



Nurses, Smoking, and Immunity: A Review

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KEY WORDS

*immune system
nurse
secondhand smoke
smoking
smoking cessation*

Nurses regularly are exposed to a variety of occupational hazards. In addition to documented occupational hazards, exposure to smoking remains a major concern. This article reviews the prevalence of smoking among nurses working in the United States and discusses their reasons for smoking. Researchers conducted a state-of-the-art review on the effects of cigarette smoking and exposure to secondhand smoke (SHS) on the immune system. Smoking prevalence among nurses working in the United States ranged from 7%–12%, and high work stress, poor work environment, shift work, and peer influence were suspected major risk factors influencing smoking behavior. A review of the effects of smoking on immunity revealed that both active smoking and exposure to SHS negatively affects immune function. When rehabilitation nurses stop smoking, their health improves and nonsmokers are exposed to less SHS. Rehabilitation nurses are encouraged to share knowledge of the immunological benefits of smoking cessation with patients to facilitate nurse-led rehabilitation programs.

Nursing is a uniquely hazardous occupation (National Institute for Occupational Safety and Health [NIOSH] Healthcare and Social Assistance Sector Council, 2009). Nurses confront a variety of biological, chemical, environmental, physical, and psychosocial hazards on a regular basis. The most common biological exposures occur when blood-borne pathogens are transmitted via needle-stick injuries (Beltrami, Williams, Shapiro, & Chamberland, 2000). Exposure to avian influenza virus, severe acute respiratory syndrome virus, and other airborne pathogens is expected while performing nursing duties (Ramsay et al., 2006). Chemical agents related to patient treatment such as antineoplastic drugs and anesthetics and substances including disinfectants, sterilants, and latex pose significant threats (Rogers, 1997). Carcinogenic chemicals such as formaldehyde and ethylene oxide commonly are encountered in clinical settings (Vecchio, Sasco, & Cann, 2003). Work-related musculoskeletal disorders are typical physical hazards affecting the nursing workforce (de Castro, 2004). Environmental and psychosocial hazards created by a poor work environment (e.g., extended work hours and shifts, high work demands due to understaffing, workplace violence, and interpersonal conflicts) can result in burnout, depression, job turnover, and sick leave, ultimately forcing nurses to leave the profession (NIOSH Healthcare and Social Assistance Sector Council, 2009).

Many occupational hazards are known to damage the immune system, which is intended to protect the body against cancer, infections, and immune-related diseases. If the immune system is not well-maintained or if it malfunctions, disease may result. This article focuses on a modifiable behavior, smoking, which negatively influences immunity. Exposure to smoking—

coupled with the occupational hazards that are part of a nurse's daily working life—puts nurses at higher risk for poor immune system functioning.

Cigarette Smoking, Exposure to Secondhand Smoke, and Health

Although cigarette smoking has declined over the years, it remains the leading cause of premature death in the United States (Mokdad, Marks, Stroup, & Gerberding, 2004; U.S. Department of Health & Human Services, 2006). Between 2000 and 2004, cigarette smoking and exposure to secondhand smoke (SHS) annually caused 443,595 premature deaths, which resulted in an estimated \$193 billion in healthcare costs plus lost productivity (Centers for Disease Control and Prevention [CDC], 2008). Of the annual deaths linked to cigarette smoking, 160,848 were attributed to cancer, 128,497 to cardiovascular diseases, and 103,338 to respiratory diseases. Annual smoking-attributed mortality of adult nonsmokers exposed to SHS accounted for 3,400 deaths due to lung cancer and 46,000 due to coronary heart disease. Cigarette smoking also has been identified as a potential risk factor for mental illnesses such as depression, anxiety, affective disorders, and schizophrenia, and it is associated with an increased prevalence of these mental illnesses (Nakata et al., 2008; Van Dongen, 1999) and higher suicide rates (Hughes, 2008). In addition, cigarette smoking and SHS exposure have negative reproductive effects leading to reduced fertility in women, early menopause, low birth weight, fetal death, and pregnancy complications (Soares & Melo, 2008). Moreover, cigarette smoking is associated with a higher risk of absence from work (Lundborg, 2007) and occupational injuries and accidents (Nakata et al., 2006) as well as an

increased likelihood of common cold infections (Arcavi & Benowitz, 2004; Bensenor et al., 2001). Despite the negative consequences of cigarette smoking, nearly 1 in 5 American adults continues to smoke (CDC, 2009).

