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The Correlation between KCNJ11 rs5215 Gene Polymorphism and Type 2 Diabetes Mellitus in Northern Population

Shubhangi Chaturvedi[1]*, Shivangi Singh[1], Pooja[1] and Dinesh Raj Modi[2]
[1]Ph.D. Scholar, Department of Biotechnology, School of Life-Sciences,
Babasaheb Bhimrao Ambedkar University, Vidya Vihar, Raibareli Road, Lucknow (Uttar Pradesh), India.
[2]Professor, Department of Biotechnology, School of Life-Sciences,
Babasaheb Bhimrao Ambedkar University, Vidya Vihar, Raibareli Road, Lucknow (Uttar Pradesh), India.

(Corresponding author: Shubhangi Chaturvedi)*
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ABSTRACT: Type 2 diabetes mellitus (T2DM) is known as a multifactorial disease that develops as a result of the interplay between various susceptibility genes and environmental components. KCNJ11 gene codes for the voltage-gated potassium channels that are responsible for glucose-stimulating insulin release. The major challenge faced during the study is the collection of samples from patients and the greater number of samples has to be processed to make the significance of this study more fruitful. The Aim of this study is to find the association between KCNJ11 gene rs5215 polymorphism and T2DM in the North Indian Population. This is a case- control study. The KCNJ11 associated rs5215 polymorphism has been genotyped using Restriction Fragment Length Polymorphism (RFLP) technique. The incidence of K allele was founded more in the group of patient, and the prevalence of EK genotype was founded more in the control group of individuals. The recessive model genotype TT was founded to be remarkably associated with the occurrence of T2DM. This study conveyed the association of KCNJ11 gene polymorphism rs5215 as a possible reason to cause T2DM.

Keywords: KCNJ11, RFLP, Polymorphism, Type 2 Diabetes mellitus, Potassium channel, North India.

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is considered worldwide as a long-standing medical issue that has many causes such as genetic factors, environmental components, unsuccessful or decreased insulin release from beta cells of the pancreas, and surrounding resistance of insulin in the body along with hyperglycemic condition (Abed *et al.,* 2013; Aka *et al.,* 2021). The major environmental component that causes the incidence of T2DM is smoking, increased cholesterol level, less physical activity, being overweight, and age (Hussieny and Alsahlawi 2021; Mutawa, 2023). Recent Study conducted in Saudi Arabia has confirmed role of KCNJ11 gene polymorphism in T2DM, KCNJ11 rs5210 is correspond with decreased depolarization- stimulated insulin exocytosis (Alqadri 2022). Usually, there are four kinds of diabetes mellitus known but T2DM is the one that occurs in a higher percentage of individuals (Muhammad and Agussalim 2018). As per different Genome-wide association studies (GWAS), various genes are known to play role in the occurrence of T2DM and those genes are KCNJ11, TCF7L2, PPAR-G, HNF1A, KCNQ1, ABCC8, CDKAL1, FTO, NOTCH2, HNF1A, IGF2BP2, and PRC1. Mutation in the KCNJ11 gene is known to cause neonatal diabetes and congenital hyperinsulinemia. As per different studies, the cases of diabetes will be increased by 2035 by a percentage of 10% (Mutawa-Al, 2023). Worldwide the percentage of diabetes cases will be increased by 2045 and will be expected to reach 693 million, since the past twenty years cases of diabetes have increased drastically in 2017 the number of cases was 451 million that will become double in the coming years as per international diabetes federation (IDF) reports. As per the analysis of The Center for Disease Control and Prevention (CDC) which is located in the United States of America 30.5 million people in the USA which is about 9.4% of the total population were diabetic in the year 2015 (Aswathi *et al.,* 2020). T2DM causes several comorbidities in the individual that are associated with the eyes, heart, and kidneys and that's why T2DM is considered a severe burden on public health (Čejková *et al.,* 2007). In many of the T2DM patient cases, diagnosis occurs usually after 6-7 years of disease and during that period several macro and micro lesions arise in the body of 25% of patients (Chistiakov *et al.,* 2009). The major action mechanism that various diabetes-causing genes do in T2DM-diagnosed individuals is still not well understood. Several gene polymorphisms that are SNPs are known to associate with T2DM that has been studied in several population and ethnic groups and every time this polymorphism shows different involvement in the occurrence of T2DM. Hence it is very important to study and find out the biomarker involve in causing the incidence of T2DM. It is analyzed in different studies that compared to the European population Indians are more likely susceptible to T2DM (Engwa *et al.,* 2018). According to research-based studies, T2DM is the cause of death in America at position seven (Gong *et al.,* 2012).

