

Can Clinico-Genomic Characterization with the Prolaris Test Reveal More Aggressive Prostate Cancer among Patients with Lower Gleason Grade Groups?

Hanan Goldberg

Gohanan@gmail.com

SUNY Upstate Medical University <https://orcid.org/0000-0002-5777-2138>

Sibtain Mustafa

<https://orcid.org/0009-0004-3295-5247>

Michael Basin

Alina Basnet

Seethram Bhat K.R.

Michael Daneshvar

UC Irvine

Thenappan Chandrasekar

Thomas Jefferson University <https://orcid.org/0000-0002-2173-5028>

Gennady Bratslavsky

SUNY Upstate Medical University

Article

Keywords:

Posted Date: September 25th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10911772/v1>

License:   This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: There is **NO** conflict of interest to disclose.

Abstract

INTRODUCTION

The prevalence of prostate cancer increases with age, and most patients over 65 are estimated to harbor the disease. Current guidelines emphasize risk stratification and life expectancy when determining next steps in management. In the growing elderly population, additional biomarkers are needed to predict outcomes beyond chronological age and to tailor treatment strategies. We evaluated age-related differences in clinico-genomic prognostic indices of aggressiveness in localized prostate cancer.

METHODS:

Clinical and genomic data for 107,471 patients from the Myriad Prolaris Prostate Cancer Database were obtained. Conventional and genomic prognostic indices were analyzed, including Prolaris treatment grouping (active surveillance (AS) vs single treatment vs multimodal treatment), cell cycle risk (CCR) scores, NCCN risk groups, tumor stage, and Gleason groups (GG). Multivariable logistic regression analyses were performed to evaluate the association of age (less than vs greater than 75), prostate-specific antigen level (PSA), tumor stage (T1a-T1c vs T2a-T2c vs T3a), positive biopsy cores (less than vs greater than 50%), and GG (1–2 vs 3–5) with Prolaris treatment scores (AS and single treatment vs multimodal treatment).

RESULTS:

With increasing age, we observed a higher proportion of high GG and more aggressive Prolaris treatment grouping. Even within the same prostate cancer GG, the suggested treatment became more aggressive with age. On multivariable analysis, there was a statistically significant increase in Prolaris treatment scores with increasing age groups (60–65 – HR 1.15; 95% CI 1.04–1.28, 65–70 – HR 1.30; 95% CI 1.18–1.43, 70–75–1.39; 95% CI 1.26–1.54, 75–80–1.56; 95% CI 1.40–1.74, > 80 – HR 1.83; 95% CI 1.60–2.08). Additionally, higher PSA levels (HR 1.21; 95% CI 1.20–1.21), GG (HR 105.70; 95% CI 96.75–115.47), tumor stages (T2a-T2c – HR 1.27; 95% CI 1.17–1.39), T3a (HR 9.94; 95% CI 6.25–15.82), and number of positive biopsy cores (HR 4.02; 95% CI 3.79–4.27) were associated with more aggressive Prolaris treatment scores.

CONCLUSIONS:

Older men may harbor worse disease, as indicated by genomic risk models, compared to their younger counterparts. Using chronological age alone can lead to significant undertreatment in this population. Clinical-genomic characterization may enable better individualized treatment decisions.

Introduction:

Currently, the average male life expectancy in the United States is 76.1 years [1]. However, once a man reaches age 75, he may live an additional 10.6 years or more [2]. At the same time, prostate cancer (PCa) is one of the most common cancers in men, with its incidence increasing with age [3, 4]. More than half of new cases are diagnosed after age 65, and over 20% occur in men aged 75 or older [3, 4]. Despite this, guidelines have recommended against screening in this age group, primarily based on life expectancy and concerns about competing mortality risks [5].

The PIVOT trial showed no significant overall survival benefit for radical prostatectomy compared with observation in men with localized PCa, especially among older individuals and those with low-risk disease [6]. These results have led to a more conservative approach in elderly patients, who are less likely to receive curative-intent therapy due to worries about competing mortality risks. Consequently, treatment choices in this group are often based on chronological age alone rather than personalized assessments of tumor biology and life expectancy [5].

