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The Risk of Prostate Cancer amongst Black Men in the United Kingdom: The PROCESS Cohort Study

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Abstract

Objectives: It is known that African American men have a greater risk of prostate cancer than white men. We investigated whether this was true for first-generation black Caribbean and black African men in the United Kingdom.

Methods: A clinical cohort study design recruiting all cases of prostate cancer diagnosed over a 5-yr period and residing in defined areas of London and Bristol. We calculated the age-standardised incidence rates and relative risk for all black men, and black Caribbean and black African men versus white men.

Results: Black men had higher age-adjusted rates of prostate cancer (166 per 100,000, 95% confidence interval [95%CI], 151–180 per 100,000) than white men (56.4 per 100,000, 95%CI, 53.3–59.5 per 100,000). The relative risks for all black, black Caribbean, and black African men were 3.09 (95%CI, 2.79–3.43; $p < 0.0001$), 3.19 (95%CI, 2.85–3.56; $p < 0.0001$) and 2.87 (95%CI, 2.34–3.53; $p < 0.0001$), respectively. There was no strong evidence that the rates for black Caribbean differed from black African men. The higher risk in black men compared with white men was more apparent in younger age groups (p value for interaction < 0.001).

Conclusions: Black men in the United Kingdom have substantially greater risk of developing prostate cancer compared with white men, although this risk is lower than that of black men in the United States. The similar rates in black Caribbean and black African men suggest a common genetic aetiology, although migration may be associated with an increased risk attributable to a gene-environment interaction.

1. Introduction

In the United Kingdom, prostate cancer is of major public health importance, being the most common cancer in men [1]. The incidence rate has risen steadily over the last 15 years, although the degree to which this rise reflects increased detection and/or a true increase in risk remains uncertain [2,3].

Data from the United States, Africa, and the Caribbean demonstrate that significant ethnic variations for prostate cancer risk exist [4–7]. The incidence rate for American black men is estimated to be around 55% greater than that for American white men [4], although this value is likely to be an underestimate because uptake of prostate-specific antigen (PSA) testing is lower in black men [8]. Incidence rates from the Caribbean and Africa are more variable and, in general, much lower than those of black men in the United States, but it is hard to know whether this information reflects a genuine lower risk or a less complete case ascertainment due to limited provision of urologic services and less thorough health information systems. One notable exception was an incidence study [9] from Kingston, Jamaica, which reported that black men had the highest incidence rate in the world at 304 per 100,000 men.

Remarkably little data exist on risk of prostate cancer amongst African Caribbean men in the United Kingdom and how this risk value compares with that of their white counterparts. Migrant studies can be very informative [10]. Because UK black men are largely first-generation migrants, it is uncertain whether they would have rates as high as US black men who have lived in the United States for many generations. Such data would help us understand whether the risk of prostate cancer changes acutely with migration. An analysis of routine mortality data by place of birth did observe elevated prostate mortality rates for both Caribbean and West African immigrants but not those from East Africa [11]. These data are not a direct measure of incidence because mortality is determined by both incidence and case fatality. Studies from the United States suggest that black men may have higher case fatality rates. A large cohort study of men and women in England and Wales (the longitudinal study) found a doubling of the standardised incidence ratios (1971–1989) for people from the Caribbean (2.2; 95%CI, 1.0–4.1) compared with the general population, but this finding was based on only nine cases and wide confidence intervals [12]. A recent audit of prostate cancer cases from East London observed a 3-fold increased risk of prostate cancer amongst black men compared with white men [13]. This smaller study had too few cases to examine whether black men from the Caribbean had any difference in risk compared with black men from the African subcontinent.

Our aim was to establish a UK population-based clinical cohort to derive, for the first time, robust estimates of incidence rates and compare the relative risks of developing prostate cancer among white men and black men of African and Caribbean ancestry.

2. Methods

We undertook a population-based retrospective cohort study by identifying all incident cases of prostate cancer in prespecified catchment populations: the PRostate Cancer in Ethnic SubgroupS (PROCESS) study. Four areas (North Bristol, South West London, South East London, and North East London) with a relatively high proportion of resident black men were chosen. For each area, we generated a list of all eligible postcodes that covered these areas using the all-fields postcode directory from the Census Dissemination Unit division of the Manchester Information & Associated Services (MIMAS) Web site [14].

Ethical approval for the study was provided by the South West Multicentre Research Ethics Committee.

