1. Introduction

Although prostate-specific antigen (PSA) is widely used to screen for prostate cancer in the asymptomatic male population, tremendous controversy exists regarding its appropriate implementation [1]. Drawbacks of PSA as a screening test include the diagnosis and treatment of some indolent tumors that would not have presented clinically and limitations in specificity whereby serum PSA levels may be elevated in benign prostatic conditions (benign prostatic hyperplasia, prostatitis) and urinary tract manipulation (eg, cystoscopy, prostate biopsy) [2]. In addition, recent studies have also shown variations in PSA levels according to genetic factors, obesity, certain medications, and standardization bias, adding to the complexity of its interpretation [3–7].

Despite these issues, there is general consensus that widespread PSA screening has led to a striking stage migration, with the vast majority of prostate cancers now diagnosed at a localized stage [8]. Numerous studies have demonstrated improved outcomes in serially screened men. For example, Roehl et al. compared radical prostatectomy...
outcomes between men diagnosed through serial screening compared with a referral population [9]. The respective 7-yr progression-free survival rates were 83% versus 77% (p = 0.002). On multivariable analysis with age at diagnosis, PSA, biopsy Gleason score, and clinical stage, screening status was significantly associated with time to biochemical recurrence. Even among men with PSA failure after radical prostatectomy, Efsthathiou et al. reported that men who were diagnosed through serial screening were more likely to have a PSA doubling time $\geq 12$ mo and had a significantly lower 10-yr cancer-specific mortality rate compared with referred patients (3.6% vs 11.3%; p = 0.0002) [10]. Several studies have also confirmed an absolute and relative reduction in metastatic prostate cancer in screened populations as compared with populations not screened [11].

With regard to screening and mortality, large randomized controlled trials of PSA-based screening were reported recently. The European Randomized Study of Screening for Prostate Cancer (ERSPC) included 162,243 men between the ages of 55 and 69 yr randomized to screening and control groups [12]. At a median follow-up of 9 yr, the intent-to-screen analysis revealed a relative risk of 0.80 (20% decrease) for prostate cancer–specific mortality, as well as a 41% decrease in metastatic disease at the time of diagnosis with screening.

In the United States, the Prostate, Lung, Colorectal and Ovary (PLCO) screening trial randomized 76,693 men 55 to 74 yr of age to annual screening versus usual medical practice in the “control” arm [13]. Because PSA testing was already widespread in the United States during this time, most men had been prescreened, and $>50$% of controls had opportunistic screening during the study. In the initial analysis of all participants, there was no difference in the stage distribution or in disease-specific mortality between the groups; however, in a subsequent subset analysis of men with no or one comorbid conditions, there was a 44% lower prostate cancer–specific mortality rate with screening [14].

The Goteborg randomized population-based screening trial included a subset of ERSPC participants [15]. It randomized 99,522 men each to biennial screening and control groups. In this study, screening was associated with a rate ratio of 0.56 (44% relative reduction) for prostate cancer mortality at a median follow-up of 14 yr but with no decrease in overall mortality as yet.

These varying results are echoed in the divergent guidelines for prostate cancer screening from professional organizations. For example, the American Cancer Society recommends an informed discussion of the risks and benefits of PSA testing beginning at age 50 for average-risk men and in the 40s for high-risk men (African American or family history) [16]. The US Preventive Services Task Force (USPSTF) issued recommendations in 2008, concluding there is insufficient evidence on the benefits and harms of screening for men $<75$ yr of age [17], whereas they recommended against screening for men $\geq 75$ yr of age. Notably, these recommendations were published before the ERSPC and PLCO reports, and updated guidelines from the USPSTF are in progress. The American College of Preventive Medicine concluded there is insufficient evidence to recommend routine prostate cancer screening and instead recommended that physicians conduct annual conversations with patients about the risks and benefits of screening [18]. The European Association of Urology also concluded that current evidence is insufficient to recommend widespread population-based screening with PSA [19].

