

Mutational Difference between the Microsatellite BAT 26 and the *TP53* Coding Gene and Survival of Patients with Oral Cavity Cancers in Senegal

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Abstract Oral cancers primarily present as oral squamous cell carcinoma and are a subgroup of head and neck cancers (HNC). The objective of this study was to identify mutational differences and assess the survival of Senegalese patients with oral cavity cancers. A total of 75 tissue samples from patients and 40 blood samples from healthy controls were analyzed. DNA extraction, polymerase chain reaction (PCR), and sequencing were performed to obtain genetic sequences. The software BioEdit and DnaSP were used to analyze polymorphism and genetic diversity in patients. Chromatograms from the BAT 26 microsatellite and the *TP53* gene were compared to the consensus sequence and the *TP53* reference sequence, respectively, using Mutation Surveyor software. The multivariate Cox analysis was performed using R software, and the impact of BAT 26 instability on patient survival was evaluated using the Kaplan–Meier method with StatView software. The results revealed greater genetic diversity in the *TP53* gene than in BAT 26, particularly in terms of the number of polymorphic and informative sites. Furthermore, microsatellite instability appeared to lead to the accumulation of a specific *TP53* mutation (c.640C>G p.His214Asp). This study indicates that Senegalese patients with oral cavity cancers harbor more *TP53* mutations than BAT 26 mutations. However, BAT 26 instability appeared to confer a survival advantage to patients.

Keywords: oral, cavity, cancer, mutations, *TP53*, BAT 26, survival

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1. Introduction

Oral cancers primarily present as oral squamous cell carcinoma (OSCC). They constitute a global health concern, with approximately 389,846 new cases diagnosed and 188,438 deaths, equivalent to one death every three minutes reported annually [1,2]. In Senegal, these cancers rank 12th, with 117 new cases and 111 deaths reported each year [1]. Due to its location and clinical heterogeneity, oral cancer has a significant impact on quality of life, and its treatment is often complex. Among recently diagnosed individuals, just over half survive beyond five years. This overall survival rate has not significantly improved in recent decades, despite advances in diagnostics techniques and treatment approaches. This statistic largely reflects the tumor stage at diagnosis, the presence of regional lymph node metastases, and the development of second primary cancers [3]. Although various prognostic markers and therapeutic targets have been proposed over the past decades, they are not yet integrated into staging systems and treatment protocols. This may partly explain the demographic rise in oral

cancer cases observed in recent years in Senegal [4]. Understanding the genetic origins of oral cancer has greatly advanced, as it is now widely accepted that solid tumors are not genetically stable. Given that these alterations influence cancer progression, considerable efforts have been made to isolate prognostic genetic markers [5]. Microsatellite instability (MSI), for instance, has been suggested as a possible prerequisite for carcinogenesis [6]. Moreover, somatic mutation analysis of *TP53* is now commonly used in clinical trials involving patient stratification based on *TP53* status, as well as in studies of new drugs targeting either wild-type or mutant *TP53* to activate an antitumor *TP53* response. Screening for *TP53* and BAT 26 mutations is thus becoming an integral part of many therapeutic and preventive strategies in clinical practice [7]. Therefore, identifying the genetic factors involved in oral carcinogenesis and patient survival can contribute to the understanding and potential prevention of oral cancer spread in Senegal. These observations highlight the need for a deeper understanding of alterations in the BAT 26 microsatellite and the *TP53* gene. Consequently, the aim of this study was to identify mutational differences and survival outcomes in Senegalese patients with oral cavity cancers.

2. Methodology

Study Population and Sampling Procedure

The study was conducted on Senegalese patients with histologically confirmed oral cancer, treated at the Department of Stomatology and Maxillofacial Surgery of Aristide Le Dantec Hospital. After obtaining approval from the Ethics Committee of Cheikh Anta Diop University, the study was carried out from February 2021 to March 2023. In total, 75 samples (including 40 tumor tissues, 20 adjacent non-tumor tissues, and 15 blood samples) collected from patients and 40 blood samples from healthy controls were included in the study. For each patient, a tumor tissue (TT), a adjacent non-tumor tissue (ANT), and peripheral blood were collected during biopsy. The adjacent non-tumor tissues were not histopathologically confirmed as normal. However, previous studies hypothesized that these tissues may have accumulated mutations identical to those found in cancerous tissues [8,9]. Therefore, to verify this hypothesis, adjacent non-tumor tissues were collected from each patient. The selection of these tissues was also based on the extent of the surgical procedures in order to minimize the risk of potential recurrence.

