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# Obesity and positive surgical margins by anatomic location after radical prostatectomy: results from the Shared Equal Access Regional Cancer Hospital database

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## Abstract

**OBJECTIVES—**To determine if there is predilection for any specific anatomical location of positive surgical margins (PSMs) after radical prostatectomy (RP) for prostate cancer in obese men, as previous studies found that obesity was associated with an increased risk of PSMs.

**PATIENTS AND METHODS—**We analysed retrospectively 1434 men treated with RP between 1989 and 2007 within the Shared Equal Access Regional Cancer Hospital database. The association between increased body mass index (BMI) and overall and site-specific PSMs was assessed using multivariate logistic regression.

**RESULTS—**After adjusting for several preoperative clinical and pathological characteristics, a higher BMI was associated with an increased risk of PSMs both overall and at all specific anatomical locations (all $P \leq 0.007$). For mildly obese men, this risk was very similar across all anatomical sites (44–78% increased risk relative to men of normal weight). When BMI was coded as a continuous variable, the odds ratio for the risk of overall PSMs or at any specific locations



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**CONFLICT OF INTEREST**
None declared.

was nearly identical at 1.05–1.06. Among men with a BMI of ≥35 kg/m$^2$, there was more variation, with the highest excess risk of PSMs at the bladder neck and apex.

**CONCLUSIONS—**Obesity was associated with an increased risk of overall PSMs and at all anatomical locations. Although the excess risk of PSMs was similar across all anatomical locations, there was a suggestion of a higher risk of apical margins among the most obese men, which if validated, further supports the importance of the apical dissection in all men and suggests added difficulty in obese patients.

### Keywords

prostate cancer; radical prostatectomy; obesity; margins

## INTRODUCTION

The object of any oncological procedure is complete removal of the cancer. As such, a positive surgical margin (PSM) is often regarded as resulting from a less than ideal operation. Indeed, many studies showed that a PSM after radical prostatectomy (RP) is associated with an increased risk of prostate cancer recurrence [1–3]. PSMs might result from extension of tumour beyond the planned limits for resection (i.e. advanced disease) and/or poor technique (i.e. iatrogenic).

Technical challenges can result from many causes, e.g. poor patient anatomy compounded by the challenges of operating within the narrow restricting confines of the prostatic fossa, the presence of scarring/inflammation resulting in difficult dissection, poorly controlled bleeding limiting visualization, and patient body habitus. One of the commonly used measures of habitus is the body mass index (BMI). While not perfect in estimating abdominal or pelvic adiposity, BMI is an easily available clinical characteristic which correlates sufficiently well with adiposity in men to warrant its use as a surrogate [4]. Indeed, previous studies showed a greater risk of PSM [5,6] and capsular incision [7] among obese men. However, whether this applies equally to all anatomical sites of PSMs is unknown. Specifically, is there an anatomical site that is more likely to be positive in an obese man? To address this, we examined the risk of PSMs both overall and at specific anatomical locations in the Shared Equal Access Regional Cancer Hospital (SEARCH) database across different BMI categories.

## PATIENTS AND METHODS

After obtaining Institutional Review Board approval from each institution to abstract and combine data, we combined data from patients undergoing RP at the Veterans Affairs Medical Centers in West Los Angeles and Palo Alto, California, Augusta, Georgia, and Durham, North Carolina into the SEARCH database [8]. This database includes information on patient age at surgery, race, height, weight, clinical stage, grade of cancer on diagnostic biopsies, preoperative PSA level, surgical specimen pathology (specimen weight, tumour grade, stage, and surgical margin status), and follow-up PSA data; the BMI was calculated for all patients. Patients treated with preoperative androgen deprivation or radiation therapy were excluded. Of the 1747 men within the SEARCH database, we excluded 50 diagnosed from a TURP, as this affects PSA level, 71 with missing preoperative PSA values, 46 with missing biopsy Gleason scores, 135 with missing clinical stage data, and 221 with missing BMI data. This resulted in a study population of 1434.

