

Fc γ R genetic polymorphisms of periodontal disease in Korean population

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I. INTRODUCTION

Recent estimates indicates that the human genome contains about 25,000 to 35,000 genes¹. Genes involved in complex multifactorial diseases such as high blood pressure, diabetes mellitus and periodontal disease, are often referred to as susceptibility genes (or more correctly susceptibility alleles). For these diseases, individuals who inherit susceptibility alleles will not develop disease unless they are exposed to deleterious environments. As for the periodontal disease, gram-negative anaerobic microorganisms, cigarette smoking, and poor oral hygiene are considered to be an important environmental risk factors².

After a 15-year longitudinal study of natural history of periodontal disease in man, Löe et al.³ were able to identify 3 different groups in individuals with poor oral hygiene and no access to dental care. Only 8% of subjects showed periodontal disease

with a rapid progression and the majority of subjects (81%) showed moderate progression. However, 11% of subjects exhibited no progression at all even they did not perform any conventional oral hygiene measures, and consequently, displayed large aggregates of plaque, calculus and stain on their teeth. This variation is thought to be attributed to either unrecognized components of the environment or different susceptibility among individuals. This susceptibility could be considered as genetic factors.

It has been recognized for years that aggressive periodontitis (formerly called juvenile periodontitis) aggregates in families^{4,5}. In the aggregation study conducted in the US, the autosomal dominant mode was favored in African- American and Caucasian families⁶. The estimated frequency of the disease allele, however, was significantly higher in African-Americans and it reflects the higher prevalence of aggressive periodontitis in this group.

Very few investigations have been performed

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with regard to family studies of probands with chronic periodontitis. In van der Velden's study⁷, 23 family units consisting of 3 more siblings, were evaluated. It showed that there also may be a genetic background in less severe forms of chronic periodontitis.

Although many efforts have been devoted to figure out the genes which really involved in the progression of periodontal disease, little is revealed yet. As the immune system is thought to play a crucial role in the pathogenesis, many studies have been focused on the identification of genetic polymorphisms in several aspects of the host immunity. There are some candidate genes that may affect the pathogenesis of periodontal disease. In particular, genetic variation in the genes of immune mediators, such as cytokines, has used to be a potential targets for susceptibility factors in relation to periodontitis.

The human genome consists of more than 3 billion pairs of bases contained in 22 pairs of chromosomes, termed autosomes, and two sex chromosomes. Genes are sequences of nucleotide bases contained in noncontiguous segments called exons. Recent estimates indicates that the human genome has about 25,000 to 35,000 genes¹. For each pair, one chromosome is inherited from the father and the other from the mother. Variant forms of a gene that can occupy a specific chromosomal site are called alleles. Two or more alleles for a given locus may exist in nature throughout evolution, however they may develop at any time. In this way, when different alleles of a given gene co-exist in the human population, we speak of genetic polymorphisms⁸.

Polymorphisms arise as a result of mutation. An alteration that changes only a single base pair is called a point mutation. Not all such mutations are repaired and they can therefore be inherited and transmitted for generations. The most common class

of point mutations is the transition, comprising the substitution of one nucleotide with another. The variation at the site harboring such changes has recently termed a single nucleotide polymorphism (SNP)⁹.

The SNP may have no effects or may have some important biological effects. For example, if a transition has taken place within the coding region of a gene, it may result in an amino acid substitution and therefore express an altered protein, perhaps with an altered function. The altered regulation due to the mutation in the promoter region may give rise to reduced or inhibited gene expression or, alternatively, result in overexpression of the gene. SNPs occur the most frequently over any other type of polymorphism; the frequency of SNPs across the human genome is estimated at every 0.3-1 kb⁹.

Genetic polymorphisms are highly useful in population genetic studies. With genotyping of individuals and assessing genotype frequencies among groups of interest, one can calculate the frequency of the alleles of them. Each of the alleles and the resulting genotypes have a particular frequency in the population. It is important to know that populations with different genotype frequencies may have comparable or equal allele frequencies. Furthermore, frequencies of genotypes and alleles may differ between a diseased group and a healthy one. Subsequently, when a given allele is identified to be associated with disease, functional studies can be started to investigate the possible role of that gene in the etiology and pathogenesis of the disease⁸.

Fc γ R is leukocyte receptors for the constant part of immunoglobulins (FcR) for IgG. FcR linked with antibodies led to the identification of complex regulatory networks that integrate innate immunity with adaptive immunity^{10,11}.

