

NIH Public Access

Author Manuscript

Cancer Causes Control. Author manuscript; available in PMC 2010 November 22

Published in final edited form as:

Cancer Causes Control. 2009 November ; 20(9): 1623-1634. doi:10.1007/s10552-009-9409-9.

CORRELATES OF SEXUALLY TRANSMITTED INFECTION HISTORIES IN A COHORT OF AMERICAN MALE HEALTH PROFESSIONALS

Siobhan Sutcliffe¹, Ichiro Kawachi², John F. Alderete³, Charlotte A. Gaydos⁴, Lisa P. Jacobson⁵, Frank J. Jenkins⁶, Raphael P. Viscidi⁷, Jonathan M. Zenilman⁴, and Elizabeth A. Platz^{5,8}

¹ Alvin J. Siteman Cancer Center and the Department of Surgery, Washington University School of Medicine, St. Louis, MO

² Department of Society, Human Development and Health, Harvard School of Public Health, Boston, MA

³ School of Molecular Biosciences, Fulmer Hall, Washington State University, Pullman, WA

⁴ Division of Infectious Diseases, Department of Medicine, Johns Hopkins Medical Institutions, Baltimore, MD

⁵ Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD

⁶ Department of Pathology, School of Medicine, and Departments of Infectious Diseases and Microbiology, School of Public Health, University of Pittsburgh, Pittsburgh, PA

⁷ Stanley Division of Developmental Neurovirology, Department of Pediatrics, Johns Hopkins Medical Institutions, Baltimore, MD

⁸ James Buchanan Brady Urological Institute and the Sidney Kimmel Comprehensive Cancer Center, Johns Hopkins Medical Institutions, Baltimore, MD

Abstract

Objective—Several epidemiologic studies have investigated sexually transmitted infections (STIs) and later risk of genitourinary conditions with suggestive positive results. While these results may reflect causal associations, other possible explanations include confounding by factors possibly related to both STI acquisition and genitourinary condition risk, such as recognized STI risk factors/ correlates, and other factors not typically considered in relation to STIs (e.g., general health-related behaviors or markers of such behaviors). Very few of these factors have been investigated in older populations in which STIs and genitourinary conditions are typically studied. Therefore, we investigated STI history correlates in one such population, the Health Professionals Follow-up Study.

Methods—We ascertained histories of potential correlates, and gonorrhea and syphilis by questionnaire (n=36,032), and performed serologic testing for *Chlamydia trachomatis*, *Trichomonas vaginalis*, human papillomavirus, and human herpesvirus type 8 infection in a subset (n=651).

Corresponding author: Dr. Elizabeth A. Platz, Department of Epidemiology, Room E6132-A, Johns Hopkins Bloomberg School of Public Health, 615 N. Wolfe Street, Baltimore, MD 21205. Tel: (410) 614-9674, Fax: (410) 614-2632, eplatz@jhsph.edu.

Department and institution in which the work was performed: Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health

The content of this work is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Results—Positive correlations were observed for African-American race, foreign birth, southern residence, smoking, alcohol consumption, ejaculation frequency, vasectomy, and high cholesterol. Inverse correlations were observed for social integration and routine health-related examinations.

Conclusions—These findings provide useful information on potential confounders for epidemiologic investigations of STIs and chronic diseases, and interesting new hypotheses for STI prevention (e.g., STI counseling before vasectomy).

Keywords

sexually transmitted diseases; epidemiology; confounding factor (epidemiology)

INTRODUCTION

Several epidemiologic studies have investigated sexually transmitted infections (STIs) and later risk of genitourinary conditions, such as prostate cancer, benign prostatic hyperplasia (BPH) and bladder cancer, with suggestive positive results (1–16). While these results may reflect causal associations, other possible explanations for some of these findings include recall, interviewer or selection biases, and confounding by factors possibly related to both STI acquisition and later risk of genitourinary conditions. These factors include previously identified risk factors or correlates of STIs, such as lower education and socioeconomic status (SES), minority race/ethnicity, residence in a Southern U.S. state, cigarette smoking, alcohol consumption, drug use, higher lifetime number of sexual partners, homosexual orientation, and lower social integration (17–28). Other possible confounding factors include those not typically considered in relation to STIs, such as general health-related behaviors (e.g., diet and exercise) or markers of such behaviors (e.g., body mass index (BMI) and cholesterol level), that might be correlated with recent or earlier acquisition of STIs and associated with later risk of genitourinary conditions. Very few of either of these types of factors has been investigated in older populations in which associations between STIs and later genitourinary conditions are typically studied. Therefore, we investigated correlates of STI histories in the Health Professionals Follow-up Study (HPFS), a cohort study of middle- to older-aged American male health professionals in which information on both STI histories (gonorrhea, syphilis, Chlamydia trachomatis, Trichomonas vaginalis, human papillomavirus (HPV) and human herpesvirus type 8 (HHV-8) infection), and an extensive array of demographic, behavioral, social integration and health-related characteristics has been collected. Although we previously investigated some of these variables in relation to STI histories in prior prostate disease publications (8,14.29), we have now expanded our investigation to include a much broader range of STIs and potential demographic, behavioral, social integration and health-related correlates.

METHODS

Study population and design

The HPFS is an ongoing cohort study of 51,529 American male health professionals aged 40– 75 at enrollment in 1986. Participants entered the study by completing a mailed baseline epidemiologic questionnaire, including questions on demographics, lifestyle behaviors and medical history, and a semi-quantitative food frequency questionnaire (FFQ). Since 1986, participants have completed epidemiologic questionnaires every two years to update exposure and disease information, and FFQs every four years to update dietary information. In 1993, participants were additionally asked to provide a blood sample for research purposes. Between 1993–1995, 18,018 participants returned a chilled, EDTA-preserved blood specimen to the Harvard School of Public Health by overnight courier. These specimens were centrifuged, separated into plasma, buffy coat and erythrocyte aliquots, and stored in liquid nitrogen.

Analysis of gonorrhea and syphilis—We selected as participants men who completed the 1992 questionnaire, which included questions on histories of gonorrhea and syphilis. For consistency with all other HPFS analyses, we excluded participants who did not complete the

Analysis of *C. trachomatis, T. vaginalis,* **HPV and HHV-8 infection**—We selected as participants incidence-density sampled controls from our previous nested case-control study of STIs and prostate cancer (8,30). As part of that study, we required controls to have completed the baseline FFQ, provided a blood sample in 1993–95, and been alive and free of a diagnosis of cancer (except non-melanoma skin cancer) as of the date of diagnosis of the case to which the control was matched. We also required controls to have had at least one prostate specific antigen (PSA) test between the date of blood draw and case diagnosis, and we individually matched controls to cases by age, year, time and season of blood draw, and PSA testing history before blood draw. Due to the low number of non-white controls (n=40), we only included white controls in the analysis (n=651).

baseline FFQ, or who were diagnosed with cancer (except non-melanoma skin cancer) before the date of return of the 1992 questionnaire. This left 32,970 white, 278 African-American,

572 Asian, and 2,212 men of other race/ethnicity in the analytic cohort.

The Harvard and Johns Hopkins Bloomberg Schools of Public Health ethical review boards approved this study.

