iJRASET

International Journal For Research in
Applied Science and Engineering Technology

INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 9 Issue: III Month of publication: March 2021

DOI: <https://doi.org/10.22214/ijraset.2021.33330>

www.ijraset.com

Call: 08813907089

E-mail ID: ijraset@gmail.com

Association of Genetic Variants in the Promoter Region of APOC3 with the Risk of NAFLD in Northern India Population: A Case Control Study

Rehana Ahmed¹, Mahrukh Hamed², Nebela Khan³, Syed Mushfiq⁴, Zubaida Rasool⁵, G. N Yattoo⁶, Sourab Singh⁷

^{1,2,3}Department of Human Genetics Sher-I-Kashmir Institute of Medical Sciences, Srinagar, Jammu and Kashmir, India.

⁴Department of Gastroenterology Sher-I-Kashmir Institute of Medical Sciences, Srinagar, Jammu and Kashmir,

⁵Department of pathology Sher-I-Kashmir Institute of Medical Sciences, Srinagar, Jammu and Kashmir,

⁶Department of Gastroenterology, Sher-I-Kashmir Institute of Medical Sciences, Srinagar, Jammu and Kashmir

⁷Department of Biotechnology Jaipur National University, Jaipur

Abstract: Non-Alcoholic Fatty Liver Disease (NAFLD) is a clinical condition defined by excessive fat accumulation in the liver in the form of triglycerides involving more than 5% of hepatocytes. The association between the APOC3 gene T-455C-(rs2854116) C-482T- (rs2854117) variant and NAFLD has been demonstrated in a number of studies, confirming that this gene is an important genetic determinant of disease development. The present study was carried out with an aim to evaluate the association of demographic features, liver enzymes, serum lipid profile and APOC3 gene variants (C-482T and T-455C) **Method:** A case-control study was designed with 150 patients with NAFLD, and 180 healthy controls. The mutations were analyzed using PCR-RFLP approach, Case-control studies assessing the relationship between APOC3 rs2854116 C/C and rs 2854117 C/T gene polymorphism with non-alcoholic fatty liver disease. **Results:** Statistically significant increased frequencies of CC+CT genotype [OR, 2.0; 95% CI (1.18-3.40); $P < 0.0006$] [OR, 2.0; 95% CI (1.38-2.91); $P < 0.0002$] and the C allele is seen in NAFLD patients compared to healthy controls. In conclusion we found that the APOC3 gene and elevated triglyceride levels in -455C/C SNP is significantly associated with the risk of NAFLD whereas the Apoc3-482C/T showed no association with NAFLD.

Keywords: Non Alcoholic fatty liver disease; Apolipoprotein; Aminotransferase; Alanine transaminase; high- density lipoprotein; Low-density lipoprotein.

I. INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD), including nonalcoholic steatohepatitis (NASH) and fibrosis, is a chronic liver disease related to hepatic insulin resistance metabolic syndrome and diabetes (Petersen KF et al 2010). Depending on the demographic area the prevalence of NAFLD differs with race and ethnicity (25.24%) (15% to 40%)- (Petersen KF et al., 2010). Currently, the population prevalence in Asia is around 25% (Jun Wang,et al., 2020). Recent advances in the molecular biology has attracted extensive attention related to polymorphisms of the NAFLD-related genes involving lipid handling, insulin signaling, and oxidative stress (Aggarwal R et al., 2012) Apolipoprotein (APOC3), a component of triglyceride (TG)-rich lipoproteins and high-density lipoprotein (HDL) is mainly synthesized in the liver and to some extent in the intestine(Karathanasis SK 1985) . APOC3 gene, mapped to chromosome 11q23, closely linked to APOA1 and APOA4 genes and is mainly involved in transport and clearance of chylomicron remnants, very-low-density lipoprotein (VLDL) and HDL from the bloodstream. A number of studies have found that polymorphisms of APOC3 are associated with the susceptibility of NAFLD among non-Asian Indian men(Petersen KF et al., 2010). APOC3 encodes a 79-amino-acid glycoprotein that is produced mainly in the liver, where it inhibits the action of lipoprotein lipase and interferes with receptor-mediated lipoprotein uptake. The presence of polymorphisms at the insulin responsive element on the promoter region leads to over-expression of the protein (Masebe T, et a.,l 2014, Groenendijk, M et al., 1999) (7,5) The APOC3 -482(rs2854116) and -455(rs2854117) promoter polymorphisms have reduced affinity for nuclear transcription factors mediating insulin responses associated with insulin resistance. The cysteine to thymine change at position -482 is associated with dyslipidemia and insulin resistance (Tarr PE et al., 2005, Green, M.L 2002) A change from thymine to cysteine at position -455 is associated with increased triglyceride levels, a risk factor for cardiovascular disease. Four common SNPs in the APOC3 gene, rs2854116, rs2854117, rs4520 and rs5128 have been identified as putative functional SNPs that influence serum APOC3 concentrations resulting in dyslipidemia by influencing triglyceride (TG) and very-low-density lipoprotein (VLDL) levels (Jiang Ye et al., 2015, Song Y et al., 2015). Many genetic studies have shown that the (SNPs) in the APOC3 gene may have implications for hypertriglyceridemia. (Talmud PJ 1997).

