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The effect of start and stop age at screening on the risk of being diagnosed with prostate cancer

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Abstract

Purpose—The aim of this study was to investigate the effect of age and number of screens on the risk of prostate cancer (PCa) diagnosis.

Materials and Methods—The Göteborg randomized population-based PCa screening trial has, since 1995, invited men biennially for prostate-specific antigen (PSA)-testing, until the upper age limit 70 years. Men with a PSA-level above the threshold 2.5 ng/ml were recommended further work-up including 10-core biopsy (sextant before 2009). The present study comprises 9,065 men born 1930–43 (1944 excluded due to different screening algorithm). Complete attendees were defined as men who accepted all screening invitations (maximum 3–9 invitations). Cumulative incidence of PCa was calculated using standard methods.

Results—Of the 3,488 (38%) complete attendees, 667 were diagnosed with PCa (follow-up 1995–30 Jun 2014). At the age 70, there was no significant difference in PCa risk between those who started screening at the age of 52 (9 screens), 55 (7 screens) or 60 (5 screens) years. However, the cumulative risk of PCa diagnosis increased dramatically with age and was 7.9% at age 60, 15% at age 65 and 21% at age 70, for men who had been screened 4 times.

Conclusion—There was no clear association between risk of PCa and the number of screens. Starting screening at an early age appears to advance the time of PCa diagnosis but does not seem to increase the risk of being diagnosed with the disease. Age at termination of screening is strongly associated with the risk of being diagnosed with PCa.

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Keywords

prostate neoplasms; prostate-specific antigen; risk factors; mass screening

Introduction

Screening for prostate cancer (PCa) with prostate-specific antigen (PSA) ought to be a careful balance, where the beneficial effects of screening; a reduced rate of advanced disease and PCa mortality,^{1, 2} have to be weighed against a number of negative consequences such as psychological harms during the screening process, complications to prostate biopsies, overdiagnosis and overtreatment.³ Overdiagnosis and overtreatment are considered the main harms of screening.³ In addition to unnecessary health-care costs, overdiagnosis and overtreatment turn “healthy” men into patients and risk negatively affecting their quality-of-life due to side-effects from curative treatment.^{4–7}

Similar to breast and cervical cancer screening, organized PSA screening within the framework of a program might be more effective in reducing PCa mortality and associated with less overdiagnosis than opportunistic screening.⁸ However, the optimal screening program, one that shifts the ratio of benefits to harms by maximizing the mortality reduction at a minimal rate of overdiagnosis, has yet to be determined. The risk of overdiagnosis is not fixed, but varies with a number of factors that relate to the screening test and the algorithm, the population screened and the disease being screened for.⁹ Previous studies have indicated that the PSA-threshold, the screening frequency/interval and increasing age at screening are important risk factors for overdiagnosis.^{6, 10–12}

In the present study, we sought to investigate the effect of age and the number of screening occasions on the risk of being diagnosed with PCa in the Göteborg randomized population-based PCa screening trial.

Materials and Methods

The Göteborg screening trial,¹ (ISRCTN54449243), which forms the basis of the present study, was approved by the Ethical committee at the University of Göteborg in 1994 and started in 1995. Since 1996, the study has contributed to the Swedish arm of the European Randomized Study of Screening for Prostate Cancer (ERSPC).

As of December 31, 1994 there were 32,298 men living in the city of Göteborg, born between 1930 and 1944 (age 50–64), of whom 10,000 men were computer randomized to a screening group and 10,000 to a control group. Men with prevalent PCa at randomization were excluded, as were those who deceased or emigrated before randomization (Figure 1). Men randomized to screening received a letter detailing the advantages and disadvantages of PCa screening together with an invitation to PSA-testing every second year. Men with a PSA-level above the cut-off (3.4 ng/ml in 1995–1998, 2.9 ng/ml in 1999–2005 and 2.5 ng/ml from 2005) were invited for further urological examination including digital rectal examination, trans-rectal ultrasound and laterally directed sextant biopsy (10-core biopsy from 2009). Men with a PSA-level below threshold and men with a benign biopsy were re-

invited after two years. The upper age-limit for screening was 70 years and invitation for screening terminated at a mean age of 69 years (67–71). A detailed description of the Göteborg screening trial has previously been reported.¹

