Examination of the Human Health Issues of Sewage Sludge and Municipal Solid Waste Incineration in North Carolina

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1. Introduction

In recent years, the Federal government has extended the Renewable Electricity Production Tax Credit to facilities that produce energy from the incineration of municipal solid waste (MSW), thereby giving these facilities a tax credit of 1.1¢/kWh produced and a financial incentive to initiate this technology as a means to produce energy while managing waste.[1] This report focuses on the facilities in North Carolina that manage the sewage sludge produced from wastewater treatment plants. North Carolina has six such extant facilities that operate air emissions sources or air cleaning devices and appurtenances in the synthetic minor fee class. Two of these have permits on file for incinerators, while the rest generate emissions from generators and storage silos.

Synthetic minor facilities are EPA-defined "major" point sources of polluting emissions that have installed a control device after November 15, 1990. These facilities are subject to federal regulations under the National Emissions Standards for Hazardous Air Pollutants (NESHAP), and must apply for a Title V permit as classified by the Clean Air Act.[2] After years of delay, in March 2011 the US EPA promulgated NESHAP for Major Sources: Industrial, Commercial and Institutional Boilers, and Process Heaters. Finalization of the standards was due to a court order from a lawsuit brought by the Sierra Club. The rules also apply to sewage sludge incinerators. On May 16, 2011, in response to an industry lawsuit, the EPA delayed implementation of the standards until all legal challenges are resolved.[3]

Although these six facilities are not necessarily designated as waste-to-energy facilities that qualify for the tax credit, they provide a model of waste incineration that portents the potential health issues that may arise when more waste-to-energy facilities

are proposed and built. The chemical compositions of MSW and sewage sludge are similar, and both processes share the risks to human health from toxic organic compounds (TOCs), like dioxins/furans and polycyclic aromatic hydrocarbons (PAHs), that are produced from incomplete combustion.

2. Synthetic minor point sources in North Carolina

There are six synthetic minor point sources in NC, two of which have permits to run incinerators. Guilford County is the home of the City of High Point – Eastside Wastewater Treatment Plant (WWTP), which runs a fluidized bed sewage sludge incinerator and carries a Venturi impingement tray scrubber as a control system. According to their latest compliance inspection report, the incinerator runs 24 hours per day, 5 days per week, for 52 weeks per year.[4] The facility burns solid sludge at a maximum rate of 2500 dry lb/hr, and the fluidized bed incinerator (FBI) has not been formally stack tested since 1998. It will complete a round of testing when the facility reaches its next allowable maximum sewage sludge charge rate of 3000 dry lb/hr, which it has already been approved for. The emissions testing values that are discussed in subsequent sections and listed in the appendices are measured by the facility operators themselves or are determined via equations as recommended by the EPA.

The Rocky River Regional WWTP in Cabarrus County runs a multiple hearth sewage sludge incinerator with a conditioning system consisting of a spray quencher and two-tray impingement cooler, and a wet electrostatic precipitator. The facility's last performance tests were conducted in July 1995, and the actual emissions that are

published in air permit reviews and in the online inventory are presumably calculated based on 2008 figures when 5543.5 tons of dry sludge were processed that year over a period of 4702 hours. The facility does keep track of its own metals and hydrocarbons concentrations in the exit gas from the incinerator stack, which is analyzed every other month.[5]

Both facilities are in compliance with the various regulations that they are subject to and emit, based on calculations, much lower levels of emissions than they are technically allowed. In the county of Iredell, the city of Statesville runs the Fourth Creek Wastewater Treatment Plant with a fuel-fired emergency generator that is listed as a synthetic minor point source. The City of Graham WWTP in Alamance County has permits for a lime silo and two diesel-powered generators with regulated NOx emissions. Also in Alamance, the city of Burlington runs the East Burlington WWTP with an activated carbon storage silo, an oxidation reactor, a hydrated lime storage silo, and two diesel fuel-fired generators. The Western Wake Regional Water Reclamation Facility in Apex, Wake County has permits for three diesel-fed generators. The rest of this report will focus on the two sewage sludge incinerators.

3. Types of emissions from these incinerators

Emission predictions vary widely between incinerators of even the same type. Multiple hearth incinerators are the most common in the United States, and generally emit less particulate matter than fluidized bed incinerators that process the same amount of sludge. However, they also tend to emit more partially combusted hydrocarbons,

potentially polycyclic aromatic hydrocarbons (PAHs) and dioxins/furans. North Carolina has one of each type; however, conclusions cannot be drawn simply from knowing the design of the incinerator. A huge variety of factors play into the types and amounts of emissions from each incinerator, and both facilities, as well as possible future ones, should be analyzed separately. Among these factors include the feed rate of sludge into the incinerator, the composition of the metals and organic matter already found in the sludge, control and scrubber features at the facility, airflow, etc. Since there are so many variables that determine the emissions from each incinerator, estimating the risks and hazards of these facilities is far from accurate, reducing the impact of any warning flags that may crop up in preliminary assessments. The EPA's most recent, from 1995, compilation of air pollutant emission factors contains a thorough explanation of the different types of incinerators and the different variables that goes into calculating the emission factors of the most notable chemicals and metals emitted.[6]

Emissions of heavy metals and partially burned hydrocarbons, containing unknown concentrations of toxic organic compounds (TOCs) like PAHs and dioxins/furans, are usually given the most public attention. Metals are known and well-understood hazards, making it reasonable for officials to plan for their output and assess their risks. However, little is known about TOCs other than that they present risks for cancer at even low doses, making it difficult to take their toxicity into account for risk assessments. Scientists have demonstrated that the most toxic dioxin congener, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), upregulates the aryl hydrocarbon (AH) receptor in human cells, inducing gene expression in a metabolic pathway and potentially initiating a broad range of symptoms at even low exposures. Other structurally-similar chemicals,

like other congeners of dioxins, furans, and polychlorinated biphenyls (PCBs), also induce toxicity via the same mechanism as TCDD, although with less efficacy because of the particular way that TCDD spatially interacts with the AH receptor.[7] Beyond this knowledge, scientists are unclear as to how inducing the AHR pathway leads to toxicity and the dosage at which these chemicals present symptoms. As such, dioxin-like compounds are quantified as toxic equivalents (TEQs) of TCDD, the most active inducer of the AHR and the most toxic of these chemical species. While these biomarkers of AHR induction are noted scientifically, their implications on human health are unclear and scientists are divided as to whether these small biochemical changes at low levels of exposure to TCDD and related compounds are merely background responses in the body or the precursors of toxicity and potential harm to health. [7]

This debate over the toxicity and level of danger that TCDD presents may be a factor in why the EPA has yet to set a reference dose (RfD; a maximal daily allowance of a substance to lead to a minimal risk, as defined by the EPA). In 2003, a group of scientists proposed an RfD based on reviews of regulatory opinions of other agencies such as the World Health Organization (WHO) and the U.K. Food Standards Agency, and findings from the 5,000+ strong arsenal of research papers devoted to the toxicity of dioxin-like compounds. Their conclusions are that while cancer risks from dioxins are present, non-cancer, mainly developmental, toxicity may be even more of a hazard. "A no-observed-adverse-effects level (NOAEL) of 13 ng/kg (maternal body burden) was identified as the most pertinent for deriving an RfD for humans." [8]

For the general populace, an RfD for dioxin (toxic equivalency) of 1-10 pg/kg-day should prevent non-cancer effects.[8] The Eastside WWTP in High Point released 1.26

mg of TCDD in 2008 over the course of a year. This number may seem insignificant, especially spread out over a wide region and considering weather patterns. However, TCDD and related compounds are extremely hydrophobic and bioaccumulate up the food chain; in fact, most human exposure is oral and is from consuming food. This may present a problem since incinerators tend to be built in rural, agricultural areas, where exposure to dioxins up the food chain is more likely from livestock and fish. There are few studies that examine the long-term risks to communities adjacent to dioxin-producing facilities, and the risks to the population surrounding the particular facilities in North Carolina were not properly examined in the case of dioxin toxicity. More longitudinal studies are needed to determine the pertinent risks of real levels of contamination.