By contrast, the National Comprehensive Cancer Network Guidelines recommend beginning the risk-benefit discussion and offering baseline digital rectal examination (DRE) and PSA at age 40 yr [20]. Annual follow-up is recommended thereafter for men who are high risk by virtue of family history, African American race, or a baseline PSA level $>1$ ng/ml, whereas average-risk men with a PSA $<1$ ng/ml are offered repeat screening again at ages 45 and 50. The American Urological Association 2009 Best Practice Statement recommends PSA screening for well-informed men who wish to pursue early diagnosis, beginning with a baseline measurement at 40 yr of age [21]. The US military services also offer PSA testing at routine physical examinations beginning at 40 yr of age [22].

Apparent in these divergent recommendations is that disagreement persists over many aspects of prostate cancer screening, including the age to begin PSA testing [23]. This paper reviews studies on baseline PSA testing at a young age ($\leq 60$ yr).

1.1. Introduction to the concept of baseline prostate-specific antigen testing

In 1995, Gann et al. published a seminal report from the Physician’s Health Study that enrolled 22,071 US male physicians 40–84 yr of age [24]. After a baseline blood draw at study inception in 1982, men were followed through 1992 for cancer diagnoses. Baseline PSA measurements were compared between 366 cases and 1098 matched controls. The results showed that, compared with men with a baseline PSA level $\leq 1$ ng/ml, the relative risk of prostate cancer was 2.2, 3.5, 5.5, 8.6, 22.2, and 145.3 with baseline PSA levels of 1.01–1.5, 1.51–2.0, 2.01–3.0, 3.01–4.0, 4.01–10.0, and $>10$ ng/ml. Compared with the reference group with PSA $\leq 1$, the relative risk (RR) of aggressive cancer in these PSA ranges was 1.9, 1.7, 6.8, 10.2, 31.0, and 461.4. Although men in this study varied considerably in age at the time of blood draw, this was among the pioneering studies to reveal the role of PSA as a predictor of future prostate cancer risk and aggressive disease in the absence of screening.

Similarly, Stenman et al. measured PSA levels in frozen serum samples from Finnish participants in the 1968–1973 Social Insurance Institution’s Mobile Clinic Health Examination Survey [25]. Using linkage to the Finnish Cancer Registry through 1980, they reported a correlation between higher PSA levels and shorter time until clinical diagnosis of prostate cancer. Because age ranged from 45 to 84 yr at blood sampling, the authors examined the results by age and showed improved discrimination of prostate cancer with PSA measured at $<65$ yr of age (area under the curve [AUC]: 0.97) versus $\geq 65$ yr of age (AUC: 0.81).
The predictive value of a baseline PSA measurement has also been demonstrated in screening populations. In the Rotterdam section of the ERSPC, Bul et al. recently reported on the 12-yr follow-up results of 15 758 men with a PSA < 3 ng/ml at the first screening round at a median age of 62.3 yr [26]. Compared with participants with a baseline PSA < 1 ng/ml, the hazard ratios for prostate cancer were 4.0 and 10.3 with initial PSA levels of 1.0–1.9 and 2.0–2.9 ng/ml (p < 0.001 for both). Men with baseline levels from 1.0 to 1.9 and from 2.0 to 2.9 ng/ml had a 4-fold and 7.6-fold increased hazard ratio for prostate cancer–specific mortality compared with baseline levels < 1 ng/ml.

In a younger population of men from the Child Health and Development Study, Whittemore et al. similarly evaluated the association between PSA in young men and later risk for prostate cancer diagnosis [27]. Subsequent to a single blood draw at a median age of 34 yr, participants were followed for several decades for cancer diagnoses. Compared with a baseline PSA in the first quartile (< 0.24 ng/ml), a baseline PSA in the fourth quartile (≥ 0.56 ng/ml) was associated with a 7.4-fold and 5.0-fold increased odds of prostate cancer diagnosis before age 65 yr in black and white men, respectively.

### 3. Evidence synthesis

#### 3.1. Baseline prostate-specific antigen at a young age and prostate cancer detection

Several other studies have specifically examined baseline PSA levels in young men (Table 1). For example, Preston et al. examined the PSA distribution in young black men and white men from the Department of Defense Serum Repository [28]. In black men, the median PSA levels were 0.38, 0.45, and 0.52 ng/ml at 20–29, 30–39, and 40–45 yr of age. In white men, the median PSA levels by age were 0.38, 0.45, and 0.40 ng/ml, respectively. Another study of 845 military officer students reported a median PSA level of 0.66 in the 40s [22]. Similarly, in the National Defense University Study, the mean PSA level was 0.9 ng/ml in 1105 men 30–59 yr of age [29].