II. MATERIALS AND METHODS

A. Subjects

For isolation of genomic DNA 3 milliliter of peripheral blood from each case and control subject was collected in EDTA vials. DNA was extracted by using a standard phenol/chloroform method. Genetic variants rs2854116 (T/C) and rs2854117 (T/T) polymorphisms in promotor region of APOC3 gene were detected by Polymerase chain reaction (PCR) followed by Restriction fragment length polymorphism (RFLP) analysis (Michael Y, et al.2005). Total volume of Polymerase chain reaction (PCR) was 25 μ l containing 50 ng genomic DNA, 1 $\times$ PCR buffer (Biotools, B & M Labs, S.A. Madrid-Spain) with 2mM MgCl₂, 0.20mM dNTPs (Biotools, B & M Labs, S.A. Madrid-Spain), 0.4 μ mol of each primer (Sigma-Aldrich Co. LLC·USA), and 1 U DNA polymerase (Biotools, B & M Labs, S.A. Madrid-Spain). PCR conditions were: initial denaturation step at 95 °C for 4 min, and 35 cycles each of denaturation for (95 °C for 30s), annealing (58°C for 30s) and extension (72 °C for 30s), followed by a final elongation at 72 °C for 7 min. The primers used for amplification of APOC3-455C/T and APOC3-482T A/G are given in Table 1. For RFLP analysis, 10 μ l of PCR products were digested with the enzymes MspI (-482C/T) and FokI (-455A/G) (Fermentas Thermo Fisher Scientific Inc. Massachusetts, USA) (1 U at 37°C for 16 h), respectively. The restriction digested products and the PCR products (Table 1), were visualized through 3% agarose gel.

B. Statistical analysis

A case-control association analysis was performed in the control and NAFLD' disease patient, groups were compared using the χ^2 test. When the assumption of the χ^2 test was violated (i.e., when one cell had an expected count of < 1, or > 20% of the cells had an expected count of < 5), Fisher's exact test was used. Odds ratios (ORs) with 95% confidence intervals (CIs) were determined for the disease susceptibility of specific genotypes and alleles. Results were considered statistically significant when the probability of findings occurring by chance was < 5% ($P < 0.05$). The statistical analysis was done with SPSS v 20 and online software via <http://vassarstats.net>.

III. RESULTS

In the present study, a total of 150 NAFLD cases and 180 healthy controls were taken. Selected demographic characteristics of the patient and control groups are demonstrated in supplementary table (1). There was no significant difference in the gender status or age between patients and control groups. In the study, the NAFLD group consisted of 86 men and 64 women, while the control group had 129 men and 51 women.

