Differences in vitamin D status may account for unexplained disparities in cancer survival rates between African and white Americans

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Keywords: cancer survival, disparities, vitamin D, ultraviolet-B, African-Americans

Considerable disparities in cancer survival rates exist between African Americans (AAs) and white Americans (WAs). Various factors such as socioeconomic status (SES), cancer stage at time of diagnosis, and treatment—which this analysis considers primary explanatory factors—have accounted for many of these differences. An additional factor not usually considered is vitamin D. Previous studies have inversely correlated higher solar ultraviolet-B (UVB) doses and serum 25-hydroxyvitamin D (25(OH)D) concentrations with incidence and/or mortality rates for about 20 types of cancer and improved survival rates for eight types of cancer. Because of darker skin pigmentation, AAs have 40% lower serum 25(OH)D concentrations than WAs. This study reviews the literature on disparities in cancer survival between AAs and WAs. The journal literature indicates that there are disparities for 13 types of cancer after consideration of SES, stage at diagnosis and treatment: bladder, breast, colon, endometrial, lung, ovarian, pancreatic, prostate, rectal, testicular, and vaginal cancer; Hodgkin lymphoma and melanoma. Solar UVB doses and/or serum 25(OH)D concentrations have been reported inversely correlated with incidence and/or mortality rates for all of these cancers. This finding suggests that future studies should consider serum 25(OH)D concentrations in addressing cancer survival disparities through both measurements of serum 25(OH)D concentrations and increasing serum 25(OH)D concentrations of those diagnosed with cancer, leading to improved survival rates and reduced disparities.

Background

Considerable disparities in cancer survival rates exist between African Americans (AAs) and white Americans (WAs). Various factors such as socioeconomic status (SES),¹ cancer stage at time of diagnosis, and treatment² have accounted for many of these disparities. Educational attainment is often used as a proxy for SES.³ Other factors include insurance status,⁴ social determinants in general⁵,⁶ and genetics.⁷,⁸ However, even when analyses of cancer survival data include all known or suspected factors affecting survival, AAs still tend to have a lower survival rate than that of WAs, possibly because of unmodeled factors such as biological differences, and perhaps as a consequence of educational level and access to health care as several authors have noted.⁹⁻¹²

Discussions of cancer survival disparities generally overlook the role of vitamin D. For 2001–2004, AAs older than 60 y had a population mean serum 25-hydroxyvitamin D [25(OH)D] concentration of 17 ng/ml compared with 25 ng/ml for WAs.¹³ Prevalence of hypovitaminosis D ([25(OH)D < 15 ng/ml]) in the South was 45% among blacks and 11% among whites.¹⁴ In patients participating in a randomized controlled trial of chemotherapy, serum 25(OH)D concentrations were lower in black patients than in white patients and patients of other race (median, 10.7 vs 21.1 vs. 19.3 ng/ml, respectively; p < 0.001), as well as in females compared with males (median, 18.3 vs 21.7 ng/ml, respectively; p = 0.0005).¹⁵ Solar ultraviolet-B (UVB) irradiance is the primary source of vitamin D for most Americans, accounting for 80–90% of vitamin D.¹⁶ AAs, with darker skin, are less efficient at producing vitamin D from UVB irradiance.¹⁷ In addition, AAs are less likely to have as much vitamin D from oral intake.¹⁸

A large body of literature supports a beneficial effect of vitamin D in reducing the risk of cancer incidence and mortality rates. The UVB-vitamin D-cancer hypothesis was proposed in 1980.¹⁹ Many ecological studies²⁰⁻²⁴ have supported this hypothesis, as have observational studies of breast and colorectal cancer.²⁵,²⁶ Two ecological studies found stronger inverse correlations between solar UVB doses and cancer mortality rates than incidence rates.²³,²⁴ Several reviews of the UVB-vitamin D-cancer hypothesis have also been published.²⁷,²⁸ Two randomized controlled trials found positive effects.²⁹,³⁰

The dose–response relation for vitamin D has been derived from observational studies for breast and colorectal cancer.²⁵ For the differences in population mean serum 25(OH)D concentrations for 2001–2004,¹³ the dose–response relations for breast and colorectal cancer indicate a 20–25% increase in incidence rate. The values for cancer incidence are not necessarily the same for cancer survival, but they do suggest the magnitude of the effect. This vitamin D-cancer dose–response relation might underestimate the effect of lower serum 25(OH)D concentrations

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Submitted: 12/11/11; Revised: 01/26/12; Accepted: 01/31/12
http://dx.doi.org/10.4161/derm.19667

Dermato-Endocrinology 4:2, 85–94; April/May/June 2012; © 2012 Landes Bioscience
for AAs since 20% of the black population is older than 60 y, in contrast to only 6% of whites; also, the risk of cancer increases more rapidly for changes of serum 25(OH)D concentration at lower concentrations.

A recent paper addressed vitamin D’s role in explaining some of the cancer survival disparities. Data from the Third National Health and Nutrition Examination Survey (NHANES III) were used to investigate the role of racial disparity from colorectal cancer; adding vitamin D deficiency to the model attenuated the mortality risk associated with being black by a statistically significant 40%. Grant and Peiris investigated vitamin D’s role in explaining disease disparities between AAs and WAs in general.

This paper surveys the literature on cancer disparities for AAs and WAs as well as the literature on epidemiological studies on vitamin D and cancer to see whether differences in serum 25(OH)D concentrations might explain many of the otherwise-unaccounted-for residual disparities.

### Results

Table 1 presents the findings regarding cancer survival with respect to serum 25(OH)D concentrations at the time of diagnosis. Significant inverse correlations between 25(OH)D and cancer survival were found for all-cancer, breast, colon, colorectal, lung,

