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The Effects of Beginning Population Based PSA Screening at Age 40

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Abstract

Objectives—To evaluate population-based CaP testing of men in their 40s, given the paucity of prospective data evaluating the consequences of PSA testing in younger men for prostate cancer (CaP).

Methods—1052 men in their 40s were followed longitudinally for prostate outcomes, from 1990 to 2010. A random subset of 268 men was selected to undergo biennial CaP testing including: PSA testing, transrectal ultrasound, and a digital rectal exam (DRE). A representative population of 609 men with a subset of 159 men who also began CaP testing in their 50s was also evaluated as a comparison group. Risk of prostate biopsy (PBx), CaP, or death from CaP was compared between CaP tested and the routine care population cohort.

Results—Median follow-up was 17.2 years. Men aged 40–49, who underwent CaP testing were 2.4× more likely to undergo a PBx (HR 2.4 95% CI: 1.8–3.3) and 2.2× more likely to be diagnosed with low risk CaP (HR 2.2, 95% CI: 1.12–4.0). Those initiating CaP testing a decade earlier were 2.2× and 1.7× more likely to be biopsied and be diagnosed with CaP for any given age (HR 2.2 95% CI 1.4–3.5 and 1.7 95% CI 1.1–2.7, respectively).

Conclusions—CaP testing in men beginning at age 40 resulted in a significant increase in the risk of PBx and diagnosis of low risk CaP, without a measurable reduction in risk of CaP-death in this low risk population. However, given the natural history of CaP, a longer follow-up is needed to confirm this finding.

Keywords

screening; prostate cancer; early detection; PSA testing

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Introduction

PSA screening for the early detection of prostate cancer (CaP) remains controversial. Guidelines from professional societies have ranged from recommending a discussion of screening in asymptomatic men beginning at age 40 by the European Association of Urology (EAU),¹ or recommending screening beginning at age 45 by the National Comprehensive Cancer Network (NCCN)² on the more conservative end. The American Urological Association (AUA) recommends screening beginning at age 55,³ and the 2012 US Preventative Services Task Force (USPSTF) recommended against PSA screening altogether.⁴ There is little evidence evaluating the screening of men under age 50, despite retrospective data from banked serum samples that suggests that younger men with an elevated baseline PSA are more likely to have adverse long-term prostate cancer outcomes.^{5–7}

Since the institution of PSA screening in the United States, the overall death rate from CaP has dropped dramatically. There remains a debate over how much of this drop is attributable to screening itself.⁸ The absolute number of deaths from CaP in the US has changed relatively little in the past 30 years since the introduction of PSA. There were 27,262 deaths from CaP in 1986 (pre-PSA era)⁹ and 26,120 in 2016;¹⁰ adjusted for population growth, the number needed to treat to prevent one CaP-specific death by PSA screening remains high.¹¹ PSA screening, when applied prospectively and compared to a population with little screening, confers a cancer-specific survival advantage; to prevent one death from CaP, the number needed to screen and to treat is 1055 and 37 respectively.^{12,13} This reduction in death from CaP does not translate into a reduction of death from any cause, however, and overall survival is equal between the groups during follow up. Furthermore, a recent Cochrane review failed to find a significant reduction in CaP mortality and noted significant harms in a meta-analysis.¹⁴

Harms of CaP screening include unnecessary biopsies, biopsy-related complications, patient anxiety, and adverse events related to the diagnosis and treatment of a potentially biologically inert ‘cancer’.¹⁵ In fact, it is estimated that as many as 1 million men in the past 20 years may have been over-diagnosed with non-lethal cancers.¹¹ Yet CaP continues to be a primary cause of cancer specific death, with more than 300,000 men dying from CaP worldwide each year.¹⁶

Several retrospective cohort studies have found that men with a higher baseline PSA in their 40s are at significantly higher risk of subsequent development of prostate cancer 20–30 years later.^{5,6,17–19} Analysis of these studies has led to the conclusion that perhaps patients could be risk-stratified with an early baseline PSA, with subsequent screening appropriately tailored. This stratified screening process could maintain the benefits of screening, while minimizing harms. However, since these studies are largely based on banked serum, it remains unclear how patients and physicians might act on prospectively analyzed PSA, digital rectal exam (DRE) and ultrasound findings.