The genotypic distributions of APOC3 -455C/T $P=0.84$ and APOC3-482A/G $P=0.86$ polymorphisms in control population were in the Hardy-Weinberg equilibrium. The amplified 196 bp PCR product was digested with MspI enzyme to genotype polymorphism C-482T, and Fok I enzyme for SNP, T-455C by incubating at 37 °C for 5 min followed by separation of fragments on 3 % polyacrylamide gel. Genotypes of -455 T/C (rs rs2854116) and 482c(rs2854117) were defined by PCR-RFLP on basis of the presence of different patterns of bands.

The digestion with MspI enzyme for C-482C/T (rs2854117) indicated by complete digestion into two bands of C/C 143 and 53 bp whereas, "T" allele, shows two bands of T/T 159 and 37 bp. Heterozygous -482C/T genotype was detected by four bands of 159, 143, 53 and 37 bp. The statistical analysis indicated the genotypic and allelic frequencies of APOC3-482 T/C polymorphism in cases and controls were not significantly different [OR, 0.70; 95% CI (0.42-1.17); $P=0.2$] and [OR, 0.70; 95% CI (0.49-1.01); $P=0.05$] respectively (Table 3).

The association between APOC3-482C (rs2854116) C/T polymorphism and clinicopathological parameters didn't show any significant difference in the results (Table 4). Restriction digestion with FokI enzyme showed no digestion for T-455C(rs2854117) allele and PCR amplicon T/T (196 bp), was left undigested. PCR product was digested into two bands of C/C 133 and 63 bp for -455T allele and for heterozygous and it results to three bands of T/C 196, 133, and 63 bp. In case of APOC3-455T/C the distribution of the genotypic and allelic frequency in cases and controls is given in Table 3.

Among the patients with NAFLD frequency of APOC3 -455T C/C: TC, CC and TT genotypes was 90(60%), 60(40%) and while in healthy controls it was 103 (57.22%), 77(42.77%) respectively. The statistical analysis indicated the genotypic and allelic frequencies of APOC3-455 C/C polymorphism in cases and controls to be significantly different [OR, 2.00; 95% CI (1.18-3.40); $P < 0.0006$] [OR, 2.00; 9.5% CI (1.38-2.91); $P < 0.0002$] (Table 3), indicating that the carriers of the C allele have a strong risk for the NAFLD predisposition. Furthermore, the association between APOC3 -455 T/C polymorphism and clinicopathological parameters was also carefully analysed (Table 4). The level of serum triglyceride is the only parameter that was significantly associated ($P < 0.001$) with the said polymorphism.

IV. DISCUSSION

In the present study, we observed the effects of two APOC3 polymorphisms (rs2854116 and rs2854117) on the risk of NAFLD. To the best of our knowledge, this is the first study from Kashmir evaluating the association of APOC3 gene variants with NAFLD. NAFLD is one of the most common liver disorders (Chalasani N et al., 2018, Younossi ZM. 2015). Although the pathogenesis of NAFLD remains largely unknown, insulin resistance, oxidative stress and inflammation play important role in the development and progression of NAFLD (Obika M 2012). Many candidate genes are reported to be associated with NAFLD risk such as TNF- α , PNPLA3, APOC3, PPAR- γ and adiponectin (Di Rosa M et al., 2012, Yu S, et al., 2013). In the present study, two SNPs of APOC3 gene which are associated with lipid metabolism were studied.

We found a strong association of [455-C (rs 2854116)] with NAFLD whereas no association of other SNP C-482 (rs 2854117) with the risk of NAFLD was observed. In addition, there was an increase in the triglyceride levels in NAFLD patients. Two polymorphisms associated with APOC3 gene T-455C and C-482T, are located in the 5' promoter region and are in strong linkage disequilibrium with each other.

A study by Petersen et al. reported that the polymorphisms C-482T and T-455C in APOC3 are associated with nonalcoholic fatty liver disease and insulin resistance (Petersen et al., 2010). APOC3 polymorphisms have been stated to be associated with many diseases such as metabolic syndrome (Povel CM et al., 2011), type 2 diabetes (Duijn CM, et al., 2011), coronary heart disease (Li XM, et al., 2016) and plasma APOC3 and lipid levels (Yang Y et al., 2015).

