

Smoking Habits, Physical Activity and BMI as Predictors of Cognitive and Affective Function in Patients with Prostate Cancer

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Objective: To assess modifiable risk factors for prostate cancer —smoking, physical activity, and obesity—in PC survivors, and their relationship with cognitive and affective functioning.

Methods: The study included 118 prostate cancer survivors. Clinical and psychological data were collected. Participants completed questionnaires on health risk behaviors. Cognitive functions were assessed using the Neurotest battery; anxiety and depression were measured via the Hospital Anxiety and Depression Scale. Statistical analyses included correlations and between-group comparisons (with post hoc tests).

Results: Smokers recalled fewer words in the Verbal Memory Test. Current smokers also performed worse on the second Verbal Memory Test trial ($p=0.035$) and had slower response times in the Visuospatial Memory Test ($p=0.013$). Obese individuals had slower Go/No-Go reaction times compared to non-obese participants ($p=0.02$). No other cognitive or affective differences were found.

Conclusion: Our preliminary findings suggest that smoking may impair memory in prostate cancer survivors. The relationship between physical activity, obesity, and cognitive and affective outcomes appears more complex and warrants further investigation.

Keywords: prostate cancer, cognitive function, smoking, physical activity, BMI, obesity

Introduction

Prostate cancer (PC) is the second most common malignancy among men worldwide and the fifth leading cause of cancer-related death.¹ Established non-modifiable risk factors include age (the most significant), ethnicity, and hereditary predisposition.² Modifiable risk factors comprise smoking, physical inactivity, diet, and obesity.^{2–4} Other risk factors are also being investigated, such as vitamin D supplementation, although their role has not been conclusively established in research.⁵

Health-risk behaviors may not only increase the likelihood of developing cancer but also influence disease trajectory, including treatment tolerance, risk of complications, and prognosis.³ These behaviors have been shown to affect sexual functioning and cardiovascular fitness, although no association with urinary incontinence has been demonstrated to date.^{6,7}

While numerous studies have investigated the influence of health-risk behaviors on PC outcomes,^{2,8} few have explored their association with mental functioning. PC survivors frequently experience persistent depressive and anxiety symptoms, as well as cognitive impairment,⁹ which may compromise treatment efficacy and overall quality of life.¹⁰ Such psychological and cognitive factors are crucial for treatment adherence and effective cooperation with healthcare providers, thereby influencing clinical outcomes.¹¹ Therefore, we aimed to investigate the associations between smoking, physical activity, and obesity with cognitive and affective functioning in PC survivors.

Smoking

Although the link between smoking and various cancers, including urological malignancies, is well established,^{12–14} the association between smoking and PC incidence remains controversial, with studies reporting positive, null, or even inverse relationships.^{15–18} Nonetheless, smoking adversely affects disease progression: smokers exhibit poorer survival, higher PC-specific mortality, and increased recurrence rates compared with non-smokers.^{3,19,20}

In the general population, smoking significantly increases the risk of cognitive decline,²¹ as well as anxiety and depressive symptoms.²² Research suggests that smoking alters neurotransmitter systems—such as dopamine, norepinephrine, and serotonin—which in turn affect cognitive abilities including learning, memory, attention, and executive functions.²¹

In their review of 148 studies, Fluharty et al found that most demonstrated associations between smoking status and depressive or anxiety symptoms, although the direction of this relationship remained unclear.²³ Some researchers propose a dual mechanism: smoking may contribute to subsequent depression or anxiety, whereas heightened anxiety sensitivity may promote smoking behavior.^{23,24}

Physical Activity

Physical activity improves PC treatment outcomes, reduces recurrence risk,^{25–27} and enhances quality of life.²⁸ Evidence also suggests that exercise may enhance cognitive functioning in men undergoing hormone therapy.^{29,30} Cognitive impairment among PC patients appears more pronounced in those receiving adjuvant therapy.⁹ In men receiving androgen deprivation therapy (ADT), Gaynor et al observed significant positive correlations between exercise levels and cognitive performance across multiple domains, including attention, memory, and executive function.²⁹

Physical activity may also help prevent or alleviate anxiety³¹ and depressive symptoms.³² Studies on the relationship between physical activity and affective symptoms have been conducted in both clinical and non-clinical populations,^{33,34} however, evidence in the PC population remains limited.

