ORIGINAL PAPER



# Prostate cancer clinical presentation, incidence, mortality and survival in Guadeloupe over the period 2008–2013 from a population-based cancer registry

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#### Abstract

*Purpose* The Caribbean population of Guadeloupe has one of the highest incidence rates of prostate cancer worldwide. In 2008, a population-based cancer registry was set up for the monitoring of cancer incidence in the aftermath of the environmental pollution with chlordecone, a persistent organochlorine insecticide formerly used in banana plantations. We describe the clinical presentation, incidence, mortality and survival of prostate cancer for the period 2008–2013.

*Methods* The Guadeloupe cancer registry has been routinely collecting all incident cases of cancer since 2008. We compared age-specific incidence rates between different populations, and calculated incidence and mortality rates standardized to the world population. Kaplan–Meier observed survival and estimated age-standardized net survival were calculated by category for age, PSA level, and Gleason score using the Pohar-Perme method.

*Results* Overall, 3,295 cases of prostate cancer were recorded. World-standardized incidence and mortality were

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respectively 184.1 [177.8–190.4] and 23.9 [21.9–25.7] per 100,000 person-years. At diagnosis, the mean age of patients was  $68 \pm 9.6$  years old and 22% were aged over 75. Median PSA level was 8.9 [IQR: 6.0–16.0] and 13.6% of the patients had a Gleason  $\geq$  8. Five-year observed and net survivals were, respectively, 79.6% [77.9–81.2] and 90.7% [88.6–92.8].

*Conclusion* The incidence of prostate cancer in Guadeloupe is among the highest in the world, along with those of the neighboring Caribbean countries and US African-Americans. We observed no decrease in incidence rates, and a decreasing but non-significant trend in mortality rates, which nonetheless remain higher than in high-income countries. Many Genome-Wide Association Studies are conducted to identify genetic markers involved in prostate cancer risk. In the Caribbean, complementary studies on both lifestyle and behavioral factors should highlight potential common risks among populations who share both genetic and environmental characteristics.

**Keywords** Prostate cancer · Incidence · Mortality · Survival · Guadeloupe · Caribbean

## Introduction

Prostate cancer has become the second most common cancer in men in high resource countries worldwide. Nevertheless, the review conducted by Center et al. showed that the highest incidence rates were observed in populations of African descent, in North America, the United Kingdom and the Caribbean [1]. The increase in incidence observed over the last few decades is mainly explained by opportunistic screening made possible by the advent of the serum prostatespecific antigen (PSA) test and its widespread use since the late 1980s. In African and most Caribbean countries where PSA testing is not commonly used, the high incidence of prostate cancer is partly determined by genetics, but the relative share of this genetic risk remains debated [1].

The expected effect of early detection of prostate cancer with PSA testing, i.e., disease-specific and overall mortality reduction through early management, is controversial. The 2013 Cochrane meta-analysis conducted from randomized controlled trials found no evidence for a reduction in prostate cancer-specific mortality due to screening [2]. Moreover, the risks of over-diagnosis [3, 4] and over treatment, with the subsequent side effects on patients' quality of life, have been underlined [5]. Likewise, the decrease in mortality rates from prostate cancer observed since the mid-1990s also shows geographic variation, and age-standardized mortality rates are still high in predominantly Africandescent populations. The age-standardized mortality rates were 29.3/100,000 person-years in the Caribbean, varying between 19 and 24 per 100,000 in sub-Saharan Africa [6].

Among risk factors for prostate cancer, endogenous factors such as family history, African-descent population, and aging are well established, but the mechanisms of certain others, such as altered androgen metabolism, diet, environmental agents, occupation or lifestyle, remain unclear [7]. Environmental exposure has been explored for endocrine disrupting chemicals (EDCs), which positively or negatively alter hormone activity, ultimately affecting reproduction, development, and/or carcinogenesis, particularly in the reproductive organs. Both epidemiological studies and in vitro analyses with cancer cell lines have investigated the association between EDCs and prostate cancer carcinogenesis and/or susceptibility. Among the chemicals involved, the review by Hu et al. listed polychlorinated biphenyls (PCBs), polyhalogenated aromatic hydrocarbons, Bisphenol A (BPA), cadmium, and inorganic arsenic [8]. In 2013, the report of the UNEP/WHO on EDCs concluded that regarding prostate cancer, sufficient evidence exists in favor of an association with exposure to mixtures of pesticides in agriculture and in pesticide manufacturing, whereas evidence is conflicting for an association with PCBs and organochlorine exposure [9]. In Guadeloupe, a Caribbean archipelago of 404,000 inhabitants who are mostly of African descent, the combined effect of environmental and genetic factors may explain the high incidence rate of prostate cancer, which was first estimated at 168 in the year 2003 [10], before the implementation of the cancer registry. Although ethnic statistics are not allowed in France, it is commonly accepted that over 80% of the population is of African descent, while Indian descent and Europeans represent approximately 15 and 5% of the population, respectively.