2.1. Case definition

The case definition for inclusion in the study was a histologically proven or a clinical diagnosis (see

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below) of prostate cancer in men resident in our prespecified catchment areas diagnosed over a specific 5-yr period. The study was piloted in the Bristol area during the period 1995–1999, but this period was changed to 1997–2001 for the other areas because there were difficulties accessing historical data in some areas.

2.2. Case ascertainment

Cases were ascertained by using a multisource approach, which included data from (1) pathology databases, (2) hospital discharge diagnosis files, (3) PSA records >10 ng/ml, (4) Cancer Registry (Bristol Centre only), and (5) urology department database (North West London centre only). We started with the pathology database and then checked for any additional cases from each source sequentially. (For the North West London centre we started by combining the pathology and urology department databases.) Records from each data source were cross-checked by local staff in the urology department, and duplicate records were removed. Hospital records were reviewed when a case was not found on the histology database but appeared to have a clinical diagnosis of prostate cancer and/or an elevated PSA result. In some cases this approach confirmed a histologic diagnosis or diagnosis other than prostate cancer. Where there was uncertainty as to the diagnosis (“grey case”), an anonymised case vignette was produced and presented to a panel of at least four urologists who had to reach a consensus as to whether the case should be included as a “clinical” (non-histologic-proven) case or excluded because there was insufficient evidence to confirm the diagnosis.

2.3. Classification of ethnicity

We classified the ethnicity of each man using a hierarchical approach. Men known to be alive were asked to complete a questionnaire that included the 2001 census questions on ethnicity. When the man was known to have died, the next of kin was contacted, if possible, as long as the death had occurred more than 6 mo in the past. If this was not possible, we next used data from the hospital administration system or medical records. In the case of North East London, the preexisting urology database had ethnicity recorded by staff. If this information was still missing and the man had died, we used place of birth from the death certificate as a proxy for ethnicity. In a few cases individual consultants reported ethnicity when they could recall it.

2.4. Census denominator data

As the resident population of some ethnic minorities changed markedly in the study areas during the 10-yr period between the 1991 census and the 2001 census, a more accurate estimate of the populations at the midpoint of the periods (1997 for Bristol and 1999 for London) over which cases were identified was obtained by using linear interpolation. Male ethnicity by age group data from the 1991 census was accessed from Casweb Local Base Statistics Table 06 and, for the 2001 census, from Casweb Standard Table 101. Because of electoral ward boundary changes, interpolation between the two time points was based on tracts created by the Social and Spatial Inequalities (SASI) group at the University of Sheffield. Tracts are amalgamations of the electoral wards existing at different points in time so that, as far as possible, a tract covers the same geographic space at each time point [15]. Some tracts also included electoral wards in which no attempt had been made to identify cases. The populations contributed by these tracts to incidence rate denominators were weighted according to the proportion of included wards in which cases had been sought.

Separate population age distributions were calculated for white, all black, black African, and black Caribbean groups. The white group was obtained by combining the populations of white British, white Irish, and white other. The black group was obtained by combining the black African, black Caribbean, mixed race black African, mixed race black Caribbean, and other black groups.

2.5. Statistical methods

Anonymised data from each area were aggregated into a master data set for analysis. Age-adjusted rates per 100,000 person years were calculated by using the direct method with the hypothetical European population as the standard population [16]. We initially calculated the rates for all black men aggregated together and then for black Caribbean and black African men. The rates in the different amalgamated ethnic groups were compared as age-adjusted rate ratios, which were calculated by using Poisson regression. In a sensitivity analysis, the potential effect of missing ethnicity data was evaluated by repeating the analysis with the assumption that all identified cases with no ethnic code were white as a worst-case scenario.

3. Results

We initially identified 2238 subjects but had to exclude 78 cases who were actually diagnosed outside our eligible time period, 5 men who were not permanent residents within the catchment areas, and 15 men who turned out not to have prostate cancer. This left 2140 incident cases over the 5-yr period (227 cases North Bristol, 456 cases South West London, 710 cases South East London, 747 cases North East London). The ethnic distribution of the cases was as follows: 1315 (61.4%) white, 435 (20.0%) black Caribbean, 102 (4.8%) black African, 10 (0.5%) black unclassified, 129 (6.0%) other ethnic groups, and 149 (7.0%) ethnicity uncoded.

