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Staining Intensity Patterns Of Foxp3 And P53 In Prostate Carcinoma: A Molecular Insights From A Sub-Saharan Cohort

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ABSTRACT

Background

Prostate cancer is the leading cause of cancer related deaths and one of the mostly diagnosed cancers among men cancer in Sub Saharan African countries including Uganda. Despite the high burden and aggressive nature of prostate cancer in men, there is limited data on the staining intensity patterns of crucial biomarkers such as Foxp3 and p53 in this population including Ugandan prostate cancer patients. Therefore, this study intended to determine the staining intensity of Foxp3 and p53 among archived prostate cancer tissue blocks from the Uganda Cancer Institute bio repository.

Methodology

Among one hundred twenty seven prostate cancer blocks that had earlier diagnosed, we managed to obtain some demographic data such as age and a few with PSA results. We stained the tissue sections with Foxp3 and p53 antibodies using immunohistochemistry and examined microscopically

Results

Our median age was 72 with an interquartile range of 65-78 years, participants with PSA values were high with >20ng/ml. Foxp3 was absent in most of the prostate tissue sections and a few showed a staining intensity 12% as strong, 6.0% moderate staining intensity and 82% didn't show a staining intensity while p53 staining intensity was abundant with moderate (61%), strongly (24%) and least being weakly stained intensity with 15%.

Conclusion

Low or absent of Foxp3 staining intensity was associated with aggressiveness of the disease and presence of p53 different staining intensity levels were attributed to p53 mutations as a disease progresses.

1. Introduction

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Prostate cancer remains a significant global health burden, being the most commonly diagnosed cancer and a leading cause of cancer-related mortality among men (1). The disease exhibits considerable geo-ethnic disparities, with men of African ancestry facing a higher risk of aggressive disease and poorer outcomes compared to other populations (2-4). Mortality rates for prostate cancer in sub-Saharan Africa are notably higher than global averages, yet there is lack of thorough molecular and genetic studies from Sub Saharan region (3, 5). Understanding the unique molecular landscape of PCa in African populations is therefore necessary to inform effective diagnostic, prognostic, and therapeutic strategies.

The tumor microenvironment plays a crucial role in cancer progression, with immune cells being key components (6). Forkhead box protein P3 (Foxp3) is a master transcriptional regulator expressed in regulatory T cells, which are known to suppress anti-tumor immune responses (7). The presence of Foxp3 Tregs within prostate tumors has been implicated in immune evasion, tumor advancement, and is often associated with worse clinical outcomes, including advanced tumor stage and reduced progression-free survival (8, 9). However, the density and significance of Foxp3 expression can vary across different tumor molecular subtypes and racial ancestries (10). In some studies, higher Foxp3+ cell density has been linked to a higher risk of metastasis among African-American patients (10).

Concurrently, the tumor suppressor protein p53 is a critical guardian of the genome, playing central roles in cell cycle arrest, DNA repair, and apoptosis (1, 11). Aberrant expression or nuclear accumulation of p53, frequently indicative of underlying p53 gene mutations, is a common event in many human cancers, including prostate cancer(1). P53 alterations are associated with aggressive disease, unfavorable clinical endpoints, and can influence the tumor immune microenvironment, including T-cell densities (12). Notably, p53 nuclear accumulation has been shown to correlate with a unique tumor immune microenvironment, including increased CD3+ T-cell density (13). While p53 mutations have significant functional implications(14), their frequency can vary, with some studies suggesting p53 nuclear accumulation may be less frequent among African-American tumors compared to European-American tumors (13).

Despite the high burden and aggressive nature of prostate cancer in men of African descent, there is limited data on the staining intensity patterns of crucial biomarkers such as Foxp3 and p53 in this population including Uganda. Therefore, this study intended to determine the staining intensity of Foxp3 and p53 among archived prostate cancer tissue blocks from the Uganda Cancer Institute bio repository. Characterizing these markers in an underrepresented PCa samples, this research seeks to provide valuable insights into the molecular heterogeneity of prostate carcinoma in Uganda, offering a critical reference point for future studies and potentially informing targeted therapeutic approaches tailored to this population.

2.0 Methodology

2.1 Study Design and Ethical Considerations

This study was embedded in a laboratory-based cross-sectional study that was focusing at Foxp3 and p53 Expression in Prostate Carcinoma and Their Correlation with Histological Grades among Archived Tissue Blocks at

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Uganda Cancer Institute that attained its ethical approval from Mbarara University of Science and Technology-Research Ethics Committee (REC Number: MUST-2024-1714), and the administrative clearance/permission letter to use archived blocks was also obtained from the Uganda Cancer Institute administration. Given the nature of utilizing archived tissue samples, a waiver of informed consent was granted.