The human leukocyte Fc γ R family consists of 3

major classes and encompasses 8 genes (CD64: Fc γ RIA, IB, and IC; CD32: Fc γ RIIA, IIB, and IIC; CD16: Fc γ RIIA and IIIB), which have been mapped to the long arm of chromosome 1 (1q21 and 23-24)¹². Functional bi-allelic polymorphisms have been identified for three Fc γ R subclasses: Fc γ RIIa, Fc γ RIIIa, and Fc γ RIIIb. Human polymorphonuclear neutrophils (PMN) constitutively express two Fc γ R subclasses, Fc γ RIIa and Fc γ RIIIb^{13,14}. Macrophage/monocytes and natural killer (NK) express Fc γ RIIIa on their surface¹².

Functional bi-allelic polymorphisms have been identified for 3 Fc γ R subclasses: Fc γ RIIa, Fc γ RIIIa, and Fc γ RIIIb. Fc γ RIIa and Fc γ RIIIb are human polymorphonuclear neutrophils origin. Fc γ RIIa bears either an arginine (Fc γ RIIa-R131) or histidine (Fc γ RIIa-H131) at amino acid position 131 in the second extracellular (EC2) Ig-like domain. This difference affects receptor affinity for IgG2 and IgG3. Fc γ RIIa-H/H131 PMN internalize human IgG2-opsonized bacteria more efficiently than Fc γ RIIa-R/R131 PMN^{12,14}.

The PMN-specific Fc γ RIIIb bears the NA1-NA2 polymorphism caused by 4 amino acid substitutions within the first extracellular (EC1) Ig-like domain. These four amino acid substitution at positions 18, 47, 64 and 88 results in glycosylation differences, affecting receptor affinity. PMNs of Fc γ RIIIb-NA2 individuals bind IgG1 or IgG3 less efficiently than those of Fc γ RIIIb-NA1 individuals^{15,16}.

Macrophages/monocytes and natural killer (NK) cells express Fc γ RIIIa, which bears a valine(V)-phenylalanine(F) polymorphism at amino acid position 158 in the EC2 domain. The Fc γ RIIIa-158V allotype exhibits higher affinity for both monomeric and immune complexed IgG1 and IgG3 than Fc γ RIIIa-158F does, and also it is capable of binding IgG4¹⁷.

In vitro works suggested that interindividual dif-

ferences in the efficacy of Fc γ R-mediated functions such as phagocytosis, cytotoxicity, and cytokine production depend on Fc γ R polymorphisms^{12,14,15,17}.

The Fc receptor for immunoglobulin G (Fc γ R) are associated with modifying effects of several infectious and autoimmune diseases¹⁸⁻²⁰. Fc γ RIIIa 158VV genotype may lower the risk for contracting acute poliomyelitis through better clearance of poliovirus¹⁸. The presence of high-binding alleles at the Fc γ RIIIa locus or at the Fc γ RIIIa-Fc γ RIIa haplotype might predispose to rheumatoid arthritis in shared epitope positive individuals¹⁹. It is also reported that individuals with Fc γ RIIIb NA2/NA2 were significantly more likely to have lupus nephritis among the Japanese patients with systemic lupus erythematosus²⁰.

There are some reports that studied the relation between periodontitis and Fc γ R polymorphisms. In Japanese population, the Fc γ RIIIa-158V allele and possibly Fc γ RIIIb-NA2 are to be associated with severity of chronic periodontitis²⁷. In Northern European Caucasians, the Fc γ RIIa-H/H131 genotype and the Fc γ RIIIa-V158 allele are considered as a putative susceptibility factor of periodontitis²¹. Colombo et al.²² described that Fc γ RIIa and Fc γ RIIIb genotype did not turn out any significant difference among refractory, successfully treated and periodontally healthy individuals.

It was the aim of this investigation to evaluate the prevalence of the genetic polymorphisms of IL-1 and Fc γ R and their associations with periodontal disease in Korean population.

II. MATERIALS AND METHODS

1. Subjects and Clinical assessments

Ninety periodontally healthy control Korean subjects (64 males and 26 females; age range 20-61

years, mean age: 27.5 ± 6.9 years), forty patients with severe chronic periodontitis (Severe CP; 24 males and 16 females; age range 34-70 years, mean age: 51.8 ± 7.2 years) and twenty three patients with aggressive periodontitis (AgP, 16 males and 7 females; age range 23-37 years, mean age: 32.1 ± 4.0 years) were included in this study.