| Cancer type | Conditions | Recurrence, vitamin D deficiency [HR (95% CI)] | Survival, vitamin D adequacy [HR or RR (95% CI)] | Survival, vitamin D deficiency [HR or RR (95% CI)] | Reference |
|-------------|------------|-----------------------------------------------|-------------------------------------------------|-------------------------------------------------|-----------|
| All         | Vitamin D supplementation in intention-to-treat | HR = 0.85 (0.68–1.06) (mortality)               |                                                  |                                                  | 33        |
| All         | Norway, 9.3-y follow up, high vs. low quartile | HR 0.36 (0.27–0.51) p < 0.01 CS                 |                                                  |                                                  | 34        |
| Bladder     | Three years of follow up, summer vs. winter diagnosis | RR = 1.0 (0.97–1.07) AC                          |                                                  |                                                  | 35        |
| Breast      | Three years of follow up, fall vs. winter diagnosis | 0.70 (0.65–0.75)                                 |                                                  |                                                  | 36        |
| Breast      | Three years of follow up, summer vs. winter diagnosis | RR = 0.75 (0.72–0.79) AC                         |                                                  |                                                  | 37        |
| Breast      | Women in Canada, 11.6-y follow-up,                    | HR = 1.71 (1.02–2.86) for distant recurrence;    | HR = 1.60 (0.96–2.64) AC                         |                                                  | 38        |
| Breast, luminal | 40-mo follow up,                                      | HR = 3.97 (1.77–8.91, p = 0.001)                 |                                                  |                                                  | 39        |
| Breast      | Continuous per 10 nmol/L decrement; distant disease, overall mortality | HR = 1.14 (1.05–1.24) p = 0.006                  | HR = 1.08 (1.00–1.17) p = 0.07                   |                                                  | 40        |
| Breast      | Norway, 9.3-y follow up, high vs. low quartile       | HR = 0.42 (0.21–0.82), p = 0.01 CS               |                                                  |                                                  | 34        |
| Chronic lymphocytic leukemia, chronic lymphocytic lymphoma | Mean follow-up 36 mo (1–86 mo)                   | HR = 1.47 (1.11–1.96) p = 0.008                  | HR = 1.47 (0.97–2.23) p = 0.07 AC               | 41        |
| Colon       | Three years of follow up, fall vs. winter diagnosis | 0.71 (0.66–0.77) men; 0.68 (0.64–0.72) women    |                                                  |                                                  | 35        |
| Colon       | Three-year follow up, summer vs. winter diagnosis, Midwest region, Norway | Men < 65 y, RR = 0.70 (0.5–0.83) AC Women < 65 y, RR = 0.77 (0.66–0.90) AC |                                                  |                                                  | 42        |
| Colon       | Norway, 9.3-y follow up, high vs. low quartile       | HR = 0.20 (0.01–1.10), p = 0.16 CS               |                                                  |                                                  | 34        |
| Colorectal  | Mean follow-up time 116 mo, used predicted 25(OH)D concentration | HR = 0.50 (0.26–0.95) CS                          |                                                  |                                                  | 43        |
| Hodgkin lymphoma | 18 and 36 mo follow up, based on season, autumn vs. winter diagnosis, Norway | RR = 0.78 (0.62–0.99) p = 0.04 AC                |                                                  |                                                  | 44        |
| Lung        | Three years of follow up, summer vs. winter diagnosis | RR = 1.00 (0.98–1.02) AC                         |                                                  |                                                  | 45        |
| Lung        | > 27.7 ng/ml vs. < 12.6 ng/ml                         | 1.08 (0.75–1.57) p = 0.76 AC                     |                                                  |                                                  | 46        |
prostate cancer, chronic lymphocytic leukemia/chronic lymphocytic lymphoma, Hodgkin lymphoma, and non-Hodgkin lymphoma. Studies also reported no significant correlation between serum 25(OH)D and survival for bladder, lung and ovarian cancer.

Table 2 presents the multifactor-adjusted hazard ratios for survival for AAAs vs. WAs for cancer-specific survival. Inclusion of SES, stage at diagnosis, and treatment in the analyses is indicated. Table 2 lists nearly all the relevant papers. The results for cancer-specific survival rates are a stronger indication of the effects of vitamin D than are all-cause survival rates because some of the all-cause deaths could be due to non-vitamin D-related diseases or to factors such as smoking. Statistically significant disparities emerged for cancer-specific survival rates for 13 types of cancer: bladder, breast, colon, endometrial, lung (non-small cell, stage III, IV), ovarian (advanced), pancreatic, prostate, rectal, testicular, vaginal cancer, Hodgkin lymphoma, stage II and melanoma. Our analysis also found statistically significant disparities for cancer-specific survival rates for two types of cancer, endometrial and ovarian cancer. There were no statistically significant findings for gastric adenocarcinoma, or head and neck, and oral cancer and leukemia.

Discussion

This review offers evidence to explain cancer survival differences between AAAs and WAs. AAAs’ lower serum 25(OH)D concentrations (mainly from reduced vitamin D photoproduction owing to darker pigmentation) may account for much of the unexplained survival disparity after consideration of such factors as SES, stage at diagnosis, and treatment. All cancers for which a disparity in cancer-specific survival was reported also have evidence for a beneficial role of vitamin D, as do most of those for which we found disparities for all-cause survival.

One reason ecological studies are strong include that vitamin D plays an important role in reducing risk of cancer initiation and angiogenesis around tumors and metastases.84,85 Since cancer can take years to decades to reach the stage of detection or death, continued high serum 25(OH)D concentrations over much of the lifetime is required for greatest risk reduction. Most recent ecological studies include various cancer risk-modifying factors in the analysis,20-24 Also, ecological studies include many cases, thereby reducing the uncertainty of the values. Among 45-y-old British citizens, casual solar UVB irradiance in summer increased breast cancer incidence rates are highest in spring and fall.87 The reasons for the seasonal variations given were increased production of vitamin D in summer and melatonin in winter. Breast cancer has several subtypes, and rate of progression can vary widely, with some being very rapid. For slower growing cancers, serum 25(OH)D concentrations in summer may be sufficient to retard or reverse the growth.

Once cancer reaches the point where it can be diagnosed, vitamin D improves cancer-specific survival by several mechanisms, including antiangiogenesis and antimetastases.84,85 The disparities for hematopoietic cancers may be weak or nonexistent because angiogenesis and metastases are less important for blood cell-related tumors than for solid tumors. Higher serum 25(OH)D concentrations also affect all-cause mortality rates88 since vitamin D protects against several major life-threatening conditions.
Table 2. Cancer-specific mortality rate disparities for AAs vs. WAs not explained by known factors for 25 types of cancer. Studies reported that AAs have significantly increased risk for 13 types of cancer after consideration of SES, cancer stage at time of diagnosis, and treatment.

