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The Epidemiology of Cancer Among Police Officers

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Abstract

Background—This review summarizes peer-reviewed studies examining cancer risks among police officers. It provides an overview of existing research limitations and uncertainties and the plausible etiologic risk factors associated with cancer in this understudied occupation.

Methods—Previous cancer studies among police officers were obtained via a systematic review of the MEDLINE, CABDirect, and Web of Science bibliographic databases.

Results—Quality observational studies of cancer among police officers are sparse and subject to limitations in exposure assessment and other methods. Results from three studies suggested possible increased mortality risks for all cancers, and cancers of the colon, kidney, digestive system, esophagus, male breast, and testis, as well as Hodgkin's disease. Few incidence studies have been performed, and results have been mixed, although some associations with police work have been observed for thyroid, skin, and male breast cancer.

Conclusions—Police are exposed to a mix of known or suspected agents or activities that increase cancer risk. Epidemiologic evidence to date is sparse and inconsistent. There is a critical need for more research to understand the biological and social processes underlying exposures and the suggested disproportionate risks and to identify effective prevention strategies.

Keywords

police; cancer; mortality; environmental; shift work

INTRODUCTION

Police officers serve a vital role in maintaining safety and order in the United States and throughout the world. According to the Federal Bureau of Investigation (FBI), there were 1, 017, 954 law enforcement employees, including 699, 850 police officers in the US as of 2007 [FBI, 2008]. Law enforcement officers suffer disproportionately from numerous health problems including, chronic heart disease (CHD), diabetes, metabolic disorders,

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psychological stress, depression, suicide, sleep disorders, and cancer [Vena et al., 1986; Franke et al., 1997; Violanti et al., 1998]. Although a majority of this workforce is exposed to various known or suspected carcinogens, there have been only a limited number of studies to assess cancer risks in this occupation. Considering the large number of cancer studies that have been performed among other occupations, such as doctors, nurses, firefighters, and military personnel, the degree to which cancer is understudied among police officers is noteworthy.

Along with the stressful and sometimes life-threatening work that officers face, they often work irregular hours including nights or rotating shifts, which have been associated with disruption of circadian rhythms [Gordon et al., 1986; Wirth et al., 2011]. Circadian rhythms help maintain homeostasis in a variety of physiological processes, such as the immune, endocrine, cardiovascular, and autonomic nervous systems, and when disrupted may increase susceptibility to disease, including cancer. There is also an elevated prevalence of alcohol use [Violanti et al., 2009a] and obesity [Ramey et al., 2009], and in some cases a reduction in physical activity [Richmond et al., 1998] or sleep quality [Charles et al., 2007a] among police officers compared to general US or other populations. Although the prevalence of smoking among police officers was similar to that of the general US population in one study [Nelson et al., 1994], exposures to other known or suspected carcinogens in this population include traffic-related airborne particulate matter [Riediker et al., 2004], other air pollutants [Hu et al., 2007], and shift work, including nights and rotating shifts [Violanti et al., 2009b; IARC, 2010]. Many of these agents have been studied extensively in relation to cancer morbidity and mortality, although not necessarily among police. This review provides a comprehensive summary and critique of cancer studies among police officers and describes carcinogenic exposures and potential avenues for prevention.

METHODS

A computer-assisted search of English language articles using MEDLINE, CABDirect, and Web of Science identified potential articles for review through October 2010. Articles were not restricted to a date range. A Boolean phrase search of key words: (police OR policemen OR police officers OR law enforcement OR policewomen OR peace officers OR patrolmen OR security officers) AND (cancer OR neoplasms OR cancer risk OR cancer incidence OR cancer mortality) was used to search for potential articles. Articles were then selected from the computer-assisted search based on relevance to the review topic including a direct risk estimate for police work. Secondary citations identified within selected articles were also reviewed. Some screening level occupational mortality analyses using country or state-based census and death certificate reports were not included since these types of studies can be susceptible to inaccurate occupational information and other limitations [McLaughlin and Mehl, 1991; Bidulescu et al., 2007; German et al., 2011]. Instead, we attempted to focus our search primarily on hypothesis driven investigations, although these were not always stated explicitly, and on studies that directly targeted police work as the exposure. A meta-analysis was not pursued due to the limited number of studies available for analysis, and since some assumptions (e.g., independence) would not have been met. Articles were also excluded if police were used as a comparison or reference group only, if police were grouped into a category with other occupations, if a direct estimate of risk was not provided or if it was estimated based on an exposure or risk assessment model, or if data were based on state or country census reports [Zahm et al., 1989; Demers et al., 1992a; Gubéran et al., 1992; Muscat et al., 1998; Pollàn et al., 2001; Borrell et al., 2003; Lope et al., 2005; Wiwanitkit et al., 2005; Bagai et al., 2007]. In addition, one study examined cancer mortality among quintiles of plasma insulin using police officers. Considering the primary focus of this study was the relationship between insulin and cancer mortality, this study was not included

[Pyorala et al., 2000]. A total of 14 studies were included in this review. Table I displays findings of the included studies by cancer type. Institutional Review Board approval was not required because this is a review article and did not utilize any data.