Prevalence of Smoking Among Nursing Professions

Cigarette smoking was a ubiquitous habit among American nurses a half century ago. In 1959 a large nationwide survey ($N = 9,498$) conducted by the American Cancer Society reported a 36% smoking prevalence in the nursing profession, and this prevalence continued through the 1960s (Garfinkel, 1976). In the 1970s, smoking among women who were registered nurses (RNs) rose to 38.9%. This rate was higher than among women in the U.S. general population (32%), and nearly twice as high as the smoking rate among physicians (21%; U.S. Department of Health & Human Services, 1980). According to reviews of smoking prevalence in the worldwide nursing profession, smoking among U.S. nurses started to decline during the 1980s (20%–33%) and fell to 14%–18% during the 1990s (Adriaanse, Van Reek, Zandbelt, & Evers, 1991; Smith & Leggat, 2007). Based on a series of National Health Interview Surveys conducted in the United States between 1974 and 1991, smoking prevalence had declined from 31.7% to 18.3% among RNs and from 37.1% to 27.2% among licensed practical nurses (LPNs; Nelson et al., 1994). Corresponding figures for physicians were 18.8% and 3.3%, indicating that cigarette smoking has declined more rapidly among physicians than nurses. Smoking prevalence among nurses now is at 7%–12% (Smith & Leggat, 2007), but it remains substantially higher than the rate among physicians, which declined to 1% in 2005 (Association of American Medical Colleges, 2007). A recent Nurses Health Study of smoking trends between 1976 and 2003 reported the percentage of current smokers dropped from 33.2% in 1976 to 13.5% in 1989, and declined further to 8.4% in 2002 and 2003 among RNs (Sarna et al., 2008). Although this trend is encouraging, readers should note that these figures mainly reflect RN and LPN behavior and not activity among nursing aides and assistants, among whom smoking prevalence is expected to be higher. A survey of Norwegian nursing aides reported that the prevalence of daily smoking was higher than 40% (Eriksen, 2006), which was much higher than the prevalence in the general Norwegian population in 2007 (21% for women and 21% for men; Statistics Norway, 2008).

Nurses as Role Models and Health Educators

Nurses are recognized as health-behavior educators and role models, and they are the largest group of healthcare professionals on the frontlines combating tobacco use (Schultz, 2003). If nurses continue to smoke, they may lose their credibility as primary instrumental partners in smoking reduction, especially among patients for whom quitting is a crucial part of their treatment (Becker et al., 1986). Consequently, occupational health strategies are needed to support smoking cessation and prevent nurses' SHS exposure.

Organizational Factors Related to Nurses' Smoking

To develop effective measures to eliminate or reduce smoking among nurses, it is important to understand why nurses smoke. Three organizational factors influence smoking behavior, smoking relapse, and continuing smoking among nurses (Rowe & Clark, 2000; Sarna, Bialous, Wewers, Froelicher, & Danao, 2005).

Work stress and poor work environment have been identified as factors that diminish quit attempts and increase smoking intensity (McKenna et al., 2003). In the Nurses' Health Study, nurses reporting severe work stress had nearly twice (11%) the rate of heavy smoking (25+ cigarettes/day) than those who reported minimal (6%) or light (6%) stress (Feskanich et al., 2002). Similarly, in a study of 218 Hungarian female nurses, heavy smoking (21+ cigarettes/day) was more prevalent among those perceiving their stress as high (10.8%) compared to those with medium (2.9%) or low (2.8%) stress levels (Piko, 1999). A study of Norwegian nursing aides revealed that smoking relapse occurred twice as often among those experiencing frequent threats and violence at work and those perceiving a poor work climate (described as nonsupportive, distrustful, and tense) after 15 months of follow-up (Eriksen, 2006). These examples suggest that nurses experiencing high stress or poor work environments may increase their cigarette consumption to cope with daily stress and find it difficult to quit smoking if such a burden continues (Hall, Munoz, Reus, & Sees, 1993). Consequently, smoking cessation programs for nurses need to include stress-management strategies (Albertsen, Borg, & Oldenburg, 2006).

Evidence supports the assumption that shift work contributes to increased smoking intensity and smoking initiation. A 2-year prospective study of 12,140 employees suggested that shift workers consumed more cigarettes and were more likely to commence smoking than day workers (van Amelsvoort, Jansen,

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& Kant, 2006). A review based on 17 studies of work schedule and health habits concluded that shift work can lead to adverse lifestyle outcomes such as poor dietary intake, smoking, and becoming overweight (Zhao & Turner, 2008). A nationally representative survey of 3,917 employed RNs in the United States reported that nurses working night shifts had a significantly higher prevalence of smoking (20.5%) than day workers (13.9%), and nurses working night shifts longer than 8 hours had a 62% increased likelihood of smoking than those working day shifts of 8 or fewer hours (Trinkoff & Storr, 1998). A combination of smoking and shift work could affect the development of poor health because shift work itself is a “probable carcinogenic agent” (Straif et al., 2007, p. 1065). Because many healthcare workers, including nurses, perform shift work and work extended schedules (Wilson, 2002), shift work may be a significant factor that aggravates nurses’ smoking behavior.

Peer and social interactions also influence smoking behavior. Wagner (1985) reported that 43% of student nurses started their smoking habits during their training. Among these students, 71% said they commenced smoking because their peers smoked. After they establish smoking habits, psychological bonds among smoking peers may prevent them from quitting (Sarna et al., 2005). Smoking peers may share common distress experiences at work that tighten their bond (Rowe & Clark, 2000; Sarna et al., 2005). Nurses who smoke and who are vulnerable to work stress may need psychological support to reduce their smoking intensity. Stress management

and work environment improvements (such as increasing social support and respect among coworkers, reducing physical and mental abuse by patients and physicians, creating a better work climate, and developing leadership) are needed to reduce smoking among nurses.