| Cancer                 | SES | Cancer stage at diagnosis | Treatment | Relative Risk (95% CI), AAs vs. WAs | Ref. |
|------------------------|-----|---------------------------|-----------|-------------------------------------|------|
| Bladder                | Y   |                           |           | 1.68 (1.28–2.21)                    | 50   |
| Bladder, males, 1- to 2-y follow-up | Y   | Y                         |           | 1.26 (1.15–1.37)                    | 12   |
| Bladder, males, 3- to 4-y follow-up | Y   | Y                         |           | 1.16 (0.96–1.41)                    | 12   |
| Bladder, females, 1- to 2-y follow-up | Y   | Y                         |           | 1.20 (1.09–1.32)                    | 12   |
| Bladder, males, 3- to 4-y follow-up | Y   | Y                         |           | 1.55 (1.21–1.98)                    | 12   |
| Bladder                | Y   | Y                         |           | 1.73 (1.23–2.43)                    | 51   |
| Bladder                | Y   | Y                         |           | 1.29 (1.24–1.36)                    | 52   |
| Breast, meta-analysis  | Y   |                           |           | 1.23 (1.05–1.20)                    | 53   |
| Breast, meta-analysis  | Y   |                           |           | 1.22 (1.10–1.37)                    | 50   |
| Breast—premenopausal   | Y   | Y                         |           | 1.41 (1.09–1.84)                    | 10   |
| Breast—postmenopausal  | Y   | Y                         |           | 1.39 (1.17–1.66)                    | 10   |
| Breast, Stage 1, 2     | Y   | Y                         |           | 1.55 (1.13–2.13)                    | 54   |
| Breast (metastasis)    | Y   | Y                         |           | 1.20 (0.96–1.50)                    | 55   |
| Breast                 | Y   | Y                         |           | 2.41 (1.21–4.79)                    | 56   |
| Cervical               |     |                           |           | No difference                       | 57   |
| Colon, meta-analysis    | Y   |                           |           | 1.13 (1.01–1.28)                    | 58   |
| Colon                  |     |                           |           | 1.19 (1.14–1.25)                    | 59   |
| Colon                  | Y   | Y                         |           | 1.15 (1.10–1.20)                    | 59   |
| Colon                  | Y   | Y                         |           | 1.08 (1.03–1.13)                    | 59   |
| Colon, early stage     | Y   | Y                         |           | 0.99 (0.67–1.45)                    | 10   |
| Colorectal             |     |                           |           | 1.33 (1.30–1.36)                    | 60   |
| Colorectal             | Y   | Y                         |           | 1.31 (1.21–1.42)                    | 61   |
| Endometrial            | Y   |                           |           | 2.08 (1.34–3.21)                    | 50   |
| Endometrial            | Y   | Y                         |           | 1.51                               | 62   |
| Endometrial            | Y   |                           |           | 1.60 (1.51–1.69)                    | 63   |
| Esophageal             | Y   | Y                         |           | 1.02                               | 64   |
| Gastric adenocarcinoma | Y   |                           |           | 1.03 (0.95–1.12)                    | 65   |
| Gastric adenocarcinoma | Y   |                           |           | 1.18 (0.94–1.49)                    | 66   |
| Head and neck          | Y   | Y                         |           | 1.06 (0.50–2.25)                    | 67   |
| Hodgkin lymphoma, stage I | Y   | Y                         |           | 1.22 (0.81–1.85)                    | 68   |
| Hodgkin lymphoma, stage II | Y   | Y                         |           | 1.35 (1.12–1.62)                    | 68   |
| Leukemia, acute myelogenous | Y   | Y                         |           | 1.05 (0.83–1.33)                    | 10   |
| Lung cancer, small cell, limited |     |                           |           | 1.11 (0.77–1.60)                    | 10   |
| Lung cancer, NSC, advanced |     |                           |           | 0.89 (0.75–1.05)                    | 10   |
| Lung cancer, NSC, stage I, II | Y   | Y                         |           | 0.97 (0.85–1.10)                    | 69   |
| Lung cancer, NSC, stage III, IV | Y   | Y                         |           | 1.24 (1.01–1.53)                    | 69   |
| Melanoma               | Y   | Y                         |           | HR, 1.60 (1.17–2.18)                | 9    |
| Multiple myeloma       | Y   | Y                         |           | 0.85 (0.70–1.03)                    | 10   |
| Nasopharyngeal         |     |                           |           | 1.00 (0.82, 1.24)                   | 70   |
| Non-Hodgkin’s lymphoma (NHL) | Y   | Y                         |           | 1.07 (0.92–1.25)                    | 71   |
| NHL, advanced          | Y   | Y                         |           | 1.17 (0.94–1.45)                    | 10   |
| Oral                   | Y   | Y                         |           | 1.1 (0.9–1.4)                       | 72   |
| Ovarian, advanced      | Y   | Y                         |           | 1.41 (1.03–2.11)                    | 10   |
| Ovarian, stage 3       | Y   | Y                         |           | 1.06 (0.61–1.79)                    | 73   |
| Ovarian, 1973–2007     | Y   | Y                         |           | 1.14 (1.07–1.21)                    | 74   |
Secondary hyperparathyroidism due to osteoblastic metastases and hungry bone syndrome has been described with advanced prostate and breast cancer, it is likely that a vitamin D replete state may minimize such occurrences. Bisphosphonates are commonly used in oncology. Pamidronate administration improved the secondary hyperparathyroidism due to “bone hunger syndrome” in a patient with osteoblastic metastases from prostate cancer. Coleman and McCloskey suggest that bisphosphonates may prevent metastases and reduce the risk of disease recurrence. Based on animal data, a vitamin D replete state may be helpful in reducing bisphosphonate induced osteonecrosis of the jaw.

Factors other than SES, stage at diagnosis, treatment, and vitamin D status might also explain the cancer survival disparities. For example, the lack of survival disparities for lung cancer may be due to a stronger effect from smoking than from vitamin D. Smoking cessation improves lung cancer survival rates associated with early-stage lung cancer.

Obesity is significantly correlated with cancer risk for nearly all types of cancer listed in Tables 1 and 2. AAs tend to have higher body mass index than WAs. One reason is that obesity is linked to poverty in the United States because of energy-dense but nutrient-poor foods are cheaper due to subsidies. A second reason is that AAs have about twice the prevalence of apolipoprotein E ε4 (ApoE4) than WAs. ApoE4 increases production of cholesterol in the liver and of insulin in the pancreas to store excess food as fat for those with sporadic food supplies, such as hunter-gatherers. Interestingly, overweight and obesity rates for white and black men differ little, whereas AA women are much heavier than WA women. Thus, obesity does not seem to be a likely explanation for cancer disparities among men but could be for women. On the other hand, serum 25(OH)D concentrations are inversely correlated with body mass index, which has implications for cancer risk. Interestingly, for pancreatic cancer incidence, higher body mass index was significantly associated with risk for AA and WA men and WA women but with only insignificantly reduced risk for AA women.

Cancer survival studies with respect to serum 25(OH)D concentrations at time of diagnosis offer strong evidence for a beneficial effect of vitamin D. All cancers with a beneficial effect of vitamin D on survival have been found inversely correlated with solar UVB doses, with the possible exception of chronic lymphocytic leukemia. There are also studies from Norway indicating improved survival for those diagnosed with breast, colon, prostate cancer and Hodgkin lymphoma in summer compared with winter.