Genetic factors play role in the arise of T2DM because polymorphism in susceptible genes such as KCNJ11-associated rs5215 alters the release of insulin through beta cells of the pancreas. Polymorphism in KCNJ11 gene swaps the shape of the Kir 6.2 protein in the beta cells which ultimately hinders the secretion of insulin in the body (Hansen *et al.*, 2005). The universality of T2DM is more in the Western region the prevalence of diabetes varies globally and it depends on many factors such as eating habits, demographic differences, and social and economic behavior variations (Isakova *et al.*, 2019). Usually, there are two kinds of diabetes one is type 1 diabetes mellitus which is also known as insulin-dependent diabetes mellitus (IDDM) and the second one is non-insulin-dependent diabetes mellitus (NIDDM) which is type 2 diabetes mellitus (Khan *et al.*, 2019). As it is confirmed after many studies done extensively that T2DM is the result of polymorphism in major genes. The gene that is an important candidate in T2DM-associated polymorphism studies is that gene that plays a major role in the release of insulin in response to elevated glucose levels. The targeted gene is the Potassium Voltage-Gated Channel Subfamily J Member 11gene (KCNJ11). This KCNJ11 gene codes for the voltage-gated potassium channel called as KATP channel and this KATP channel has two subunits that are an inner subunit that is a rectifier of the potassium channel known as Kir6.2 and the other part of the subunit called a subunit of sulfonylurea receptor (SUR) (Khan *et al.*, 2020; Makhzoom *et al.*, 2019). KCNJ11 gene has one exon and its location is at 11p15.1, KATP channel in the pancreatic beta cells helps in the release of insulin by linking the metabolic state of the cell and membrane potential. The level of glucose in the bloodstream alters the activity of the KATP channel. A higher level of glucose lowers the activity of the KATP channel which causes the influx of calcium ions and lastly leads to insulin secretion (Aswathi *et al.*, 2020; Liu *et al.*, 2013). T2DM is known as a heterogeneous disease that turns out because of the E23K polymorphism of the KCNJ11 gene which increases the "open prospect" of the kir6.2 channel that conducts drop off in the insulin secretion process (Muftin and Jubair 2019; Sethi *et al.*, 2015). The pancreatic beta cell KATP channel is made by the SUR1 and Kir6.2 protein, it is experimentally confirmed that mice that don't have the kir6.2 gene have diminished glucose and tolbutamide stimulated insulin release (Rastegari *et al.*, 2015). The KCNJ11-associated rs5215 SNP is caused due to transition of (C) to (T) KATP channel of KCNJ11 gene functions in the maintenance of pancreatic beta cell membrane (Hussieny and Alsahlawi 2021). Mutation in the KCNJ11 gene that results in the causing heterozygous condition is responsible for the occurrence of permanent neonatal diabetes (PNDM), adult onset of diabetes, and Maturity-onset diabetes of the young (MODY) (Sethi *et al.*, 2015).

MATERIAL AND METHOD

Population Study. This work has been done in the Biotechnology department of Babasaheb Bhimrao Ambedkar University in Lucknow. This study is a case-control-based investigation, this study included 120 individuals of the North Indian Population specifically from Lucknow city in Uttar Pradesh and all are above the age of 42 years. The patient category has 60 total T2DM cases samples that are confirmed diabetic according to O.P Chaudhary Hospital from where diabetic patient samples have been collected and have the serum plasma glucose level in a fasting condition more than 100mg/dl and random glucose value in the cases is more than 200mg/dl. The control classification includes 60 individuals having fasting glucose levels less than 100mg/dl and having no family history of T2DM in lineage. The research work has been conducted since year 2019 to present including almost every age group of samples and have covered all parameters to conduct a study with significant result.

Inclusion and Exclusion Standard. In those cases, samples have been avoided to taken from a person who has any comorbidity associated such as Liver linked to disease, type 1 diabetes, heart-related issue, and severe gastrointestinal disease also patients having hyperglycemic levels is avoided because abnormal glucose ratio can be due to any infection after surgeries affect and thyroid can also be a reason for that. This case–control investigation has got permission from the ethical committee of O.P Chaudhary Hospital and Research Center, Lucknow and the signed consent letter for sample collection has been taken from all the patients. The study has been for about four years.

Sample Collection. A blood sample of about 5 ml is collected 3 ml in the two separate EDTA vials and 2 ml in fluoride vials using this set of the vial glucose level of the individual's sample can be quantified. On the other hand sample in one EDTA vial will be used in measuring the hemoglobin A1c and the other EDTA vial sample has been used for the isolation of DNA and to investigate genetic variation in the diabetic patient's sample.