Recent population-based data suggest that this approach may not fully assess the oncologic risk in older men. Analyses of men aged ≥ 75 years with clinically localized T1c prostate cancer show that those who undergo definitive local therapy, either surgery or radiotherapy, have significantly better overall survival compared with observation or androgen-deprivation therapy alone [7]. Genomic biomarkers have become more widely used to distinguish indolent from biologically aggressive disease, aiming to spare low-risk patients from overtreatment while identifying those who might benefit from definitive therapy.

The Prolaris assay, which combines a cell-cycle progression signature with clinicopathologic features, has been added to the NCCN guidelines for PCa management [11]. The test measures the expression of 31 genes involved in cell cycle progression from biopsy samples to produce a Cell-Cycle Progression (CCP) score, which aids in predicting disease progression and guiding treatment decisions [8]. Prolaris is designed for men with localized PCa, especially those with low-risk or favorable intermediate-risk disease and a 10-year life expectancy [8, 9].

We aimed to assess whether molecular biomarkers could help elucidate the relationship between age and aggressive disease features in PCa, potentially guiding management strategies for older men. In this study, we analyzed age-related differences in clinical-genomic prognostic indices of tumor aggressiveness in localized PCa.

Methods:

We conducted a retrospective cohort study using the Myriad Prolaris Prostate Cancer Database, a prospectively maintained national commercial genomic testing registry. The database includes men with biopsy-proven localized PCa who underwent Prolaris genomic testing between 2013 and 2023 according to the discretion of the patient's physician. All patient records were deidentified in accordance with

HIPAA guidelines prior to analysis. The study was exempt from institutional review board approval because it used de-identified data.

Eligible patients had histologically confirmed prostate adenocarcinoma with localized disease at diagnosis (clinical stage T1–T3a, N0, M0), available Prolaris results including Cell Cycle Progression and Cell Cycle Risk scores, and clinical variables such as age at diagnosis, prostate-specific antigen (PSA) level, Gleason grade group, clinical T stage, and biopsy core data. Patients missing data for any of these core variables were excluded from the primary analysis but included in sensitivity analyses to evaluate the effect of missing data.

The Prolaris assay is a validated gene expression test of cell cycle progression performed on formalin-fixed paraffin-embedded prostate biopsy tissue. The test measures the expression of 31 genes involved in cell cycle progression and 15 housekeeping genes using quantitative reverse transcription PCR (RT-qPCR). The CCP score indicates the average expression of the 31 cell cycle progression genes normalized to the housekeeping genes. The CCR score is a combined measure that integrates the CCP score with standard clinicopathologic factors such as PSA, Gleason grade group, and clinical T stage to provide a personalized risk assessment. Based on the CCR score, patients are classified into treatment recommendation groups: active surveillance (AS), single treatment (ST), or multimodal treatment (MT). These groupings are based on validated algorithms that incorporate both genomic and clinical risk factors to help guide management decisions in men with localized prostate cancer who have an estimated life expectancy of at least 10 years.

Clinical variables collected included age at diagnosis, categorized as 60, 60–64, 65–69, 70–74, 75–79, and ≥ 80 years; PSA level at diagnosis (ng/mL), analyzed as a continuous variable and categorized as 10, 10–20, and > 20 ng/mL; Gleason grade group (1–5 according to the International Society of Urological Pathology 2014 modified Gleason grading system); clinical T stage (T1, T2, T3a); and percentage of positive biopsy cores, categorized as $\leq 50\%$ or $> 50\%$. Patients were stratified into National Comprehensive Cancer Network (NCCN) risk groups (very low, low, favorable intermediate, unfavorable intermediate, high, and very high) based on established criteria that incorporate PSA, Gleason grade group, clinical stage, and biopsy core involvement.

The primary endpoint was the association between age and Prolaris treatment grouping (AS, ST, or MT). Secondary endpoints included associations between age and Cell Cycle Risk score, NCCN risk group, and Gleason grade distribution.