The UVB-vitamin D-cancer hypothesis receives its strongest support from ecological studies. Observational studies also provide good support if the various studies are examined carefully and a good reason is found for why many observational studies have not found a beneficial effect of vitamin D in reducing the risk of cancer. Nested case-control studies have a reduced strength since only a single serum 25(OH)D concentration measurement or oral intake assessment is made at time of enrollment, with follow-up periods lasting between 3 and 28 y. As the follow-up time increases beyond about 3–7 y, the single measurement is less meaningful. Case-control studies, on the other hand, use serum 25(OH)D concentration or vitamin D oral intake values at the time of diagnosis. A review of observational studies of breast and colorectal cancer incidence with respect to serum 25(OH)D concentration found statistically significant inverse correlations for breast cancer out to 3 y and for colorectal cancer out to 12 y of...
follow-up. Thus, the recently reported results from the Vitamin D Pooling Project study of rarer cancer types (endometrial, esophageal, gastric, ovarian, pancreatic, and renal cancer and non-Hodgkin lymphoma) probably failed to find an inverse correlation between incidence of these cancers and prediagnostic serum 25(OH)D concentrations because the mean follow-up period was 6.63 y and because there were so few cases that the 95% confidence intervals were about 50%. The correlation between serum 25(OH)D concentrations measured at different times decreases with time, dropping to a regression coefficient of 0.40 after 14 y.

Several ways exist to test the UVB-vitamin D-cancer hypothesis as an additional contributing factor for cancer survival disparities. One would be to measure serum 25(OH)D concentrations of newly diagnosed cancer patients and at several intervals during the course of the cancer. Another would be to supplement newly diagnosed cancer patients with sufficient vitamin D to bring serum 25(OH)D concentrations up to 40–80 ng/ml and compare results for those not supplemented, perhaps from previous patients in the same practice. A recent publication described the rationale for vitamin D supplementation, which is being done in some cancer treatment centers.

Increasing serum 25(OH)D concentrations would also reduce the risk of severe sepsis associated with cancer surgery as well as many other comorbid diseases.

Study caveats. We acknowledge that while it appears very likely that vitamin D is an important and often ignored factor in the biology of cancer, the issue of cancer etiology is complex and is clearly multifactorial. Moreover, outcomes studies may have skewed results since AA men are less likely to participate in cancer screening trials. Black women may be less physically active. An inverse relationship between physical activity and breast cancer in AA women has been reported. Some of the adverse cancer outcomes may relate to less than optimal care. Esnaola et al. reported that AA patients are less likely to receive resection in non-metastatic rectal cancer. Rolnick et al. demonstrated that AA colorectal cancer survivors are less likely to receive post-treatment colorectal surveillance. Similar findings have been found in prostate cancer. These may not necessarily reflect racism in that physicians may make recommendations based on a patient’s access to health care, presence of insurance, etc. In addition, poor health literacy in AA women may also impact access to available health care strategies.

Cultural differences may also play a role with cultural insensitivity among providers compounding the issue. Margolis et al. demonstrated significant racial differences in belief prior to lung cancer surgery. Some of these differences result in refusal of surgery on the part of AA patients. AAs have less trust in their health providers and may not accept physicians’ assertions regarding treatment. Spiritually based health interventions may be more effective in AAs. Van Ness et al. indicates that lack of religiousness maybe associated with poor cancer survival in AA women. Church attendance may be associated with greater emotional and social support, which is linked to better outcomes in breast cancer.

We must also consider the possibility that apart from direct cellular benefits of vitamin D on cancer that vitamin D deficiency has indirect effects which are hard to quantify but may have a significant impact on cancer outcomes. Vitamin D deficiency is also associated with a higher prevalence of depression and neurocognitive symptoms, which makes patients intrinsically less likely to seek medical attention. Treating vitamin D deficiency may ameliorate symptoms of depression.

Some risk factors such as diet can be modified and increased consumption of vegetables may decrease the risk of breast cancer in AAs, possibly by altering estrogen/progesterone receptor status. Fortunately, it does appear that tumors are not intrinsically more aggressive in AAs. In Veterans with equal access to health care, lung and colon cancer are not necessarily more aggressive diseases in AAs. Dignam reported that black women, diagnosed at comparable disease stage as white women and treated appropriately, tend to experience similar breast cancer prognoses and survival.

Some of the residual disparity for prostate cancer may be due to the higher prevalence of the ApoE4 allele among AAs than WAs, which is related to increased cholesterol production. Cholesterol is an important risk factor for high-grade prostate cancer. Increased low-density lipoprotein concentrations increased the risk of prostate cancer for AAs but not WAs.

Conclusion

Lower serum 25(OH)D concentrations among AAs than WAs may explain many of the cancer survival disparities after consideration of SES, stage at time of diagnosis, and treatment. More research is required to confirm this hypothesis. If substantially correct, programs to increase serum 25(OH)D concentrations among AAs could reduce the cancer disparities. This approach would work not only for those of the ages where cancer is more likely but also for those younger. Vitamin D can reduce the risk of cancer at the initiation stage and in the advanced stages, as well as raising serum 25(OH)D concentrations to over 40 ng/ml shortly after cancer diagnosis. Given the biologic plausibility, the currently available evidence of beneficence, and the lack of harm with moderate Vitamin D replacement, we recommend Oncologists consider a more proactive stance on this issue pending additional studies.

Materials and Methods

Being a review, this analysis summarizes papers in the journal literature. Papers cited in this study came from the National Library of Medicine’s PubMed database (http://www.pubmed.gov). As of December 28, 2011, a search for cancer disparity papers (search terms “cancer disparities survival, African-American”) identified 457 articles. We reviewed the titles and abstracts of many of these. We examined in more detail those reporting hazard ratios for AAs vs. WAs for survival. The tables in this review do not include the component papers of meta-analyses cited. Thus, the papers listed in the tables are representative rather than exhaustive. We inspected the results in the papers, preferring those with disparities in survival that included all three factors—SES, stage at diagnosis, and treatment,
in addition to race—over those including fewer or none of these factors. We examined the papers to see whether survival was cancer-specific or all-cause.

In addition, papers reporting cancer survival with respect to serum 25(OH)D concentration were also sought.

Disclosure of Potential Conflicts of Interest

W.B.G. receives funding from the UV Foundation (McLean, VA), Bio-Tech Pharmacal (Fayetteville, AR), the Vitamin D Council (San Luis Obispo, CA), and the Vitamin D Society (Canada).

