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Introduction

Included in this compendium are articles and reports by those associated with the National Institute of Environmental Health Sciences (NIEHS) Worker Education and Training Program. The articles discuss issues important to hazardous materials training and the safety and health of workers.

The 1986 Superfund Amendments and Reauthorization Act of 1986 (SARA) authorized a program of grants for health and safety training for workers involved with hazardous materials and waste removal, containment, and emergency response. NIEHS was assigned responsibility for administering the program and making awards to non-profit organizations with demonstrated experience and ability in reaching target populations and operating worker health and safety programs. The Department of Energy also has a cooperative agreement with NIEHS to make training awards. In addition, Congress appropriated additional money for a Minority Worker Training Program. This appropriation established a series of national pilot programs to test a range of strategies for the recruitment and training of young persons who live near hazardous waste sites or in communities at risk of exposure to contaminated properties for work in the environmental field.

Since the NIEHS Worker Education and Training Program began in 1986, more than 100 organizations from across the country have trained workers to better protect themselves, their colleagues, and the communities in which they work from the dangers of hazardous materials. As a result of these programs, trained employees work more effectively at their job sites to promote safer processes and procedures. Twenty awardee groups and consortia, representing labor-management, labor, and academia, have developed model curricula and delivered training to more than 800,000 workers.

CANCER MORTALITY PATTERNS AMONG HAIRDRESSERS

AND BARBERS IN 24 U.S. STATES, 1984-1995.

(pending publication in the Journal of Occupational and Environmental Medicine)

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ABSTRACT

Hairdressers and barbers are exposed occupationally to a number of different chemical products. In order to evaluate overall and site-specific cancer mortality patterns among these occupational groups, we examined data enumerating cause of death and occupation and industry codes from 38,721 death certificates, collected between 1984-1995 from 24 states. We calculated mortality odds ratios (MORs) and 95% confidence intervals for cancer sites according to age, race, geographic region, and calendar year. Among female hairdressers of both races, significant increase in mortality occurred from non-Hodgkin's lymphoma (NHL), leukemia/aleukemia, and lymphoid leukemia. White women hairdressers had significant excess of mortality from cancers of the stomach, colon, pancreas, lung, breast, and bladder; black women hairdressers had significant excess of lung cancer. Women hairdressers had mortality elevations for many other sites. White male hairdressers had significantly elevated mortality from nonmelanoma skin cancer and NHL. Deficits for specific cancer sites were also noted for hairdressers according to race and gender. Significant mortality from stomach and pharyngeal cancer was observed among white male barbers and black male barbers respectively with excess mortality from multiple myeloma and leukemia/aleukemia. No specific patterns could be established for both occupations according to age and region. Elevations in cancer mortality for hairdressers and barber suggest that their occupations entail exposures that are probably carcinogenic. Further detailed studies evaluating their exposure to individual chemicals at work are required.

INTRODUCTION

Hairdressers and barbers represent a large occupational group with frequent exposures to products that are mutagens and carcinogens. The terms “hairdresser,” “cosmetologist,” and “beautician,” seem to be used interchangeably. Of the 500,000 to 750,000 professional cosmetologists and hairdressers in the United States, about 80 to 85 percent are women (Cosmetic, Toiletry, and Fragrance Association, 1992). Apart from using bleaches, hair dyes, shampoos, conditioners, hair sprays, and nail and skin care products, these professionals work with a number of “hair-preparations” for hair-styling, creating permanent waves, and hair straightening. Together, these formulations contain several thousand chemicals. Barbers, who are mainly men, generally cut only men’s hair and have less exposure to hair dyes and other cosmetic products. The products used by hairdressers and cosmetologists are, with a few exceptions, similar to the retail products sold for home use. Thus, potential exposure for consumers, either in beauty salons or at home, would be to a similar range of chemical substances as for hairdressers, with differences, however, in the frequency and duration of exposure.

Studies of occupational exposures among hairdressers have focused on hair dyes for several reasons. In the 1970s, a number of the aromatic amines and related nitro compounds in permanent hair dyes were found to be mutagens and animal carcinogens (Ames et al., 1975; NCI, 1978). It was also determined that these compounds could be absorbed through the skin (Kiese and Rauscher, 1968) and urinary mutagens have been found in the urine of cosmetologists (Babish et al., 1991). Several epidemiologic cohort studies of female hairdressers found increased risks of many cancers, most notably non-Hodgkin’s lymphoma (NHL) and ovarian cancer (Giles et al., 1984; Lynge & Thygesen, 1988; Pukkala et al., 1992; Teta et al., 1984; Kono et al., 1983). Personal use of hair dyes, particularly use of permanent dark dyes (Zahm et al, 1992; Thun et al., 1994), was

also associated with an elevated risk of NHL in several studies (Milham, 1983; Cantor et al., 1988; Zahm et al., 1992; Thun et al, 1994), but not in others (Hennekens, 1979; Grodstein et al., 1994; Holly et al.,1998). A recent study found higher NHL death rates only for prolonged use (10 or more years) of black or brown dyes (Altekruse et al., 1999). Use of permanent dark hair dyes has also been associated with elevated risk for leukemia (Mele et al., 1994)

The epidemiologic evidence for cancer among male hairdressers and barbers is limited to a consistent excess of bladder cancer found in five large cohort studies (Alderson, 1980; Guberan et al., 1985; Skov et al., 1990; Malker et al., 1987; Lynge and Thygesen, 1988). Although these studies did not adjust for smoking, two of these studies (Guberan et al., 1985; Lynge and Thygesen, 1988) found excesses of bladder cancer that were not accompanied by appreciable excesses of lung cancer.

In light of the fact that a number of studies have found increased cancer risks for hairdressers and barbers, we investigated cancer mortality among these occupational groups by examining more than 38,000 death certificates from 24 U.S. states over 12 years.

METHODS

The National Cancer Institute, the National Institute for Occupational Safety and Health, and the National Center for Health Statistics have supported the coding of usual occupation and industry titles (United States Department of Commerce, 1982) on death certificates from 24 states since 1984. This coding of industry and occupation on death certificates serves as a tool for national surveillance of occupational disease. We used death certificates from these 24 states to evaluate mortality patterns among hairdressers and cosmetologists (Standard Occupation Code [SOC] 458) as well as barbers (SOC 457). Our analysis included 38,721 deaths among those 20 years of age and older, from 24 states, over the years 1984-1995. We did not include the state of Alaska because mortality records

from Alaska were available only for one year with fewer than 2,000 deaths reported. Racial groups other than whites and blacks were also excluded due to small numbers.