Baseline characteristics were summarized using descriptive statistics. Continuous variables were reported as median and interquartile range (IQR) or mean and standard deviation (SD), depending on their distribution. Categorical variables were presented as frequencies and percentages. Comparisons across age groups were made using the Kruskal-Wallis test for continuous variables and the chi-square test for categorical variables. Multivariable logistic regression models assessed associations between various factors and the Prolaris treatment assignment (MT vs AS/ST). Covariates included age, PSA, Gleason grade group, clinical T stage, and biopsy positivity $> 50\%$. Odds ratios (OR) with 95% confidence

intervals (CI) were reported. Statistical significance was set at $p < 0.05$. Sensitivity analyses were conducted, excluding cases with missing data.

Results:

A total of 107,471 men with localized PCa underwent Prolaris genomic testing and were included in the analysis. Baseline characteristics stratified by age group are shown in Table 1. The median age at diagnosis was 66 years (IQR 61–72). As age increased, men presented with higher PSA levels, higher Gleason grade groups, and more advanced clinical stages, leading to a shift toward higher NCCN risk groups.

Table 1

Baseline characteristics of men undergoing Prolaris genomic testing for localized prostate cancer, stratified by age group

Characteristic	< 60	60–64	65–69	70–74	75–79	≥ 80
Number of patients, n (%)	21,590 (20.1)	22,064 (20.5)	26,972 (25.1)	20,914 (19.5)	11,323 (10.5)	4,608 (4.3)
Mean PSA, ng/mL (SD)	7.39 (7.19)	7.79 (7.56)	7.80 (7.18)	8.09 (7.28)	8.99 (8.49)	12.41 (11.97)
ISUP grade group, n (%)						
Grade 1	11,265 (52.2)	10,343 (46.9)	12,470 (47.2)	8,610 (41.2)	3,930 (34.7)	1,127 (24.5)
Grade 2	7,122 (33.0)	7,743 (35.1)	9,407 (34.9)	7,837 (37.5)	4,238 (37.4)	1,508 (32.7)
Grade 3	2,155 (10.0)	2,553 (11.6)	3,069 (11.4)	2,771 (13.2)	1,829 (16.2)	973 (21.1)
Grade 4	712 (3.3)	948 (4.3)	1,197 (4.4)	1,136 (5.4)	831 (7.3)	602 (13.1)
Grade 5	336 (1.6)	477 (2.2)	559 (2.1)	560 (2.7)	495 (4.4)	398 (8.6)
Clinical stage, n (%)						
T1	19,507 (90.4)	19,800 (89.7)	24,329 (90.2)	18,755 (89.7)	10,037 (88.6)	3,988 (86.6)
T2a	1,226 (5.7)	1,322 (6.0)	1,647 (6.1)	1,375 (6.6)	790 (7.0)	307 (6.7)
T2b/c	815 (3.8)	888 (8.0)	939 (3.4)	739 (3.5)	457 (4.1)	178 (6.0)
T3a	42 (0.2)	54 (0.2)	57 (0.2)	45 (0.2)	39 (0.3)	25 (0.5)
NCCN risk group, n (%)						
Very low/low	10,008 (46.4)	8,989 (40.7)	11,115 (41.2)	7,422 (35.5)	3,218 (28.4)	799 (17.3)
Favorable intermediate	5,140 (23.8)	5,903 (26.8)	7,515 (27.9)	6,196 (29.6)	3,256 (28.8)	1,028 (22.3)
Unfavorable intermediate	4,830 (22.4)	5,190 (23.5)	5,884 (21.8)	5,039 (24.1)	3,140 (27.7)	1,445 (31.4)
High/very high	1,612 (7.5)	1,982 (9.0)	2,458 (9.1)	2,257 (10.8)	1,709 (15.1)	1,336 (29.0)
Prolaris CCR treatment group, n (%)						