Along with the neighboring island of Martinique, the French Department of Guadeloupe shares one of the highest incidence rates of prostate cancer worldwide. The wide use of pesticides, namely organochlorine and particularly chlordecone (Kepone), in banana plantations between 1973 and 1993 led to widespread pollution of the soil, drinking water, and some vegetable and animal food resources. The resulting contamination of the population has raised major public health concerns. In 2008, a population-based cancer registry was set up for the monitoring of cancer incidence in Guadeloupe. In parallel, both ecological analyses [11–13] and epidemiological studies have been conducted to assess the impact of this contamination on wildlife [14] and humans [15]. The monitoring of the effects of this contamination is still ongoing and specific measures are being implemented to prevent further contamination of the population through consumption of vegetables or water.

We describe the clinical presentation of prostate cancer, and estimate its incidence, mortality, and survival rates, to compare our results with those of European, US, and other Caribbean populations in this particular context.

#### Methods

We analyzed data from the population-based cancer registry of Guadeloupe for the period 2008-2013. This registry is member of the French network of Cancer registries [16] and of the International Association of Cancer Registries (IACR). It has been routinely recording all incident cases of cancer occurring in Guadeloupe since 2008. Potential cases are identified from multiple sources: pathology reports and hospital discharge records, long term illness registration by the health insurance system, and medical files. The mandatory data collected include demographic data (date and place of birth, gender, place of residence) and tumor characteristics (date of diagnosis, histological type). Other variables (PSA serum level at diagnosis, Gleason score, type, and date of first treatment) are routinely recorded when available. We use IACR rules for dealing with multiple tumors, for incidence. Population data for each year of incidence were obtained from the French National Institute of Statistics and Economic Studies (Insee) [17]. Data on death from prostate cancer for patients resident in Guadeloupe were obtained from the French epidemiological center on the medical causes of death run by the French National Institute of Health and Medical Research (CépiDc, Inserm). The CepiDc is the French national death registry and is responsible for producing annual national statistics on the medical causes of death. For follow-up of the cohort, and with the authorization of the French national authority for the protection of privacy and personal data (CNIL), data regarding the vital status of all individuals are provided by the CépiDc. Through this national system, which is fully integrated into the usual administrative formalities for all French citizens, a satisfactory exhaustiveness of death information is achieved.

The registry has also implemented an active follow-up of vital status at 5 years based on hospital records, laboratory data, and administrative databases.

We described population characteristics at diagnosis and calculated the age-specific incidence rates by 5-year periods, and the cumulative rate of prostate cancer for men aged 0-74 years, as presented in Globocan online analyses. Quantitative variables are reported as mean  $\pm$  standard deviation [4] or median [interquartile range] and qualitative variables as number (percentage).

Age-standardized incidence and mortality rates per 100,000 person-years [95% confidence interval (CI)], were calculated for the period 2008–2013 using the direct method and the world standard population, affording comparisons over time and across countries with different age compositions [18, 19]. Data from the SEER program for the US [20] and from the Globocan 2012 populations were used to build comparative curves of age-specific incidence rates. We performed joinpoint regression model and permutation tests [21] to identify changes in trends in incidence and mortality over the period using the software package Joinpoint Regression Program, Version 4.5.0.1—June 2017 of the SEER Statistical Methodology and Applications Branch, Surveillance Research Program, National Cancer Institute.

Observed survival after diagnosis was estimated using the Kaplan–Meier method [22] and presented with 95% confidence intervals. The endpoint date was set at 31 December 2015, which was the last update for patients' vital status from CepiDc or through an active search of the last follow-up date from various sources. Patients lost to follow-up and not identified by CepiDc were censored at the date of their last visit (recorded hospitalization or medical consultation).