The protocol for processing RP specimens was similar across sites, with two of four using step-sectioning with 3–5 mm intervals and embedding all sections for analysis. The third centre used representative sections of the apex, base, inferior, mid and superior aspects of

the gland, including any grossly evident tumour, and seminal vesicles as per the protocol outlined by experienced genitourinary pathologists [9]. At the fourth centre, the distal periurethral plane was removed and sectioned perpendicular to the distal margins. The prostate was sectioned from distal to proximal margin, including the seminal vesicles. The bladder neck was examined separately. Margins were categorized as positive or negative at each given anatomical location (i.e. apex, bladder neck, left peripheral or right peripheral). Information on the number of positive foci at each location was not available, thus patients were defined as either positive or negative at each location.

We explored differences in the distribution of clinicopathological characteristics across BMI groups of normal weight (<25 kg/m$^2$), overweight (25–29.9), obese (30–34.9), and moderately and severely obese (≥35) using ANOVA for continuous variables or the chi-squared test for categorical variables. The odds ratio (OR) of each binary adverse pathological feature (overall and site-specific PSMs, extracapsular extension, and seminal vesicle invasion) was estimated for BMI categories using logistic regression. Few men had lymph node metastases (19 only). BMI was entered into all multivariable models as a series of indicator variables for each BMI category. We tested for trend by entering the median BMI of each BMI category as a continuous term into the model and evaluating the coefficient by the Wald test. We adjusted for clinical characteristics, i.e. preoperative PSA level (continuous variable), age at RP (continuous), year of surgery (continuous), race (white, black vs other), and centre (categorical). We also adjusted for pathological variables (prostate weight, pathological Gleason score, extracapsular extension, seminal vesicle invasion, and lymph node metastasis). Because the data for preoperative PSA level and prostate weight were not normally distributed, we examined the data after logarithmic transformation. The distribution of all clinicopathological variables was similar among the SEARCH sites. Therefore, data from all centres were combined for analyses.

## RESULTS

The clinical and pathological features of the patients are listed in Table 1. Men with a higher BMI were younger and treated more recently (both $P < 0.001$), and had lower preoperative PSA levels even though they had larger prostates (both $P = 0.02$). A higher BMI was associated with higher tumour grade in the biopsy ($P \leq 0.07$) and final pathological examination ($P = 0.10$), although neither were statistically significant. Overall, the incidence of PSMs was 45% (635 men). The overall incidence of positive left (22%) and right (21%) peripheral margins were very similar to the incidence of apical PSMs (21%); positive bladder neck margins were uncommon (6%). A higher BMI was associated with a higher incidence of overall PSMs ($P = 0.04$) and in all specific anatomical locations (all $P \leq 0.03$). There was no significant association between BMI and any other adverse pathological features.

After adjusting for several preoperative clinical and pathological characteristics, a higher BMI was not significantly associated with odds of extracapsular extension ($P$ trend = 0.81) or seminal vesicle invasion ($P$ trend = 0.44; data not shown). However, a higher BMI was associated with a significantly greater incidence of overall PSMs ($P < 0.001$) with more than double the risk among moderately and severely obese men (Table 2). Similarly, a higher BMI was significantly associated with a greater risk of PSMs at all specific anatomical locations (all $P$ trend ≤0.007, Table 2). For example, men with a BMI of 30–34.9 kg/m$^2$ had a 44–78% increased odds of PSMs, depending on the location. Furthermore, when BMI was treated as a continuous variable, the OR associated with each 1-point increase in BMI for the risk of overall PSMs or at any specific locations was nearly identical, at 1.05–1.06. There was a suggestion that extreme obesity (≥35 kg/m$^2$), might be more strongly associated with apical and bladder neck margins (OR 3.11 and 3.74, respectively), although the 95% CIs

overlapped with the estimates for the other site-specific PSMs. Thus, there was no compelling evidence that any one specific anatomical site of PSMs was more or less strongly associated with BMI than any other site.