Cigarette Smoking and Immunity

Research that focuses on smoking among nurses rarely addresses the ways in which smoking deteriorates health. Nurses may become more motivated to quit smoking or change their attitude about smoking if they have more knowledge about the health risks. This knowledge also will contribute to their role as health educators for patients who smoke. Smoking is known to promote chronic illness by altering immune function. Smoking affects function and quantity of immune components in a dynamic way (Sopori, 2002). **Table 1** features a list of blood immune markers in smoking-immune studies and the major roles of these components.

As shown in **Table 2**, smoking elevates white blood cell counts. On average, smoking results in 30% higher white blood cell counts (Arcavi & Benowitz, 2004). Increased white blood cell counts are a sign of systemic inflammation, which in turn has been identified as a powerful predictor of cardiovascular disease mortality (Margolis et al., 2005) and cancer mortality (Shankar et al., 2006) as evidenced by several large-scale prospective studies.

Among lymphocytes, smoking selectively increases helper T (CD4+) cells, especially memory T

Table 1. Major Roles of Immune Markers Used in Smoking-Immune Studies in Humans

Immune Markers	Major Roles
Neutrophils	Destroy antigens during an acute inflammatory response.
Monocytes	Digest dead or damaged cells and provide immunological defence against antigens. Monocytes migrate into tissues and develop into macrophages.
Lymphocytes	Kill antigens and produce antibodies. Main lymphocytes are T, B, and NK cells.
Natural killer (NK) cells	Kill certain tumor and virus-infected cells.
B cells	Antibody (immunoglobulin, gammaglobulin) production.
T cells	Directly attack foreign antigens and regulate the immune system.
Cytotoxic T (CD8+) cells	Lysis of virus-infected cells, tumor cells, or allografted cells.
Helper T (CD4+) cells	Facilitate B-cell proliferation and differentiation, immunoglobulin synthesis, assist cytotoxic T cells attacking antigens.
Memory T (CD4+CD45RO+) cells	Subset of helper T cells that respond to previously encountered antigens. This cell can reproduce to mount a faster and stronger immune response than the first time the immune system responded to the antigens.
Naive T (CD4+CD45RA+) cells	Subset of helper T cells that have not yet encountered antigens. This cell responds to the newly encountered antigens and will turn into a reservoir of memory T cells.
Immunoglobulin G, A, M (IgG, IgA, IgM)	Neutralize bacteria, viruses, and other environmental pathogens.
Immunoglobulin E (IgE)	Protect against parasite invasion. Responsible for allergic reactions.

Table 2. Summary of the Effects of Cigarette Smoking, Smoking Cessation, and Exposure to Secondhand Smoke (SHS) on Peripheral Blood Immune Markers^a

Immune Markers	Effect Direction and Strength ^f			
	Current Smoking ^b	Smoking Cessation ^c	Exposure to SHS ^d	
			Adults	Nonadults ^e
Quantitative (numerical) markers				
Leukocytes (cells/mm ³)	↑↑		↑	
Neutrophils	↑↑			↓
Monocytes	↑↑			
Lymphocytes	↑↑		↑	
NK (CD56+/CD16+/CD57+) cells	↓↓		↑	
B (CD19+) cells	↑→		↑	
Total T (CD3+) cells	↑↑		↑	
Cytotoxic T (CD8+) cells	↑			
Helper T (CD4+) cells	↑↑	↓	↑	
Memory T (CD4+CD45RO+) cells	↑↑↑		↑↑	
Naive T (CD4+CD45RA+) cells	↑↑		↑↑	
Immunoglobulins				
Immunoglobulin G (IgG)	↓↓	↑↑		
Immunoglobulin A (IgA)	↓	↑		
Immunoglobulin M (IgM)	↓	↑↑		
Immunoglobulin E (IgE)	↑↑↑		↑→	↑↑
Qualitative (functional) markers				
Natural killer cell activity (NKCA)	↓↓↓	↑↑	↑	
Lymphocyte proliferation (against mitogens)	↓		→	↑
CD4+ to CD8+ (CD4+/CD8+) ratio	↑			

^aResults shown in the table are based on the effects on peripheral blood immune parameters. Results may differ if not measured in the peripheral blood (i.e., bronchoalveolar fluid, sputum, etc.); ^bCompared to lifetime nonsmokers; ^cSmoking cessation effects on immunity observed within 1-6 months of cessation; ^dExposure to SHS in lifetime nonsmokers; ^eIncluding neonates, infants, and children; ^fEffect direction and strength: ↑(↓) weak-to-moderate increase (decrease), ↑↑(↓↓) moderate-to-strong increase (decrease), ↑↑↑(↓↓↓) strong-to-very-strong increase (decrease), → no change, ↑ → enhanced or no change.