Molecular analyses

DNA from tissue and blood samples was extracted using the Zymo Research Kit, following the manufacturer's instructions for each type of biological material.

- ✓ The BAT 26 microsatellite was amplified using the primer pair (F 5'-CTGCGGTAATCAAGTTTTAG-3' and R 5'-AACCATTCAACATTTTAAACCC-3'). A reaction volume of 25 µL was used, containing: 12.5 µL of Master Mix, 1 µL of MgCl₂, 0.5 µL of each primer, 8.5 µL of MilliQ water, and 2 µL of cDNA. PCR was performed under the following conditions: initial denaturation at 95°C for 5 minutes; 35 cycles of denaturation at 95°C for 30 seconds, annealing at 47°C for 1 minute, and extension at 70°C for 1 minute; followed by a final extension at 70°C for 10 minutes.
- ✓ The region from exon 5 to exon 6 of the *TP53* gene was amplified using the primer pair (F 5'-GTTTCTTTGCTGCGTCTTC-3' and R 5'-CTTAACCCCTCCTCCAGAG-3'). A reaction volume of 25 µL was used, containing: 12.5 µL of Master Mix, 0.5 µL of each primer, 5 µL of MilliQ water, and 2 µL of cDNA. PCR was performed under the following conditions: initial denaturation at 94°C for 7 minutes; 35 cycles of denaturation at 94°C for 1 minute, annealing at 64°C for 1 minute, and extension at 72°C for 1 minute; followed by a final extension at 72°C for 10 minutes.

After visualization under blue light, PCR products for which the primers had successfully annealed were purified and sequenced. Sequencing was based on a specific PCR reaction that, in addition to the usual components (DNA template, polymerase, primers, dNTPs, and Mg²⁺), included modified nucleotides dideoxynucleotides (ddNTPs). These ddNTPs are coupled to fluorescent: ddATP (green), ddTTP (red), ddCTP (blue), and ddGTP (yellow), and lack a hydroxyl group at the 3' end of the

deoxyribose. The incorporation of these ddNTPs by the polymerase therefore terminates the elongation of the complementary DNA strand being synthesized, generating fragments of different lengths.

Sequencing reactions were carried out in an MJ Research PTC-225 Peltier thermal cycler using the ABIPRISM BigDye™ Terminator Cycle kits. Each sample was sequenced using the sense primer. Fluorescent fragments were purified following the BigDye XTerminator purification protocol. Finally, the samples were suspended in distilled water and subjected to electrophoresis in an ABI 3730xl sequencer (Applied Biosystems).

Genetic Analyses

Genetic Diversity Analyses

The sequences obtained from tissue samples (both cancerous and healthy) and blood were carefully checked, aligned, and corrected using BioEdit software version 7.1.9 [10]. A comparative analysis between the two markers (BAT 26 and *TP53*) was conducted by evaluating several parameters: the number of variable sites, the number of non-informative variable sites, the number of informative variable sites, the total number of mutations (Eta), the average number of nucleotide differences between sequences (k), the number of haplotypes¹, nucleotide diversity indices (π), and haplotype diversity indices (Hd) across cancerous tissues, healthy tissues, and blood samples for each marker. Variable sites are positions in the sequence that show at least two different nucleotide states, but with differing frequencies. Non-informative variable sites are those where at least two types of nucleotides are present, with one occurring at a much higher frequency than the other. Informative variable sites, on the other hand, are those where at least two nucleotide types are present, and each is found in at least two of the sequences being compared. The number of mutations refers to the total count of synonymous and non-synonymous sites. Nucleotide diversity is the probability that two randomly selected sequences from a population differ at a given site, while haplotype diversity is the probability that two randomly selected haplotypes from a population are different. All of these variables were determined using DnaSP software version 5.10 [11].