References

1. Byers TE, Wolf HJ, Bauer KR, Bolick-Aldrich S, Chen VW, Finch JL, et al. Patterns of Care Study Group. The impact of socioeconomic status on survival after cancer in the United States: findings from the National Program of Cancer Registries Patterns of Care Study. Cancer 2008; 113:582-91; PMID:18613122; http://dx.doi.org/10.1002/cncr.23567

2. Brawley OW. Is race really a negative prognostic factor for cancer? J Natl Cancer Inst 2009; 101:970-1; PMID:19567421; http://dx.doi.org/10.1093/jnci/djp185

3. Siegel JA, Cinar P, Mobasher M, Ziogas A, Meyskens FL, Jr. A social determinants of health approach to reduce health inequities. Public Health Rep 2010; 125(Suppl 4):6-7; PMID:20629251

4. DeSantis C, Jemal A, Ward E. Disparities in breast cancer prognostic factors by race, insurance status, and education. Cancer Causes Control 2010; 21:1445-50; PMID:20506039; http://dx.doi.org/10.1007/s10552-010-9757-z

5. Gerend MA, Pai M. Social determinants of Black-white disparities in breast cancer mortality. Cancer Causes Control 2010; 21:121; PMID:20507436; http://dx.doi.org/10.1007/s10552-010-9752-1

6. Satcher D. Include a social determinants of health approach when calculating survival. Am J Public Health 2010; 101:984-92; PMID:19584328; http://dx.doi.org/10.1158/0005-8160-JCO.2010.7255

7. Grant WB, Holick MF. Benefits and requirements of vitamin D for optimal health: a review. Altern Med Rev 2005; 10:94-111; PMID:15989370

8. Zell JA, Cinar P, Mobasher M, Ziogas A, Meyskens FL, Jr. A social determinants of health approach to reduce health inequities. Public Health Rep 2010; 125(Suppl 4):6-7; PMID:20629251

9. Zell JA, Cinar P, Mobasher M, Ziogas A, Meyskens FL, Jr., Anton-Culver H. Survival for patients with invasive cutaneous melanoma among ethnic groups: the effects of socioeconomic status and treatment. J Clin Oncol 2008; 26:66-75; PMID:18165642; http://dx.doi.org/10.1200/JCO.2007.12.3004

10. Albain KS, Unger JM, Crowley JJ, Coltman CA, Jr., Hershman DL. Racial disparities in cancer survival: a reanalysis of the Women’s Health Initiative (WHI) limited-access data set. J Natl Cancer Inst 2011; 103:1186-92; PMID:21029996; http://dx.doi.org/10.1093/jnci/djr280

11. Arnold LD, Patel AV, Yan Y, Jacobs EJ, Thun MJ, Calle EE, et al. Are racial disparities in pancreatic cancer explained by smoking and overweight/obesity? Cancer Epidemiol Biomarkers Prev 2008; 17:2913-23; PMID:19899731; http://dx.doi.org/10.1158/1055-9965.EPI-07-0633

12. Zell JA, Cinar P, Mobasher M, Ziogas A, Meyskens FL, Jr., Anton-Culver H. Survival for patients with invasive cutaneous melanoma among ethnic groups: the effects of socioeconomic status and treatment. J Clin Oncol 2008; 26:66-75; PMID:18165642; http://dx.doi.org/10.1200/JCO.2007.12.3004

13. Ginde AA, Liu MC, Camargo CA, Jr. Demographic differences and trends of vitamin D insufficiency in the US population, 1988-2004. Arch Intern Med 2009; 169:626-32; PMID:19970572; http://dx.doi.org/10.1001/archinternmed.2008.604

14. Egan KM, Signorelli LR, Munro HM, Hargreaves MK, Hollis BW, Blot WJ. Vitamin D insufficiency among African-Americans in the southeastern United States: implications for cancer latency and mortality. Cancer Causes Control 2008; 19:527-35; PMID:18219582; http://dx.doi.org/10.1007/s10552-008-9115-z

15. Ng K, Sargent DJ, Goldberg RM, Meyerhardt JA, Green EM, Pinot HC, et al. Vitamin D status in patients with stage IV colorectal cancer: findings from Intergroup trial N9741. J Clin Oncol 2011; 29:1599-606; PMID:21422438; http://dx.doi.org/10.1200/JCO.2010.31.7255

16. Grant WB, Holick MF. Benefits and requirements of vitamin D for optimal health: a review. Altern Med Rev 2005; 10:94-111; PMID:15989370

17. Harris SS. Vitamin D and African Americans. J Nutr 2006; 136:1162-9; PMID:16549493

18. Nesby-O'Dell S, Scanlon KS, Gogweil ME, Gillespie C, Hollis BW, Looker AC, et al. Hypervitaminosis D prevalence and determinants among African American and white women of reproductive age: third National Health and Nutrition Examination Survey, 1988-1994. Am J Clin Nutr 2002; 76:187-92; PMID:12081833

19. Garland CF, Garland FC. Do sunlight and vitamin D reduce the likelihood of colon cancer? Int J Epidemiol 1989; 9:227-31; PMID:7440046; http://dx.doi.org/10.1093/ije/9.3.227

20. Grant WB, Garland CF. The association of solar ultraviolet B (UVB) with reducing risk of cancer: multifactorial ecologic analysis of geographic variation in age-adjusted cancer mortality rates. Anticancer Res 2006; 26:44A:2687-99; PMID:16886679

21. Grant WB. Lower vitamin-D production from solar ultraviolet B and vitamin D reduce the risk of cancer?: results of a randomized trial. Am J Clin Nutr 2007; 85:1568-91; PMID:17566907

22. Bolland MJ, Grey A, Gamble GD, Reid IR. Calcium and vitamin D supplements and health outcomes: a reanalysis of the Women’s Health Initiative (WHI) limited-access data set. Am J Clin Nutr 2011; 94:1144-9; PMID:21880848; http://dx.doi.org/10.3945/ajcn.110.810530

23. Fiscella K, Winters P, Tancerdi C, Hendren S, Franks P. Racial disparity in death from colorectal cancer: does vitamin D deficiency contribute? Cancer 2011; 117: 1061-9; PMID:20945439; http://dx.doi.org/10.1002/1097-0256/crd.20647

24. Grant WB, Peiris AN. Possible role of serum 25-hydroxyvitamin D in black-white health disparities in the United States. J Am Med Dir Assoc 2010; 11:617-28; PMID:20129996; http://dx.doi.org/10.1016/j.jamda.2010.03.013

25. Avnell A, Maclennan GG, Jenkinson DJ, McPherson GC, McDonald AM, Pant PR, et al. the RECORD Trial Group. Long-Term Follow-Up for Mortality and Cancer in a Randomized Placebo-Controlled Trial of Vitamin D3 and/or Calcium (RECORD Trial). J Clin Endocrinol Metab 2012; 97:614-22; PMID:22128804; http://dx.doi.org/10.1210/jc.2011-1309