RESULTS

Cohort Studies

In a study of municipal workers from Buffalo, New York, Vena et al. [1986] examined mortality among 2, 376 white male police officers on active duty between 1950 and 1979 with a minimum work duration of 5 years. Overall, standardized mortality ratios (SMRs) among police officers were comparable to the US white male population (SMR = 1.06; 95% confidence interval (95% CI) = 0.98–1.14). However, mortality from all malignant neoplasms (SMR = 1.27; 95% CI = 1.08–1.49), cancers of the digestive organs and peritoneum (SMR = 1.57; 95% CI = 1.18–2.04), esophagus (SMR = 2.86; 95% CI = 1.23–5.64), and colon (SMR = 1.80; 95% CI = 1.10–2.79) were elevated. When duration of employment was examined, statistically significant increases in SMRs for digestive, lymphatic or hematopoietic tissues were observed for officers working 10–19 years or more than 40 years. Those working more than 40 years also had a fourfold increase in bladder cancer mortality, and brain cancer mortality was elevated among officers employed 20–29 years [Vena et al., 1986]. This retrospective cohort mortality study was the only study out of the three cohort mortality studies to look at cancer mortality by years worked and latency. Internal cohort analyses were also conducted to account for the healthy worker effect by comparing mortality rates among police officers to all other municipal workers, the majority of whom were firefighters. Police officers compared to firefighters had elevated rate ratios for all malignant neoplasms, all digestive neoplasms including the esophagus and colon, and cancer of the respiratory system.

In 1998, Violanti and Vena et al. updated the Buffalo Police cohort and extended follow-up through December 31, 1990. The cancer mortality patterns remained with elevated mortality rates from all malignant neoplasms and for cancers of the digestive organs and peritoneum, esophagus, and colon. Mortality rates for kidney cancer (SMR = 2.08; 95% CI = 1.00–3.82) and Hodgkin's disease (SMR = 3.13; 95% CI = 1.01–7.29) also were elevated [Violanti et al., 1998].

Gu et al. examined cancer incidence among 2, 234 white male police officers with at least 5 years of police service in Buffalo, New York. This cohort is the retrospective counterpart to the prospective Buffalo Cardio-Metabolic Occupational Police Stress (BCOPS) cohort. The BCOPS study provides a prospective framework for examining biological processes through which stressors associated with police work may mediate adverse health outcomes. The protocol combines the characterization of stress biomarkers, subclinical CVD measures, psychosocial factors, and shift work to examine their potential associations with psychological disturbances and chronic diseases afflicting police officers [Violanti et al., 2006; Violanti et al., 2009a]. The period of this retrospective investigation was January 1, 1976 until December 31, 2006. There were a total of 406 observed cancers. The standardized incidence ratio (SIR) was similar to the US white male population (SIR = 0.94, 95% CI = 0.85–1.03). A statistically significantly elevated SIR was observed for Hodgkin's lymphoma (SIR = 3.34, 95% CI = 1.22–7.26). Increased SIRs were observed for kidney, brain, and thyroid cancer, although they were not statistically significant. There also were statistically significant decreases in skin (SIR = 0.54, 95% CI = 0.26–0.98) and bladder cancer risk (SIR = 0.64, 95% CI = 0.39–0.99) [Gu et al., 2011].

Mortality among firefighters employed for at least 1 year in Seattle, Tacoma (WA), and Portland (OR) was examined between 1944 and 1979 with follow up until 1989 in a

retrospective cohort study that examined whether smoke exposure among firefighters increased cancer risk [Demers et al., 1992b]. Ironically, Demers et al. used police as a comparison population for their study and the mortality experience in this population of police officers from the same cities was compared with national rates. Inclusion criteria were not specified and little information is provided about the dynamics or characteristics of the 3, 676 police officers with follow-up that yielded 714 deaths. Police officers had an SMR for all cancers combined of 0.95, and decreased colon cancer mortality (SMR = 0.50; 95% CI = 0.22–0.99) compared to white US males. There were no statistically significant observed increases in mortality for other cancer types [Demers et al., 1992b], although the point estimates for lymphosarcoma and leukemia were elevated. The SMR reported for lung cancer was 0.92, 95% CI = 0.69–1.19. Cancer of the esophagus was not reported, and no analyses were performed for duration of employment or latency among police. Similarly, Rosenstock et al. focused on respiratory mortality in a subset of the Demers et al. population with inclusion limited to those employed at least 1 day after January 1, 1980 and actively employed for at least 1 day after January 1, 1945 with follow-up through 1984. The police comparison cohort was limited to Portland and Tacoma as "Seattle records could not provide complete assessment of the police cohort." The police cohort consisted of 2, 074 men with 389 observed deaths. Elevated mortality was observed for malignant neoplasms of the trachea, bronchus, and lung among police officers compared to the US population, although the increase was not statistically significant (SMR = 1.09; 95% CI = 0.75–1.26) [Rosenstock et al., 1990]. No other cancer mortality findings are reported as this study focused on respiratory mortality. These two papers therefore lack analytical approaches that fully inform the issues of cancer mortality among police officers.

Morton et al. examined risk of leukemia incidence among various occupational groups in the Portland–Vancouver metropolitan area from 1963 through 1977. Usual occupation of the case was used to calculate the SIR for each occupational group based on the 1970 census population counts by occupation. As shown in Table I, there was a statistically significant increase in total leukemia incidence (SIR = 2.6; $P < 0.05$) among police officers compared to the general population, which was accounted for primarily by an elevated SIR for nonlymphatic leukemia (SIR = 3.4, $P < 0.05$). Although limited by time period (case ascertainment included the years 1963–1977), this incidence study contributes to the body of evidence that police officers may be at increased risk of leukemia [Morton and Marjanovic, 1984].

A total of 3, 868 urban police officers in Rome, who were investigated using a retrospective cohort design, had increased mortality rates from cancers of the colon, bladder, and kidneys, as well as non-Hodgkin's lymphoma; the mortality risk estimates were imprecise and not statistically significant [Forastiere et al., 1994]. Subjects with 20–29 years of employment duration had increased mortality risks from colon (SMR = 2.32; 95% CI = 1.32–3.76), breast (SMR = 21.82; 95% CI = 2.64–78.82), and endocrine gland cancers (SMR = 5.88; 95% CI = 1.21–17.19). Also of note were two cases of male breast cancer (SMR = 14.36; 95% CI = 1.73–51.90). Results from a case–control analysis by job category indicated bladder cancer mortality was elevated among car drivers (odds ratio (OR) = 4.17; 95% CI = 1.14–15.24), especially those with 10 or more years of experience (OR = 12.2; 95% CI = 2.93–51.32). Increased non-Hodgkin's lymphoma mortality was also observed among motorcyclist officers, although the effect was not statistically significant [Forastiere et al., 1994].