2.2 **Study Site**

The study was carried out at the UCI pathology laboratory, which serves as a national referral pathology laboratory in Uganda. UCI is a public healthcare center focusing on research, training, consulting, prevention, and cancer treatment across various oncology specializations. It is located at 0°20′29.0″ N, 32°34′40.0″ E in Kampala, Uganda. The Pathology Department at UCI comprises five pathologists, around eight technologists, and some residents, providing services to patients from all regions of Uganda and neighboring countries including Rwanda, Tanzania, Kenya, Southern Sudan, and the Democratic Republic of Congo. The laboratory used to receive around 300 prostate cancer biopsies annually, with histological diagnoses primarily based on H&E staining, as markers for prostate cancer were not routinely performed.

2.3 **Study Population and Sample Collection**

Archived formalin-fixed paraffin-embedded prostate carcinoma tissue blocks were retrieved from the pathology archives of the Uganda Cancer Institute. A total of 127 prostate cancer tissue blocks, collected between January 1, 2021, and December 2024, were included in the study.

2.4 **Sampling Procedure**

A purposive sampling strategy was employed to select archived prostate cancer blocks from the histopathology laboratory of UCI from January 2021 to December 2024. We managed to retrieve 127 blocks, with an initial goal of 116 as our minimum sample size. Histologically diagnosed prostate cancer blocks, documented in the Laboratory Information Management System registry, were traced and retrieved using their corresponding laboratory identifier numbers.

2.4.1 **Selection Criteria**

Inclusion Criteria: All formalin-fixed paraffin-embedded prostate carcinoma tissue blocks archived in the Histopathology Laboratory that were well-processed and not damaged were included in the study.

Exclusion Criteria: Tissue blocks that were damaged or missing essential demographic data like age and were excluded. Depleted due to multiple sectioning and those that didn't show tumour during H&E examination were also excluded

2.5 **Tissue Processing and Sectioning**

The retrieved FFPE blocks were fixed on a microtome chuck and sectioned at a thickness of 3 microns using a disposable microtome knife on a rotary microtome. Tissue sections were then floated onto a 40°C water bath (5°C less than the paraffin wax melting point) to straighten wrinkles and subsequently scooped perpendicularly onto well-

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labeled, positively charged glass slides. After sectioning, all slides (for H&E and immunohistochemistry) were air-dried at room temperature for one hour, followed by overnight incubation in an oven set at 52°C.

2.6 **Laboratory Procedures**

2.6.1 **Hematoxyline and Eosin Staining**

Tissue sections were dewaxed in two changes of Xylene for 5 minutes each. Rehydration was performed through a series of decreasing alcohol concentrations (100%, 90%, 70%, 50%) to water, with 15 dips in each container. Harris's hematoxylin stain, a progressive basic dye, was then applied to stain acidic elements (nuclei) blue (15). Sections were subsequently washed, blued for 5 minutes, and counterstained with Eosin, an acidic dye that stains basic cellular elements (cytoplasm) pink.

2.6.2 Immunohistochemistry Staining

IHC staining was performed on charged slides. Sections were deparaffinized in two changes of xylene (3 minutes each), then rehydrated through descending grades of alcohol (100%, 95%, 90%, 80%) to distilled water.

Antigen Retrieval: Heat-induced antigen retrieval was performed by placing the sections in a citrate retrieval buffer within the working chamber of a Bio SB Tinto Retriever pressure cooker. The pressure cooker was set to high pressure (110°C) for 15 minutes. After the cycle, the pressure was released, and the lid removed.

Primary Antibodies: Primary antibodies were sourced from Abcam and used at a dilution of 1:300. Foxp3: Mouse monoclonal, Clone 236A/E7 and P53: Rabbit monoclonal, Clone AB1101 were used. For detection system**,** The UltraView Universal DAB Detection Kit was used for visualization, with DAB (3,3'-Diaminobenzidine) chromogen as the substrate. Sections were counterstained with hematoxylin and eosin for microscopic analysis.