(CD4+CD45RO+) cells (Chavance, Perrot, & Annesi, 1993; Nakata et al., 2007), but the function of these cells is greatly reduced (Sopori, 2002). Memory T cells facilitate B cell proliferation and differentiation and immunoglobulin synthesis. Immunoglobulins (Igs) such as G, A, and M are produced by plasma B cells that neutralize bacteria, viruses, and other environmental pathogens. An excess proliferation of memory T cells stimulated by repeated exposure to tobacco particulates accelerates cellular aging, leading to impaired responses to new and previously encountered antigens (Schroder & Rink, 2003). Consequently, production of antibodies (IgG, IgA, IgM) by B cells is reduced in cigarette smokers (Arcavi & Benowitz, 2004). Aging of the lymphocytes coincides with the

fact that cumulative exposure to smoking accelerates telomere shortening in circulating lymphocytes (Morla et al., 2006).

In addition to the proaging effect of smoking on T cells, evidence indicates an increase of memory T cells is associated with subclinical atherosclerosis. In a study of healthy men age 60–75, there was a positive relationship between the number of circulating memory T cells and mean intima-media thickness of the common carotid artery (Tanigawa et al., 2003). Cumulative lifelong exposure to smoking as represented by a combination of duration and intensity of smoking (i.e., pack years) had a strong association ($r > .439$) with increases of memory T cells (Nakata et al., 2007). It has been reported that atherosclerotic

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lesions show an accumulation of T lymphocytes, and that the majority of these cells are memory T cells (Stemme, Holm, & Hansson, 1992). Collectively, these observations suggest that increased memory T cells potentially mirror the extent of damage created in atherosclerotic lesions.

In contrast to decreases of IgG, IgA, and IgM, smoking increases the level of IgE (Oryszczyn et al., 2000). IgE plays a role in protecting against parasite invasion and is a major factor in the body's allergic response. Elevation of the IgE level by smoking could be a consequence of an allergic response to tobacco particulates.

Smoking inhibits both the function and quantity of natural killer (NK) cells (Mehta, Nazzal, & Sadikot, 2008; Tollerud et al., 1989). Suppressed NK cells cannot effectively kill tumor cells and virus-infected cells, leading to an increased risk for developing cancer and infection. An 11-year prospective study of 3,625 residents of Japan revealed that subjects grouped into the lower third of NK cell activity (NKCA) had respectively 1.59 and 1.69 times higher risk of cancer incidence compared to subjects with upper and medium thirds of NKCA levels (Imai, Matsuyama, Miyake, Suga, & Nakachi, 2000). A review of cigarette smoking and infection concluded that cigarette smokers are at higher risk for infection than nonsmokers because of impaired NK cell function (Arcavi & Benowitz, 2004). Exposure to SHS among nonsmokers also has been associated with an increased risk of infection. In a study of healthcare workers, women who were lifetime nonsmokers but who were passively exposed to cigarette smoke had a 1.3 times higher risk of frequent colds than their unexposed counterparts (Bensenor et al., 2001).

Exposure to SHS and Immunity

Many chemical components of cigarette smoke, including ammonia, benzene, nicotine, and carbon monoxide, exist in sidestream smoke in higher concentrations than in mainstream smoke (Brownson, Eriksen, Davis, & Warner, 1997). *Mainstream smoke* is the inhaled and exhaled smoke created from taking a puff on a lit cigarette; *sidestream smoke* is the smoke emitted from the end of a smoldering cigarette. Sidestream smoke is known as the major component of SHS exposure. The effect of exposure to SHS on immune function generally has been reported to be smaller than that of active smoking, but it produces a significant negative impact on immunity in adults (Johnson, Houchens, Kluwe, Craig, & Fisher, 1990). For example, in a study of 670 healthy workers, lifetime nonsmokers chronically exposed to a high level of SHS exhibited 30%–40% higher counts of memory and naïve T cells compared with their nonexposed

counterparts but expressed 20%–30% lower counts of those lymphocytes than current smokers (Nakata, Tanigawa, Araki, Sakurai, & Iso, 2004). Similarly, a marginal elevation of total IgE level was found in nonsmoking passive smokers compared with nonpassive women smokers who did and did not have asthma, but current smokers exhibited higher levels of IgE than any other nonsmoking subgroups (Oryszczyn et al., 2000).

Another important issue related to SHS exposure is the effects on the immune systems of nonadults such as fetuses, neonates, infants, and children. Early exposure to cigarette smoking has been reported to disturb normal development of the immune system (Prescott, 2008). Maternal smoking during pregnancy has been associated with higher total IgE levels (Magnusson, 1986), decreased NKCA (Castellazzi et al., 1999), increased lymphoproliferation (Devereux, Barker, & Seaton, 2002), reduced type 1 helper-T cell response to polyclonal stimulation (Noakes, Holt, & Prescott, 2003), and lower counts of neutrophils in cord blood (Merzelina-Roumans, Breukers, Ubachs, & van Wersch, 1996). In a study of 9-year-old Italian children, boys who had parents who smoked had significantly higher levels of total IgE and eosinophil counts than boys with nonsmoking parents (Ronchetti et al., 1990). These immunological alterations may explain the higher frequency of asthma and increased susceptibility to infections and cancer in children of smokers (Tebow et al., 2008). Smokers should be encouraged to keep in mind that nonsmokers exposed to SHS include vulnerable children who are put at risk for smoking-attributable diseases.

