U.S. Breast Cancer Incidences by State explained by Air Toxics, Alcohol and Income

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Abstract

Increasing interest exists in correlating environmental factors to human cancer incidence rate data. In this study, women breast cancer incidence variations across the 50 United States were fitted to predicted variations in air toxics concentrations by state in the United States, alcohol consumption, and household income. In the regression equations developed, the intercept accounted for 70-77%, the air toxics represented 7-10%, state household median income 13%, and alcohol explained 10-13% of the breast cancer incidences, which compares with a 5% alcohol contribution to breast cancer incidences reported in a recent epidemiological study. The EPA National-scale Air Toxics Assessment is the source of the air toxics cancer risk estimates. Three air toxics in combination with alcohol consumption explained women breast cancer incidence rate variations by state with a correlation coefficient squared (r^2) of 0.68. Adding state median household income increased the correlation coefficient squared (r^2) to 0.72.

Implications

Air toxics are estimated to explain 7-10% of women breast cancer incidence rates in the United States. The uncertainty assigned to the air toxics contribution is at a minimum a factor of 2.5. The air toxics of importance are organic compounds that include benzene and trichloroethylene. These findings may be used to prioritize research, monitoring, and regulatory intervention activities that reduce potential cancer risks to the United States population.

Introduction

In the early 1990s, the U.S. EPA undertook the Cumulative Exposure Project (CEP) with the goal of modeling annual ambient air concentrations of 148 air toxics and predicting their

associated cancer risk (Rosenbaum et al., 1999; Woodruff et al., 1998). Historical modeled national estimates suggested that ambient concentrations of HAPs exceeded benchmark risk levels for cancer and non-cancer end points in many areas of the country (Caldwell et al., 1998; Woodruff et al., 1998 & 2000). Boffetta and Nyberg (2003) examined the contribution of environmental factors to cancer risk and concluded just the opposite that there was insufficient data to estimate the cancer risk posed by outdoor air pollution. Ten years after Bofetta and Nyberg (2003) in 2013, the specialized cancer agency of the World Health Organization, the International Agency of Research on cancer (IARC), announced that it had classified outdoor air pollution as carcinogenic to humans.

The availability of nationwide ambient monitoring for the criteria air pollutants (carbon monoxide, lead, nitrogen dioxide, ozone, particulate matter, and sulfur dioxide) makes assessment of exposure and risk for these chemicals feasible. Considerably less is known about the distribution of exposure to and risk from the wide range of hazardous air pollutants (HAPs; also known as "air toxics") identified by Congress in the Clean Air Act Amendments. Less information is known because nationwide ambient monitoring is impractical given the number of air toxics and their diverse chemical monitoring needs (Caldwell et al., 1998; Morello et al., 2001; Woodruff et al., 1998). To analyze whether a data relationship exists between cancer risk and air toxics, this study used the U.S. EPA's (2015) 2011 National-scale Air Toxics Assessment (NATA). The NATA estimates aggregated at the State level were used. The NATA estimates were compared with breast cancer incidence rate variations across the fifty United States.

Materials and Methods

Epidemiological studies were downloaded and reviewed to identify air toxics with a reported link to breast cancer incidence rates in women. The U.S. EPA (2015) National Air Toxics Assessment study estimated the cancer risk associated with 179 air toxics plus diesel combustion particulate matter (PM) for each of the fifty United States. Alcohol consumption per capita by state were downloaded from the National Institute on Alcohol Abuse and Alcoholism (La Vallee and Yi, 2011). The National Cancer Institute (2016) latest 5-year average breast cancer incidence rate for each of the fifty United States were the observed data.

A simple regression analysis was conducted to identify those air toxics with a positive correlation (r) with the breast cancer incident rate variations across the fifty United States. Those air toxics with a positive correlation to breast cancer incidence rates variations by state were reviewed for confirmation of causality by epidemiological studies. Only positively correlated air toxics with epidemiological support coupled with alcohol consumption were considered for data fit using a multiple linear regression program.

This study used a multiple linear regression program to determine whether the NATA 2011 predicted cancer risk from air toxics could help explain the variation in breast cancer incidence rates across the fifty United States. To understand whether this study's linear regression equations might have predictive capability, the risk factor alcohol with a quantified breast cancer incidence rate was included into the analysis. The Seitz et al. (2012) epidemiological study assigned a 5% breast cancer incidence rate from alcohol consumption in the United States.