Association between BAT 26 Instability and *TP53* Mutations

Mutation Surveyor software version 5.0.1 (<https://softgenetics.com/products/mutation-surveyor/>) allows for the comparison of chromatograms of any submitted gene with the reference sequence of that gene. To determine the presence of mutations and their positions within the gene, the raw sequences of BAT 26 and *TP53* were analyzed using this software. Given the significant difference between the microsatellite sequences available in reference databases and those of our study population, a consensus sequence was constructed from our control samples. This consensus sequence was obtained from the 40 control samples, all of which showed a repeat of 25 adenines (A). However, this consensus sequence does not represent the entire Senegalese population but was used in this study as a first investigation of its kind. Microsatellite instability (MSI) was considered positive when the

¹ Individuals who have the same nucleotide sequence.

sequence of any patient tissue showed different sizes or mutations compared to the consensus sequence. An association analysis was performed between BAT 26 microsatellite instability and *TP53* mutations with a frequency equal to or greater than 50% among patients. Fisher's exact test was used under R version 4.2.2 (R Core Team, 2022).

Association analyses and risk assessment

Classical multifactorial risk and prognostic factors were evaluated using the Cox model. Parameters such as age were considered risk factors, while tumor stage, lymph node involvement, and tumor grade were analyzed as prognostic factors. This analysis was performed using R software.

Survival Tests

The average patient survival time was determined using statistical tests. To assess whether BAT 26 instability impacts patient survival, survival analyses were conducted using the Kaplan-Meier method [12] with StatView software version 5.0.1. The Kaplan-Meier method allows the generation of survival curves. Comparison of survival times was performed using the log-rank test [13]. Due to the small sample size, survival analyses related to *TP53* mutations were not included to avoid a lack of statistical significance that could result from the limited population size.

The significance level was set at 5% (*P*-value 0.05) for all tests.

3. Results

Genetic Diversity Difference

The comparative analysis of genetic diversity rates in oral tissues reveals that cancerous tissues, adjacent non-tumor tissues, and blood from affected individuals are more polymorphic for *TP53* than for BAT 26, particularly in terms of the number of polymorphic sites and the

number of informative sites. Moreover, for the *TP53* gene, 228, 109, and 38 mutations were identified in cancerous tissues, adjacent non-tumor tissues, and blood samples, respectively. In contrast, only 36, 23, and 14 mutations were observed for BAT 26 in the same tissue types. This greater diversity of *TP53* in oral tissues is further highlighted by the significantly higher number of nucleotide differences compared to those observed in BAT 26. However, it is important to note that blood samples appear to be less polymorphic than tissue samples for both BAT 26 and *TP53*. Additionally, for both genetic markers, high haplotype diversity and low nucleotide diversity were observed. Nevertheless, nucleotide diversity indices were lower in cancerous and adjacent non-tumor tissues for BAT 26 when compared to *TP53*. This suggests that the microsatellite polymorphism evolves more rapidly than that of *TP53* in Senegalese patients with oral cavity cancer. These results are presented in Table 1.

Association between BAT 26 Instability and *TP53* Mutations

The results show that individuals exhibiting instability at the BAT-26 locus accumulate more *TP53* mutations. However, only the c.640C>G p.His214Asp mutation is significantly associated with microsatellite instability (*p* = 0.05). These findings therefore suggest that BAT 26 instability may promote the accumulation of the c.640C>G p.His214Asp *TP53* mutation in Senegalese patients (Table 2).

Multivariate analysis using the Cox model

The multivariate analysis indicates that only lymph node involvement appears to have a prognostic effect in patients (*HR* = 1.47), although this effect is not statistically significant. Other parameters, such as age, stage, and tumor grade, do not seem to have a prognostic impact in Senegalese patients.

Table 1. Genetic Diversity Parameters between BAT 26 and *TP53*

	BAT 26			TP53		
	Cancerous tissue	Adjacent non-tumor tissues	Blood	Cancerous tissue	Adjacent non-tumor tissues	Blood
Genetic diversity parameters						
Number of variable sites	29	23	13	152	92	32
Non informative sites	13	7	7	37	51	26
Informative sites	16	11	6	115	41	6
Total number of mutation (Eta)	36	23	14	228	109	38
Number of haplotypes (h)	25	15	8	25	14	5
Average number of nucleotide differences (K)	4.48	4.86	3.87	41.10	26.98	9.25
Genetic diversity indices						
Haplotype diversity indice (Hd)	0.963±0.015	0.958±0.033	0.909±0.065	0.923±0.00	0.990±0.00	0.722±0.02
Nucleotide diversity indice (π)	0.042±0.005	0.046±0.006	0.034±0.006	0.099±0.00	0.065±0.00	0.022±0.00