Net survival, the survival which would be observed if prostate cancer were the only cause of death, was estimated with the unbiased Pohar-Perme estimator method [23]

Table 1Characteristics ofpatients with prostate cancer atdiagnosis by age group

using expected mortality rates derived from the observed mortality rates available by sex, annual age, year of death, and department of residence given by the French National Institute of Statistics and Economic Studies. This method, which does not require the cause of death, is recommended by Roche et al. for survival analysis of cancer registry data [24]. Results are given with 95% confidence intervals.

Statistical analyses were performed using Stata 14.0 (Stata Corp LLC, College Station, TX, USA) and a twosided p value less than 0.05 was considered statistically significant. Incidence, mortality, and net survival curves were built with Microsoft Excel<sup>®</sup> software.

# Results

From January 2008 to December 2013, 3,295 cases of prostate cancer were recorded. Mean age of the patients was  $68 \pm 9.6$  years. The diagnosis was based on histology of the primary tumor in 96.5% of cases, on specific tumor marker in 3.1% of cases and on histology of a metastasis in 0.3% of cases. The median PSA level was 8.9 [IOR: 6.0-16.0] and a Gleason score  $\geq 8$  was observed in 13.6% of cases. Table 1 presents the characteristics of the patients at diagnosis by age group. Over the study period, the crude incidence of prostate cancer was 291.9 per 100,000 person-years and the world-standardized incidence and mortality were 184.1 [177.8-190.4] and 23.9 [21.9-25.7] per 100,000 person-years (Fig. 1), respectively. The Annual percentage changes from the Join point analyses of respectively 0.5 for incidence and -6.8 for mortality did not differ significantly from zero at the alpha = 0.05 level for both incidence and mortality.

The cumulative risk of prostate cancer for men aged 0-74 years old was 36.1% over the study period. The

|  | All, <i>N</i> =3,295 | <60 years <i>N</i> =708 | 60–74 years $N = 1,857$ | $\geq$ 75 years $N =$ 730 |
|--|----------------------|-------------------------|-------------------------|---------------------------|
| PSA level, $n$ (%)                         |                      |                         |                         |                           |
| <10  | 1566 (47.5)          | 416 (58.8)              | 991 (53.4)              | 159 (21.8)                |
| [10-99]                                    | 1067 (32.4)          | 187 (26.4)              | 572 (30.8)              | 308 (42.2)                |
| ≥100                                       | 177 (5.4)            | 25 (3.5)                | 73 (3.9)                | 79 (10.8)                 |
| Unknown                                    | 485 (14.7)           | 80 (11.3)               | 221 (11.9)              | 184 (25.2)                |
| Gleason, n (%)                             |                      |                         |                         |                           |
| ≤6   | 1694 (51.4)          | 446 (63.0)              | 997 (53.7)              | 251 (34.4)                |
| 7  | 977 (29.6)           | 178 (25.1)              | 548 (29.5)              | 251 (34.4)                |
| ≥8   | 447(13.6)            | 49 (6.9)                | 216 (11.6)              | 182 (24.9)                |
| Unknown                                    | 177 (5.4)            | 35 (5.0)                | 96 (5.2)                | 46 (6.3)                  |
| Number of deaths (%)                       | 649 (19.7)           | 45 (6.4)                | 267 (14.4)              | 337 (46.2)                |
| Length of follow-up in years, median [IQR] | 4.0 [2.7–5.8]        | 4.1 [2.9–6.0]           | 4.2 [2.9–5.8]           | 3.4 [1.5–5.1]             |

Guadeloupe cancer registry 2008–2013

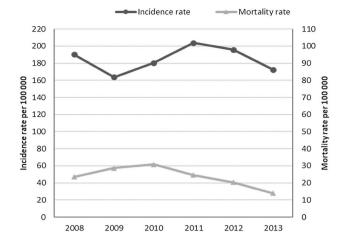
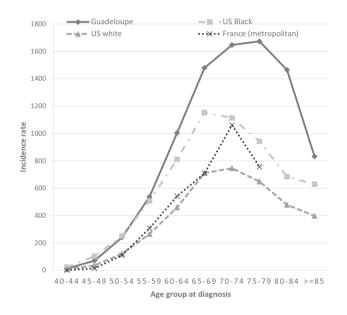


Fig. 1 World-standardized incidence and mortality rates of prostate cancer in Guadeloupe for the period 2008–2013