Patient classification was done on the criteria described by Kornman et al.²³ and the one proposed in the 1999 American Academy of Periodontology (AAP) world workshop. The study population consists of staffs and students of Seoul National University Dental Hospital and patients referred to the department of periodontology of Seoul National University Dental Hospital. The study was approved by the Institution Review Board at Seoul National University Hospital and written informed consents were obtained from all subjects.

Clinical parameters including probing depth (PD), clinical attachment level (CAL), bleeding on probing (BOP), gingival index (GI)²⁴, supragingival plaque accumulation (PI, O'Leary plaque control record method)²⁵, alveolar bone loss (BL) were assessed. Probing depth (PD) and clinical attachment level (CAL) were recorded using the Florida Probe® (Florida Probe Co., Gainesville, FL, USA) in 6 sites of all existing tooth and they were categorized into 3 groups (<4mm, 4 to 6mm, and >6mm). Full mouth radiographs were taken by digital X-ray to evaluate alveolar bone levels using V-works version 3.5 (Cybermed, Seoul, Korea). Alveolar bone loss was evaluated by the modified method of Schei et al.²⁶. Alveolar bone loss (BL) was measured from the cemento-enamel junction (CEJ) to the alveolar bone crest and expressed as a percentage of the length from CEJ to root apex. The percentage of sites that showed more than 50% bone loss was calculated.

2. Isolation of genomic DNA and genotyping

Genomic DNA was obtained from peripheral blood of the subject by using a commercially available DNA extraction kit (Puregen Genra System, Minneapolis, MN) according to the manufacture's instructions. The genotyping was performed according to the previously described method by Kobayashi et al.²⁷.

(1) Fc γ RIIIa-158V/F genotyping

Fc γ RIIIa-158V/F genotyping also used a allele-specific PCR procedure previously described by Kobayashi et al.¹¹. Three different primers were used for the detection of one base transition (nt 559-G/T). Following primer was used as common Fc γ RIIIa-specific forward primer: 5'-TCA CAT ATT TAC AGA ATG GCA ATG G-3'; nt 449-473. The nt 559G-specific reverse primer (5'-TCT CTG AAG ACA CAT TTC TAC TCC CTA C-3'; nt 586-559) was used for finding Fc γ RIIIa-158V allele, and The 559T-specific reverse primer (5'-TCT CTG AAG ACA CAT TTC TAC TCC CTA A-3'; nt 586-559) was used for finding Fc γ RIIIa-158F allele. PCR was performed with PCR premixture (Bioneer, Koera), 100 ng genomic DNA and 200 nM of each primer in a 50 μ l reaction volume. PCR conditions were as follows: 1 cycle at 95 $^{\circ}$ C for 9 min, 37 cycles at 95 $^{\circ}$ C for 30s, 60 $^{\circ}$ C for 30s, and 72 $^{\circ}$ C for 20s, with a final extension at 72 $^{\circ}$ C for 10 min. The PCR products were determined by electrophoresis on a 2% agarose gel stained with ethidium bromide. PCR product with 138bp can be found at the electrophoresis (Figure 1).

(2) Fc γ RIIIb-NA1/NA2 genotyping

A PCR using allele-specific primers was used for Fc γ RIIIbNA1/NA2 genotyping previously as described by Kobayashi et al.²⁷. In order to genotype for NA1, 100ng of genomic DNA was added to PCR premixture

Figure 1. The result of agar-gel electrophoresis of Fc γ RIIIa-158V/F by PCR, Lane 1, Size marker: Lane 2,3, product of 158VV (138bp at lane 2): Lane 4,5, product of 158VF (138bp at lane 4, 5): Lane 6,7, product of 158FF (138bp at lane 7)

Figure 2. The result of agar gel electrophoresis of Fc γ RIIIb-NA1/NA2 by PCR, Lane 1, Size marker: Lane 2,3, product of NA1NA1 (141bp at lane 2): Lane 4,5, product of NA1NA2 (141bp at lane 4 and 169bp at lane 5): Lane 6,7, product of NA2NA2 (169bp at lane 7)

(Bioneer, Korea) with the sense primer (5'-CAG TGG TTT CAC AAT GTG AA-3'; nt 208-227) or the anti-sense primer (5'-CAT GGA CTT CTA GCT GCA CCG-3'; nt 329-349). PCR conditions were as follows: 1 cycle at 95°C for 9 min, 35 cycles at 95°C for 30s, 63°C for 30s, and 72°C for 30s, ending at 72°C for 10 min.