Obesity

Obesity is a well-established risk factor for multiple cancers.³⁵ However, the relationship between obesity, PC risk, and cognitive or affective functioning remains complex and inconclusive.^{36,37} Among men with PC, obesity has been associated with accelerated disease progression and increased mortality—each 5 kg/m² rise in body mass index (BMI) corresponds to a 49% increase in PC-specific mortality.^{36,38} Research also shows a linear correlation between BMI and prostate volume.³⁹ Obesity in midlife has been linked to cognitive impairment, whereas in later life, it may exert a protective effect.⁴⁰

Excess weight, even below the obesity threshold, increases cancer risk, particularly when accompanied by metabolic complications.⁴¹ Body weight also strongly influences PC progression: it has been associated with more aggressive and higher-grade disease, earlier progression, metastasis development, and an elevated risk of recurrence, as well as higher all-cause and PC-specific mortality.^{36,42}

Several mechanisms have been proposed to explain the link between obesity and poorer PC outcomes, including chronic low-grade inflammation, activation of inflammatory cytokines, oxidative stress, adipokine signaling dysregulation, adiponectin deficiency, hormonal imbalances, and alterations in hormone and insulin levels.^{43–45} These same mechanisms are also implicated in cognitive impairment.⁴⁶ Therefore, it is plausible that obesity may correlate with more severe cognitive deficits. This hypothesis is supported by findings from Miller and Spencer⁴⁷ and Wu et al,⁴⁸ although their studies did not include PC patients.

Anxiety and depression frequently accompany obesity. Metabolic changes associated with excess weight, along with body image concerns, increase vulnerability to these affective disorders.⁴⁹ However, this relationship is bidirectional: anxiety and depression may impair dietary control and adherence to lifestyle modifications aimed at weight reduction.⁵⁰

Despite the growing evidence on these links, few studies have examined cognitive outcomes among PC survivors after radical prostatectomy in relation to health-risk behaviors. This study therefore aims to evaluate the associations of smoking, physical activity, and obesity with cognitive and affective functioning in this population.

Materials and Methods

The study enrolled 118 patients with histopathologically confirmed prostate adenocarcinoma who had undergone radical prostatectomy, on average 1.5 years prior to assessment. All participants were Caucasian men with a mean age of 66 years. They were treated at the Urological Outpatient Department of Jan Biziel University Hospital No. 2 in Bydgoszcz, Poland.

Participants received detailed information about the study's objectives and voluntarily consented to participate. Prior to enrollment, written informed consent was obtained from all subjects. Eligibility criteria included the ability to comprehend the study objectives, willingness to participate, and absence of cognitive or physical incapacitation. Individuals diagnosed with dementia, psychiatric disorders, or unstable medical conditions were excluded. The study adhered to the principles of the Declaration of Helsinki and received approval from the Bioethical Commission of the Nicolaus Copernicus University, Collegium Medicum in Bydgoszcz (approval No. 476/2017).

Participants completed a detailed questionnaire on health-risk behaviors, including smoking (number of cigarettes smoked and smoking duration), physical activity (frequency of intentional activity per week), education (years of schooling), and anthropometric parameters (weight, height, BMI). Additionally, prostate cancer-related data were collected through patient questionnaires and medical records, including cancer grade (Grade Group—classification according to the International Society of Urological Pathology⁵¹), treatment history, disease course, and comorbidities. Since some comorbidities were rare, we also use a count of comorbidities for statistical analysis.