**Fig. 2** Age-specific incidence rates of prostate cancer in Guadeloupean men (cancer registry 2008–2013), US Black and White populations (SEER program 2009–2013) [20], and metropolitan France (Globocan 2012) [6]

age-specific incidence rates were higher in Guadeloupean men aged  $\geq$  55 years for the period 2008–2013 as compared to US populations (blacks and whites) and French metropolitan populations in the year 2012 (Fig. 2). However, above 75 years of age, the different age-grouping pattern, precluded comparison of the 4-way population rates. The median follow-up was 4 years [95% CI 2.7–5.8]. Up to December 2015, 649 (19.7%) deaths were notified. Overall, 5-year observed and net survival were, respectively, 79.6% [78.0–81.3] and 90.7% [88.6–92.8]. Table 2 presents observed and net survival at 1, 3, and 5 years after diagnosis. Observed survival was lower than net survival for all age groups and each time period. However, patients over 75 had greater differences, indicating that this age class had more deaths linked to causes other than prostate cancer.

Five-year net survival was 63% for patients with a Gleason score  $\geq$ 8 and 42.7% for patients with a PSA level > 100. Figure 3a–c present net survival by age group (3a), Gleason score (3b), and PSA level (3c).

## Discussion

This is the first report of prostate cancer incidence and mortality in Guadeloupe from a population-based cancer registry. The age-standardized incidence rate of 184.1 per 100,000 was much higher than the Globocan 2012 rate estimated from national mortality data [6]. We observed no significant trends in incidence and mortality rates. The age-specific incidence rates were higher for men over 55 years old compared to both US and French populations. The cumulative risk of prostate cancer was 36.1% for men aged 0–74 years over the study period. The 5-year net survival of 90.7% for this Caribbean population was comparable to high-income countries.

The last worldwide estimated incidences of prostate cancer age-adjusted to the IACR world population were given by the Globocan program 2012. These data showed that the highest rates were observed in the Caribbean, namely Barbados (123.1), Trinidad and Tobago (123.9) and Martinique (227.2) [6].

From the SEER Program, the incidence rate of prostate cancer in the US, age-standardized on the IACR world population, was 90.2 for the global population, 84.5 in white males, and 145.8 in black males over the period 2009-2013 [20]. These results put the Caribbean region and the US at the forefront of this major public health problem. It is known that incidence and trends in prostate cancer are closely linked to variations in the diagnosis of latent cancers with PSA testing, possibly leading to over-diagnosis, defined as the detection, through PSA testing, of prostate cancer that would not otherwise have been diagnosed within the patient's lifetime [3]. From a simulation model on PSA testing and subsequent prostate cancer diagnosis from the SEER registry data, Etzioni et al. found that an over-diagnosis rate of 29% in whites and 44% from blacks from PSA testing [3]. Moreover, the performance characteristics of PSA are not the same in all races. Serum PSA levels are higher in men of African descent than in whites [25] and PSA testing has a higher positive predictive value in black men than in white men [26].

As demonstrated by Telesca et al., prostate cancer incidence trends due to widespread implementation of screening show an initial increase, followed by a decline closely tied G : 1 @ 105@ CH