Controls: Positive Controls, we used Tonsil tissue for Foxp3 and gastric carcinoma tissue for P53, both known to exhibit expression of the respective markers. For negative Controls, a normal colon tissue for Foxp3 and normal liver tissue for P53, where expression was expected to be absent were used

2.6.3 **Tissue Mounting**

Tissue sections were mounted by covering the stained sections with a cover glass using DPX mounting medium.

2.7 **IHC Staining Interpretation and Scoring**

IHC stained slides were independently evaluated by the principal investigator and two independent pathologists. A semi-quantitative grading system for Foxp33 and p53 expression was adopted, based on criteria similar to Wang et al. (16, 17). The intensity of cells showing positive expression was graded using a x10 objective. Staining was assessed in the nucleus for both Foxp3 and p53.

Scoring Criteria: Negative (for Foxp3: None): No positive reaction, weakly expressed (+): Less than 25% of cells stained positive, moderately expressed (++): 26%-50% of cells expressed positivity. Strongly expressed (+++): Greater than 50% expression.

The scoring criteria were applied uniformly for both Foxp3 and p53 expression.

Inter-observer Variability: To ensure the reliability and reproducibility of the scoring, 10% of the slides were

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reviewed by an external pathologist, who confirmed 100% agreement with the results of the internal observers/ pathologists.

2.8 **Quality Control**

Quality control measures implemented in our study included:

- Inter-observer agreement: Slide sections were read by the principal investigator and two independent pathologists, with an additional 10% of slides reviewed by external pathologists who demonstrated 100% agreement with the initial scoring.
- Confirmation of tissue sections was performed by the PI and a pathologist before being subjected to IHC.
- In-house quality controls were utilized for IHC antibodies.
- Freshly prepared IHC reagents were used.
- All procedures were performed according to standard operating procedures.
- Correct labeling was ensured at every level of the process.
- The microtome and surrounding area were thoroughly cleaned before sectioning.

2.9 **Statistical Analysis**

Data collected was entered into an Excel spreadsheet, cleaned, and then imported into STATA statistical software, version 17.0. Descriptive statistics were used to characterize the specimens, with percentages and frequencies for categorical variables, and mean and standard deviation for continuous variables. Tables and figures were employed for data representation. The expression of Foxp3 and p53 among prostate carcinoma cases was categorized as Weak, Moderate, and Strong, and presented as percentages.

2.11 **Dissemination of Results**

The study results were shared with the UCI administration, the MLS department, the MUST library, and the MUST Annual Research conference. A manuscript will also be prepared and submitted for publication.

3. Results

3.1 Demographic and Clinical Characteristics

The demographic and clinical characteristics of the 127 prostate cancer patients included in the study are summarized in Table 1. The median age of the patients was 72 years. The majority of patients were in the 71-80 age group (35.4%), closely followed by the 61-70 years group (33.9%). A significant proportion of patients (53.5%) did not have documented PSA values. Among those with recorded PSA levels, 39.4% had values greater than 20ng/ml.

Table 1: Prostate cancer Characteristics of Prostate cancer cases at Uganda Cancer Institute.

Variable	**Frequency (Percentage, %) n=127**
Age(years): Median (IQR)	72 (65-78)
Age groups (Years)	
≤60	19 (15.0%)

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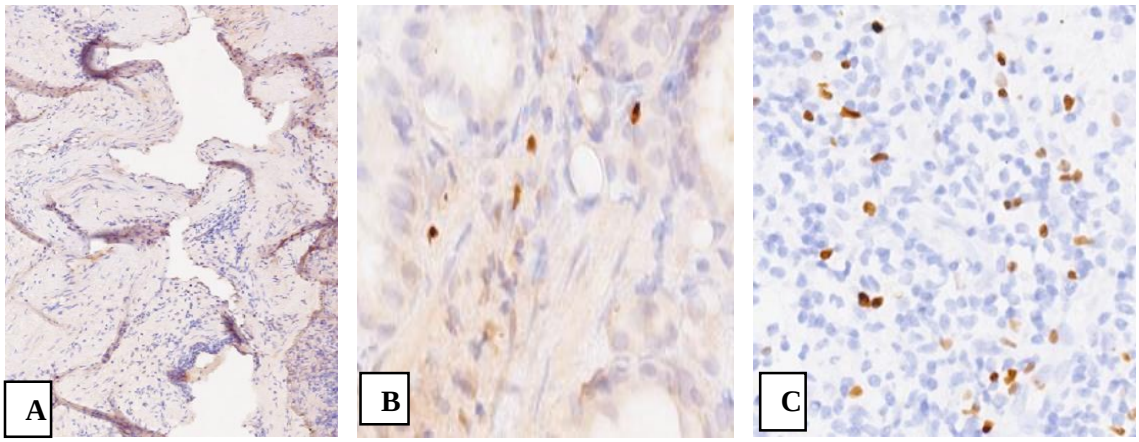
61-70	43 (33.9%)
71-80	45 (35.4%)
>80	20 (15.7%)
PSA values (ng/ml)	
None	68 (53.5%)
<10	1 (0.8%)
10-20	8 (6.3%)
>20	50 (39.4%)