Assessment of possible STI correlates

Most of the variables explored were assessed on the 1992 or prior questionnaires. These included previously identified STI correlates, such as 1) demographic and geographic variables (e.g., race/ethnicity, place of birth); 2) risk-taking behavioral variables (e.g., cigarette smoking, alcohol consumption) measured both earlier in life, and thus nearer the likely time of STI acquisition, and later in life; and 3) measures of social integration. We assessed social integration by components of the Berkman-Syme social network index. This index measures four types of social connections: marital status, sociability (number of and contact with close friends and relatives), religious group affiliation, and social or community group membership (31). We also explored general health-related behaviors or markers of such behaviors (e.g., physical activity, BMI), because we hypothesized that unhealthy behaviors (i.e., unprotected sexual activity and other unhealthy behaviors) might be correlated. Further rationale for the choice of each specific variable is provided in the Results section. We explored each variable in relation to each STI, even if the hypothesis was specific to one STI, to keep the analyses consistent across infections.

Assessment of STIs

Gonorrhea and syphilis—We obtained information on histories of gonorrhea and syphilis from the 1992 questionnaire, which asked participants to report whether they had ever had a diagnosis of syphilis, gonorrhea, or neither. We then categorized gonorrhea and syphilis separately as: no, yes, or unknown for men who did not respond to the STI question.

C. trachomatis, *T. vaginalis*, HPV and HHV-8 infection—We assessed histories of *C. trachomatis*, *T. vaginalis*, HPV and HHV-8 infection by plasma IgG antibody detection using specimens provided in 1993–5. We assessed *C. trachomatis* serostatus by the Ani Labsystems *C. trachomatis* IgG enzyme immunoassay (Ani Labsystems, Helsinki, Finland), *T. vaginalis* by an in-house enzyme-linked immunosorbent assay (ELISA) (8), HPV-16, -18 and -33 by three in-house ELISAs (32) and HHV-8 by an in-house monoclonal antibody-enhanced immunofluorescent assay (33). Further details of testing are described in previous publications (8,30).

Statistical analysis

We calculated means and proportions of variables by each STI, and compared these values by t-tests and Pearson's Chi-squared (or Fisher's exact) tests, respectively. We assessed trends in proportions by the Cochran-Armitage trend test. We investigated confounding by age and identified correlates, and mediation by marital status by linear or logistic regression, as appropriate. When assessing the presence or absence of correlations, we gave greater weight to patterns of correlations across STIs than to the statistical significance of individual correlations because of the large number of statistical comparisons made and the varying sample size of each STI analysis.

To investigate whether the age of participants influenced observed correlations, we stratified the analyses by age in 1992 (<65, \geq 65 years) to assess whether stronger correlations were observed among younger men than among older men, the latter of whom may have been depleted of men with higher variable values due to premature death (e.g., men who died of lung cancer due to heavy smoking). This analysis also allowed us to investigate whether secular trends in STI prevalence or self-reporting, or antibody waning since the likely time of STI acquisition (usually adolescence/early adulthood) affected the results. To investigate whether our cancer exclusion criterion influenced observed correlations, we repeated the self-reported analysis retaining men with cancer in 1992. We also repeated this analysis using the same inclusion/exclusion criteria as the nested prostate cancer case-control study to determine whether differing criteria affected observed correlations. Finally, to investigate whether we missed correlates due to the lower expected extent of STI history in the HPFS than in other study populations (i.e., one or two episodes of infection versus several episodes), we repeated the analysis comparing men with a history of \geq 2 STIs to those without a history of STIs.

RESULTS

Of the 36,032 eligible participants, 2.5% of white, 22.3% of African-American, 1.9% of Asian and 2.6% of participants of other race/ethnicity reported a history of gonorrhea, for an overall lifetime prevalence of 2.7%. 0.2% of both white and non-white participants reported a history of syphilis. The proportion of men with unknown histories of gonorrhea and syphilis did not vary appreciably by race (data not shown). All further analyses were limited to white participants because the reported lifetime prevalence of STIs differed greatly by race, and sufficient numbers of African-American or Asian men were not available to perform stratified analyses. Among white participants, the reported lifetime prevalence of gonorrhea decreased with increasing age, that of syphilis remained relatively constant with age, and the likelihood of not responding to the STI question increased with age (Table 1).

For the analysis of serologically detected STIs (n=651), 3.2% of white participants were seropositive for *C. trachomatis*, 9.2% for *T. vaginalis*, 8.8%, 5.7%, and 6.4% for HPV-16, -18, and -33, respectively, 16.6% for any of the three tested HPV types, and 18.1% for HHV-8. The prevalence of these STIs did not vary significantly with age (Table 1).

When we examined correlations among STIs, a self-reported history of gonorrhea was positively correlated with a history of syphilis and HHV-8 seropositivity. *C. trachomatis* and *T. vaginalis* seropositivity were both positively correlated with HPV infection, and *T. vaginalis* seropositivity was also weakly positively correlated with HHV-8 infection. With respect to HPV seropositivity, each HPV type (16, 18 and 33) was strongly positively correlated with each other type, and HPV-16 was also weakly positively correlated with HHV-8 infection (Table 2).

Geographic correlates

As HHV-8 seroprevalence is higher in certain parts of Africa and the Mediterranean (34), we investigated each infection in relation to foreign birth and Southern European ancestry. Men with histories of gonorrhea, syphilis and *C. trachomatis* infection were more likely to have been born in a foreign country than men without histories of infection, whereas no appreciable difference was observed by Southern European ancestry. Men with histories of gonorrhea and syphilis, but none of the serologically detected STIs, were also more likely to report living in the South at a young age (Table 3).

