Published in final edited form as:

Cancer Causes Control. 2013 February ; 24(2): 383–390. doi:10.1007/s10552-012-0124-6.

Risk of Localized and Advanced Prostate Cancer Among Immigrants Versus Native-Born Swedish Men: A nation-wide, population-based study

Stacy Loeb, MD, Linda Drevin, PhD, David Robinson, MD PhD, Erik Holmberg, PhD, Sigrid Carlsson, MD PhD, Mats Lambe, MD PhD*, and Pär Stattin, MD PhD*

Department of Urology (SL), New York University, NY, NY, the Regional Cancer Centre (LD, ML), Uppsala University Hospital, Uppsala, Sweden, the Department of Urology (DR), Jönköping County Council, Sweden, the Regional Cancer Centre (EH), Sahlgrenska University Hospital, Gothenburg, Sweden, the Department of Oncology (EH) and Urology (SC), Sahlgrenska Academy at University of Gothenburg (SC), Sweden, the Department of Surgery, Urology Service, Memorial Sloan-Kettering Cancer Center (SC, PS), New York, New York, USA, the Department of Surgical and Perioperative Sciences (PS), Urology and Andrology, Umeå University, Umeå, Sweden, and the Department of Medical Epidemiology and Biostatistics (ML), Karolinska Institutet, Stockholm, Sweden

Abstract

Purpose—Prostate cancer (PCa) incidence and prognosis vary geographically. We examined possible differences in PCa risk by clinical risk category between native-born and immigrant populations in Sweden. Our hypothesis was that lower PSA-testing uptake among foreign-born men would result in lower rates of localized disease, and similar or higher risk of metastatic disease.

Methods—Using the Prostate Cancer database Sweden (PCBaSe), we identified 117,328 men with PCa diagnosed from 1991–2008, of which 8,332 were foreign-born. For each case, 5 cancer-free matched controls were randomly selected from the population register. Conditional logistic regression was used to compare low-risk, intermediate-risk, high-risk, regionally metastatic, and distant metastatic PCa based upon region of origin.

Results—Across all risk categories, immigrants had significantly lower PCa risk than native-born Swedish men, except North Americans and Northern Europeans. The lowest PCa risk was observed in men from the Middle East, Southern Europe and Asia. Multivariable adjustment for socioeconomic factors and comorbidities did not materially change risk estimates. Older age at immigration and more recent arrival in Sweden were associated with lower PCa risk. Non-native men were less likely to be diagnosed with PCa through PSA-testing during a health check-up.

Conclusions—The risk for all stages of PCa was lower among first-generation immigrants to Sweden compared to native-born men. Older age at immigration and more recent immigration

Corresponding author: Stacy Loeb, MD, NYU Department of Urology, 550 1st Ave, VZ30, 6th floor (612), New York, NY 10016, Phone: 646-825-6358, Fax: 212-263-4549, stacyloeb@gmail.com.

*Mats Lambe and Pär Stattin shared equal responsibility as senior author.

were associated with particularly low risks. Patterns of PSA testing appeared to only partly explain the differences in PCa risk, since immigrant men also had a lower risk of metastatic disease.

Keywords

prostate cancer; immigrants; risk; prognosis; geography

Introduction

Migrant studies are frequently used to examine the role of genetic and environmental factors in disease etiology. Prior studies have shown divergent rates of numerous diseases between immigrant and native-born populations.[1–3]

Prostate cancer (PCa) incidence and mortality vary widely between populations.[4, 5] Together with results from migrant studies, this suggests that environment, lifestyle and dietary factors play important roles in PCa risk, in conjunction with diagnostic differences.[6]

High PCa incidence is reported in North America, particularly among African-Americans, whereas rates are relatively low in Asians.[7] Bray et al. reported a five-fold variation in age-adjusted PCa incidence rates between 24 European countries from 2001–2005, with the highest rates in Scandinavia.[8] In Sweden, men have a ~18% lifetime risk of a PCa diagnosis and PCa is the leading cause of male cancer death [9].