Genotyping Genetic Variation. Isolation of DNA is done by the manual method which is using the phenol-chloroform method and the isolated DNA yield has been measured using a spectrophotometer or by running the isolated DNA sample on 1% agarose gel using electrophoresis. Isolated DNA to be used for polymerase Chain reaction (PCR) and RFLP are stored at − 20°C to prevent degradation and for long-term storage. SNPs correlated with the KCNJ11 gene that is rs5215 will be genotype using RFLP and PCR techniques.

KCNJ11 gene-rs5215 will be amplified using (GeNei India) PCR Mastermix and subsequent set of Primer set:

Forward: (5'- GAGACCATGGCTCAGGACAG- 3')
Reverse: (5'- TGTGCCCATTGTAGCTGAGG -3')

The PCR was done using the following set of background: Firstly Initial denaturation at 95 °C for 5 min, carried by 35 cycles of (a) 95 °C for 30 s (denaturation), (b) 61 °C for 30 s (annealing), (c) 72°C for 30 s (elongation) and final elongation done at 72 °C for 10 min. The KCNJ11 gene amplicon size is (240 bp) and the amplified PCR product will be digested using the restriction endonuclease Ban II (NEB, India)

at the temperature of 37 °C for 1 hour. The reaction mixture for Restriction Digestion is 10 µl of PCR product, 3 µl 10X NEB buffer, 0.2 µlBanII enzyme, and 1.8 µl autoclaved Milli-Q water. The digested product from the Ban II enzyme was run on 3% agarose gel and separated bands will be seen using the UV transilluminator.

Statistical Analysis. For statistical analysis, the statistical application SPSS 16 was employed. The genotype distributions were examined for Hardy-Weinberg equilibrium using the chi-square test. Using the chi-square test, the frequency of the genotypes and alleles was compared between the two groups. Multiple Regression Analyses has done using SPSS Software, One-Sample Kolmogorov- Smirnov Normal Test was done and different relationship-associated graphs have been generated using SPSS Software. Regression Standardized Residual has been shown through Histogram, with the aid of a logistic regression model, the odds ratios were determined. Statistical significance was defined as a P-value of 0.05 or lower.

RESULTS AND DISCUSSION

The biochemical readings from both case and control samples have been evaluated for better analysis of the study. All the biological and scientific readings of the patient and healthy individuals are listed in (Table 1).

Table 1: Comparison of biological parameters of both patient and healthy individuals.

Variables	Patients (Number = 60)	Controls (Number = 60)	*P-value*
Fasting Glucose	109.82± 6.74	85.4 ± 8.58	0.300
Postprandial Glucose	225.37 ± 51.7	116.80 ± 9.48	0.328
Very Low density Lipoprotein	21.88 ± 14.14	15.05 ± 8.35	0.284
High Density Lipoprotein	52.78 ± 11.76	51.78 ± 5.29	0.240
Age	43.1 ± 14.05	34.2 ± 6.7	0.499

Noteworthy variation has been seen in the value of hemoglobin A1c, fasting glucose level and body mass index (BMI). No major changes have been reported on the basis of age and sex. But an association between Fasting Blood Sugar and Age has been observed in case of T2DM patients and on other hand in case of controls no such relationship has been observed that is explained using graph that is a relationship graph (Fig. 5).

The major difference between the Postprandial Sugar (PPS) of case and control has been illustrated using the graphs, Comparing these graphs one can analyze the difference in level of PPS in both groups easily (Fig.1 and 2).

Regression Standardized Residual. In Fig. 1 variances in the level of glucose in healthy individuals has been shown whom are Controls of the study, in all the controls PPS is under control that is under 180 mg/dl.

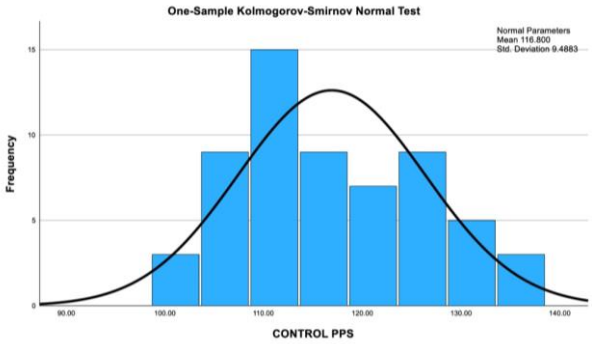

Fig. 1.