In this study, we evaluate how CaP testing initiated in men in their 40s would affect the subsequent risks of undergoing a prostate biopsy, receiving a CaP diagnosis, and tumor

characteristics when followed prospectively compared to a representative population cohort undergoing routine care. We also compare these risks to men who commence CaP testing starting at age 50.

Materials and Methods

The principal study population consisted of men (n=1052) ages 40-49, living in Olmsted County, MN, USA enrolled in a prospective cohort study entitled “Natural History of Prostatism: The Olmsted County Study” (DK058859). This study, in part, was designed to prospectively assess the natural history of benign prostatic hyperplasia and to understand how surrogate measures of prostate growth, such as PSA, changed over time. A random sample of these men was chosen for clinic evaluation as described below (n=268). An additional random sample (n=161) of men were selected to undergo CaP testing, ages 50-59, and were chosen from a population cohort (n=609) undergoing routine care. This ‘older’ cohort was followed and compared to the men entering the study in their 40s. The entire cohort has been described previously.²⁰ Beginning in 1990, 3,874 men living in Olmsted County between the ages of 40-79 were invited to participate; 2,115 (55%) of eligible subjects enrolled at baseline and completed biennial questionnaires about overall health status, urinary symptoms and sexual function. Men in the first few years of the study who died or were lost to follow-up were replaced during rounds 2 and 3 (in 1992, 1994), resulting in a total of 2,447 study participants, of which 1052 were ages 40-49, and 609 were ages 50-59 at study entry. A random sample, including 268 and 161 men between ages 40-49 and 50-59 respectively, was selected as a ‘clinic cohort,’ with 87% participating in a biennial CaP testing. This included a DRE, PSA screening, and a transrectal ultrasound of the prostate. Any patient with an abnormal DRE result, elevated PSA (>4.0 ng/mL), or suspicious lesion on TRUS were further evaluated with a prostate biopsy. Two men, ages 50-59 at baseline, had a previous biopsy and were excluded from subsequent analysis leaving a cohort of 159. Men not included in the CaP testing cohort completed questionnaires only and were free to undergo CaP testing at the discretion of their primary care physicians. The study population was then maintained as a closed cohort and followed biennially. This study received institutional review board approval from the Mayo Clinic and Olmsted Medical Center.

The community medical records of all study participants were abstracted to obtain information on CaP diagnosis, prostate biopsy, death and cause of death, if applicable.

If a PSA above the age-specific norm was identified during the course of the study (>2.5 ng/mL for 40-49, >3.5 ng/mL 50-59, >4.5 ng/mL for 60-69, and >5.5 ng/mL for 70-79)²⁰, or if an abnormality in texture (nodule or asymmetry) was noted on DRE, a letter to the patient advised them to seek medical attention.

Differences between men randomly selected to CaP testing were analyzed by chi-square, Fisher's exact test, or Wilcoxon log rank as appropriate. These comparisons were also made according to which decade of life PSA screening commenced. Risk of subsequent biopsy or cancer diagnosis was calculated using Kaplan-Meier curves (graphing failure instead of survival), log rank test and proportional hazard ratios (HR). To account for the differential

risk of cancer between men in their 50s vs. 40s at study entry, Kaplan-Meier curves used age on the x-axis. Proportional hazards assumptions were not met for CaP diagnosis in patients after 18 years of follow-up, therefore follow-up was censored in these patients at 18 years for these analyses. Incidence was calculated dividing the incident cases during a specified time period by years of at risk follow-up during that time period, and standardized against US incidence rates from 1992-2008. Estimates of new cases were calculated using 2011 US census data and Olmsted County incidence rates. All analyses were performed using JMP 9.0.1 (SAS Institute, Cary, NC, USA). All tests of statistical significance were two-sided with an alpha of 0.05.

Results

A comparison of baseline statistics can be found in Table 1, stratified according to whether subjects were randomly selected to participate in the screening cohort or whether they began protocol screening in the 4th or 5th decade of life. There were only small, clinically insignificant differences in baseline risk.

Comparison of Screened Cohort vs. Routine Care Beginning ages 40-49

The risks of undergoing a biopsy, developing CaP, and death from any cause are presented in Supplemental Table 1. Notably, men who began CaP screening at age 40 had a more than two-fold higher risk of undergoing prostate biopsy than age-matched population controls (HR 2.4, 95% CI: 1.7-3.3). The screened men were also twice as likely to be diagnosed with low risk prostate cancer (HR 2.2, 95% CI: 1.12-4.0). Negative biopsy rates were high in both cohorts (Supplemental Table 1) though they were 2.6-fold higher in the screened cohort. Similar findings were noted on survival curves depicting risk of subsequent prostate biopsy or CaP (Figure 1). In the 40-49-year-old-screened cohort, no man (n=0/268) went on to develop high-risk prostate cancer (Gleason Score >7) and only one man in the non-screened cohort did (n=1/759).