In 2008, PCa accounted for ~272,223 deaths worldwide i, with considerable regional variation.[7] The National Longitudinal Mortality Study demonstrated that the risk of life-threatening PCa also differs by country of origin. Specifically, immigrants to the US had a 33% lower PCa mortality risk compared to US-born men after demographic adjustments.[10]

Possible explanations include genetic differences and environmental exposures, including divergent dietary patterns. However, striking differences in incidence rates must also be interpreted in the context of diagnostic intensity and screening behavior. Thus, lower PCa incidence among immigrants may reflect lower healthcare access or utilization, particularly PSA testing.[11]

To better elucidate possible underlying mechanisms, we took advantage of detailed clinical information available in a large, nationwide database. To our knowledge, no prior studies in this area have included information on disease aggressiveness. This way, we were able to compare PCa risk by clinical risk category between native-born men and first-generation immigrants in Sweden. We hypothesized that lower PSA testing rates in foreign-born men would be reflected in lower incidence of localized PCa compared to Swedish men, while advanced disease would be similar or higher in foreign-born compared to Swedish-born men.

Materials and Methods

We conducted a register-based study using the Prostate Cancer database Sweden (PCBaSe) version 2.0, as previously described.[12] Briefly, the study population was identified in the National Prostate Cancer Register (NPCR) containing comprehensive information on >96% of incident PCa cases in Sweden including age, staging, Gleason score, PSA, primary treatment, and details on the evaluation leading to PCa diagnoses.[13, 14] Using the individually unique personal identity number assigned to all Swedish residents, NPCR was linked to numerous other health care and demographic nation-wide population-based registries including the Population Register with country of origin and date of immigration, the National Patient Register with inpatient care, and the LISA database with occupation, income, education and marital status etc.

In PCBaSe, for all men with PCa registered between 1991–1995, we selected two PCa-free men from the same county and birth year; for those registered between 1996–2008, we selected five PCa-free men from the same county and birth year. Thus, for each PCa case, PCBaSe 2.0 contains two or five matched men without PCa as a comparison cohort.

From PCBaSe, we identified 117,328 men with PCa diagnosed between 1991–2008 of which 8,332 were foreign-born. As described above, for each case, 2–5 controls were randomly selected from the Swedish population register, living in the same county and born the same year as the case, but free from PCa diagnosis at the end of the year that the index case was diagnosed. Approximately 10–15% of the Swedish population is comprised of first-generation immigrants, such that they are represented in the background population sample.

Using data from the National Patient Register[15], we calculated the Charlson Comorbidity Index (CCI) [16, 17]. Immigrants were also evaluated based upon age at immigration and duration of residence in Sweden. Categories were chosen based on prior studies, and because diagnoses during the first few years upon arrival may represent prevalent cases.[18]

To examine for incidence differences by risk category, PCa cases were further classified into five risk groups, based upon a modification of the NCCN guidelines: (1) low-risk (clinical stage T1–2, Gleason score ≤6 and PSA <10 ng/mL), (2) intermediate-risk (T1–2, Gleason 7 and/or PSA 10–20), (3) high-risk (T3 and/or Gleason 8–10 and/or PSA 20–50), (4) T4 and/or regionally metastatic disease (N1 and/or PSA 50–100 ng/mL without distant metastases) and (5) distant metastases (M1 and/or PSA ≥100).[19]

Statistical methods

Conditional logistic regression was used to compare PCa risk overall and in the 5 risk categories based upon birthplace. Simulations were used to estimate the possible influence of over-coverage of immigrants in the Population Register due to non-notification of return to countries of birth. Separate analyses were performed to examine for differences by age at immigration and duration of residence in Sweden. Finally, we assessed possible differences in the method of diagnosis (PSA testing of asymptomatic men versus evaluation of symptoms) between Swedish and foreign-born PCa cases. Multivariable adjustment for

socioeconomic, education, comorbidities and marital status was performed for all subjects with covariate data available. Statistical analyses were performed using R. The study was approved by the Research Ethics Board of Umeå University Hospital.

Results

The study population included 108996 Swedish cases, 505561 Swedish controls, 8332 immigrant cases and 57083 immigrant controls. Median age was 72 and 68 years for Swedish and foreign-born men, respectively. Other Nordic countries contributed the largest immigrant group, followed by Western Europe. Foreign-born men resided in Sweden for a median of 38 years (range, 11–53).

Table 1 shows the demographics in Swedish-born and foreign-born cases and matched controls. All comparisons were statistically significant between cases and controls, and between Swedish-born versus immigrant men ($p < 0.001$).

Among prostate cancer cases, 22.5%, 22.7%, 25.7%, 8.8%, and 17.4% were classified as low-risk, intermediate-risk, high-risk, regionally metastatic and distant metastatic at diagnosis (Table 2). 3450 (2.9%) patients could not be classified into clinical risk categories due to missing data.