Table 2. Association between TP53 Variants and BAT 26 Instability

TP53 Variants	Instability	Stability	Odd Ratio (P-value)
c.638G>A p.213Arg>Gln	16	11	1 (0.69)
c.640C>G p.214His>Asp	15	6	4 (0.05*)
c.643A>G p.215Ser>Gly	13	6	2.6 (0.23)
c.644G>A p.215Ser>Asn	17	10	2.4 (0.27)
c.647T>G p.216Val>Gly	16	8	0.3 (0.16)
c.653T>G p.218Val>Gly	12	7	0.5 (0.51)
c.655C>G p.219Pro>Ala	12	7	1.6 (0.51)
c.661G>A p.221Glu>Lys	12	7	1.6 (0.51)
c.662A>T p.221Glu>Val	13	6	2.6 (0.19)

Note. The yellow line highlights the significant association between BAT-26 instability and the TP53 c.640C>G p.His214Asp mutation.

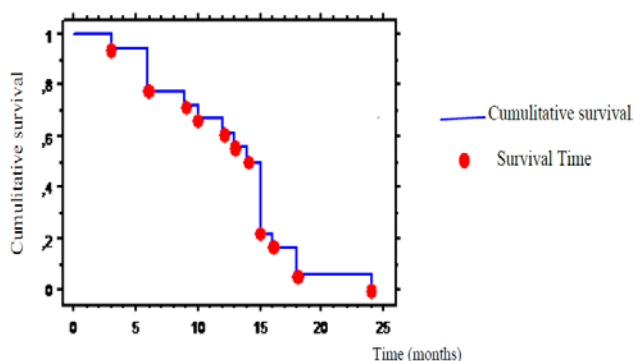
Table 3. Multivariate analysis using the Cox model

Factors	HR	95 % CI	P-value
Age	0.994	0.95-0.03	0.79
Stage	0.58	0.15-2.17	0.42
Lymph node	1.47	0.45-4.79	0.51
Tumor grade	0.63	0.15-2.62	0.53

Note. HR=Hazard ratio; CI=confidence interval

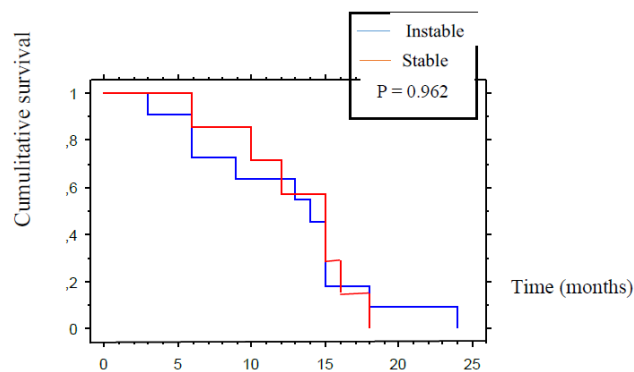
Overall Survival

Figure 1 shows that the mean survival time of patients is 12.77 ± 1.24 months, with a range from 4 to 24 months.

**Figure 1. Overall Survival Curve of Patients with Oral Cancer**

Correlation between Survival Time and MSI Status

The analysis shows that instability appears to confer a survival advantage in patients (Figure 2). However, this correlation is not statistically significant.

**Figure 2. Survival Curve according to BAT 26 Stability and Instability**

4. Discussion

This study was conducted with Senegalese patients treated at the Stomatology and Maxillofacial Surgery

Department of Aristide Le Dantec Hospital. The aim was to identify the mutational differences between the BAT 26 microsatellite and the TP53 gene, as well as the survival of patients with oral cavity cancers in Senegal. For the BAT 26 microsatellite, there were 36 mutations in cancerous tissues, 23 in adjacent non-tumor tissues, and 14 in blood samples. In contrast, 228, 109, and 38 mutations were found in cancerous tissues, adjacent non-tumor tissues, and blood, respectively, for the TP53 gene. These results indicate that Senegalese patients exhibit a mutational difference between BAT 26 and TP53. In other words, patients with oral cancer harbor more TP53 mutations than BAT 26 mutations. Furthermore, mutations were found both in cancerous tissues and adjacent non-tumor tissues, with a slightly higher number of mutations in cancerous tissues than in adjacent non-tumor tissues. This suggests a closeness between cancerous and adjacent non-tumor tissues. These findings align with those of Samb *et al.* [9], who observed a similarity between healthy and cancerous tissues for both markers in Senegalese patients with oral cancers. These data confirm the hypothesis of a genetic similarity between the two types of tissues studied. Indeed, these results could help refine surgical margins in order to reduce the risk of recurrence. The high haplotypic diversity combined with low nucleotide diversity suggests a rapid evolution of polymorphism at the BAT 26 microsatellite, increasing its instability and potentially promoting the accumulation of TP53 gene mutations in Senegalese patients. This interpretation is supported by the observed association between BAT 26 instability and the accumulation of the TP53 c.640C>G variant, as demonstrated by our results.