Although exposure to SHS likely impairs aspects of immune function, more studies are needed to reach conclusions because most studies are based on lower levels of evidence, such as cross-sectional study designs.

Smoking Cessation and Immunity

Smoking cessation can help smokers regain immune function. In addition, cessation may be beneficial for nonsmokers whose immune function is altered by exposure to SHS. When smoking ceases, an increase in NKCA and immunoglobulin levels (IgG and IgM) can be observed within 1 to 6 months, followed by decreases in circulating CD4+ T and CD8+ T lymphocytes that approach nonsmokers' levels (Meliska, Stunkard, Gilbert, Jensen, & Martinko, 1995; Miller, Goldstein, Murphy, & Ginns, 1982). A study of young cigarette smokers (age 21–35) who quit smoking showed that NKCA recovery was detectable 31 days after cessation (Meliska et al.). In contrast, a prospective study of 6-month smoking cessation on immunity found a

small NKCA increase after cessation among quitters younger than age 65, but not among quitters who were at least 65 years old (Ioka et al., 2001). Immune function recovery through smoking cessation may be influenced by cumulative pack years of smoking before cessation and levels of SHS exposure after cessation (Nakata et al., 2004, 2007), as well as changes in other behavioral factors. Although the effects of smoking cessation on immune outcomes are limited in human subjects and require further evidence, smoking cessation appears to be beneficial to health through recovery of immune function.

Clinical Implications for Rehabilitation Nursing

Smoking cessation appears to be the most effective intervention or treatment to reduce mortality in patients with primary and secondary stroke (Kawachi et al., 1995) and coronary artery disease (Wilhelmsen, 1998). Because nurse-led rehabilitation programs are effective in improving smoking behavior of patients with these conditions (McHugh et al., 2001; Michael & Shaughnessy, 2006), rehabilitation nurses are encouraged to share knowledge of the immunological benefits of smoking cessation with patients who continue to smoke. Providing feedback about smokers' biomarkers also may be a useful method with which to motivate or reinforce attempts to quit (McClure, 2001). In addition to classical biomarkers of smoking such as carbon monoxide, serum and urinary cotinine, and pulmonary function levels, immunological indicators may help improve motivation to quit smoking and prevent relapses in smoking behavior.

Conclusions

Smoking among nurses remains prevalent not only in the United States but around the world. High levels of work stress caused by high physical and psychological job demands and poor work environment characterized by a lack of social support and communication, poor work climate and leadership, frequent physical and mental abuse, demanding work schedules, and peer influence are major organizational risk factors influencing nurses' smoking behavior. Studies have shown that smoking is a strong behavioral factor that disturbs normal immune functioning and leads to the development of cancer, heart disease, stroke, and respiratory diseases. Exposure to SHS also may disrupt immune function, increasing the risk of smoking-attributable diseases. Smoking cessation among rehabilitation nurses is strongly recommended not only to improve the health of smoking nurses but to

Key Practice Points

1. Although the prevalence of cigarette smoking has declined over the years among nursing professionals, approximately 1 in 10 U.S. nurses still smoke.
2. Among nurses, work stress, poor work environment, shift work, and peer and social influences have been identified as key organizational factors that contribute to smoking intensity and initiation of smoking.
3. Both active smoking and exposure to secondhand smoke are detrimental to immune functioning among adults, infants, and neonates.
4. Smoking cessation in rehabilitation nurses is strongly recommended to improve their health, prevent the health decline of nonsmoking patients exposed to secondhand smoke, and provide positive influences for smoking patients.

prevent worsening the health of nonsmokers exposed to SHS. In addition, by not smoking, nurses fulfill their function as role models and health educators and are better able to positively influence patients in rehabilitation programs who are smokers.

Acknowledgments

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of NIOSH.

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References

- Adriaanse, H., Van Reek, J., Zandbelt, L., & Evers, G. (1991). Nurses' smoking worldwide. A review of 73 surveys on nurses' tobacco consumption in 21 countries in the period 1959–1988. *International Journal of Nursing Studies*, 28(4), 361–375.
- Albertsen, K., Borg, V., & Oldenburg, B. (2006). A systematic review of the impact of work environment on smoking cessation, relapse and amount smoked. *Preventive Medicine*, 43(4), 291–305.