Mortality odds ratios (MORs) and 95% confidence intervals were calculated according to Miettinen and Wong (Miettinen and Wong, 1981) separately for female hairdressers and cosmetologists (hereafter called hairdressers) and for male barbers. The number of deaths among female barbers was too low for calculation of cancer MORs. All noncancer deaths were used as the referent group. Age-specific (20 to 39, 40 to 59, 60 to 74, and 75+) MORs were calculated where numbers permitted. The analyses were also performed separately for 5 regions of the country: East (Maine, New Hampshire, New Jersey, Rhode Island, Vermont), North Central (Indiana, Ohio, Wisconsin), South Central (Kansas, Oklahoma, Missouri, Nebraska), South (Kentucky, Georgia, North Carolina, South Carolina, Tennessee, West Virginia), and West (Colorado, Idaho, Nevada, New Mexico, Utah, Washington). We evaluated overall mortality, mortality from all cancers combined, and for specific cancer sites while reporting MORs for cancer sites only if there were 5 or more deaths in one of the gender/race groups.

RESULTS

Among hairdressers, there were a total of 26,617 deaths: 19,980 white women; 3,602 black women; 2,641 white men; and 394 black men. Mortality from all malignant neoplasms combined was significantly elevated among women hairdressers of both races. There was a significant deficit in mortality from all malignant neoplasms for white male hairdressers and it was lower than expected for black men (Table 1). Among women hairdressers, mortality from cancer was significantly elevated among whites for the following sites: stomach, colon, pancreas, lung, breast, and bladder. Mortality from all lymphopoietic cancers, non-Hodgkin's lymphoma (NHL), leukemia/aleukemia, and lymphoid leukemia was also significantly elevated for white females. Mortality from the same cancers was also elevated among black women, with significant elevations for lung cancer and all lymphatic and hematopoietic cancers. Both

white and black women hairdressers had elevated mortality from cancers of the digestive organs and peritoneum, kidney, brain, and from Hodgkin's disease, multiple myeloma, and myeloid leukemia. Elevations in mortality for nasopharyngeal and pharyngeal cancers and cancers of the connective tissue and skin (non-melanoma) were also observed among white women hairdressers. Mortality from cancers of the liver, cervix, uterus, and ovary was elevated among black women. Non-significant deficits in mortality among women hairdressers occurred for cancers of the esophagus and bone and joints with significant deficits for monocytic leukemia. Black women hairdressers had mortality deficits for cancers of lip/ salivary gland/buccal cavity, larynx, connective tissue, and thyroid.

White male hairdressers had significantly elevated mortality from nonmelanoma skin cancer and NHL. Among black men, with the exception of all lymphatic and hematopoietic cancer, all other sites with elevations in mortality had 4 or fewer deaths. Significant deficits in mortality were observed among white men for cancers of the stomach, colon, lung, skin (melanoma), breast, kidney, brain, and for leukemia and monocytic leukemia. Nonsignificant decreases in mortality were observed for cancers of the pancreas, prostrate, bladder, and from myeloid leukemia for white male hairdressers.

An analysis of the cancer mortality patterns of hairdressers by geographic region (data not shown) revealed excess mortality from all cancers among white women in every region, which was significant in the east (MOR=1.29; 95% CI, 1.18, 1.42), north central (MOR=1.21; 95% CI, 1.14, 1.28), south central (MOR=1.11; 95%CI, 1.01, 1.21) and south (MOR=1.08; 95% CI,1.01, 1.14). Among black women, overall mortality was elevated in every region except the west with significant elevation in the north central states (MOR=1.37; 95% CI, 1.19, 1.59).

Among white women hairdressers, elevations in mortality in all five regions were seen for pancreatic cancer, lung cancer, all lymphatic and hematopoietic cancers, multiple myeloma, leukemia and aleukemia, and lymphoid leukemia. Elevations in mortality from cancer at other sites were scattered over the five regions. For black women, no single cancer site presented elevated mortality in all five regions. The significant elevation in mortality from cancers at individual sites generally occurred among white women

40 years of age and older and among black women 60 and older. Unlike white women, mortality from cancers of the stomach, colon, and ovary was elevated in black women only age 60 and above.

Among white male hairdressers, the deficit of mortality from cancers at individual sites was consistent across all regions (data not shown). Among black male hairdressers, mortality deficits were observed in all regions except the south and west. Mortality from all malignant neoplasms combined was lower than expected for white men under age 75 and for black men under 60. With the exception of the south central states, NHL was elevated in all states and in all ages below 75 for white men. NHL was significantly elevated in the youngest white men, ages 20-39 (MOR=2.11; 95% CI, 1.38, 3.21). For black male hairdressers, all regional and age group specific estimates for individual cancer sites were based on fewer than 5 deaths.

In the case of male barbers, 12,104 deaths occurred between 1984 and 1995. Of those, 10,572 were white men and 1,532 were black men. Overall cancer mortality was slightly lower than expected for white barbers and very close to expected for black barbers. Mortality among white male barbers and black male barbers was significantly elevated for stomach and pharyngeal cancer respectively (Table 2). Among barbers of both races, MORs were elevated for multiple myeloma, and leukemia/aleukemia. Mortality from specific cancers among barbers differed somewhat from that of male hairdressers.

There were significant mortality deficits for all malignant neoplasms and for cancers of the pancreas, lung, and prostrate for white male barbers and esophageal cancer for black male barbers. Mortality from cancer of the lip/salivary glands/buccal cavity, digestive organs, larynx, melanoma, NHL, and Hodgkin's disease was lower than expected among both races. Significant excess of mortality from cancer of the bladder occurred only among white male barbers in the east (MOR=1.69; 95% CI, 1.09, 2.62) and south central (MOR=1.76; 95% CI, 1.11, 2.80) regions. No discernible patterns in mortality according to age were observed for the male barbers.