In the next phase, cognitive function was assessed using the Neurotest computerized test battery (described below). Affective functioning was also evaluated using the Hospital Anxiety and Depression Scale (HADS) to assess the presence of depressive and anxiety symptoms.⁵²

The Neurotest battery comprises several computerized tasks assessing different domains of cognitive function. The following tests were used:

- Simple Reaction Time (SRT): Assesses attention, reaction speed, and response accuracy. Participants press a button as quickly as possible when a green circle appears on the screen.⁵³
- Go/No-Go Test: Evaluates cognitive inhibition, reaction time, and executive functions. The task consists of two stimuli: pressing a key when a green square appears (Go response) and withholding a response when a blue square appears (No-Go response). The number of correct and incorrect responses, as well as reaction times, are recorded.⁵⁴
- Verbal Memory Test (VMT): Assesses short-term verbal memory and learning. The researcher reads a list of 10 words, and participants are asked to recall as many as possible in any order (VM1). The procedure is repeated five times (VM2–VM5), with recall tested after each trial.⁵⁵
- Verbal Memory Delayed Test (VMDT): Evaluates delayed recall, administered 20 minutes after completion of the VMT. Participants are asked to recall the previously presented words without prompting.
- Visual Working Memory Test (VWMT): Measures visuospatial working memory. Seven playing cards are shown sequentially in different screen positions. In the second stage, the same cards reappear, and the participant must indicate their original locations. Reaction times and the number of correct responses are recorded.⁵⁵
- Hospital Anxiety and Depression Scale (HADS): Assesses anxiety (HADS-A) and depressive (HADS-D) symptom severity. The scale consists of 14 items—seven for anxiety and seven for depression—rated on a 4-point scale. Respondents select the statement that best describes their current state.⁵²

Statistical analyses were performed using STATISTICA 13.1. As the data did not meet normal distribution criteria, nonparametric tests were applied. Correlations were evaluated using Spearman's rank correlation test. Differences between two groups were assessed with the Mann–Whitney *U*-test, while comparisons among multiple groups were conducted using ANOVA. The Bonferroni correction was applied to analyses conducted using the Mann–Whitney *U*-test. Post hoc analyses for ANOVA results were performed using Fisher's Least Significant Difference (LSD) test ([Supplementary Tables](#)).

Results

Clinical and demographic characteristics of the study participants are summarized in Table 1. Analysis revealed differences in selected parameters across subgroups defined by smoking status, physical activity, and BMI. Age was excluded from further analysis when it did not represent a significant differentiating factor.

Initial analyses demonstrated that smokers performed significantly worse on the second ($p = 0.04$) and third ($p = 0.048$) trials of the VMT. Although performance on the fourth and fifth trials was also poorer among smokers, these differences only approached statistical significance. Smokers additionally exhibited longer reaction times in the VWMT, with the difference nearing statistical significance (Table 2).

Table 1 Demographic and Clinical Data

Parameter		All Group (n=118)
Age (y)		66,0 (60,0–70,0)
BMI (kg/m ²)		27,2 (25,6–29,7)
Education	Basic (n,%)	6 (5%)
	Vocational (n,%)	33 (28%)
	Secondary (n,%)	36 (30,5%)
	Higher (n,%)	43 (36,5%)
GRADE group	1 (n)	70 (59,3%)
	2 (n)	35 (29,7%)
	3 (n)	5 (4,2%)
	4 (n)	4 (3,4%)
	5 (n)	4 (3,4%)
GLEASON Score	1 (n)	2 (1,5%)
	5 (n)	70 (59,5%)
	6 (n)	38 (32%)
	7 (n)	4 (3,5%)
	8 (n)	4 (3,5%)
Time from surgery [m]		19,0 (12,0–39,0)
Hypertension		65 (55%)
Diabetes		19 (16%)
Myocardial infarction		10 (8,5%)
Stroke		7 (6%)
Additional treatment		24 (20,5%)
Radiotherapy		22 (18,5%)
Hormonotherapy		9 (7,5%)

(Continued)

Table 1 (Continued).

