

Epidemiomorphology of prostate cancer in Cameroon: about 1047 cases

Abstract

Objective: To determine the epidemiological and histological profile of prostate cancer in Cameroon.

Material and methods: It was a descriptive and retrospective study over a period of 12 years (2004-2015), concerning prostate cancers in five regions (Center, Littoral, West, North-west South-west). The studied parameters were frequency, age, PSA, histological type and Gleason score.

Results: We collected 1047 prostate cancers, which represented 81.42% of all urogenital cancers. The average age was 66.88 ± 9.58 years (range 32–98 years). The most affected age group was 60-79 years (736 cases), however, 3.15% (33 cases) were under 50 years. Histologically, we found a predominance of adenocarcinoma with 1044 cases (99.71%). Moderately differentiated tumors (Gleason score 7-8) represented the majority 56.56% (590 cases) of cases.

Conclusion: Prostate cancers are the predominant malignant uro-genital tumors in Cameroon. We noted the significant presence of relatively young men. Adenocarcinoma is still the main histological type. Moderately differentiated tumors are the most common.

Keywords: Cancer; prostate; epidemiology; histopathology; Cameroon.

1. INTRODUCTION

The prostate cancer is the most frequent neoplasm in humans and the second leading cause of cancer death after bronchopulmonary cancers [1]. This incidence, once underestimated, has been increasing since the popularization of screening methods, namely rectal examination and the PSA rate, thus representing a real public health challenge. In the United States, it is the first cancer with an incidence of 187 per 100,000 population (116 Black and 71 Caucasian) with an average age of diagnosis 61 years [3, 4]. In Europe the incidence varies between 25-193 per 100 000 inhabitants, in France it is 187, with a standardized mortality rate of 10. The survival rate at 5 years is 88% higher than the European average of 83 %, the mean age of the diagnosis was 71 years [5-6]. In Africa, for example in Togo, the prostate cancer accounts for 74.63% of all urological cancers, with an average age of diagnosis of 67.8 years, and 8.3% of cases were recorded in people younger than 50 years [7]. Unlike the West, the survival rate in Africa does not seem to exceed 50%, for example in Uganda the 5-year survival rate was estimated at 47.2% [8]. Many risk factors are mentioned for this cancer including age, race and family history of prostate cancer [9]. Other risk factors are emerging, such as diet, lifestyle and especially genetic factors [9]. In the management of this pathology in the country, the various treatments used are surgery, radiotherapy, chemotherapy and hormone therapy [3]. In Cameroon, the urogenital apparatus is the target of 6.5% of all malignant neoplasms [10], prostate cancer accounts for the vast majority of (72%) of these cancers, with 6.32% of patients diagnosed before the age of 50 years [11]. Numerous studies show that the dominant histological type in our country is adenocarcinoma [10-12]. However, it should be noted that these studies are regional studies and

so on. This is why we propose to carry out a national study in order to establish an epidemiological profile and the histological aspects of prostate cancers in Cameroon.

2. MATERIAL AND METHODS

This is a retrospective and descriptive study. It covers cases of prostate cancer diagnosed over a period of 12 years (2004 to 2015) in the approved anatomy-pathology laboratories of different cities in Cameroon (Yaoundé, Douala, Bafoussam, Bamenda and Buéa). Patients were consulted in the urology or oncology departments of different health facilities spread throughout the national territory, patients had benefited from a biopsy sample or from an enlarged surgical resection of the tumor. For each case recorded, the results of the anatomopathological examinations which had been carried out after fixation of the biopsy parts and of the operative parts with 10% formalin were investigated. Only patients whose diagnosis of prostate cancer was confirmed by histological examinations were included in this series. The data consisted of independent variables such as frequency, patient age, prostate specific antigen (PSA), histologic type of tumor, and Gleason score. The dependent variable was prostate cancer confirmed by histology. The analysis of the variables was carried out using the Statistical Package for Social Sciences (SPSS) software, version 16.0. The elements of the descriptive statistics made it possible to calculate frequencies and proportions. The data have been shown in tables and figures.

3. RESULTS

3.1. Frequency:

In our study, we found 1286 cases of urogenital cancers in Cameroon, of which the prostate was the first with 1047 cases, 81.42% followed by that of the kidney, 110 cases (8.55%), bladder 6.30%, the testis-30 cases (2.33%), the penis (0.78%), the urethra-4 cases (0.31%) and the ureter-4 (0.31%) (Figure 1).