Numerous studies have also reported on the association between baseline PSA measurements at a young age and prostate cancer risk. In the Baltimore Longitudinal Study of Aging, Fang et al. reported a median PSA level of 0.6 ng/ml for men in their 40s and 0.71 ng/ml for men in their 50s [30]. For men in their 40s with PSA levels less than the age-specific median, the 25-yr cumulative freedom from a clinical diagnosis of prostate cancer was 89.6% versus 71.6% in men with levels greater than their age-specific median. Similarly, for men in their 50s with baseline PSA levels below the age-specific median, 25-yr prostate cancer-free survival rates were 83.6% versus 58.9% for men with levels above their age-specific median.

In the Duke Prostate Database, Tang et al. reported on the relationship between baseline PSA levels and prostate cancer detection on biopsy performed for PSA or DRE indications [31]. Overall, the median PSA level was 0.7 ng/ml for both black and white men < 50 yr of age. Among black men, prostate cancer was diagnosed in 7 men (0.4%) with a baseline PSA ≤ 0.6 ng/ml versus 11 men (4%) with baseline values from 1.5 to 2.5 ng/ml (RR: 11.1; 95% confidence

### Table 1 – Prostate-specific antigen distributions in young men in selected published series

<table>
<thead>
<tr>
<th>Study population</th>
<th>Age range, yr</th>
<th>PSA distribution, ng/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>Department of Defense Serum Repository [28]**</td>
<td>20–29</td>
<td>0.38/0.38</td>
</tr>
<tr>
<td></td>
<td>30–39</td>
<td>0.45/0.45</td>
</tr>
<tr>
<td></td>
<td>40–45</td>
<td>0.52/0.40</td>
</tr>
<tr>
<td>The Army War College Study [22]</td>
<td>40s</td>
<td>0.66</td>
</tr>
<tr>
<td>National Defense University Study [29]</td>
<td>30–59</td>
<td>0.9 (mean)</td>
</tr>
<tr>
<td>Baltimore Longitudinal Study of Aging [30]</td>
<td>40–49.9</td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td>50–59.9</td>
<td>0.71</td>
</tr>
<tr>
<td>Duke Prostate Database [31]</td>
<td>≤ 30</td>
<td>0.7</td>
</tr>
<tr>
<td>Malmo Preventive Project [33]</td>
<td>44–50</td>
<td>0.63</td>
</tr>
<tr>
<td>Catalona Screening Study [35]</td>
<td>40–49</td>
<td>0.7</td>
</tr>
<tr>
<td></td>
<td>50–59</td>
<td>0.9</td>
</tr>
<tr>
<td>Child Health and Development Study [27]***</td>
<td>&lt; 30</td>
<td>0.48/0.31, 0.50/0.32</td>
</tr>
<tr>
<td></td>
<td>30–39</td>
<td>0.38/0.33, 0.44/0.37</td>
</tr>
<tr>
<td></td>
<td>40–55</td>
<td>0.63/0.41, 0.50/0.48</td>
</tr>
</tbody>
</table>

PSA = prostate-specific antigen.
* Median unless otherwise noted.
** African American/white.
*** African American case/control, white case/control.

The evidence for this review was obtained through PubMed searches of publications through April 2011 on keywords baseline PSA and prostate cancer. Additional references were obtained from the bibliography of articles identified during the initial search and through the collaborative group. Only English-language publications were evaluated. For the evidence synthesis, we focused on studies involving young men (< 60 yr of age) at the baseline PSA assessment. The data were then categorized into studies on baseline PSA testing and prostate cancer risk prediction, and those related to baseline PSA measurements and tumor aggressiveness or disease-specific outcomes.
interval [CI], 4.3–19.0; \( p < 0.001 \). Similarly, in white men, prostate cancer was diagnosed in 40 men (0.7%) with a baseline PSA level \( \leq 0.6 \) ng/ml versus 10 men (2.2%) with baseline values from 1.5 to 2.5 ng/ml (RR: 7.6; 95% CI, 3.2–18.4; \( p = 0.001 \)).

