PN-ACA-787

ASSESSING CHRONIC HEALTH RISKS FROM STATIONARY SOURCE AIR EMISSIONS IN VOLGOGRAD, RUSSIA: A CASE STUDY IN QUANTITATIVE HEALTH RISK ASSESSMENT

The Volgograd Health Risk Assessment Working Group

Environment Discussion Paper No. 18
May 1997

NIS-EEP PROJECT
Newly Independent States Environmental Economics and Policy Project

This paper was sponsored by the Harvard Institute for International Development (HIID) under a cooperative agreement with the United States Agency for International Development (USAID).

The Volgograd Health Risk Assessment Working Group includes Boris Filatov, local team leader, Institute of Hygiene and Toxicology, Volgograd; Scott Wolff, lead technical consultant, Harvard School of Public Health, Cambridge; Larissa Vishnavetskaya, emissions inventories and Ecolog modeling, Agroproject Institute, Volgograd; Simon Avaliani, HIID Risk Assessment Coordinator, Moscow; Dmitry Shaposhnikov, HIID Research Assistant, Moscow; Haluk Ozkaynak, Harvard School of Public Health, Cambridge; and Bruce Larson, HIID, Moscow. Main authors of this English-language report are Scott Wolff, Bruce Larson, and Haluk Ozkaynak. Main authors of a parallel Russian-language report are Boris Filatov, Simon Avaliani, and Dmitry Shaposhnikov.

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Acknowledgments: Sydney Rosen, HIID, Cambridge, provided substantial and substantive editorial guidance on this report. Elena Bondarchuk, HIID, Moscow, provided invaluable logistic, administrative, and management support to the team during completion of this study. Many thanks are also due to John Evans, the USAID/Russia Office of Environment and Health, the Ecological Committee and others in Volgograd City, and the USEPA.

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For additional information please contact: International Environment Program, Harvard Institute for International Development, One Eliot Street, Cambridge, MA 02138.

Tel: (617) 495-5176. Fax: (617) 496-8040.

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1. INTRODUCTION

Protecting human health, and more specifically reducing risks that damages to health will occur in the future, is a fundamental rationale for governmental control of pollution. Such control, however, imposes costs on polluting enterprises and on government budgets. As a result, environmental policy makers must continually try to evaluate multiple types of health risks while keeping the costs of environmental regulations acceptable to society.

Industrial activities impose two general kinds of health risks on the community at large (as opposed to workers): normal or expected levels of emissions and unexpected or accidental levels of emissions. A wide variety of emissions are standard by-products of industrial processes, depending on the type of industry and technology, the inputs used, and the equipment used to reduce emissions. These 'normal' emissions, which are more or less predictable, are transported through various environmental media (air, water, soil) and come into contact with humans, thereby creating some possibility of negative health impacts. In this situation, the main risks to human health are determined by the level of human exposure and the impact on human health of such exposure.[1]

Beginning in the 1980s, pollution control efforts in Russia for normal emissions evolved into a system that combines facility and stack-specific emission limits (called PDV in Russian) with a system of pollution charges. The choice of emission limits was based mainly on concerns with satisfying ambient environmental standards during short time periods. In principle, enterprises first proposed to local environmental authorities allowable emission levels (both maximum short-term emission rates and tons per year of emissions). Environmental authorities, often with the assistance of scientific institutes, then conducted an air dispersion modeling exercise to evaluate if Russian ambient environmental standards for that pollutant (called PDK in Russian) would ever be expected to be violated anywhere outside the enterprise's sanitary protection zone during a 20-minute period using a worst-case modeling scenario.

This existing approach for regulating normal industrial emissions, which is focused mainly on acute health concerns, ignores the large and important issue of chronic health effects from longer periods of exposure to pollutants.[2] Thus, while environmental authorities in Russia have 20-minute PDKs to guide their evaluation of short-term environmental conditions, there are no equivalent annual PDKs to assist with the evaluation of long-term health concerns. There are also no other accepted and practical methods that environmental authorities can use to take into account chronic health risks when determining acceptable emissions levels (PDVs) for industrial facilities.