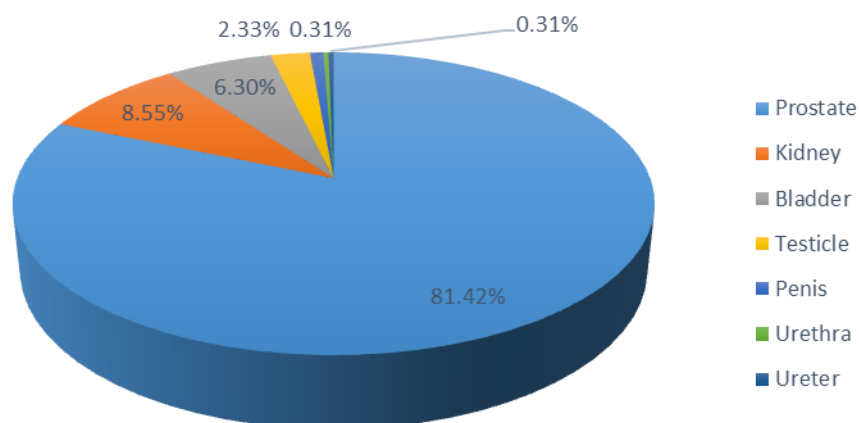
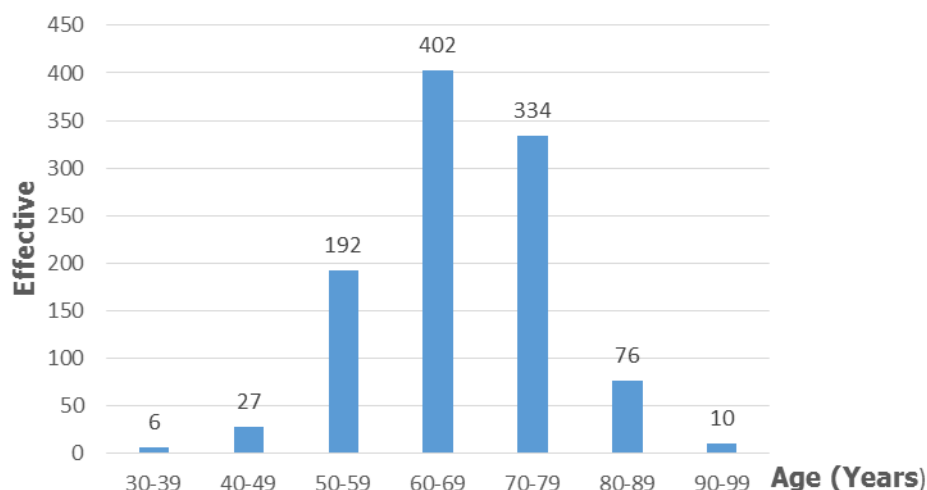


Fig.1. Distribution of urogenital cancers by seat

3.2. Distribution according to age groups

The mean age of diagnosis was 66.88 ± 9.58 years, with extremes ranging from 32 to 98 years. As shown in Figure 2, 70.30% (736 cases) of patients were between the ages of 60 and 79

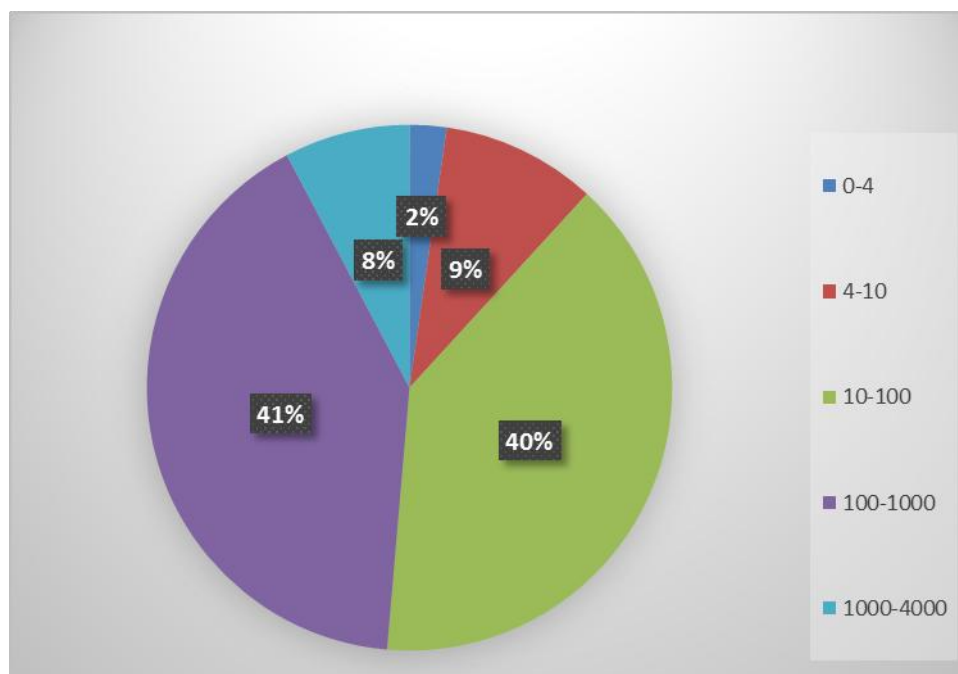
73 years, 3.15% (33 cases) of patients were under 50 years of age, of which 06 were younger than
74 40 years of age.