Connolly et al. used data from the Northern Ireland Cancer Registry to examine the relationship of PSA measurements drawn in 1994–1998 to subsequent prostate cancer diagnoses and deaths ascertained through 2003 [32]. Of the 68 354 men, 8443 were \( < 50 \) yr of age at the baseline PSA assessment. In this subset, the hazard ratios for prostate cancer diagnosis were 4.65, 24.35, 138.96, 157.46, and 335.12 with baseline PSA levels of 1–1.9, 2–2.9, 3–3.9, 4–9.9, and \( \geq 10 \) ng/ml compared with the reference group with a baseline PSA of 0–0.9 ng/ml.

Baseline PSA levels were also studied extensively in men from the Malmo Preventive Project that enrolled 21 277 Swedish men from 1974 to 1986. In 2007, Lilja et al. reported that PSA measured at 44–50 yr of age was a strong predictor of a clinical diagnosis of prostate cancer during the subsequent two decades [33]. On receiver operating characteristic (ROC) analysis, the AUC was 0.76 for the prediction of prostate cancer based on this measurement. In a more recent analysis from the same source population with updated follow-up, Lilja et al. similarly found a robust association between PSA levels at \( \leq 50 \) yr of age with subsequent prostate cancer diagnosis (AUC: 0.719; \( p < 0.0005 \)) [34]. In the subset with PSA measurements at \( < 40 \) yr of age, they reported an even stronger association between the baseline PSA and later prostate cancer diagnosis (AUC: 0.74; \( p < 0.0005 \)).

The usefulness of baseline PSA testing was also examined in 13 943 men \( \leq 60 \) yr of age from a large prospective prostate cancer screening trial in the United States, in which biopsy was performed for a PSA >2.5 ng/ml or suspicious findings on DRE [35]. High-risk men in their 40s with a baseline PSA level between the age-specific median (0.7 ng/ml) and the biopsy threshold of 2.5 ng/ml had a 14.6-fold increased risk of prostate cancer compared with those with a baseline PSA below the age-specific median. Similarly, compared with men in their 50s with a PSA below the age-specific median (0.9 ng/ml), those with a PSA from 0.9 to 2.5 ng/ml had a 7.6-fold increased risk of prostate cancer. Taken together, these studies suggest that baseline PSA levels at a young age provide useful information regarding the risk of future prostate cancer diagnosis. Indeed, across distinct populations, higher baseline PSA values were associated with an increased prostate cancer risk over the next 20–25 yr.

### 3.2. Baseline prostate-specific antigen at a young age and prognosis

Several studies have evaluated the association between baseline PSA levels with prostate cancer aggressiveness. For example, in men from Northern Ireland, Connolly et al. showed a direct relationship between increasing baseline PSA levels with the risk of high-grade disease and prostate cancer–specific mortality [32].

In the Malmo Preventive Project population, there was not only a strong relationship between baseline PSA levels with the overall risk of clinically detected prostate cancer but also a significant association with the risk of aggressive disease. In fact, Lilja et al. showed an even stronger relationship between baseline PSA levels at 44–50 yr of age with advanced prostate cancer at diagnosis (AUC: 0.751). Overall, 81% of advanced prostate cancers at diagnosis occurred in men with baseline PSA levels above the population median (0.63 ng/ml) [34].

Vickers et al. performed a nested case-control study of 1167 men from the Malmo Preventive Project born in 1921, who provided blood samples at 60 yr of age and were followed through 2006 [36]. The baseline PSA level at 60 yr of age was significantly associated with subsequent prostate cancer metastasis (AUC: 0.86; \( p < 0.001 \)) and disease-specific mortality (AUC: 0.90; \( p < 0.001 \)). In this study, 90% of those with metastases and 95% of prostate cancer deaths occurred in men with a PSA level greater than or equal to the median (1 ng/ml) at 60 yr of age. This led the authors to suggest a threshold of 1 ng/ml to focus subsequent screening efforts.

Similarly, Kuller et al. examined stored serum samples collected from 1973 to 1975 as part of the Multiple Risk Factor Intervention Trial in men 35–57 yr of age at baseline, who were then followed through 1999 [37]. After matching 63 men who died from prostate cancer with 63 controls who did not, they found significantly higher baseline PSA levels in the group who died from prostate cancer at an average of 18 yr later (mean: 1.1 ng/ml controls versus 2.84 ng/ml fatal prostate cancer; \( p = 0.002 \)).

