

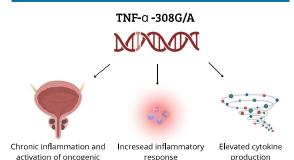


TNF-a -308G/A polymorphism (rs1800629) and prostate cancer risk in samples of men from Bahia state

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BACKGROUND



AIM

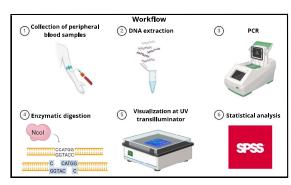
signaling pathways

response

The present study aims to analyze the role of the TNF-a -308G/A (rs1800629) polymorphism in susceptibility to PCa.

METHODS

A total of 136 samples were analyzed, including 51 cases and 85 controls, using the PCR/RFLP technique with the Ncol restriction endonuclease. The digestion products were visualized on a 4,5% agarose gel stained with ethidium bromide under UV transilluminator.



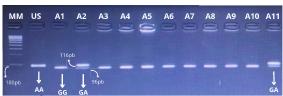


Figure 1: Electrophoresis pattern of the PCR/RFLP product for TNF-α -308G/A

Caption: MM: molecular marker; US: undigested sample; A1 to A11: patients ple; GG: wild homozygote; GA: heterozygote; AA: mutant homozygote

RESULTS

The analyzed samples were not in Hardy-Weinberg equilibrium, and this deviation could be explained by the sample size in the study. The allelic frequency of G^* and A^* was 0.667 and 0.333 in the cases, and 0.859 and 0.141 in the controls, respectively. Furthermore, genotypic and genic differentiation was observed among the samples (p<0.05), indicating a possible association between genotype and investigated phenotype.

Table 1: Genotype frequency and risk of PCa

Genotype	Cases	Controls	P Value	OR(Cl95%)
TNF-a -308G/A				
GG	30 (58.8%)	66 (77.6%)	<0.05	-
GA	8 (15.7%)	14 (16.5%)	<0.05	-
AA	13 (25.5%)	5 (5.9%)	<0.05	-
Dominant model				
GG	30	66	Ref	1.257
GA+AA	21	19	0.032*	(0.477-3.316)
Recessive model				
AA	13	5	Ref	0.220
GA+GG	38	80	0.002*	(0.057-0.846)

Table 2: Allele frequency of TNF-a -308G/A polymorphism

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Popu l ation	*G	*A		
Cases	0.667	0.333		
Controls	0.859	0.141		
Brazilians+	0.894	0.106		
Africans*	0.876	0.123		
Europeans*	0.830	0.169		
Asians*	0.926	0.073		

https://abraom.ib.usp.br/search.php+ https://www.ncbi.nlm.nih.gov/snp/rs689466*

CONCLUSIONS

This study contributes to the comprehension of the role of TNF-a polymorphism in prostate cancer. However, future studies should incorporating samples from a diverse genomic composition and a larger number of samples. Moreover, measuring cytokine levels in plasma may contribute to a better understanding of the association between inflammation pathophysiology of PCa.

REFERENCES