Complete attendees were defined as men in the screening group who accepted all screening invitations and incomplete attendees were defined as men accepting at least one screening invitation. Complete attendees who were diagnosed with PCa, who deceased or emigrated within 2 years of their last screen, were also regarded as complete attendees. Because men were in the age range 50 to 64 years at the start of screening, the maximum number of screens for each age cohort were as follows; 3 screens for men born 1930–31, 4 for men born 32–33, 5 for men born 34–35, 6 for men born 36–37, 7 for men born 38–39, eight for men born 1940–41, nine for men born 1942–43, and ten for men born 1944. Men born 1944 were excluded from this study because they followed a separate algorithm as part of a pilot side-study embedded within the 10th screening round, which in addition to a lower cut-off for biopsy also incorporated magnetic resonance imaging and targeted biopsies.

The screening database, containing both the screening and control group, is linked to the Swedish Cancer Register and to the Population Register four times yearly to identify men diagnosed with PCa and men who has deceased or emigrated. Last date of follow-up in the present study was June 30, 2014. For all men diagnosed with PCa, medical records were retrieved.

Cumulative PCa incidence for the entire study population (n=9,065) was calculated as 1 minus the Kaplan Meier estimator.¹³ Cumulative PCa incidence for complete attendees was calculated for each age cohort with the Kaplan-Meier method and competing risk estimates were calculated using the stcomp macro for Stata described by Gooley et al.¹⁴ All cases of PCa, regardless of method of diagnosis (screen-detected or interval cancers) were included for analysis. Interval cancers were defined as PCa diagnosed within 2 years of the last screening occasion. The follow-up time was calculated as time from date of randomization to date of PCa diagnosis or date of censoring (date of death, emigration or last follow-up of June 30 2014). As the number of men with prevalent PCa was unevenly distributed across the age cohorts these cases were added to each cohort when calculating cumulative PCa incidence. Statistical analyses were performed with STATA Statistical Software 13.1 (StataCorp, College Station, TX, USA) and IBM SPSS Statistics 20 (IBM, IBM Corp., Somers, NY, USA).

Results

With a follow-up until Jun 30 2014, 3,488 (38%) men were complete attendees to all screening invitations, 3,470 (38%) were incomplete attendees and 2,107 (23%) never attended (Figure 1). The proportion of men in each cohort who were complete attendees ranged from 29–52% (Table 1). The number of PSA-tests varied more between the cohorts than did the proportion of men who underwent a biopsy or who received a PCa diagnosis (Table 1). Of those with a positive screening test, 87% complied with the biopsy recommendation.

A total of 667 (19%) cases of PCa (screen-detected and interval cancers) were diagnosed among complete attendees and 310 (8.9%) cancers among incomplete attendees (Table 1). The majority of PCa were diagnosed during the first two biopsy occasions; very few cases were detected in men with more than four negative biopsies (Table 2). For the entire cohort, the median age at diagnosis was 65.1 years (IQR 61.9–67.5), higher for those who started screening later in life and stabilized around 63 years for those who started screening below 60 years of age (Table 1). Complete attendees were younger at diagnosis than incomplete attendees (65.0 years, versus 65.8 years). The distribution of tumor risk group varied between age cohorts. The proportion of low-risk PCa increased with the number of screens but also stabilized from those who started screening below the age of 60 and no further increase was observed in men with more screens (Table 1).

Prostate cancer incidence was strongly affected by age at termination of screening but not by the number of screens or age at the start of screening. The incidence reached a “steady state” after four screens and thereafter only increased with age but not with additional screens (Table 3). When men reached the upper age-limit for invitation (70 years) PCa incidence was similar between the age cohorts (Table 3 fifth column, Figure 2). The results were comparable with competing risk estimates (Figure 3). No marked rise in PCa incidence was observed when the PSA-threshold was lowered or the number of biopsy cores was increased, indicating that these factors had minor or no influence on the incidence rate (Figure 4).