DISCUSSION AND CONCLUSIONS

We observed significant increases in mortality from cancers of the lung and all lymphohematopoietic cancers among women hairdressers of both races in 24 states. For white women hairdressers, significant mortality also occurred from cancer of the stomach, colon, pancreas, breast, bladder, and from NHL, leukemia/aleukemia, and lymphoid leukemia. The increases in risk, however, were not very large. There was a statistically significant deficit among white male hairdressers for mortality from all malignant neoplasms and cancers of the stomach, colon, lung, melanoma, brain, and leukemia/aleukemia, whereas mortality from nonmelanoma skin cancer and NHL was significantly elevated. White male barbers also had a significant deficit of mortality from all malignant neoplasms and from cancers of the pancreas, lung, and prostrate. In contrast to white male hairdressers, white male barbers did not exhibit elevated mortality from rectal cancer, nonmelanoma skin cancer, or NHL. Mortality from stomach cancer and pharyngeal cancer was significantly elevated among whites and blacks respectively and mortality from multiple myeloma and leukemia/aleukemia was elevated among both white and black barbers.

In our analysis, women hairdressers had significantly elevated mortality from malignant neoplasms. In contrast to women hairdressers, male hairdressers and barbers had an overall deficit in mortality from all malignant neoplasms. A previous analysis of cancer mortality among cosmetologists in Connecticut found excess cancer mortality among women (Teta et al., 1984) but most cohort studies in other countries have not observed excess cancers overall (Alderson, 1980; Guberan et al., 1985; Kono et al, 1983; Pukkala et al., 1992). Previous epidemiologic studies of hairdressers and barbers in Europe, the United States, and Japan have observed elevated rates of various cancers with excesses for bladder, lung, ovarian, and lymphatic and hematopoietic cancers reported most frequently (IARC, 1993). We noted excess mortality from cancers of the bladder, lung, and lymphohematopoietic cancers among female hairdressers of both races, but ovarian cancer was elevated only among black female hairdressers. Male hairdressers and barbers also had excess mortality from specific lymphatic and hematopoietic cancers.

We observed elevated pharyngeal cancer mortality among women hairdressers and male barbers. Excess of buccal cavity and pharyngeal cancer has been observed in the past only among male hairdressers (Guberan et al., 1985); another study from the United Kingdom failed to find increased risk for these sites (Alderson, 1980). Excessive alcohol consumption has been shown to increase risk of developing cancers of

the mouth and pharynx (Kato, 1990; Tonnesen, 1994). Female cosmetologists were one of the occupational groups with the highest standard mortality rates for cirrhosis of the liver in one study (Leigh and Jiang, 1993). However, lack of information on alcohol consumption trends among hairdressers precludes association between excessive alcohol intake and the observed elevation in mortality from pharyngeal cancer. We observed a significant increase in stomach cancer mortality among white women hairdressers and white male barbers. Significant excess of stomach cancer among Japanese women beauticians (Kono et al., 1983) and excess of stomach cancer among female hairdressers (Milham, 1983; Office of Population Censuses and Surveys, 1986) has been noted previously. Socioeconomic status is also known to influence health status. Hairdressers and barbers have been placed in the "secondary blue collar" category in previous sociological research, which is indicative of the lowest socioeconomic status (SES) (Barnett et al., 1997). Cancers of the pharynx and stomach have been associated with lower levels of education (Ferraroni, 1989) and lower SES (Pukkala, 1986) as well as smoking (Ferraroni, 1989).

We noted a significant elevation in mortality from cancer of the lung for women hairdressers in our results. Elevated rates of lung cancer have been consistently observed in cohort studies of women hairdressers (Kono et al, 1983; Teta et al., 1984; Skov et al., 1990; Malker et al, 1987; Pukkala et al., 1992) with few exceptions (Lynge and Thygesen, 1988). One U. S. study found significant elevation in lung cancer mortality among women hairdressers (Rubin et al., 1994). Both formaldehyde, used in shampoos and nail products, and vinyl chloride, previously used as a propellant in hair sprays compounds have been associated with lung cancer in some occupational studies. However, the epidemiologic data are not strong or consistent (Blot and Fraumeni, 1996). A more probable explanation for this observed excess of lung cancer may be a higher prevalence of smoking among hairdressers as was noted in Sweden (Skov et al., 1990; Malker et al., 1987) and in the U.S. (Leigh, 1996). Another U.S. study (Osorio et al., 1986) found no association between lung cancer and specific occupational tasks or exposures of female hairdressers after controlling for smoking status.

Our results showed an excess of pancreatic cancer mortality among women hairdressers and black male barbers. Hairdressers are exposed to solvents from nail products and lacquer/enamel removers, and to aromatic amines from hair dyes; exposure to the latter has also been linked with high rates of pancreatic

cancer (Frumkin, 1994). Pancreatic cancer is clinically associated with pancreatitis, which in turn has been associated with occupational exposure to organic solvents (Redlich and Brodkin, 1994). Nonsignificant elevations in risk for pancreatic cancer have been found for male barbers in Massachusetts (Dubrow and Wegman, 1982, 1983, 1984) and for male hairdressers in Finland (Pukkala et al., 1992). Smoking increases the risk of pancreatic cancer several-fold and has been consistently associated with pancreatic cancer (Zheng, 1993; Howe, 1994; Silverman, 1994; Boyle, 1996; Fuchs, 1996; Harnack 1997; Weiderpass, 1998). However, specific etiological agents for pancreatic cancer have not been identified (Redlich and Brodkin, 1994).

Mortality due to non-melanoma skin cancer was elevated in our analysis for white female hairdressers, with significant elevation for white male hairdressers. Increased risk for non-melanoma skin cancer was observed among women hairdressers in a Finnish cohort study (Pukkala, 1992).

We observed significantly elevated mortality from breast cancer in white women hairdressers and non-significant elevation for black women. Evidence of excess breast cancer risk among hairdressers has been inconsistent in studies conducted in the past. Significant excess of breast cancer in female cosmetologists has been noted in the U.S. (Teta et al., 1984) and Japan (Kato, 1990) and non-significant excess in other U.S. (Koenig et al., 1991) and Finnish (Pukkala et al., 1992) studies. A review of occupational studies on female breast cancer found limited evidence of an association with employment as cosmetologist (Goldberg, 1996), but a recent study found excess breast cancer risk for Swedish hairdressers and beauticians (Pollan et al., 1999). Excess breast cancer was reported for personal use of hair dye by women in New York (Shafer and Schafer, 1976) and in Washington state (Cook et al., 1999). But six case-control studies (Kinlen et al., 1977; Shore et al., 1979; Stavrasky et al., 1979; Nasca et al., 1980; Wynder and Goodman, 1983; Koenig et al., 1991) and one cohort study (Hennekens et al., 1979) found no significant excess of breast cancer among hair dye users.