The age-specific rates, crude rates, and age-standardised rates are shown in Table 1. In each age strata, except for 85 yr and older, the rates were greater for black men (relative rates: 2.0–5.5). These differences appeared larger for younger ages. The age-standardised rate for white men was 56.4 per 100,000 (95%CI, 53.3–59.5 per 100,000) and 166 per 100,000 (95%CI, 151–180 per 100,000) for black men. When we examined this information by subgroups, we found that the age-adjusted rate for black Caribbean men was 173 per 100,000 (95%CI, 156–190 per 100,000) and 139 per 100,000 (95%CI, 110–168 per 100,000) for black Africans. In both cases the age-adjusted rates were larger than the crude rates, although this difference was much more marked for the black African men because they have a much younger population (black Caribbean men <40 yr: 66.8%; black African men <40 yr: 78.8%; Caucasian men <40 yr: 61.9%). A statistical test of interaction between ethnicity and age group confirmed the observation that the higher relative rates for black men compared with white men were more marked for younger age groups ($p < 0.001$ for all black men, $p = 0.002$ for black Caribbean men, $p = 0.02$ for black African men).

The age-adjusted relative rates are shown in Table 2. Black men were around three times more likely to be diagnosed with prostate cancer than white men (relative risk for black versus white men: 3.09; 95%CI, 2.79–3.43; $p < 0.0001$). Black African men had slightly lower relative risks than black Caribbean men, but this finding was compatible with chance variation (relative risk for black African versus black Caribbean men: 0.90; 95%CI, 0.73–1.12; $p = 0.35$).

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Table 1 – Age-specific, crude, and age-adjusted rates of prostate cancer comparing all black, black Caribbean, and black African men with white men (overall adjusted rates use the hypothetical European population)

Age group	White			Black			Black Caribbean			Black African		
	Cases	Person years	Rate/100,000	Cases	Person years	Rate/100,000	Cases	Person years	Rate/100,000	Cases	Person years	Rate/100,000
0-4	0	129835	0	0	73345	0	0	25515	0	0	38485	0
05-9	0	106425	0	0	66100	0	0	25135	0	0	32675	0
10-14	0	99365	0	0	59560	0	0	25590	0	0	27045	0
15-19	0	98970	0	0	51335	0	0	22440	0	0	23470	0
20-24	0	197595	0	0	45450	0	0	19055	0	0	21915	0
25-29	0	297610	0	0	50800	0	0	19725	0	0	27285	0
30-34	0	254710	0	0	63325	0	0	23295	0	0	36035	0
35-39	0	204455	0	0	59215	0	0	24360	0	0	31265	0
40-44	1	158175	0.6	0	43935	0	0	17210	0	0	24735	0
45-49	5	125645	4	4	26035	15	3	11060	27	1	14085	7
50-54	28	123385	23	16	19100	84	7	9685	72	9	8810	102
55-59	51	98260	52	46	16210	284	30	10565	284	14	5130	273
60-64	137	87630	156	107	21350	501	80	15185	527	26	5460	476
65-69	203	79420	256	139	16485	843	113	12775	885	25	3250	769
70-74	256	70905	361	136	10325	1317	114	8510	1340	18	1475	1220
75-79	311	57000	546	68	5285	1287	58	4395	1320	8	745	1074
80-84	195	34805	560	24	2100	1143	23	1640	1402	1	390	256
85+	126	21075	598	6	985	609	6	880	682	0	85	0
Crude rate	58.5 per 100,000			86.5 per 100,000			156.7 per 100,000			33.7 per 100,000		
Age-adjusted rate	56.4 per 100,000			165.5 per 100,000			173.1 per 100,000			139.3 per 100,000		

The relative risks were slightly attenuated (relative risk for all black versus white men: 2.77; 95%CI, 2.50–3.06; $p < 0.0001$) when we repeated the analysis by assigning all cases with missing ethnicity as white men. We also repeated the analyses excluding cases from the North East London centre. This centre previously published an analysis on an independent subset of cases [13] (not necessarily the same cases as those ascertained in the PROCESS study). If anything, the relative rates were even larger (3.57; 95%CI, 3.16–4.04; $p < 0.0001$) with the exclusion of this centre, indicating that our results were independent of that previous publication.

To compare our results with US data, we used US age-specific rates for black men in 1999 (taken from www.seer.cancer.gov) to calculate age-standardised rates using the European standard population. This calculation resulted in a US black rate of 283 per 100,000 compared with 166 per 100,000 for UK black men (US white rate was 172 per 100,000).