Discussion

Prostate cancer screening has potential to reduce morbidity from advanced disease and PCa mortality but is associated with a considerable overdiagnosis.² With up to 50% of all screen-detected PCa being overdiagnosed¹⁰ we must find “smarter” screening strategies.¹⁵ The results from this study show that starting screening at an early age advances the time of diagnosis but does not seem to increase the risk of PCa (Figure 2, 3 and Table 3 fifth column) This is a surprising finding as it could be hypothesized that each screening occasion would be associated with a certain risk of PCa diagnosis and that starting early therefore would increase the overall risk of PCa diagnosis. Risk of PCa was however strongly associated with age at termination of screening; the cumulative risk of being diagnosed with PCa after 4 screens at the age of 60 years was 7.9%, compared to 15% at 65 years and 21% at 70 years (Table 3).

The optimal age for participation in PCa screening is currently unknown and the Göteborg trial as well as the core age group of the ERSPC showed a benefit of screening men ages 50–70 and 55–69 years, respectively.² The Göteborg screening trial is the only high-quality randomized controlled trial that has evaluated PSA screening also for men 50–55 years.¹ In the present study, there was no significant difference in PCa incidence at termination of screening regardless if men started screening at age 51, 55 or 60 and underwent nine, seven or five screens (Table 3 fifth column). This is also supported by the fact that the proportion of low-risk tumors was similar in the age cohorts that started screening below the age of 60 (Table 1). This is an important finding as the lower starting age is thought to be one of the beneficial features, together with the long follow-up, of the larger relative risk reduction in PCa mortality seen in the Göteborg trial as compared to the ERSPC trial.^{1,2} In addition,

lowering the PSA-threshold and increasing the number of biopsy cores, appeared to have a marginal effect on the risk of PCa diagnosis as no change in the shape of the incidence curve was observed after implementations of these changes (Figure 4). While age at start of screening seems to be of minor importance for overdiagnosis, our results indicate that age at stop of screening is strongly associated with risk of PCa diagnosis and probably also the risk of overdiagnosis.

Our findings are consistent with a recent modeling study comparing different screening policies. In that study, decreasing the stopping age had a larger effect on overdiagnosis than reducing the screening frequency or basing re-screening algorithms on PSA-levels and age. However, increasing the starting age had only a minor effect on overdiagnosis. The authors concluded that stopping screening at the age of 70 years would be a reasonable way to reduce the harms and keep the benefits of PCa screening.¹² A study by Schaeffer et al. showed that men 75 to 80 years old with a “normal” PSA (<3 ng/mL) are unlikely to develop life threatening PCa during their remaining life, indicating that PSA-testing can be discontinued for these men.¹⁶ Gulati et al. recently developed a model-based nomogram to predict an individual’s risk of being overdiagnosed. Age had the largest effect on risk of overdiagnosis and each additional year of age at diagnosis increased the odds of overdiagnosis by 12.9%.¹⁷

On the other hand, a general recommendation to stop screening for all men at the age of 70 year may miss preventable morbidity and mortality. Today, the median age of dying from PCa in Sweden is 80 years and the remaining life-expectancy for Swedish men 65 years of age is almost 19 years.¹⁸ A previous report from the Göteborg study has indicated that the protective effect of screening lasts approximately nine years after screening termination.¹⁹ One alternative to discontinuing screening for all men at the age of 70 years would be to apply individual cessation ages based on for example comorbidity status. Landsdorp-Voogelar et al. reported in a study that the optimal age for screening cessation based on comorbid conditions varied with a 10 year interval around the age cut point of 74 years.²⁰ These findings are also supported in a quality-of-life analysis from the ERSPC where it was found that screening until age 75 years for men with low comorbidity has approximately the same adjustments in quality-of-life as screening until the age of 69 for the general population.⁶ Another strategy could be to risk stratify men to individualized screening intervals and stopping ages based on PSA-levels in early to late midlife.^{21–23}