This polymorphism leads to a roughly 30% higher plasma concentration of APOC3 and postprandial hypertriglyceridemia and as a result of which individuals with carrier of those polymorphisms T-455C(rs2854116) can take up increased amounts of lipid from the chylomicron remnant leading to NAFLD and hepatic insulin resistance (Kotronen A, et al., 2012.). In the present study, we found a higher prevalence of triglycerides among males in comparison with females.

This finding is consistent with a previous study where male patients had significantly higher rates of NAFLD as well as NASH (Williams CD et al., 2011). Lipid metabolism in the liver comprises four major pathways including fatty acid uptake, de novo fatty acid synthesis, oxidation of fatty acids and secretion of VLDL. Changes in any of these pathways can disturb the vigorous balance of the homeostasis of lipids in liver cells, causing abnormal lipid depletion or accumulation. Many studies using high-throughput microarray technique verified by qRT-PCR assays were carried out to explain lipid metabolism pathways in the liver (Zhu L et al., 2011).

A recent study with non-obese patients carrying apolipoprotein C3 gene variants (C-482T, T-455C or both) showed elevated activity of the LDL receptor in steatosis (Petersen KF et al., 2010). In the present study, there was no significant difference in the total cholesterol, HDL and LDL levels between patients and controls.

However in 455C (rs 2854116) triglyceride level were found to be elevated significantly in patients in comparison with the controls which is in accordance with previous reports where triglyceride level was found to be significantly high in NAFLD patients (Petersen KF et al., 2010) (Bianchi G, et al., 2001) showed that 80 % of patients with NAFLD were obese. In a study carried out by (Deepa et al. Chawla M, et al., 2009) only 21 % of the population was found to have normal BMI while the remaining 79 % were either overweight or obese. (Golander et al., 2006) reported that patients with NAFLD had a significantly higher BMI.

However, in the present study, we did not find an association between BMI and NAFLD. Subsequent studies in Hispanic, European American, African American and European subjects have turned futile to confirm the association of APOC3 variants with NAFLD (Al-Serri A, et al., 2011, Kotronen A, et al., 2012) reported a similar lack of association between the two APOC3 gene polymorphisms and NAFLD in a Finnish population. In their study, two SNPs of the APOC3 gene were genotyped and they also measured the liver fat using magnetic resonance spectroscopy and plasma concentration of APOC3, where they concluded that the SNP C-455 (rs2854116) of APOC3 is associated with the risk of NAFLD.

Our results are in concordance with results from the study by (Kumar A, et al., 2014) where the CC+CT genotype was found to be associated with elevated triglyceride levels and the patients had an increased risk of NAFLD. In conclusion, the present study showed vital association of genotype -455C/C of SNP, rs 2854116 in APOC3 gene with NAFLD, and increased serum triglyceride levels were found to be strongly associated with the disease.

V. CONCLUSION

In assumption, the present study revealed a significant association of genotype -455C/C of SNP, rs2854116 in APOC3 gene with NAFLD. An increased serum triglyceride levels were also found to be strongly associated with NAFLD. However, the present study identified a strong genotype -455 C/C)- phenotype (elevated serum triglycerides) correlation in cases with NAFLD.