#### Table 2 Observed and Net survival at 1, 3, and 5 years after prostate cancer diagnosis

|               | Survival, % [95% CI] |                    |                  |                     |                  |                    |  |  |
|---------------|----------------------|--------------------|------------------|---------------------|------------------|--------------------|--|--|
|               | 1 year               |                    | 3 years          |                     | 5 years          |                    |  |  |
|               | Observed             | Net                | Observed         | Net                 | Observed         | Net                |  |  |
| Age groups    |                      |                    |                  |                     |                  |                    |  |  |
| <60           | 98.5 [97.2–99.2]     | 99.2 [98.3-100.2]  | 95.6 [93.7–97.0] | 97.9 [96.3–99.6]    | 94.3 [92.0–96.0] | 98.2 [96.2-100.3]  |  |  |
| [60–74]       | 97.9 [97.1–98.5]     | 99.7 [99.0-100.4]  | 91.9 [90.4–93.1] | 97.3 [95.9–98.7]    | 84.8 [82.7-86.7] | 93.8 [91.6–96.0]   |  |  |
| ≥75           | 88.4 [85.8–90.6]     | 94.4 [91.8–97.0]   | 71.2 [67.6–74.5] | 87.6 [83.3–92.0]    | 53.5 [49.1–57.7] | 76.3 [70.1–83.1]   |  |  |
| Gleason score | e                    |                    |                  |                     |                  |                    |  |  |
| ≤6            | 97.9 [97.1–98.5]     | 100.0 [99.3–101.0] | 93.9 [92.5–95.0] | 100.0 [99.0-102.0]  | 87.7 [85.5–89.6] | 98.3 [95.9–101.0]  |  |  |
| 7             | 97.0 [95.7–98.0]     | 99.8 [98.7–101.0]  | 88.2 [85.9–90.2] | 96.2 [93.8–98.7]    | 78.8 [75.6–81.6] | 90.5 [86.8–94.4]   |  |  |
| ≥8            | 87.5 [84.0-90.4]     | 91.0 [87.7–94.4]   | 66.5 [61.7–70.9] | 74.3 [69.2–79.9]    | 52.4 [47.0-57.6] | 63.0 [56.3–70.4]   |  |  |
| Unknown       | 91.9 [86.7–95.1]     | 94.4 [90.3–98.7]   | 88.4 [82.6–92.3] | 95.8 [90.7-101.2]   | 80.8 [73.8-86.2] | 93.2 [85.9-101.1]  |  |  |
| PSA           |                      |                    |                  |                     |                  |                    |  |  |
| <10           | 98.6 [97.8–99.1]     | 100.0 [99.8–101.0] | 95.8 [94.6–96.8] | 101.0 [100.3–103.0] | 90.6 [88.7–92.3] | 100.0 [97.8–102.0] |  |  |
| [10–99]       | 95.8 [94.4–96.9]     | 98.8 [97.5-100.1]  | 87.7 [85.5–89.6] | 96.5 [94.2–98.9]    | 78.8 [75.8–81.4] | 92.4 [88.9–96.2]   |  |  |
| ≥100          | 81.2 [74.3-86.4]     | 84.5 [78.4–91.0]   | 50.4 [42.4–57.9] | 56.8 [48.5–66.4]    | 35.2 [26.9-43.6] | 42.7 [33.5–54.5]   |  |  |
| Unknown       | 92.6 [89.7–94.8]     | 96.0 [93.4–98.7]   | 77.5 [73.1–81.2] | 86.3 [81.7–91.0]    | 62.9 [57.4–67.9] | 74.7 [68.0-82.1]   |  |  |

Guadeloupe cancer registry 2008-2013

to the lead time (i.e., the amount of time by which screening advances the diagnosis). This lead time has been estimated to be 6.8 years [5.42–8.20] in black men and 4.59 years [3.24–5.93] in white men [27].

As a French overseas department, free access to healthcare is available in Guadeloupe, through the French universal social welfare system. In the absence of any organized screening program for prostate cancer, PSA testing, a routine test, became even more common after the media scandal caused in 2007 by the disclosure of soil contamination by the organochlorine insecticide chlordecone, and the resulting risks to the population. Considering the lead time of 6.8 years estimated by Telesca et al. and the succession of events linked to the chlordecone scandal in our population in 2007, it is likely that the prostate incidence rates we report for the period 2008–2013 include a substantial number of latent prostate cancers. Over-diagnosis could therefore partly explain our observed incidence rate, but we were unable to verify this likelihood model of a decrease in incidence trends due to the short length of follow-up for our registry.

In Guadeloupe, first among all risk factors, environmental pollution with pesticides is being closely monitored. The Karuprostate case-control study conducted between 2003 and 2007, provided some evidence for an association between exposure to chlordecone and risk of prostate cancer. A positive and significant linear dose–response relationship was found between exposure to chlordecone, as estimated by plasma concentration, and the risk of developing prostate cancer [28]. The investigators showed that the risk was increased in patients with variant alleles of functional polymorphisms of chlordecone reductase. Genetic polymorphisms of different metabolic enzymes were also investigated. Emeville et al. studied the Glutathione S-Transferase genes T1 (GSTT1), whose main role in the conjugation of reactive metabolites may be altered by chemical exposure. They hypothesized, from a case-control study, that copy number of GTT1 and combined GSTM1/GSTT1 were associated with prostate cancer risk in Guadeloupe [29].

Along with environmental factors, genetic susceptibilities in African-descent populations are also studied to explain this high incidence of prostate cancer in our population. Thus, Brureau et al., studied the association between polymorphisms of five estrogen-related genes (CYP17, CYP19, CYP1B1, COMT, and UGT1A1) and prostate cancer risk in two different populations of African ancestry (Guadeloupe and the Democratic Republic of Congo). The AA genotype and the A allele of rs4680 [30] were inversely associated with prostate cancer risk in both populations [31]. These studies need to be interpreted with caution due to small sample sizes and need to be replicated in other populations.