Characteristic	< 60	60–64	65–69	70–74	75–79	≥ 80
Active surveillance	13,719 (63.5)	12,687 (57.5)	15,265 (56.6)	10,634 (50.8)	4,754 (42.0)	1,241 (26.9)
Single modality	6,163 (28.5)	7,228 (32.8)	8,953 (33.2)	7,764 (37.1)	4,667 (41.2)	1,912 (41.5)
Multimodal	1,708 (7.9)	2,149 (9.7)	2,754 (10.2)	2,516 (12.0)	1,902 (16.8)	1,455 (31.6)

The distribution of Prolaris treatment recommendations changed notably with increasing age (Fig. 1). Among men under 60 years, 42% were recommended for Active Surveillance (AS), compared to only 21% of men aged 80 years or older. Conversely, the use of Multimodal Treatment (MT) increased gradually with age, from 11% in men under 60 to 34% in men 80 or older. This trend was consistent across Gleason grade groups, with older patients showing greater treatment intensification within each grade category. CCR scores increased with age and were highest in men 80 or older. Older men with Gleason grade groups 1–2 had significantly higher CCR scores, indicating age-related genomic aggressiveness that may not be captured by histopathology alone (Fig. 2).

In adjusted models, age was independently linked to higher Prolaris treatment scores, even after accounting for PSA, Gleason grade, tumor stage, and biopsy positivity. Compared to men under 60, those aged 60–65 had a 15% greater risk of being assigned to multimodal treatment. This risk increased steadily with age, with men over 80 having nearly double the likelihood of being recommended for multimodal therapy (OR 1.83, 95% CI 1.60–2.08; p-trend < 0.001) compared to men younger than 60 (Table 2).

Table 2
Multivariable logistic regression model assessing associations of
clinicopathologic factors with Prolaris treatment score

Variable	OR	95% CI	p-value
Age group (ref: <60)			
60–64	1.15	1.04–1.28	0.007
65–69	1.30	1.18–1.43	< 0.001
70–74	1.39	1.26–1.54	< 0.001
75–79	1.56	1.40–1.74	< 0.001
≥80	1.83	1.60–2.08	< 0.001
PSA level (ng/mL; log)	1.21	1.20–1.21	< 0.001
Tumor stage (ref: T1a-T1c)			
T2a-T2c	1.27	1.17–1.39	< 0.001
T3a	9.94	6.25–15.82	< 0.001
Gleason grade group (ref: 1–2)			
3–5	105.70	96.75–115.47	< 0.001
Positive biopsy cores (ref: ≤50%)			
>50%	4.02	3.79–4.27	< 0.001

Clinicopathologic variables also demonstrated strong associations. Each 10 ng/mL increase in PSA was associated with a 21% higher likelihood of an aggressive Prolaris assignment. Men with T2 disease had a modest but significant increase in risk compared with T1 (OR 1.27, 95% CI 1.17–1.39), while those with T3a tumors faced nearly a tenfold higher risk of multimodal classification. Gleason grade also showed a significant association: men with GG5 disease were over 100 times more likely to be categorized as multimodal than those with GG1 (OR 105.70, 95% CI 96.75–115.47).

The association between age and aggressive Prolaris scores persisted within each Gleason grade category, reinforcing the notion that older age may confer an additional biological risk, as reflected in the genomic profile.

Discussion:

Our study shows that increasing age in men with localized PCa may be independently associated with more aggressive disease features, seen not only in traditional clinical and pathological traits but also in Prolaris genomic risk scores. Older men had higher PSA levels, higher Gleason grade groups, and more advanced clinical stages, and they were more often treated with multiple therapies even after adjusting

for known risk factors. Importantly, this association persisted among men with lower-grade disease, indicating that chronological age may reflect a biological risk not fully captured by histopathology alone [12–16].

These findings align with a broader pattern observed in population-based cohorts, in which increasing age at diagnosis is associated with a higher likelihood of high-grade and high-risk PCa at presentation [28]. Similarly, SEER-based analyses indicate that men aged ≥ 75 years more often present with advanced disease and make up a disproportionate share of prostate cancer-specific deaths [13, 29].