A retrospective cohort study was conducted among 22, 197 Ontario police officers or retirees from 1964 to 1995 from department rosters, with cancer incidence data from the Ontario Cancer Registry and mortality data from the Ontario Mortality Database [Finkelstein, 1998]. A lower incidence of lung cancer (SIR = 0.66; 90% CI = 0.52–0.82) and all cancer combined (SIR = 0.90; 90% CI = 0.83–0.98) was observed among male officers

from the time of cohort entry to the end of 1995. An increased incidence of melanoma was also reported (SIR = 1.45; 90% CI = 1.10–1.88). In this study, there were a number of melanomas as well as testicular tumors diagnosed between the actual hire date and the date at which complete cohort identification was possible in their police department, possibly leading to an underestimate of cases. When rates beginning in 1964 or the date of hire to the end of 1995 were examined for all police officers, the incidence of testicular cancer (SIR = 1.33; 90% CI = 1.0–1.74) or melanoma (SIR = 1.37; 90% CI = 1.08–1.72) was elevated, whereas the rate for all solid tumors was reduced (SIR = 0.83; 90% CI = 0.77–0.90). The investigators suggested radar and sunlight exposures as plausible risk factors that could have accounted for the increased rates of testicular cancer and melanoma, respectively [Finkelstein, 1998]. Although a major strength is the measure of incidence outcome, this study had relatively short latencies between cohort entry and diagnosis. An entry criterion was enrollment on the police roster in 1970; however, Toronto (n = 5, 416) and Provincial police (n = 5, 366) were not added until 1981 and 1987, respectively. Follow-up was to the end of 1995. The majority of the cohort had less than a 15-year follow-up.

Another study examined the hypothesis that radar devices contribute to increases in testicular cancer in a retrospective cohort of 340 officers in response to reports of a cancer cluster in two police departments in Washington State [Davis and Mostofi, 1993]. Six cases of testicular cancer were confirmed with a resulting SMR of 6.9 ($P < 0.01$). All those diagnosed with testicular cancer had been working as police officers for the majority of their occupational life and the average work duration as a police officer prior to diagnosis was 14.7 years [Davis and Mostofi, 1993].

A cancer cluster in a British Columbia police detachment followed 20 current and 154 previous employees associated with the worksite since occupancy began in 1963 [van Netten et al., 2003]. A total of 16 cancer cases were reported. The age at diagnosis was between 22 and 44 years. Excess cases of testicular, cervical, and colon cancer were identified, along with melanoma, leukemia, and lymphoma, although not all achieved statistical significance. The total number of male cancer cases was 2.3 times higher than expected in the general British Columbia population ($P = 0.05$, based on a one-sided significance test) [van Netten et al., 2003].

In the Netherlands, a population-based prospective cohort study of men employed in various occupations was performed beginning in 1986 with follow-up data until 1993 [Zeegers et al., 2004]. The study employed a case–cohort approach, collecting data on 830 cases and 1, 525 subcohort members. Study participants were grouped based on occupation, and incident prostate cancers were ascertained via a cancer registry. Police work as the position held at the time of baseline, which was defined as the last occupation held at the time of the baseline questionnaire in September 1989, was associated with a substantial increase in prostate cancer risk (relative risk (RR) = 4.00; 95% CI = 1.59–10.02), as was police work for most of the career (RR = 3.91; 95% CI = 1.53–9.99). Ever having worked as a police officer was associated with a 67% increase in prostate cancer risk for each decade of work [Zeegers et al., 2004].

Case–Control and Other Study Designs

The role of occupation as a risk factor for cancer among 58, 134 male patients over 25 years of age was evaluated between 1980 and 1993, as recorded in the Swiss Cancer Registries of Basel, Geneva, St. Gall, Vaud, and Zurich [Bouchardy et al., 2002]. A case–control method was applied where cancers of interest were evaluated and all other cancer cases served as referents. Police officers were more likely to have cancer of the lung (small cell, OR = 1.5; 95% CI = 1.0–2.2), prostate (OR = 1.3; 95% CI = 1.0–1.5), or bladder (OR = 1.4; 95% CI = 1.0–1.9). Increased ORs were also noted among police officers for cancers of the colon,

rectum, liver, lung, kidney, and thyroid gland, as well as Hodgkin's disease, acute leukemia, and melanoma, although none of these achieved statistical significance [Bouchardy et al., 2002]. Use of cancer cases as controls could lead to an underestimation of risk if there was an unknown or undescribed association between police work with one or more of the referent cancers.

Acoustic neuromas were examined in a case–control study that included 793 cases from the entire Swedish population from 1987 to 1999. Compared to all men, the OR for police officers was greater following 10 years of work experience prior to their diagnosis (OR = 2.5; 95% CI = 1.1–5.6). A similar effect was observed for those with more than 10 years of experience, but was not statistically significant (OR = 1.9; 95% CI = 0.8–4.5) [Prochazka et al., 2010].

A proportional mortality study of New Jersey police included 567 officers between 1974 and 1980. Analyses were restricted to the white male population. An elevated proportional mortality ratio (PMR) was identified for cancers of the large intestine (PMR = 1.58) and for melanoma (PMR = 2.10), and the PMR for digestive cancer increased with increasing duration of employment [Feuer and Rosenman, 1986].