## DISCUSSION

In the present study, obesity increased the risk of overall PSMs and at all site-specific anatomical locations examined. Although the risk of PSMs was increased at all sites, there was a possible suggestion of greater risk for apical and bladder neck margins among the most obese men. Importantly, we did not detect a significant association between higher BMI and either extracapsular extension or seminal vesicle invasion, suggesting that the excess risk of PSMs in obese men results from suboptimal technique rather than an extension of tumour outside the prostate (i.e. advanced disease). Given that the excess risk was apparent at all sites, care must be taken during the entire operation to ensure clean margins. The possible greater risk at the apex and bladder neck requires verification, but if validated suggests that special emphasis should be placed on dissection in these locations to prevent iatrogenic PSMs in obese men.

The reported incidence of PSMs after RP is 11–39% [1,2,10–12]; in the present study the rate was 45%, which is at the higher end of the range. This probably reflects a combination of more advanced disease in this racially mixed equal-access database dating back many years, a high incidence of obesity, and pooled data from many surgeons (both high- and low-volume). We hypothesise that a PSM might result from one of two broadly classified reasons, i.e. iatrogenic, or extension of tumour beyond planned adequate limits for resection (i.e. advanced disease). Regardless of the aetiology, many studies found that PSMs are an independent predictor of recurrence [10,11]. In addition, previous work from the SEARCH database also found that PSMs increase the risk of PSA recurrence [13].

Similarly, obesity is yet another predictor that increases the risks of high-grade disease, as well as biochemical recurrence after RP [5,6,14,15]. Beyond a more aggressive biology in obese men [16], obesity can also complicate complete surgical extirpation due to technical issues [7]. Moreover, these technical challenges are apparent regardless of the method of surgical approach [17]. When the prostate is approached through a retropubic incision, the excess transabdominal fat makes access to the prostate more difficult. Intra-abdominal adiposity can similarly affect access in the laparoscopic approach. Although perineal prostatectomy avoids both transabdominal and intra-abdominal fat, obese men are still at increased risk of PSMs through a perineal approach [17]. It has also been shown that obese men have larger prostates [18] and these in turn can be difficult to remove through a perineal approach, thereby resulting in an iatrogenic PSM. The net result is that obesity increases the risks of having PSMs [12,14,19]. In the present study, the risk of overall PSMs was 45% higher in mildly obese men and 128% higher in moderately and severely obese men. These results very closely mirror a previous study of nearly 2900 men from Johns Hopkins, in which although the overall PSM rate was much lower (13%), mildly obese and moderately/severely obese men were 96% and 157% more likely to have PSMs than were normal weight men, respectively [5]. Siddiqui *et al.* [12] also found a significantly increased PSM rate of 46% in obese men, vs 33% among normal weight men.

Although obesity increases the risk of overall PSMs it is unknown whether obesity results in a greater incidence at all specific anatomical sites. Using pooled data from several institutions, we found that obesity increased the risk of PSMs at all anatomical sites assessed. For mildly obese men, this risk was very similar across all anatomical sites (44–78% increased risk). However, for men with a BMI of ≥35 kg/m$^2$, there was more variation, with the highest excess risk of a PSM at the bladder neck and apex. However, in the present

study, there were relatively few men with positive bladder neck margin (78), and thus these risks should be interpreted with caution. However, there was a robust sample size for apical PSMs (280). The apex of the prostate is the most distal portion and would theoretically be the most difficult part to visualize, particularly in an obese man. Thus, the current data would suggest that although care should be taken in general when operating on obese men, perhaps additional care is needed in the apical area to prevent iatrogenic PSMs.