Another danger, however, is the huge volume of particulate matter emitted from these facilities. The size of these particles allows them to carry other harmful substances, like heavy metals and TOCs, over large distances aerially and directly into human bodies. Even disregarding their potential to act as a vector of harmful chemicals, particulate matter has been shown to irritate the respiratory system. Asbestos, a proven carcinogen, can be classified as particulate matter and provide a well-known example of the health hazards of particulate matter.

An annotated bibliography published by the American Lung Association in 2006 compiled research highlights from recent papers that noted the harmful effects of particulate matter on human health. The summary included (with citations): [9]

- A long-term study showing risk of premature death attributable to PM is three times greater than previously reported

- Studies linking daily exposures in PM with increased hospital admissions for strokes, congestive heart failure, heart attacks, COPD and other respiratory problems
- A toxicology study showing links between exposure to PM2.5 at levels near or below the current standards and development of atherosclerotic plaques
- Many studies elucidating the biological mechanisms and pathways for cardiovascular effects
- Studies linking prenatal exposure to air pollution with increased risk of low birth weight, preterm birth, infant mortality, and cancer
- Research showing that coarse particles exacerbate respiratory disease
- Intervention studies showing that reductions in air pollution yield measurable improvement in children's respiratory health and reduction in premature deaths
- Policy analyses showing the need for strong annual and daily fine particle standards to protect susceptible populations and provide equivalent levels of protection to different regions of the country.

Although particulate matter is ubiquitous in the environment and has natural as well as anthropogenic sources, the release of high levels of PMs, particularly fine materials less than 10 micrometers in diameter, may have harmful and unforeseen health effects in communities near incineration facilities. In 2008 the Rocky River WWTP released about 400 pounds of PM2.5 and another 400 pounds of PM10, and although these figures seem insignificant over 365 days, the amount adds up over time and with exposure. Studies should be conducted in these communities for evidence of negative health effects, such as increased incidence of respiratory disease, asthma, and cardiovascular problems.

4. Recommendations of outside agencies

The Health Protection Agency (HPA) of the U.K. published "The Impact on Health of Emissions to Air from Municipal Waste Incinerators" in September 2009 and concluded that "modern, well managed incinerators make only a small contribution to local concentrations of air pollutants. It is possible that such small additions could have an impact on health but such effects, if they exist, are likely to be very small and not detectable."[10] In reaching this conclusion, the HPA cited a 2004 report created for the Department for Environment, Food, and Rural Affairs (Defra, U.K.) by the outside firm Enviros Consulting Ltd. The report estimates for 2000 in the U.K. emissions on the order of 400 ng/T TEQ of dioxins and furans, and 0.1 mg/T TEQ of dioxin-like PCBs. Despite such levels, the study cites other papers that deny links between distance to incinerators and blood dioxin levels, and any connections between living in proximity to incinerators and cancer. Therefore, the Enviros study also rejects the link between living near an incinerator and incurring toxic health effects.

Whereas this study, although thorough, deems inconclusive and incomplete evidence as not enough to implicate incinerators, another review published by the British Society for Ecological Medicine (BSEM) on "The Health Effects of Waste Incinerators" in June 2008 reaches different conclusions from the same set of evidence. The BSEM concludes that the inconclusive evidence should prevent future incinerators from getting permitted and insists that more tests are necessary to establish the safety of municipal waste incinerators. The BSEM specifically points to new evidence that highlights the previously understated risks of particulate matter on respiratory and cardiovascular health. Another worry of the BSEM is that the published emissions of dioxins are

underreported, and that the current technology cannot accurately capture the true output of dioxins, particularly those produced at shutdown and startup of the facilities.

The BSEM's conservative approach to risk assessment is more inclined towards protecting health and holding the standard of health above all other considerations (the group is composed of doctors, after all). In light of their strong sense of duty toward maintaining health, their recommendations hold more weight in terms of determining the health effects of other municipal waste incinerators in different localities and run under different circumstances. Their conclusions are as follows: [12]

1. Incineration does not remove waste. It simply converts it into another form (gas, particulates, ash) and these new forms are typically more hazardous though less visible than in the original form.
2. Large epidemiological studies have shown higher rates of adult and childhood cancers and of birth defects around incinerators. Smaller studies and a large body of related research support these findings, point to a causal relationship, and suggest that a much wider range of illnesses may be involved.
3. Recent research has confirmed that particulate pollution, especially the fine particulate (PM2.5) pollution, which is typical of incinerator emissions, is an important contributor to heart disease, lung cancer, and an assortment of other diseases, and causes a linear increase in mortality. The latest research has found there is a much greater effect on mortality than previously thought and implies that incinerators will cause increases in cardiovascular and cerebrovascular morbidity and mortality with both short-term and long-term exposure. Particulates

from incinerators will be especially hazardous due to the toxic chemicals attached to them.

4. Other pollutants emitted by incinerators include heavy metals and a large variety of organic chemicals. These substances include known carcinogens, endocrine disruptors, and substances that can attach to genes, alter behaviour, damage the immune system and decrease intelligence. There appears to be no threshold for some of these effects, such as endocrine disruption. The dangers of these are self-evident. Some of these compounds have been detected hundreds to thousands of miles away from their source.
5. The danger of incinerating radioactive waste deserves special mention. Incineration converts radioactive waste into billions of radioactive particulates. These particulates make a near perfect delivery system for introducing the radioactive matter into the human body, where it can then act as an internal emitter of alpha or beta radiation. This type of radiation is qualitatively different, far more dangerous and far more sinister, than background radiation. There can be no justification for using this method of dealing with radioactive waste.
6. Modern incinerators produce fly ash which is much more toxic than in the past, containing large quantities of dioxin-rich material for which there is no safe method of disposal, except vitrification, a method not being used in the UK. Disposal of incinerator ash to landfill sites is associated with long-term threats to aquifers and water tables and the potential for accidents serious enough to require evacuation of an area.

7. The risks to local people that occur when incinerators operate under nonstandard working conditions have not been addressed, particularly the emissions at start-up and shutdown which may be associated with the release, within 2 days, of more dioxin than over 6 months of working under standard conditions.
8. The greatest concern is the long-term effects of incinerator emissions on the developing embryo and infant, and the real possibility that genetic changes will occur and be passed on to succeeding generations. Far greater vulnerability to toxins has been documented for the very young, particularly foetuses, with risks of cancer, spontaneous abortion, birth defects or permanent cognitive damage. A worryingly high body burden of pollutants has recently been reported in two studies of cord blood from new-born babies.
9. Waste incineration is prohibitively expensive when health costs are taken into account. A variety of studies, including that from the government, indicate that a single large incinerator could cost the tax payer many millions of pounds per annum in health costs. Put simply, the government's own data is demonstrating that incinerators are a major health hazard. With the predicted inclusion of the waste industry within the EU European Emissions Trading Scheme, local taxpayers, in areas with incinerators, will not only have to live within a polluted area but will be saddled with costs, under ETS, of millions of pounds per annum to pay for it.
10. Waste incineration is unjust because its maximum toxic impact is on the most vulnerable members of our society, the unborn child, children, the poor and the chemically sensitive. It contravenes the United Nations Commission on Human

Rights, the European Human Rights Convention (the Right to Life), and the Stockholm Convention, and violates the Environmental Protection Act of 1990 which states that the UK must prevent emissions from harming human health.