2.2 Foxp3 Staining Intensity

The immunohistochemical staining intensity (Figure 1) for Foxp3 in the 127 archived prostate tissue blocks is presented in a pie chart (Figure 2). A substantial majority of the samples, 104 out of 127 (82%), exhibited no FOXP3 staining. Moderate Foxp3 staining was observed in 8 cases (6%), while 15 cases (12%) showed strong FOXP3 staining.

FIGURE 1: Micrographs showing different staining intensity of Foxp3



***Magnification**: X200, **A**-Slide with no staining intensity of Foxp3 antibody**, B**- slide with moderate staining intensity. **C**- Slide with strong Foxp3 staining intensity.*

***FIGURE 2: A pie-chart showing staining intensity percentages of FOXP3 among archived prostate tissue blocks at UCI** |*

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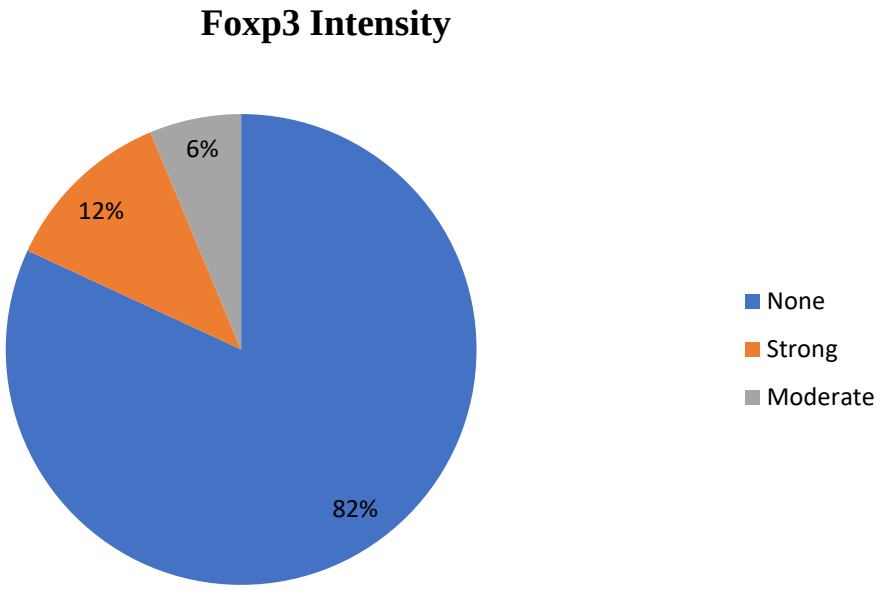
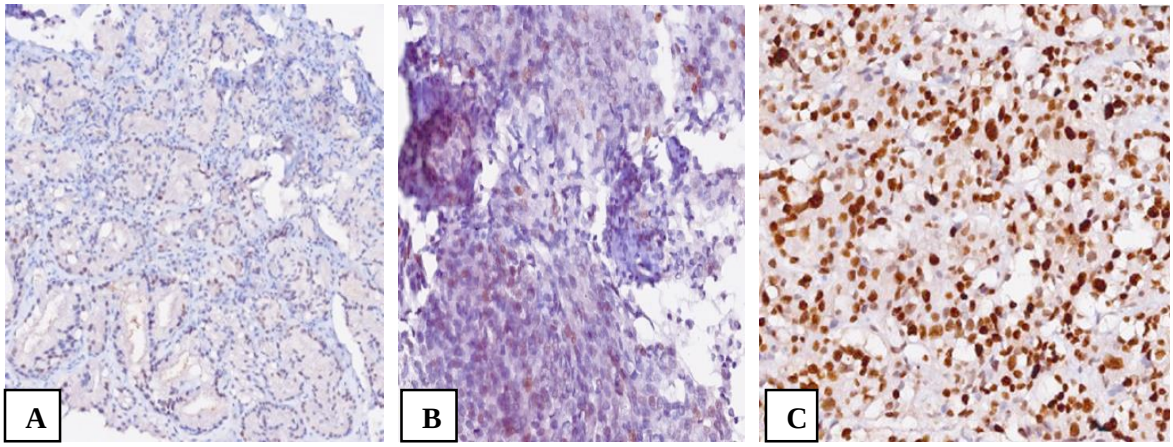