Comparison of Effect of Beginning PSA screening in the 4th or 5th decade of life

To determine the effect of the age at which PSA screening is initiated on the risk of undergoing prostate biopsy and CaP, we compared men who entered the study in their 50s vs. 40s. We observed that the risk of prostate biopsy by age 50 in men who began screening in their 40s was 5.3% (95% CI 2.6-8.1) (Figure 2a). The risk of biopsy increased at a faster rate than those who began screening at age 50. By age 67 (the median age of CaP diagnosis in the US), the risk of biopsy in the younger cohort was 41% vs 22% in the older cohort (HR 2.5, 95% CI 1.4-3.5). Men who began screening in their 40s remained significantly more likely to have a biopsy when compared age for age (Figure 2b).

The risk of CaP diagnosis was 1.7-fold higher in the men who began screening a decade earlier, when controlling for family history (Figure 3), (HR 1.7, 95% CI 1.1-2.7). By age 67, men who began screening in their 40s had an estimated 24% risk of CaP diagnosis (95% CI: 4.5-43%) which is well above the lifetime risk of prostate cancer for men in the US. By contrast, men who began CaP screening in their 50s only had a 6% risk of CaP at this age. In

the 50-59-year-old screened cohort, only one man (n=1/159) went on to develop high-risk prostate cancer (Gleason Score >7) vs. four men in the non-screened cohort (n=4/450).

Incidence and Standardized Incidence Ratios

The majority of incident cancers identified in both cohorts (Supplemental Table 2) were low risk; 78% were Gleason <7, PSA <10. There were only two cancer related deaths in these 1661 men, precluding any predictive models of cancer-specific death. Incidence rates in Olmsted County during the study period for those who began PSA screening in their 40s were 2-3 times higher than those observed in all men in the US during the same time period (Supplemental Table 3), and 1-3 times higher than men in Olmsted County not randomized to the screening cohort. We note, however, that the confidence intervals of the incidence rates are wide and include both the US rates and the routine-care incidence rates, and therefore may represent random variation. We also note that being selected to participate in the screening cohort in men entering the study ages 50-59 did not increase the risk of cancer diagnosis. We hypothesize that this may in part due to the fact that PSA screening was recommended beginning at age 50 during the study period, and there has been traditionally high compliance (61% in men age>50) with CaP screening in Olmsted County²¹ vs. 40% in men of comparable age and time period in the US population as a whole.²²

Discussion

The principal finding of our study is that population-based screening, if initiated in younger men aged 40-49 years, resulted in a greater risk of these men undergoing a biopsy (HR 2.4, 95% CI: 1.7-3.3) and receiving the diagnosis of low risk CaP (HR 2.2, 95% CI: 1.12-4.0) compared to men who underwent routine care. Those who initiated screening at age 40 in our study had a CaP diagnosis rate of nearly 24% (Figure 3) during the ensuing 18 years of follow up. For comparison, the current estimated lifetime risk of CaP for the average man in the US is only 16%. There were no significant differences in the two cohorts with regards to the subsequent development of intermediate or high-risk cancers. Whether screening was started at age 40 or 50, the proportion of patients developing intermediate or high-risk cancers was only 0.1% (Supplemental Table 2).

Some professional societies have recommended that men begin PSA screening early to increase the odds of identifying localized cancer, although more recently these recommendations have been qualified with the consideration of family history, race, or other risk factors.^{23,24} Several of these groups had previously advocated initiating screening as early as age 40. This had stemmed, in part, from studies demonstrating that men presenting with cancer outside the gland at the time of definitive treatment were seven times more likely to die of CaP than those with localized disease.²⁵ There is also data to suggest that a higher baseline PSA in younger men may be predictive of developing prostate cancer 20-30 years later.⁵⁻⁷ What has not been clear, however, is whether formalized screening programs for these younger patients would result in a net benefit. Our data seem to suggest that a baseline PSA is useful in discriminating long term risk of CaP,²⁶ although it must be balanced by the risks of increased CaP testing and diagnosing low risk CaP.