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- Arcavi, L., & Benowitz, N. L. (2004). Cigarette smoking and infection. *Archives of Internal Medicine*, 164(20), 2206–2216.
- Association of American Medical Colleges. (2007). *Physician behavior and practice patterns related to smoking cessation*. A report prepared for the American Legacy Foundation. Retrieved June 1, 2010, from www.aamc.org/workforce/smoking-cessation-summary.pdf.
- Becker, D. M., Myers, A. H., Sacci, M., Weida, S., Swank, R., Levine, D. M., et al. (1986). Smoking behavior and attitudes toward smoking among hospital nurses. *American Journal of Public Health*, 76(12), 1449–1451.
- Beltrami, E. M., Williams, I. T., Shapiro, C. N., & Chamberland, M. E. (2000). Risk and management of blood-borne infections in health care workers. *Clinical Microbiology Reviews*, 13(3), 385–407.
- Bensenor, I. M., Cook, N. R., Lee, I. M., Chown, M. J., Hennekens, C. H., Buring, J. E., et al. (2001). Active and passive smoking and risk of colds in women. *Annals of Epidemiology*, 11(4), 225–231.
- Brownson, R. C., Eriksen, M. P., Davis, R. M., & Warner, K. E. (1997). Environmental tobacco smoke: Health effects and policies to reduce exposure. *Annual Review of Public Health*, 18, 163–185.
- Castellazzi, A. M., Maccario, R., Moretta, A., De Amici, M., Gasparoni, A., Chirico, G., et al. (1999). Effect of active and passive smoking during pregnancy on natural killer-cell activity in infants. *Journal of Allergy and Clinical Immunology*, 103(1, Pt. 1), 172–173.
- Centers for Disease Control and Prevention. (2008). Smoking-attributable mortality, years of potential life lost, and productivity losses—United States, 2000–2004. *Morbidity and Mortality Weekly Report*, 57(45), 1226–1228.
- Centers for Disease Control and Prevention. (2009). State-specific prevalence and trends in adult cigarette smoking—United States, 1998–2007. *Morbidity and Mortality Weekly Report*, 58(9), 221–226.
- Chavance, M., Perrot, J. Y., & Annesi, I. (1993). Smoking, CD45RO+ (memory), and CD45RA+ (naive) CD4+ T cells. *American Review of Respiratory Disease*, 148(1), 237–240.
- de Castro, A. B. (2004). Handle with care: The American Nurses Association's campaign to address work-related musculoskeletal disorders. *Online Journal of Issues in Nursing*, 9(3), 3.
- Devereux, G., Barker, R. N., & Seaton, A. (2002). Antenatal determinants of neonatal immune responses to allergens. *Clinical and Experimental Allergy*, 32(1), 43–50.
- Eriksen, W. (2006). Work factors as predictors of smoking relapse in nurses' aides. *International Archives of Occupational and Environmental Health*, 79(3), 244–250.
- Feskanich, D., Hastrup, J. L., Marshall, J. R., Colditz, G. A., Stampfer, M. J., Willett, W. C., et al. (2002). Stress and suicide in the Nurses' Health Study. *Journal of Epidemiology and Community Health*, 56(2), 95–98.
- Garfinkel, L. (1976). Cigarette smoking among physicians and other health professionals, 1959–1972. *CA: A Cancer Journal for Clinicians*, 26(6), 373–375.
- Hall, S. M., Munoz, R. F., Reus, V. I., & Sees, K. L. (1993). Nicotine, negative affect, and depression. *Journal of Consulting and Clinical Psychology*, 61(5), 761–767.
- Hughes, J. R. (2008). Smoking and suicide: A brief overview. *Drug and Alcohol Dependence*, 98(3), 169–178.
- Imai, K., Matsuyama, S., Miyake, S., Suga, K., & Nakachi, K. (2000). Natural cytotoxic activity of peripheral-blood lymphocytes and cancer incidence: An 11-year follow-up study of a general population. *Lancet*, 356(9244), 1795–1799.
- Ioka, A., Nakamura, M., Shirokawa, N., Kinoshita, T., Masui, S., Imai, K., et al. (2001). Natural killer activity and its changes among participants in a smoking cessation intervention program—A prospective pilot study of 6 months' duration. *Journal of Epidemiology*, 11(5), 238–242.
- Johnson, J. D., Houchens, D. P., Kluwe, W. M., Craig, D. K., & Fisher, G. L. (1990). Effects of mainstream and environmental tobacco smoke on the immune system in animals and humans—A review. *Critical Reviews in Toxicology*, 20(5), 369–395.