To assess uncertainty in the contribution of components in the linear regression equations, this study reviewed the range in the % contribution of air toxics and alcohol to breast cancer incidence from different multiple linear regression equations developed in this study and also examined the difference in the linear regression predicted contribution of alcohol compared with that arrived at by the epidemiological study. For the linear regression models reported, each air toxic constituent and alcohol were included only if meaningful at the 95% confidence interval.

Epidemiological studies that have linked air toxics to breast cancer

Epidemiological studies have examined the link between air toxics in the occupational workplace and breast cancer incidence and mortality. Occupational exposures among enlisted women in the U.S. Army[12] and women in various professions in Israel (Shaham et al., 2006) and a study of a fairly small sample of women from their work at a shoe factory in Florence, Italy (Costantini et al., 2009), all support a relationship between exposure to benzene and later development of breast cancer. Hansen (1999) and Band et al. (2000 and 2002) linked breast cancer cases with solvent-using occupations - after adjusting for age, alcohol use, family history of breast cancer, and previous breast biopsy. Cantor et al. (1995) linked workplace exposure to styrene and several organic solvents (methylene chloride, carbon tetrachloride, and formaldehyde). Organic solvent-using occupations include laundry and dry-cleaning, aircraft and motor vehicle repair,

the publishing and printing industries, and farm workers. Animal studies examining the carcinogen of metals such as arsenic, hexavalent chromium, nickel, cadmium, and cobalt have not reported increases in mammary tumors (National Academies Press, 2012). 4,4'-Methylenebis(2-chloroaniline) (MBOCA) is an organic chemical that is a byproduct of polyurethane processing that includes rubber manufacturing. MBOCA has caused breast cancer in rats, mice and dogs that ate MBOCA for over a year (ATSDR, 2016).

Breast cancer incidence rate data

The most recent five-year average female breast cancer incidence rates available for each of the fifty United States were downloaded from the National Cancer Institute (2016) and reviewed. The breast cancer incidence data for the United States do not include data for Nevada but does include data for the District of Columbia. Arkansas has the lowest reported breast cancer incident rate at 108 per 100,000 women. The District of Columbia has the highest reported breast cancer incident rate at 142 per 100,000 women. The difference between the highest and lowest incidence rates by state is 34 breast cancer incidences per 100,000 women or 30% of the lowest breast cancer incidence rate of 108 per 100,000 women in Arkansas. Breast cancer incidence rates in the developed world (e.g., United States) are significantly higher than in the developing world. For example, breast cancer incidence rates in the U.S. are about 2.3 times higher than in developing countries when age adjusted (Ferlay et al., 2012).

U.S. EPA's NATA.

The NATA and its predecessor the Cumulative Exposure Project established a means for using source emission data to derive estimates of human exposure to air toxics (Rosenbaum et al., 1998) and human cancer risk (Caldwell et al., 1998; Woodruff et al., 1998 & 2000). This study downloaded the NATA 2011 cancer risk estimates by air toxic aggregated at the State level (EPA, 2015). The U.S. EPA's most recent national-scale air toxics assessment was conducted using the 2011 air toxic emissions inventory. This NATA calendar year 2011 study offers a snapshot in time of the predicted health risks associated with 179 air toxic chemicals. NATA combines source emission data (i.e., TRI data, databases from the U.S. EPA's Maximum

Achievable Control Technology program, and emissions estimates for mobile and area sources) with meteorology (wind speed and direction) in a Gaussian dispersion model called the Human Exposure Model version 1.3.1 (HEM-3) for the 179 HAPs and diesel PM. A photo-chemical grid model, Community Multi-Scale Air Quality Modeling System (CMAQ), is then used for a subset of 40 HAPs and diesel PM to improve mass conservation. The air toxic annual ambient concentrations are input into the Hazardous Air Pollution Exposure Model 7 (HAPEM7) that incorporates activity patterns influence on personal exposure to ambient air toxic concentrations. From the HAPEM7 human concentration exposure estimates, air toxic concentrations from the NATA 2011 study were converted to cancer risk by applying inhalation unit risk factors following the U.S. EPA standard methods. For cancer, even though the type (e.g., liver, blood, lung) and weight of evidence (e.g., known, suspected, or possible) varied by chemical, aggregate risk was estimated as the sum of individual chemical risks. The cancer risk estimates are considered by the U.S. EPA to be "upper-bound" estimates" that are a plausible upper limit to the true probability that an individual will contract cancer over a 70 year lifetime as a result of a given hazard (such as exposure to a toxic chemical)".