Risk-taking behaviors

With respect to *early adulthood* risk-taking behaviors, men with histories of gonorrhea and syphilis, but none of the serologically detected STIs, were more likely to report smoking cigarettes at a young age than men without histories of infection. Those with a history of gonorrhea were also more likely to report early alcohol consumption. These correlations attenuated after mutual adjustment. With respect to frequency of ejaculation from ages 20–29, a possible correlate of early life frequency of sexual activity and consequent exposure to STIs, men with histories of gonorrhea, syphilis and HPV infection reported higher frequencies than men without histories of infection (Table 3). No appreciable difference was observed by adolescent/early adulthood height, BMI, body habitus and physical activity (data not shown). We investigated these factors because we hypothesized that physical appearance and athleticism might influence opportunity for sexual relationships earlier in life when men are more likely to acquire STIs.

With respect to *later adulthood* risk-taking behaviors, men with histories of gonorrhea and syphilis, but none of the serologically detected STIs, were more likely to report cigarette smoking than men without histories of infection. In general, total reported alcohol consumption was higher and habitual consumption of >3 drinks per day was more common among men with than without histories of STIs. These correlations attenuated for gonorrhea and syphilis, but not for serologically detected STIs, after mutual adjustment. Reported ejaculation frequency from ages 40–49 and in 1991 was higher among men with histories of gonorrhea, syphilis and possibly HPV infection. Although not very pronounced, blood donation in the past 30 years was inversely correlated with histories of each infection. We investigated this variable as a surrogate for high STI-risk behaviors not asked about on HPFS questionnaires (e.g., injection drug use and male sexual contact) because these behaviors do not meet eligibility criteria for blood donation. No appreciable difference was observed by history of blood transfusion, which has been proposed as a risk factor for HHV-8 infection (35). Finally, men with histories of each infection, except syphilis and HHV-8, were more likely to report having had a vasectomy (Table 3). When we stratified this analysis by marital status, similar results to overall were observed among never-married men, married men who divorced or separated during followup, and men in stable marriages. No additional notable difference was observed by age at vasectomy. Too few men had vasectomies after 1992/blood draw to investigate the temporality of this correlation (data not shown).

Health-related behaviors

No appreciable pattern was observed by adult BMI, body habitus, level of physical activity or use of sunscreen. In general, men with histories of infection were less likely to report routine (i.e., not for symptoms) health screening, particularly physical and digital rectal examinations, than men without histories of infection. These differences persisted after additionally considering examinations for symptoms. They attenuated slightly after adjustment for marital status. Although not different by blood pressure, men with histories of infection were generally more likely to have had a diagnosis of high cholesterol than men without histories of infection.

No appreciable difference was observed by diet, including total energy, total fat, animal fat, red meat, fish, fruit, vegetable, and calcium intake (Table 3 and data not shown).

Social integration

In general, men with histories of each infection, except HHV-8, were less likely to have ever been or to currently be married, to attend religious meetings/services, irrespective of their religious affiliation, and to participate in group activities than men without histories of infection. These latter differences attenuated slightly after adjustment for marital status. Few differences were observed by participation in social sports, such as tennis, squash or racquetball. Men with histories of each infection, except HHV-8, were also less likely to report having children, close relatives or friends and less likely to report seeing their children, close relatives or friends regularly than men without histories of infection. These differences attenuated when only men with at least one child, close relative or friend were considered and when the results were adjusted for marital status. In general, men with histories of each infection were also less likely to consume meals prepared at home, a possible marker of either marital support or time spent with their spouse/family. These results were largely unchanged after adjustment for marital status. When we combined information from several of the abovementioned variables to form a modified version of the Berkman-Syme social network index, men with histories of each infection, except HPV and HHV-8, had lower mean indices, suggesting lower social integration, than men without histories of infection (Table 4 and data not shown).

With the exception of attenuated correlations mentioned above, similar results were generally observed when we adjusted each identified correlate for the other correlates. Additionally, none of the identified correlates influenced previously observed associations between specific STIs and prostate cancer, the most thoroughly investigated genitourinary outcome in the HPFS (8, 29,30). Similar inferences were also obtained when we 1) stratified the analyses by age in 1992; 2) repeated the self-reported analysis retaining men with cancer in 1992; 3) repeated the self-reported analysis retaining men with case-control study inclusion/ exclusion criteria; and 4) compared men with a history of ≥ 2 STIs to those without a history of STIs (data not shown).

DISCUSSION

In this cohort of older, professional American men, we identified several correlates of STI histories, including some previously observed in other typically higher STI-risk populations, such as African-American race, foreign birth, southern residence (26), cigarette smoking (26, 28), greater alcohol consumption (26,27), higher ejaculation frequency as a crude surrogate for higher frequency of sexual activity and greater opportunity for exposure to STIs, and lower social integration (17–25). Additional identified correlates included having had a vasectomy or a diagnosis of high cholesterol, and having a lesser degree of routine health-related examinations. Smoking and alcohol consumption correlations attenuated after mutual adjustment, and social integration and health-related examination correlations attenuated after adjustment for marriage. Importantly, none of the identified correlates confounded previously observed associations between specific STIs and prostate cancer in the HPFS.

To our knowledge, our study is the first to observe a correlation between having had a vasectomy and STIs. However, as our analysis was cross-sectional, there are several possible explanations for this correlation, some more likely than others. First, although STIs are typically acquired in adolescence/early adulthood (36) whereas vasectomies are usually performed after 30 years of age (37), STI acquisition is unlikely to have precipitated having a vasectomy. Another possibility is confounding by factors common to both STI acquisition and vasectomy. We believe that a general aversion or allergy to condoms is unlikely to be such a

factor because it ignores the abundance of female birth control options. Another unlikely possibility is confounding by marital status because similar results to overall were observed among never-married men, married men who divorced or separated during follow-up, and stably married men. A final possibility is that men with vasectomies no longer rely on condoms within or outside of their marriages. If this last possibility is true, then it may be important to counsel men about the need for continuing STI prevention possibly in and certainly outside of their marriages after vasectomy.

Before conducting this study, we hypothesized that men with STI histories would be more likely to report other unhealthy behaviors or markers of such behaviors than men without histories of STIs. While we did not observe correlations for all behaviors/markers examined, we did observe that men with histories of STIs were more likely to report cigarette smoking, heavy alcohol consumption, and high cholesterol, and less likely to report routine health-related examinations than men without histories of STIs. The latter health-related examination correlations attenuated after adjustment for marital status, suggesting that they may have been mediated by being married, while the remaining correlations did not. These remaining correlations may imply a general lesser attention to personal health among men with than among men without STI histories. While not obviously translatable to public health practice, these correlations may be important for future epidemiologic studies of STIs and chronic diseases, as they may confound observed associations.