Parameter		All Group (n=118)
Physical activity	None (n,%)	40 (34%)
	1/week (n,%)	24 (20,5%)
	2-3/week (n,%)	22 (18,5%)
	>3/week (n,%)	32 (27%)
Smoking	Currently (n,%)	11 (9,5%)
	Former smoker (n,%)	41 (34,5%)
	Never smoke (n,%)	66 (56%)
BMI (kg/m ²)		27,2 (25,6–29,7)
Weight	Norma (n,%)	24 (20,5%)
	Overweight (n,%)	68 (57,5%)
	Obesity (n,%)	26 (22%)

Note: Data are shown as median (25th – 75th quartile) or number (%).

Abbreviations: Grade Group, Grade of cancer advancement according to the International Society of Urological Pathology classification; BMI, body mass index; M, months.

Table 2 Results of Cognitive Testing (Neurotest) and Affective Measures (HADS) Among Smokers and Non-Smokers

	Non-Smokers (n = 66)	Smokers (n = 52)	p
Age, y	66,0 (60,0–70,0)	67,0 (62,0–71,0)	0,49
SRT correct answers, n	25,0 (25,0–25,0)	25,0 (25,0–25,0)	0,86
SRT reaction time, ms	284,2 (234,6–328,1)	278,8 (236,5–334,3)	0,76
G/nG incorrectNoGo, n	5,0 (2,0–8,0)	6,0 (3,0–8,0)	0,74
G/nG correct Go, n	74,0 (73,0–75,0)	74,0 (72,0–75,0)	0,97
G/nG reaction time, ms	363,0 (312,5–405,9)	366,3 (338,4–423,3)	0,13
VM1	6,0 (4,0–7,0)	5,0 (4,0–6,0)	0,49
VM2	7,0 (7,0–8,0)	7,0 (6,0–8,0)	0,04
VM3	8,0 (7,0–9,0)	7,0 (6,0–8,0)	0,048
VM4	8,0 (7,0–10,0)	8,0 (6,0–9,0)	0,06
VM5	8,0 (7,0–10,0)	8,0 (6,0–9,0)	0,055
VMDT, n	6,0 (5,0–7,0)	5,0 (4,5–7,0)	0,70
VWMT, no answers	0,0 (0,0–0,0)	0,0 (0,0–1,0)	0,48
VWMT, incorrect time, ms	3410 (0–4516)	3655 (2454–5515)	0,49
VWMT, correct time, ms	2844 (2421–4054)	3476 (2541–4703)	0,053

(Continued)

Table 2 (Continued).

	Non-Smokers (n = 66)	Smokers (n = 52)	p
VWMT, n	6,0 (5,0–7,0)	5,0 (4,0–6,0)	0,65
HADS A	2,0 (1,0–5,0)	4,0 (1,0–7,0)	0,13
HADS D	2,0 (1,0–5,0)	3,0 (1,0–4,5)	0,93

Notes: Data are presented as medians with 25th and 75th percentiles. Statistically significant results are marked in bold.

Abbreviations: SRT, Simple Reaction Test; G/nG, Go/No-Go test (incorrect No-Go answers, correct Go answers, reaction time); VM1–5, Verbal Memory Test, trials 1–5; VMDT, Verbal Memory Delayed Test; VWMT, Visual Working Memory Test (number of no answers, incorrect answers time, correct answers time, number of correct answers); HADS, Hospital Anxiety and Depression Scale (A – anxiety subscale, D – depression subscale).

Further subgroup analysis classified participants as non-smokers, former smokers, or current smokers. Current smokers performed significantly worse on the second VMT trial and showed significantly slower reaction times in the VWMT ([Supplementary Table 1](#)).

Regarding physical activity, when participants were grouped as physically active or inactive, the active group demonstrated faster SRT reaction times, a result approaching statistical significance ([Table 3](#)). However, no significant differences were observed when the active group was further divided by activity frequency ([Supplementary Table 2](#)).