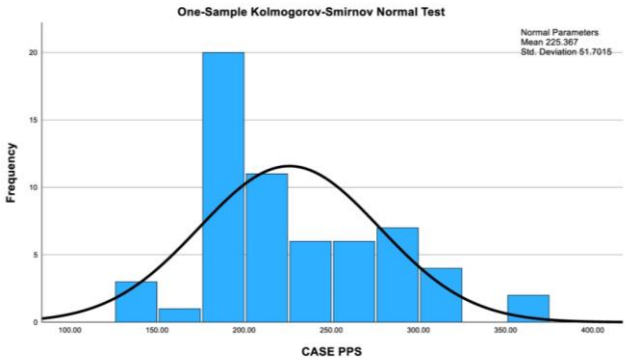

Fig. 2. Fluctuations in level of Sugar after two hours of eating in cases or patients have been shown in this Graph in case of patients elevations have been seen specifically at glucose level of 200mg/dl.

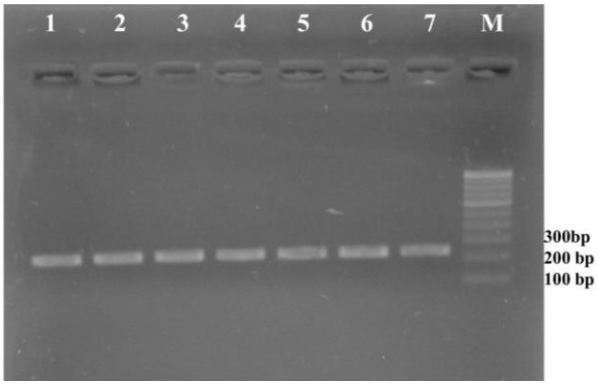

Fig. 3. The PCR amplification of KCNJ11-rs5215 gives a product of (240 bp), PCR amplified products run on 2% Agarose gel. Lane M has 100 bp loaded in it and rest lanes as 1,2,3,4,5,6,7 have PCR product loaded (240 bp).

When PCR reaction done, Amplicon of 240 bp was obtained (Fig. 3).

When restriction digestion was done using the Ban II enzyme on the amplified PCR product and it gives different size of bands:

Wild type homozygote will give CC gives one band of size 240 bp.

Mutant homozygote TT gives two bands of 156 and 84 bp size.

Heterozygote CT will give three bands of size 240, 156 and 84 bp, (Fig. 4).

The reoccurrence of the genotypes in patients and control group gives different result for patient's category (CC-CT-TT) genotypes were coming as (35% - 50% - 15%), and the control group's results are (17% – 51% - 3.3%), each. The variation that comes between the two groups is found to be statistically significant (P-value = 0.03). All individuals' genotypic frequencies have been come according to Hardy-Weinberg equilibrium.

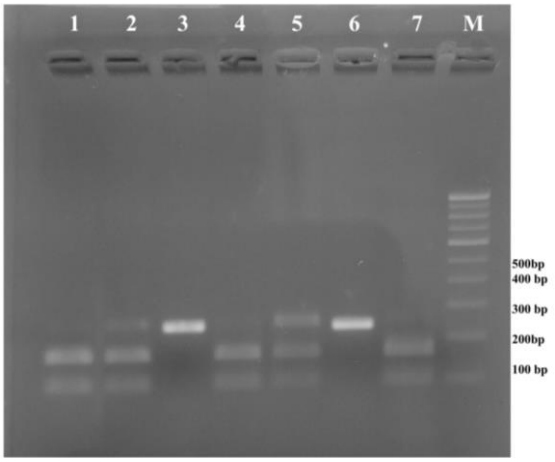

Fig. 4. In this figure restriction digestion product of KCNJ11-rs5215 has been loaded. In lane 2 and 5 heterozygote CT is loaded: Lane 1,4 and 7 mutant homozygote TT is loaded: In Lane 3 and 6 wild homozygote CC is laoded.

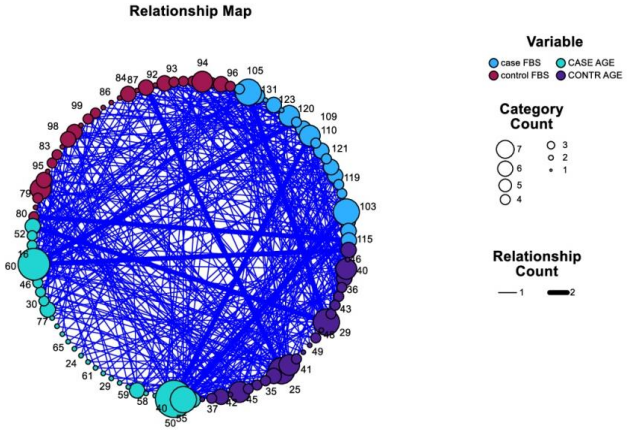

Fig. 5. In case of Diabetic patients or cases Fasting Blood Sugar (FBS) has seen elevated at the age of between 50-60 Years, and that elevations are much higher compared to healthy individual or Control FBS. While in case of healthy ones no such relationship or elevation at particular age is observed.