Occupational exposure to aromatic amines may explain up to 25 percent of bladder cancers in some areas of Western countries (Vineis, 1997). The excess rates of bladder cancer among hairdressers has been of particular interest because of their frequent exposure to hair coloring products that contain mutagens and

possible bladder carcinogens (Hartge et al., 1982; IARC, 1993). Urine mutagenicity was increased in hairdressers exposed to hair dyes compared to those without exposure (Babish et al., 1991) suggesting that the dye components are absorbed systemically.

In our analysis, women hairdressers and black male barbers experienced excess mortality from bladder cancer. Excess bladder cancer incidence and mortality has been observed in previous studies (Teta et al., 1984; Guberan et al., 1985; Skov et al., 1990; Lynge and Thygesen, 1988) but not consistently (Risch et al., 1988), especially among women (Kono et al., 1983; Malker et al, 1987; Pukkala e al., 1992). The results from numerous case-control studies of bladder cancer that evaluated occupation as a barber or hairdresser are not consistent and were usually limited by small numbers (IARC, 1993). Overall, allowance for smoking was lacking or inadequate in most studies. The largest study, a population-based case-control study in 10 areas of the United States, found a thirty percent increased risk among white male barbers and hairdressers and a forty percent increased risk among white female hairdressers (Silverman et al., 1989). A recent Canadian case-control study found strongly increased risks of bladder cancer for hairdressers (Teschke, 1997) -- all three cases among hairdressers had applied hair dyes.

We found excess mortality from brain cancer in women hairdressers and white male barbers; however, there was a significant deficit among white male hairdressers. Teta et al. found excess of brain cancer among male and female cosmetologists in Connecticut (Teta et al., 1984). Another investigation of a brain cancer cluster in Missouri found significantly elevated brain cancer for hairdressers and cosmetologists (Neuberger, 1991). In Canada, significant elevation in brain cancer was also noted among adults using hair dye or hair spray (Burch et al., 1987).

We observed excess mortality from all lymphatic and hematopoietic cancers among all hairdressers and barbers, although the specific cancer varied according to gender and profession. Non-Hogkin's lymphoma

(NHL) related mortality was elevated among female hairdressers and white male hairdressers. Mortality from Hodgkin's disease, multiple myeloma, leukemia/aleukemia, and lymphoid and myeloid leukemia was also elevated among women hairdressers. Barbers had excess mortality from multiple myeloma, leukemia/aleukemia, and myeloid leukemia. Most studies have found elevated risks of NHL or all lymphomas (Decoufle, 1977; Boffetta, 1994) or other lymphopoietic cancers (Spinelli et al., 1984; Guidotii et al, 1982; Teta et al., 1984; Menck, et al, 1977; Kono et al., 1983; Guberan et al., 1985) among hairdressers. These studies have been mainly conducted in Europe and included white women. Significant excess for NHL among female hairdressers was found in Australia (Giles et al., 1984) and Denmark (Boffeta et al., 1994) while the risk of NHL decreased in Sweden (Boffeta et al., 1994). A non-significant excess of NHL was noted among men and women hairdressers in Denmark (Lynge & Thygesen, 1988), among male hairdressers in the U.S. (Blair et al., 1993), and for women hairdressers in Italy (Constantini et al., 1998; Miligi et al., 1999) .

The overall incidence of non-Hodgkin's lymphoma has risen steadily over the past four decades (Smith, 1996). NHL and Kaposi's sarcoma are AIDS-defining illnesses (Smith et al., 1998) and HIV infection is most strongly correlated with the increasing incidence of NHL in the United States (Chassagne-Clement et al., 1999). Increased AIDS mortality rates observed for male hairdressers (Lamba, 1998) and better diagnosis of NHL might partly explain the excess mortality from NHL observed in male and female hairdressers. However, HIV-associated disease accounts for only a small part of the increase in this lymphoma (Smith, 1996). Another explanation may be hairdressers' exposure to solvents in nail care products. In a review of 45 studies on possible association between NHL and exposure to organic solvents, 13 defined or suggested organic solvents as possible risk factors for NHL (Rego, 1998).

Nonsignificant excess for Hodgkin's disease for female hairdressers has been noted in other studies (Office of Population Censuses and Surveys, 1986; Hrubec et al., 1992; Costantini et al., 1998; Miligi et al., 1999;

Robinson et al., 1999) as well as in our analysis. A case-control study of personal exposure to hair dyes (Zahm et al., 1992) found increased risk of Hodgkin's disease in women who used hair dyes; the risk was higher for use of permanent hair dyes.

Excess of multiple myeloma among hairdressers has been observed in the U.S. (Guidotti et al., 1982; Milham 1983), Europe (McLaughlin et al., 1988; Costantini et al., 1998; Miligi et al., 1999), and Australia (Giles et al., 1984) with a sixfold increase for female hairdressers in one study (Spinelli et al., 1984). In one study from Finland (Pukkala et al, 1992), no excess for multiple myeloma was observed for women hairdressers. Among barbers, excess of multiple myeloma has been found in the U. S. (Hrubec et al., 1992), and a significant increase in Canada (Gallagher et al., 1989). In the U. S., significant elevation in risk for multiple myeloma was noted for men who used hair dyes (Brown et al., 1992). Risk of multiple myeloma among women has also been associated with use of dark permanent hair dyes (Zahm et al., 1992), especially black permanent hair dyes (Altekruse, 1999).

Studies of Italian hairdressers have found a sixfold increase in risk for chronic myeloid leukemia (Mele et al., 1994) and excess risk for lymphocytic leukemia (Miligi et al., 1999). Significant association between acute lymphocytic leukemia and hair dye use was found in the U.S. (Markowitz et al., 1985; Cantor et al., 1988) and a slight increased risk was noted in one Italian study (Miligi et al., 1999) for women using permanent hair dyes.