26. Tretti S, Schwartz GG, Tønjesen PA, Robshaim TE. Serum levels of 25-hydroxyvitamin D and survival in Norwegian patients with cancer of breast, colon, lung, and lymphoma: a population-based study. Cancer Causes Control 2012; 23:363-70; PMID:22193937; http://dx.doi.org/10.1007/s10552-011-9885-6

27. Pompomucci AC, Dahlback A, Moan J. Sun exposure and cancer survival in Norway: changes in the risk of death with season of diagnosis and latitude. Adv Exp Med Biol 2008; 624:43-54; PMID:18348446; http://dx.doi.org/10.1007/978-0-387-77574-6_4

28. Robshaim TE, Tretti S, Dahlback A, Moan J. Vitamin D3 from sunlight may improve the prognosis of breast-, colon- and prostate cancer (Norway). Cancer Causes Control 2004; 15:1549-58; PMID:15017127; http://dx.doi.org/10.1023/B:CAOC.0000019494.34403.03

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A.N.P. acknowledges use of resources at Mountain Home VAMC. The services of the Library staff at Mountain Home VA and affiliated sites are gratefully acknowledged. This paper does not represent the position of the Department of Veterans Affairs or the US government.
Poroznicu AC, Lagouna Z, Roebsham TE, Berg JP, Dalhback A, Moan J. Changes in risk of death from breast cancer with season and latitude: sun exposure and breast cancer survival in Norway. Breast Cancer Res Treat 2007; 102:325-8; PMID:17028983; http://dx.doi.org/10.1007/s10549-006-9331-8

Goodwin PJ, Ennis M, Pritchard KL, Koo J, Hood N. Prognostic effects of 25-hydroxyvitamin D levels in early breast cancer. J Clin Oncol 2009; 27:3755-60; PMID:19451439; http://dx.doi.org/10.1200/JCO.2008.07.2072

Kim HJ, Lee YM, Ko BS, Lee JW, Yu JH, Son BH, et al. Vitamin D deficiency is correlated with poor outcomes in patients with luminal-type breast cancer. Ann Surg Oncol 2011; 18:1830-6; PMID:21573699; http://dx.doi.org/10.1245/s10434-010-1465-6

Yteland A, Hein R, Abbas S, Schneeweiss A, Flesch-Jansy D, Chang-Claude J. Serum 25-hydroxyvitamin D and postmenopausal breast cancer survival: a prospective patient cohort study. Breast Cancer Res 2011; 13:R74; PMID:21791949; http://dx.doi.org/10.1186/bcr2920

Shanafelt TD, Drake MT, Maurer MJ, Allmer C, Rabe J, et al. Vitamin D status and survival in patients with hematologic malignancies. J Clin Oncol 2009; 27:3755-60; PMID:19451439; http://dx.doi.org/10.1200/JCO.2008.07.2072

Hollenbeck BK, Dunn RL, Ye Z, Hollingsworth JM, Lee CT, Birkmeyer JD. Racial differences in treatment and outcomes among patients with early-stage bladder cancer. Cancer 2010; 116:560-6; PMID:19877112

Yee DS, Ishill NM, Lowrance WT, Herr HW, Elkin EB. Ethnic differences in bladder cancer survival. Urology 2011; 78:544-5; PMID:21782222; http://dx.doi.org/10.1016/j.urology.2011.02.023

Newman LA, Griffith KA, Jatoi I, Simon MS, Crowe JP, Colditz GA. Meta-analysis of survival in African American and white American patients with breast cancer: ethnicity compared with socioeconomic status. J Clin Oncol 2006; 24:1342-9; PMID:16549828; http://dx.doi.org/10.1200/JCO.2005.03.3472

Betz JP, Johnston K, Backus B, Dottos G, Rose AJ, Pierre S, et al. The influence of black race on treatment and mortality for early-stage breast cancer. Med Care 2009; 47:969-92; PMID:19648837

Schoolmard M, Jeffer DB, Gillanders WE, AF R. Racial disparities in the development of breast cancer metastases among older women: a multilevel study. Cancer 2009; 115:731-40; PMID:19130463; http://dx.doi.org/10.1002/cncr.234087

Adams SA, Barber LA, Spangenberg HE, Spangenberg EM, Delage AF, et al. Racial disparities in breast cancer mortality in a multiethnic cohort in the Southeast. Cancer 2011; 117:2553-61; PMID:21288370; http://dx.doi.org/10.1002/cna.23670

Fedele C, Allison J, Dola C, Tafi S, Galandak J, Jacob C, et al. Relationship among age, race, medical funding, and cervical cancer survival. J Natl Med Assoc 2010; 102:199-205; PMID:20355349

Du XL, Meyer TE, Franke L, Zinn P, Zell JA. Survival of distinct Asian groups among colorectal cancer patients in California. Cancer 2007; 109:2161-70; PMID:17455219; http://dx.doi.org/10.1002/cncr.222664

Le H, Ziegas A, Taylor TH, Lipkin SM, Zell JA. Survival of distinct Asian groups among colorectal cancer patients in California. Cancer 2007; 109:2161-70; PMID:17455219; http://dx.doi.org/10.1002/cncr.222664

Soneji S, Iyer SS, Armstrong K, Aitch DA. Racial disparities in stage-specific colorectal cancer mortality: 1990-2005. Am J Public Health 2010; 100:1912-6; PMID:20724684; http://dx.doi.org/10.2105/aje.2009.141929

White A, Vernon SW, Frankau L, Du XL. Racial disparities in colorectal cancer survival: to what extent are racial disparities explained by differences in treatment, tumor characteristics, and hospital characteristics? Cancer 2011; 116:2432-31; PMID:20626015; http://dx.doi.org/10.1002/cncr.23593

Randall TC, Armstrong K. Differences in treatment and outcome between African-American and white women with endometrial cancer. J Clin Oncol 2003; 21:4200-6; PMID:14615448; http://dx.doi.org/10.1200/JCO.2003.01.218

Wright JD, Fiorelli J, Schiff PB, Burke WM, Kansler GP, Colditz GA. Meta-analysis of survival in African American and white women with colorectal cancer: a population-based cohort of elderly patients with non-Hodgkin lymphoma. Cancer 2008; 113:3231-41; PMID:18937267; http://dx.doi.org/10.1002/cncr.23914

Arbogast PK, Olshan AF, Clapan DJ, Schoenbach VJ, Slade GD, Symons MJ. Factors contributing to the poorer survival of black Americans diagnosed with oral cancer (United States). Cancer Causes Control 1999; 10:513-23; PMID:10661821; http://dx.doi.org/10.1021/acs.jradiol.1030100