Another notable correlation that deserves further mention is that between social integration and STI history. Although this correlation has been previously observed in several high-risk populations (17–25), our study is the first, to our knowledge, to observe such a correlation in an older low-risk population. Whether this correlation reflects a relation between low early life social integration (with persistent low social integration) and early acquisition of STIs, or low later life social integration and later life acquisition of STIs (e.g., because of riskier partner choices, such as commercial or casual sex partners) is difficult to discern, but merits further investigation given the paucity of data in older populations. Interestingly, while correlations between measures of social integration and STIs were observed consistently across most STIs, they were not observed for HHV-8. This pattern was unexpected for marital and parental status because HHV-8 seroprevalence is frequently higher among homosexual men (38), and thus might be expected to be inversely related to being married or having children. We could not directly investigate this hypothesis because HPFS participants were not asked about sexual orientation. The lack of correlation for other measures of social integration was less unexpected because homosexual orientation has not been observed to be associated with global social support (39) and because, unlike the other predominantly sexually transmitted infections considered in this analysis, HHV-8 is believed to have other important routes of transmission, such as via saliva (40), which might be less associated with social integration. In general, although we did not expect HHV-8 infection to share all of the same correlates as other STIs, we included it in this analysis because it is believed to be sexually transmissible and thus might share some correlates with the other STIs.

Just as some correlates were observed for all STIs except HHV-8 infection, other correlates (i.e., age, southern residence, smoking, and ejaculation frequency) were observed for self-reported but not serologically detected STIs. This difference may be explained by several possible reasons. First, these two groups of STIs may have different epidemiology. Gonorrhea and syphilis are currently localized to populations with high-risk health behaviors, and have been for several decades, whereas *C. trachomatis*, *T. vaginalis*, HPV and HHV-8 infection are more evenly spread in the U.S. population than gonorrhea and syphilis (26). Second, assessment of gonorrhea and syphilis relied on participant recall and willingness to report STI histories, whereas assessment of other STIs relied on biomarker detection. Therefore, associations between gonorrhea, syphilis and certain correlates may have been observed simply

Sutcliffe et al.

because participants who were willing to report their STI histories may have also been willing to report other less socially desirable characteristics. Alternatively, the sensitivity of assessing self-reported histories of gonorrhea and syphilis, two frequently symptomatic, well-recognized STIs, may have been greater than for serologic detection of other STIs because of waning antibody titers over time, an initially weak immune response, or other problems with test sensitivity, thereby resulting in a possibly lesser degree of non-differential misclassification and attenuation towards the null. However, why either of these two possible explanations would apply to only certain correlates and not others (e.g., alcohol consumption) is unclear. Finally, although the inclusion/exclusion criteria for the analyses of self-reported and serologically detected STIs differed, we do not believe these differences influenced the results because similar results were observed for self-reported STIs using the nested-case control study requirements as when using the original requirements.

With respect to the generalizability of our findings, we suspect that STI histories in our study population likely reflect a small number of lifetime episodes of infection, given the education, SES and racial/ethnic distribution of participants. Therefore, some of the observed correlates and the strength of these correlations may be more reflective of correlations with minimal STI history than extensive history. We attempted to evaluate this hypothesis by repeating the analyses comparing men with a history of ≥ 2 STIs to those without a history of STIs. No additional correlates were identified in this analysis, although the distribution of STIs among men with a history of ≥ 2 STIs was fairly narrow (range: 2–4; median=2). A further consideration in interpreting observed correlations is the possibility that a certain proportion of participants may have been educated under the G.I. and subsequent veterans' bills. Therefore, their STI histories may be more reflective of war/military training exposures than their prior or subsequent lifetime risk behaviors. Although these considerations may potentially affect the generalizability of our findings to younger high-risk populations, they should not affect their generalizability to other study populations of older men in which STIs and later genitourinary conditions are typically explored. Additionally, our choice of variables, while limited to those frequently collected in chronic disease-focused studies rather than STI studies (e.g., diet questions rather than drug use questions), may also be more relevant to study populations typically used to explore STIs and chronic diseases.

In summary, we observed several similar STI correlates in our cohort of older male health professionals as in other typically higher STI-risk populations (e.g., social integration), as well as several newly identified correlates (vasectomy, health-related markers/examinations). These findings provide useful information on potential confounding variables for epidemiologic investigations of STIs and later chronic diseases, and possible interesting new hypotheses for STI prevention (e.g., STI counseling before vasectomy).

Acknowledgments

Sources of financial support: Siobhan Sutcliffe was supported by a Doctoral Research Award from the Canadian Prostate Cancer Research Initiative/Canadian Institutes of Health Research, and the Fund for Research and Progress in Urology, John Hopkins Medical Institutions. This work was supported by research grants CA55075, HL35464 (Harvard), and P50CA58236 (Hopkins) from the National Institutes of Health, and the Summer Epidemiology Program Fund, Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health.

We are grateful to Jill Arnold, Elizabeth Frost-Hawes, Mira Kaufman, Laura Sampson, Alvin Wing, and Mildred Wolff for their continued help in conducting the Health Professionals Follow-up Study. We thank Billie J. Wood, Te-Hung Chang, Barbara Silver and Stefanie Morosky for performing *C. trachomatis, T. vaginalis,* HPV and HHV-8 antibody testing, respectively. We also thank Ligia A. Pinto and Dr. Allan Hildesheim for generous provision of HPV-16 antibody positive control serum and Dr. Patti E. Gravitt for advice regarding HPV antibody testing.