The major advantage of the data we used is the large numbers of deaths, which allowed us to compare mortality patterns among both men and women hairdressers and barbers by race, age, and geographic regions across the country. However, age- and region-based comparisons in mortality were limited by small numbers for many of the cancer sites. Limitations of an analysis that uses coding of occupation on death certificates to define exposure includes the questionable accuracy of the coding of occupation and some causes of death. There is the potential for misclassification errors, which would tend to bias risk estimates toward the null (Checkoway et al., 1989). The accuracy of coding for cause of death is quite good for cancers of the stomach, pancreas, lung, prostrate, thyroid, and multiple myeloma, but not as good for colon, rectum, connective tissue, bone, cervix, and eye (Percy et al., 1981).

Other limitations of this analysis were lack of detailed information on occupation and industry and on confounding lifestyle factors such as smoking and diet. While death certificates ask for the "usual" or lifetime occupation and industry, the information entered may be more representative of occupation or industry at the time of death (Stout and Bell, 1991). Death certificates also do not provide information on other occupations held by the deceased in the past, so that duration of exposure and latency cannot be analyzed.

The International Agency for Research on Cancer has concluded that occupation as a hairdresser or barber entails exposures that are probably carcinogenic (IARC, 1993). Our results showed excess mortality from many cancers among these groups that were generally consistent with cancer excesses reported in the literature, especially among women hairdressers. Male hairdressers and barbers had deficits of cancer overall and at many sites; however, mortality from certain lymphatic and hematopoietic cancers was elevated. While the observed excesses in mortality might be attributed to the various chemicals and chemical mixtures hairdressers and barbers are exposed to occupationally, the effects of lifestyle risk factors for cancer-related mortality such as alcohol consumption, smoking, sexual habits, and of low socioeconomic status cannot be ignored. Synergistic effects between chemical exposures and lifestyle factors may also lead to increased risk..

For hairdressers, current exposure to hair dye components differs from that in the past. Over the past twenty years, many of the chemicals discovered to be mutagenic and carcinogenic in hair dyes and other hair preparations have been banned from use in the U.S. and Europe. However, commercially used hair dyes still contain many carcinogenic compounds. Substitutes used by the hair dye manufacturers might also be potentially carcinogenic since chemically, they are close structural relatives of the banned chemicals.

Effects of exposure to chemical mixtures on hairdressers and barbers are by and large unknown and there is limited availability of human data for such exposures. Further detailed studies with emphasis on exposure assessment in hairdressing salons and barber shops are required. Apart from ascertaining duration and frequency of exposure, such studies should focus on occupational exposure to specific products like hair

dyes, hair sprays, nail treatments, and other exposures in hairdressing salons in order to tease out the individual effects of different products and chemicals.

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Table 1. MORs Among Hairdressers by Race and Gender for Select Cancer Sites

Cancer Site	**White Women**	**Black Women**	**White Men**	**Black Men**
All malignant neoplasms	1.13 (5643)	1.15 (867)	0.71[i] (375)[ii]	0.81 (39)
	1.10-1.17	1.06-1.24	0.64-0.79[iii]	0.59-1.12
Lip, Salivary glands, and buccal cavity	1.06 (33)	0.60 (3)	0.72 (4)	0.00(0.963)[exp.]
	0.75-1.48	0.20-1.84	0.27-1.89	
Nasopharynx	1.30 (6)	1.23 (1)	0.91 (1)	0.00 (0.181)[exp]
	0.59-2.90	0.18-8.52	0.13-6.39	
Pharynx	1.36 (24)	1.23 (5)	0.46 (2)	0.00 (1.044) [exp]
	0.91-2.03	0.51-2.94	0.12-1.83	
Digestive organs and peritoneum	1.11 (1221)	1.10 (234)	0.66 (76)	0.64 (8)
	1.05-1.18	0.97-1.26	0.52-0.83	0.33-1.24
Esophagus	0.92 (38)	0.63 (10)	0.54 (7)	0.00 (2.505) [exp]
	0.67-1.27	0.34-1.17	0.26-1.12	

Stomach	1.21 (114)	1.11 (29)	0.47 (7)	0.00 (2.053) [exp]
	1.01-1.45	0.77-1.60	0.23-0.99	
Large Intestine excluding rectum (Colon)	1.12 (561)	1.18 (103)	0.46 (20)	0.58 (2)
	1.03-1.22	0.97-1.43	0.30-0.72	0.16-2.04
Rectum and rectosigmoid joint	0.95 (66)	1.20 (13)	1.11 (9)	5.00 (4)
	0.75-1.21	0.70-2.06	0.58-2.13	2.04-12.26
Liver	0.87 (18)	1.68 (7)	1.86 (8)	0.00 (0.803) [exp]
	0.54-1.37	0.80-3.52	0.94-3.69	
Pancreas	1.24 (312)	1.01 (51)	0.75 (17)	0.52 (1)
	1.11-1.39	0.77-1.34	0.47-1.21	0.08-3.35
Larynx	1.36 (21)	0.66 (2)	0.42 (2)	0.00 (0.853) [exp]
	0.89-2.08	0.17-2.62	0.11-1.64	
Trachea, bronchus, and lung	1.32 (1413)	1.26 (162)	0.71 (114)	0.84 (12)
	1.25-1.40	1.07-1.47	0.59-0.86	0.48-1.47
Bone and joints	0.63 (6)	0.70 (1)	0.40 (1)	0.00 (0.213) [exp]
	0.28-1.39	0.10-4.92	0.06-2.84	
Connective tissue	1.06 (39)	0.89 (6)	0.31 (2)	1.71 (1)
	0.78-1.46	0.40-1.97	0.08-1.22	0.25-11.85

Cancer Site	**White Women**	**Black Women**	**White Men**	**Black Men**
Melanoma	0.98 (63) 0.76-1.25	0.50 (1) 0.07-3.51	0.44 (9) 0.23-0.85	12.85 (1) 2.21-68.12
Skin (non-melanoma and nos)	1.27 (16)	1.22 (2)	2.92(11)	2.72 (1)
	0.78-2.07	0.31-4.86	1.62-5.25	0.39-18.74