75
76 **Fig.2. Distribution of Patients by Age**

77 **3.3. PSA**

78 The average PSAT rate was 419.71 ng / ml, with extremes ranging from 3 to 4000.
79 According to Figure 3, of the 219 PSAs in our study, we identified 5 patients (2.28%). with a
80 PSAT level of less than 4 ng / ml; 90 (41.09%) patients with a rate between 100 and 1000 and 17
81 (7.76%) patients with a rate higher than 1000



82
83 **Fig.3. distribution of Patients according to total PSA level**

84 **3.4. Anatomopathology**

85 **3.4.1. Types of sampling**

Of the 1047 cases of prostate cancer identified in the study, 885 were from biopsies and 162 from surgical specimens (88 pieces of prostatectomy and 74 pieces of adenomectomy)

3.4.2. pTNM Classification

Among prostatectomy specimens, 43 were classified as pT2a, 16 were classified as pT2b and 29 pT3. The margin status was not specified on the different samples (Table 1).

Table 1. Pathological classification of prostatectomy parts.

Classification pTNM	Prostatectomy Parts	
pT2a	43	
pT2b	16	
pT3	29	
Total	88	

3.4.3. Histological type

In total, 1044 (99.71%) were adenocarcinomas. We also identified two (02) lymphomas in 64, 68 year olds, and one (01) sarcoma in a 49-year-old patient.

3.4.4. Gleason Score

As shown in FIG. 5, among the adenocarcinomas, the moderately differentiated tumors (Gleason score 7-8) accounted for the majority 56.56% (590 cases) of the cases; followed by well-differentiated tumors (Gleason score 4-6). 36.33% (379 cases) and undifferentiated tumors (Gleason score 9-10) 7.09% (74 cases) of the cases.