- Kawachi, I., Colditz, G. A., Stampfer, M. J., Willett, W. C., Manson, J. E., Speizer, F. E., et al. (1995). Prospective study of shift work and risk of coronary heart disease in women. *Circulation*, 92(11), 3178–3182.
- Lundborg, P. (2007). Does smoking increase sick leave? Evidence using register data on Swedish workers. *Tobacco Control*, 16(2), 114–118.
- Magnusson, C. G. (1986). Maternal smoking influences cord serum IgE and IgD levels and increases the risk for subsequent infant allergy. *Journal of Allergy and Clinical Immunology*, 78(5, Pt. 1), 898–904.
- Margolis, K. L., Manson, J. E., Greenland, P., Rodabough, R. J., Bray, P. F., Safford, M., et al. (2005). Leukocyte count as a predictor of cardiovascular events and mortality in postmenopausal women: The Women's Health Initiative Observational Study. *Archives of Internal Medicine*, 165(5), 500–508.
- McClure, J. B. (2001). Are biomarkers a useful aid in smoking cessation? A review and analysis of the literature. *Behavioral Medicine*, 27(1), 37–47.
- McHugh, F., Lindsay, G. M., Hanlon, P., Hutton, I., Brown, M. R., Morrison, C., et al. (2001). Nurse led shared care for patients on the waiting list for coronary artery bypass surgery: A randomised controlled trial. *Heart*, 86(3), 317–323.
- McKenna, H., Slater, P., McCance, T., Bunting, B., Spiers, A., & McElwee, G. (2003). The role of stress, peer influence and education levels on the smoking behaviour of nurses. *International Journal of Nursing Studies*, 40(4), 359–366.
- Mehta, H., Nazzari, K., & Sadikot, R. T. (2008). Cigarette smoking and innate immunity. *Inflammation Research*, 57(11), 497–503.
- Meliska, C. J., Stunkard, M. E., Gilbert, D. G., Jensen, R. A., & Martinko, J. M. (1995). Immune function in cigarette smokers who quit smoking for 31 days. *Journal of Allergy and Clinical Immunology*, 95(4), 901–910.
- Mercelina-Roumans, P. E., Breukers, R. B., Ubachs, J. M., & van Wersch, J. W. (1996). Hematological variables in cord blood of neonates of smoking and nonsmoking mothers. *Journal of Clinical Epidemiology*, 49(4), 449–454.
- Michael, K. M., & Shaughnessy, M. (2006). Stroke prevention and management in older adults. *Journal of Cardiovascular Nursing*, 21(5 Suppl. 1), S21–S26.
- Miller, L. G., Goldstein, G., Murphy, M., & Ginns, L. C. (1982). Reversible alterations in immunoregulatory T cells in smoking. Analysis by monoclonal antibodies and flow cytometry. *Chest*, 82(5), 526–529.
- Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual causes of death in the United States, 2000. *Journal of the American Medical Association*, 291(10), 1238–1245.
- Morla, M., Busquets, X., Pons, J., Saulea, J., MacNee, W., & Agustí, A. G. (2006). Telomere shortening in smokers with and without COPD. *European Respiratory Journal*, 27(3), 525–528.
- Nakata, A., Ikeda, T., Takahashi, M., Haratani, T., Hojou, M., Fujioka, Y., et al. (2006). Non-fatal occupational injury among active and passive smokers in small- and medium-scale manufacturing enterprises in Japan. *Social Science & Medicine*, 63(9), 2452–2463.
- Nakata, A., Takahashi, M., Ikeda, T., Hojou, M., Nigam, J. A., & Swanson, N. G. (2008). Active and passive smoking and depression among Japanese workers. *Preventive Medicine*, 46(5), 451–456.
- Nakata, A., Takahashi, M., Irie, M., Fujioka, Y., Haratani, T., & Araki, S. (2007). Relationship between cumulative effects of smoking and memory CD4+ T lymphocyte subpopulations. *Addictive Behaviors*, 32(7), 1526–1531.
- Nakata, A., Tanigawa, T., Araki, S., Sakurai, S., & Iso, H. (2004). Lymphocyte subpopulations among passive smokers. *Journal of the American Medical Association*, 291(14), 1699–1700.
- Nelson, D. E., Giovino, G. A., Emont, S. L., Brackbill, R., Cameron, L. L., Peddicord, J., et al. (1994). Trends in cigarette smoking among US physicians and nurses. *Journal of the American Medical Association*, 271(16), 1273–1275.