The emphasis of the BSEM report is minimizing the impact on human health, while the Enviros report for Defra seeks to find compromise in the best-use policy between the disparate waste management practices, combining concern for human health with pragmatism in U.K. society. Defra's conclusion is that waste incineration is not a poor strategy, with no red flags that should prevent its use. BSEM, however, under more stringent analysis, holds that more information is needed about incineration and reasonable alternatives should be explored.

5. Conclusion

The most important thing to draw from the different reports and official recommendations regarding waste incineration is the difficulty of establishing a risk factor for this practice. The sheer amount of studies and variables that exist are overwhelming, since any data could skew a conclusion one way or the other even though they are drawn from the same body of evidence. Both sides of the argument have rich sources of data; there are studies that both establish and reject linkages to health effects on communities living in proximity to incinerators.

In the morass of this information, the truth and irony is that not enough information is present. In 1998, the Science and Environmental Health Network published the Wingspan consensus statement on the Precautionary Principle, an ethical

guide that seeks environmental justice by placing the burden of proof for the safety of a process or activity on the instigators and proponents, not the public. Since the official declaration of the Principle by the SEHN and its affiliates, including the University of Massachusetts-Lowell, global organizations such as the United Nations, parts of the European Union, and the City of San Francisco have ratified the basic tenets of the Precautionary Principle as part of their environmental policies. The premise of the Principle is that "when an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically."[13] In this case with the incineration of municipal waste, the Principle applies because the cause and effect relationships between incineration emissions and the hazards to human health are so unclear and controversial; the risks are minimized if we as a society err on the side of caution and put the public's safety first.

As the issues with the new NESHAP Major Source regulations indicate, relying on governmental agencies to protect public health is at best frustrating and at worst risky. Not only did it require a lawsuit from a non-profit agency to finalize the NESHAP standards, those same standards are still being blocked by further bureaucratic red tape. More studies, particularly longitudinal ones of communities impacted by incinerators, are necessary to draw more rigorous conclusions. In light of the lack of information, among all of these studies and mounds of reports, the conservative approach that the British Society of Ecological Medicine advocates is the most reasonable for the goal of preserving human health. Waste incineration as a practice should be limited and used as a last ditch effort to prevent other more harmful waste management techniques, such as

mass landfills (depending on the type of waste). It is easy and convenient to assume that our society's massive waste problems can be simply burned into thin air with municipal waste incineration; however, even this process is a temporary fix that converts one format of waste into another.

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Appendix 1: List of allowable emitted substances from synthetic minor point sources in North Carolina

Acetaldehyde: http://www.epa.gov/ttnatw01/hlthef/acetalde.html

Acetaldehyde is mainly used as an intermediate in the synthesis of other chemicals. It is ubiquitous in the environment and may be formed in the body from the breakdown of ethanol. Acute (short-term) exposure to acetaldehyde results in effects including irritation of the eyes, skin, and respiratory tract. Symptoms of chronic (long-term) intoxication of acetaldehyde resemble those of alcoholism. Acetaldehyde is considered a probable human carcinogen (Group B2) based on inadequate human cancer studies and animal studies that have shown nasal tumors in rats and laryngeal tumors in hamsters.

Acetonitrile: http://www.epa.gov/ttnatw01/hlthef/acetonit.html

Acetonitrile has many uses, including as a solvent, for spinning fibers, and in lithium batteries. It is primarily found in air from automobile exhaust and manufacturing facilities. Acute (short-term) inhalation exposure results in irritation of mucous membranes. Chronic (long-term) exposure results in central nervous system effects, such as headaches, numbness, and tremors. No data are available on its carcinogenic effects in humans; EPA has classified it as a Group D, not classifiable as to human carcinogenicity. It can metabolize to hydrogen cyanide, which is the source of the toxic effects.

Acrolein: http://www.epa.gov/ttnatw01/hlthef/acrolein.html

Acrolein is primarily used as an intermediate in the synthesis of acrylic acid and as a biocide. It may be formed from the breakdown of certain pollutants in outdoor air or from the burning of organic matter including tobacco, or fuels such as gasoline or oil. It is toxic to humans following inhalation, oral or dermal exposures. Acute (short-term) inhalation exposure may result in upper respiratory tract irritation and congestion. No information is available on its reproductive, developmental, or carcinogenic effects in humans, and the existing animal cancer data are considered inadequate to make a determination that acrolein is carcinogenic to humans.

Acrylonitrile: http://www.epa.gov/ttnatw01/hlthef/acryloni.html

Exposure to acrylonitrile is primarily occupational: it is used in the manufacture of acrylic acid and modacrylic fibers. Acute (short-term) exposure of workers to acrylonitrile has been observed

to cause mucous membrane irritation, headaches, dizziness, and nausea. No information is available on the reproductive or developmental effects of acrylonitrile in humans. Based on limited evidence in humans and evidence in rats, EPA has classified acrylonitrile as a probable human carcinogen (Group B1).

Antimony: http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=331&tid=58

Exposure to antimony at high levels can result in a variety of adverse health effects. Breathing high levels for a long time can irritate your eyes and lungs and can cause heart and lung problems, stomach pain, diarrhea, vomiting, and stomach ulcers. In short-term studies, animals that breathed very high levels of antimony died. Animals that breathed high levels had lung, heart, liver, and kidney damage. In long-term studies, animals that breathed very low levels of antimony had eye irritation, hair loss, lung damage, and heart problems. Problems with fertility were also noted. In animal studies, problems with fertility have been seen when rats breathed very high levels of antimony for a few months. Ingesting large doses of antimony can cause vomiting. We don't know what other effects may be caused by ingesting it. Long-term animal studies have reported liver damage and blood changes when animals ingested antimony. Antimony can irritate the skin if it is left on it. Antimony can have beneficial effects when used for medical reasons. It has been used as a medicine to treat people infected with parasites.

Arsenic: http://www.cdphe.state.co.us/rma/resourcenotebook/Chemicals/arsenic.pdf

Chronic poisoning may involve weakness, anorexia, hepatomegaly, jaundice and gastrointestinal complaints followed by conjunctivitis and irritation of throat and respiratory tract, perforation of the nasal septum, hyperkeratosis, hyperpigmentation, eczematoid, and allergic dermatitis. Central nervous system symptoms may include numbness, burning, tingling of the hands and feet, fasciculation, gross tremors, ataxia, incoordination, shuffling gait, and mental confusion. Long-term inhalation of inorganic arsenic may injure blood vessels and/or heart. Arsenic is a known human carcinogen. Inhalation of arsenic increases frequency of chromosomal aberrations in peripheral lymphocytes. No data are available on the reproductive or developmental effects of arsenic.

Benzene: http://www.epa.gov/ttnatw01/hlthef/benzene.html

Benzene is found in the air from emissions from burning coal and oil, gasoline service stations, and motor vehicle exhaust. Acute (short-term) inhalation exposure of humans to benzene may cause drowsiness, dizziness, headaches, as well as eye, skin, and respiratory tract irritation, and, at high levels, unconsciousness. Chronic (long-term) inhalation exposure has caused various

disorders in the blood, including reduced numbers of red blood cells and aplastic anemia, in occupational settings. Reproductive effects have been reported for women exposed by inhalation to high levels, and adverse effects on the developing fetus have been observed in animal tests. Increased incidence of leukemia (cancer of the tissues that form white blood cells) have been observed in humans occupationally exposed to benzene. EPA has classified benzene as a Group A, human carcinogen.