Figure 1a shows unadjusted PCa risk in foreign-born men by region of birth compared to Swedish men. Odds ratios of PCa were 0.96 (95% CI=0.84–1.10) for immigrants from North America and 0.92 (95% CI= 0.85–1.00) for men from Northern Europe, as compared to native-born Swedish men. By contrast, Middle Eastern (overall OR 0.47, 95 % CI 0.42–0.52), Southern European (OR 0.48, 95% CI 0.45–0.52), and Asian (OR 0.53, 95% CI 0.48–0.59) men had significantly lower PCa risk compared to native Swedes. These risk estimates remained virtually unchanged following adjustment for marital status, education, comorbidity and socioeconomic status (Figure 1a).

By clinical risk category, all immigrant groups had lower risks of low-risk, intermediate-risk, high-risk, regionally metastatic, and distant metastatic disease compared to Swedish men, with the exception of immigrants from North America and Northern Europe (Figure 1b). For example, Middle Eastern men had a significantly lower risk of both localized low-risk PCa (OR 0.40, 95% CI 0.32–0.49) and distant metastatic disease (OR 0.38 95% CI 0.26–0.56) compared to native-born Swedish men. We also looked specifically at PCa risk in immigrants from each Nordic country compared to native-born Swedes with generally similar results: Denmark (OR 0.80, 95% CI 0.75–0.86), Finland (OR 0.81, 95% CI 0.78–0.84), and Norway (OR 0.89, 95% CI 0.83–0.96). This did not reach statistical significance for immigrants from Iceland (OR 0.77, 95% CI 0.49–1.21), although possibly due to small sample size ($n=19$).

Older age at immigration and short time since arrival to Sweden were significantly associated with lower PCa risk (Table 3). Despite regional differences in average age at immigration, these findings were consistent across immigrant groups within each region (Table 4).

Finally, compared to native Swedes, foreign-born men (including other Nordic countries) were significantly less likely to be diagnosed through PSA testing as part of an asymptomatic check-up (Table 5). For example, Middle Eastern men had a 74% lower risk of diagnosis through PSA testing (OR 0.26; 95 % CI 0.20–0.35). The frequency of lower urinary tract symptoms leading to PCa diagnosis was also significantly lower in these groups.

Discussion

To our knowledge, this is the first study to compare clinical features of PCa and diagnostic modalities in immigrants compared to a native-born population. In the current nation-wide population-based study, we found significantly lower overall PCa risk in all immigrant groups in Sweden (other Nordic countries, Southern, Eastern and Western Europe, Central and South America, Africa and Asia) compared to native-born men, except those from North America and Northern Europe. While the likelihood of being diagnosed through PSA testing was significantly lower among foreign-born compared to native Swedes, foreign-born men had lower PCa risk across all five clinical risk categories (ranging from localized low-risk to metastatic PCa). Similar to findings in prior studies,[18] stratified analyses found evidence that younger age at immigration, and increasing time since arrival, were associated with higher PCa risk.

Our initial hypothesis was that a lower uptake of PSA testing and subsequent lower frequency of work-up among asymptomatic immigrant men would result in a lower risk of localized PCa, which was supported by our findings of less PSA testing and low-risk PCa among immigrants. However, we also observed a lower risk of advanced and metastatic PCa among immigrants compared to native-born Swedish men, suggesting that disparities in screening and diagnostic testing could only partly explain the observed trends. This finding was further supported by virtually no change in the results following multivariable adjustment for socioeconomic factors and comorbidities.

Our findings corroborate results from prior studies that have examined PCa incidence among men emigrating from low to high-risk regions, such as Asian immigrants to the US compared to those living in Asia. Shimizu et al. reported age-adjusted annual PCa incidence rates of 8.4/100,000 in Japan, versus 32.2/100,000 for Japanese immigrants in California. [20] Also, PCa incidence rates are lower in Asian-Americans compared to other US ethnic groups.[21] The estimated age-specific PCa incidence rate was 14.3/100,000 among the Hmong (Southeast Asian) population in California, versus 148.8/100,000 for non-Hispanic whites residing in the same area.[11]

Similarly, a lower PCa incidence has previously been reported in foreign-born compared to native-born Swedish men, although these studies did not include data about disease aggressiveness or means of diagnosis.[2, 18, 22] Hemminki et al. found that immigrants had a lower incidence of all cancers (standardized incidence rate (SIR) 0.95, 95% CI 0.94–0.97) and PCa (SIR 0.75, 95% CI 0.72–0.78) compared to men born in Sweden.[2] This database used only included foreign-born men fathering children in Sweden, and therefore subjects were young at the time of immigration (median age 24 years).