DISCUSSION

Police officers are exposed to a number of known or potential carcinogens, although to the authors' knowledge there are no systematic reviews of this subject. Among the epidemiologic investigations that examined relationships between police work and cancer, statistically significant increases in mortality due to: all cancer, digestive organ malignancies, and cancer of the esophagus, colon, kidney, bladder, brain, lymphatic and hematopoietic tissues, endocrine glands, and breasts, as well as testicular cancer, melanoma, and Hodgkin's disease have been reported [Feuer and Rosenman, 1986; Vena et al., 1986; Davis and Mostofi, 1993; Forastiere et al., 1994; Violanti et al., 1998]. Evidence for dose–response with increasing years of police work was noted in a few studies [Vena et al., 1986; Forastiere et al., 1994]. When cancer incidence was examined, some consistency with cancer mortality studies was observed. Statistically significant increases in cancer incidence have been reported for all cancer combined, and for cancers of the prostate, bladder, and thyroid, as well as testicular cancer, Hodgkin's lymphoma, and melanoma [Finkelstein, 1998; Bouchardy et al., 2002; van Netten et al., 2003; Zeegers et al., 2004; Gu et al., 2011].

There were noteworthy limitations among most of the studies reviewed, including: the use of surrogate exposures (e.g., job title) and the absence of quantitative exposure assessment for known or suspected carcinogens; a lack of control for potential confounding factors; lack of consideration of the promoting or potentiating effects of physiological stress; or behavioral, lifestyle, or coping factors such as alcohol consumption, exercise, or shiftwork and sleep disruption. The "healthy worker" effect, which stems from working populations typically being healthier than the general population [Meijers et al., 1989], results in a tendency to underestimate risk when population based referents are used. This type of bias was implicated as a reason for reduced risks observed in several studies among police [Finkelstein, 1998; Violanti et al., 1998; Borrell et al., 2003; Kang et al., 2008]. To avoid this limitation, investigators can compare police officers to other working populations with similar characteristics, or evaluate dose response within the population of interest. Only one study conducted internal cohort analyses [Vena et al., 1986] and only a few studies assessed work duration or latency [Vena et al., 1986; Violanti et al., 1998]. Other issues inherent in the study of law enforcement populations include: sample size limitations with corresponding reductions in statistical power, the potential for selection bias due to a lack of participation, or relatively short follow-up periods, which can lead to incomplete

ascertainment of outcome and an underestimation of risk. These concerns were acknowledged in many studies included in this review [Rosenstock et al., 1990; Forastiere et al., 1994; Muscat et al., 1998; Pyorala et al., 2000; Bagai et al., 2007], and may be particularly relevant to studies among police officers. For example, police officers have duties and schedules that render compliance with research protocols somewhat difficult. Studies focusing on cancer usually require extended follow-up periods to account for latencies of a decade or more, and they may select for officers who are better adapted to the stress associated with police work and therefore healthier or more resistant to disease.

Epidemiologic evaluations of law enforcement as a cancer risk factor are hampered not only by methodological limitations but also by the limited number of studies conducted to date. However, a complementary literature has documented exposures to carcinogens among police officers. Such exposures increase biological plausibility and provide supporting evidence that officers are at increased risk for cancer development. These occupational hazards are briefly described below (Table II). Note that police officers can be simultaneously exposed to multiple carcinogenic risk factors, such as shift work and obesity. However, the potential impacts and relative contributions of these agents to cancer risk have not been well described and represent an important area for future research.

Air Pollution, Particulate Matter, and Chemical Agents

Traffic police often spend at least several hours per day driving or directing traffic in congested areas. These activities may result in exposure to carcinogenetic airborne pollutants derived from motor vehicle exhaust, such as benzene, polycyclic aromatic hydrocarbons (PAHs), and persistent organic pollutants, for example dioxin [Forastiere et al., 1994; Ritter et al., 2007]. Personal benzene exposures among police officers were three to five times higher than those observed among controls [Pilidis et al., 2009], and traffic police from Bangkok had estimated cancer risks of 1.8 per 100, 000 based on a benzene exposure model [Wiwanitkit et al., 2005]. Particulate matter (PM) is a major air pollution constituent that can induce inflammation, free radical formation, oxidative DNA damage, cytotoxicity, and mutagenesis within the respiratory tract [Valavanidis et al., 2008]. The physiologic effects of in-vehicle, roadside, and ambient $PM_{2.5}$ were investigated among young, healthy, non-smoking, male North Carolina Highway Patrol troopers [Riediker et al., 2004]. Nine officers were monitored on four successive days while working the 3 PM to midnight shift. In-vehicle $PM_{2.5}$ (average: 24 $\mu g/m^3$) was associated with decreased lymphocyte counts (−11% per 10 $\mu g/m^3$) and increased concentrations of red blood cells, C-reactive protein, and von Willebrand factor [Riediker et al., 2004; Hanahan and Weinberg, 2011]. Hu et al. [2007] found traffic police have PAH exposures that exceeded national- and WHO-recommended exposure guidelines.

Other chemical agents that officers may potentially be exposed to include those associated with clandestine methamphetamine manufacturing. In 2004, there were 17, 033 methamphetamine lab-related seizures by US law enforcement agencies [Mitka, 2005]. Some chemicals involved in these activities are carcinogenic (e.g., benzene, methylene chloride). Although the frequency or extent of exposure to these agents among most law enforcement officers is not anticipated to be extensive, particularly with appropriate training and safety precautions, potential exposure to these agents represents a growing concern within this occupation [Thrasher et al., 2009].

Radar

Reports that police officers rest active radar guns against their lap, chest, or other body parts while not in use led to the hypothesis that irradiation of localized tissues may increase risks for leukemia, and testicular, brain, eye, or skin cancer [Lotz et al., 1995; Breckenkamp et al.,

2003; van Netten et al., 2003]. Traffic radar devices emit microwave radiation (10.5–36.0 GHz) that can enhance tumor growth by: changing ion flux across cellular membranes; reducing the activity of DNA repair enzymes; modifying gene expression (e.g., oncogenes); increasing cell proliferation rates; disrupting immune or endocrine function thus promoting tumor growth, or by enhancing the absorption of carcinogenic substances into cells [Lyle et al., 1983; Neubauer et al., 1990; Stuchly, 1998; Verschaeve and Maes, 1998; Akdag et al., 1999; French et al., 2001; Krewski et al., 2001].