4. Discussion

This is the first large study that compares incidence rates of first-generation black migrants from either Africa or the Caribbean with rates of white men in the United Kingdom. Black men have a 3-fold greater risk of developing prostate cancer, and there is little difference between men of Caribbean or African origin. This relative difference is far larger than that seen in the United States because of the lower rate of prostate cancer amongst white men in the United Kingdom. Incidence rates for US black men in 1999 standardised to the same European standard population used for the United Kingdom rates are around

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Table 2 – Relative rates of prostate cancer among white, all black, black Caribbean, and black African men

Ethnic group	Crude risk ratio			Age-adjusted risk ratio		
	RR	95%CI	p value	RR	95%CI	p value
Black men vs. white men	1.48	1.34–1.64	0.0006	3.09	2.79–3.43	<0.0001
Black Caribbean vs. white men	2.68	2.40–2.99	<0.0001	3.19	2.85–3.56	<0.0001
Black African vs. white men	0.58	0.47–0.71	<0.0001	2.87	2.34–3.53	<0.0001
Black African vs. black Caribbean men	0.22	0.17–0.27	<0.0001	0.90	0.73–1.12	0.35

RR = relative rate; 95%CI = 95% confidence interval.

70% higher than what we have observed in the United Kingdom. The greater routine use of PSA testing in the United States would be expected to result in higher incidence rates because of the inclusion of screen-detected cases, but the differences are greater than would be expected on the basis of simulation predictions [17].

4.1. International data

Data from routine cancer registries from the Caribbean and Africa are hard to interpret but suggest much lower rates for black men than those observed from the United States or United Kingdom [5,6] (Table 3). These results may underestimate the true rates either because of limited provision of urologic services and/or limitations with the completeness of registry data in these areas [18]. One study [19] from Nigeria reported a higher incidence rate of 127 per 100,000, but this finding was restricted to men aged 45 yr and older; hence, the total population rate is much lower (see Table 3 for estimated total population rate). Some publications from the Caribbean suggest much higher rates amongst black men [20], in particular a study from Kingston, Jamaica [9].

The study of clinical cases from Kingston ascertained all cases diagnosed from 1989 to 1994 using multiple sources. It reported the highest age-standardised incidence rate in the world of 304 per 100,000 men in stark contrast to another publication [21] from the same population but based on registration data, which reported an age-standardised rate of 56.4 per 100,000. We could not fully check the paper by Glover and colleagues [9] because they do not present the age distribution of their population. However, the crude rate can be calculated from tabulated figures, giving a much lower rate of around 70 per 100,000. It is unlikely that standardization to the 1970 US population could have produced a 4-fold difference. One possible explanation is that the reported rate may have been the cumulative 5-yr rate rather than the annual rate, although this would not explain the period-specific rates reported in Table 2 of the paper.

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Table 3 – Reported incidence rates for clinically detected prostate cancer in selected West African and Caribbean countries

Author	Year	Country	Crude rate per 100,000	ASR (world) per 100,000
Incidence studies				
Ekwere [31]	1984–1994	Nigeria	6.1 ^a	
Osegbe [19]	1988–1993	Nigeria	21.2 ^b	
Angwafo [32]	1986–1990	Cameroon	0.2 ^c	
Hanchard [21]	1993–1997	Jamaica	39	56.4
Glover [8]	1989–1993	Jamaica	69.6	304 ^d
Mallick [20]	1999	Guadeloupe	112.7	88.8 ^e
Registry-based estimates				
GLOBOCAN [6]	2004	Caribbean	47.7	52.4
GLOBOCAN [6]	2004	Bahamas	45.8	65.3
GLOBOCAN [6]	2004	Barbados	102.3	99.7
GLOBOCAN [6]	2004	Cuba	37.5	28.2
GLOBOCAN [6]	2004	Dominican Republic	53	85.3
GLOBOCAN [6]	2004	Haiti	18.7	38.1
GLOBOCAN [6]	2004	Jamaica	36.6	42.3
GLOBOCAN [6]	2004	Puerto Rico	126.7	100.1
GLOBOCAN [6]	2004	Trinidad and Tobago	53.9	60.5
IARC report [5]	1997–1998	Gambia	2.0	4.7
IARC report [5]	1996–1999	Guinea, Conakry	2.7	9.7
IARC report [5]	1988–1997	Mali, Bamako	1.9	5.7
IARC report [5]	1993–1999	Niger, Niamey	2.2	10.8
IARC report [5]	1998–1999	Nigeria, Ibadan	7.7	19.8

ASR = age-standardised rate.

^a This paper presents the 10-yr incidence of 61.3 per 100,000.

^b This paper presents the 6-yr cumulative incidence (127/100,000) for men older than 45 yr. The estimated crude rate is much lower.