REFERENCES

- [1] Petersen KF, Dufour S, Hariri A, Nelson-Williams C, Foo JN, Zhang XM, Dziura J, Lifton RP and Shulman GI. Apolipoprotein C3 gene variants in nonalcoholic fatty liver disease. *N Engl J Med* 2010; 362: 1082-1089.
- [2] Association between APOC3 polymorphisms and non-alcoholic fatty liver disease risk: a meta-analysis Jun Wang¹, Chuncui Ye¹, Sujuan Fei
- [3] Duseja A and Aggarwal R. APOC3 and PNPLA3 in non-alcoholic fatty liver disease: Need to clear the air. *J Gastroenterol Hepatol* 2012; 27: 848-851.
- [4] Karathanasis SK: Apolipoprotein multigene family: tandem organization of human apolipoprotein AI, CIII, and AIV genes. *Proc Natl Acad Sci U S A* 1985, 82:6374-6378
- [5] Masebe T, Bessong PO, Ndip RN, Meyer D: Genetic variants of APOC3 promoter and HLA-B genes in an HIV infected cohort in northern South Africa: a pilot study. *Int J Mol Sci* 2014, 15:11403-11415.
- [6] Tarr PE, Taffe P, Bleiber G, Furrer H, Rotger M, Martinez R, Hirschel B, Battegay M, Weber R, Vernazza P, Bernasconi E, Darioli R, Rickenbach M, Ledergerber B, Telenti A, Swiss HIVCS: Modeling the influence of APOC3, APOE, and TNF polymorphisms on the risk of antiretroviral therapy-associated lipid disorders. *J Infect Dis* 2005, 191:1419-1426.
- [7] (5)Groenendijk, M.; Cantor, R.M.; Blom, N.H.; Rotter, J.I.; de Bruin, T.W.; Dallinga-Thie, G.M. Association of plasma lipids and apolipoproteins with the insulin response element in the apoC-III promoter region in familial combined hyperlipidemia. *J. Lipid Res.* 1999, 40, 1036-1044.
- [8] (6)Green, M.L. Evaluation and management of dyslipidemia in patients with HIV infection. *J. Gen. Intern. Med.* 2002, 17, 707-810.
- [9] (12)Tarr, P.E.; Taffe, P.; Bleiber, G.; Furrer, H.; Rotger, M.; Martinez, R.; Hirschel, B.; Battegay, M.; Weber, R.; Vernazza, P.; et al. Modeling the influence of APOC3, APOE, and TNF polymorphisms on the risk of antiretroviral therapy-associated lipid disorders. *J. Infect. Dis.* 2005, 191, 1419-1426
- [10] Jiang Y, Ma J, Li H, Liu Y, You C. Effect of apolipoprotein C3 genetic polymorphisms on serum lipid levels and the risk of intracerebral hemorrhage. *Lipids Health Dis.* 2015; 14: 48. doi: 10.1186/s12944-015-0047-9 PMID: 25994187
- [11] Song Y, Zhu L, Richa M, Li P, Yang Y, Li S. Associations of the APOC3 rs5128 polymorphism with plasma APOC3 and lipid levels: a meta-analysis. *Lipids Health Dis.* 2015; 14: 32. doi: 10.1186/s12944-015-0027-0 PMID: 25928461
- [12] Talmud PJ, Humphries SE. Apolipoprotein C-III gene variation and dyslipidaemia. *Curr Opin Lipidol.* 1997; 8: 154-8.
- [13] Guettier JM, Georgopoulos A, Michael Y, et al. Polymorphisms in the fatty acid-binding protein 2 and apolipoprotein C-III genes are associated with the metabolic syndrome and dyslipidemia in a South Indian population. *J Clin Endocrinol Metab.* 2005;90:1705-11s.
- [14] Chalasani N, Younossi Z, Lavine JE, Charlton M, Cusi K, Rinella M, Harrison SA, Brunt EM, Sanyal AJ. The diagnosis and management of nonalcoholic fatty liver disease: Practice guidance from the American Association for the Study of Liver Diseases. *Hepatology.* 2018 Jan;67(1):328-357.
- [15] Younossi ZM. Non-alcoholic fatty liver disease - A global public health perspective. *J Hepatol.* 2018 Nov 9. pii: S0168-8278(18)32517-0. doi:10.1016/j.jhep.2018.10.033.
- [16] Obika M, Noguchi H. Diagnosis and evaluation of nonalcoholic fatty liver disease. *Exp Diabetes Res.* 2012; 2012: 145754.