Age-related differences are also evident in conservative management. Among men under active surveillance, older age is linked to a higher chance of grade reclassification at biopsy or radical prostatectomy [12, 30]. This suggests that tumors initially deemed indolent in older patients may carry more aggressive features not fully detected in initial sampling. Our findings of an increased CCR score among elderly males with Gleason Grade Group 1–2 tumors may suggest that the risk posed by such histopathology could underestimate the biological aggression among elderly patients, although the increase in scores may be due to non-tumorigenic alterations in gene expression related to age.

Beyond pathological features, age also affects survival outcomes. Analyses adjusting for grade and stage show that age at diagnosis independently affects prostate cancer-specific mortality, while competing-risk models highlight the interaction between tumor risk and age in determining cancer-specific versus other-cause mortality [15, 31]. Therefore, age may influence both the tumor's biological behavior and the balance of oncologic and other health risks.

Despite this increased biological risk, older men are less likely to receive definitive therapy. Age has been strongly associated with not receiving curative-intent treatment, even after accounting for tumor characteristics and life expectancy [16]. Population-based analyses confirm persistent age-related disparities in the use of surgery and radiotherapy [21, 22]. Although appropriately selected elderly men may derive significant oncologic benefit from definitive local treatment, treatment intensity often decreases with advancing age [32, 33].

As the population continues to age, the burden of PCa among older men will increase significantly [11]. Management decisions should therefore be based not only on chronological age but also on an assessment of biological risk and life expectancy [2, 5]. From a molecular perspective, aging is associated with the accumulation of chromosomal changes and an increased somatic mutational load in normal tissues. Broad genomic analyses show that mutation burden increases across tumor types with age [24–27].

Our results also follow the same trend. Prolaris scores were higher in older men, including those with low-grade disease, and age remained an independent determinant of a higher genomic risk category even after adjusting for clinicopathological characteristics. These results can be interpreted in two ways – either tumors not considered aggressive by traditional assessment show greater aggressiveness at the molecular level in older patients, or the higher scores reflect the age of the tissue. Using genomic

classifiers like Prolaris in clinical decision-making could lead to treatment intensification, better matching of treatment intensity to the tumor's underlying biology, and help prevent age-related undertreatment.

Previous studies examining novel biomarkers, such as Decipher, showed more aggressive disease features in elderly PCa patients with low-grade disease. In a multicenter study of over 25,000 patients, increasing age was independently associated with higher Decipher scores at both biopsy and radical prostatectomy after adjusting for conventional clinicopathologic factors, with the effect most pronounced in men with grade group 1–2 disease. [12]

It should be noted, however, that higher genomic scores in older men may not necessarily reflect more aggressive tumor biology but rather the well-documented age-related accumulation of somatic mutations in normal tissues [26, 27]. Whether this biological phenomenon influences the clinical interpretation of genomic classifier scores in elderly patients remains an open question and warrants further investigation.

Despite using a large, well-characterized, clinically validated commercial genomic database, this study has several important limitations. First, although the database is maintained prospectively, the retrospective study design introduces potential selection bias. Specifically, CCR testing may have been preferentially performed on patients with higher-risk clinical features, a bias that is likely amplified among older men referred for diagnostic biopsy. As a result, elderly patients included in this cohort may disproportionately represent biologically aggressive disease.

Second, the database lacks detailed clinical variables, treatment data, and long-term oncologic outcomes, limiting the ability to evaluate downstream management and preventing validation of CCR prognostic performance across age groups. Third, demographic information was incomplete, including race and family history, both of which are established prognostic factors and were not included in the analysis. These limitations mean we should interpret the association between increasing age and higher CCR scores cautiously and highlight the need for prospective studies with detailed clinical data and long-term follow-up.

Overall, advanced age was shown to be an independent predictor of Prolaris scores, even among men with favorable histopathology (Gleason grade groups 1–2). This could reflect unmeasured genomic risk not captured by histopathology, or it could be due to aging-related changes in gene expression that are not specific to tumor aggression. Given the retrospective nature of our analysis, we cannot make this distinction. However, our results make it clear that genomic classifier scores obtained in older men must account for this unresolved biological issue, and chronological age should not be used to exclude these men from diagnostic assessment and risk stratification when they have significant life expectancy. Using genomic risk stratification with Prolaris may help improve risk assessment in select older patients, though it remains unclear how much age-related increases in genomic risk translate into meaningful clinical benefits from definitive treatment.