^c This paper reports an incidence rate of 93.8 per 100,000 for men older than 40 yr, but this rate is for cases ascertained over 5 yr and the calculation is incorrect. We have recalculated the crude rate assuming the male population is half the total population.

^d It is uncertain whether the data from Glover and colleagues represent the 5-yr or annual incidence rate. Standardisation used the 1970 US population data.

^e The study from Guadeloupe reported a range of incidence rates between 1995–2003. We selected the 1999 data as representative of rates prior to screening and comparable to our ascertainment period.

4.2. Limitations of this study

It is important to consider several important limitations to our study. We were not able to ascertain cases that were diagnosed and managed solely in the private sector except in one area in which private biopsies were processed through the National Health Service hospital. This omission is unlikely to have resulted in many missed cases because our men resided mainly in relatively deprived areas. However, it may have biased our results to overestimate the black–white differences, because we suspect that private cases would have been more likely to be white than black. Because of boundary changes, we had to aggregate areas to make them directly comparable between census years and then interpolate between the ethnic group and age-specific populations to estimate the populations during the study period. This approach is unlikely to have produced bias in the relative risks. Our age-standardised rate for all cases was 75.2 per 100,000 (including men of unknown ethnicity), which is consistent with routine cancer registration data for 1999 in similar parts of London (between 59 to 88 per 100,000) [22]. In 7% of cases, ethnic status was unknown. Our sensitivity analysis demonstrated that the results were not changed dramatically even under the extreme worst-case scenario that all such men were white.

4.3. Implications for aetiology and public health

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Our observation that younger black men were at greater relative risk is interesting and potentially important because such men have longer remaining life expectancy than their older counterparts and may have more to benefit from screening and early treatment. A case for screening would be further supported if one believes that black men have a worse prognosis [23,24] as suggested from US data. However, our own UK data do not suggest that black men have worse prognosis than white men (manuscript in preparation). Any decision to introduce population-based screening must have strong evidence that the benefits of screening outweigh the potential adverse effects [25].

Most of the black African men in our study originated from West Africa and hence would be genetically similar to black Caribbean men because the latter were transported from the West Coast of Africa to the Caribbean to work on plantations [26]. It is reasonable to assume that almost all our black men were born overseas and migrated to Britain sometime in their adulthood [27]. It is therefore likely that these men share a common genetic susceptibility for an increased risk of prostate cancer [28,29]. If one accepts that the rates in West Africa and the Caribbean are lower than those in the United Kingdom and United States, then the data suggest a "migrant" effect, whereby first-generation migrants in the United Kingdom have a greater risk than that in their country of origin, whilst African American men, who have had many more generations of exposure to a Westernised life style, have the greatest risk, assuming that PSA screening does not fully explain the much higher rates in the United States. This would strongly support the notion of an interaction between common environmental factors acting across the adult life course [30], which is related to Westernisation, and increased genetic susceptibility [3]. If one accepts the validity of the high rates from Jamaica, then the high rates observed in the United Kingdom and United States are, if anything, less than one would have predicted given better access to health care and PSA screening.

5. Conclusions

Our study has demonstrated that black men in the United Kingdom have a much higher relative risk of developing prostate cancer than white men and that this risk is more marked for younger men, although the absolute risk for black men in the United Kingdom is still less than that of African American men. Future research needs to confirm whether black men in the Caribbean and Africa have similar, higher, or lower rates than those for black men in the United Kingdom and United States so that we have a clear idea as to the role of migration on prostate cancer risk. If migration is associated with an increased risk, then epidemiologic studies need to identify potentially modifiable risk factors to guide primary prevention programmes.

Conflicts of interest

None of the authors has any conflict of interest related to this manuscript.

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Appendix A

The PROCESS study group included members from Bristol, UK (P. Abrams, D. Dickerson, S. Falk, R. Feneley, D. Gillatt, C. Gingell, J. Graham, F. Keeley, H. Newman, R. Persad, J. Probert, H. Schwaibold, G. Sibley, A. Timoney, R. Wells, T. Whittlestone, M. Wright); South West London (C. Anderson, K. Anson, M. Bailey, R. Kirby); South East London (D. Cahill, P. Dasgupta, J. Glass, S. Khan, G. Muir, T. O'Brien, R. Popert, J. Poulsen, P. Thompson, R. Tiptaft, K. Walsh), and North East London (N. Buchholz, F. Chinegwundoh, C. Fowler, I. Junaid, V. Nargund, A. Paris, J. Pati).

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