These genetic and environmental risk factors could also be associated with lifestyle factors and diet, as suggested by the marked variation in prostate cancer incidence across geographic and ethnic groups and the observed changes in risk in migrants [32]. A possible association between global obesity measured by Body Mass Index (BMI) and prostate cancer has been suggested. From the Melbourne Collaborative Cohort Study on more than 17,000 men, BMI was found to increase the risk of aggressive prostate cancer and mortality from prostate cancer [33]. In the

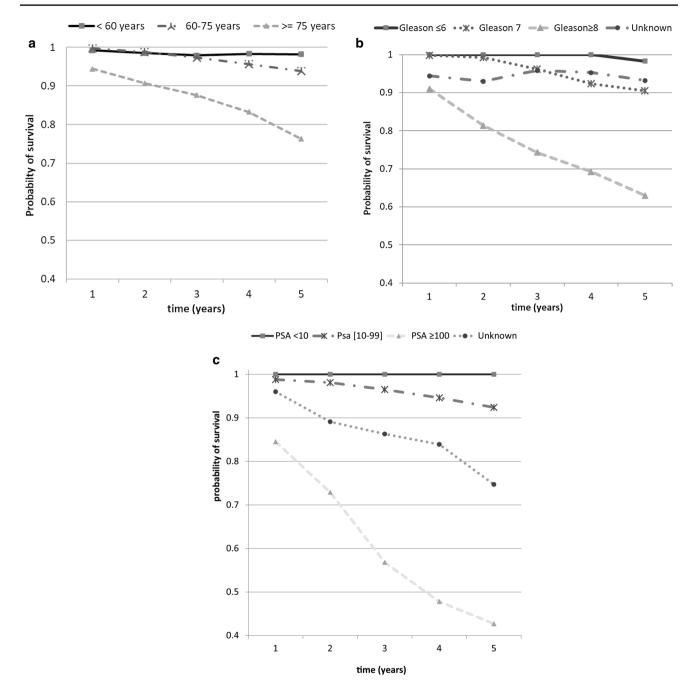


Fig. 3 a Net survival by age group, Guadeloupe cancer registry, 2008–2013. b Net survival by Gleason score, Guadeloupe cancer registry, 2008–2013. c Net survival by PSA level at diagnosis, Guadeloupe cancer registry, 2008–2013

US, a 35% obesity rate was found in blacks compared to 23.7% in whites [34]. For patients with prostate cancer, Khan et al. found an obesity rate of 39.8% in Black men compared to 37.8% in white men but obesity was associated with highly aggressive prostate cancer in white men only [35]. On the other hand, Parke et al. in the Multiethnic Cohort Study, found no racial/ethnic differences in prostate cancer risk associated to lifestyle factors [36]. In Guadeloupe, the prevalence of obesity was estimated to be

18% in men in the general population [37], but no data are available for men with prostate cancer.

These findings on BMI and prostate cancer risk need further studies. BMI is known to be an imperfect estimate of adiposity [38], particularly in men, mainly because of greater muscle mass. Von Hafe et al. showed, with a measure of body fat by computed tomography, that visceral fat rather than overall adiposity was more predictive of prostate cancer [39].

While differences in incidence rates are mainly explained by differences in PSA testing, ethnicity and/or environmental factors, the differences in mortality rates more likely reflect differences in practice and underlying risk. In the Caribbean, among the 21 English and Dutch speaking countries of the Caribbean Public Health Agency, prostate cancer was the leading cause of cancer deaths with age-standardized mortality rates ranging from 15.1 to 74.1 per 100,000. However, large disparities are observed, with high rates (41.3-74.1)for the Bahamas, Antigua & Barbuda and Dominica, middle rates (25.7-41.3) for Trinidad and Tobago, Barbados and Jamaica and low rates (9.4-25.7) for Puerto Rico and Guyana [40]. In the French speaking island of Guadeloupe, the mortality rate of 23.9 was among the lowest in the Caribbean area, and showed a non-significant decreasing trend. It remained, however, 2.3 times higher than the estimated rate of 10.0 in mainland France [6].