Our data also suggests that a shorter screening interval will not lead to any substantial increase in overdiagnosis as the risk of PCa diagnosis was similar regardless if men had been screened five or nine times (Table 3). These findings are consistent with a recent cost-effectiveness analysis from the ERSPC which showed that much of the overdiagnosis was already covered by a four-year interval and that shortening the screening interval only had minor effect on overdiagnosis, yet resulted in a larger reduction in PCa mortality.²⁴ Another ERSPC study showed that screening with a 2-year interval significantly reduced the incidence of advanced PCa compared to a 4-year interval but that a 2-year interval also increased the overall risk of PCa.²⁵

The main strength of this study is the population-based design inviting a previously unscreened group of men.¹ In addition, the study population comprises men who started screening early in life, who have attended repeated screening up to nine times and been followed up to 20 years. This allowed a unique possibility to evaluate the long-term effects of repeated PSA screening starting at different ages. Another strength is that competing risk analyses were performed to rule out that the results were not biased due to different risks of competing causes of death in the age cohorts of these ageing men (Figure 2 and 3). A third strength is the completeness of the data; the Swedish Cancer Registry has an almost complete coverage.²⁶ Because the risk of overdiagnosis could not be measured directly in this material the cumulative risk of PCa were used as an estimation, which is a limitation. A high PCa incidence may not correspond to a high risk of overdiagnosis, even though it is likely. As expected, the number of men with prevalent PCa and the number of men who had already died from PCa before study start were not evenly distributed between the different age cohorts, but were more common in the oldest cohorts. We tried to control for this by adding in the prevalent cases of PCa to each cohort. We could not control for PCa deaths occurring before study start, however, the number of PCa death before study start in each age cohort was small. According to the Swedish National Board of Health and Welfare, 32 men born 1930–44 died from PCa between 1980 and study start in 1995 (Hornblad J. National Board of Health and Welfare, personal communication, 24 Feb 2015).

Conclusion

In conclusion, starting screening at an early age appears to advance the time of PCa diagnosis but does not seem to increase the risk of being diagnosed with the disease, indicating that starting early does not increase the risk of overdiagnosis. However, age at termination of screening seems to be strongly associated with risk of being diagnosed with PCa. To reduce the risk of overdiagnosis and keep the benefit, screening must be performed more selectively among older men. When to stop screening should be an individual decision between the doctor and patient based on risk factors such as baseline PSA, comorbidity status and remaining life-expectancy.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. Hugosson J, Carlsson S, Aus G, et al. Mortality results from the Göteborg randomised population-based prostate-cancer screening trial. *Lancet Oncol.* 2010; 11:725. [PubMed: 20598634]