Beiki et al. evaluated PCa risk in 3,385,160 Swedish-born men versus 372,663 immigrants. [18] Overall, they found nearly a 40% lower risk of PCa in foreign-born men (OR 0.62, 95% CI 0.61–0.63). After stratification for age at immigration (<40 versus ≥40 years) or duration of residence in Sweden (<35 versus ≥35 years), foreign-born men had a consistently lower PCa risk. More recently, Mousavi et al. examined cancer risk in immigrants compared to native-born Swedish men in the Swedish Family-Cancer Database.[22] Similar to our study, they found reduced PCa incidence among immigrants to Sweden, except North Americans. The authors of the above studies proposed several possible underlying mechanisms, including differences in genetics, lifestyle factors, PSA testing rates or access to diagnostic procedures.

Overall, Sweden has undergone dramatic demographic transitions in recent decades. Low birth rates and increased longevity have resulted in an aging population, and large immigration has increased ethnic diversity substantially. In 2005, Sweden reported more immigrants per capita than the U.S., including a large immigrant population from the Middle East. Accordingly, the present study provides a timely opportunity to improve the understanding of differences in PCa risk and diagnosis in this growing population. Nevertheless, our findings should be confirmed in other contemporary populations globally, including those with different health care systems.

To our knowledge, our study is the first to examine tumor characteristics and relationship to PSA testing of asymptomatic men, which are both strongly related to overall PCa incidence, in an immigrant compared to a native-born population. Additional strengths of our study included the use of population based data with high completeness. Through record linkage to other nation-wide population-based registries, we had data on socioeconomic factors (ex: education and marital status) and comorbidity scores. Furthermore, five controls were available from the general Swedish male population matched for birth year and county of residence, for whom data on covariates of interest were also available.

Limitations of our study included missing grade or stage for a minority of men, precluding risk classification. Data on birthplace or date of immigration to Sweden were also unavailable for a small proportion of foreign-born men. There was also a disparity between nationalities in average age at immigration, and there may be other unmeasured differences between immigrants and native-born Swedish men. However, analyses stratified by region and multivariable adjustment for comorbidities and socioeconomic variables did not materially change the results. Finally, since all immigrants may not report if they return to their country of birth, there may be over-coverage of immigrants recorded as residing in Sweden in the Population Register. The exact extent of this misclassification is unknown, but has been estimated to be 1% and 2.8% for Nordic and other immigrants.[23] Nevertheless, in simulated scenarios of an over-coverage of 1 %, 2.8 % and 8 %, risk estimates remained virtually unchanged (data not shown).

The current dataset did not allow a comparison of specific genetic or lifestyle factors between men from different geographic regions. Recent technological advances have facilitated the identification of genetic variants which influence PCa risk.[24] Multiple environmental factors have also been suggested to possibly be associated with PCa risk.[25–

27] For example, observational data suggests a role for intake of calcium, dairy products, and charred meat in the etiology and/or progression of PCa. It is possible that generally lower consumption of these foods in the Asian diet might be associated with the lower overall PCa risk compared to the US and Scandinavia.

Of additional interest is the possible role of sunlight and vitamin D. Prior studies have shown an inverse relationship between geographic UV exposure and PCa mortality [28], such that this might also help explain our observation of lower PCa risk among men born in southern regions (ex: Southern Europe, Asia, Africa, Central and South America), and the findings of increased risk with longer duration of residence in Sweden. Because it was not possible to test these hypotheses directly in the current population, further study is necessary to define the relative contribution of genetic factors and environmental exposures on PCa risk and progression.

Conclusion

First-generation immigrants to Sweden had lower PCa risk compared to age-matched Swedish-born men. The lowest PCa risk was observed in men immigrating at an older age or recently settling in Sweden, which was consistent across risk categories. Although low-risk and PSA-detected PCa were less frequent among foreign-born men, the risk of advanced disease was also significantly lower. The observed risk estimates remained virtually unchanged following adjustment for socioeconomic status and comorbidities. Taken together, patterns of health care-seeking behavior and means of detection appeared to only partly explain the differences in PCa risk between the immigrant and non-immigrant Swedish population as immigrant men also had lower risk of metastatic disease.

Acknowledgments

Funding was provided by the Swedish Research Council 825-2008-5910 and The Swedish Cancer Foundation 11 0471, Västerbotten County Council and Lion's Cancer Research Foundation at Umeå University. SL is supported by the Louis Feil Charitable Lead Trust.