References

1. Siegel RL, Miller KD, Fuchs HE, Jemal A. Cancer statistics, 2024. *CA Cancer J Clin*. 2024.
2. Droz JP, et al. Management of prostate cancer in older men: ESMO–SIOG guidelines. *Lancet Oncol*. 2014.
3. Rawla P. Epidemiology of prostate cancer. *World J Oncol*. 2019.
4. Siegel RL, Miller KD, Jemal A. Cancer statistics for men by age. *CA Cancer J Clin*. 2020.
5. Mohler JL, et al. NCCN Guidelines: Prostate Cancer. *J Natl Compr Canc Netw*. 2023.
6. Wilt TJ, Brawer MK, Jones KM, et al. Radical prostatectomy versus observation for localized prostate cancer. *N Engl J Med*. 2012;367:203–213.
7. Gleicher S, et al. Management of localized T1c prostate cancer among men 75 years and older: A National Cancer Database study. *Clin Genitourin Cancer*. 2023.
8. Jones CU, et al. Radiotherapy and short-term androgen deprivation for localized prostate cancer (RTOG 9408). *N Engl J Med*. 2011;365:107–118.
9. Hamdy FC, et al. Ten-year outcomes after monitoring, surgery, or radiotherapy for localized prostate cancer (ProtecT trial). *N Engl J Med*. 2016;375:1415–1424.
10. Cuzick J, et al. Prognostic value of a cell cycle progression signature in prostate cancer. *Lancet Oncol*. 2011;12:245–255.
11. Shore ND, et al. The clinical utility of genomic classifiers in prostate cancer. *Prostate Cancer Prostatic Dis*. 2020;23:77–91.
12. Goldberg H, et al. Clinical-genomic characterization unveils more aggressive disease features in elderly prostate cancer patients with low-grade disease. *Eur Urol Focus*. 2021;7(4):797–806.
13. Smith BD, et al. Future of cancer incidence in the United States: burdens upon an aging, changing nation. *J Clin Oncol*. 2009;27:2758–2765.
14. Druskin SC, et al. Older age predicts biopsy and radical prostatectomy grade reclassification to aggressive prostate cancer in men on active surveillance. *J Urol*. 2019;201:98–104.
15. Scosyrev E, et al. Prostate cancer in the elderly: frequency of advanced disease at presentation and disease-specific mortality. *Cancer*. 2012;118:3062–3070.
16. Daskivich TJ, et al. Prediction of long-term other-cause mortality in men with early-stage prostate cancer. *Urology*. 2015;85:92–100.
17. Gandaglia G, et al. The effect of age at diagnosis on prostate cancer mortality: a grade-for-grade and stage-for-stage analysis. *Eur J Surg Oncol*. 2014;40:1706–1715.
18. de Camargo Cancela M, et al. Age remains the major predictor of curative treatment non-receipt for localized prostate cancer. *Br J Cancer*. 2013;109:272–279.
19. Daskivich TJ, et al. Questioning the 10-year life expectancy rule for high-grade prostate cancer. *Urology*. 2016;93:68–76.