References

- 1. Dennis LK, Dawson DV. Meta-analysis of measures of sexual activity and prostate cancer. Epidemiology 2002 Jan;13(1):72–9. [PubMed: 11805589]
- Taylor ML, Mainous AG 3rd, Wells BJ. Prostate cancer and sexually transmitted diseases: a metaanalysis. Fam Med 2005 Jul–Aug;37(7):506–12. [PubMed: 15988645]
- Fernandez L, Galan Y, Jimenez R, Gutierrez A, Guerra M, Pereda C, et al. Sexual behaviour, history of sexually transmitted diseases, and the risk of prostate cancer: a case-control study in Cuba. Int J Epidemiol 2005 Feb;34(1):193–7. [PubMed: 15375086]
- 4. Huang WY, Hayes R, Pfeiffer R, Viscidi RP, Lee FK, Wang YF, et al. Sexually transmissible infections and prostate cancer risk. Cancer Epidemiol Biomarkers Prev 2008 Sep;17(9):2374–81. [PubMed: 18768506]
- Sarma AV, McLaughlin JC, Wallner LP, Dunn RL, Cooney KA, Schottenfeld D, et al. Sexual behavior, sexually transmitted diseases and prostatitis: the risk of prostate cancer in black men. J Urol 2006 Sep; 176(3):1108–13. [PubMed: 16890703]
- Lightfoot N, Conlon M, Kreiger N, Sass-Kortsak A, Purdham J, Darlington G. Medical history, sexual, and maturational factors and prostate cancer risk. Ann Epidemiol 2004 Oct;14(9):655–62. [PubMed: 15380796]
- Hoffman LJ, Bunker CH, Pellett PE, Trump DL, Patrick AL, Dollard SC, et al. Elevated seroprevalence of human herpesvirus 8 among men with prostate cancer. J Infect Dis 2004 Jan 1;189(1):15–20. [PubMed: 14702148]
- Sutcliffe S, Giovannucci E, Alderete JF, Chang TH, Gaydos CA, Zenilman JM, et al. Plasma antibodies against *Trichomonas vaginalis* and subsequent risk of prostate cancer. Cancer Epidemiol Biomarkers Prev 2006 May;15(5):939–45. [PubMed: 16702374]
- Adami HO, Kuper H, Andersson SO, Bergstrom R, Dillner J. Prostate cancer risk and serologic evidence of human papilloma virus infection: a population-based case-control study. Cancer Epidemiol Biomarkers Prev 2003 Sep;12(9):872–5. [PubMed: 14504197]
- Oishi K, Okada K, Yoshida O, Yamabe H, Ohno Y, Hayes RB, et al. A case-control study of prostatic cancer in Kyoto, Japan: sexual risk factors. Prostate 1990;17(4):269–79. [PubMed: 2251222]
- Signorello LB, Tzonou A, Lagiou P, Samoli E, Zavitsanos X, Trichopoulos D. The epidemiology of benign prostatic hyperplasia: a study in Greece. BJU Int 1999 Aug;84(3):286–91. [PubMed: 10468723]
- Joseph MA, Harlow SD, Wei JT, Sarma AV, Dunn RL, Taylor JM, et al. Risk factors for lower urinary tract symptoms in a population-based sample of African-American men. Am J Epidemiol 2003 May 15;157(10):906–14. [PubMed: 12746243]
- 13. Araki H, Watanabe H, Mishina T, Nakao M. High-risk group for benign prostatic hypertrophy. Prostate 1983;4(3):253–64. [PubMed: 6189108]
- Sutcliffe S, Giovannucci E, De Marzo AM, Willett WC, Platz EA. Sexually transmitted infections, prostatitis, ejaculation frequency, and the odds of lower urinary tract symptoms. Am J Epidemiol 2005 Nov 1;162(9):898–906. [PubMed: 16177142]
- 15. Michaud DS, Platz EA, Giovannucci E. Gonorrhoea and male bladder cancer in a prospective study. Br J Cancer 2007 Jan 15;96(1):169–71. [PubMed: 17164760]
- La Vecchia C, Negri E, D'Avanzo B, Savoldelli R, Franceschi S. Genital and urinary tract diseases and bladder cancer. Cancer Res 1991 Jan 15;51(2):629–31. [PubMed: 1985779]
- Mazzaferro KE, Murray PJ, Ness RB, Bass DC, Tyus N, Cook RL. Depression, stress, and social support as predictors of high-risk sexual behaviors and STIs in young women. J Adolesc Health 2006 Oct;39(4):601–3. [PubMed: 16982400]
- Ekstrand ML, Coates TJ. Maintenance of safer sexual behaviors and predictors of risky sex: the San Francisco Men's Health Study. Am J Public Health 1990 Aug;80(8):973–7. [PubMed: 2368861]
- 19. St Lawrence JS, Brasfield TL, Jefferson KW, Allyene E, Shirley A. Social support as a factor in African-American adolescents' sexual risk behavior. J Adolesc Res 1994;9(3):292–310.
- Smith CA. Factors associated with early sexual activity among urban adolescents. Soc Work 1997 Jul;42(4):334–46. [PubMed: 9228830]

Page 9

- 21. Sabo DF, Miller KE, Farrell MP, Melnick MJ, Barnes GM. High school athletic participation, sexual behavior and adolescent pregnancy: a regional study. J Adolesc Health 1999 Sep;25(3):207–16. [PubMed: 10475497]
- Resnick MD, Bearman PS, Blum RW, Bauman KE, Harris KM, Jones J, et al. Protecting adolescents from harm. Findings from the National Longitudinal Study on Adolescent Health. JAMA 1997 Sep 10;278(10):823–32. [PubMed: 9293990]
- Henrich CC, Brookmeyer KA, Shrier LA, Shahar G. Supportive relationships and sexual risk behavior in adolescence: an ecological-transactional approach. J Pediatr Psychol 2006 Apr;31(3):286–97. [PubMed: 15827352]
- 24. Crosby RA, DiClemente RJ, Wingood GM, Cobb BK, Harrington K, Davies SL, et al. HIV/STDprotective benefits of living with mothers in perceived supportive families: a study of high-risk African American female teens. Prev Med 2001 Sep;33(3):175–8. [PubMed: 11522158]
- Boyer CB, Tschann JM, Shafer MA. Predictors of risk for sexually transmitted diseases in ninth grade urban high school students. J Adolesc Res 1999 Oct;14(4):448–65. [PubMed: 12322581]
- Aral, SO.; Holmes, KK. Social and behavioral determinants of the epidemiology of STDs: Industrialized and developing countries. In: Holmes, K.; Sparling, P.; Mardh, P.; Lemon, S.; Stamm, W.; Piot, P., et al., editors. Sexually transmitted diseases. 3. New York: The McGraw-Hill Companies, Inc; 1999. p. 39-76.
- Cook RL, Clark DB. Is there an association between alcohol consumption and sexually transmitted diseases? A systematic review. Sex Transm Dis 2005 Mar;32(3):156–64. [PubMed: 15729152]
- Wolf R, Freedman D. Cigarette smoking, sexually transmitted diseases, and HIV/AIDS. Int J Dermatol 2000 Jan;39(1):1–9. [PubMed: 10651955]
- Sutcliffe S, Giovannucci E, De Marzo AM, Leitzmann MF, Willett WC, Platz EA. Gonorrhea, syphilis, clinical prostatitis, and the risk of prostate cancer. Cancer Epidemiol Biomarkers Prev 2006 Nov;15(11):2160–6. [PubMed: 17119041]
- 30. Sutcliffe S, Giovannucci E, Gaydos CA, Viscidi RP, Jenkins FJ, Zenilman JM, et al. Plasma antibodies against *Chlamydia trachomatis*, human papillomavirus, and human herpesvirus type 8 in relation to prostate cancer: a prospective study. Cancer Epidemiol Biomarkers Prev 2007 Aug;16(8):1573–80. [PubMed: 17684131]
- 31. Berkman, L. Doctoral dissertation. Berkeley: University of California at Berkeley; 1977. Social networks, host resistance, and mortality: a follow-up study of Alameda County residents.
- 32. Viscidi RP, Ahdieh-Grant L, Clayman B, Fox K, Massad LS, Cu-Uvin S, et al. Serum immunoglobulin G response to Human Papillomavirus type 16 virus-like particles in Human Immunodeficiency Virus (HIV)-positive and risk-matched HIV-negative women. JID 2003;187:194–205. [PubMed: 12552444]
- Jenkins FJ, Hoffman LJ, Liegey-Dougall A. Reactivation of and primary infection with human herpesvirus 8 among solid-organ transplant recipients. J Infect Dis 2002 May 1;185(9):1238–43. [PubMed: 12001040]
- Franceschi S, Geddes M. Epidemiology of classic Kaposi's sarcoma, with special reference to mediterranean population. Tumori 1995 Sep-Oct;81(5):308–14. [PubMed: 8804445]
- Blackbourn DJ, Ambroziak J, Lennette E, Adams M, Ramachandran B, Levy JA. Infectious human herpesvirus 8 in a healthy North American blood donor. Lancet 1997 Mar 1;349(9052):609–11. [PubMed: 9057733]
- 36. Centers for Disease Control and Prevention. Sexually transmitted disease surveillance, 2005. Atlanta, GA: U.S. Department of Health and Human Services; Nov. 2006
- Barone MA, Johnson CH, Luick MA, Teutonico DL, Magnani RJ. Characteristics of men receiving vasectomies in the United States, 1998–1999. Perspect Sex Reprod Health 2004 Jan-Feb;36(1):27– 33. [PubMed: 14982674]
- Engels EA, Atkinson JO, Graubard BI, McQuillan GM, Gamache C, Mbisa G, et al. Risk factors for human herpesvirus 8 infection among adults in the United States and evidence for sexual transmission. J Infect Dis 2007 Jul 15;196(2):199–207. [PubMed: 17570106]
- 39. Valanis BG, Bowen DJ, Bassford T, Whitlock E, Charney P, Carter RA. Sexual orientation and health: comparisons in the women's health initiative sample. Arch Fam Med 2000 Sep-Oct;9(9):843–53. [PubMed: 11031391]