References

1. Singh GK, Siahpush M. Ethnic-immigrant differentials in health behaviors, morbidity, and cause-specific mortality in the United States: an analysis of two national data bases. *Hum Biol.* 2002; 74(1):83–109. [PubMed: 11931581]
2. Hemminki K, Li X, Czene K. Cancer risks in first-generation immigrants to Sweden. *Int J Cancer.* 2002; 99(2):218–228. [PubMed: 11979437]
3. Beiki O, Hall P, Ekblom A, Moradi T. Breast cancer incidence and case fatality among 4.7 million women in relation to social and ethnic background: a population-based cohort study. *Breast cancer research : BCR.* 2012; 14(1):R5. [PubMed: 22225950]
4. Muir CS, Nectoux J, Staszewski J. The epidemiology of prostatic cancer. Geographical distribution and time-trends. *Acta oncologica.* 1991; 30(2):133–140. [PubMed: 2029395]
5. Jemal A, Bray F, Center MM, Ferlay J, Ward E, Forman D. Global cancer statistics. *CA: a cancer journal for clinicians.* 2011; 61(2):69–90. [PubMed: 21296855]
6. Lindeberg, S. Food and western disease: health and nutrition from an evolutionary perspective. United Kingdom: John Wiley & Sons Ltd.; 2010.
7. [Accessed November 17, 2011] World Health Organization Global Health Observatory Repository Data. <http://apps.who.int/ghodata/?vid=10011>

8. Bray F, Lortet-Tieulent J, Ferlay J, Forman D, Auvinen A. Prostate cancer incidence and mortality trends in 37 European countries: an overview. *European journal of cancer*. 2010; 46(17):3040–3052. [PubMed: 21047585]
9. Kvale R, Auvinen A, Adami HO, Klint A, Hernes E, Moller B, Pukkala E, Storm HH, Tryggvadottir L, Tretli S, Wahlqvist R, Weiderpass E, Bray F. Interpreting trends in prostate cancer incidence and mortality in the five Nordic countries. *J Natl Cancer Inst*. 2007; 99(24):1881–1887. [PubMed: 18073376]
10. Singh GK, Siahpush M. All-cause and cause-specific mortality of immigrants and native born in the United States. *Am J Public Health*. 2001; 91(3):392–399. [PubMed: 11236403]
11. Mills PK, Yang RC, Riordan D. Cancer incidence in the Hmong in California, 1988–2000. *Cancer*. 2005; 104(12 Suppl):2969–2974. [PubMed: 16247793]
12. Hagel E, Garmo H, Bill-Axelson A, Bratt O, Johansson JE, Adolfsson J, Lambe M, Stattin P. PCBaSe Sweden: a register-based resource for prostate cancer research. *Scandinavian journal of urology and nephrology*. 2009; 43(5):342–349. [PubMed: 19921977]
13. Adolfsson J, Garmo H, Varenhorst E, Ahlgren G, Ahlstrand C, Andren O, Bill-Axelson A, Bratt O, Damber JE, Hellstrom K, Hellstrom M, Holmberg E, Holmberg L, Hugosson J, Johansson JE, Pettersson B, Tornblom M, Widmark A, Stattin P. Clinical characteristics and primary treatment of prostate cancer in Sweden between 1996 and 2005. *Scandinavian journal of urology and nephrology*. 2007; 41(6):456–477. [PubMed: 17934985]
14. ARSRAPPORT Nationella Prostatacancerregistret 2009. [Accessed November 28, 2011] http://www.roc.se/prostata/rapport/rapport_2009.pdf (*in Swedish*).
15. The National Board of Health and Welfare. Statistics in the Areas of Health and Medical Care. 2007
16. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Dis*. 1987; 40(5):373–383. [PubMed: 3558716]
17. Kastner C, Armitage J, Kimble A, Rawal J, Carter PG, Venn S. The Charlson comorbidity score: a superior comorbidity assessment tool for the prostate cancer multidisciplinary meeting. *Prostate Cancer Prostatic Dis*. 2006; 9(3):270–274. [PubMed: 16770340]
18. Beiki O, Ekblom A, Allebeck P, Moradi T. Risk of prostate cancer among Swedish-born and foreign-born men in Sweden, 1961–2004. *Int J Cancer*. 2009; 124(8):1941–1953. [PubMed: 19107943]
19. National Comprehensive Cancer Network Clinical Practice Guidelines in Oncology version 2011. [Accessed January 14, 2011] http://www.nccn.org/professionals/physician_gls/pdf/prostate.pdf.
20. Shimizu H, Ross RK, Bernstein L, Yatani R, Henderson BE, Mack TM. Cancers of the prostate and breast among Japanese and white immigrants in Los Angeles County. *Br J Cancer*. 1991; 63(6):963–966. [PubMed: 2069852]
21. [Accessed November 28, 2011] Cancer of the Prostate- SEER Stat Fact Sheet. <http://seer.cancer.gov/statfacts/html/prost.html#incidence-mortality>
22. Mousavi SM, Fallah M, Sundquist K, Hemminki K. Age and time dependent changes in cancer incidence among immigrants to Sweden: Colorectal, lung, breast and prostate cancers. *Int J Cancer*. 2011
23. Qvist, J. Problems of coverage in the Population Register. Report 1999:1. Sweden, S., editor. Stockholm: 1999.
24. Zheng SL, Sun J, Wiklund F, Smith S, Stattin P, Li G, Adami HO, Hsu FC, Zhu Y, Balter K, Kader AK, Turner AR, Liu W, Bleecker ER, Meyers DA, Duggan D, Carpten JD, Chang BL, Isaacs WB, Xu J, Gronberg H. Cumulative association of five genetic variants with prostate cancer. *N Engl J Med*. 2008; 358(9):910–919. [PubMed: 18199855]
25. John EM, Dreon DM, Koo J, Schwartz GG. Residential sunlight exposure is associated with a decreased risk of prostate cancer. *J Steroid Biochem Mol Biol*. 2004; 89–90(1–5):549–552.
26. Hori S, Butler E, McLoughlin J. Prostate cancer and diet: food for thought? *BJU Int*. 2011; 107(9): 1348–1359. [PubMed: 21518228]