In order to genotype for NA2, 2 different primers were used; NA2 sense primer (5'-CTC AAT GGT ACA GCG TGC TT-3'; nt 128-147) and NA2 anti-sense primer (5'-CTG TAC TCT CCA CTG TCG TT-3'; nt 277-296). The amplification protocol was as follows: 1 cycle at 95°C for 9 min, 35 cycles at 95°C for 30s, 64°C for 15s, and 72°C for 30s, ending at 72°C for 10 min. After gel electrophoresis the end products of the reactions were 141bp for NA1 and

169bp for NA2 (Figure 2).

3. Statistical analysis

Differences in clinical parameters between each groups were compared by ANOVA test ($p < 0.05$). The associations of the genotype frequencies between each groups were determined by χ^2 test or Fisher's exact test, using software (SPSS 10.0, SPSS Inc., IL, USA).

III. RESULTS

1. Clinical parameters

All subject's clinical characteristics were shown in

Table 1. Clinical characteristics of subjects with control, severe chronic periodontitis (CP), aggressive periodontitis(AgP)

Clinical Parameters	Control (n= 90)	Severe CP (n= 40)	AgP (n= 23)
Male/Female	64/26	24/16	16/7
Age(years)	27.5±6.9	51.8±7.2	32.1±4.0
Number of Teeth	28.32±1.63	25.65±4.00*	28.39±2.39+
Number of Missing teeth	0.40±0.97	3.43±3.44*	1.22±2.02+
Gingival index	0.21±0.13	0.91±0.41*	0.93±0.36*
Plaque index	42.84±18.64	69.35±15.27*	69.77±15.65*
mean % BOP	11.64±10.59	54.82±19.01*	59.84±16.21*
mean % of Bone loss	8.58±1.53	40.80±7.32*	37.87±9.44*†
% of BL > 50%	0	33.03±13.56*	25.69 ±16.38*†
Smoking/Non Smoking	21/69	15/25	13/10

* Significantly different from Control subject groups (p<0.05)

† Significantly different from Severe CP patient groups (p<0.05)

Table 2. Periodontal characteristics of subjects with control, severe chronic periodontitis(CP) and aggressive periodontitis(AgP)

Clinical Parameters	Control (n= 90)	Severe CP (n= 40)	AgP (n= 23)
Probing depth(PD) mm (mean±SD)	2.06±0.21	3.69±0.72*	3.83±0.83*
% site of PD <4mm	100	63.11±11.54*	56.81±17.81*†
% site of PD 4-6mm	0	24.09±9.20*	31.21±12.41*†
% site of PD >6mm	0	12.81±10.78*	11.99±11.54*
Clinical attachment loss mm (mean±SD)	2.10±0.22	4.37±0.97*	4.36±0.99*
% site of CAL <4mm	100	50.26±15.94*	47.21±20.83*
% site of CAL 4-6mm	0	29.79±11.81*	33.93±13.76*
% site of CAL >6mm	0	19.95±14.86*	18.87±13.67*

* Significantly different from Control subject groups (p<0.01)

† Significantly different from Severe CP patient groups (p<0.01)

Table 1 and 2.

The number of missing teeth was greater in severe CP than in control and AgP (p<0.01). Consequently the number of remaining teeth was smaller in severe CP than in control and AgP (p<0.01). The control group showed significant difference with severe CP and AgP in some clinical parameters such as gingival index (GI), plaque index(PI) and mean percentage of bleeding on probing (BOP) (p<0.01). It meant that the clinical features of severe and AgP were similar to each other and were different from control. However, all

three groups were significantly different in mean percentage of bone loss and percentage of bone loss >50%(p<0.01).

The mean probing depth, clinical attachment loss and the percentage of sites greater than 4mm of them in severe CP and AgP were significantly higher than control (p<0.01) (Table 2).