The existing Russian Law on Environmental Protection does allow environmental risks to be considered when determining acceptable emissions levels for pollution permits. Unfortunately, as local officials have pointed out to the authors, there is no guidance on how to undertake such a consideration.

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[1] Industrial accidents can lead to unexpected high levels of specific emissions, usually but not always for a relatively short period of time. These 'accidental' emissions, such as explosions of chemical storage tanks, also create human health risks. For accidental emissions, environmental policy must address multiple questions of technology safety conditions, as well as financial and criminal liability for accidents when they occur.

[2] There are 24-hour PDKs that in principle can be used for evaluating longer time periods. Most 24-hour PDKs, with the exception of vinyl chloride and B(a)P, were established without considering carcinogenic effects. Even though there are 24-hour PDKs, emission limits (PDVs) are set solely on the basis of 20-minute PDKs.

To begin to fill this gap in the Russian environmental regulatory framework, the Volgograd Health Risk Assessment Working Group undertook a quantitative health risk assessment of current, stationary-source industrial air emissions in the city of Volgograd, Russia. Since facilities in Volgograd, an industrial city of about one million people, emit a large number of pollutants that generate multiple and sometimes not comparable risks, the Working Group decided to focus on two types of health risks affecting the general population of Volgograd: (1) additional risks of cancer; and (2) additional mortality risks from particulates.

The purpose of this research is to show how modern methods of quantitative health risk assessment as recommended by the U.S. Environmental Protection Agency (U.S. EPA), combined with existing Russian environmental data and technical air dispersion modeling technologies and capabilities, can be used to evaluate health risks from normal industrial emissions. While there is widespread understanding in Russia of the basic theory and methods of health risk assessment as it is used in the United States and other places, there has been to date a lack of practical applications in Russia that: (i) are clearly documented and replicable; (ii) apply techniques well accepted in the international community; and (iii) provide a clear link to individual pollution sources for environmental policy makers. It is hoped that the results of this case study, along with those of a growing body of other health risk assessment activities currently underway in Russia, will provide a foundation for future environmental policy making.

This paper is organized as follows. Section 2 provides a short introduction to quantitative health risk assessment techniques, with special reference to inhalation risks from carcinogens and particulates. Section 3 provides a step-by-step description of the process and results obtained from the Volgograd analysis. Not surprisingly, a number of assumptions had to be made to implement a quantitative health risk assessment with existing Russian data and modeling technologies, and these are also discussed in Section 3. Section 4 then concludes with a discussion of the main results of the study and its implications for future quantitative health risk assessment activities in Russia.

2. A BRIEF INTRODUCTION TO METHODOLOGY WITH SPECIAL REFERENCE TO INHALATION RISKS FROM AIR POLLUTANTS

2.1 Basic Approach

Health risk assessment methods began to be used in the United States in the 1970s to estimate risks to human health from specific chemicals and levels of exposure (Andrews 1995). Since that time, a wide variety of risk assessment methods have been developed to address different kinds of risk and different reasons for conducting the assessment. Common to all of these methods, however, is the fact that each step of the risk assessment process requires making multiple assumptions and judgments. From the outset, then, it is emphasized that risk assessment is a *process* used to estimate expected health impacts. This process is based on available scientific information to the fullest extent possible. But scientific understanding is not adequate, and will not be adequate, to eliminate uncertainty from risk assessments. A healthy respect for the uncertainties associated with conducting risk assessments is needed by all concerned (the risk assessors, the public, and the authorities responsible for environmental and health issues). The results of risk assessments are likely to provide additional, relevant

information for policy makers, but such information will seldom if ever provide final, indisputable answers to hard government policy decisions.

The Volgograd air pollution health risk assessment utilizes a methodology based primarily on risk assessment methods recommended by the U.S. EPA. The U.S. EPA guidance is provided in several documents, including *Risk Assessment Guidance for Superfund, Volume 1: Human Health Assessment Manual*, the *Superfund Exposure Assessment Manual*, and the *Exposure Factors Handbook* (U.S. EPA 1996; 1989; 1988). The methodology divides risk assessment into four general, sequential steps: hazard identification; exposure assessment; dose-response assessment; and risk characterization. A short description of each step, focusing on methods appropriate for assessing health risks from the inhalation of carcinogens and particulates, is provided below.