27. AICR. , editor. World Cancer Research Fund / American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington, DC: 2007.
28. Hanchette CL, Schwartz GG. Geographic patterns of prostate cancer mortality. Evidence for a protective effect of ultraviolet radiation. *Cancer*. 1992; 70(12):2861–2869. [PubMed: 1451068]
29. Berglund A, Garmo H, Tishelman C, Holmberg L, Stattin P, Lambe M. Comorbidity, treatment and mortality: a population based cohort study of prostate cancer in PCBaSe Sweden. *J Urol*. 2011; 185(3):833–839. [PubMed: 21239002]

(a)

(b)

Figure 1.

Odds ratios for prostate cancer in immigrant men compared to Swedish men by country of birth*, including (a) overall unadjusted and adjusted rates, and (b) stratifications by clinical risk category in PCBaSe 2.0.

* Based upon categorizations from Statistics Sweden, regions of birth were grouped as: Sweden, other Nordic countries (Denmark, Finland, Iceland, Norway), Northern Europe (Estonia, Ireland, Latvia, Lithuania, United Kingdom of Great Britain and Northern Ireland), Southern Europe (Albania, Andorra, Bosnia and Herzegovina, Gibraltar, Greece, Italy,

Yugoslavia, Kosovo, Croatia, Macedonia, Malta, Montenegro, Portugal, San Marino, Serbia, Serbia and Montenegro, Slovenia, Spain, Vatican City), Eastern Europe (Bulgaria, Moldavia, Poland, Romania, Russia, Slovakia, Czech Republic, Ukraine, Hungary, Belarus), Western Europe (Belgium, France, Liechtenstein, Luxembourg, Monaco, The Netherlands, Switzerland, Germany, Austria), North America, Central and South America, Africa, the Middle East, Asia, and Other.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Demographics of Swedish-born and immigrant prostate cancer cases and their matched controls in Prostate Cancer data Base Sweden (PCBaSe) 2.0.