40. Dukers NH, Rezza G. Human herpesvirus 8 epidemiology: what we do and do not know. AIDS 2003 Aug 15;17(12):1717–30. [PubMed: 12891058]

Table 1

Lifetime prevalence of select sexually transmitted infections by age^{*} among white participants in the Health Professionals Follow-up Study, 1992–1995.

	≥46	4654	55-64	65–74	≥75	p-trend
Infections assessed by self-report:						
N	32,970	11,548	10,375	8,652	2,395	
Gonorrhea (%, 95% CI)	2.5 (2.4, 2.7)	3.4	2.3	1.9	1.9	<0.01
Syphilis (%, 95% CI)	$0.23\ (0.18,\ 0.28)$	0.23	0.24	0.23	0.17	0.78
Unknown (%, 95% CI)	8.5 (8.2, 8.8)	6.4	8.2	10.4	12.7	<0.01
Infections assessed by plasma anti	body detection:					
Z	651	74	202	316	59	
C. trachomatis (%, 95% CI)	3.2 (1.9, 4.6)	6.8	3.5	1.9	5.1	0.21
T. vaginalis (%, 95% CI)	9.2 (7.0, 11.4)	8.1	8.9	9.5	10.2	0.64
HPV-16 (%, 95% CI)	8.8 (6.6, 10.9)	8.1	7.9	9.8	6.8	0.80
HPV-18 (%, 95% CI)	5.7 (3.9, 7.5)	6.8	5.0	5.7	6.8	0.91
HPV-33 (%, 95% CI)	6.4 (4.6, 8.3)	4.0	5.0	8.2	5.1	0.26
HPV-16, -18 or -33 (%, 95% CI)	16.6 (13.7, 19.4)	14.9	14.4	18.4	17.0	0.34
HHV-8 (%, 95% CI)	18.1 (15.2, 21.1)	12.2	22.3	17.7	13.6	0.78

Cancer Causes Control. Author manuscript; available in PMC 2010 November 22.

Age in 1992 for infections assessed by self-report and age at blood draw (1993–1995) for infections assessed by plasma antibody detection. Participants were required to be 40 to 75 years of age at enrollment in 1986. Therefore, participants were 46 to 81 years of age in 1992.

 $\overrightarrow{r}_{\mbox{Calculated}}$ by the Cochran-Armitage trend test.

Table 2

Correlations between histories of select sexually transmitted infections among white participants in the Health Professionals Follow-up Study, 1992–1995.

Sutcliffe et al.

		*		*				:		:		
	History of	gonorrhea/ · *	History	of syphilis'	C. trachomati	is seropositive ^s	T. vaginalis	seropositive	HPV-16, -18 or	-33 seropositive	HHV-8 sei	opositive.
	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No
	n=836	n=29,343	n=75	n=30,102	n=21	n=618	n=60	n=591	n=108	n=543	n=118	n=533
History of (%):												
Gonorrhea			24.0	2.7***	0.0	2.4	1.7	2.7	3.7	2.4	5.9	1.9^{***}
Syphilis	2.2	0.2^{****}			//	11	//	11	11	11	//	11
Seropositivity aga	uinst (%):											
C. trachomatis							3.3	3.2	5.6	2.8*	2.5	3.4
T. vaginalis					9.5	9.4			14.8	8.1^{***}	11.0	8.8
HPV-16					23.8	7.9***	15.0	8.1^{**}			11.0	8.3
HPV-18					4.8	5.7	11.7	5.1^{**}			6.8	5.4
HPV-33					14.3	6.2^*	11.7	5.9**			5.1	6.8
8-VHH					14.3	18.0	21.7	17.8	20.4	17.7		
* 0.10≤P<0.20												
** 0.05≤P<0.10												
*** 0.01≤P<0.05												
$^{****}_{P<0.01}$												
P values were calcul	lated by Pearso	on's Chi-square	or Fisher's	exact tests, as	appropriate.							
$\dot{\tau}_{{ m Results}}$ for men wi	th unknown hi	istories of gonori	thea and sy	philis are not	shown.							
[‡] Adjusted for age.												
s Results for men wi	th equivocal C	7. trachomatis an	tibody test	results are no	t shown.							

Cancer Causes Control. Author manuscript; available in PMC 2010 November 22.

 $^{\prime\prime}$ None of the participants tested for serologic evidence of a history of STIs reported a history of sybhilis.