2.2 Hazard Identification and Exposure Assessment

The purpose of the first step of a risk assessment, hazard identification, is to identify the specific chemicals that should be included in the risk assessment due to their potential to affect human health adversely. Hazard identification focuses the risk assessment on the potentially most toxic substances first. For broad analyses like an assessment of health risks from stationary source air emissions in a city, as was done in Volgograd, this step also includes selecting the subset of facilities that will be included in the risk assessment due to their high levels of emissions.[3]

The second step is exposure assessment, which determines the level of exposure of humans to the hazard (i.e. chemical) and the types of exposures that will be included in the risk assessment. Several questions need to be answered in the exposure assessment step, including: what are the significant pathways through which human exposure occurs (air, water, soil, food, skin, lungs); what population is potentially exposed to the hazard; and what are the estimated concentration levels to which they are exposed. For risk assessments from air pollution, it is possible to use either directly monitored concentrations in the ambient environment or to use air dispersion models to estimate concentrations based on inventories of industrial and other types of emissions, depending on what data are available.

Since directly measured ambient concentrations are not usually available for a wide variety of important pollutants, as is the case in Russia, exposure assessment will often include the process of modeling air concentrations from emissions data. While adding an extra dimension of complexity to the process of assessing risks, modeling has the added advantage for environmental policy purposes that the sources responsible for creating health risks are identified directly as part of the risk assessment process. Such information is usually needed to evaluate policy options for reducing health risks.

Based on measurements or estimates of concentrations of the emitted chemicals in environmental media at specific locations or receptor points in the study area (sometimes called exposure point concentrations (EPCs)), it is possible to calculate chemical intake levels in the potentially exposed populations. For chronic (long-term) exposure, which is the focus of the Volgograd study, these intake levels are called chronic daily intakes (CDIs).

[3] At the hazard identification stage, it is also common to collect data on annual estimates of the total mass of products produced by each facility, the general kinds of production processes, the type of pollution control equipment currently in operation, and the year the facility was built.

Exposure to pollutants can be estimated for different population groups based on their demographic and geographic attributes. For example, exposures can be calculated separately for children, elderly people, or the individuals with maximum exposure based on their residence location, type of employment, etc. Most commonly, average exposures are calculated for the entire population. This is the approach taken in the Volgograd study.

2.3 Dose-Response Assessment

The third step, dose-response assessment, quantifies the link between exposure (i.e. intake of a chemical--the dose) and the resulting adverse health effects (the response). Two types of adverse health effects are evaluated in risk assessments, carcinogenic and noncarcinogenic effects. Carcinogens are compounds that produce tumors after long-term, chronic exposures, generally assumed as an entire lifetime in health risk assessments. Carcinogens are thought to increase the probability of tumor formation no matter how low the dose level (i.e. nonthreshold effects). Noncarcinogens are substances that produce adverse health effects other than cancer.[4] Noncarcinogenic health effects include both mortality and morbidity and can be exhibited following either short-term (acute) or long-term (chronic) exposure.

The potential carcinogenicity of chemicals is assessed in two ways. The first uses epidemiological studies to link known human chemical exposures with increases in the incidence of cancer. Although this method is highly relevant, epidemiology studies are demanding since they require a great deal of data, substantial increases in observed cancer rates over background rates, and accurate exposure information. These studies are further complicated by other variables, such as dietary habits and smoking, which can make correlations of chemical exposure with cancer incidence difficult.

In the absence of long-term human exposure data, the other method used to evaluate the carcinogenicity of chemicals is laboratory tests in which animals such as rats and mice are exposed to chemicals over a period of a few months to two years. If an increase in tumor incidence is observed in the exposed animals compared to the control group, the implication is that the chemical could be carcinogenic in humans.[5]

For carcinogens, dose response estimates called "slope factors" (or "cancer potency factors") make the link between chemical dose and increased individual probability of cancer over a lifetime. Slope factors, listed in units of $(mg/kg\text{-}day)^{-1}$ (i.e. reciprocal units of exposure), are based on laboratory experiments with animals and then extrapolated to dose levels likely to be encountered by human populations in the environment. Extrapolation of the dose-response curve to low doses is necessary since laboratory animals are tested at doses which are often much

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[4] Noncarcinogens are often believed to act via threshold mechanisms, which assumes that a specified dose level must be exceeded before toxic effects are exhibited.