EPA (2015) reports the following emission source categories are included in the inventory and subsequent assessment. Major emissions sources were "stationary facilities that emit or have the potential to emit 10 tons of any one toxic air pollutant or 25 tons of more than one toxic air pollutant per year" (e.g., electric utility power plants, oil refineries). Area and other emissions sources were "sources that generally have smaller emissions on an individual basis than 'major sources' and are often too small or ubiquitous in nature to be inventoried as individual sources"; this may include smaller facilities (e.g., dry cleaning facilities, gas station/automobile repair) or other sources such as wildfires. On-road mobile sources were "vehicles found on roads or highways." Nonroad mobile sources were "mobile sources not found on roads and highways (e.g., airplanes, trains, lawn mowers, construction vehicles, farm machinery)." In addition, background concentrations are estimated, which represent exposure from "natural sources, persistence in the environment of past years' emissions and long-range transport from distant sources." NATA cancer risk estimates were calculated for the year 2011 and use 2010 census tracts.

Alcohol

Seitz et al. (2012) report a significant increase of the order of 4% in the risk of breast cancer is present at intakes of up to one alcoholic drink/day. Heavy alcohol consumption, defined as three or more drinks/day, is associated with an increased risk by 40-50%. They report this translates into up to 5% of breast cancers attributable to alcohol in northern Europe and North America. La Vallee and Yi (2011) report on apparent alcohol consumption per capita for each of the fifty United States. These data were downloaded and used in this study's analysis. Utah has the lowest and New Hampshire the highest apparent alcohol consumption at 1.33 gallons per capita per year and 4.38 gallons per capita per year. The difference between the highest and lowest alcohol consumption rates is 3.05 gallons per capita per year, which is 230% of the lowest alcohol consumption rate per capita in Utah.

Results

Breast cancer incidence rate data at the State level were the observed or y-variables. The NATA 2011 predicted cancer risk estimates by air toxic for each of fifty United States areas (Nevada omitted and the District of Columbia included) were the independent variables. Alcohol consumption per capita variation by state for each of the fifty United States areas was another added independent variable.

A linear regression program was run to identify those air toxics with a significant positive correlation compared with the breast cancer incidence variation by state. Benzene (r=0.66), 1,3-butadiene (0.62), naphthalene (0.59), beryllium (0.39), ethylbenzene (0.38), trichloroethylene (0.35), tetrachloroethylene (0.30), and arsenic (0.32) showed the highest positive data correlations. The NATA 2011 cancer risk for the metals including arsenic, beryllium and cadmium reflect the fact that these metal emissions in the 2011 emission inventory represented only the mass of the metal. 4,4'-Methylenebis(2-chloroaniline) (MBOCA) (r=0.23) also showed a positive data correlation. Formaldehyde and styrene linked to breast cancer in epidemiological and ecological studies (Coyle et al., 2005) showed no correlation with state variations in breast cancer incidence rates.

The NATA 2011 predicted variation of cancer risk from the air toxics benzene, 1,3-butadiene, ethylbenzene, and napthalene by state are highly inter-correlated. The correlation (r) between benzene and 1,3-butadiene is 0.95, between benzene and ethylbenzene is 0.71, and benzene and napthalene is 0.84. This study removed all but the highest correlated air toxic benzene from the multiple regression model analysis. Similarly, the correlation (r) between arsenic, beryllium and cadmium are also highly inter-correlated across the 50 United States. The correlation (r) between arsenic and beryllium is 0.77 and between arsenic and cadmium is 0.74.

This study identified two multiple regression equations that are statistically significant at the 95% confidence interval in explaining breast cancer incidence rate variations across the fifty United States. The two regression equations used NATA calendar year 2011 cancer risk estimates for the air toxics benzene and trichloroethylene along with alcohol consumption per capita by state. The two equations differ in whether a third air toxic (4,4'-Methylenebis(2-chloroaniline) (MBOCA)) is included. The two data fit multiple regression equations that have correlation coefficient squared (r^2) values of 0.60 and 0.68 are as follows:

$$\text{Breast Cancer Incidence Rate (per 100k women)} = 96 + 2.68\ CR_{Benzene} + 31.3\ CR_{TCE} + 5.35\ \text{Alcohol} \qquad (r^2=0.60) \qquad (1)$$

$$\text{Breast Cancer Incidence Rate (per 100k women)} = 95 + 2.64\ CR_{Benzene} + 35.8\ CR_{TCE} + 5.54\ \text{Alcohol} + 322\ CR_{MBOCA} \qquad (r^2=0.68) \qquad (2)$$