Shift Work and Sleep Disruption

Police officers are usually required to work irregular hours, including nights, which can elicit circadian rhythm dysregulation and sleep insufficiency [Gordon et al., 1986; Akerstedt, 1998; Shields, 2002; Burch et al., 2005; Harada et al., 2005; Eriksen and Kecklund, 2007; Burch et al., 2009]. For example, up to 90% of police were tired upon awakening, 72% struggled with sleepiness during the daytime, and nearly 60% reported suboptimal sleep duration the previous night (<6.5 hr) [Charles et al., 2007a]. Neuroendocrine or immune system dysregulation may serve as a biologically plausible mechanism whereby shiftwork or sleep disruption may foster tumor development [Akerstedt and Nilsson, 2003; Youngstedt and Kripke, 2004; Burch et al., 2005, 2007; Grandner and Drummond, 2007; Blask, 2009]. The epidemiologic evidence examining relationships between sleep disorders and cancer risk is limited and inconsistent, despite a compelling literature linking sleep disruption with inflammation and other pathways that facilitate carcinogenesis. For example, either long (≥8 hr) or short duration sleep (≤6 hr) has been associated with increased breast cancer risks in several large cohort studies [Pinheiro et al., 2006; Kakizaki et al., 2008; Wu et al., 2008]. It is possible that both long and short duration sleep share common underlying pathophysiological processes, although this issue remains to be elucidated. In contrast, the relationship between shiftwork and cancer risk is more compelling. Shift work has been linked with increased risks for prostate, breast, and colorectal cancer, as well as non-Hodgkin's lymphoma, and the International Agency for Research on Cancer (IARC) concluded that shift work is a (Group 2A) probable human carcinogen [IARC, 2010]. Several recent studies that were published after the IARC review support and extend these observations [Lie et al., 2011; Hansen and Stevens, 2012; Knutsson et al., 2012; Menegaux et al., 2012].

Stress and Coping

Stress can elicit significant pathophysiological effects and perpetuate inappropriate coping behaviors or lifestyles that predispose individuals to increased cancer risk [Juster et al., 2010]. Common stressors among police officers include: the need to make immediate and sometimes life and death decisions, excessive work hours, fatigue, and abruptly changing duties from mundane to demanding tasks [Brown et al., 1996; Berg et al., 2005]. Severe stressors among Norwegian police included inadequate support from supervisors, serious incidents (murder or vehicular accidents), handling seriously injured people, injuries to fellow officers, and personal injury while on duty [Berg et al., 2005]. When the brief symptom inventory (BSI) was used to examine self-reported police stress, an elevated mean global severity index score (GSI, 0.42) was observed relative to normative values (0.30), indicating greater overall psychological distress among officers compared to a control population [Andrew et al., 2008]. Based on data acquired from the general health questionnaire (GHQ) [Goldberg and Blackwell, 1970], more than 40% of officers were considered a psychologically distressed "case" [Collins and Gibbs, 2003], whereas only 23% of medical inpatients met this definition [Maguire et al., 1974]. Stress can activate several biological processes that enhance tumor development, including pro-inflammatory cytokine secretion, immune system dysregulation, and alterations in the tumor microenvironment [Chida et al., 2008; Wirth et al., 2011].

Stress among police can be accompanied by either healthy (e.g., increased exercise or sleep) or unhealthy coping strategies (poor diet, alcohol abuse). Smoking and excessive alcohol consumption both have been associated with increased cancer risks [Toh et al., 2010]. Results from the National Health Interview and other surveys indicate that US police officers have a prevalence of smoking (~14–39%) similar to the average among all US adult men (30.1%) [Nelson et al., 1994; Franke et al., 1997; Joseph et al., 2009; Violanti et al., 2009b]. Nonetheless, improved stress reduction and smoking cessation efforts can reduce tobacco-related cancer morbidity and mortality among police officers. The largest obstacles to smoking cessation among police officers were loss of stress relief (55%) and loss of smoking enjoyment (50%) [Reichert et al., 2006].

The link between stress and alcohol abuse has been established, and approximately 25% of police officers have been characterized as alcohol dependent [Kroes, 1985; Fenlon et al., 1997; Kohan and O'Connor, 2002]. In one study, only 22% of police officers refrained from alcohol consumption, and nearly 15% consumed more than seven drinks per week [Violanti et al., 2009a]. In another study, 52% of male and 29% of female officers drank alcohol at least once a week [Zukauskas et al., 2009], and 48% and 40% of male and female officers, respectively, drank excessive (weekly consumption exceeding eight drinks for men and six for women in a row, two or more times a month) amounts of alcohol [Meyers and Perrine, 1996; Richmond et al., 1998]. Using a similar excessive drinking definition, CDC's Behavioral Risk Factor Surveillance Survey found that only 17.1% of US adults are considered excessive drinkers [Kanny, 2012]. In addition to work-related stress, a factor frequently cited as contributing to elevated alcohol use among police is the workplace culture [Dietrich and Smith, 1986; OHSC, 1992; Fenlon et al., 1997]. Increased alcohol consumption has been associated with esophageal, hepatocellular, rectal, and breast cancer [Mukamal and Rimm, 2008], and an increased incidence or mortality associated with these cancers has been observed among some studies of police officers (Table I).