Breast	1.10 (1027)	1.15 (153)	0.00 (0.636) [exp]	13.10 (1)
	1.03-1.17	0.98 (1.36)		2.38-72.22
Cervix uteri	0.91 (93)	1.10 (32)	-	-
	0.74-1.12	0.78-1.55)		
Corpus uteri, uterus nos, chorionepithelioma	0.89 (108)	1.12 (33)	-	-
	0.73-1.07	0.79-1.57		
Ovary, fallopian tube, uterine adnexa	1.00 (285)	1.22 (37)	-	-
	0.89-1.12	0.89-1.69		
Prostrate	-	-	0.80 (27)	0.53 (2)
			0.54-1.17	0.14-2.01
Testis	-	-	0.46 (2)	0.00 (0.143) [exp]
			0.12-1.84	
Bladder, urethra, and other urinary organs	1.36 (88)	1.19 (15)	0.59 (6)	0.00 (0.482) [exp]
	1.10-1.68	0.72-1.98	0.27-1.31	
Kidney and renal pelvis	1.08 (91)	1.15 (12)	0.36 (5)	0.86 (1)
	0.88-1.33	0.66-2.03	0.15-0.85	0.14-5.22
Brain	1.09 (132)	1.29 (10)	0.41 (11)	0.00 (1.037) [exp]
	0.91-1.29	0.70-2.39	0.23-0.74	
Thyroid	0.58 (7)	0.98 (2)	1.07 (1)	0.00 (0.041) [exp]
	0.27-1.21	0.25-3.91	0.15-7.48	
All lymphatic and hematopoietic cancer	1.15 (569)	1.31 (84)	0.99 (70)	1.05 (6)
	1.15-1.25	1.05-1.62	0.78-1.25	0.49-2.22
Non-Hodgkin's lymphoma	1.15 (227)	1.13 (17)	1.49 (43)	0.47 (1)
	1.01-1.31	0.70-1.81	1.10-2.01	0.08-2.83

Hodgkin's disease	1.38 (23)	2.40 (4)	0.92 (6)	1.76 (1)
	0.92-2.08	0.91-6.33	0.41-2.05	0.26-11.68
Multiple Myeloma	1.18 (102)	1.20 (30)	0.98 (7)	3.18 (3)
	0.97-1.43	0.84-1.72	0.47-2.05	1.07-9.40
Cancer Site	**White Women**	**Black Women**	**White Men**	**Black Men**
Leukemia and aleukemia	1.15 (200)	1.29 (26)	0.42 (11)	0.50 (1)
	1.00-1.32	0.88-1.90	0.23-0.75	0.10-2.51
Lymphoid leukemia	1.32 (59)	1.43 (8)	0.62 (4)	0.00 (0.436) [exp]
	1.02-1.71	0.71-2.85	0.24-1.65	
Myeloid leukemia	1.07 (83)	1.34 (12)	0.54 (7)	0.98 (1)
	0.86-1.32	0.76-2.35	0.26-1.12	0.20-4.86
Monocytic leukemia	0.39 (1)	0.00 (0.291) [exp]	0.00 (0.354) [exp]	0.00 (0.022) [exp]
	0.05-2.77			

Table 2. MORs Among Barbers by Race for Select Cancer Sites

Cancer Site	White Men	Black Men
All malignant neoplasms	0.93[iv] (2198)[v]	0.99 (373)
	0.88-.97[vi]	0.88-1.12
Lip, Salivary glands, and buccal cavity	0.84 (18)	0.67 (3)
	0.53-1.34	0.22-2.05
Nasopharynx	0.38 (1)	1.56 (1)
	0.05-2.67	0.22-10.92
Pharynx	0.60 (10)	1.92 (10)
	0.32-1.11	1.04-3.58
Digestive organs and peritoneum	0.93 (509)	0.95 (88)
	0.85-1.02	0.76-1.18
Esophagus	0.91 (48)	0.47 (8)
	0.69-1.21	0.24-0.94
Stomach	1.26 (81)	0.76 (12)
	(1.01-1.57)	0.43-1.34
Large Intestine excluding rectum (Colon)	0.95 (216)	1.14 (33)
	0.83-1.08	0.81-1.61
Rectum and rectosigmoid joint	0.72 (27)	1.02 (5)
	0.50-1.06	0.43-2.44
Liver	1.02 (17)	1.55 (5)
	0.63-1.64	0.65-1.71

Pancreas	0.78 (81)	1.07 (18)
	0.62-0.96	0.68-1.71
Larynx	0.69 (16)	0.87 (5)
	0.42-1.12	0.37-2.07
Trachea, bronchus, and lung	0.90 (700)	0.88 (105)
	0.83-0.98	0.71-1.07
Bone and joints	1.82 (7)	1.57 (1)
	0.87-3.82	0.22-10.91
Connective tissue	0.94 (11)	2.13 (3)
	0.52-1.69	0.69-6.56
Melanoma	0.68 (22)	0.00 (0.506)[exp]
	0.45-1.04	
Cancer Site	**White Men**	**Black Men**
Skin (non-melanoma and nos)	0.86 (12)	2.50 (4)
	0.49-1.51	0.96-6.51
Breast	0.38 (1)	0.00 (0.487) [exp]
	0.05-2.69	
Prostrate	0.86 (288)	1.11 (80)
	0.77-0.97	0.88-1.39
Testis	0.62 (1)	0.00 (0.157) [exp]
	0.09-4.40	
Bladder, urethra, and other urinary organs	0.98 (75)	1.48 (9)
	0.78-1.23	0.77-2.5

Kidney and renal pelvis	0.98 (52)	1.14 (7)
	0.74-1.28	0.55-2.39
Brain	1.17 (53)	1.28 (4)
	0.89-1.53	0.48-3.39
Thyroid	1.54 (5)	0.00 (0.298) [exp]
	0.64-3.69	
All lymphatic and hematopoietic cancer	1.02 (233)	1.08 (28)
	0.89-1.16	0.74-1.56
Non-Hodgkin's lymphoma	0.88 (72)	0.79 (5)
	0.70-1.11	0.33-1.87
Hodgkin's disease	0.85 (5)	0.00 (0.728) [exp]
	0.35-2.04	
Multiple myeloma	1.24 (49)	1.23 (11)
	0.94-1.64	0.68-2.22
Leukemia and Aleukemia	1.09 (97)	1.10 (10)
	0.89-1.33	0.59-2.04
Lymphoid leukemia	1.00 (29)	0.34 (1)
	(0.69-1.43)	0.05-2.33
Myeloid leukemia	1.16 (39)	1.11 (4)
	0.85-1.58	0.42-2.94
Monocytic leukemia	0.00 (1.244) [exp]	0.00 (0.090) [exp]

[i] MOR

[ii] Number of deaths

[iii] 95% Confidence intervals

[exp] Number of expected deaths where number of observed deaths was zero

[iv] MOR

[v] Number of deaths

[vi] 95% Confidence interval

[exp] Number of expected deaths where number of observed deaths was zero

IMPROVING THE STUDY OF THE IMPACT OF WORKER HEALTH AND SAFETY TRAINING

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This brief paper addresses two related aspects of training impact research: (1) how the various audiences for impact information affect the design of impact research and (2) several selected approaches to enhance research on the impact of worker health and safety training. The experiences of the Midwest Consortium for Hazardous Waste Worker Training are used to illustrate this necessarily brief discussion. The Midwest Consortium consists of nine training organizations in eight states and a centralized evaluation group.