For obesity, patients with obesity showed significantly longer reaction times in the Go/No-Go test ($p = 0.02$) ([Table 4](#)). When overweight individuals were included in the analysis, the difference in reaction time was no longer statistically significant ([Supplementary Table 3](#)).

Table 3 Results of Cognitive Testing (Neurotest) and Affective Measures (HADS) in Study Subgroups with and Without Physical Activity (PA)

	No PA (n = 65)	PA (n = 53)	P
Age, y	66,0 (60,0–70,0)	66,0 (62,0–70,0)	0,89
SRT correct answers, n	25,0 (25,0–25,0)	25,0 (25,0–25,0)	0,91
SRT reaction time, ms	285,4 (249,12–328,1)	276,4 (231,4–330,7)	0,055
G/nG incorrectNoGo, n	5,0 (2,0–8,0)	6,0 (3,0–8,0)	0,74
G/nG correct Go, n	74,0 (73,0–75,0)	74,0 (72,0–75,0)	0,97
G/nG reaction time, ms	363,0 (312,5–405,9)	366,5 (345,0–424,5)	0,55
VM1	6,0 (5,0–7,0)	5,0 (4,0–6,0)	0,075
VM2	7,0 (7,0–8,0)	7,0 (6,0–8,0)	0,33
VM3	8,0 (7,0–9,0)	7,0 (6,0–8,0)	0,57
VM4	8,0 (7,0–10,0)	8,0 (6,0–9,0)	0,40
VM5	8,0 (7,0–10,0)	8,0 (6,0–9,0)	0,53
VMDT, n	6,0 (4,0–7,0)	5,0 (4,0–7,0)	0,46
VWMT, no answers	0,0 (0,0–0,0)	0,0 (0,0–1,0)	0,61
VWMT, incorrect time, ms	3477 (0–4516)	3655 (2454–5515)	0,30
VWMT, correct time, ms	3329 (2546–4282)	3476 (2541–4703)	0,16

(Continued)

Table 3 (Continued).

	No PA (n = 65)	PA (n = 53)	P
VWMT, n	6,0 (5,0–7,0)	4,0 (2,5–6,0)	0,93
HADS A	2,0 (1,0–5,0)	4,0 (1,0–7,0)	0,15
HADS D	2,0 (1,0–5,0)	3,0 (1,0–4,0)	0,28

Note: Data are presented as medians with 25th and 75th percentiles.

Abbreviations: SRT, Simple Reaction Test; G/nG, Go/No-Go test (incorrect No-Go answers, correct Go answers, reaction time); VM1–5, Verbal Memory Test, trials 1–5; VMDT, Verbal Memory Delayed Test; VWMT, Visual Working Memory Test (number of no answers, incorrect answers time, correct answers time, number of correct answers); HADS, Hospital Anxiety and Depression Scale (A – anxiety subscale; D – depression subscale).

Table 4 Results of Cognitive Testing (Neurotest) and Affective Measures (HADS) in Study Subgroups with and Without Obesity (PA)

	Non – Obesity (n=92)	Obesity (n=26)	P
Age, y	66,0 (59,5–70,0)	68,0 (63,0–71,0)	0,25
SRT correct answers, n	25,0 (25,0–25,0)	25,0 (25,0–25,0)	0,93
SRT reaction time, ms	282,6 (233,6–329,9)	283,8 (252,0–343,9)	0,57
G/nG incorrectNoGo, n	5,0 (2,0–8,0)	6,5 (3,0–8,0)	0,36
G/nG correct Go, n	74,0 (72,0–75,0)	74,0 (73,0–75,0)	0,62
G/nG reaction time, ms	360,2 (315,9–406,7)	396,8 (358,3–439,1)	0,02
VM1	5,0 (4,0–7,0)	5,5 (4,0–7,0)	0,56
VM2	7,0 (6,0–8,0)	8,0 (6,0–9,0)	0,17
VM3	7,0 (7,0–8,0)	8,0 (7,0–9,0)	0,35
VM4	8,0 (7,0–10,0)	8,0 (7,0–9,0)	0,31
VM5	8,0 (7,0–10,0)	7,5 (7,0–9,0)	0,35
VMDT, n	6,0 (5,0–7,0)	6,0 (5,0–7,0)	0,46
VWMT, no answers	0,0 (0,0–0,5)	0,0 (0,0–1,0)	0,91
VWMT, incorrect time, ms	3496 (0,0–4941)	3695 (0,0–5355)	0,51
VWMT, correct time, ms	3133 (2551–4712)	3211 (2355–4234)	0,99
VWMT, n	5,0 (4,0–6,5)	5,0 (2,0–6,0)	0,46
HADS A	3,0 (1,0–6,5)	3,0 (1,0–6,0)	0,59
HADS D	3,0 (1,0–5,0)	2,0 (1,0–4,0)	0,36