In another recent study from the Duke Prostate Center Database, Tang et al. reported on 4568 men diagnosed with prostate cancer \( \geq 6 \) mo after a baseline PSA measurement, and evaluated its relationship with disease-specific mortality [38]. On ROC analysis, the AUC was 0.839 for the discrimination of prostate cancer–specific mortality using baseline PSA. On multivariable analysis, the baseline PSA level, an older age at the baseline PSA test, and African American race were all significantly associated with death from prostate cancer. A competing-risk estimate revealed a greater proportion of all-cause mortality contributed by prostate cancer as the baseline PSA increased.

### 3.3. Limitations of baseline prostate-specific antigen testing at a young age

Although practice patterns related to PSA testing vary widely, PSA screening for men in their 40s is not commonly performed worldwide. Even in the United States where prostate cancer screening is widespread, only 22.5% of men 40–49 yr of age reported a PSA test in the last year, compared with 53.7% of men \( \geq 50 \) yr of age in the 2002 Behavioral Risk Factor Surveillance System [39]. Screening in the 40s was more frequent in young black non-Hispanic men compared with white non-Hispanic men. Other studies have reported that screening at a young age is significantly more common in obese men [40] as well as in individuals with a family history of prostate cancer [41], consistent with several professional guidelines.
Autopsy studies have shown that the incidence of prostate cancer is age related [42]. For example, in a population of US organ donors, Yin et al. reported only a 0.5% incidence of autopsy-detected prostate cancer among men <49 yr of age [43]. By contrast, in a European population, Soos et al. reported histologic prostate cancer on autopsy in 0%, 15%, and 26.6% of men 18–30, 31–40, and 41–50 yr of age, respectively [44].

Although prostate cancer deaths begin to occur in the early 40s based on data from the Surveillance Epidemiology and End Results program, it is a relatively infrequent event [8]. Of all prostate cancer deaths from 2003 to 2007, only 0.1% and 1.4% were reported in men 35–44 and 45–54 yr of age, respectively. Indeed, the average ages at prostate cancer diagnosis and death were 67 yr and 80 yr, respectively. It is unclear whether a proportion of these later deaths could have been prevented through earlier screening. However, the results of this review suggest that baseline PSA measurements in men in their 40s provide valuable prognostic information.

Because numerous professional guidelines recommend earlier screening for men with a positive family history, performing a baseline PSA measurement on men in their 40s would seem rational, since it is a more robust predictor of risk than family history. Certainly the risk-to-benefit ratio of early PSA testing may be very different in specific high-risk subpopulations (eg, BRCA mutation carriers) [45].

Nevertheless, several authors have expressed strong concern that initiating earlier prostate cancer screening in average-risk young men might increase overdiagnosis with resultant overtreatment. Although active surveillance is increasingly offered among the standard management options for low-risk localized prostate cancer and may help reduce overtreatment, most programs are specifically targeted toward men with a more limited life expectancy [46]. Thus it is unclear how useful this strategy would be with a shift to earlier ages at diagnosis.

In addition, there is evidence from randomized trials demonstrating a survival advantage of radical prostatectomy over watchful waiting for men <65 yr of age [47]. Treatment-related morbidity is also significantly lower in younger men, including lower rates of incontinence and erectile dysfunction [48]. These combined findings suggest that active treatment may be more effective, with less associated side effects, in younger men.

However, another potential limitation to starting regular screening in the 40s is the large number needed to screen to diagnose a single case of prostate cancer [32]. For example, in a study with 1 178 high-risk men in their 40s who underwent screening, only 48 (4%) underwent a biopsy and 26 (2%) were diagnosed with prostate cancer during a mean follow-up of 38 mo [35]. Because these men were high risk (African American or positive family history), these rates would likely be even lower in an unselected population. In an Austrian screening study from 1993 to 1994, 2054 men were enrolled at 45–49 yr of age, and only 28 (1.4%) underwent a biopsy with three prostate cancers detected [49].