2. Distribution of genotypes and alleles

The distributions of Fc γ R genotype frequencies were shown in Table 3 and allele frequencies in

Table 3. Distribution of genotypes in subjects with control, severe chronic periodontitis (CP) and aggressive periodontitis(AgP)

	Control (n=90) number (%)	Severe CP (n=40) number (%)	AgP (n=23) number (%)
Fc γRIIIa			
158V/V	25 (27.78)	7 (17.50)	1 (4.35)
158V/F	21 (23.33)	18 (42.50)	6 (26.09)
158F/F	44 (48.89)	15 (37.50)	16 (69.56)
Fc γRIIIb			
NA1/NA1	24 (26.67)	10 (25.00)	8 (34.78)
NA1/NA2	53 (58.89)	23 (57.50)	12 (52.18)
NA2/NA2	13 (14.44)	7 (17.50)	3 (13.04)
Fc γR Genotype (NA2 + 158V)			
Genotype (+)	33 (36.67)	17 (42.50)	4 (17.39)
Genotype (-)	57 (63.33)	23 (57.50)	19 (82.61)

* Significantly different from Control subject groups ($p < 0.01$)

+ Significantly different from Severe CP patient groups ($p < 0.01$)

Table 4. Distribution of alleles in subjects with control, severe chronic periodontitis (CP) and aggressive periodontitis(AgP)

	Control (n=90) (%)	Severe CP (n=40) (%)	AgP (n=23) (%)
Fc γRIIIa			
158V	71 (39.44%)	32 (40.00%)	8 (17.39%)
158F	109 (60.56%)	48 (60.00%)	38 (82.61%)
Fc γRIIIb			
NA1	101 (56.11%)	43 (53.75%)	28 (60.87%)
NA2	79 (43.89%)	37 (46.25%)	18 (39.13%)

Table 4.

Fc γ RIIIa and Fc γ RIIIb genotypes were also evaluated, Fc γ RIIIa 158VF showed statistically significant difference between 3 groups ($p < 0.05$). The control had more 158VV genotype than severe CP and AgP ($p < 0.05$) and AgP had more 158FF genotype than severe CP ($p < 0.05$).

In Fc γ RIIIb genotyping, half of all genotypes were Fc γ RIIIb NA1/NA2 (control: 58.89%, severe CP: 57.50%, AgP: 52.18%). It was found that all

three groups showed similar genotype distribution and the proportion of NA1/NA1 alleles were slightly more than NA2/NA2 alleles.

The Fc γ R composite genotype (Fc γ RIIIb-NA2 plus Fc γ RIIIa 158V) which was studied by Kobayashi et al.²⁷ was examined, The proportion of Fc γ RIIIb-NA2 plus Fc γ RIIIa 158V genotypes in AgP was less than in control and severe CP. However, there was no statistical significance among three groups ($p > 0.05$).

IV. DISCUSSION

Fc γ R are the leukocyte receptors for the constant (or Fc-) part of immunoglobulins for IgG. FcR which was linked with antibodies led to the immune response^{10,11}. Fc γ RIIIa is expressed on the surface of macrophage/monocytes and natural killer (NK) cells and Fc γ RIIIb is expressed on the surface of human polymorphonuclear neutrophils.

The Fc γ RIIIa-158V allele type exhibits higher affinity for both monomeric and immune complexed IgG1 and IgG3 than Fc γ RIIIa-158F does, and is capable of binding IgG4¹⁷. In Japanese population, more than 50% subjects have Fc γ RIIIa 158FF genotype, and there was no statistical difference among 3 different groups (healthy controls, AP patients and G-EOP patients)²⁸. In our study, however, the prevalence of Fc γ RIIIa polymorphism was different between groups. Severe CP and AgP showed statistically significant difference from control subjects and there was also statistically significant difference between severe CP and AgP groups ($p < 0.05$). The control has more 158V than other 2 groups (Table 4). Only one subject(1/23) has Fc γ RIIIa 158VV genotype in AgP.

Fc γ RIIIb is a neutrophil-specific receptor and has two polymorphisms (NA1 and NA2). In Japanese population, generalized aggressive periodontitis patients were different from control and chronic periodontitis patients in the proportion of Fc γ RIIIb polymorphisms. Fc γ RIIIb NA2 was found much frequently in Japanese generalized aggressive periodontitis patients (55% allelic frequency) than in other groups (40% in chronic periodontitis patients and 38% in healthy control subjects)²⁸. In our study, all three groups showed similar distribution of genotypes and allelic frequencies and didn't showed any statistically significant difference ($p > 0.05$).