It remains clear that these younger men are at a very low risk of 10-20-year risk of death from CaP, making impossible for a cohort as small as ours to detect a difference in CaP survival. In a retrospective, case-control study of Swedish men, Vickers et al reported the risk of CaP metastases at 15 years being as low as 0.6% for men at age 40, even for those who had relatively high PSAs in their age group (1.6 ng/mL).²⁷ Another study constructed a Markov model combining ERSPC and Australian age-specific incidence data comparing screened and unscreened cohorts, and found very low CaP-specific death over 10 years among those who would have hypothetically begun screening at age 40.²⁸ Therefore, determining whether PSA screening of men in their 40s could reduce CaP-specific death would require immense numbers, very long follow up, or both.

Limitations to our study include the fact that it was not designed to be a PSA screening trial, and we assessed secondary endpoints. CaP-specific death was also extremely rare in our cohort, with only two (out of 1661) men dying from CaP during follow up, for a rate of 28 deaths/100,000 patient years of follow-up. The diagnosis of intermediate/high risk prostate cancer was also low for the entire cohort, and it was even lower among men in the screening cohort (Supplemental Table 1). The low rate of intermediate/high risk disease and death from CaP is likely a reflection of our relatively small sample size. We are therefore unable to definitively conclude whether the increased burden of testing and cancer diagnosis is offset by a risk reduction in death from CaP. Another limitation is the fact that follow-up was relatively short considering the known natural history of prostate cancer. Given that our cohort was comprised of younger, healthy individuals, additional years of follow-up would likely be needed to identify a clear difference should one exist. Current prostate cancer screening strategies also routinely incorporate the use of novel biomarkers and multiparametric magnetic resonance imaging, neither of which were available for routine use during the study period. Our study also did not assess quality of life variables, which is an important variable to study as part of future screening trials.

Our findings have implications for clinicians in understanding the risks and benefits of commencing PSA screening at age 40.

Conclusion

We found no evidence that initiation of biennial CaP screening at age 40 offers any advantage over initiation at age 50 in reducing CaP related morbidity in this small, prospectively followed cohort. However, these results should be tempered by the fact that younger populations likely require a longer follow-up than our study was able to confer. A larger study cohort may also be required to validate our findings. CaP screening in men beginning at age 40 in our study resulted in a significant increase in risk of prostate biopsy and the diagnosis of low risk CaP compared to age matched population controls.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1. Risk of Prostate Biopsy (A) and Cancer (B) according to PSA screening status men ages 40-49

Figure 2.
Risk of Prostate Biopsy according to decade in which PSA screening begins i.e. 40-49 vs. 50-59. (A) With all data, (B) Excluding those with biopsy before age 50.

Figure 3. Risk of CaP according to decade in which PSA screening commences

Table 1
Baseline Characteristics men ages 40-49 stratified according to PSA screening status (A)
and Characteristics at baseline in screened men according to when they commenced PSA
Screening (B)

A.				
Characteristics at baseline	All Men (n=1052)	Screened (n=268)	Routine Care (n=784)	P value
Age at entry in years, median (IQR)	44.4 (42.9-46.7)	44.8 (43.3-46.7)	44.3 (42.7-46.6)	0.027
Median Follow-up in years (IQR)	17.2 (15.1-18.5)	16.9 (15.0-17.6)	17.7 (15.1-18.6)	<0.0001
Family History of CaP, n (%)	90 (8.6%)	19 (7.1%)	71 (9.0%)	0.33
Baseline AUA symptom score (IQR)	4 (2-7)	5 (2-9)	4 (1-7)	0.081

B.			
	Screened Starting 40-49	Screened Starting 50-59	P value
Age at entry in years, median (IQR)	44.8 (43.3-46.7)	54.3 (51.7-56.1)	<0.0001
Median Follow-up in years (IQR)	16.9 (15.0-17.6)	17.1 (16.3-17.9)	0.043
Family History of CaP, n (%)	19 (7.1%)	9 (5.7%)	0.67
Baseline PSA (median, ng/mL)	0.7 (0.5-1.0)	0.9 (0.6-1.4)	0.0002
Baseline DRE Abnormal	29 (11%)	27 (17%)	0.86

CaP – Prostate Cancer

DRE – Digital Rectal Exam

IQR – Interquartile Range

PSA – Prostate Specific Antigen