2. Schroder FH, Hugosson J, Roobol MJ, et al. Screening and prostate cancer mortality: results of the European Randomised Study of Screening for Prostate Cancer (ERSPC) at 13 years of follow-up. *Lancet*. 2014; 384:2027. [PubMed: 25108889]
3. Moyer VA. U. S. Preventive Services Task Force. Screening for prostate cancer: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2012; 157:120. [PubMed: 22801674]
4. Punnen S, Cowan JE, Chan JM, et al. Long-term Health-related Quality of Life After Primary Treatment for Localized Prostate Cancer: Results from the CaPSURE Registry. *Eur Urol*. 2015; 68:600. [PubMed: 25242555]
5. Davis KM, Kelly SP, Luta G, et al. The association of long-term treatment-related side effects with cancer-specific and general quality of life among prostate cancer survivors. *Urology*. 2014; 84:300. [PubMed: 24975711]
6. Heijnsdijk EA, Wever EM, Auvinen A, et al. Quality-of-life effects of prostate-specific antigen screening. *N Engl J Med*. 2012; 367:595. [PubMed: 22894572]
7. Carlsson S, Aus G, Bergdahl S, et al. The excess burden of side-effects from treatment in men allocated to screening for prostate cancer. The Goteborg randomised population-based prostate cancer screening trial. *Eur J Cancer*. 2011; 47:545. [PubMed: 21087857]
8. Arnsrud Godtman R, Holmberg E, Lilja H, et al. Opportunistic testing versus organized prostate-specific antigen screening, outcome after 18 years in the Gothenburg Randomized Population-Based Prostate Cancer Screening Trial. *Eur Urol*. 2015; 68:354. [PubMed: 25556937]
9. Loeb S, Bjurlin MA, Nicholson J, et al. Overdiagnosis and Overtreatment of Prostate Cancer. *Eur Urol*. 2014; 65:1046. [PubMed: 24439788]
10. Draisma G, Boer R, Otto SJ, et al. Lead times and overdiagnosis due to prostate-specific antigen screening: estimates from the European Randomized Study of Screening for Prostate Cancer. *J Natl Cancer Inst*. 2003; 95:868. [PubMed: 12813170]
11. Gulati R, Gore JL, Etzioni R. Comparative effectiveness of alternative prostate-specific antigen--based prostate cancer screening strategies: model estimates of potential benefits and harms. *Ann Intern Med*. 2013; 158:145. [PubMed: 23381039]
12. de Carvalho TM, Heijnsdijk EA, de Koning HJ. Screening for prostate cancer in the US? reduce the harms and keep the benefit. *Int J Cancer*. 2015; 136:1600. [PubMed: 25123412]
13. Kaplan EL, Meier P. Nonparametric estimation from incomplete observations. *J Am Stat Assoc*. 1958; 53:457.
14. Gooley TA, Leisenring W, Crowley J et al. Estimation of failure probabilities in the presence of competing risks: new representations of old estimators. *Stat Med*. 1999; 18:695. [PubMed: 10204198]
15. Vickers A, Carlsson S, Laudone V, et al. It Ain't What You Do, It's the Way You Do It: Five Golden Rules for Transforming Prostate-Specific Antigen Screening. *Eur Urol*. 2014; 66:188. [PubMed: 24411991]
16. Schaeffer EM, Carter HB, Kettermann A, et al. Prostate specific antigen testing among the elderly--when to stop? *J Urol*. 2009; 181:1606. [PubMed: 19246059]
17. Gulati R, Inoue LY, Gore JL, et al. Individualized estimates of overdiagnosis in screen-detected prostate cancer. *J Natl Cancer Inst*. 2014; 106:367.
18. Statistiska, centralbyrån. Statistics Sweden. Life expectancy by sex and age. Year 2013–2110. [cited; Nov 12, 2014] Available from: www.scb.se.
19. Grenabo Bergdahl A, Holmberg E, Moss S, et al. Incidence of prostate cancer after termination of screening in a population-based randomised screening trial. *Eur Urol*. 2013; 64:703. [PubMed: 23721957]
20. Lansdorp-Vogelaar I, Gulati R, Mariotto AB, et al. Personalizing age of cancer screening cessation based on comorbid conditions: model estimates of harms and benefits. *Ann Intern Med*. 2014; 161:104. [PubMed: 25023249]
21. Lilja H, Cronin AM, Dahlin A et al. Prediction of significant prostate cancer diagnosed 20 to 30 years later with a single measure of prostate-specific antigen at or before age 50. *Cancer*. 2011; 117:1210. [PubMed: 20960520]