[5] Carcinogens can be classified according to the strength of evidence for their carcinogenicity depending on the results of either or both kinds of studies described above. The U.S. EPA and the International Agency for Research on Cancer (IARC) evaluate chemicals in this manner. The U.S. EPA classifies chemicals from Group A, known human carcinogens, to Group E, which are chemicals showing no evidence for carcinogenicity. The U.S. National Toxicology Program (NTP) currently lists approximately 25 substances or groups of substances as known human carcinogens (NTP, 1996).

higher than environmental concentrations.[6] According to U.S. EPA (1992b), slope factors are characterized as upper-bound estimates. The true risk to humans, while not identifiable, is not likely to exceed the upper-bound estimate and in fact may be lower. Slope factors are published for both the ingestion and inhalation exposure routes.

Since air dispersion modeling results are usually presented in terms of ambient concentrations ($\mu g/m^3$), it is often more convenient to convert carcinogenic slope factors into Unit Risk Factors (URFs). URFs represent the additional cancer risk per unit concentration of the chemical in the medium (e.g. $\mu g/m^3$) where human contact occurs (U.S. EPA, 1989). Since the Volgograd analysis focuses on the potential human health risks associated with the inhalation of chemicals, air is the environmental medium of concern in this report. URFs for the inhalation exposure route, presented in units of $(\mu g/m^3)^{-1}$, are calculated assuming lifetime exposure to the chemical and a daily adult inhalation rate of 20 m^3/day. Additional factors to account for differences in absorption and units may also be included. URFs for air are calculated by U.S. EPA (1989) as:

$$URF = (SF^* b^* c)/a, \qquad (1)$$

where URF=Unit Risk Factor $(\mu g/m^3)^{-1}$; SF=Slope Factor $(mg/kg\text{-}day)^{-1}$; a = 70 kg assumed human body weight; b = 20 m^3 /day assumed adult human inhalation rate; and c = 0.001 which is a units conversion from milligrams to micrograms.

Noncarcinogenic effects are all other potential health effects caused by chemical exposures that do not result in the production of tumors. These may include respiratory irritation, physiological effects such as liver toxicity, mental effects such as impaired coordination, reproductive effects such as sterility, and death. As with carcinogenicity, the potential noncarcinogenic effects of environmental pollutants are evaluated from the results of human epidemiology studies or laboratory animal studies.

For health risk assessments, many noncarcinogenic effects are assumed to occur as a result of exceeding a threshold or safe-dose level. Such threshold doses are called Reference Doses (RfDs) and Reference Concentrations (RfCs) by the U.S. EPA. RfDs are derived from human or animal studies by adjusting either the "No Observed Adverse Effect Level" (NOAEL) or the "Lowest Observed Adverse Effect Level" (LOAEL) for various types of uncertainty.[7] For a few pollutants such as SO2, NOx, and particulates, there are the equivalent of slope factors or unit risk factors that can be used to construct a direct dose-response relationship between exposures and health effects.

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[6] Cancer slope factors are developed with a statistical extrapolation model, based on a linearized multistage model, that estimates the largest possible linear slope (within a 95% upper confidence limit) based on animal experimental data extrapolated to lower environmental concentrations.

[7] RfDs are derived using a number of health conservative methods, among them the application of uncertainty factors to NOAELs and LOAELs determined by epidemiological or animal studies. NOAELs represent the highest doses which have no apparent effect on exposed individuals above background exposures, while LOAELs represent the lowest doses thought to cause adverse effects. The experimental dose levels are multiplied by uncertainty factors, typically ranging from 1 to 10, to account for the duration of exposure in the animal or human study, the extrapolation of dose-response data from one species to another, the quality of the study, the severity of any effects noted, and a presumed range of human sensitivity to effects of the chemical. This approach provides an additional margin of safety in protecting public health. The current methodology for deriving inhalation RfCs is described in U.S. EPA (1990).