B[a]P: see PAHs

Beryllium: http://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=184&tid=33

Beryllium can be harmful if you breathe it. The effects depend on how much you are exposed to and for how long. If beryllium air levels are high enough (greater than 1000 $\mu g/m^3$), an acute condition can result. This condition resembles pneumonia and is called acute beryllium disease Occupational and community air standards are effective in preventing most acute lung damage. Some people (1-15%) become sensitive to beryllium. These individuals may develop an inflammatory reaction in the respiratory system. This condition is called chronic beryllium disease (CBD), and can occur many years after exposure to higher than normal levels of beryllium (greater than 0.5 $\mu g/m^3$). This disease can make you feel weak and tired, and can cause difficulty in breathing. It can also result in anorexia, weight loss, and may also lead to right side heart enlargement and heart disease in advanced cases. Some people who are sensitized to beryllium may not have any symptoms. The general population is unlikely to develop acute or chronic beryllium disease because ambient air levels of beryllium are normally very low (0.00003-0.0002 $\mu g/m^3$). Swallowing beryllium has not been reported to cause effects in humans because very little beryllium is absorbed from the stomach and intestines. Ulcers have been seen in dogs ingesting beryllium in the diet. Beryllium contact with skin that has been scraped or cut may cause rashes or ulcers. Long term exposure to beryllium can increase the risk of developing lung cancer in people. The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have determined that beryllium is a human carcinogen. The EPA has determined that beryllium is a probable human carcinogen. EPA has estimated that lifetime exposure to 0.04 $\mu g/m^3$ beryllium can result in a one in a thousand chance of developing cancer.

Cadmium: http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=47&tid=15

Breathing high levels of cadmium can severely damage the lungs. Eating food or drinking water with very high levels severely irritates the stomach, leading to vomiting and diarrhea. Long-term exposure to lower levels of cadmium in air, food, or water leads to a buildup of cadmium in the kidneys and possible kidney disease. Other long-term effects are lung damage and fragile bones.

Carbon tetrachloride: http://www.epa.gov/ttnatw01/hlthef/carbonte.html,
http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=195&tid=35

- Chronic inhalation or oral exposure to carbon tetrachloride produces liver and kidney damage in humans and animals.
- EPA has not established a Reference Concentration (RfC) for carbon tetrachloride.
- The California Environmental Protection Agency (CalEPA) has established a chronic reference exposure level of 0.04 milligrams per cubic meter (mg/m^3) for carbon tetrachloride based on liver effects in guinea pigs. The CalEPA reference exposure level is a concentration at or below which adverse health effects are not likely to occur. It is not a direct estimator of risk, but rather a reference point to gauge the potential effects. At lifetime exposures increasingly greater than the reference exposure level, the potential for adverse health effects increases.
- ATSDR has established an acute duration (1-14 days) inhalation minimal risk level (MRL) of 1.3 mg/m^3 (0.2 parts per million [ppm]) based on liver effects in rats, and an intermediate duration (14-365 days) MRL of 0.3 mg/m^3 (0.05 ppm) also based on liver effects in rats. The MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure.
- The Reference Dose (RfD) for carbon tetrachloride is 0.0007 milligrams per kilogram per day (mg/kg/d) based on the occurrence of liver lesions in rats. The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious noncancer effects during a lifetime.
- EPA has medium confidence in the RfD based on (1) high confidence in the principal study on which the RfD was based because the study was well conducted and good dose-response was observed in the liver, which is the target organ for carbon tetrachloride toxicity; and (2) medium confidence in the database because four additional subchronic studies support the RfD. but reproductive and teratology endpoints are not well investigated; and, consequently, medium confidence in the RfD.

Chlorobenzene: http://www.epa.gov/ttnatw01/hlthef/chlorobe.html

Chlorobenzene is used primarily as a solvent, a degreasing agent, and a chemical intermediate. Limited information is available on the acute (short-term) effects of chlorobenzene. Acute inhalation exposure of animals to chlorobenzene produced narcosis, restlessness, tremors, and muscle spasms. Chronic (long-term) exposure of humans to chlorobenzene affects the central nervous system (CNS). Signs of neurotoxicity in humans include numbness, cyanosis, hyperesthesia (increased sensation), and muscle spasms. No information is available on the

carcinogenic effects of chlorobenzene in humans. EPA has classified chlorobenzene as a Group D, not classifiable as to human carcinogenicity.

Chloroform: http://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=52&tid=16

Breathing about 900 parts of chloroform per million parts air (900 ppm) for a short time can cause dizziness, fatigue, and headache. Breathing air, eating food, or drinking water containing high levels of chloroform for long periods of time may damage your liver and kidneys. Large amounts of chloroform can cause sores when chloroform touches your skin. It isn't known whether chloroform causes reproductive effects or birth defects in people. Animal studies have shown that miscarriages occurred in rats and mice that breathed air containing 30 to 300 ppm chloroform during pregnancy and also in rats that ate chloroform during pregnancy. Offspring of rats and mice that breathed chloroform during pregnancy had birth defects. Abnormal sperm were found in mice that breathed air containing 400 ppm chloroform for a few days.

Cobalt: http://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=372&tid=64

Cobalt can benefit or harm human health. Cobalt is beneficial for humans because it is part of vitamin B12. Exposure to high levels of cobalt can result in lung and heart effects and dermatitis. Liver and kidney effects have also been observed in animals exposed to high levels of cobalt. Exposure to large amounts of radiation from radioactive cobalt can damage cells in your body from the radiation. You might also experience acute radiation syndrome that includes nausea, vomiting, diarrhea, bleeding, coma, and even death. This would be a rare event.

Chromium: http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=61&tid=17

Breathing high levels of chromium(VI) can cause irritation to the lining of the nose, nose ulcers, runny nose, and breathing problems, such as asthma, cough, shortness of breath, or wheezing. The concentrations of chromium in air that can cause these effects may be different for different types of chromium compounds, with effects occurring at much lower concentrations for chromium(VI) compared to chromium(III). The Department of Health and Human Services (DHHS), the International Agency for Reseach on Cancer (IARC), and the EPA have determined that chromium(VI) compounds are known human carcinogens. In workers, inhalation of chromium(VI) has been shown to cause lung cancer. Chromium(VI) also causes lung cancer in animals. An increase in stomach tumors was observed in humans and animals exposed to chromium(VI) in drinking water.

DEHP: http://www.epa.gov/ttnatw01/hlthef/eth-phth.html

Incineration is not the main source in ambient air; see also phthalates

Bis(2-ethylhexyl) phthalate (DEHP) is used in the production of polyvinyl chloride (PVC). It exhibits low toxicity from acute (short-term) and chronic (long-term) exposures. Acute exposure to large oral doses of DEHP can cause gastrointestinal distress in humans. No information is available on the chronic, reproductive, developmental, or carcinogenic effects of DEHP in humans. Animal studies have reported increased lung weights and increased liver weights from chronic inhalation exposure to DEHP. Oral exposure has resulted in developmental and reproductive effects in rats and mice. A study by the National Toxicology Program (NTP) showed that DEHP administered orally increased the incidence of liver tumors in rats and mice. EPA has classified DEHP as a Group B2, probable human carcinogen