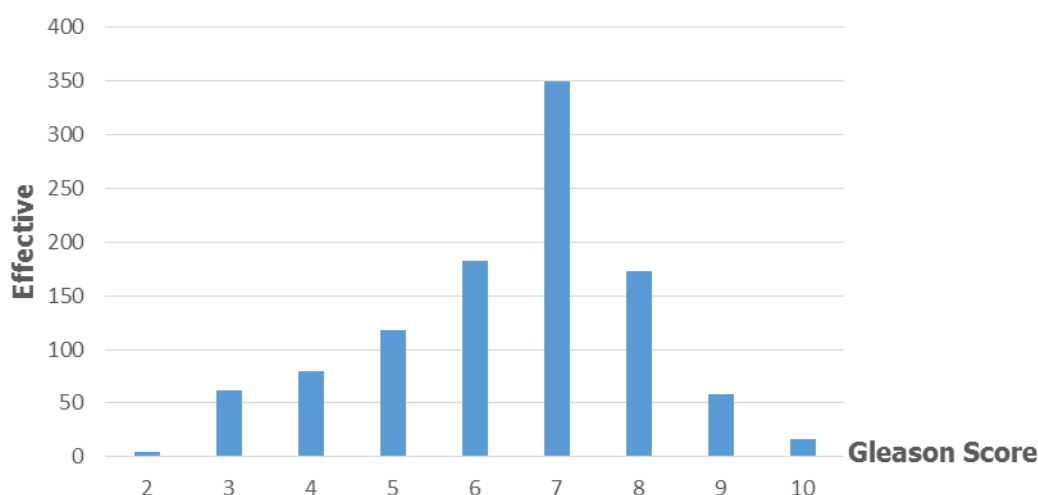


Fig.5. Distribution according to Gleason score

4. DISCUSSION

From the national point of view, there is an increase in the incidence of prostate cancer compared to other urological cancers. Indeed, in 2006, Sow et al found a frequency of cancers of the prostate 60% compared to other urological; in 2012 Engbang et al 72% [11, 12]. However It should be noted, that these two studies were regional studies, while our studies are national in character. In the West, prostate cancer is also the leading cause of cancer in men [1, 2, 5]. In England, the frequency among urological cancers (69%) is clearly lower than ours [13]. The incidence of prostate cancer would be increased by the relative popularization of the PSA test in our country. Indeed, it has been observed that in many developing countries, as in developed countries, the introduction of these tests has clearly increased the incidence of this pathology [14].

In our study, prostate cancer ranked first with 1047 cases (81.42%). This result is similar to that found by Engbang et al in the Littoral and Darré et al in Togo respectively 72.70% and 74.63% [7,11]. These rates have been found by several authors to confirm the prevalence of prostate cancer among urologic malignant tumors in the world, and which would be the number one in front of lung, stomach and colorectal cancers [1].

The mean age of diagnosis was 66.88 ± 9.58 years, with extremes ranging from 32 to 98 years. 3% (32 cases) of patients were under 50 years of age, of which 05 were younger than 40 years of age. This average age is comparable to that found by Gueye et al in Senegal, 69 years [15]. But it remains lower than those found in Guinea-Conakry, Morocco and France respectively 70 and 71 years in Morocco and France [6, 16, 17]. However, the average age of our patients is higher than that of the United States, which is 61 years [4]. Our relatively low values could be explained first by our very recent data compared to the above-mentioned African studies made for the most recent study in 2006, when the use of SAP was not yet widespread on the continent. This is compounded by the number of cases under the age of 50. For this young population, the recovered rate (3%) is higher compared to those of some African countries which hardly find patients under 50 years of age. Nevertheless, other studies of the continent find rates much higher than ours; including a Senegalese study of autopsy documents, which found 3% for the 20-29 age group, 28% for the 30-39 age group and 40% for the 40-49 age group with

extremes of 20 and 29 years. 90 years, and a Togolese study 8.3% of subjects under 50 years of age [7, 18]. These last cases re-emerge the question of the age of onset of screening, especially in the African subject. The African population, according to several authors would be more affected by prostate cancer and the average age at diagnosis seems lower both in Africa and in African-Americans unlike the Caucasians [4, 19]. It has been shown that men of African origin are 2.5 times more likely to develop prostate cancers than Caucasians who are even more at risk than Asian men. One cause is attributed to specific genetic variation within these groups, including the high prevalence of SRD5A1 gene (steroid-5-alpha-reductase, alpha polypeptide 1 (3-oxo-5-alpha-steroid delta 4 -dehydrogenase alpha 1)), which forms the enzyme, Steroid 5-alpha-reductase (EC 1.3.99.5) catalyzing the conversion of testosterone to more potent androgen, dihydrotestosterone (DHT) [20].

With 77.55% of patients over the age of 60 years, other authors agree that advanced age is a risk factor for prostate cancer and the cause of this can be attributed to genetic alterations emanating from the aging process [21]. Thus, several reasons are mentioned, namely, the variation of hormonal levels and the alteration of the testosterone ratio E2 [22]. Age is likely to result in decreased plasma levels of androgens and an increase or a constant of estrogens, thus causing a decrease in the androgenic ratio to plasma estrogen [23]. This is why other authors discuss the probable role of the enzyme UGT2B28 (Uridine diphospho-glucuronosyltransferases) in the tumor process of the prostate, knowing that it has the capacity to glucuronide both testosterone and oestradiol and thus play on the equilibrium of this ratio [24].