2.4 Risk Characterization

Risk characterization, the last step in the risk assessment process, combines the chemical intake estimates from the exposure assessment with the quantitative health criteria from the dose-response assessment.[8] For carcinogens, risk characterization combines EPCs with slope factors to estimate additional cancer risks. Two main kinds of risks are usually considered. *Individual lifetime cancer risk* is defined as the additional risk of an individual developing cancer over an entire lifetime of exposure to specific chemical concentration levels. *Annual population cancer risk* is defined as the number of additional cancer cases in the study area expected to occur each year as a result of exposure to the carcinogenic chemical.

Additional lifetime cancer risk for an individual is a function of three main factors: (1) the inhalation CDI estimated from ambient air concentrations at the exposure points (usually based on air dispersion modeling results); (2) the probability that a particular chemical promotes tumor formation; and (3) the duration over which exposure occurs. Using U.S. EPA methods, these factors are combined by multiplying the slope factor, the exposure-point concentrations, and the total fraction of the individual's lifetime over which exposure occurs. Thus:

Individual Lifetime Cancer Risk = CDI x SF x a,

where Individual Lifetime Cancer Risk = individual excess lifetime cancer risk for a specific chemical in the potentially exposed residential population; CDI = chronic daily intake of the specific chemical at the modeled exposure point (mg/kg-day); SF = slope factor for inhalation of the chemical $(mg/kg\text{-}day)^{-1}$; and a = 1 = 70/70 represents the number of years that the exposed individuals are assumed to live in the study area at the same receptor location (70 years) divided by the total number of years assumed in a lifetime (70 years) (U.S. EPA, 1989). The calculation for the annual population cancer risk takes the calculated individual risk, multiplies by the exposed population (50,000 at each receptor point in this study), and then divides by the exposure period used for the individual risk calculation (in the example above, 70 years).

For noncarcinogens, risk can be calculated by comparing estimated daily intakes to RfDs or estimated/monitored concentrations to RfCs.[9] As noted above, there are some noncarcinogens such as particulates for which the equivalent of slope factors or unit risk factors exist or can be developed. In such cases, risk calculation for noncarcinogens is similar to the above calculation for carcinogens.

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[8] The term "risk assessment" has a wide variety of meanings depending on the selected methodology and ultimate purpose of the analysis. Risk assessments conducted for estimating the potential health impacts of environmental contaminants use the U.S. EPA-recommended methodology described in this report. The ultimate use of this kind of risk assessment is typically to inform policy, regulatory, or investment decisions that will affect public health. The kinds of risks to be evaluated (maximum individual risk, average individual risk, average population risk) will also depend on the ultimate purpose of the analysis. For new facilities in the United States, it is common practice to focus on the maximum exposed individual (MEI) and to estimate the total population cancer burden in the study area. Policy implications are likely to be different for different definitions of risk.

[9] Such threshold logic for all types of pollutants is commonly used in Russian health and environmental policy, where measured or estimated ambient concentrations are compared to Russian ambient standards (20 minute or 24-hour PDKs).

2.5 Uncertainties in Risk Assessments

An overview of basic risk assessment methods is not complete without a discussion of the uncertainties inherent in this four-step process. As emphasized in Section 2.1, risk assessment is a process used to estimate expected health impacts, and a healthy respect for the uncertainties inherent in this process is needed. Risk assessments require information about many different variables, including sources of exposure; transport and environmental fate of pollutants; inhalation, ingestion, and dermal exposure pathways; exposed population groups; types of activities, conditions, and locations affecting exposures; administered dose and amount of biologically effective dose received by target organs; and health effects in response to exposures based on toxicologic and epidemiologic data. Each variable introduces its own uncertainties that can include, for example, variation in impacts among individuals and uncertainties regarding scenarios, models, and/or parameters.[10]

There is a large literature on the uncertainties inherent in risk assessment methods (e.g., Wilson, Crouch, and Zeise 1985; IAEA 1989; U.S. EPA 1992c; Suter 1993; U.S. EPA 1992b; Bogen and Spear 1987; Morgan and Henrion 1990). The interested reader can consult this and other literature for further details. Discussion of the uncertainties in the Volgograd study is contained in Section 3 of this report.