Dichlorobenzene(p): http://www.epa.gov/ttn/atw/hlthef/dich-ben.html

- Chronic exposure to 1,4-dichlorobenzene by inhalation in humans results in effects on the liver, skin, and CNS (e.g., cerebellar ataxia, dysarthria, weakness in limbs, and hyporeflexia).
- Animal studies have reported effects on the respiratory system, liver, and kidneys from inhalation exposure to 1,4-dichlorobenzene, while oral studies have reported effects on the blood, liver, and kidneys.
- The Reference Concentration (RfC) for 1,4-dichlorobenzene is 0.8 milligrams per cubic meter (mg/m^3) based on increased liver weights in rats. The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups), that is likely to be without appreciable risk of deleterious noncancer effects during a lifetime. It is not a direct estimator of risk but rather a reference point to gauge the potential effects. At exposures increasingly greater than the RfC, the potential for adverse health effects increases. Lifetime exposure above the RfC does not imply that an adverse health effect would necessarily occur.
- EPA has medium confidence in the study on which the RfC was based because the critical study employed an extensive reproductive protocol including histopathologic examination of tissues of adults and offspring; medium confidence in the database because there are a number of supporting studies for the developmental and reproductive toxicology database; and, consequently, medium confidence in the RfC.
- EPA has not established a Reference Dose (RfD) for 1,4-dichlorobenzene.
- ATSDR has established an intermediate oral minimal risk level (MRL) of 0.4 milligrams per kilogram body weight per day (mg/kg/d) based on liver effects in rats. The MRL is an estimate of

the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure.

Ethyl benzene: http://www.epa.gov/ttnatw01/hlthef/ethylben.html

Ethylbenzene is mainly used in the manufacture of styrene. Acute (short-term) exposure to ethylbenzene in humans results in respiratory effects, such as throat irritation and chest constriction, irritation of the eyes, and neurological effects such as dizziness. Chronic (long-term) exposure to ethylbenzene by inhalation in humans has shown conflicting results regarding its effects on the blood. Animal studies have reported effects on the blood, liver, and kidneys from chronic inhalation exposure to ethylbenzene. Limited information is available on the carcinogenic effects of ethylbenzene in humans. In a study by the National Toxicology Program (NTP), exposure to ethylbenzene by inhalation resulted in an increased incidence of kidney and testicular tumors in rats, and lung and liver tumors in mice. EPA has classified ethylbenzene as a Group D, not classifiable as to human carcinogenicity.

Ethylene dichloride:

Exposure to low levels of ethylene dichloride can occur from breathing ambient or workplace air. Inhalation of concentrated ethylene dichloride vapor can induce effects on the human nervous system, liver, and kidneys, as well as respiratory distress, cardiac arrhythmia, nausea, and vomiting. Chronic (long-term) inhalation exposure to ethylene dichloride produced effects on the liver and kidneys in animals. No information is available on the reproductive or developmental effects of ethylene dichloride in humans. Decreased fertility and increased embryo mortality have been observed in inhalation studies of rats. Epidemiological studies are not conclusive regarding the carcinogenic effects of ethylene dichloride, due to concomitant exposure to other chemicals. Following treatment by gavage (experimentally placing the chemical in the stomach), several tumor types were induced in rats and mice. EPA has classified ethylene dichloride as a Group B2, probable human carcinogen.

Formaldehyde: http://www.epa.gov/iaq/formalde.html

Formaldehyde, a colorless, pungent-smelling gas, can cause watery eyes, burning sensations in the eyes and throat, nausea, and difficulty in breathing in some humans exposed at elevated levels (above 0.1 parts per million). High concentrations may trigger attacks in people with asthma. There is evidence that some people can develop a sensitivity to formaldehyde. It has also been shown to cause cancer in animals and may cause cancer in humans. Health effects include eye, nose, and throat irritation; wheezing and coughing; fatigue; skin rash; severe allergic reactions. May cause cancer. May also cause other effects listed under "organic gases."

Hexachlorodibenzo-p-dioxin: see TCDD and dioxin-like compounds

Hexane, n-: http://www.epa.gov/ttnatw01/hlthef/hexane.html

Hexane is used to extract edible oils from seeds and vegetables, as a special-use solvent, and as a cleaning agent. Acute (short-term) inhalation exposure of humans to high levels of hexane causes mild central nervous system (CNS) effects, including dizziness, giddiness, slight nausea, and headache. Chronic (long-term) exposure to hexane in air is associated with polyneuropathy in humans, with numbness in the extremities, muscular weakness, blurred vision, headache, and fatigue observed. Neurotoxic effects have also been exhibited in rats. No information is available on the carcinogenic effects of hexane in humans or animals. EPA has classified hexane as a Group D, not classifiable as to human carcinogenicity.

Hydrogen chloride: http://www.epa.gov/ttnatw01/hlthef/hydrochl.html

Hydrochloric acid has many uses. It is used in the production of chlorides, fertilizers, and dyes, in electroplating, and in the photographic, textile, and rubber industries. Hydrochloric acid is corrosive to the eyes, skin, and mucous membranes. Acute (short-term) inhalation exposure may cause eye, nose, and respiratory tract irritation and inflammation and pulmonary edema in humans. Acute oral exposure may cause corrosion of the mucous membranes, esophagus, and stomach and dermal contact may produce severe burns, ulceration, and scarring in humans. Chronic (long-term) occupational exposure to hydrochloric acid has been reported to cause gastritis, chronic bronchitis, dermatitis, and photosensitization in workers. Prolonged exposure to low concentrations may also cause dental discoloration and erosion. EPA has not classified hydrochloric acid for carcinogenicity.

Lead: http://www.epa.gov/ttn/atw/hlthef/lead.html

Lead is used in the manufacture of batteries, metal products, paints, and ceramic glazes. Exposure to lead can occur from breathing contaminated workplace air or house dust or eating lead-based paint chips or contaminated dirt. Lead is a very toxic element, causing a variety of effects at low dose levels. Brain damage, kidney damage, and gastrointestinal distress are seen from acute (short-term) exposure to high levels of lead in humans. Chronic (long-term) exposure to lead in humans results in effects on the blood, central nervous system (CNS), blood pressure, kidneys, and Vitamin D metabolism. Children are particularly sensitive to the chronic effects of lead, with slowed cognitive development, reduced growth and other effects reported. Reproductive effects, such as decreased sperm count in men and spontaneous abortions in women, have been associated with high lead exposure. The developing fetus is at particular risk from maternal lead exposure,

with low birth weight and slowed postnatal neurobehavioral development noted. Human studies are inconclusive regarding lead exposure and cancer.

MEK (methyl ethyl ketone): http://www.epa.gov/ttnatw01/hlthef/methylet.html

Not so important → Methyl ethyl ketone is used as a solvent. Acute (short-term) inhalation exposure to methyl ethyl ketone in humans results in irritation to the eyes, nose, and throat. Limited information is available on the chronic (long-term) effects of methyl ethyl ketone in humans. Chronic inhalation studies in animals have reported slight neurological, liver, kidney, and respiratory effects. No information is available on the developmental, reproductive, or carcinogenic effects of methyl ethyl ketone in humans. Developmental effects, including decreased fetal weight and fetal malformations, have been reported in mice and rats exposed to methyl ethyl ketone via inhalation and ingestion. EPA has classified methyl ethyl ketone as a Group D, not classifiable as to human carcinogenicity.

Mn & compounds: http://www.epa.gov/ttnatw01/hlthef/manganes.html

Manganese is naturally ubiquitous in the environment. Manganese is essential for normal physiologic functioning in humans and animals, and exposure to low levels of manganese in the diet is considered to be nutritionally essential in humans. Chronic (long-term) exposure to high levels of manganese by inhalation in humans may result in central nervous system (CNS) effects. Visual reaction time, hand steadiness, and eye-hand coordination were affected in chronically-exposed workers. A syndrome named manganism may result from chronic exposure to higher levels; manganism is characterized by feelings of weakness and lethargy, tremors, a mask-like face, and psychological disturbances. Respiratory effects have also been noted in workers chronically exposed by inhalation. Impotence and loss of libido have been noted in male workers afflicted with manganism.