According to some authors, the genetic factor plays an important role in the development of prostate cancer, about 42% of the total risk [25]. Some genes have been identified as susceptible to prostate cancer because they are involved in cell cycle control, DNA repair, androgen response, metastatic development, as well as in other defense activities; they are essentially BRCA2, PON1, CHEK2, RNASEL, ELAC2 and many others [26]. Many other factors are involved in the development of prostate cancer. It has been shown that populations whose origins are not exposed to this pathology (eg, Asians) have seen their risk increase due to their displacement in areas with more affected populations [27]. This confirms the fact that certain factors, in particular exogenous factors, play a role in the transition from so-called latent prostate cancer to clinical prostate cancer. These include factors such as food, sexual behavior, alcohol consumption, exposure to ultraviolet radiation, occupational exposure [28]. However, factors such as obesity, metabolic syndromes and decreased physical activity may also increase this risk [28].

In the study, the average PSAT rate was 419.71, with extremes ranging from 3 to 4000. The average PSA regimen is very high compared to England, where it is about 15ng / ml with extremes ranging from 3.4 to 79.5, and other African countries, including Senegal where it is 72.2 ng / ml and Morocco where it is 10ng / ml [13, 15, 17]. This fact, correlated with the later age of diagnosis underlies a long evolution of the disease before diagnosis. This set of PSA makes us ask the question of a peculiarity of PSA high among Cameroonians, nevertheless our low number of PSA does not allow us to conclude. The PSA remains an essential element in the diagnosis of prostate cancer, a diagnosis that combines a rectal examination and biopsy data [29]. If this test is to have a detection rate higher than the rectal examination, it should not be forgotten that the sensitivity is low (36%) [30]. This is the reason why any high PSA value cannot be taken as prostate cancer because such values are also found in prostatitis and benign prostatic hyperplasia [30]. There are no agreed standards defining the PSA measure; the latter remains a continuous parameter, with a strong estimate of the probability of prostate cancer in a subject [28]. Many men may suffer from prostate cancer, but have a low serum PSA, even if the

Gleason is greater than or equal to 7 [31]. The popularization of the use of this test has favored the growing number of over-diagnosis of cancer, especially for so-called indolent forms; 90% of men newly diagnosed with prostate cancer undergo treatment for cancer with no clinical consequences or life expectancy [32]. This is why serum PSA, Gleason score, and T-stage are more useful together than alone in the final prediction of the pathological stage [33].

Adenocarcinoma was the most frequent tumor, as found in other authors on the continent and elsewhere [1, 3, 7, 11], the prostate being an organ consisting of very large glandular cells. The moderately differentiated tumors accounted for the majority of adenocarcinomas. These results are in contrast to those found on the continent, notably in Guinea and Togo [7, 79], in which well-differentiated tumors ranked first. However, we know that Gleason prostate tumors > 7 are worse prognostic with a survival rate. These results seem to indicate an increased aggressiveness of prostate cancer in Cameroon compared to its neighbors on the continent. However, it would be desirable to dissect the Gleason score of biopsies and that of prostate tumors. Concerning the first, several authors have shown the crucial importance of the Gleason score on these types of play. Malavaud et al. concluded that Gleason's biopsy score was the only preoperative factor informing the clinician on secondary biological progression, an independent risk factor for biological recidivism, but more significant than PSAi [34, 35]. Gleason score of the operative piece is one of the main predictors of recurrence after radical prostatectomy [36, 37]. Thus, it is necessary to understand that the serum PSA association, Gleason score, stage T is necessary, or even indispensable the final prediction of the pathological stage [33].

5. CONCLUSION

Prostate cancer remains the first malignant tumor of the urogenital shope in Cameroon. The most common histologic type is adenocarcinoma, with moderately differentiated tumors being the most common. The organization of awareness-raising campaigns and the establishment of cancer registries would notably allow not only the control of real epidemiology, but also the organization of the fight against this pathology.

CONSENT

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Globocan (2012) Prostate cancer incidence and mortality worldwide in 2012. http://globocan.iarc.fr/Pages/summary_table_site_sel.aspx
2. Belot A, Grosclaude P, Bossard N, et al. Cancer incidence and mortality in France over the period 1980-2005. *Rev Epidemiol Sante Publique* 2008; 56 (3): 159–75
3. Hutiev TS, Engbang NJP, Owona MLJ, Hosroev RG, Beslekoiev US. Epidemiology of prostate cancer in North-Ossetian Republic (Russia). *J Afr Cancer* 2015; 7:122-126
4. SEER 9 areas and US Mortality Files (National Center for Health Statistics, CDC). Rates are age-adjusted to the 2000 US Std Population (19 age groups - Census P25-1103). Regression lines are calculated using the Joinpoint Regression Program Version 4.2.0, April 2015, National Cancer Institute.

