This study will offer a forward-looking perspective on diabetic diagnostics and treatment, novel drug development, drug design, and pathogenesis processes. The creation of new bioinformatics tools or software for the detection of Mirror Repeats (MRs) will also benefit from this study. Future studies may be required to determine the exact role of mirror repeats within the diabetic genes of *Rattus norvegicus* and other organism genomes.

Financial support
This research received no external funding.

Author Contribution Statement
Parveen Kumar: Conceptualization, Methodology, Software, Supervisor, Reviewing, Editing.
Babli: Data curation, Investigation, Writing-Original draft preparation.
Kavita Saini: Writing-Review Editing.

Conflict of interest
The authors do not have any conflict of interest.

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