- [17] Di Rosa M, Malaguarnera L. Genetic variants in candidate genes influencing NAFLD progression. *J Mol Med (Berl).* 2012; 90: 105-18.
- [18] Wang J, Guo X, Wu P, Song J, Ye C, Yu S, et al. Association between the Pro12Ala polymorphism of PPAR- γ gene and the non-alcoholic fatty liver disease: a meta-analysis. *Gene* 2013; 528: 328-34 PubMed .
- [19] Peterson et al.
- [20] Povel CM, Boer JM, Reiling E, Feskens EJ. Genetic variants and the metabolic syndrome: a systematic review. *Obes Rev.* 2011;12: 952-67 PubMed .
- [21] van Hoek M, van Herpt TW, Dehghan A, Hofman A, Lieverse AG, van Duijn CM, et al. Association of an APOC3 promoter variant with type 2 diabetes risk and need for insulin treatment in lean persons. *Diabetologia* 2011; 54: 1360-7 PubMed .
- [22] Zhang JZ, Xie X, Ma YT, Zheng YY, Yang YN, Li XM, et al. Association between Apolipoprotein C-III Gene Polymorphisms and Coronary Heart Disease: A Meta-analysis. *Aging Dis.* 2016; 7: 36-44 PubMed .
- [23] Song Y, Zhu L, Richa M, Li P, Yang Y, Li S. Associations of the APOC3 rs5128 polymorphism with plasma APOC3 and lipid levels: a meta-analysis. *Lipids Health Dis.* 2015; 14: 32. 46. Zhang H, Chen L, Xin Y, Lou Y, Li.
- [24] Williams CD, Stengel J, Asike MI, Torres DM, Shaw J, Contreras M. Prevalence of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis among a largely middle-aged population utilizing ultrasound and liver biopsy: a prospective study. *Gastroenterology.* 2011;140:124-31.
- [25] Lazo M, Clark JM. The epidemiology of nonalcoholic fatty liver disease: a global perspective. *Semin Liv Dis.* 2008;28:339-50.
- [26] Zhu L, Baker SS, Liu W, et al. Lipid in the livers of adolescents with nonalcoholic steatohepatitis: combined effects of pathways on steatosis. *Metabolism.* 2011;60:1001-11.
- [27] Petersen KF, Dufour S, Hariri A, et al. Apolipoprotein C3 gene variants in nonalcoholic fatty liver disease. *N Engl J Med.* 2010;362:1082-9
- [28] Marchesini G, Brizi M, Bianchi G, et al. Nonalcoholic fatty liver disease: a feature of the metabolic syndrome. *Diabetes.* 2001;50:1844-50.
- [29] Uchil D, Pipalia D, Chawla M, et al. Non-alcoholic fatty liver disease (NAFLD)-the hepatic component of metabolic syndrome. *J Assoc Physicians India.* 2009;57:201-4.
- [30] Goland S, Shimon S, Zornitzki T, et al. Cardiac abnormalities as a new manifestation of nonalcoholic fatty liver disease: echocardiographic and tissue Doppler imaging assessment. *J Clin Gastroenterol.* 2006;40:949-55.
- [31] Valenti L, Nobili V, Al-Serri A, et al. The APOC3 T-455C and C-482 T promoter region polymorphisms are not associated with the severity of liver damage independently of PNPLA3 I148M genotype in patients with nonalcoholic fatty liver. *J Hepatol.* 2011;55:1409-14.
- [32] Hyysalo J, Stojkovic I, Kotronen A, et al. Genetic variation in PNPLA3 but not APOC3 influences liver fat in non-alcoholic fatty liver disease. *J Gastroenterol Hepatol.* 2012;27:951-6.
- [33] Samuel VT, Petersen KF, Shulman GI. Lipid-induced insulin resistance: unravelling the mechanism. *Lancet* 2010;375: 2267-77 PubMed .
- [34] Guettier JM, Georgopoulos A, Michael Y, et al. Polymorphisms in the fatty acid-binding protein 2 and apolipoprotein C-III genes are associated with the metabolic syndrome and dyslipidemia in a South Indian population. *J Clin Endocrinol Metab.* 2005;90:1705-1