Table 1

	Swedish-born men		Immigrants	
	Cases (n = 108 996)	Controls (n = 505 561)	Cases (n = 8 332)	Controls (n = 57 083)
Age category, no. (%)				
<65 yrs	27,014 (24.8)	130,944 (25.9)	2,614 (31.4)	20,069 (35.2)
65–74 yrs	39,763 (36.5)	187,464 (37.1)	3,496 (42.0)	23,993 (42.0)
75+ yrs	42,219 (38.7)	187,153 (37.0)	2,222 (26.7)	13,021 (22.8)
Age, yrs				
Median (IQR)	72 (65,78)	71 (64,78)	69 (63,75)	68 (62,74)
Marital status *				
Married	74,185 (68.1)	329,181 (65.1)	5,550 (66.6)	37,121 (65.0)
Divorced	12,386 (11.4)	61,881 (12.2)	1,484 (17.8)	10,776 (18.9)
Widower	12,607 (11.6)	54,067 (10.7)	677 (8.1)	4,125 (7.2)
Never married	9,810 (9.0)	59,483 (11.8)	618 (7.4)	5,033 (8.8)
Missing	8 (0.0)	949 (0.2)	3 (0.0)	28 (0.0)
Socio-economic status *				
Low	54,599 (50.1)	266,008 (52.6)	4,352 (52.2)	29,591 (51.8)
High	53,692 (49.3)	234,658 (46.4)	3,007 (36.1)	16,695 (29.2)
Not gainfully employed	480 (0.4)	3,575 (0.7)	43 (0.5)	383 (0.7)
Missing	225 (0.2)	1,266 (0.3)	930 (11.2)	10,408 (18.2)
Comorbidity				
0	70,647 (64.8)	335,840 (66.4)	5,482 (65.8)	38,963 (68.3)
1	20,027 (18.4)	90,291 (17.9)	1,439 (17.3)	9,573 (16.8)
2	10,743 (9.9)	45,857 (9.1)	772 (9.3)	4,902 (8.6)
3+	7,579 (7.0)	33,573 (6.6)	639 (7.7)	3,645 (6.4)
Education				
Low	49,948 (45.8)	243,736 (48.2)	2,935 (35.2)	20,525 (36.0)
Middle	35,971 (33.0)	163,925 (32.4)	3,174 (38.1)	20,300 (35.6)
High	19,654 (18.0)	83,335 (16.5)	1,691 (20.3)	10,344 (18.1)
Missing	3,423 (3.1)	14,565 (2.9)	532 (6.4)	5,914 (10.4)

Based upon linkage to the LISA database^{*}

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 2

Prostate Cancer Features Among Swedish-Born and Immigrant PCa Cases in PCBaSe 2.0.

	Swedish-born PCa cases (n = 108 996)		Immigrant PCa cases (n = 8 332)	
Year of diagnosis, no. (%)				
1991–1993	5,258	(4.8)	126	(1.5)
1994–1996	5,974	(5.5)	234	(2.8)
1997–1999	17,158	(15.7)	1,004	(12.0)
2000–2002	20,763	(19.0)	1,542	(18.5)
2003–2005	26,094	(23.9)	2,210	(26.5)
2006–2009	33,749	(31.0)	3,216	(38.6)
Gleason score				
2–6	47,337	(43.4)	3,850	(46.2)
7	36,831	(33.8)	2,777	(33.3)
8–10	22,328	(20.5)	1,514	(18.2)
Missing	2,500	(2.3)	191	(2.3)
Serum PSA level (ng/mL)				
<4	7,016	(6.4)	690	(8.3)
4–10	32,857	(30.1)	2,902	(34.8)
10–20	22,431	(20.6)	1,760	(21.1)
20–50	19,265	(17.7)	1,253	(15.0)
50–100	9,021	(8.3)	567	(6.8)
>100	14,396	(13.2)	898	(10.8)
Missing	4,010	(3.7)	262	(3.1)
Serum PSA level (ng/mL)				
Median (IQR)	14	(7, 42)	11	(6, 30)
Risk category [*]				
Low risk	24,233	(22.2)	2,169	(26.0)
Intermediate risk	24,508	(22.5)	2,103	(25.2)
High risk	28,211	(25.9)	1,948	(23.4)
Regionally metastatic	9,673	(8.9)	642	(7.7)
Distant metastases	19,201	(17.6)	1,190	(14.3)
Missing	3,170	(2.9)	280	(3.4)

* Modification of the National Comprehensive Cancer Network guidelines[19]: (1) low risk (clinical stage T1–2, Gleason score ≤ 6 and PSA < 10 ng/mL), (2) intermediate risk (T1–2, Gleason score 7 and/or PSA 10 to < 20 ng/mL, (3) high risk: T3 and/or Gleason score 8–10 and/or PSA 20 to < 50 ng/mL, (4) T4 and/or regionally metastatic disease: N1 and/or PSA 50 to < 100 ng/mL in the absence of distant metastases (M0 or Mx) and (5) distant metastases: M1 and/or PSA ≥ 100 ng/mL.

Table 3

Odds ratios for prostate cancer in immigrant men (compared to Swedish-born) based upon age at immigration and duration of residence in Sweden in PCBaSe 2.0.