Notes: Data are presented as medians with 25th and 75th percentiles. Statistically significant results are marked in bold.

Abbreviations: SRT, Simple Reaction Test; G/nG, Go/No-Go test (incorrect No-Go answers, correct Go answers, reaction time); VM1–5, Verbal Memory Test, trials 1–5; VMDT, Verbal Memory Delayed Test; VWMT, Visual Working Memory Test (number of no answers, incorrect answers time, correct answers time, number of correct answers); HADS, Hospital Anxiety and Depression Scale (A – anxiety subscale, D – depression subscale).

Correlation analyses between age, clinical parameters (eg, BMI, pack-years, smoking duration, comorbidities), and cognitive or affective performance revealed no significant associations.

No statistically significant differences were found among subgroups regarding anxiety or depressive symptoms (Tables 2–4). Nevertheless, none of the observed significances were maintained after implementation of correction tests.

Discussion

Smoking

In our PC cohort, 11 participants were current smokers, 41 were former smokers, and 66 had never smoked (Table 1). Large-scale studies report smoking prevalence ranging from 20% to 60%, with 45–60% being former smokers.⁵⁶ In Poland, approximately 20% of men over the age of 65 smoke. The lower rate observed in our sample may reflect higher cessation rates following a cancer diagnosis. Notably, however, the majority of participants had a history of smoking, which may have contributed to disease development.

We first examined whether smokers and non-smokers differed in cognitive performance (Table 2). The VMT revealed reduced short-term verbal memory among smokers, who recalled fewer words in four of the five trials. While no group difference was observed in the first trial, subsequent trials demonstrated less efficient recall with repeated exposure in smokers (Table 2).

When participants were categorized as current, former, or never smokers, significant differences emerged in the VMT (VM2 trial) and the VWMT. Post hoc analyses indicated that these effects were partly driven by differences between current and former smokers, suggesting that smoking cessation may improve learning ability (Supplementary Table 1). Smokers also exhibited slower response times in the VWMT, whereas other reaction times were unaffected. This pattern suggests that verbal and visuospatial memory may be particularly susceptible to smoking-related damage.

The relationship between smoking and cognition is complex. Although smoking is generally associated with cognitive decline, some evidence points to transient cognitive enhancement—particularly in attention and memory domains.⁵⁷ Nicotine withdrawal can temporarily impair cognitive performance, whereas long-term cessation reduces the risk of cognitive decline.⁵⁸ Genetic studies by Elshafei et al demonstrate that smokers harbor more somatic mutations, which may influence both disease progression and cognitive function.⁵⁹

No significant differences in anxiety or depression were found between smokers and non-smokers (Table 2). Similarly, Kizilkan et al⁶⁰ reported no such differences in PC patients, though both studies involved relatively small cohorts. Other research indicates that depressive symptoms may be more prevalent among low-risk PC patients.⁶¹

Physical Activity

Although sedentary behavior has not been conclusively linked to PC risk, higher physical activity levels are associated with reduced PC-specific mortality.¹⁶ Physically active patients—particularly those with faster walking speed—also tend to exhibit superior cognitive performance.^{29,62}