Our study has a several limitations; there was no centralised analysis of prostate specimens. This might lead to variations in assessing surgical margin status due differences in specimen processing and interobserver variation among pathologists. However, adherence to standardized protocols by individual institutions would have mitigated the effects of interobserver variation. Moreover, the multi-institutional nature of the pathological assessment might lead to the results being more generally applicable to community practices. In addition, each pathologist and centre processed and read the specimens similarly, regardless of BMI. Thus, their variations are unlikely to be differential by BMI and thus their effect on the current findings is unclear. Also, we controlled for centre in our analysis to adjust for case mix and pathological processing/interpretation differences among centres. Overall, there were more PSMs in our series than in several contemporary series in which most of the surgery was done by one surgeon or a small group of high-volume surgeons [5,11]. However, our results are comparable with the margin-positive rates noted in the multi-institutional Cancer of the Prostate Strategic Urologic Research Endeavor database, in which patient care is delivered through a group of physicians with differing levels of experience and expertise, and wherein the PSM rate was reported as 34%, with an additional 6% of patients having indeterminate margins [2]. Even in the hands of experienced surgeons practising at major urban centres, Eastham *et al.* [20] reported that PSM rates were 10–48%. Moreover, the case mix of the current study shows higher risk disease (higher PSA level, more obese, smaller prostates) than many series [5,17], which could have further contributed to the high PSM rate. Regardless of the overall incidence of PSMs, the current findings are in line with previous data from high-volume tertiary-care referral centres, showing that obesity is associated with a greater risk of PSMs [5,17].

In conclusion, in a multi-institutional series, obesity was associated with a greater risk of overall PSMs and at all specific anatomical locations. Although in general the excess risk of PSMs associated with obesity was similar at all sites, there was a suggestion of a particularly high risk of apical margins among the most obese men, which if validated, further supports the importance of apical dissection in all men, and suggests added difficulty in obese patients.

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## Abbreviations

| | |
|---|---|
| **SEARCH** | Shared Equal Access Regional Cancer Hospital |
| **BMI** | body mass index |
| **OR** | odds ratio |
| **PSM** | positive surgical margin |

**RP** radical prostatectomy

## References


1. Ohori M, Wheeler TM, Kattan MW, et al. Prognostic significance of positive surgical margins in radical prostatectomy specimens. J Urol. 1995; 154:1818–24. [PubMed: 7563355]
2. Grossfeld GD, Chang JJ, Broering JM, et al. Impact of positive surgical margins on prostate cancer recurrence and the use of secondary cancer treatment: data from the CaPSURE database. J Urol. 2000; 163:1171–7. [PubMed: 10737489]
3. Swindle P, Eastham JA, Ohori M, et al. Do margins matter? The prognostic significance of positive surgical margins in radical prostatectomy specimens. J Urol. 2005; 174:903–7. [PubMed: 16093984]
4. Romero-Corral A, Somers VK, Sierra-Johnson J, et al. Accuracy of body mass index in diagnosing obesity in the adult general population. Int J Obes (Lond). 2008; 32:959–66. [PubMed: 18283284]
5. Freedland SJ, Grubb KA, Yiu SK, et al. Obesity and risk of biochemical progression following radical prostatectomy at a tertiary care referral center. J Urol. 2005; 174:919–22. [PubMed: 16093988]
6. Freedland SJ, Isaacs WB, Mangold LA, et al. Stronger association between obesity and biochemical progression after radical prostatectomy among men treated in the last 10 years. Clin Cancer Res. 2005; 11:2883–8. [PubMed: 15837737]
7. Freedland SJ, Grubb KA, Yiu SK, et al. Obesity and capsular incision at the time of open retropubic radical prostatectomy. J Urol. 2005; 174:1798–801. [PubMed: 16217290]
8. Hamilton RJ, Aronson WJ, Presti JC Jr, et al. Race, biochemical disease recurrence, and prostate-specific antigen doubling time after radical prostatectomy: results from the SEARCH database. Cancer. 2007; 110:2202–9. [PubMed: 17876838]
9. Hall GS, Kramer CE, Epstein JI. Evaluation of radical prostatectomy specimens. A comparative analysis of sampling methods. Am J Surg Pathol. 1992; 16:315–24. [PubMed: 1373577]
10. Blute ML, Bostwick DG, Bergstralh EJ, et al. Anatomic site-specific positive margins in organ-confined prostate cancer and its impact on outcome after radical prostatectomy. Urology. 1997; 50:733–9. [PubMed: 9372884]
11. Eastham JA, Kuroiwa K, Ohori M, et al. Prognostic significance of location of positive margins in radical prostatectomy specimens. Urology. 2007; 70:965–9. [PubMed: 18068455]
12. Siddiqui SA, Inman BA, Sengupta S, et al. Obesity and survival after radical prostatectomy: a 10-year prospective cohort study. Cancer. 2006; 107:521–9. [PubMed: 16773619]
13. Freedland SJ, Aronson W, Presti JC Jr, et al. Should a positive surgical margin following radical prostatectomy be pathological stage T2 or T3? Results from the SEARCH database. J Urol. 2003; 169:2142–6. [PubMed: 12771736]
14. Amling CL, Riffenburgh RH, Sun L, et al. Pathologic variables and recurrence rates as related to obesity and race in men with prostate cancer undergoing radical prostatectomy. J Clin Oncol. 2004; 22:439–45. [PubMed: 14691120]
15. Bassett WW, Cooperberg MR, Sadetsky N, et al. Impact of obesity on prostate cancer recurrence after radical prostatectomy: data from CaPSURE. Urology. 2005; 66:1060–5. [PubMed: 16286124]
16. Rodriguez C, Freedland SJ, Deka A, et al. Body mass index, weight change, and risk of prostate cancer in the Cancer Prevention Study II Nutrition Cohort. Cancer Epidemiol Biomarkers Prev. 2007; 16:63–9. [PubMed: 17179486]
17. Fitzsimons NJ, Sun LL, Dahm P, et al. A single-institution comparison between radical perineal and radical retropubic prostatectomy on perioperative and pathological outcomes for obese men: an analysis of the Duke Prostate Center database. Urology. 2007; 70:1146–51. [PubMed: 18158036]
18. Freedland SJ, Platz EA, Presti JC Jr, et al. Obesity, serum prostate specific antigen and prostate size: implications for prostate cancer detection. J Urol. 2006; 175:500–4. [PubMed: 16406980]