Where $CR_{Benzene}$, CR_{TCE}, and CR_{MBOCA} denote the NATA calendar year 2011 modeled cancer risks variations across the 50 United States for these compounds. Alcohol denotes the per capita alcohol consumption in gallon per year by state that varies across the 50 United States. The intercept of 95-96 per 100 k women in the multiple regression equations [1] and [2] account for 77% and the air toxics benzene and trichloroethylene with or without MBOCA explain 10% of the breast cancer incidence rate data. Alcohol explains 13% of the breast cancer incidence rate data, which compares with alcohol accounting for 5% of the United States breast cancer incidences according to an epidemiological study (Seitz et al, 2012). The air toxics benzene, TCE and MBOCA can be thought of as surrogates for a number of other correlated air toxics.

Figure 1 and **Figure 2** graphically show the three and four term multiple regression equations [1] and [2] data fit to the breast cancer incidence rate for the fifty United States. Because the regression equations predict alcohol explains 13% of breast cancer incidences, which is about 2.5 times that reported by an epidemiological study, the explained contribution from air toxics (10%) to breast cancer should at a minimum be assigned a similar factor of 2.5 uncertainty.

Figure 1 here

Figure 2 here

Discussion

This study developed two regression equations that explain breast cancer incidence rate (2008-2012) variations across the fifty United States. The two regression equations explain breast cancer incidences using NATA 2011 cancer risk estimates for two or three air toxics coupled with alcohol consumption per capita by state. The strongest multiple regression correlation squared (r^2=0.68) was obtained using NATA cancer risk estimates for three air toxics (benzene, TCE and MBOCA) combined with alcohol consumption per capita. One-quarter or 25% of breast cancer incidences are explained by the three air toxics in combination with alcohol consumption. 75% of breast cancer incidences are from factors that the causation is more complex than air toxics and alcohol can explain.

As noted earlier, breast cancer incidence rates per 100,000 women in the U.S. are about 2.3 times higher than in developing countries when age adjusted (Ferlay et al., 2012). One explanation for why fewer breast cancer incidences are reported in less developed countries is that lower air toxic concentrations are present in less developed countries. For example, McCarthy et al. (2006) identified the mean remote concentration of benzene and tetrachloroethylene as 0.14 $\mu g/m^3$ and 0.26 $\mu g/m^3$, respectively, or about 7 times and 30 times lower than in United States urban areas. The authors concluded that background levels make up 6-17% of a typical United States urban concentration for a number of air toxics.

Other studies have documented racial and economic disparities in the location of Toxic Release Inventories and other treatment, storage, and/or disposal facilities in the United States (Morello-Frosch et al., 2001; Pastor et al, 2001). These studies have suggested that the lowest income populations have little exposure because of lack of economic and industrial development, and

areas with the highest income have little exposure because of increased mobility and political will. According to their hypothesis, the burden of exposure would fall on low- to middle-income working-class populations.

Because of these other studies thoughts, this work investigated whether household income added as a factor would improve the predicted explanation of breast cancer incidences across the United States. What this study found was that the U.S. Census Bureau (2016) reported variation in state household median income (2011-2013 average) was positively correlated and does not show a negatively correlated or a U-shaped pattern compared with breast cancer incidence variations by state. Mississippi has the lowest and Maryland the highest median household incomes at $40,194 per year and $69,826 per year. The difference between the highest and lowest median household incomes is $29,632 or about 75% of the lowest median household income state of Mississippi.