- 232 5. Ferlay J, Steliarova-Foucher E, Lortet-Tieulent J, Rosso S, Coebergh JWW, Comber H,
233 Forman D, Bray F. Cancer incidence and mortality patterns in Europe: Estimates for 40
234 countries in 2012. *European Journal of Cancer* 2013; 49:1374– 1403
- 235 6. Trama A, Foschi R, Larran N, Sant M, Fuentes-Raspall R, Serraino D, Tavilla A, Van
236 Eycken L, Nicolai N, and the EUROCare-5 Working Group¹. Survival of male genital
237 cancers (prostate, testis and penis) in Europe 1999–2007: Results from the EUROCare-
238 5 study. *European Journal of Cancer* 2015 ; 51 :2206– 2216
- 239 7. Darré T, Amégbor K, Kpatcha M, et al. Cancers urologiques au Togo : profil
240 hísticoépidémiologique à propos de 678 cas. *J Afr Cancer* 2014; 6 : 27-31
- 241 8. Sankaranarayanan R, Swaminathan R, Jayant K, Brenner H. An overview of cancer
242 survival in Africa, Asia, the Caribbean and Central America: the case for investment in
243 cancer health services. *IARC Sci Publi* 2011; (162):257-91.
- 244 9. Gann PH. Risk factors for prostate cancer. *Rev urol* 2002; 4(5): S3-S10.
- 245 10. Mbakop A, Essame Oyono JL, Ngbangalm C, Abondo A. Epidémiologie actuelle des
246 cancers au Cameroun. *Bull Cancer* 1992 ; 79 : 1101-1104.
- 247 11. Engbang NJP, Sala B, Moby H, Fonkwa C, Essomba B, Essam Sime JD, Ateba G, Fewou
248 A. Cancers urogénitaux dans la région du littoral-Cameroun : épidémiologie et
249 histopathologie. *Revue de Médecine et de de pharmacie* 2014 ; 4(2) : 440-446
- 250 12. Sow M, Nkegoum B, Essame Oyono JL, Garoua, Nzokou A. Aspects épidémiologiques
251 et histopathologiques des tumeurs urogénitales au Cameroun. *Progrès en Urologie* 2006 ;
252 16 :36-39
- 253 13. Maruthappu M, Barnes I, Sayeed S, Ali R. Incidence of prostate and urological cancers in
254 England by ethnic group, 2001-2007: a descriptive study. Maruthappu et al. *BMC Cancer*
255 2015; 15:753.
- 256 14. Torre LA, Bray F, Siegel RL, Ferlay et al. *Global Cancer Statistics, 2012. CA. A Cancer*
257 *Journal for Clinicians* 2015; 65: 87-108.
- 258 15. Gueye SM, Jalloh M, Labou I et al. : Profil clinique du cancer de la prostate au Senegal.
259 *African journal of urology* 2004; 10(4):203-207.
- 260 16. Diallo AB, Youwe N, Barry AM et al. Caractéristiques cliniques du cancer de la prostate
261 en guinée. Résultats sur la période 2000-2006. *African journal of urology* 2007 ;
262 13(4)280-287.
- 263 17. Ammani A, Janane A, Chafiki J, Sossa J, El harrech Y et al. Profil Epidémiologique Du
264 Cancer De La Prostate Dans Le Service D’urologie De L’hôpital Mohammed V De
265 Rabat. *J Maroc Urol* 2007 ; 5:11-14.
- 266 18. Kpatcha TM, Anoukoum T, Darre T, Botcho G et al. Prévalence du cancer de la prostate
267 à l’autopsie chez le noir africain. *J. RECH. Sci. Univ. Lomé (Togo)* 2013;15(3): 393-397.
- 268 19. Edwards SM, Eeles RA. Unravelling the genetics of prostate cancer. *Am J Med Genet C*
269 *Semin Med Genet* 2004; 129C:65-73.