Police officers tend to report increased food consumption, a high fat diet, and decreased physical activity in response to high levels of occupational stress [Grencik, 1973; Violanti et al., 1986]. The average BMI among male and female police officers in Buffalo, NY was 29.2 ± 3.9 and was 26.1 ± 4.6, respectively [Charles et al., 2007b]. These values are higher than adult men over the age of 20 in the general US population (28.4 ± 0.14), but lower than the average for women (28.4 ± 0.19) [McDowell et al., 2008]. Among nine Mid-western states in the US, nearly 83% of police officers were overweight (BMI > 25) [Ramey et al., 2004], and similar findings were reported among retired police officers from Milwaukee (85% with a BMI > 25) [Ramey et al., 2009], which is much greater than the percentage of US adults who are overweight or obese (66%) [Ogden et al., 2006]. One study found that 25% of police officers had reduced leisure-time physical activity in 1996 compared to their physical activity in 1981 [Sorensen et al., 2000]. Similarly, 38% of officers self-reported regular exercise in 1981, whereas only 23% reported regular exercise in 1993 [Franke et al., 1997]. In another study, 9% of officers claimed they rarely exercised, 38% exercised 1–2 days a week, 32% exercised 3–4 days a week, and only 21% exercised 5 or more days a week [Richmond et al., 1998]. These proportions are lower than the prevalence of regular physical activity (i.e., at least 30 min of moderate intensity physical activity most days of the week) among women (47%) and men (50%) in the general US population [MMWR, 2008]. Reduced physical activity has been associated with several cancers that are elevated among police officers, including colon cancer [Friedenreich et al., 2010].

CONCLUSION

Police work is associated with exposure to a variety of carcinogenic agents, lifestyles, or risk factors. Available epidemiologic evidence suggests that police officers may be subject to

increased overall risks for cancer incidence and mortality, particularly Hodgkin's lymphoma, melanoma, and bladder or testicular cancer. Studies of cancer among police have been subject to imprecise exposure assessment, the healthy worker effect or other forms of selection bias, and a lack of characterization of confounding, among other limitations. Little is known of how multiple carcinogenic exposures combine or interact to elicit changes in cancer incidence or mortality. Despite the limitations, an ample literature describes biologically plausible pathways that can predispose or potentiate cancer risks among police officers. There is a clear need to evaluate these impacts in this understudied occupation.

The law enforcement workplace is dynamic, and effective programs for disease or injury prevention require ongoing monitoring of hazards and health in order to assure the adequacy of disease prevention strategies. To optimize prevention efforts, potential carcinogenicity among police can be addressed more effectively by characterizing and controlling exposures or practices that elicit increased risks, and by monitoring biological perturbations resulting from those activities. Future interventions should focus on the most common cancers, or cancer sites most strongly attributable to police work in order to prevent or ameliorate their impacts.

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TABLE I

Cancer Studies Among Police Officers by Cancer Site

Refs.	Study dates	Study design	Total population	Observed deaths or cases	Effect measure: risk estimate (95% CI)
All cancer					
Vena et al. [1986]	1950–1979	Cohort	2, 376	150	SMR:1.27 (1.08–1.49)
Violanti et al. [1998]	1950–1990		2, 593	247	SMR:1.25 (1.10–1.41)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	291	SMR: 0.95(0.81–1.11)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	557	SMR:1.02(0.89–1.10)
Finkelstein [1998]	1964–1995	Cohort	22, 197	561	SIR: 0.90(0.83–0.98)[a]
Feuerand Rosenman [1986]	1974–1980	Proportionate mortality study	901	129	PMR:1.11
van Netten et al. [2003]	1963–2002	Case report	174	6	SMR: 2.30[b, c]
Gu et al. [2011]	1976–2006	Cohort	2, 234	406	SIR: 0.94 (0.85–1.03)
Prostate					
Vena et al. [1986]	1950–1979	Cohort	2, 376	6	SMR: 0.64 (0.24–1.40)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	12	SMR: 0.72 (0.37–1.26)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	30	SMR:1.02 (0.51–1.82)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	7	SMR: 0.77(0.31–1.50)
Finkelstein [1998]	1964–1995	Cohort	22, 197	85	SIR:1.16 (0.93–1.43)[a]
Zeegers et al. [2004]	1986–1993	Cohort	58, 279	693 650 695	RR: everpolice1.62 (0.62–4.27)[d] RR: longest profession 3.91 (1.14–13.42)[e] RR: baseline police 4.00 (1.19–13.37)[f]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	129	OR:1.3 (1.00–1.50)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	104	SIR: 0.88 (0.72–1.07)
Lung and respiratory tract					
Vena et al. [1986]	1950–1979	Cohort	2, 376	42	SMR:1.14 (0.82–1.54)
Violanti et al. [1998]	1950–1990		2, 593	82	SMR:1.23 (0.97–1.52)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	30	SMR: 0.92(0.69–1.19)
Rosenstock et al. [1990]	1945–1984	Cohort	6, 466	95	SMR:1.09(0.72 –1.26)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	82	SMR:1.05(0.84–1.30)
Finkelstein [1998]	1964–1995	Cohort	22, 197	77	SIR: 0.66(0.52–0.82)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	101	OR: 0.90(0.70–1.10)[g]