22. Loeb S, Carter HB, Catalona WJ, et al. Baseline prostate-specific antigen testing at a young age. *Eur Urol*. 2012; 61:1. [PubMed: 21862205]
23. Carlsson S, Assel M, Sjoberg D, et al. Influence of blood prostate specific antigen levels at age 60 on benefits and harms of prostate cancer screening: population based cohort study. *BMJ*. 2014; 348:g2296. [PubMed: 24682399]
24. Heijnsdijk EA, de Carvalho TM, Auvinen A, et al. Cost-effectiveness of Prostate Cancer Screening: A Simulation Study Based on ERSPC Data. *J Natl Cancer Inst*. 2015; 107:366. [PubMed: 25505238]
25. van Leeuwen PJ, Roobol MJ, Kranse R, et al. Towards an optimal interval for prostate cancer screening. *Eur Urol*. 2012; 61:171. [PubMed: 21840117]
26. Barlow L, Westergren K, Holmberg L, et al. The completeness of the Swedish Cancer Register: a sample survey for year 1998. *Acta Oncol*. 2009; 48:27. [PubMed: 18767000]

Figure 1.
CONSORT diagram of the Göteborg Randomized Population-Based Prostate-Cancer Screening Trial (PSA=prostate-specific antigen)

Figure 2.
Cumulative incidence of prostate cancer among complete attendees stratified by age cohorts,
Kaplan-Meier estimates (number of men included in the analysis=3,488)

Figure 3.

Cumulative incidence of prostate cancer stratified by age cohort among complete attendees, competing risk estimates (number of men included in the analysis=3,488)

Figure 4.
Cumulative incidence of prostate cancer (number of men included in analysis=9,065)

Table 1

Number of PSA-tests, biopsies and prostate cancers (screen-detected and interval cancers)

Birth cohort	Number of men	Number of complete attendees	Number of PSA-tests	Number of PSA-tests, complete attendees	Number and proportion of men biopsied	Number and proportion of men biopsied, complete attendees	Number of biopsies	Number of biopsies, complete attendees		Number of prostate cancers (median age at diagnosis)	Number of prostate cancers, complete attendees (median age at diagnosis)
1930–31	1108	580	1943	1601	241 (22%)	186 (32%)	366	301		96 (67.1)	84 (66.9)
									Low	46	41 (7.1%)
									Intermediate	36	32 (5.5%)
									High	10	7 (1.2%)
									Advanced	4	4 (0.7%)
1932–33	1106	523	2496	1882	264 (24%)	179 (34%)	425	303	Unknown	0	0
									Low	54	49 (9.4%)
									Intermediate	40	29 (5.5%)
									High	9	9 (1.7%)
									Advanced	5	3 (0.6%)
1934–35	1083	467	3056	2134	270 (25%)	171 (37%)	485	325	Unknown	0	0
									Low	65	53 (11%)
									Intermediate	47	36 (7.7%)
									High	7	5 (1.1%)
									Advanced	2	2 (0.4%)
1936–37	1190	475	3789	2506	295 (25%)	182 (38%)	533	358	Unknown	1	1 (0.2%)
									Low	92	66 (14%)
									Intermediate	43	34 (7.2%)
									High	5	3 (0.6%)

Birth cohort	Number of men	Number of complete attendees	Number of PSA-tests	Number of PSA-tests, complete attendees	Number and proportion of men biopsied	Number and proportion of men biopsied, complete attendees	Number of biopsies	Number of biopsies, complete attendees		Number of prostate cancers (median age at diagnosis)	Number of prostate cancers, complete attendees (median age at diagnosis)
									Advanced	2	2 (0.4%)
									Unknown	0	0
1938–39	1409	480	5136	2943	366 (26%)	190 (40%)	731	411		146 (64.9)	91 (63.6)
									Low	88	58 (12%)
									Intermediate	49	30 (6.3%)
									High	5	2 (0.4%)
									Advanced	2	0
1940–41	1453	467	6018	3280	373 (26%)	167 (36%)	753	382	Unknown	2	1 (0.2%)
										156 (63.7)	93 (62.5)
									Low	103	60 (13%)
									Intermediate	41	26 (5.6%)
									High	10	6 (1.3%)
1942–43	1716	496	7749	3878	456 (27%)	197 (40%)	918	454	Advanced	0	0
									Unknown	2	1 (0.2%)
										207 (63.5)	107 (62.9)
									Low	120	64 (13%)
									Intermediate	71	35 (7.1%)
Total	9065	3488	30187	18224	2265	1272	4211	2534	High	14	7 (1.4%)
									Advanced	1	0
									Unknown	1	1 (0.2%)
										977	667

Table 2

Screen-detected prostate cancer divided by age cohort and biopsy round (interval cancers not included). Proportions within parenthesis represent prostate cancer detection rates at that biopsy round.