Mercury & compounds: http://www.epa.gov/airtoxics/hlthef/mercury.html

Chronic exposure to methyl mercury in humans also affects the CNS with symptoms such as paresthesia (a sensation of pricking on the skin), blurred vision, malaise, speech difficulties, and constriction of the visual field. Methyl mercury exposure, via the oral route, has led to significant developmental effects. Infants born to women who ingested high levels of methyl mercury exhibited mental retardation, ataxia, constriction of the visual field, blindness, and cerebral palsy. The major effect from chronic exposure to inorganic mercury is kidney damage. Animal studies have reported effects such as alterations in testicular tissue, increased resorption rates, and abnormalities of development. Mercuric chloride (an inorganic mercury compound) exposure has

been shown to result in forestomach, thyroid, and renal tumors in experimental animals. Chronic (long-term) exposure to elemental mercury in humans also affects the CNS, with effects such as erethism (increased excitability), irritability, excessive shyness, and tremors. Human studies are inconclusive regarding elemental mercury and cancer.

Methylchloroform: http://www.epa.gov/ttnatw01/hlthef/trichlor.html

Methyl chloroform is used as a solvent and in many consumer products. Effects reported in humans due to acute (short-term) inhalation exposure to methyl chloroform include hypotension, mild hepatic effects, and central nervous system (CNS) depression. Cardiac arrhythmia and respiratory arrest may result from the depression of the CNS. Symptoms of acute inhalation exposure include dizziness, nausea, vomiting, diarrhea, loss of consciousness, and decreased blood pressure in humans. After chronic (long-term) inhalation exposure to methyl chloroform, some liver damage was observed in mice and ventricular arrhythmias in humans. EPA has classified methyl chloroform as a Group D, not classifiable as to human carcinogenicity.

Methylene chloride: http://www.epa.gov/ttnatw01/hlthef/methylen.html

Methylene chloride is predominantly used as a solvent. The acute (short-term) effects of methylene chloride inhalation in humans consist mainly of nervous system effects including decreased visual, auditory, and motor functions, but these effects are reversible once exposure ceases. The effects of chronic (long-term) exposure to methylene chloride suggest that the central nervous system (CNS) is a potential target in humans and animals. Human data are inconclusive regarding methylene chloride and cancer. Animal studies have shown increases in liver and lung cancer and benign mammary gland tumors following the inhalation of methylene chloride.

Naphthalene: http://www.epa.gov/ttnatw01/hlthef/naphthal.html

Naphthalene is used in the production of phthalic anhydride; it is also used in mothballs. Acute (short-term) exposure of humans to naphthalene by inhalation, ingestion, and dermal contact is associated with hemolytic anemia, damage to the liver, and neurological damage. Cataracts have also been reported in workers acutely exposed to naphthalene by inhalation and ingestion. Chronic (long-term) exposure of workers and rodents to naphthalene has been reported to cause cataracts and damage to the retina. Hemolytic anemia has been reported in infants born to mothers who "sniffed" and ingested naphthalene (as mothballs) during pregnancy. Available data are inadequate to establish a causal relationship between exposure to naphthalene and cancer in humans. EPA has classified naphthalene as a Group C, possible human carcinogen.

Nickel: http://www.epa.gov/ttnatw01/hlthef/nickel.html

Nickel occurs naturally in the environment at low levels. Nickel is an essential element in some animal species, and it has been suggested it may be essential for human nutrition. Nickel dermatitis, consisting of itching of the fingers, hands, and forearms, is the most common effect in humans from chronic (long-term) skin contact with nickel. Respiratory effects have also been reported in humans from inhalation exposure to nickel. Human and animal studies have reported an increased risk of lung and nasal cancers from exposure to nickel refinery dusts and nickel subsulfide. Animal studies of soluble nickel compounds (i.e., nickel carbonyl) have reported lung tumors. EPA has classified nickel refinery dust and nickel subsulfide as Group A, human carcinogens, and nickel carbonyl as a Group B2, probable human carcinogen.

Perchloroethylene: http://www.epa.gov/ttnatw01/hlthef/tet-ethy.html

Tetrachloroethylene is widely used for dry-cleaning fabrics and metal degreasing operations. The main effects of tetrachloroethylene in humans are neurological, liver, and kidney effects following acute (short-term) and chronic (long-term) inhalation exposure. Adverse reproductive effects, such as spontaneous abortions, have been reported from occupational exposure to tetrachloroethylene; however, no definite conclusions can be made because of the limitations of the studies. Results from epidemiological studies of dry-cleaners occupationally exposed to tetrachloroethylene suggest increased risks for several types of cancer. Animal studies have reported an increased incidence of liver cancer in mice, via inhalation and gavage (experimentally placing the chemical in the stomach), and kidney and mononuclear cell leukemia in rats. In the mid-1980s, EPA considered the epidemiological and animal evidence on tetrachloroethylene as intermediate between a probable and possible human carcinogen (Group B/C). The Agency is currently reassessing its potential carcinogenicity.

Phenol: http://www.epa.gov/ttnatw01/hlthef/phenol.html

Chronic inhalation exposure of animals to phenol has shown central nervous systems (CNS), kidney, liver, respiratory, and cardiovascular effects. The Reference Dose (RfD) for phenol is 0.6 milligrams per kilogram body weight per day (mg/kg/d) based on reduced fetal body weights in rats. The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious noncancer effects during a lifetime. It is not a direct estimator of risk but rather a reference point to gauge the potential effects. At exposures increasingly greater than the RfD, the potential for adverse health effects increases. Lifetime exposure above the RfD does not imply that an adverse health effect would necessarily occur. EPA has low confidence in

the study on which the RfD was based because the dose was administered by gavage; medium confidence in the database because it contains several supporting studies (subchronic, chronic, and reproductive/ developmental); and, consequently, low-to-medium confidence in the RfD. EPA has established a provisional Reference Concentration (RfC) for phenol of 0.006 milligrams per cubic meter (mg/m^3) based on no effects in rats, mice, or monkeys. The provisional RfC is a value that has had some form of Agency review, but it does not appear on IRIS.

Polycyclic aromatic hydrocarbons (PAHs): http://www.epa.gov/ttnatw01/hlthef/polycycl.html

Chronic Effects (Noncancer): Skin exposures to mixtures of carcinogenic PAHs cause skin disorders in humans and animals, and adverse skin effects have been noted in humans and animals following application of solutions containing benzo[a]pyrene. An epidemiological study of workers exposed by inhalation to benzo[a]pyrene and other particulate matter reported some respiratory effects. The role of benzo[a]pyrene in this association, however, is unclear. Animal studies have reported effects on the blood and liver from oral exposure to benzo[a]pyrene and a slight hypersensitivity response from dermal exposure to benzo[a]pyrene. EPA has not established a Reference Concentration (RfC) or a Reference Dose (RfD) for POM (PAHs) or for benzo[a]pyrene.

Reproductive/Developmental Effects: No information is available on the reproductive or developmental effects of POM in humans. Animal studies have indicated that benzo[a]pyrene, via oral exposure, induces reproductive toxicity, including a reduced incidence of pregnancy and decreased fertility. Developmental effects, such as a reduced viability of litters and reduced mean pup weight, have also been noted from oral exposure to benzo[a]pyrene in animals.