Bristow RE, Uela S, Gerardi MA, Aji Boye OB, Iheama OA. Analysis of racial disparities in stage IIIB epithelial ovarian cancer care and outcomes in a tertiary gynecologic oncology referral center. Gynecol Oncol 2011; 122:319-23; PMID:21632009; http://dx.doi.org/10.1016/j.ygyno.2011.04.047

Terplan M, Schultenner N, McNamara EJ, Tracey LJ, Temkin SM. Have racial disparities in ovarian cancer increased over time? An analysis of SEER data. Gynecol Oncol 2011; 122:319-23; PMID:21632009; http://dx.doi.org/10.1016/j.ygyno.2011.04.047

Eloubeidi MA, Desmond RA, Wilcox CM, Wilson RJ, Manchulalapati P, Fouad MM, et al. Prognostic factors for survival in pancreatic cancer: a population-based study. Ann Surg 2006; 243:322-9; PMID:16920462; http://dx.doi.org/10.1097/01.aso.0000216328.03231.0f

Zella JA, Rhce JM, Ziegas A, Lipkin SM, Antion-Culver H. Race, socioeconomic status, stage, and survival time among pancreatic cancer cases in California. Cancer Epidemiol Biomarkers Prev 2007; 16:5465-52; PMID:17372250; http://dx.doi.org/10.1158/1055-9965.EPI-06-0893

Evans S, Mercalle C, Ibrahim F, Persad R, Ben-Shlomo Y. Investigating Black-white differences in prostate cancer prognosis: A systematic review and meta-analysis. Int J Cancer 2008; 123:430-5; PMID:18452170; http://dx.doi.org/10.1002/ijc.23500

Cheng L, Wirtz JS, McClure LA, Shema SJ, Cockburn MC, John EM, et al. Socioeconomic status and prostate cancer incidence and mortality rates among the diverse population of California. Cancer Causes Control 2009; 20:1451-40; PMID:19562519; http://dx.doi.org/10.1007/s10555-009-9360-0

66. Stessin AM, Sherr DL. Demographic disparities in patterns of care and survival outcomes for patients with resected gastric adenocarcinoma. Cancer Epidemiol Biomarkers Prev 2011; 20:223-33; PMID:21200617; http://dx.doi.org/10.1158/1055-9966.EPI-10-0158

67. Chen LM, Li G, Reitzel LR, Pytnia KB, Zafereo ME, Wei Q, et al. Matched-pair analysis of race or ethnicity in outcomes of head and neck cancer patients receiving similar multidisciplinary care. [Phila Pa]. Cancer Prev Res (Phila) 2009; 2:782-91; PMID:19737985; http://dx.doi.org/10.1158/1940-6207.CAPR-09-0154
92. Berruti A, Sperone P, Fasolis G, Torta M, Fontana D, Anderson JL, May HT, Horne BD, Bair TL, Hall NL, Grant WB. An estimate of the global reduction in prostate cancer. Prostate 1997; 33:252-5; PMID:9937197; http://dx.doi.org/10.1002/(SICI)1097-0045(19971201)33:4<252::AID-JPRO535-3.0.CO;2-O

93. Coleman RE, McClurey EV. Bisphosphonates in oncology. Bone 2011; 49:71-6; PMID:21320652; http://dx.doi.org/10.1016/j.bone.2011.02.003

94. Hokago A, Christensen R, Chung EM, Sung EC, Felsenfeld AL, Sayre JW, et al. Increased prevalence of bisphosphonate-related osteonecrosis of the jaw with vitamin D deficiency in rats. J Bone Miner Res 2010; 25:1337-49; PMID:20209058; http://dx.doi.org/10.1002/jbmr.23

95. Parsons A, Daley A, Beph R, Ayeday P. Influence of smoking cessation after diagnosis of early stage lung cancer on prognosis: systematic review of observational studies with meta-analysis. BMJ 2010; 340:b5569; http://dx.doi.org/10.1136/bmj.b5569

96. Lichtman MA. Obesity and the risk for a hematological malignancy: leukemia, lymphoma, or myeloma. Oncologist 2010; 15:1083-101; PMID:20930095; http://dx.doi.org/10.1634/theclinologist.2010-0206

97. Murthy NS, Mukherjee S, Ray G, Ray A. Dietary factors and cancer chemoprevention: an overview of obesity-related malignancies. J Postgrad Med 2009; 45:55-54; PMID:19242081; http://dx.doi.org/10.4103/0022-3859.47551

98. Drewnowski A. Obesity, diets, and social inequalities. Nut Rev 2009; 67(Suppl 1):S36; PMID:19453676; http://dx.doi.org/10.1038/3487.2009.00517.x

99. Grant WB. A multicountry ecological study of risk-modifying factors for prostate cancer: apolipoprotein E ε4 as a risk factor and cereals as a risk reduction factor. Anticancer Res 2010; 30:189-99; PMID:20150635

100. Laganova Z, Porojnicu AC, Grant WB, Brunold O, Moan JE. Obesity and increased risk of cancer: does decrease of serum 25-hydroxyvitamin D level with increasing body mass index explain some of the association? Mol Nutr Food Res 2010; 54:1127-33; PMID:20512788

101. Grant WB. Effect of interval between serum draw and time of measurement on relative risk of cancer incidence. J Nutr 2010; 140:333-40; PMID:20166967; http://dx.doi.org/10.3944/jn.2010-0284

102. Lim U, Freedman DM, Hollis BW, Horst RL, Purdue MP, Carter RA, et al. A prospective investigation of serum 25-hydroxyvitamin D and risk of lymphoid cancers. Int J Cancer 2010; 129:974-86; PMID:19935445; http://dx.doi.org/10.1002/ijc.23984

103. Fielder KJ, VDPF Steering Committee. Overview of the Cohort Consortium Vitamin D Pooling Project of Rarer Cancers. Am J Epidemiol 2010; 173:418-27; PMID:20562193; http://dx.doi.org/10.1093/aje/kwq119

104. Jorde R, Sneve M, Hutchinson M, Emaus N, Eggeveen S, Grimnes G. Tracking of serum 25-hydroxyvitamin D levels during 14 years in a population-based study and during 12 months in an intervention study. Am J Epidemiol 2010; 171:903-8; PMID:20297663; http://dx.doi.org/10.1093/aje/kwq005

105. Grant WB. Benefits of vitamin D in reducing the risk of cancer: Time to include vitamin D in cancer treatment. J Soc Integr Oncol 2010; 8:88-1