It is noteworthy that the benefits of screening have been shown to differ based on the initial PSA level. Van Leeuwen et al. compared 43 987 men 55–74 yr of age from the screening arm of the ERSPC to 42 503 men from the population of Northern Ireland where screening is uncommon [50]. Although prostate cancer–specific mortality was lower in the ERSPC screening group compared with the Northern Ireland group, the adjusted absolute difference in prostate cancer–specific mortality between the groups increased with higher baseline PSA values.

If the exclusive objective of PSA testing at a young age was to detect early prostate cancers, this could imply a large number of unnecessary PSA tests in young men depending on their baseline PSA level. However, if the primary purpose is risk assessment and establishing a baseline PSA and/or PSA velocity, these measurements would instead provide important prognostic information. Indeed, multiple studies have reported a higher AUC on ROC analysis for prostate cancer detection with both PSA and PSA velocity measurements in men in their 40s compared with those ≥50 yr of age [51,52]. This finding has been possibly attributed to improved performance characteristics in younger men before the onset of BPH and other confounding conditions.

Baseline measurements also may be put to practical use in guiding the subsequent screening strategy. For example, Loeb et al. reported that men in their 40s with a PSA above the age-specific median were 1.96-fold (p = 0.007) and 2.1-fold (p < 0.0001) more likely to reach the biopsy threshold (2.5 ng/ml) by 1 and 5 yr, respectively, compared with those with baseline levels below the age-specific median [53]. This study demonstrated that baseline PSA testing in the 40s led to relatively few additional biopsies but did identify patient subgroups with strikingly different prostate cancer risk profiles.

In 30 495 men from the PLCO trial, Crawford et al. evaluated the relationship between baseline PSA levels at 55–74 yr of age and subsequent PSA levels during the next 5 yr [54]. Among participants with baseline PSA levels <1, 1–2, 2–3, and 3–4 ng/ml, the rates of conversion to a PSA >4 ng/ml by 5 yr were 1.5%, 7.4%, 33.5%, and 79%, respectively. Based on these findings, the authors concluded that a reduced testing frequency may be explored for men with a baseline PSA <1 ng/ml. Because the study population did not include men in their 40s, however, the generalizability of these findings to younger men is unclear.

Nevertheless, these results suggest that baseline PSA levels may be useful to determine patient subgroups in whom a longer screening interval might be appropriate. For example, Ross et al. performed a Monte Carlo simulation based on a Markov model to assess the comparative efficiency of different PSA screening approaches, by comparing the number of prostate cancer deaths prevented per the number of PSA tests and prostate biopsies [55]. The model suggested that a strategy of screening men at 40 and 45 yr of age followed by biennial screening beginning at 50 yr of age (or in the 40s if PSA >2 ng/ml) was more efficient than annual screening starting at 50 yr of age. Overall, additional prospective studies are necessary to determine the optimal screening frequency and triggers for biopsy for men in their
40s with a sufficient life expectancy who elect to undergo screening. A limitation of this review is that randomized trials have not been performed to evaluate PSA screening for men in their 40s. Additionally, it is possible that verification bias affected the results. However, it is encouraging that baseline PSA measurements were significantly associated with prostate cancer risk and prognosis in multiple populations involving clinically diagnosed cases, in a setting where PSA screening was not commonly practiced.

4. Conclusions

Currently, a minority of men in their 40s undergoes PSA testing, and several professional organizations do not recommend considering PSA screening until age 50. However, baseline PSA measurements at a young age are stronger predictors of prostate cancer risk than race and family history. Baseline PSA measurements at a young age are robust predictors of aggressive prostate cancer, metastasis, and disease-specific mortality many years later. Thus baseline PSA testing might be useful for risk stratification and to individualize screening protocols.

Author contributions: Stacy Loeb had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Loeb, Carter, Catalona, Moul, Schroder. Acquisition of data: Loeb, Moul, Schroder. Analysis and interpretation of data: Loeb, Carter, Catalona, Moul, Schroder. Drafting of the manuscript: Loeb, Schroder. Critical revision of the manuscript for important intellectual content: Loeb, Carter, Catalona, Moul, Schroder. Statistical analysis: None. Obtaining funding: None. Administrative, technical, or material support: Loeb. Supervision: Carter, Catalona, Moul, Schroder. Other (specify): None.

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References