Refs.	Study dates	Study design	Total population	Observed deaths or cases	Effect measure: risk estimate (95% CI)
Feuerand Rosenman [1986]	1974–1980	Proportionate mortality study	901	43	PMR: 0.95
Gu et al. [2011]	1976–2006	Cohort	2, 234	83	SIR: 0.97 (0.77–1.20)
Colon					
Vena et al. [1986]	1950–1979	Cohort	2, 376	20	SMR:1.80 (1.10–2.79)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	35	SMR:1.87 (1.29–2.59)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	24	SMR: 0.50 (0.22–0.99)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	16	SMR:1.47 (0.84–2.3)
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	32	OR:1.10 (0.90–1.50)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	29	SIR: 0.80 (0.53–1.14)
Rectum					
Vena et al. [1986]	1950–1979	Cohort	2, 376	6	SMR:1.40 (0.51–3.04)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	8	SMR:1.41 (0.61–2.79)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	8	SMR:1.11 (0.36–2.59)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	7	SMR:1.07 (0.43–2.20)
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	38	OR:1.30 (0.90–1.80)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	14	SIR:1.22 (0.67–2.05)
Leukemia					
Violanti et al. [1998]	1950–1990	Cohort	2, 593	11	SMR:1.45 (0.72–2.58)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	15	SMR:1.56 (0.78–2.80)
Morton and Marjanovic [1984]	1964–1977	Cohort	1, 678	4	SIR: 2.60[b]
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	5	SMR: 0.67 (0.22–1.50)
Finkelstein [1998]	1964–1995	Cohort	22, 197	12	SIR: 0.60 (0.31–1.05)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	18	OR:1.10 (0.70–1.80)[g]
Feuerand Rosenman [1986]	1974–1980	Proportionate mortality study	901	3	PMR: 0.63
Gu et al. [2011]	1976–2006	Cohort	2, 234	15	SIR:1.21 (0.68–2.00)
All Lymphatic cancer					
Vena et al. [1986]	1950–1979	Cohort	2, 376	15	SMR:1.32 (0.74–2.19)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	23	SMR:1.24 (0.78–1.86)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	37	SMR:1.22 (0.75–1.86)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	3	SMR:1.02 (0.59–1.60)

Refs.	Study dates	Study design	Total population	Observed deaths or cases	Effect measure: risk estimate (95% CI)
Gu et al. [2011]	1976–2006	Cohort	2, 234	18	SIR:1.00 (0.59–1.58)
Hodgkin's lymphoma					
Violanti et al. [1998]	1950–1990	Cohort	2, 593	5	SMR: 3.13 (1.01–7.29)
Finkelstein [1998]	1964–1995	Cohort	22, 197	8	SIR: 0.84 (0.36–1.66)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	5	OR:1.20 (0.50–2.90)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	6	SIR: 3.34 (1.22–7.26)
Non-Hodgkin's lymphoma					
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	6	SMR:1.51 (0.55–0.32)
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	12	OR: 0.90(0.50–1.50)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	12	SIR: 0.74 (0.38–1.29)
All digestive cancer					
Vena et al. [1986]	1950–1979	Cohort	2, 376	56	SMR:1.57 (1.18–2.04)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	83	SMR:1.51 (1.20–1.87)
Finkelstein [1998]	1964–1995	Cohort	22, 197	130	SIR: 0.92(0.77–1.09)[a]
Feuerand Rosenman [1986]	1974–1980	Proportionate mortality study	901	43	PMR:1.58[b]
Gu et al. [2011]	1976–2006	Cohort	2, 234	89	SIR:1.01 (0.81–1.24)
Esophagus					
Vena et al. [1986]	1950–1979	Cohort	2, 376	8	SMR: 2.86 (1.23–5.64)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	10	SMR: 2.13 (1.01–3.91)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	2	SMR: 0.55 (0.07–1.90)
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	7	OR: 0.70 (0.30–1.60)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	8	SIR:1.39 (0.60–2.73)
Stomach					
Vena et al. [1986]	1950–1979	Cohort	2, 376	9	SMR:1.20 (0.55–2.29)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	24	SMR:1.09(0.70–1.60)
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	29	OR:1.10 (0.70–1.50)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	12	SIR:1.28 (0.66–2.24)
Liver					
Vena et al. [1986]	1950–1979	Cohort	2, 376	3	SMR:1.14 (0.82–1.54)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	5	SMR:1.27 (0.41–2.97)

Refs.	Study dates	Study design	Total population	Observed deaths or cases	Effect measure: risk estimate (95% CI)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	4	SMR:1.40 (0.38–3.59)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	12	SMR: 0.88 (0.45–1.50)
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	16	OR:1.20 (0.70–2.00)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	3	SIR: 0.82 (0.16–2.39)
Pancreas					
Vena et al. [1986]	1950–1979	Cohort	2, 376	9	SMR:1.35 (0.62–2.56)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	12	SMR:1.15 (0.59–2.01)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	7	SMR: 0.87 (0.35–1.70)
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	13	OR: 0.80 (0.50–1.40)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	9	SIR: 0.87 (0.40–1.66)
Brain					
Vena et al. [1986]	1950–1979	Cohort	2, 376	5	SMR:1.63(0.52–3.79)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	7	SMR:1.40 (0.56–2.88)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	22	SMR:1.36 (0.59–2.69)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	3	SMR: 0.52 (0.13–1.20)
Finkelstein [1998]	1964–1995	Cohort	22, 197	16	SIR: 0.84 (0.48–1.36)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	9	OR: 0.80 (0.40–1.60)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	9	SIR:1.61 (0.73–3.05)
Oral cavity					
Finkelstein [1998]	1964–1995	Cohort	22, 197	24	SIR: 0.71 (0.45–1.06)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	16	OR: 0.90(0.50–1.50)[g]
Buccal cavity and pharynx					
Vena et al. [1986]	1950–1979	Cohort	2, 376	7	SMR:1.87 (0.73–3.86)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	9	SMR:1.62(0.74–3.08)
Gu et al. [2011]	1976–2006	Cohort	2, 234	10	SIR: 0.66 (0.31–1.21)
Larynx					
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	5	SMR: 0.71 (0.23–1.60)
Finkelstein [1998]	1964–1995	Cohort	22, 197	13	SMR: 0.98 (0.52–1.68)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	8	OR: 0.80 (0.40–1.60)[g]
Thyroid					