19. Freedland SJ, Aronson WJ, Kane CJ, et al. Impact of obesity on biochemical control after radical prostatectomy for clinically localized prostate cancer: a report by the Shared Equal Access Regional Cancer Hospital database study group. J Clin Oncol. 2004; 22:446–53. [PubMed: 14691122]

20. Eastham JA, Kattan MW, Riedel E, et al. Variations among individual surgeons in the rate of positive surgical margins in radical prostatectomy specimens. J Urol. 2003; 170:2292–5. [PubMed: 14634399]

**TABLE 1**

Clinical and pathological features of men undergoing RP, segregated by BMI

| Variable | Normal weight | Overweight | Mildly obese | Moderately and severely obese | *P** |
|---|---|---|---|---|---|
| BMI, kg/m$^2$ | <25 | 25–29.9 | 30–34.9 | ≥35 | |
| No. patients | 365 | 649 | 298 | 122 | |
| Median year of surgery | 2000 | 2001 | 2001 | 2002 | <0.001† |
| Mean (SD) age, years | 62.0 (6.5) | 61.1 (6.4) | 60.7 (6.3) | 59.0 (6.2) | <0.001† |
| Race, *n* (%) | | | | | |
| White | 185 (51) | 318 (49) | 145 (49) | 58 (48) | 0.09 |
| Black | 153 (42) | 276 (43) | 140 (47) | 61 (50) | |
| Other | 27 (7) | 54 (8) | 13 (4) | 3 (2) | |
| PSA level, ng/mL | | | | | |
| Mean (SD), median | 10.2 (7.0), 7.6 | 9.1 (7.0), 7.1 | 9.9 (11.6), 7.0 | 8.3 (7.4), 6.1 | 0.02† |
| Biopsy Gleason sum, *n* (%) | | | | | 0.07 |
| 2–6 | 248 (68) | 381 (59) | 191 (64) | 71 (58) | |
| 7 (3 + 4) | 68 (19) | 147 (23) | 56 (19) | 32 (26) | |
| ≥4 + 3 | 49 (13) | 121 (19) | 51 (17) | 19 (16) | 0.56 |
| Clinical stage, *n* (%) | | | | | |
| T1c | 188 (54) | 339 (54) | 167 (58) | 70 (59) | |
| T2/T3 | 163 (46) | 284 (46) | 121 (42) | 49 (41) | |
| Prostate weight, g | | | | | |
| Mean (SD), median | 41.4 (20.6), 36 | 44.1 (22.2), 40 | 46.2 (26.6), 39 | 46.4 (19.8), 43 | 0.01† |
| Pathological Gleason sum, *n* (%) | | | | | 0.10 |
| 2–6 | 166 (46) | 264 (41) | 106 (36) | 41 (34) | |
| 7 (3 + 4) | 121 (33) | 233 (36) | 125 (42) | 47 (39) | |
| ≥4 + 3 | 75 (21) | 146 (23) | 64 (22) | 32 (27) | |
| PSMs, *n* (%) | 147 (41) | 282 (44) | 141 (48) | 65 (55) | 0.04 |
| Left peripheral | 64 (19) | 128 (21) | 66 (24) | 34 (32) | 0.03 |
| Right peripheral | 61 (18) | 117 (20) | 69 (25) | 30 (29) | 0.03 |
| Apex | 55 (16) | 130 (21) | 57 (21) | 38 (35) | 0.001 |
| Bladder neck | 16 (5) | 31 (5) | 17 (6) | 14 (12) | 0.02 |