Age at immigration (years)	No. of cases	OR	95% CI	Duration of residence (years)	No. of cases	OR	95% CI
Swedish-born men	108,996	Ref		Swedish-born men	108,996	Ref	
<20	969	0.84	(0.79 – 0.89)	<5	197	0.47	(0.41 – 0.54)
20–30	3,048	0.76	(0.73 – 0.79)	5–25	954	0.50	(0.47 – 0.53)
30–40	1,486	0.72	(0.68 – 0.76)	25–50	4,306	0.73	(0.71 – 0.76)
40+	1,244	0.51	(0.48 – 0.54)	50+	1,290	0.85	(0.80 – 0.90)
Date of immigration unknown	1,585	0.91	(0.87 – 0.96)	Date of immigration unknown	1,585	0.91	(0.87 – 0.96)

Table 4

Odds ratios for prostate cancer in immigrant men (compared to Swedish-born) by age at immigration and duration of residence in Sweden

Country of birth	Age at immigration				Duration of residence							
	No.	<30 yrs OR	95% CI	No.	30 yrs OR	95% CI	No.	<35 yrs OR	95% CI	No.	35 yrs OR	95% CI
Sweden	108,996	Ref		108,996	Ref		108,996	Ref		108,996	Ref	
Other Nordic countries	2,239	0.83	(0.79–0.86)	1,034	0.76	(0.72–0.81)	691	0.70	(0.65–0.75)	2,582	0.84	(0.81–0.87)
Eastern Europe	332	0.72	(0.65–0.80)	356	0.70	(0.63–0.78)	298	0.66	(0.59–0.74)	390	0.75	(0.68–0.83)
Northern Europe	175	0.88	(0.76–1.02)	82	0.66	(0.53–0.82)	70	0.65	(0.52–0.82)	187	0.86	(0.75–0.99)
Southern Europe	349	0.54	(0.48–0.60)	380	0.44	(0.40–0.48)	322	0.41	(0.37–0.46)	407	0.55	(0.50–0.61)
Western Europe	637	0.86	(0.79–0.93)	187	0.74	(0.64–0.85)	119	0.65	(0.55–0.78)	705	0.87	(0.80–0.93)
The Middle East	56	0.53	(0.41–0.69)	237	0.45	(0.40–0.51)	252	0.45	(0.40–0.51)	41	0.57	(0.42–0.78)
Asia, other	85	0.57	(0.46–0.70)	205	0.45	(0.39–0.52)	221	0.45	(0.39–0.51)	69	0.59	(0.47–0.75)
Africa	59	0.74	(0.57–0.96)	71	0.52	(0.41–0.65)	87	0.53	(0.43–0.65)	43	0.80	(0.59–1.08)
Central and South America	35	0.72	(0.52–1.01)	119	0.56	(0.46–0.66)	137	0.56	(0.47–0.66)	17	0.89	(0.55–1.43)
North America	42	0.98	(0.72–1.33)	41	0.77	(0.56–1.04)	41	0.84	(0.62–1.14)	42	0.88	(0.65–1.19)
Date of immigration unknown	1,585	0.91	(0.87–0.96)	1,585	0.91	(0.87–0.96)	1,585	0.91	(0.87–0.96)	1,585	0.91	(0.87–0.96)

Comparison of mode of detection and presence of symptoms between Swedish born and immigrant cases.

Table 5

Country of birth	Health examination			Lower urinary tract symptoms		
	No.	OR	95% CI	No.	OR	95% CI
Sweden	16,961	Ref		20,499	Ref	
Other Nordic countries	604	0.67	(0.62 – 0.73)	951	0.93	(0.87 – 0.99)
Eastern Europe	129	0.58	(0.49 – 0.69)	183	0.91	(0.78 – 1.05)
Northern Europe	98	0.96	(0.79 – 1.18)	102	0.92	(0.75 – 1.11)
Southern Europe	142	0.36	(0.30 – 0.42)	239	0.72	(0.63 – 0.82)
Western Europe	218	0.86	(0.75 – 0.99)	214	0.87	(0.76 – 0.99)
The Middle East	49	0.26	(0.20 – 0.35)	109	0.79	(0.65 – 0.95)
Asia, other	57	0.37	(0.28 – 0.47)	96	0.73	(0.59 – 0.89)
Africa	28	0.39	(0.27 – 0.56)	39	0.89	(0.65 – 1.22)
Central and South America	39	0.51	(0.37 – 0.69)	40	0.71	(0.52 – 0.97)
North America	30	0.84	(0.59 – 1.20)	46	1.14	(0.85 – 1.52)
All immigrants	1,401	0.58	(0.55 – 0.62)	2,025	0.87	(0.83 – 0.91)