These findings are consistent with those of Nikbakht *et al.* [14], who found that MSI was more frequently observed in tumors with the heterozygous arginine/proline genotype of TP53. Since BAT 26 is part of the mismatch repair (MMR) system, which interacts with the p53 protein to maintain genomic integrity, microsatellite instability may influence the prevalence of TP53 mutations. Indeed, oral cavity cancers often exhibit significant genomic instability, leading to initiation and

progression events that transform normal epithelial cells into cancerous cells [9]. MMR system anomalies and p53 mutations contribute to tumorigenesis in various cells. Furthermore, MMR-deficient cells show defective activation of p53 family members when exposed to alkylating agents or cisplatin [14]. However, our results need to be confirmed by larger studies in order to achieve statistical significance regarding a possible association between MSI and other *TP53* variants.

This study showed that patients with oral cancers have a mean survival of 12.77 ± 1.24 months. This result is lower than that of Adduri *et al.* [15], who found a median survival of 30.5 months, and also lower than the findings of Mbaye *et al.* [16], who reported a median survival of 23 months in Senegalese patients with breast cancer. This shorter survival time could be due to differences in tumor aggressiveness; oral cavity cancers may therefore be more aggressive than breast cancers among Senegalese patients. It could also reflect the fact that most cases are diagnosed at an advanced stage of the disease. Indeed, Samb *et al.* [4] reported that 80% of their patients presented with very advanced tumors (stages T3–T4). Several factors may explain this diagnostic delay: the trivialization of minor ulcerations, prolonged reliance on traditional treatments, socioeconomic hardship, the high cost of medical care, and the high rate of illiteracy observed in developing countries, particularly in Senegal. In addition, the scarcity of healthcare infrastructure, the shortage of qualified medical personnel, the lack of public awareness campaigns, and the persistence of cultural beliefs all contribute to delayed diagnosis and, consequently, late presentation of the disease. Furthermore, the lack of adequate equipment and the limited number of trained professionals can sometimes lead to dental extractions performed without prior radiological or thorough clinical examinations [17]. As a result, when a tooth extraction is poorly performed especially in a tumoral context the consequences can be severe, even fatal for the patient [18]. Moreover, the nutritional status of patients with oral tumors often deteriorates, further increasing the risk of mortality. Altogether, these factors may explain the significantly shorter average survival time compared to that reported in studies conducted in Western countries.

Our results also indicated that microsatellite instability might confer a survival advantage among Senegalese patients, although this association was not statistically significant. These results align with those of Mbaye *et al.* [16], who showed that BAT 26 instability is significantly associated with longer post-operative survival. Numerous studies have demonstrated that MSI tumors have a better survival rate [19,20]. Additionally, Carvalho *et al.* [21] found that in colorectal cancers, the 5-year survival rate was 85% for patients with BAT 26 instability compared to those who were stable for BAT 26.

The reasons for this survival advantage remain unclear, but they could be related to a self-destructive mechanism resulting from the large number of mutations accumulated in the cellular genome. These mutations may affect genes involved in the variability of the tumor clone [22].

Furthermore, the relationship between survival and prognostic factors showed no statistical significance

within this cohort. However, other studies [23,24] suggest that survival rates for this type of cancer are closely related to tumor size, lymph node involvement, the presence of distant metastases, and the degree of tumor differentiation. The clinical stage of the lymph nodes has been reported as a significant prognostic factor for survival, and the TNM classification system correlates well with overall survival [25,26]. It appears that prognosis is better for early-detected tumors that are well-differentiated and show no evidence of regional or distant metastases. Therefore, prognosis worsens with advanced disease and increasing inaccessibility of the tumor, and the TNM stage significantly affects five-year survival [27].