Composite genotype of Fc γ R was studied by Kobayashi et al.^{27,28}. In Japanese non-smoking population, statistically significant difference in the frequency of Fc γ R composite genotype (Fc γ RIIIb-NA2 plus Fc γ RIIIa-158V) was observed between severe (46.0%) and moderate CP (15.4%) groups and between severe CP and control groups(17.2%). In our study, the distribution of composite genotype between control and severe CP patients was similar (Table 3). The frequency of Fc γ R composite genotype (Fc γ RIIIb-NA2 plus Fc γ RIIIa-158V) positive of AgP was less than that of control and severe CP. However there was no statistical significance between AgP and others ($p > 0.05$). In comparison with Japanese data, the frequency of Fc γ R composite genotype was higher in our study. The frequencies of control and severe CP in our study (36.67%, 42.50%) were also similar to that of Japanese severe CP patients (46.0%)²⁸.

It has been known that the freshly isolated NK cells from Fc γ RIIIa-158VV individuals carried more cytophilic IgG than from Fc γ RIIIa-158FF individuals. It is reported that combined genotype with Fc γ RIIIa was known to have relation with immune thrombocytopenic purpura. Although only Fc γ RIIIa 158V/F is closely related with the severity of periodontitis in our study, other genes such as Fc γ RIIIa-131R/H and Fc γ RIIIb NA1/NA2 showed relation with periodontitis in other studies in specific ethnic groups. Therefore further studies are needed to elucidate the relationship.

Within the limit of our study, the result of this investigation have shown that genetic polymorphisms of Fc γ R may be used as a periodontal disease susceptibility marker in Korean population. It is also considered that the frequency of genetic polymorphism in periodontal disease is different from ethnic groups.

V. CONCLUSION

It was our aim to evaluate the prevalence of the genetic polymorphisms of Fc γ R and their associations with periodontal disease in Korean population. Within the limit of our experiment, it is concluded that Fc γ RIIIa 158V/F polymorphism have correlation with periodontal disease characteristics. Comparing with other ethnic groups, it is assumed that there was ethnic difference in the prevalence of Fc γ R genetic polymorphism in periodontal disease.

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한국인 치주질환 환자에서 Fc γ R 유전자다형성에 관한 연구

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Genomic Project 이후로 다양한 질환에 있어서 유전적인 영향에 관한 연구가 진행되고 있다. 이 연구의 목적은 한국인 치주질환 환자에서 Fc γ R 유전자의 유전자다형성과 치주질환 특성과의 관련성을 알아보는 것이다.

치주적으로 건강한 한국인 90명 (대조군, 남자 64명, 여자 26명), 중도 만성 치주염환자 40명 (severe chronic periodontitis patients; severe CP, 남자 24명, 여자 16명), 급진성 치주염환자 23명 (aggressive periodontitis patients; AgP, 남자 16명, 여자 7명)을 대상으로 임상지수 (치주낭깊이, 임상부착소실, 치은지수, 치태지수, 탐침 후 출혈지수, 치조골소실)를 측정하였다. 또한 이들의 정맥혈에서 추출한 DNA를 PCR(Polymerase Chain Reaction)법, 전기영동법 등을 이용하여 Fc γ RIIIa, Fc γ RIIIb의 대립유전자의 존재여부를 확인하였다. 이를 바탕으로 각 유전자의 다형성 및 Fc γ R 복합유전자형 (Fc γ R composite genotype)을 확인하여, 각 군 간을 비교하였다.

치주질환의 특성과 유전자다형성과의 관련성을 알아보기 위하여 Fc γ R 유전자에 대한 유전자다형성을 조사한 결과 다음과 같은 결과를 얻을 수 있었다.

1. Fc γ RIIIa에 대한 유전자다형성 연구결과 대조군과 severe CP, AgP군 사이에서, severe CP와 AgP군 사이에서는 대립유전자분포가 서로 유의성 있는 차이를 나타내었지만($p < 0.05$), Fc γ RIIIb에서는 유의성 있는 차이를 보이지 않았다($p > 0.05$).
2. Fc γ R 복합유전자형간의 비교에서 유의성있는 차이를 발견할 수 없었다($p > 0.05$).

이와 같은 결과를 종합하여 볼 때 실험대상 한국인 치주염환자에서 Fc γ R 유전자에 대한 다형성분석에서 Fc γ RIIIa 대립유전자가 치주염에 대한 감수성과 관련되어 있다고 생각된다. 이 연구의 결과는 유전자의 차이가 치주질환의 감수성 판단의 자료로 활용할 수 있는 가능성을 보여주고 있다.

주요어 : 한국인, 치주질환, 유전자다형성, Fc γ R, 대립유전자