3.3 P53 Staining Intensity

The immunohistochemical staining intensity (*Figure 3)* for p53 in the same 127 archived prostate tissue blocks is detailed in *Figure 4* **(**pie chart). The most prevalent staining intensity pattern was moderate, observed in 77 cases (61%). Strong p53 staining was detected in 31 cases (24%), while weak staining was present in 19 cases (15.0%).

FIGURE 3: Micrographs showing different staining intensity of Foxp3



Magnification*: X200,* ***A****-Slide with weakly stained intensity of p53 antibody****, B****- slides with moderate staining intensity.* ***C****- Slide with strong p53 staining intensity.*

FIGURE 4: Pie – chart 2: indicating Staining intensity of p53 among archived prostate tissue blocks at UCI

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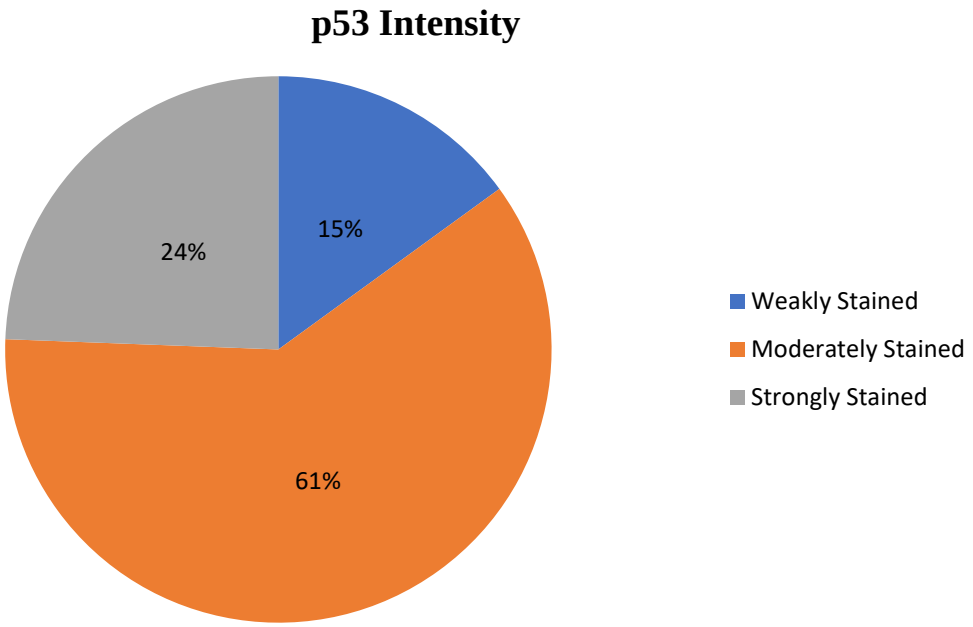

4. Discussion

This study provides valuable insights into the staining intensity of Foxp3 and p53 in prostate cancer tissue blocks from Ugandan patients, a population often underrepresented in molecular pathology research. Our findings reveal distinct distributions for both markers, underscoring the molecular heterogeneity of prostate cancer in this cohort.

The observation that a significant majority (82%) of prostate cancer samples showed no Foxp3 staining is noteworthy. Foxp3 is a key marker for regulatory T cells, which typically suppress anti-tumor immunity(18). While some studies have shown that a high staining density of intra-tumoral Foxp3 stained cells are linked to more advanced tumor stages, higher Ki67 labeling index, and poorer progression-free survival in some prostate cancer studies (8, 19), our results suggest a limited presence or activity of Foxp3 within the prostate tumor microenvironment in a large proportion of these Ugandan patients. This could imply a potentially less immune-suppressive environment regarding Foxp3 Tregs staining intensity in these specific cases, or it might point to other dominant mechanisms of immune evasion. Conversely, the presence of moderate (6%) to strong (12%) Foxp3 staining intensity in a subset of patients (total 18.1%) indicates that immune suppression via Foxp3- intensifying cells may still be a relevant factor in those specific individuals, potentially influencing disease progression or response to future immunotherapies. This highlights the importance of characterizing Foxp3 staining intensity across diverse populations, as the mechanistic roles of immune modulatory pathways in PCa may vary(10). Loss of Foxp3 staining intensity has been linked to PCa aggressiveness (20, 21).This is in agreement with our findings that showed absence of Foxp3 staining intensity in most of our prostate carcinoma tissue blocks.