20. Bandini M, et al. Radical prostatectomy or radiotherapy reduce prostate cancer mortality in elderly patients: population-based propensity score adjusted analysis. *World J Urol.* 2018;36:7–13.
21. Yan Y, et al. Primary treatment choices for men with clinically localized prostate carcinoma detected by screening. *Cancer.* 2000;88:1122–1130.
22. Abdollah F, et al. Competing-risks mortality after radiotherapy vs observation for localized prostate cancer. *Int J Radiat Oncol Biol Phys.* 2012;84:95–103.
23. Konety BR, et al. Patterns of primary and secondary therapy for prostate cancer in elderly men. *J Urol.* 2008;179:1797–1803.
24. Bratt O, et al. Undertreatment of men in their seventies with high-risk nonmetastatic prostate cancer. *Eur Urol.* 2015;68:53–58.
25. Dell'Oglio P, et al. Treatment trends and Medicare reimbursements for localized prostate cancer in elderly patients. *Can Urol Assoc J.* 2018;12:E338–E344.
26. Risques RA, Kennedy SR. Aging and the rise of somatic cancer-associated mutations in normal tissues. *PLoS Genet.* 2018;14:e1007108.
27. Blokzijl F, et al. Tissue-specific mutation accumulation in human adult stem cells during life. *Nature.* 2016;538:260–264.
28. Wahl DR, et al. Pan-cancer analysis of genomic sequencing among the elderly. *Int J Radiat Oncol Biol Phys.* 2017;98:726–732.
29. Milholland B, et al. Age-related somatic mutations in the cancer genome. *Oncotarget.* 2015;6:24627–24635.
30. Muralidhar V, et al. Association between older age and increasing Gleason score and risk classification in prostate cancer. *Cancer Epidemiol.* 2015.
31. Bernard B, et al. Age and metastatic prostate cancer outcomes. *Cancer.* 2020.
32. Wang X, et al. Association between age and upgrading during active surveillance for prostate cancer: systematic review and meta-analysis. 2021.
33. Daskivich TJ, et al. Age, tumor risk, and competing mortality in prostate cancer. *JAMA.* 2013.
34. Wong YN, et al. Survival associated with treatment vs observation in elderly men with localized prostate cancer. *JAMA.* 2006.
35. Bandini M, et al. Radical prostatectomy or radiotherapy reduce prostate cancer mortality in elderly patients: propensity score analysis. *World J Urol.* 2018.