It is important to note at the outset that training alone is a weak intervention. Without regular use of the knowledge and skills and/or absent frequent - more than once per year - refresher training, workers knowledge and skills will degrade substantially (see C. Vaught's paper in this publication for an excellent example). Workers may not be ale to utilize their knowledge and skills for many reasons, e.g., no need or opportunity to use them or organizational barriers to their utilization. Thus, it is not realistic to expect large workplace impacts from health and health safety training. However, the accumulation of small impacts over millions of workers exposed to hazardous materials can result in important benefits to all concerned.

Audiences For Training Impact Information

How do we choose what impacts to evaluate? One way to think about this question is to couch it in terms of the stakeholders and potential audiences for our research. Who are the Midwest Consortium's audiences and what are their information needs? First and foremost, those who employ our trainees want to documented evidence for the benefits of health and safety training. The range of employer benefits is large, e.g., a better trained workforce, compliance with regulations, reduced insurance or workman's compensation premiums, a safer workplace, reduced exposures among workers, increased productivity to name a few of the more obvious ones. Trainers, training organizations, course developers need impact information to evaluate and revise course content and to build more appropriate performance exercises. Indirectly, future trainees benefit from the results of impact studies by having improved training programs available to them. The Midwest Consortium's external advisory board has responsibility to help shape the Consortium's training effort and thus needs information on training effectiveness and training impact.

In its role as funding agency, the NIEHS program office requires information on training impact to document funding needs and funding successes to Congress, to make performance comparisons and to allocate program dollars across grantees. To illustrate, the Midwest Consortium (along with other grantees) has pilot tested several ways to elicit reliable and valid information on employment of trainees after they leave the training institution. Tracking Grantees also can learn from each other as results of evaluation/impact studies (both successful and unsuccessful) are shared. Finally, research on the impact of health and safety training may contribute to the knowledge base of the scientific/professional communities.

Clearly, information needs vary across audiences. What may not be as obvious is that the same information may be used by different audiences for different purposes and that different operational definitions of the same construct may more or less satisfactory for different audiences. Assume for the moment that we can agree upon an operational definition of "impact". Some audiences may find that the "average" impact across a sample of workers is sufficient information. Others may need to know under what conditions and among what types of workers does training have differential impacts. More complex questions require larger sample sizes. Such different research questions have implications for the study design. For example, addressing the contingency question, ("under what conditions") forces the researcher to examine the organizational contexts of work, not just worker behaviors. Impact studies now take on the character of transfer of training studies (Ford & Fisher, 1994) which require a broadening of the conceptual and theoretical frameworks within which research is conduced and the ability to analyze data collected across multiple levels of the organization (i.e., linked individual and group level data).

Audience needs have implications for at least three other aspects of training impact research . Some audiences may be interested in why health and safety training had differential effects; the theory question. The Midwest Consortium has focused on the concept of self-efficacy - the subjective evaluation or belief that a particular task or job can be performed competently (Bandura, 1997). We have tried to measure individual self-efficacy using a variety of specific actions (e.g., donning a respirator, using the NIOSH Pocket Guide, reading placards, etc.). Our results to date suggest that our approach has produced measures of self-efficacy that are factorially complex and which probably vary by type of trainee. A different approach might be to adapt a more generic and shorter measurement model of self-efficacy (Jones, 1986). The positive consequences of adopting a generic approach to self-efficacy measurement are to reduce the response burden on trainees who complain about an overabundance of paper work and to be able to compare results across training populations The negative consequence of using a generic model of self-efficacy is loss of task specificity. This trade-off in gains and losses from a design choice is but one example of a measurement model decision that simultaneously meets the information demands of the scientific community but reduces the value of the information for course modification.

Second, audiences differ in terms of how much confidence they have in the results. Because the cost of information is so high, managerial decisions and course revision decisions typically be driven by incomplete information. Professional and scientific communities place more stringent conditions on the sorts of impact information that are acceptable to them. Finally, most audiences are (or should be) interested in the

cost-effectiveness of different modes of delivering health and safety training; especially with the advent of advanced technology modes of delivery.

Towards improved training impact research

Space permits only a limited discussion of challenges and potential solutions to the design of training impact studies. We have chosen to discuss two issues that we believe can materially improve research on the impacts of health and safety training. First, we will address a constellation of themes that fall under the heading of measurement. Second we will address the need to refocus our attention on intervention processes, like the training itself and, perhaps, features of the work context.

Measurement Themes. Assessing the impact of health and safety training is a challenge, especially since the enterprise relies so heavily on self-reports of trainees, workers, and employers. Training impact studies should incorporate current psychometric principles whenever possible. We add the last caveat to acknowledge that in early stages of development, operational measures may have to be crude and cannot reach acceptable psychometric standards. Such developmental efforts should be applauded, but not used as an excuse for shoddy measurement.

There is an ongoing field of research that seeks to understand the nature and direction of biases in self-reports and how these findings might be incorporated into the practice of survey research (see e.g., Schwarz & Sudman, 1996; Stone, et al, 2000) . As one illustration, Kruger (1999) has shown that people systematically rate themselves below average when they were evaluating performance on challenging tasks; a healthy antidote to the argument that trainee ratings are inflated by the so-called Lake Wobegon effect. At a minimum, researchers should incorporate this new knowledge into their survey instruments. Ideally, researchers should begin to investigate the effects of question wording on the results of impact studies.