Differences in mortality have not only been attributed to genetic, nutritional, or hormonal factors, but also to socioeconomic and behavioral factors. Accordingly, differences in knowledge and access to health care, cultural misconceptions about prostate cancer and delays in seeking medical care, as well as treatment disparities have all been highlighted in many studies, mainly in the US [41, 42]. Africandescent populations in the US have poorer survival than whites at every stage of the disease. In a study from the California cancer registry, Robbins et al. showed that the large difference in prostate cancer survival between white men and black men was completely explained by known prognostic factors, with potentially modifiable disparities playing the largest role [43].

A tendency towards more advanced Gleason grades in the biopsy specimens was found among black men, who also had a proportionately higher percentage of locally advanced disease and higher pathological Gleason scores in the radical prostatectomy specimens. Thus, the hypothesis that prostate cancer is more aggressive among black men has repeatedly been suggested. In Guadeloupe, the A allele at rs16901979 was associated with both risk of prostate cancer and risk of aggressive prostate cancer defined by a Gleason score  $\geq$ 7 [44], in agreement with the study by Okobia et al. of African-Caribbean men from Tobago [45]. Our data shows that Guadeloupe cases with Gleason seven have lower net survival compared to those with a Gleason score below six, but the observed and net survival findings also indicate that a substantial portion of cases have limited and potential endemic, rather than aggressive disease. Therefore, the risk of overtreatment leading to urinary, sexual, and bowel health-related quality of life issues over time must be carefully assessed.

Comparison of patient survival between different populations warrants careful attention. Net survival allows comparisons of the ability of different countries to effectively treat patients by overcoming the disparities in mortality associated with other causes of death in these countries. In our population, the overall 5-year observed survival was 79.6%, while net survival was 90.7%. This net survival was comparable to data for mainland France (90.5%), given by the CONCORD-2 study for an earlier period (2005–2009). In the US, net survival was above 97%. Few data were available for the Caribbean countries, except for Cuba, with a reported net survival of 56.1% [46]. Survival estimates using expected mortality rates have proven to be a more reliable estimate of the net survival from cancer than cause-specific survival. Nevertheless, for some cancers diagnosed at an early stage (i.e., prostate, breast, colon, and rectum), relative survival has been shown to be greater than 100%, indicating that the life tables may not be appropriate for representing survival [47-49]. A "healthy screening effect" can also be discussed. Thus, men who seek screening on a regular basis are more health conscious and may not have the same expected mortality as the general population. Our data show some survival estimates higher than 100%, mainly for patients with PSA < 10 and Gleason < 6, in line with the idea that these populations have better follow-up and, therefore, better health status than the general population.

Our data confirm that access to care is satisfactory in Guadeloupe for patients with prostate cancer, partially offsetting the potential aggressiveness of the disease. Nevertheless, the increasing incidence and the persistently high mortality are not yet controlled.

Along with environmental risk factors and lifestyle changes, genetic markers involved in prostate cancer risk are being explored with Genome-Wide Association Studies (GWAS) pooling resources and clinical data of consortia [50]. Thus, prostate cancer risk has been associated with 76 susceptibility loci. Within the Caribbean, along with these GWAS, complementary studies on both lifestyle and behavioral factors, should highlight potential common risks [51]. Indeed, as reported by Odedina et al., most African-Caribbean and African-Americans have ancestries from Benin, Nigeria, Ghana, Gambia, Senegal, Mozambique, and Angola because of the transatlantic slave trade [52] and may share common polymorphisms involved in the risk of prostate cancer, and/or in predicting high-grade disease.

The limitations of our study include those inherent to a general cancer registry, mainly exhaustiveness and incomplete data for variables that are not mandatory. Unlike some other cancer localizations that require management outside the island, prostate cancer has good coverage of diagnosis and care, performed locally by the three pathologists and the three main health care facilities. The registry has complete access to all these sources, via a specific module to extract data for the pathologists, who use the same management software, and from on-site consultation of medical records. Furthermore, access to the national database of hospital records, as well as cause-specific mortality data and individual vital status from the French national database allow us to guarantee a satisfactory exhaustiveness and quality of the data presented.

## Conclusion

Prostate cancer incidence and mortality are a major concern worldwide and raise particular concern in populations of African descent. Black populations in the Caribbean share environmental, nutritional, cultural and genetic history. More collaborative studies are needed to assess risk and prognostic factors of prostate cancer in populations of African descent with the common goal of reducing the burden of the disease in these populations.

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