Cancer Risk: Epidemiologic studies have reported an increase in lung cancer in humans exposed to coke oven emission, roofing tar emissions, and cigarette smoke. Each of these mixtures contains a number of POM compounds (e.g., certain PAHs). Animal studies have reported respiratory tract tumors from inhalation exposure to benzo(*a*)pyrene and forestomach tumors, leukemia, and lung tumors from oral exposure to benzo[a]pyrene. EPA has classified seven PAHs (benzo[*a*]pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene) as Group B2, probable human carcinogens. EPA uses mathematical models, based on animal studies to estimate the probability of a person developing cancer from ingesting water containing a specified concentration of a chemical. EPA has calculated an oral cancer slope factor of 7.3 $(mg/kg/d)^{-1}$ for benzo[a]pyrene. For a detailed discussion of the confidence in the potency estimates, please see IRIS. The California Environmental Protection Agency (CalEPA) has calculated an inhalation unit risk estimate of 1.1×10^{-3} $(\mu g/m^3)^{-1}$ for benzo[a]pyrene.

Selenium compounds: http://www.epa.gov/ttnatw01/hlthef/selenium.html

No information is available on the chronic effects of selenium in humans from inhalation exposure.

Selenium is a naturally occurring substance that is toxic at high concentrations but is also a nutritionally essential element. Hydrogen selenide is the most acutely toxic selenium compound. Acute (short-term) exposure to elemental selenium, hydrogen selenide, and selenium dioxide by inhalation results primarily in respiratory effects, such as irritation of the mucous membranes, pulmonary edema, severe bronchitis, and bronchial pneumonia. Epidemiological studies of humans chronically (long-term) exposed to high levels of selenium in food and water have reported discoloration of the skin, pathological deformation and loss of nails, loss of hair, excessive tooth decay and discoloration, lack of mental alertness, and listlessness. Epidemiological studies have reported an inverse association between selenium levels in the blood and cancer occurrence and animal studies have reported that selenium supplementation, as sodium selenate, sodium selenite, and organic forms of selenium, results in a reduced incidence of several tumor types. The only selenium compound that has been shown to be carcinogenic in animals is selenium sulfide, which resulted in an increase in liver tumors from oral exposure. EPA has classified elemental selenium as a Group D, not classifiable as to human carcinogenicity, and selenium sulfide as a Group B2, probable human carcinogen.

Sulfuric acid: http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=255&tid=47

Touching sulfuric acid will burn your skin, and breathing sulfuric acid can result in tooth erosion and respiratory tract irritation. Drinking sulfuric acid can burn your mouth, throat, and stomach; it can result in death. If you get sulfuric acid in your eyes, it will cause your eyes to water and will burn. People who have breathed large quantities of sulfuric acid at work have shown an increase in cancers of the larynx. However, most of the people were also smokers who were exposed to other chemcals and acids as well. The ability of sulfuric acid to cause cancer in laboratory animals has not been studied. The International Agency for Research on Cancer (IARC) has determined that occupational exposure to strong inorganic acid mists containing sulfuric acid is carcinogenic to humans. IARC has not classified pure sulfuric acid for its carcinogenic effects.

Trichloroethylene (TCE): http://www.epa.gov/ttnatw01/hlthef/tri-ethy.html

Most of the trichloroethylene used in the United States is released into the atmosphere from industrial degreasing operations. Acute (short-term) and chronic (long-term) inhalation exposure to trichloroethylene can affect the human central nervous system (CNS), with symptoms such as dizziness, headaches, confusion, euphoria, facial numbness, and weakness. Liver, kidney,

immunological, endocrine, and developmental effects have also been reported in humans. A recent analysis of available epidemiological studies reports trichloroethylene exposure to be associated with several types of cancers in humans, especially kidney, liver, cervix, and lymphatic system. Animal studies have reported increases in lung, liver, kidney, and testicular tumors and lymphoma. The Agency is currently reassessing the cancer classification of trichloroethylene.

2,3,7,8-Tetrachlorodibenzodioxin (TCDD) and other dioxins: http://www.epa.gov/ttnatw01/hlthef/dioxin.html

Chronic Effects (Noncancer): Chloracne is also the major effect seen from chronic (long-term) exposure to 2,3,7,8-TCDD in humans. Animal studies have reported hair loss, loss of body weight, and a weakened immune system from oral exposure to 2,3,7,8-TCDD. EPA has not established a Reference Concentration (RfC) or a Reference Dose (RfD) for 2,3,7,8-TCDD. ATSDR has calculated a chronic oral minimal risk level (MRL) of 1 x 10^{-9} milligrams per kilogram body weight per day (mg/kg/d) based on neurological effects in monkeys. The MRL is an estimate of daily exposure to a dose of a chemical that is likely to be without appreciable risk of adverse noncancerous effects over a specified duration of exposure. Exposure to a level above the MRL does not mean that adverse effects will occur. The MRL is used by public health professionals as a screening tool.

Reproductive/Developmental Effects: The results of available reproductive and developmental studies in humans are inconclusive. Animal studies have reported developmental effects, such as skeletal deformities, kidney defects, and weakened immune responses in the offspring of animals exposed to 2,3,7,8-TCDD during pregnancy. Reproductive effects, including altered levels of sex hormones, reduced production of sperm, and increased rates of miscarriages, have been seen in animals exposed to 2,3,7,8-TCDD.

Cancer Risk: Human studies, primarily of workers occupationally exposed to 2,3,7,8-TCDD by inhalation, have found an association between 2,3,7,8-TCDD and lung cancer, soft-tissue sarcomas, lymphomas, and stomach carcinomas, although for malignant lymphomas, the increase in risk is not consistent. No information is available on the carcinogenic effects of 2,3,7,8-TCDD in animals following inhalation exposure. Animal studies have reported tumors of the liver, lung, tongue, thyroid, and nasal turbinates from oral exposure to 2,3,7,8-TCDD. EPA has classified 2,3,7,8-TCDD as a Group B2; probable human carcinogen. EPA has calculated an inhalation cancer slope factor of $1.5x10^{5}$ $(mg/kg/d)^{-1}$ and an inhalation unit risk estimate of 3.3 x 10^{-5} $(pg/m^{3})^{-1}$ for 2,3,7,8-TCDD. EPA has calculated an oral cancer slope factor of 1.5 x 10^{5} $(mg/kg/d)^{-1}$ and an oral unit risk factor of 4.5 $(\mu g/L)^{-1}$ for 2,3,7,8-TCDD.

Tetrachloroethane: http://www.epa.gov/ttnatw01/hlthef/tetrachl.html

As 1,1,2,2-tetrachloroethane is no longer used much in the United States, current air emissions predominantly result from its use as a chemical intermediate during the manufacture of other chemicals. Low levels have been detected in air. The main effects of 1,1,2,2-tetrachloroethane are liver and neurological effects. Acute (short-term) inhalation exposure to very high levels of 1,1,2,2-tetrachloroethane has resulted in effects on the liver and respiratory, central nervous, and gastrointestinal systems in humans. Chronic (long-term) inhalation exposure to 1,1,2,2-tetrachloroethane in humans results in jaundice and an enlarged liver, headaches, tremors, dizziness, numbness, and drowsiness. Animal studies have shown a significantly increased incidence of liver tumors in mice orally exposed to 1,1,2,2-tetrachloroethane. EPA has classified 1,1,2,2-tetrachloroethane as a Group C possible human carcinogen.