5. Conclusion

Based on this study, we can conclude that Senegalese patients with oral cavity cancers harbor more *TP53* mutations than BAT 26 mutations. There appears to be an association between BAT 26 instability and a specific mutation in the *TP53* gene. Thus, *TP53* and BAT 26 could serve as relevant molecular markers in oral cancers. Although this trend is not statistically significant, BAT 26 instability seems to confer a survival advantage in patients. Despite the frequent alterations of BAT 26 and *TP53* in this cohort, their prognostic value needs to be confirmed through larger, multi-institutional studies.

Therefore, it would be necessary to develop follow-up strategies to prevent patient loss, which would provide a clearer view of their survival. Furthermore, future studies should focus on the role of MSI in the expression of cancer-related genes and whether changes in the length of intronic regions affect the expression of these genes. This information could be useful in determining cancer progression, which is crucial for deciding on the appropriate type of treatment.

Conflict of Interest

The authors declare no conflict of interest.

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Ethical Approval

Approval was granted by the Ethics Committee of the Cheikh Anta DIOP University of Dakar (date 2018/N°0272).

Consent to Participate

Informed consent was obtained from all individual participants included in the study.

References

- [1] Ferlay J, Ervik M, Lam F, Laversanne M, Colombet M, Mery L, Piñeros M, Znaor A, Soerjomataram I, Bray F (2024). Global Cancer Observatory: Cancer Today. Lyon, France: International Agency for Research on Cancer.
- [2] Bray, F. *et al.* Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA. Cancer J. Clin.* 68, 394-424.
- [3] Kim, D. W., Lee, S., Kwon, S., Nam, W., Cha, I. H., & Kim, H. J. (2019). Deep learning-based survival prediction of oral cancer patients. *Scientific reports*, 9(1), 6994.
- [4] Samb, M. D., Mbaye, F., Diatta, H., Ndiaye, M. M., Touré, S., and Sembene, M. (2024). Epidemiology and association of risk factors with molecular data of oral cancer in Senegal Sub-Sahara region. *Journal of Cancer Research and Experimental Oncology*, 15(1), 1-12. Sahara region. *Journal of Cancer Research and Experimental Oncology*, 15(1), 1-12.
- [5] Ludwig, J. A., & Weinstein, J. N. (2005). Biomarkers in cancer staging, prognosis and treatment selection. *Nature Reviews Cancer*, 5(11), 845-856.
- [6] Corso, G., Pedrazzani, C., Marrelli, D., Pascale, V., Pinto, E., & Roviello, F. (2009). Correlation of microsatellite instability at multiple loci with long-term survival in advanced gastric carcinoma. *Archives of Surgery*, 144(8), 722-727.
- [7] Friedlander, P. L. (2001). Genomic instability in head and neck cancer patients. *Head & neck*, 23(8), 683-691.
- [8] Diatta, H., Mbaye, F., Gueye M. D., Ndiaye M. M., **Samb M. D.**, Fall, M., Diallo, B. K., Toure, S., and Sembene, M. (2021). Genetic mutations of exon 4 at *TP53* gene in oral cavity cancers. *Oral Health and Care*, 6, 2399-9640.
- [9] Samb, M. D., Mbaye, F., Ndiaye, M. M., Toure, S., & Sembene, M. (2023). Bat 26 Microsatellite Instability in Oral Cavity Cancers in Senegal. *Journal of Cancer Therapy*, 14(1), 25-37
- [10] Hall, T. A. (1999, January). BioEdit: a user-friendly biological sequence alignment editor and analysis program for Windows 95/98/NT. In *Nucleic acids symposium series* (Vol. 41, No. 41, pp. 95-98). [London]: Information Retrieval Ltd., c1979-c2000.
- [11] Rozas J., Librado P., Sánchez-Del Barrio J.C., Messeguer X. and Rozas R. 2012. DnaSP Version 5 Help Contents [Help File]. Available with the program at [http:// www.ub.edu/dnasp/](http://www.ub.edu/dnasp/).
- [12] Kaplan E.L. & Meir P. 1958. Estimation from Incomplete Observations. *Journal of the American Statistical Association*. 53 (282): 457-481.