- National Institute for Occupational Safety and Health (NIOSH) Healthcare and Social Assistance Sector Council. (2009). State of the sector—Healthcare and social assistance: Identification of research opportunities for the next decade of NORA. Cincinnati, OH: NIOSH.
- Noakes, P. S., Holt, P. G., & Prescott, S. L. (2003). Maternal smoking in pregnancy alters neonatal cytokine responses. *Allergy*, 58(10), 1053–1058.
- Oryszczyn, M. P., Annesi-Maesano, I., Charpin, D., Paty, E., Maccario, J., & Kauffmann, F. (2000). Relationships of active and passive smoking to total IgE in adults of the Epidemiological Study of the Genetics and Environment of Asthma, Bronchial Hyperresponsiveness, and Atopy (EGEA). *American Journal of Respiratory and Critical Care Medicine*, 161(4, Pt. 1), 1241–1246.
- Piko, B. (1999). Work-related stress among nurses: A challenge for health care institutions. *Journal of the Royal Society for the Promotion of Health*, 119(3), 156–162.
- Prescott, S. L. (2008). Effects of early cigarette smoke exposure on early immune development and respiratory disease. *Pediatric Respiratory Reviews*, 9(1), 3–10.
- Ramsay, J., Denny, F., Szirotnyak, K., Thomas, J., Corneliussen, E., & Paxton, K. L. (2006). Identifying nursing hazards in the emergency department: A new approach to nursing job hazard analysis. *Journal of Safety Research*, 37(1), 63–74.
- Rogers, B. (1997). Health hazards in nursing and health care: An overview. *American Journal of Infection Control*, 25(3), 248–261.
- Ronchetti, R., Macri, F., Ciofetta, G., Indinnimeo, L., Cutrera, R., Bonci, E., et al. (1990). Increased serum IgE and increased prevalence of eosinophilia in 9-year-old children of smoking parents. *Journal of Allergy and Clinical Immunology*, 86(3), 400–407.
- Rowe, K., & Clark, J. M. (2000). Why nurses smoke: A review of the literature. *International Journal of Nursing Studies*, 37(2), 173–181.
- Sarna, L., Bialous, S. A., Jun, H. J., Wewers, M. E., Cooley, M. E., & Feskanich, D. (2008). Smoking trends in the Nurses' Health Study (1976–2003). *Nursing Research*, 57(6), 374–382.
- Sarna, L., Bialous, S. A., Wewers, M. E., Froelicher, E. S., & Danao, L. (2005). Nurses, smoking, and the workplace. *Research in Nursing & Health*, 28(1), 79–90.
- Schroder, A. K., & Rink, L. (2003). Neutrophil immunity of the elderly. *Mechanisms of Ageing and Development*, 124(4), 419–425.
- Schultz, A. S. (2003). Nursing and tobacco reduction: A review of the literature. *International Journal of Nursing Studies*, 40(6), 571–586.
- Shankar, A., Wang, J. J., Rochtchina, E., Yu, M. C., Kefford, R., & Mitchell, P. (2006). Association between circulating white blood cell count and cancer mortality: A population-based cohort study. *Archives of Internal Medicine*, 166(2), 188–194.
- Smith, D. R., & Leggat, P. A. (2007). An international review of tobacco smoking research in the nursing profession, 1976–2006. *Journal of Research in Nursing*, 12(2), 165–181.
- Soares, S. R., & Melo, M. A. (2008). Cigarette smoking and reproductive function. *Current Opinion in Obstetrics & Gynecology*, 20(3), 281–291.
- Sopori, M. (2002). Effects of cigarette smoke on the immune system. *Nature Review Immunology*, 2(5), 372–377.
- Statistics Norway. (2009). *Smoking in Norway, 2008: Steady decline in number of daily smokers*. Retrieved July 6, 2010, from www.ssb.no/royk_en/arkiv/art-2009-02-03-01-en.html.
- Stemme, S., Holm, J., & Hansson, G. K. (1992). T lymphocytes in human atherosclerotic plaques are memory cells expressing CD45RO and the integrin VLA-1. *Arteriosclerosis & Thrombosis*, 12(2), 206–211.
- Straif, K., Baan, R., Grosse, Y., Secretan, B., El Ghissassi, F., Bouvard, V., et al. (2007). Carcinogenicity of shift-work, painting, and fire-fighting. *Lancet Oncology*, 8(12), 1065–1066.
- Tanigawa, T., Kitamura, A., Yamagishi, K., Sakurai, S., Nakata, A., Yamashita, H., et al. (2003). Relationships of differential leukocyte and lymphocyte subpopulations with carotid atherosclerosis in elderly men. *Journal of Clinical Immunology*, 23(6), 469–476.
- Tebow, G., Sherrill, D. L., Lohman, I. C., Stern, D. A., Wright, A. L., Martinez, F. D., et al. (2008). Effects of parental smoking on interferon gamma production in children. *Pediatrics*, 121(6), e1563–e1569.
- Tollerud, D. J., Clark, J. W., Brown, L. M., Neuland, C. Y., Mann, D. L., Pankiw-Trost, L. K., et al. (1989). Association of cigarette smoking with decreased numbers of circulating natural killer cells. *American Review of Respiratory Disease*, 139(1), 194–198.
- Trinkoff, A. M., & Storr, C. L. (1998). Work schedule characteristics and substance use in nurses. *American Journal of Industrial Medicine*, 34(3), 266–271.
- U.S. Department of Health & Human Services. (1980). *The health consequences of smoking for women: A report of the surgeon general*. Rockville, MD, Washington, DC: Author.
- U.S. Department of Health & Human Services. (2006). *The health consequences of involuntary exposure to tobacco smoke: A report of the surgeon general*. Rockville, MD, Washington, DC: Author.
- van Amelsvoort, L., Jansen, N. W. H., & Kant, I. (2006). Smoking among shift workers: More than a confounding factor. *Chronobiology International*, 23(6), 1105–1113.
- Van Dongen, C. J. (1999). Smoking and persistent mental illness: An exploratory study. *Journal of Psychosocial Nursing and Mental Health Services*, 37(11), 26–34.
- Vecchio, D., Sasco, A. J., & Cann, C. I. (2003). Occupational risk in health care and research. *American Journal of Industrial Medicine*, 43(4), 369–397.
- Wagner, T. J. (1985). Smoking behavior of nurses in western New York. *Nursing Research*, 34(1), 58–60.
- Wilhelmsen, L. (1998). Effects of cessation of smoking after myocardial infarction. *Journal of Cardiovascular Risk*, 5(3), 173–176.
- Wilson, J. L. (2002). The impact of shift patterns on health-care professionals. *Journal of Nursing Management*, 10(4), 211–219.
- Zhao, L., & Turner, C. (2008). The impact of shift work on people's daily health habits and adverse health outcomes. *Australian Journal of Advanced Nursing*, 25(3), 8–22.

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