Adding household median income variations by state improves the linear regression data fit squared correlation (r^2=0.72) above that of either equations [1] or [2]. In this linear regression expression, each of the five terms shown below is meaningful at the 95% confidence level:

$$\text{Breast Cancer Incidence Rate (per 100k women)} = 88 + 1.98\ CR_{Benzene} + 29.1\ CR_{TCE} + 4.42\ \text{Alcohol} + 313\ CR_{MBOCA} + 0.248 \times \text{3-Year Average Median Household Income (\$ k)} \quad (r^2=0.72) \qquad [3]$$

The intercept of 88 per 100 k women in this multiple regression equation [3] accounts for 70% and the three air toxics combined explain 7% of the breast cancer incidence rate data. Alcohol explains 10% of the breast cancer incidence rate data, which is twice the 5% breast cancer incidences identified by an epidemiological study (Seitz et al., 2012). Household median income (3-year average) explains 13% of the breast cancer incidence rate data. The air toxics benzene, TCE and MBOCA can be thought of as surrogates for a number of other correlated air toxics. Monitoring studies conducted in communities have shown personal exposures tend to be higher relative to parallel measurements made indoors and outdoors (Payne-Sturges, 2004). For many air toxics (e.g., benzene) indoor concentrations dominate exposure. Compounds associated with vehicle emissions were found to have similar indoor and outdoor concentrations. In a similar study in three Minnesota communities, personal exposure to VOCs was consistently higher than

indoor and outdoor concentrations (Sexton et al., 2004). The Minnesota work has been confirmed in other United States locales (Municipality of Anchorage, 2011).

In conclusion, this work developed three linear regression models to explain breast cancer incidence rate variations across the fifty United States. Higher income, alcohol consumption and air toxics exposure explain 13%, 10-13%, and 7-10%, respectively, of breast cancer incidence variations by state across the United States. The regression models predictions that alcohol explains 10-13% of breast cancer incidences ranges from 2 to 2.5 times more than the 5% contribution assigned to alcohol reported by an epidemiological study (Seitz et al., 2012). This study uses the regression models over prediction factor of 2 to 2.5 for alcohol compared to the epidemiological study finding as the minimum likely uncertainty that should be assigned to the regression models assigned contributions of air toxics (7-10%) and household income (13%) to breast cancer incidences by state in the United States. This study finds that states with higher economic status have higher breast cancer incidences. These findings may be used to prioritize research, monitoring, and regulatory intervention activities that reduce potential cancer risks to the United States population.

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Figure Captions

Figure 1. Three term regression equation (1).

Figure 2. Four term regression equation (2).

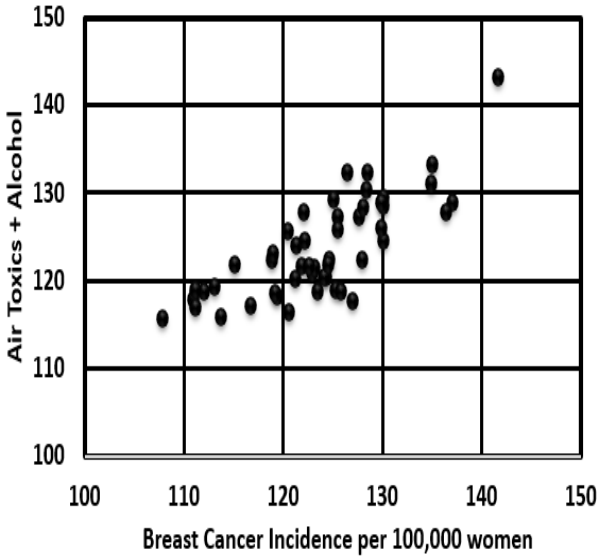

Figure 1. Three term regression equation (1).

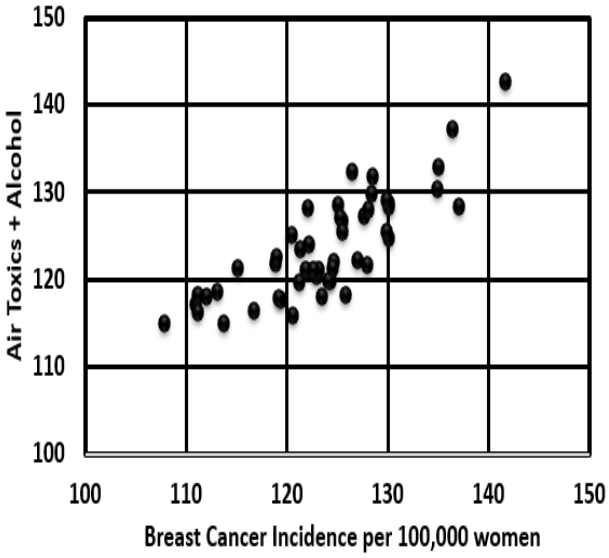

Figure 2. Four term regression equation (2)