_
_
_
_
_
_
- U
~
-
-
_
<u> </u>
-
<u> </u>
_
\sim
U U
_
_
_
~
~
0
<u> </u>
_
_
_
10
0)
-
0
~
_
_

NIH-PA Author Manuscript

Table 3

	i.
	Ъ.
	9.
	÷
	2
	9.
	÷,
	٠.
	2
	З
	Ę
	\mathbf{v}
	d
	Ä
	÷
	2
	Ц
	Ы
	Ľ.
	6
	÷,
	5
	ō
	· 🔁
	ŝ
	ല
	0
	Ě.
	Ц
	Ч
	H
	За
	Ψ
	щ
	Ð
	8
	Ξ
	.⊟
	S
	Ц
	.9
	Ξ.
	8
	Ę
	Ξ.
	
	ŏ
	Ē
	· =
	Ц
	S
	ar
	Ξ
	Ξ
	-
	al
	n
	×
	Se
	نَت
	S.
	<u>e</u>
	e)
	<u>,</u>
	Эf
	~
	S
	٠Ē
	õ
	ŝ
	Ë
	÷.
	N
	بے
	\mathbf{ts}
	Ц
	ğ
	ipa
	icipa
	rticipa
	articipa
	participa
	e participa
	nite participa
	/hite participa
	white participa
	f white participa
	of white participa
	i^{T} of white participa
	cs^{T} of white participa
	tics ^{τ} of white participa
	istics ⁷ of white participa
-	stristics ^{τ} of white participa
	teristics ⁷ of white participa
-	icteristics ^{τ} of white participa
	racteristics ^{τ} of white participa
-	haracteristics ⁷ of white participa
-	Tharacteristics ⁷ of white participa

									HPV-16	18 or - 33		
	History of	° gonorrhea ^{‡,§}	History	of syphilis [‡]	C. trachomat	is seropositive//	T. vaginalis	seropositive	seropo	sitive	HHV-8 sei	opositive.
	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No
	n=836	n=29,343	n=75	n=30,102	n=21	n=618	n=60	n=591	n=108	n=543	n=118	n=533
Geographic characteristics:												
Foreign birth (%)	4.0	2.4 ****	5.3	2.5*	19.0	2.1^{****}	1.7	2.7	3.7	2.4	2.5	2.6
Southern European ancestry (%)	25.3	25.2	30.7	25.3	23.8	21.4	16.7	22.2	25.0	21.0	21.2	21.8
Residence in the South at (%):												
Age 15	22.4	18.7^{****}	24.0	18.8	14.3	15.5	8.3	16.2^{*}	13.9	15.8	16.1	15.4
Age 25	25.1	21.4***	28.0	21.5*	9.5	19.3	13.3	19.6	16.7	19.5	16.1	19.7
Risk-taking behaviors:												
Early adulthood:												
Smoked cigarettes before age 30 (%)	61.3	48.4	57.3	48.6*	42.9	50.6	46.7	51.3	50.9	50.8	50.8	50.8
Consumed alcohol from ages 18–22 (%)	76.8	64.9****	66.7	65.4	52.4	68.6*	75.0	67.7	62.0	69.6*	70.3	67.9
Mean ejaculation frequency from ages 20–29 (times/ month)	15.6	14.1 ^{****}	15.7	14.1 ^{***}	13.6	14.0	14.3	14.0	14.7	13.9*	13.5	14.1
Later adulthood:												
Current smoker (%)	8.6	6.8***	12.0	6.9 ^{**}	4.8	4.8	3.3	5.1	6.5	4.6	6.8	4.5
Mean alcohol consumption (g/day)	14.3	10.4^{****}	13.1	10.5^{*}	10.3	10.4	14.0	10.2^{*}	11.8	10.3	13.3	10.0^{**}
Ever usually had >3 drinks/ day (%)	28.4	14.7***	24.0	15.0^{***}	14.3	16.7	13.3	17.8	25.9	15.6***	22.0	16.3^{*}
Mean ejaculation frequency (ti	mes/month)											
From ages 40–49	12.0	10.9^{****}	13.0	10.9^{****}	11.1	11.0	11.3	11.0	11.7	10.9	10.7	11.1
In 1991	8.0	7.1****	9.0	7.1	5.2	5.9	5.9	5.8	6.3	5.8	5.6	5.9
Blood donation in the past 30 years (%)	65.2	66.0	62.7	66.1	52.4	68.9*	65.0	68.9	67.6	68.7	66.1	69.0

_
_
~
_
_
_
_
_
0
~
-
~
+
_
-
\mathbf{O}
<u> </u>
_
_
-
0
~
_
_
-
<u> </u>
10
0
-
0
<u> </u>
0
<u> </u>
-

Sutcliffe et al.

	History of	gonorrhea ^{‡,§}	History	of syphilis [‡]	C. trachomati	is seropositive//	T. vaginalis	seropositive	HPV-16, - seropc	18 or - 33 Sitive	HHV-8 sei	opositive
	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No
	n=836	n=29,343	n=75	n=30,102	n=21	n=618	n=60	n=591	n=108	n=543	n=118	n=533
Blood transfusion before study blood draw (%) $\dot{\tau}\dot{\tau}$					9.5	6.5	5.0	6.6	5.6	6.6	6.8	6.4
History of vasectomy (%)	27.0	25.6	25.3	25.8	42.9	25.1**	30.0	25.7	35.2	24.3	23.7	26.6
Health-related behaviors an	d markers:											
Current mean body mass index (kg/m ²)	25.5	25.5	25.2	25.5	25.4	25.6	25.5	25.6	26.1	25.5**	25.9	25.5
Current body habitus $(\%)^{\ddagger\ddagger}$:												
Lean	40.1	39.4	40.0	39.4	38.1	36.6	35.0	37.1	29.6	38.3	30.5	38.3
Muscular	31.2	31.7	29.3	31.6	23.8	36.4	35.0	35.9	40.7	34.8	38.1	35.3
Fat	18.6	18.0	13.3	18.0	19.0	21.0^{*}	21.7	20.6	19.4	21.0^{*}	25.4	19.7
Any vigorous leisure-time physical activity (%)	63.3	60.3**	50.7	60.5**	71.4	59.1	53.3	60.2	61.1	59.3	60.2	59.5
Use of sunscreen $\ge 75\%$ of the time during the past summer (%)	34.9	34.4	33.3	34.6	47.6	33.3*	36.7	33.3	29.6	34.4	33.0	33.8
Routine physical exam at least every 2 years since 1986 (%)	28.9	31.0	16.0	30.9****	47.6	40.0	40.0	39.9	33.3	41.2*	38.1	40.3
Routine rectal exam at least every 2 years since 1986 (%)	23.4	26.8 ^{***}	16.0	26.6 ^{***}	19.0	38.5**	30.0	38.4	32.4	38.7	37.3	37.7
Routine prostate specific antigen test at least every 2 years up to 2000 (%)	34.9	34.3	28.0	34.2	61.9	55.3	56.7	54.8	50.0	56.0	54.2	55.2
At least one routine sigmoidoscopy or colonoscopy since 1986 (%)	33.0	32.9	37.3	32.7	42.9	40.9	36.7	41.3	38.9	41.2	37.3	41.6
High blood pressure (%)	31.1	28.6^*	29.3	28.4	28.6	31.2	33.3	30.3	32.4	30.2	29.7	30.8
High serum cholesterol (%)	40.5	36.4***	32.0	36.4	47.6	43.7	48.3	43.2	40.7	44.2	49.2	42.4 [*]
* 0.10≤P<0.20												