Figures

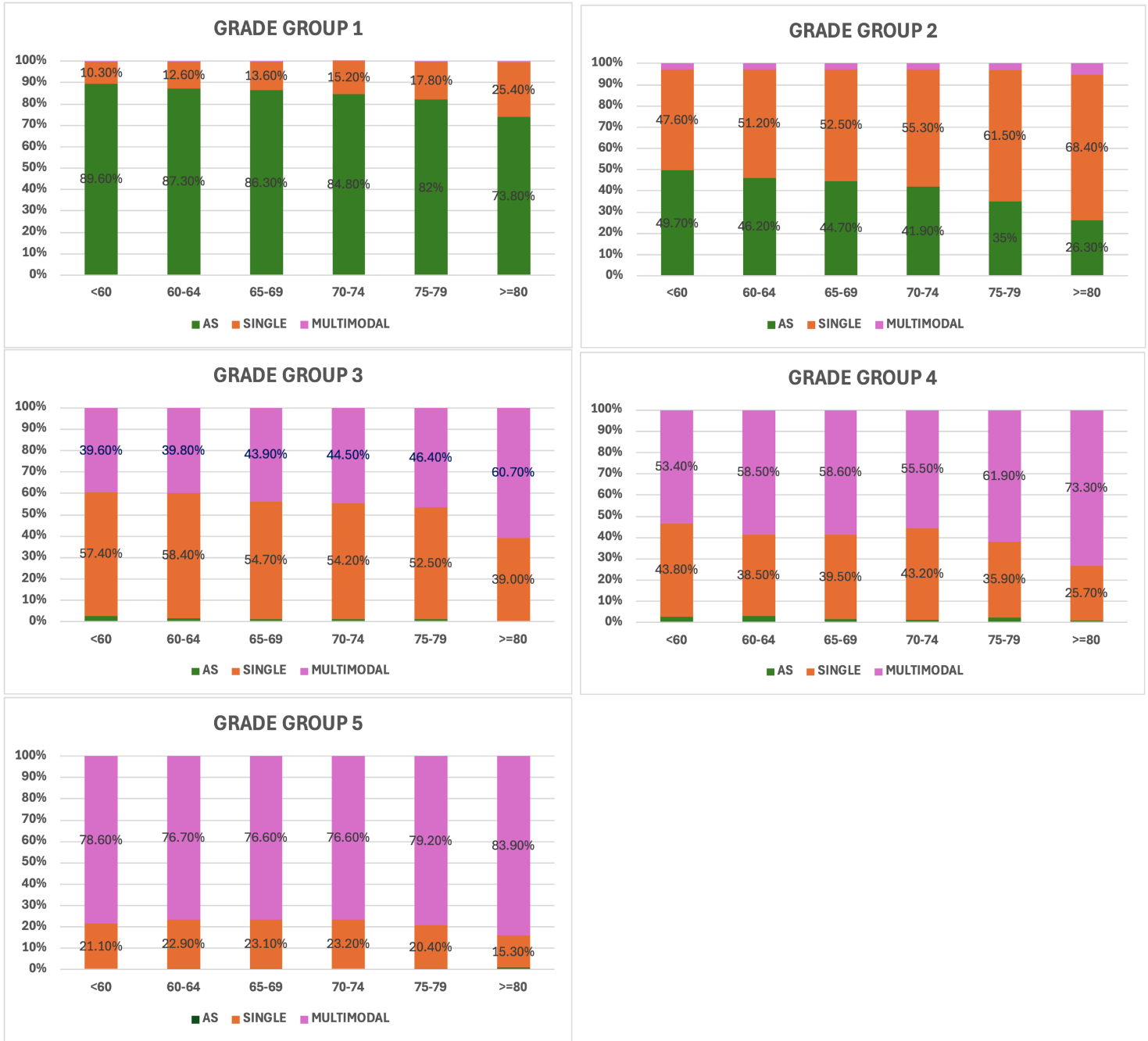


Figure 1

Prolaris treatment recommendation (active surveillance, single modality, or multimodal treatment) by Gleason grade group and age group.

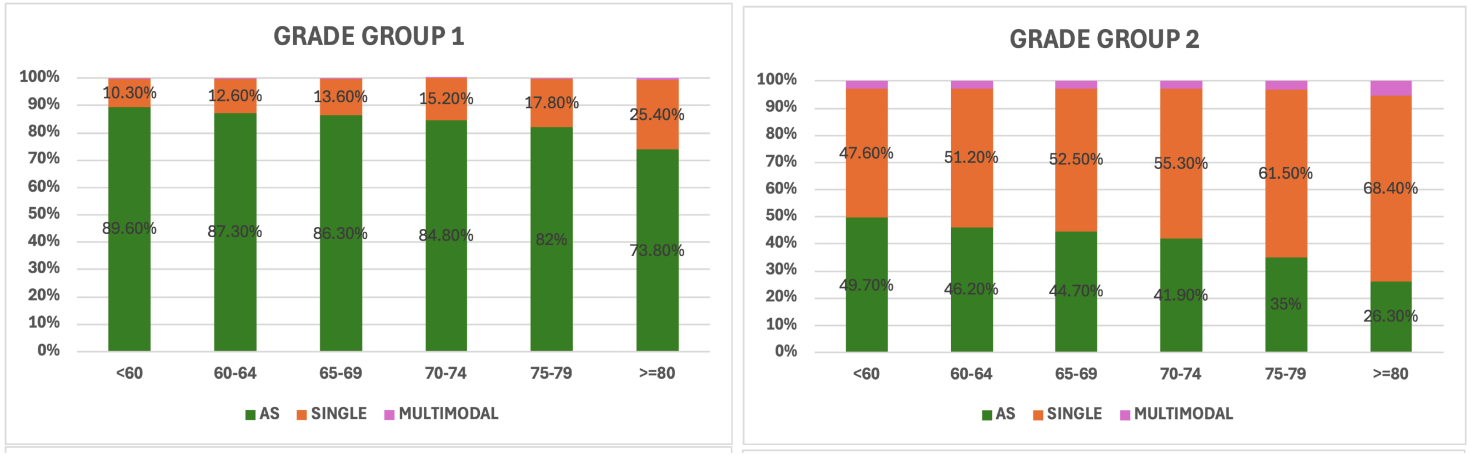


Figure 2

Close-up of Prolaris treatment recommendation by age among men with Gleason grade groups 1 and 2.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Table1BaselineCharacteristics.xlsx](#)
- [Table2MultivariableModel.xlsx](#)