106. Vash PG, Trukova K, Lammersfeld CA, Braun DP, Gupta D. Impact of oral vitamin D supplementation on serum 25-hydroxyvitamin D levels in oncology. Nutr J 2010; 9:60; PMID:20192237; http://dx.doi.org/10.1186/1475-2891-9-60

107. Peppone LJ, Histon AJ, Reid ME, Rosier RN, Zdaharla Y, Trump DL, et al. The effect of various vitamin D supplementation regimens in breast cancer patients. Breast Cancer Res Treat 2011; 127:171-7; PMID:21388416; http://dx.doi.org/10.1007/s10549-011-1415-4

108. Jeng L, Yamschikov AV, Judd SE, Blumberg HM, Martin GS, Ziegler TR, et al. Aberrations in vitamin D status and anti-microbial peptide levels in patients in the intensive care unit with sepsis. J Transl Med 2009; 7:28; PMID:19389235; http://dx.doi.org/10.1186/1475-2876-7-28

109. Ford ME, Havstad SL, Davis SD. A randomized trial of recruitment methods for older African-American patients in the Prostate, Lung, Colorectal and Ovarian (PLCO) Cancer Screening Trial. Clin Trials 2004; 1:343-51; PMID:16279272; http://dx.doi.org/10.1117/11704775407406293

110. Smith AW, Allan GE, Reeve BB, Irwin ML, Bernstein L, Baumgartner K, et al. Race/ethnicity, physical activity, and quality of life in breast cancer survivors. Cancer Epidemiol Biomarkers Prev 2009; 18:556-63; PMID:19190157; http://dx.doi.org/10.1158/1055-9966.EPI-08-0352

111. Bernstein L, Patel AV, Unsin G, Sullivan-Halley J, Press MF, Deapen D, et al. Lifetime recreational exercise activity and breast cancer risk among black women and white women. J Natl Cancer Inst 2005; 97:1671-9; PMID:16288120; http://dx.doi.org/10.1093/jnci/dji374

112. Esnaola NF, Stewart AK, Feig BW, Skibber JM, Rodriguez-Bigas MA. Age-, race-, and ethnicity-related differences in the treatment of nonmetastatic rectal cancer: a patterns of care study from the national cancer data base. Ann Surg Oncol 2008; 15:306-47; PMID:18712449; http://dx.doi.org/10.1245/s10434-008-0106-9

113. Rolnick S, Henley Alfred S, Kaucer GP, Forman K, Ulickas Yood M, Jankowski M, et al. Racial and age differences in colon examination surveillance follow-up: a diagnosis of colorectal cancer. J Natl Cancer Inst Monogr 2005; 35:96-101; PMID:16287893; http://dx.doi.org/10.1093/jncimonographs/lgi045

114. Scapira MM, McAuliffe TL, Nuttige AB. Treatment of localized prostate cancer in African-American compared with Caucasian men. Less use of aggressive therapy for comparable disease. Med Care 1995; 33:1079-88; PMID:7475418; http://dx.doi.org/10.1097/00005650-199511000-00002

115. OMalley MS, Earp JA, Hawley ST, Schell MJ, Mathews HF, Mitchell J. The association of race/ ethnicity, socioeconomic status, and physician recommendation for mammography: who gets the message about breast cancer screening? Am J Public Health 2001; 91:49-54; PMID:11189825; http://dx.doi.org/10.2105/AJPH.91.1.49

116. Sharp LK, Zurawski JM, Roland PY, OToole C, Hines J. Health literacy, cervical cancer risk factors, and distress in low-income African-American women seeking colposcopy. Ethn Dis 2002; 12:541-6; PMID:12477141

117. Margolis ML, Christie JD, Silvestri GA, Kaiser L, Santiago S, Hansen-Flaschen J. Racial differences pertaining to a belief about lung cancer surgery: results of a multicenter survey. Ann Intern Med 2003; 138:538-63; PMID:14530226

118. Holt CL, Wynn TA, Litsker MS, Southward P, Jeannes S, Schulz E. A comparison of a spiritually based and non-spiritually based educational intervention for informed decision making for prostate cancer screening among church-attending African-American men. Urol Nurs 2009; 29:249-58; PMID:19718941

119. Van Noss PH, Kauf SL, Jones BA. Religion, race, and breast cancer survival. Int J Psychiary Med 2003; 33:557-75; PMID:15152786; http://dx.doi.org/10.2190/LEXP-6CCR-G728-5W9H
120. Jorde R, Sneve M, Fugenschau Y, Svartberg J, Waterkoo K. Effects of vitamin D supplementation on symptoms of depression in overweight and obese subjects: randomized double blind trial. J Intern Med 2008; 264:599-609; PMID:18793245; http://dx.doi.org/10.1111/j.1365-2796.2008.02008.x

121. Boggs DA, Palmer JR, Wise LA, Spiegelman D, Stampfer MJ, Adams-Campbell LL, et al. Fruit and vegetable intake in relation to risk of breast cancer in the Black Women’s Health Study. Am J Epidemiol 2010; 172:1268-79; PMID:20937636; http://dx.doi.org/10.1093/aje/kwq293

122. McLeod DG, Schellhammer PF, Vogelzang NJ, Soloway MS, Sharifi R, Block NL, et al. The Casodex Combination Study Group. Exploratory analysis on the effect of race on clinical outcome in patients with advanced prostate cancer receiving bicalutamide or flutamide, each in combination with LHRH analogues. Prostate 1999; 40:218-24; PMID:10420149; http://dx.doi.org/10.1002/(SICI)1097-0045(19990901)40:4<218:AID-PROS2>3.0.CO;2-6

123. Akerley WL, 3rd, Moritz TE, Ryan LS, Henderson WG, Zacharski LR. Racial comparison of outcomes of male Department of Veterans Affairs patients with lung and colon cancer. Arch Intern Med 1993; 153: 1681-8; PMID:8333805; http://dx.doi.org/10.1001/archinte.1993.00410140063008

124. Dignam JJ. Differences in breast cancer prognosis among African-American and Caucasian women. CA Cancer J Clin 2000; 50:50-64; PMID:10735015; http://dx.doi.org/10.3322/canjclin.50.1.50

125. Mondul AM, Clipp SL, Helzlsouer KJ, Platz EA. Association between plasma total cholesterol concentration and incident prostate cancer in the CLUE II cohort. Cancer Causes Control 2010; 21:61-8; PMID:19806465; http://dx.doi.org/10.1007/s10552-009-9434-8

126. Moses KA, Abd TT, Goodman M, Hsiao W, Hall JA, Marshall FF, et al. Increased low density lipoprotein and increased likelihood of positive prostate biopsy in black americans. J Urol 2009; 182:2219-25; PMID:19758611; http://dx.doi.org/10.1016/j.juro.2009.07.039