- 270 20. Park SY, Haiman CA, Cheng I, Park SL, Wilkens LR, Kolonel LN, Le Marchand L,
271 Henderson BE. Racial/ethnic differences in lifestyle related factors and prostate cancer
272 risk: the Multiethnic Cohort Study. *Cancer Causes Control* 2015; 26(10):1507-15
- 273 21. Scosyrev E, Messing EM, Mohile S, Golijanin D, Wu G. Prostate cancer in elderly.
274 *Cancer* 2012, 118(12):3062-3070.
- 275 22. Black A, Pinsky PF, Grubb RL, Falk RT, Hsing AW et al. Sex steroid hormone
276 metabolism in relation to risk of aggressive prostate cancer. *Cancer Epidemiol*
277 *Biomarkers Prev* 2014; 23(11): 2374-2382.
- 278 23. Burton A.J, Tilling KM, Holly JM, Hamdy FC, Rowlands MA, Donovan JL, Martin RM.
279 Metabolic imbalance and prostate cancer progression. *Int J Mol Epidemiol Genet* 2010;
280 1(4): 248-271.
- 281 24. Belledant A, Hovington H, Garcia L, Caron P, Brisson H et al. The UGT2B28 Sex-
282 steroid Inactivation Pathway Is a Regulator of Steroidogenesis and Modifies the Risk of
283 Prostate Cancer Progression. *Eur Urol* 2016; 69(4):601-9
- 284 25. Cancel-Tassin, G. and O. Cussenot. Genetic susceptibility to prostate cancer. *BJU Int*
285 2005; 96(9): 1380-1385.
- 286 26. Eeles, R., C. Goh, E. Castro, E. Bancroft, M. Guy, A. A. Al Olama, D. Easton, Z. Kote-
287 Jarai. The genetic epidemiology of prostate cancer and its clinical implications. *Nat Rev*
288 *Urol* 2014; 11(1): 18-31.
- 289 27. Kolonel LN, Altshuler D, Henderson BE. The multiethnic cohort study: exploring genes,
290 lifestyle and cancer risk. *Nat Rev Cancer* 2004; 4(7):519–527.
- 291 28. Leitzmann MF, Rohrmann S. Risk factors for the onset of prostatic cancer: age, location,
292 and behavioral correlates. *Clin Epidemiol* 2012; 4:1-11.
- 293 29. Buyyounouski MK, Pickles T, Kestin LL, Allison R, Williams SG. Validating the
294 Interval to Biochemical Failure for the Identification of Potentially Lethal Prostate
295 Cancer. *J Clin Oncol* 2012; 30:1857-1863.
- 296 30. Comparison of Digital Rectal Examination and Prostate Specific Antigen in Early
297 Detection of Prostate Cancer; Ivan Karner Josip Galić; *Collegium antropologicum* 2003;
298 27(1): 61-6
- 299 31. Thompson IM, Pauler DK, Goodman PJ, et al. Prevalence of prostate cancer among men
300 with a prostate-specific antigen level < or = 4.0 ng per milliliter. *N Engl J Med* 2004;
301 350(22):2239-46.
- 302 32. Pepe P, Panella P, D'Arrigo L, Savoca F, Pennisi M, Aragona F. Should Men with
303 Serum Prostate-Specific Antigen ≤ 4 ng/ml and Normal Digital Rectal Examination
304 Undergo a Prostate Biopsy? *Oncology* 2006; 70:81–89
- 305 33. Eggener SE, Badani K, Barocas DA, Barrisford GW, Cheng JS, et al. Gleason 6 Prostate
306 Cancer: Translating Biology into Population Health. *J Urol* 2015; 194(3):626-34
- 307 34. Malavaud B, Game X, Villers A, Mouzin M, Mazerolles C,
308 Rischmann P, et al. Secondary biological recurrence after radical
309 prostatectomy: multivariate analysis of prognostic clinical, biological, and

- 310 histologic factors. Prog En Urol J Assoc Fr Urol Société Fr Urol.
311 2001; 11(2):277-82.
- 312 35. D'Amico AV, Whittington R, Malkowicz SB, Schultz D, Schnall M,
313 Tomaszewski JE, et al. A multivariate analysis of clinical and pathological
314 factors that predict for prostate specific antigen failure after radical
315 prostatectomy for prostate cancer. J Urol 1995; 154(1):131 – 8
- 316 36. Pierorazio PM, Walsh PC, Partin AW, Epstein JI. Prognostic Gleason
317 grade grouping: data based on the modified Gleason scoring system.
318 BJU Int 2013; 111(5):753–60.
- 319 37. Hernandez DJ, Nielsen ME, Han M, Trock BJ, Partin AW, Walsh
320 PC, et al. Natural History of Pathologically Organ-Confining (pT2),
321 Gleason \leq 6 Prostate Cancer Following Radical Prostatectomy Urology 2008;
322 72(1):172–6.
- 323