Toulene: http://www.epa.gov/ttnatw01/hlthef/toluene.html

Toluene is added to gasoline, used to produce benzene, and used as a solvent. Exposed to toluene may occur from breathing ambient or indoor air. The central nervous system (CNS) is the primary target organ for toluene toxicity in both humans and animals for acute (short-term) and chronic (long-term) exposures. CNS dysfunction and narcosis have been frequently observed in humans acutely exposed to toluene by inhalation; symptoms include fatigue, sleepiness, headaches, and nausea. CNS depression has been reported to occur in chronic abusers exposed to high levels of toluene. Chronic inhalation exposure of humans to toluene also causes irritation of the upper respiratory tract and eyes, sore throat, dizziness, and headache. Human studies have reported developmental effects, such as CNS dysfunction, attention deficits, and minor craniofacial and limb anomalies, in the children of pregnant women exposed to toluene or mixed solvents by inhalation. Reproductive effects, including an association between exposure to toluene and an increased incidence of spontaneous abortions, have also been noted. However, these studies are not conclusive due to many confounding variables. EPA has classified toluene as a Group D, not classifiable as to human carcinogenicity.

Vinyl chloride: http://www.epa.gov/ttnatw01/hlthef/vinylchl.html

Most vinyl chloride is used to make polyvinyl chloride (PVC) plastic and vinyl products. Acute (short-term) exposure to high levels of vinyl chloride in air has resulted in central nervous system effects (CNS), such as dizziness, drowsiness, and headaches in humans. Chronic (long-term) exposure to vinyl chloride through inhalation and oral exposure in humans has resulted in liver damage. Cancer is a major concern from exposure to vinyl chloride via inhalation, as vinyl

chloride exposure has been shown to increase the risk of a rare form of liver cancer in humans. EPA has classified vinyl chloride as a Group A, human carcinogen.

Vinylidene chloride: http://www.epa.gov/airtoxics/hlthef/di-ethyl.html

Vinylidene chloride is used as an intermediate in chemical synthesis and to produce polyvinylidene chloride copolymers. The primary acute (short-term) effects in humans from vinylidene chloride exposure are on the central nervous system (CNS), including CNS depression and symptoms of inebriation, convulsions, spasms, and unconsciousness at high concentrations. Low-level, chronic (long-term) inhalation exposure of vinylidene chloride in humans may effect the liver. Animal studies indicate that chronic exposure to vinylidene chloride can affect the liver, kidneys, CNS and lungs. Human data are considered inadequate in providing evidence of cancer from exposure to vinylidene chloride. The most recent cancer classification for vinylidene chloride can be found on IRIS.

Xylene: http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=295&tid=53

No health effects have been noted at the background levels that people are exposed to on a daily basis. High levels of exposure for short or long periods can cause headaches, lack of muscle coordination, dizziness, confusion, and changes in ones's sense of balance. Exposure of people to high levels of xylene for short periods can also cause irritation of the skin, eyes, nose, and throat; difficulty in breathing; problems with the lungs; delayed reaction time; memory difficulties; stomach discomfort; and possibly changes in the liver and kidneys. It can cause unconsciousness and even death at very high levels. Both the International Agency for Research on Cancer (IARC) and the EPA have found that there is insufficient information to determine whether or not xylene is carcinogenic.

Appendix 2: Most recent listing of actual emissions reported from synthetic minor point sources in NC

Pollutant	**CAS Code**	**Assumed emissions reported**				
Criteria		**Tons**				
		4th Creek WWTP (2006)	Graham WWTP (2008)	East Burlington WWTP (2007)	High Point - Eastside WWTP (2008)	Rocky River WWTP (2008)
CO	CO	1.1	0.1	36	5.8	85.9
CO2	124389		15		7391.3	
CH4	74-82-8		0		0.2	
NOx	NOx	4.6	0.3	1.2	8.2	13.9
N20	10024972		0		0.1	
PM(TSP)	PM(TSP)	0.1	0	0.1	0.3	0.7
PM10	PM10	0.1	0	0.1	0.3	0.2
PM2.5	PM2.5	0.1	0	0.1	0.12	0.2
SO2	SO2	0.8		0.1	1.9	1.8
VOC	VOC		0	5.9	2	4.4
Hazardous/ toxic		**Pounds**				
Acetaldehyde	75-07-0		0	0.1		1.8
Acetonitrile	75-05-8					8.3
Acrolein	107-02-8		0	0		
Acrylonitrile	107-13-1				157.08	5.4
Antimony & compounds	SBC					2.7
Antinomy unlisted compounds	SBC-other					2.7
Arsenic & compounds	ASC (7778394)				0.24	1.996
Arsenic compounds-arsine gas	7784-42-1					1.996
Arsenic metal, elemental, unreacted	7440-38-2				0.24	
Arsenic unlisted compounds	ASC-other					0.001
Benzene	71-43-2	2.1	0.1	0.3	1.85	1.9
B[a]P	50-32-8		0	0		
Beryllium & compounds	BEC			0	0.12	0.1
Beryllium unlisted compounds	BEC-other			0		0.1
Cadmium & compounds (total mass)	CDC				0.13	1.22

Cadmium metal, elemental, unreacted	7440-43-9					1.22
Cadmium unlisted	CDC-other				0.13	0
Carbon tetrachloride	56-23-5				0.111	0
Chlorobenzene	108-90-7				0.084	2.9
Chloroform	67-66-3				18.5	5.4
Cobalt & compounds	COC			0		5
Cobalt unlisted compounds	COC-other			0		5
Cr(VI) non-specific	NSCR6				0.41	0.665
Cr(VI) non-specific unlisted	NSCR6-other				0.41	0.665
Cr -All/total	CRC				1.72	
Cr unlisted	CRC-other				1.31	
DEHP	117-81-7				1087.7	3.5
Dichlorobenzene(p), 1,4-	106-46-7				4091	4.5
Ethyl benzene	100-414					0.2
Ethylene dichloride	107-06-2					0.1
Formadeyde	50-00-0		0	0.9		4.4
Hexachloro-dibenzo-p-dioxin 1,2,3,6,7,8	57653-85-7				8.3×10^{-6}	0.001
Hexane, n-	110-54-3			20.6		
Hydrogen chloride	7647-01-0				850	110.9
Lead & compounds	PBC					332.6
Lead unlisted compounds	PBC-other					332.6
MEK (methyl ethyl ketone)	78-93-3					0.6
Mn & compounds	MNC				5.11	9.4
Mn unlisted compounds	MNC-other					9.4
Mercury & compounds	HGC			0.008	0.584	4.214
Mercury unlisted compounds	HGC-other			0.008	0.584	0
Mercury, vapor	7439-97-6					4.213
Methyl chloroform	71-55-6				4.42	15.5
Methylene chloride	75-09-2				6.5	4.4
Naphthalene	91-20-3		0	0	878	99.8
Nickel & compounds	NIC			0	1.8	0

Nickel unlisted compounds	NIC-other			0	1.8	0
Perchloroethylene	127-18-4				1.1	2.2
Phenol	108-95-2					20
Polycyclic organic matter (7 PAHs for NIF)	56553/PAH		0	0		
Polycyclic organic matter (PAH, dioxins, etc. NC & AP 42 historic)	POM			0.1		
Polycyclic organic matter (specific compounds from OAQPS for TV)	83329/POMTV		0	0	878	99.8
Selenium compounds	SEC			0	554	0.7
Sulfuric acid	7664-93-9				1022	554.4
Trichloroethylene	79-01-6				0.277	5
TCDD	1746-01-6				$2.77x\ 10^{-6}$	0
Tetrachloroethane ,1,1,2,2-	79-34-5					133
Toulene	108-88-3	0.8	0.1	0.1	17	7.2
Vinyl chloride	75-01-4				34.19	41.02
Vinylidene chloride	75-35-4					2.77
Xylene (mixed sources)	1330-20-7	0.5	0	0.1		10.5