Cancer Causes Control. Author manuscript; available in PMC 2010 November 22.

** 0.05<P<0.10 *** 0.01<P<0.05 **NIH-PA Author Manuscript**

NIH-PA Author Manuscript

 $^{****}_{P<0.01}$

P values were calculated by t-tests for continuous variables, and Pearson's Chi-square or Fisher's exact tests, as appropriate, for binary and categorical variables.

 † All characteristics were assessed on the 1986, 1988, 1990 or 1992 questionnaires unless otherwise specified.

 \sharp Results for men with unknown histories of gonorrhea and syphilis are not shown.

[§]Adjusted for age.

 $\left\|\right\|_{K}$ Results for men with equivocal C trachomatis antibody test results are not shown.

 $\dot{\tau}^{\dagger}$ Assessed on the 2000 questionnaire. Lifetime prevalences may underestimate the true lifetime prevalences of blood transfusion in this cohort because the chronologic order of blood draw and blood transfusion (solicited by age at blood transfusion in five year intervals) could not be discemed for all men.

 $\sharp\sharp$ Numbers may not sum to 100% due to missing responses.

Sutcliffe et al.

_
_
_
_
U
-
_
<u> </u>
-
_
-
0
\sim
_
_
<
01
<u>u</u>
_
_
ć
5
0)
õ
0
_
- i - i
7
0
-

NIH-PA Author Manuscript

Table 4

Social integration characteristics † of white participants by select sexually transmitted infections in the Health Professionals Follow-up Study, 1992–1995.

	History of	gonorrhea [‡] ,§	History	of syphilis \sharp	C. trachomatis	: seropositive//	T. vaginalis	s seropositive	HPV-16, -1 seropos	l8 or -33 átive	HHV-8 sei	ropositive
	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No
	n=836	n=29,343	n=75	n=30,102	n=21	n=618	n=60	n=591	n=108	n=543	n=118	n=533
Ever married (%)	79.8	84.7****	68.0	84.6****	81.0	90.8^{*}	86.7	90.7	86.1	91.2^{*}	92.4	6.68
Currently married (%)	67.3	78.7***	57.3	78.4***	76.2	87.1*	85.0	86.8	83.3	87.3	88.1	86.3
Attend any religious meetings/ services (%)	45.6	60.5***	49.3	60.1**	52.4	67.8*	58.3	68.4*	63.0	68.3	69.5	67.0
Participate in any group activities $t^{\dagger \dagger}$ (%)	40.0	52.2	46.7	51.9	38.1	60.2^{***}	46.7	60.4***	56.5	59.7	60.2	58.9
Spend any time playing tennis, squash or racquetball (%)	15.3	16.2	10.7	16.3*	23.8	15.2	16.7	15.4	12.0	16.2	19.5	14.6*
Only child $\ddagger \ddagger 1 \%$ (%)	8.5	10.8^{***}	13.3	10.7	9.5	11.6	8.3	11.7	11.1	11.4	15.2	10.5^{*}
Any living children (%)	69.69	79.8***	60.0	79.5****	81.0	86.4	81.7	86.5	81.5	86.9^{*}	87.3	85.7
Any close relatives (%)	79.0	80.0	74.7	80.0	76.2	84.6	81.7	84.6	83.3	84.5	83.0	84.6
Any close friends (%)	81.0	81.6	77.3	81.6	81.0	88.2	80.0	88.7**	86.1	88.2	89.0	87.6
≥3 close friends (%)	61.0	66.3****	53.3	66.1^{***}	61.9	74.3	61.7	75.0***	70.4	74.4	78.8	72.6*
See at least once/month (%):												
Children	58.8	71.4***	54.7	71.1	66.7	73.6	66.7	73.9	65.7	74.8**	72.9	73.4
Close relatives	54.3	60.3****	61.3	60.2	52.4	62.5	51.7	63.3**	60.2	62.6	61.0	62.5
Close friends	77.8	79.4	78.7	79.3	76.2	86.1	83.3	86.0	83.3	86.2	86.4	85.6
See at least 3 close friends/ month (%)	48.6	54.8***	46.7	54.5*	47.6	63.6*	60.0	63.4	60.2	63.7	69.5	61.7*
Meals prepared at home ≥ 5 times	s/week ^{§§} (%	:()										
Midday	24.6	29.6***	25.3	29.2	33.3	37.1	31.7	37.9	33.3	38.1	29.7	39.0 ^{**}
Evening	64.7	72.0****	68.0	71.8	66.7	75.7	80.0	75.3	70.4	76.8*	76.3	75.6
Mean modified Berkman- Syme social network index// //	2.8	3.2****	2.8	3.2***	3.1	3.3*	3.2	3.3*	3.3	3.3	3.4	3.3

Cancer Causes Control. Author manuscript; available in PMC 2010 November 22.

* 0.10≤P<0.20

** 0.05<P<0.10

*** 0.01≤P<0.05

 $^{****}_{P<0.01}$

P values were calculated by t-tests for continuous variables, and Pearson's Chi-square or Fisher's exact tests, as appropriate, for binary variables.

Sutcliffe et al.

 $\dot{\tau}$ All characteristics were assessed on the 1988 questionnaire unless otherwise specified.

[§]Adjusted for age.

''Results for men with equivocal C. trachomatis antibody test results are not shown.

 †† Includes social, work, church-connected, self-help, charity, public service or community group activities.

 \ddagger^{\ddagger} Assessed on the 1996 questionnaire.

SS Assessed on the 1986 questionnaire.

//// Calculated by assigning one point each for affirmative responses to the questions on currently married, any religious attendance, any group activities, and any close friends, and then summing these points to obtain an index ranging from zero to four.