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Regarding p53 expression, our study found varied staining intensities, with moderate staining being the most common (61%), followed by strong (24%) and weak (15.0%) staining intensities. This varied staining intensity of p53 is consistent with the understanding that altered p53 protein accumulation, often indicative of underlying p53 proteins' mutations, is a common feature in prostate cancer and can be linked to disease progression and an aggressive phenotype (1, 12). Strong p53 staining intensity typically reflects the nuclear accumulation of mutated p53 protein, which loses its tumor suppressor function and can even promote oncogenesis (22, 23). The high prevalence of moderate and strong staining intensity in our cohort (85%) suggests a substantial proportion of these Ugandan PCa cases may harbor p53 pathway alterations. This aligns with findings that p53 nuclear accumulation is associated with a unique tumor immune microenvironment (24, 25). While some studies have suggested that p53 nuclear accumulation might be less frequent in African-American tumors compared to European-American tumors (13, 26), our findings indicate a significant prevalence of altered p53 intensity, reinforcing the need for more granular studies in Ugandan (African prostate carcinoma patients). This is therefore that functional implications of these p53 intensity alterations, such as specific p53 mutations and Foxp3 , are critical to understand their role in tumor behavior(20, 27).

Overall, these findings underscore the molecular heterogeneity of prostate cancer in Ugandan patients, contributing valuable, specific data to a region often underrepresented in cancer research (28, 29). The observed patterns of Foxp3 and p53 staining intensities provide initial insights into the immune landscape and tumor suppressor pathways within these tumors. The high proportion of cases with altered p53 suggests a potentially aggressiveness of the disease.

Limitations: This was a nested study embedded in cross-sectional analysis using immunohistochemistry that had a limited scope tied to the parent study that resulted into dependency therefore future research should aim to corroborate these findings using more advanced molecular techniques, such as sequencing for p53 mutations and genetic analysis for other relevant markers like TMPRSS2-ERG fusion genes, which are common genetic alterations in prostate cancer (30). Correlating Foxp3 and p53 expression with detailed clinical outcomes, including disease progression, recurrence, and response to therapy, would further elucidate their prognostic and predictive utility in this population. Larger cohort studies with comprehensive clinical data are needed to fully characterize the molecular landscape of prostate cancer in Uganda and sub-Saharan Africa.

4. Conclusion and Recommendations

Foxp3 was lost in most of the prostate tissue blocks and a few showed a staining intensity 12% as strong, 6% moderate staining intensity and 82% didn't show a staining intensity that might be associated with aggressiveness of the disease while p53 staining intensity was abundant with moderate (61%), strongly (25%) and least being weakly stained with 15% indicating that p53 different staining intensities are observed in all prostate carcinoma blocks that is attributed to p53 mutations. Based on the findings of this study, we provide the following recommendations for different stakeholders:

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To the Clinicians and Pathology staff, Consider the potential implications of altered p53 staining intensity, indicated by moderate to strong staining, as a marker for potentially aggressive disease in prostate cancer patients in Uganda, encourage the routine collection and documentation of full clinical data, including PSA values, for all prostate cancer patients to facilitate comprehensive research and improved patient management and Integrate Foxp3 and p53 immunohistochemistry into routine pathology workflows where resources permit, to provide additional prognostic information among prostate cancer patients.

To the Researchers, Conduct further studies to correlate Fox3 and p53 expression with specific clinical outcomes, such as overall survival or response to different treatment modalities, in Ugandan prostate cancer patients, or Conduct a larger prospective study while Collaborating internationally to build larger cohorts and leverage advanced genomic technologies to characterize the unique molecular classification of prostate cancer in sub-Saharan Africa including Uganda.

To the **Policy Makers,** Invest in infrastructure and training to enhance pathology capabilities, including immunohistochemistry and genetic sequencing, within national and regional cancer treatment centers. Also fund local research efforts focused on understanding region-specific cancer biology to inform tailored diagnostic and therapeutic strategies, ultimately improving patient outcomes in Ugandan population and Africa in general.

Data Availability Statement

Data is available upon the request from the corresponding author

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Conflict of interest

No conflict of interest declared

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