Refs.	Study dates	Study design	Total population	Observed deaths or cases	Effect measure: risk estimate (95% CI)
Vena et al. [1986]	1950–1979	Cohort	2, 376	2	SMR:7.64 (0.86–27.59)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	2	SMR: 5.23 (0.59–18.80)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	3	SMR: 3.44 (0.71–10.00)
Finkelstein [1998]	1964–1995	Cohort	22, 197	3	SIR: 0.86 (0.32–1.87)[a]
Lope et al. [2005]	1971–1989	Cohort	1, 103	13	RR: 2.12 (1.23–3.66)
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	4	OR:1.10 (0.40–2.80)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	5	SIR:1.99(0.64–4.64)
Male breast					
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	2	SMR:14.36 (1.73–51.00)
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	1	OR: 0.80 (0.10–6.00)[g]
Bladder					
Vena et al. [1986]	1950–1979	Cohort	2, 376	6	SMR:1.47 (0.54–3.21)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	7	SMR:1.13 (0.45–2.33)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	2	SMR: 0.91 (0.25–2.34)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	13	SMR:1.27 (0.67–2.10)
Finkelstein [1998]	1964–1995	Cohort	22, 197	30	SIR: 0.93(0.63–1.33)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	50	OR:1.40 (1.00–1.90)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	20	SIR: 0.64 (0.39–0.99)
Kidney					
Vena et al. [1986]	1950–1979	Cohort	2, 376	5	SMR:1.75 (0.56–4.08)
Violanti et al. [1998]	1950–1990	Cohort	2, 593	10	SMR: 2.08(1.00–3.82)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	7	SMR:1.39 (0.56–2.80)
Finkelstein [1998]	1964–1995	Cohort	2, 234	23	SIR: 0.96 (0.61–1.44)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	19	OR:1.20 (0.70–1.90)[g]
Gu et al. [2011]	1976–2006	Cohort	2, 234	19	SIR:1.56 (0.94–2.43)
Testis					
Finkelstein [1998]	1964–1995	Cohort	22, 197	23	SIR:1.30 (0.89–1.84)[a]
Davis and Mostofi [1993]	1979–1991	Cohort	340	6	SMR: 6.90[b]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	15	OR: 0.90(0.50–1.60)[g]
Skin					

Refs.	Study dates	Study design	Total population	Observed deaths or cases	Effect measure: risk estimate (95% CI)
Demers et al. [1992b]	1945–1979	Cohort	4, 546	6	SMR: 0.94 (0.26–2.41)
Forastiere et al. [1994]	1972–1991	Cohort	3, 868	4	SMR: 2.34(0.64–6.0)
Finkelstein [1998]	1964–1995	Cohort	22, 197	41	SIR:1.45 (1.10–1.88)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	19	OR: 0.90(0.60–1.40)[g]
Feuerand Rosenman [1986]	1974–1980	Proportionate mortality study	901	7	PMR: 2.10[b]
Gu et al. [2011]	1976–2006	Cohort	2, 234	10	SIR: 0.54 (0.26–0.98)
Bone					
Finkelstein [1998]	1964–1995	Cohort	22, 197	2	SIR: 0.82 (0.10–3.00)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	2	OR:1.40 (0.30–5.60)[g]
Soft tissue					
Finkelstein [1998]	1964–1995	Cohort	22, 197	7	SIR:1.12 (0.45–2.31)[a]
Bouchardy et al. [2002]	1980–1993	Case–referent	58, 134	3	OR: 0.70 (0.20–2.30)[g]

CI, confidence interval; SMR, standardized mortality ratio; SIR, standardized incidence ratio; PMR, proportionate mortality ratio; OR, odds ratio; RR, relative risk.

Violanti et al. [1998] was an extension of Vena et al. [1986].

Demers et al. [1992b] used the same source population as Rosenstock et al. [1990].

[a] 90% confidence intervals.

[b] Significant at $P \leq 0.05$.

[c] P values based on one-sided significance tests.

[d] Ever worked as a police officer.

[e] Police officer as longest profession ever held.

[f] Police officer as occupation held at baseline.

[g] Case–referent method—cancers of interest were evaluated and all other cancer cases served as referents.

TABLE II

Potential Police Exposures and Associated Cancers

Hazardous exposure	Associated cancer site	Refs.
Radar emissions	Prostate, eye, brain, skin, testicular, leukemia	Zeegers et al. [2004], Breckenkamp et al. [2003], Lotz et al. [1995]
Sunlight (UV exposure)	Skin	Finkelstein [1998], Narayanan et al. [2010]
Shift work and circadian disruption	Prostate, breast, non-Hodgkin's lymphoma, colorectal	Kubo et al. [2006], Zhu et al. [2005], Schernhammer et al. [2001, 2003], Hoffman et al. [2009]
Obesity	Prostate, colorectal, kidney, endometrial, thyroid, breast, esophagus, leukemia, skin, adenocarcinoma, non-Hodgkin's lymphoma, multiple myelomas	Roberts et al. [2010]
Physical activity	Prostate, colon, endometrial, breast, lung	Friedenreich and Orenstein [2002], Fredriksson et al. [1989]
Tobacco use	Colon, bladder, esophagus, pancreas, kidney, oral cavity, lung, pharynx, larynx	Sasco et al. [2004], Ozlu and Bulbul [2005]
Alcohol consumption	Breast, colorectal, liver, larynx, oral cavity, pharynx, esophagus	Mukamal and Rimm [2008], Boffetta and Hashibe [2006]
Methamphetamine labs (trichloroethylene)	Breast, bladder, esophagus, kidney, liver, non-Hodgkin's lymphoma, all malignant neoplasms	Scott and Chiu [2006]
Air pollution, benzene, and particulate matter	Leukemia, non-Hodgkin's lymphoma, lung, kidney, liver	Wiwanitkit et al. [2005], Forastiere et al. [1994], Blair et al. [1993], Lewtas [2007]
Gun cleaning solvents	Hodgkin's lymphoma, leukemia	Violanti et al. [1998]
Stress and post-traumatic stress disorder	All malignant neoplasms	Chida et al. [2008]