| Variable | Normal weight | Overweight | Mildly obese | Moderately and severely obese | *P*[*] |
|---|---|---|---|---|---|
| Extraprostatic extension | 79 (22) | 166 (26) | 62 (21) | 35 (30) | 0.15 |
| Seminal vesicle invasion | 33 (9) | 65 (10) | 24 (8) | 13 (11) | 0.73 |
| Lymph node involvement | 7 (2) | 7 (1) | 3 (1) | 2 (2) | 0.13 |

[*] *P* by chi-squared, except

[†] by ANOVA.

## TABLE 2

OR (95% CI) of PSMs at the time of RP by BMI (relative to normal weight, <25 kg/m$^2$; definitions as in Table 1)

| | OR (95% CI)* | *P* |
|---|---|---|
| Overall PSMs | | 0.001† |
| Overweight | 1.20 (0.88–1.64) | |
| Mild obesity | 1.45 (1.00–2.09) | |
| Moderate and severe obesity | 2.28 (1.38–3.76) | |
| BMI as continuous variable | 1.05 (1.02–1.08) | <0.001 |
| Left peripheral PSMs | | 0.007† |
| Overweight | 1.14 (0.79–1.67) | |
| Mild obesity | 1.49 (0.96–2.30) | |
| Moderate and severe obesity | 1.98 (1.13–3.46) | |
| BMI as continuous variable | 1.05 (1.02–1.08) | 0.002 |
| Right peripheral PSMs | | 0.001† |
| Overweight | 1.19 (0.80–1.75) | |
| Mild obesity | 1.78 (1.14–2.78) | |
| Moderate and severe obesity | 2.21 (1.24–3.95) | |
| BMI as continuous variable | 1.06 (1.03–1.10) | <0.001 |
| Apical PSM | | <0.001† |
| Overweight | 1.46 (0.99–2.15) | |
| Mild obesity | 1.44 (0.91–2.26) | |
| Moderate and severe obesity | 3.11 (1.78–5.41) | |
| BMI as continuous variable | 1.06 (1.03–1.10) | <0.001 |
| Bladder neck PSM | | 0.002† |
| Overweight | 1.21 (0.61–2.38) | |
| Mild obesity | 1.68 (0.79–3.60) | |
| Moderate and severe obesity | 3.74 (1.61–8.73) | |
| BMI as continuous variable | 1.06 (1.01–1.11) | 0.016 |

*Adjusted for age, preoperative PSA level, year of surgery, race, centre, pathological Gleason score, pathological specimen weight, extracapsular extension, seminal vesicle invasion and lymph node involvement;

†P trend determined using the median BMI of each category as a continuous variable.