Relying on self-reports to evaluate training impact produces a serious, but more subtle, limitation on the interpretation of impact studies. When both the outcome measure (e.g., changes in work practice, attitudes, or beliefs) and the predictor variables (e.g., self efficacy, aspects of the training, perceptions of the organizations safety climate), are self-reported on the same survey instrument, the measure share common methods variance. This sharing of methods variance in the predictor and outcome variables inflates the empirical relationships among the variables. This problem is cannot be managed statistically; it is an inherent problem in that particular design. The solution - measure the outcome and predictor variables independently - is obvious, but difficult and expensive to implement.

At the outer edges of the difficulty and expense continua, Ecological Momentary Assessment techniques (EMA), also known as the Experience Sampling Method, can be applied to assess training impact variables. (For an introduction to EMA, see Shiffman & Stone, 1998. To see its difficulties, see Schwartz & Stone, 1998.). Applied to training impact studies, EMA would require repeatedly collecting self-reported data from workers on the job. The goal of EMA would be to obtain a representative sample of workers' behaviors as that behavior occurs. Depending upon the purpose of the research, instances of the focal behavior could collected using random intervals, regular intervals, or whenever the focal behavior occurs on the job. More recent applications of EMA utilize hand-held computers to cue participants and to record the actual time of data entry.

Although EMA relies on self-report, the data are collected independently of a survey questionnaires, significantly reducing methods bias. Moreover, EMA eliminates recall biases which are inherent in survey methods. On the negative side, EMA places a huge response burden on workers, requires cooperation from workers and their employers, requires expensive hardware, and demands an extremely high level of statistical sophistication to model pooled within-worker data. Despite its complexity, we believe that EMA is a viable candidate methodology for training impact research.

In addition to EMA, there are more conventional sources of outcome data that are independent of worker surveys. These sources include, but are not limited to, diaries, observational methods, group processes (e.g., focus groups), open-ended interviews, management information systems, and/or other data archives. No one data source or data collection methodology is error free or bias free. Multiple measures and multiple methods should frame our future research in order to produce results in which we can have the most confidence.

Intervention and Process Variables. Unless we can learn why our training had the effects it had, we will not be able to improve worker health and safety in this and in other contexts. Consider the following hypothetical scenario. Results of our study suggest that workers did not use any of the skills they learned on the job. How are we to explain this finding? To list just a few: (1) poorly delivered training, (2) poorly conceived training, (3) training not implemented as designed (4) unempowered workers, (5) unconcerned employers, (6) concerned but unaware employers, (7) lax regulatory enforcement, (8) inadequate theory of organizational processes. Unless we describe and monitor the processes from training through the work environment, we will not be able to understand either failures or successes, the negative impacts or the positive impacts.

To illustrate the point, Berger et al (1996) measured three self-reported measures of ability among trainees who completed either a long (24 or 40 hours) course containing lots of hands-on learning or a short (8 hour) course with little hands-on training. The study used the retrospective pretest-posttest design first described by Howard (1980). The retrospective pretest design is an alternative model to the more traditional pretest-posttest design. In the retrospective pretest design, trainees report on their perceived level of ability after completing the training and then how they perceived their levels of ability to have been prior to taking the training. The differences between retrospective pretraining means and posttraining means was substantially greater among trainees in the long courses than in the short course. A plausible explanation for this result is that longer and more involving health and safety training courses produce greater perceived improvements in ability than do shorter and less involving training courses. These results support what we all :"know" intuitively, but can now be supported empirically. The larger challenge is to recognize the value of describing and monitoring our interventions and linking these descriptions to outcomes. One paradigm that explicitly incorporates process measures into program evaluation is participatory action research. Such total immersion in the organization is not necessary to start monitoring intervention processes.

While thinking about and measuring process variables can be done atheoretically, we can make even larger strides if we think theoretically. Consider the following. We have all learned that the *sine non qua* of experimental design is the so-called pretest post control group design with random assignment. But is that always the case? In a provocative article, Rogers and Hunter (1996) argue otherwise. They describe two situations, not unlike those we face in our training impact research.. In their first situation, an intervention is implemented across the entire organization, with top management's support. Results of a preintervention -postintervention study shows a 50% improvement. In Rogers and Hunter's second situation, intervention and control groups are randomly assigned within the same organization. The interventions were implemented without visible top management support. (Top management strongly supported the study, but could not directly support the intervention in the selected groups). Analysis of this pretest-posttest control group design with random assignment produced 5% net improvement for the intervention group.

How might we interpret these two study results; a 50% improvement from a poorly designed study or a 5% improvement from an ideally designed study. Many of us would argue for the more modest 5% improvement based upon knowledge of research design. Rogers and Hunter warn us against such a reflexive conclusion. We know empirically that top management support for an intervention is strongly associated with maximum benefits from that intervention. In the first study a weak research design was paired with a strong intervention strategy. In the second study, a strong evaluation design was coupled with a weak intervention strategy.

They conclude:

The difference between the findings...is due, however to the difference

between effective and ineffective implementation and actually has nothing to do with the use of a control group (Rogers & Hunter, 1996, p.193).

Knowledge of intervention theory and empirical findings helped us draw a more informed conclusion about these two pieces of impact research.

Summary

This paper focused on but two of many challenges that confront research on the impact of health and safety training. We want to highlight the need to learn what information our various stakeholders and potential audiences demand of us and the most effective ways of presenting such information to them. These are empirical questions. One place to start is the vast continuous quality improvement literature where they are concerned with data displays and information formats.

We stressed several aspects of measurement that need to be addressed , including adequate psychometrics, minimizing response bias, awareness of the common methods variance in our design. The psychometric quality of our research is readily solved; their is lots of expertise available. Minimizing response bias is more challenging. There are no cookbooks and the knowledge base is increasing exponentially. Collectively, our research would become more credible if we at least could begin to address that issue. Reducing the problem of common method variance is extremely difficult. Grantees are not funded to conduct research - especially expensive research. Short of additional mechanism to fund impact research on worker health and safety training, common methods bias will continue to limit the validity of impact evaluations.

Secondly, we addressed the importance of describing and monitoring our interventions. It is equally important that we understand how theories of interventions, whether they focus on individuals, organizations, engineering controls, or pedagogical process, can inform the interpretation of our training impact studies. How do we address this issue? It is not a technical/methods issue. It is a “not thinking outside of the box” issue..

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