Tables -1

Primer sequence, Annealing temperatures, Restriction Enzymes & lengths of digested fragments of APOC3- gene for polymorphic variants

Polymorphism	rs number	Primer Sequence	Restriction Enzyme	AT*	Amplicon size (bp)	RFLP pattern
rs2854116	F- 5'-GGCTGTGA	GAGCTCAGCCCT-3'	FokI	58 °C		APOC3- 455c 196, 133,64
APOC3-482	rs2854117	R- 5' TCACACTGGAAT	TTCAGGCC-3'	MspI		143 143,159,53 ,37

AT* :Annealing

£-The same primer set was used to amplify for both polymorphisms variants.

Table:II

Frequency distribution analysis of selected demographic and risk factors of NAFLD cases and controls.

Variables	Cases N=150(%)	Controls N=180(%)	p-value chi sq
Age	90 (60)	103(57.22)	0.34
≥50=90	60 (40)	77(42.77)	
<50=60			
Dwelling			0.20
Rural=84	84(56)	92(61.34)	
Urban=66	66(44)	58(38.66)	

Table: III

Genotypic frequencies of Apoc3 Gene Polymorphism in the NAFLD cases and Controls

Genotype	Apoc3 455c (cases)(150)	Controls(180)	O.D-ratio	p-value
455c				
TT	107	150	2.00(1.18-3.40)	0.0006
TC/CC	43	30		
Allele 2N	214	300		
T	86	60	2.00(1.38-2.91)	0.0002
C				
482				
CC	118	130	0.70(0.42-1.17)	0.2
CT/TT	32	50		
Allele 2N	236	260	0.70(0.49-1.01)	0.05
C	64	100		
T				

Table:IV

Association of APOC3 Gene polymorphism(snp's 455c and 482T) and various clinical and lab features of NAFLD Patients

Parameter	Cases=150				Controls=180					
	455c		P-VALUE	O-RATIO	482T		P-VALUE	O.RATIO		
	TT=107	TC+CC=43			CC=118	CT+TT=32				
AGE										
≥50=90	60(66.66)	30(33.34)	0.20	1.64(0.77-3.48)	69(76.66)	21(23.34)	0.54	1.35(0.59-3.06)		1.47(0.68-3.16)
<50=60	47(78.33)	13(21.67)			49(81.66)	11(18.34)				
SEX										
Male=86	67(77.90)	19(22.10)	0.04	0.47(0.23-0.96)	68(79.07)	18(20.93)	1	0.94(0.42-2.07)		
Female=64	40(62.5)	24(37.5)			50(78.13)	14(21.87)				
DWELLING										
Rural=84	62(73.81)	22(26.19)	0.28	0.76(0.37-1.54)	72(85.72)	12(14.28)	0.01	0.38(0.17-0.85)		
Urban=66	45(68.19)	21(31.81)			46(69.69)	20(30.31)				
HDL										
High/low=31	20(64.52)	11(35.48)	0.37	1.49(0.64-3.46)	24(77.42)	07(22.58)	1	1.09(0.42-4.83)		1.47(0.68-3.16)
Normal=119	87(73.10)	32(26.90)			94(78.99)	25(21.01)				
LDL										
High/low=30	19(63.34)	11(36.66)	0.36	1.59(0.68-3.70)	25(83.33)	05(16.67)	0.33	0.68(0.24-1.97)		
Normal=120	88(73.34)	32(26.66)			93(77.5)	27(22.5)				
TRIGLYCERIDE										
High/low=104	87(83.65)	17(16.35)	0.001	0.15(0.06-0.32)	84(80.76)	20(19.24)	0.23	0.67(0.29-1.53)		
Normal=46	20(43.47)	26(56.53)			34(73.91)	12(26.09)				
AST										
HIGH=50	34(36.66)	16(63.34)	0.56	1.27(0.60-2.66)	30	20	0.03	2.23(1.07-4.64)		
NORMAL=100	73(45)	27(55)			77	23				
ALT										
HIGH=96	63(44.79)	33(55.21)	0.05	2.30(1.02-5.15)	74(77.09)	22(22.91)	0.54	1.30(0.56-3.01)		
NORMAL=54	44(38.88)	10(61.12)			44(81.48)	10(18.52)

10.22214/IJRASET

45.98

IMPACT FACTOR:
7.129

IMPACT FACTOR:
7.429

INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Call : 08813907089 (24*7 Support on Whatsapp)