(C – T) allele prevalence in the patient group has come (63.3 – 37%), and in the control group frequency was (83.3 – 17%). Analysis of variation in both the types shows that the difference was statistically insignificant, (P – value = 0.0132).

To assess the impact of the KCNJ11- rs5215 polymorphism on T2DM, four models were examined. For each model, the odds ratio and P-value were determined. (Table 2).

Table 2: Genetic traits of patients and controls groups.

Variables	Patients = 60	Control = 60	OR (95% CI)	*P- value*
Genotypes				
C/C	21 (35%)	10 (17%)	Reference	
C/T	30 (50%)	38 (51%)	0.1754 (0.03658 – 0.7857)	0.0252
T/T	9 (15%)	2 (3.3%)	0.5392 (0.3796 – 0.8906)	0.103
Dominant model				
C/C	10 (17%)	20 (33.3%)	Reference	
C/T + T/T	50 (83.3%)	40 (67%)	0.4000 (0.1662 – 0.9153)	0.0350
Recessive model				
C/T + T/T	55 (92%)	45 (75%)	Reference	
T/T	5 (8.3 %)	15 (25%)	3.667 (1.272 – 9.648)	0.0143
Alleles				
C	38 (63.3%)	50 (83.3%)	Reference	
T	22 (37%)	10 (17%)	0.3455 (0.1451 – 0.8433)	0.0132

One of the most common non-fatal diseases in the world, T2DM is a challenging health issue. It is thought that there are a variety of different hereditary variables that contribute to type 2 diabetes mellitus. Investigating these genetic variables that are linked to the risk of T2DM is therefore necessary. A Different meta-analysis conducted that shows the correlation of KCNJ11 gene polymorphism with arise of T2DM which makes this association and its result more comprehensive (Moazzam-Jazi *et al.*, 2022). T2DM is a complicated illness that is influenced by several environmental and genetic variables, as well as the effects of a single genetic component on Individuals who do not develop T2DM in the same ways because of differences in their environments. The large importance (four times) in our study in a sample of the northern population as opposed to other groups may be explained by the possibility that these potent environmental factors may magnify the influence of the genetic factor in the risk allele carriers. Such as we did not include people in the control group who had a genealogy of T2DM, which may have decreased the likelihood that the controls would have SNPs contributing concerns for T2DM, such as the KCNJ11-rs5215 genotype, and hence might cause bias in the selection of people. This may also be the reason for our study's risky value. This is demonstrated by (Keshavarz *et al.*, 2014) who examined a population in Iran and found no correlation between the rs5215 polymorphism of the KCNJ11 gene and type 2 diabetes. Nevertheless, an association was seen when the results were restricted to obese people. This might attest to the genetic factor's growing influence in the context of potent environmental factors. In this study, no relevant significant difference has been shown between VLDL and HDL in case and control. The relationship between KCNJ11 polymorphic changes has not been studied before in the northern population, but this study has confirmed the association between KCNJ11 rs5215 and the incidence of T2DM. Various investigations were done which reveal the fact that Indian ethnicity have increased body fat and higher adiposity at a particular BMI level in comparison with another ethnic group this makes Indian more susceptible to condition of insulin resistance and Dyslipidemia (Shitomi-Jones *et al.*, 2023).

CONCLUSIONS

The research found a link between type 2 diabetes mellitus and the KCNJ11 gene's rs5215 polymorphism in a sample of the North Indian Population. This research bolsters the pathophysiology of T2DM's KCNJ11 rs5215 polymorphism. During the work of this study collection of samples from Diabetic patients has been a major challenge, but we counsel patients about the study and then take their consent to take blood samples. The important contribution has been made by us is to increase the sample size which increased the relevance of this study. Bigger investigations need to be conducted to verify this finding.

FUTURE SCOPE

This kind of study is very fruitful as medical purpose as these kind of polymorphisms work as a biomarker. These kind of studies can provide more significant results if conducted over larger sample size, and such case control study on major Diabetes causing genes associated polymorphism will also helps in the field of personalized medicine.

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Conflict of Interest. None.

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