In our study, 27% of respondents reported high physical activity (≥ 4 times/week), 19% reported moderate activity (2–3 times/week), 20.5% engaged in occasional activity, and 34% reported no physical activity (Table 1). This distribution is comparable to that observed in the general population.⁶³

Participants who did not engage in regular exercise demonstrated slower SRT responses, although the differences approached but did not reach statistical significance (Table 3). Despite previous findings linking exercise to improved executive functioning and cognitive control,^{29,64} we did not observe similar associations. Physically active and inactive individuals showed no significant differences in learning, memory, or executive function performance (Table 3).

Mundell et al reviewed the effects of exercise in PC patients receiving ADT—a treatment known to impair cognition (eg, executive function, visuomotor skills, attention, and verbal memory). Their findings indicate that exercise can mitigate these cognitive deficits.³⁰

Yang et al identified a strong association between handgrip strength and cognitive performance among cancer survivors, including PC patients. Interestingly, this association was linear in women but followed an inverted U-shaped curve in men.⁶⁵ Both aerobic and anaerobic exercise appear to support cognitive function, as confirmed by meta-analyses.⁶⁶

Anxiety and depressive symptoms did not differ significantly between physically active and inactive participants, despite prior evidence suggesting psychological benefits of exercise in PC patients.⁶⁷

Obesity

The global obesity epidemic contributes to the risk of multiple cancers and affects disease progression. In our cohort, 22% (26 participants) were classified as obese (BMI > 30 kg/m²), and 57.5% (68 participants) were overweight (BMI > 25 kg/m²), exceeding general population estimates.⁶⁸

We observed only one cognitive difference between obese and non-obese participants: non-obese individuals demonstrated faster reaction times in the Go/No-Go task (Table 4). Other tests revealed no significant differences, even when normal-weight participants were analyzed separately (Supplementary Table 3). This isolated finding is unlikely to be clinically meaningful but merits further investigation.

Although excess body weight is generally associated with cognitive decline,⁶⁹ findings are inconsistent. Xing et al (2022) reported an inverse relationship between obesity and cognition in patients with type 2 diabetes, suggesting a possible protective effect.⁷⁰

PC patients undergoing ADT often experience increased fat mass, loss of muscle, and sarcopenic obesity—factors that may contribute to cognitive impairment.^{71,72} However, this was not observed in our sample.

Vidal and Freedland have proposed that obesity's influence on PC progression may depend on tumor molecular characteristics, such as TMPRSS2–ERG fusion status.⁴⁵ Such molecular heterogeneity may also account for the inconsistent associations between obesity and cognition.

We found no significant differences in depression or anxiety between obese and non-obese participants (Table 4). The absence of such associations may reflect the small number of participants receiving adjuvant therapy, which is typically linked to worse mood outcomes.

Limitations

This study has several limitations. The primary limitation is the relatively small sample size, which may restrict the statistical power and generalizability of the findings, especially considering that statistical analyses were frequently conducted on subgroups. Another limitation lies in the use of unvalidated, self-reported measures to assess smoking status and physical activity, which may introduce response bias. Furthermore, the assessment of physical activity referred only to its frequency, without evaluating its intensity, which may also have contributed to the obtained results. In the context of examining the relationship between obesity and cognitive functioning, another limitation of our study is the lack of information regarding the use of weight-loss medications.

Conclusion

PC outcomes are influenced by health-risk behaviors such as smoking, physical inactivity, and obesity.⁷³ However, there remains insufficient evidence regarding their impact on cognitive and affective functioning among PC survivors. Our preliminary findings suggest that smoking may impair verbal memory, and to a lesser extent, visuospatial memory. The relationships among body weight, physical activity, and cognitive or affective symptoms are complex and warrant further investigation in larger, well-characterized cohorts.

Data Sharing Statement

Data available on request from the corresponding author due to ethical policy.

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