Birth cohort	Biopsy round								
	1	2	3	4	5	6	7	8	9
1930–31	245 biopsies 61 cancers (25%)	102 biopsies 24 cancers (24%)	26 biopsies 2 cancers (7.7%)						
1932–33	267 biopsies 72 cancers (27%)	113 biopsies 28 cancers (25%)	39 biopsies 3 cancers (7.7%)	12 biopsies 0 cancers -					
1934–35	270 biopsies 77 cancers (29%)	114 biopsies 26 cancers (23%)	58 biopsies 8 cancers (14%)	31 biopsies 2 cancers (6.5%)	12 biopsies 2 cancers (17%)				
1936–37	297 biopsies 89 cancers (30%)	125 biopsies 29 cancers (23%)	63 biopsies 9 cancers (14%)	29 biopsies 5 cancers (17%)	17 biopsies 2 cancers (12%)	4 biopsies 0 cancers -			
1938–39	366 biopsies 101 cancers (28%)	165 biopsies 22 cancers (13%)	93 biopsies 8 cancers (8.6%)	56 biopsies 2 cancers (3.6%)	27 biopsies 3 cancers (11%)	16 biopsies 1 cancer (6.3%)	6 biopsies 0 cancers -		
1940–41	372 biopsies 91 cancers (24%)	178 biopsies 32 cancers (18%)	101 biopsies 21 cancers (21%)	51 biopsies 5 cancers (9.8%)	27 biopsies 1 cancer (3.7%)	12 biopsies 0 cancer -	7 biopsies 0 cancer -	3 biopsies 0 cancer -	
1942–43	456 biopsies 123 cancers (27%)	213 biopsies 33 cancers (15%)	117 biopsies 28 cancers (24%)	60 biopsies 9 cancers (15%)	36 biopsies 6 cancers (17%)	18 biopsies 2 cancers (11%)	8 biopsies 0 cancers -	3 biopsies 0 cancers -	1 biopsy 0 cancers -
Total number of biopsies and prostate cancers	2273/614	1010/194	497/79	239/23	119/14	50/3	21/0	6/0	1/0

Table 3

Number of complete attendees in each age cohort and cumulative incidences of prostate cancer (screen-detected and interval cancers) at different ages. Within parenthesis is the number of screens. Prevalent cases of prostate cancers are added to each age cohort.

Birth cohort	Number of complete attendees	Cumulative incidence at 60 years	Cumulative incidence at 65 years	Cumulative incidence at 70 years
1930–31	580		6.4 (1)	16.2 (3) (95% CI: 13.5–19.4)
1932–33	523		8.5 (2)	18.2 (4) (95% CI: 16.0–22.7)
1934–35	467	1.4 (1)	12.7 (3)	20.6 (5) (95% CI: 17.2–24.7)
1936–37	475	5.8 (1–2)	14.8 (4)	23.2 (6) (95% CI: 19.6–27.3)
1938–39	480	6.8 (2–3)	12.9 (5)	20.9 (7) (95% CI: 17.5–24.9)
1940–41	467	7.4 (3–4)	15.7 (6)	20.8 (8) (95% CI: 17.1–24.9)
1942–43	496	8.4 (4–5)	15.9 (7)	23.1 (9) (95% CI: 19.5–27.2)
Cumulative incidence for those with 4 screens (all cohorts)		7.9% (95% CI: 6.2–10.1)	15.0% (95% CI: 13.5–16.7)	21.2% (95% CI: 19.7–22.8%)