- [13] Peto R., Pike M.C., Armitage P., Breslow N.E., Cox D.R. & Haward S.V. 1979. Organization and analysis of comparative therapeutic trials involving long-term patient follow-up. *Revue d'Épidémiologie et de Santé Publique*. 27: 167-255.
- [14] Nikbakht, D. M., & Miromohammad, S. H. (2010). *TP53* codon 72 heterozygosity may promote microsatellite instability in sporadic colorectal cancer.
- [15] Adduri, R. S., Kotapalli, V., Gupta, N. A., Gowrishankar, S., Srinivasulu, M., Ali, M. M., ... & Bashyam, M. D. (2014). P53 nuclear stabilization is associated with FHIT loss and younger age of onset in squamous cell carcinoma of oral tongue. *BMC Clinical Pathology*, 14, 1-8.
- [16] Mbaye, F., Ka, S., Dem, A., Kane, M., & Sembéne, M. (2015). Clinicopathological and survival significance of BAT-25 and BAT-26 instability in breast cancer among Senegalese patients. *International Journal of Cancer Research and Experimental Oncology*, 7(2), 13-19.
- [17] World Health Organisation. (2012). Key Interventions for Cancer Prevention and Control in Africa: A Handbook for Leaders and Managers.
- [18] Mfutu, C. M., Pakasa, N. M., Sekele, J., Nzudjom, A., Bolenge, J., & Nyembwe, D. (2020). Clinical and histopathological profiles of oral cavity tumors after dental extraction at the University Clinics of Kinshasa in the Democratic Republic of Congo. *International Journal of Advanced Research*, 6(2), 286-289.
- [19] Jass J.R. 1999. Towards a molecular classification of colorectal cancer. *International Journal Colorectal Disease*. 14 (4-5): 194-200.
- [20] Gryfe R. & Gallinger S. 2001. Microsatellite instability, mismatch repair deficiency, and colorectal cancer. *Surgery*. 130 (11): 17-21.
- [21] Carvalho F., Santos E.M.M., Pena S.D., Vettore A., Lopes A., Ferreira F.O., Junior S.A., Nakagawa W.T. & Rossi B.M. 2005. Clinicopathological significance of BAT-26 instability in 184 patients with colorectal cancer. *Applied cancer research*. 25 (1): 36-40.
- [22] Shin, D. M., Mao, L., Papadimitrakopoulou, V. M., Clayman, G., El-Naggar, A., Shin, H. J., Lee, J. J., Lee, J. S., Gillenwater, A., Myers, J., Lippman, S. M., Hittelman, W. N., & Hong, W. K. (2000). Biochemopreventive therapy for patients with premalignant lesions of the head and neck and p53 gene expression. *Journal of the National Cancer Institute*, 92(1), 69-73.
- [23] Yang, M., Kim, W. H., Choi, Y., Lee, S. H., Kim, K. R., Lee, H. S., & Tae, K. (2006). Effects of ERCC1 expression in peripheral blood on the risk of head and neck cancer. *European journal of cancer prevention: the official journal of the European Cancer Prevention Organisation (ECP)*, 15(3), 269-273.
- [24] Zhang, Z., Shi, Q., Liu, Z., Sturgis, E. M., Spitz, M. R., & Wei, Q. (2005). Polymorphisms of methionine synthase and methionine synthase reductase and risk of squamous cell carcinoma of the head and neck: a case-control analysis. *Cancer Epidemiology Biomarkers & Prevention*, 14(5), 1188-1193.
- [25] Amit, M., Yen, T. C., Liao, C. T., Binenbaum, Y., Chaturvedi, P., Agarwal, J. P., ... & International Consortium for Outcome Research (ICOR) in Head and Neck Cancer. (2013). Clinical nodal stage is a significant predictor of outcome in patients with oral cavity squamous cell carcinoma and pathologically negative neck metastases: results of the international consortium for outcome research. *Annals of surgical oncology*, 20, 3575-3581.
- [26] Ong, T. K., Murphy, C., Smith, A. B., Kanatas, A. N., & Mitchell, D. A. (2017). Survival after surgery for oral cancer: a 30-year experience. *British Journal of Oral and Maxillofacial Surgery*, 55(9), 911-916.
- [27] Warnakulasuriya, S. (2009). Global epidemiology of oral and oropharyngeal cancer. *Oral oncology*, 45(4-5), 309-316.



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