3. THE VOLGOGRAD CASE STUDY: CARCINOGENIC AND PARTICULATE-INDUCED MORTALITY RISKS FROM MAJOR STATIONARY SOURCES

3.1 Preliminaries

Volgograd is an industrial city of approximately one million people in southern Russia. The city was known previously as Stalingrad, where brutal and famous battles took place during the Great Patriotic War (also known as World War II). Following the almost complete destruction of its infrastructure, housing, and industry during the war, the city was rebuilt during the next two decades.

As shown in Map 1, Volgograd is a long, narrow city on the western bank of the Volga River. The distance by road between the city's northern and southern borders is about 70 km. Industrial facilities are scattered throughout the city, although there are main concentrations of industry in the north and south.

The purpose of this section is to report on the detailed methods used and results obtained during the Volgograd air pollution health risk assessment. The risk assessment was carried out by a Working Group comprised of scientists and environmental policy experts from Volgograd scientific institutes, the Harvard Institute for International Development, and the Harvard School of Public Health. From the outset, four principles guided the Working Group's analysis.

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[10] Different types of uncertainty include *scenario uncertainty*, which arises from a lack of knowledge required to specify fully the problem; *model uncertainty*, arising from a lack of knowledge required to formulate the appropriate conceptual or computational models; and *parameter uncertainty*, a lack of knowledge about the true value or distribution of a model parameter (U.S. EPA 1992b). Variability among individuals must also be distinguished from other kinds of uncertainty (Bogen and Spear 1987; IAEA 1989; Morgan and Henrion 1990).

(1) *Use existing data for Volgograd.* The Working Group needed to use existing data because there was not adequate time or financial resources to collect new data.

(2) *Use data that are likely to be available for many other Russian cities as well.* The Working Group wanted to use existing data so that the process developed for Volgograd can easily be replicated in other parts of Russia. Principles 1 and 2 suggested that the study should focus on air health risks from stationary sources. Emissions data for stationary source air polluters are available because of the framework of pollution permits and pollution charges that exists in Russia. Stationary sources have been a main focus of Russian air pollution laws and regulations as well.

(3) *Apply the standard four-step quantitative health risk assessment methodology described in Section 2.* The purpose of this analysis was to attempt to apply a standard, internationally-accepted methodology within the constraints imposed by the need to use existing data that are commonly available in Russia.

(4) *Make the link between health risks and the facilities that generate the risks explicit in the risk assessment to permit subsequent identification of cost-effective policy options for reducing health risks.* The Working Group concluded that a direct link between health risks and contributors to such risk was necessary to make the results of the risk assessment clearly relevant to the current environmental policy debate in the city. Since there is not an appropriate air monitoring network in Volgograd for most pollutants, as is the case in the rest of Russia and most other countries, it was not feasible to use ambient concentration data in any case. Instead, emissions inventories from stationary source air polluters provided the core data for the health risk assessment, and the link between industrial facilities and health risks was made through modeling the dispersion of air emissions throughout the city.

To select an air dispersion modeling strategy for estimating ambient air concentrations at specific locations in the city (exposure-point concentrations or EPCs, as discussed in Section 2), the Working Group again applied the principle of using information resources--in this case air dispersion modeling capabilities--that are commonly available in Russia. Thus, Ecolog, a computer program based on the standard Russian air dispersion model for developing stationary source air pollution limits, was chosen for the analysis.[11] Due in part to modeling and data constraints, it was also decided that inhalation would be the main pathway for which health risks would be assessed in this study.

The remainder of this section contains a step-by-step explanation of the process used and results obtained in the assessment of air pollution health risks from stationary sources in Volgograd. To provide guidance for future risk assessments in Russia that rely on existing data and modeling technologies, each step in the Volgograd risk assessment process, along with supporting assumptions, is described in detail.

[11] Air dispersion modeling will be discussed in more detail below under exposure assessment.

3.2 Hazard Identification in Volgograd

Given the focus on stationary source air emissions in Volgograd, the Working Group developed a list of polluters in the city based on information provided by local environmental authorities. While Volgograd has more than two hundred facilities regulated under the pollution permit system, only 29 of them account for more than 90 percent of total reported stationary source air emissions in the city (in terms of total tons per year during 1995).[12] These 29 facilities, which individually have total annual chemical and particulate emissions greater than 10 tons/year based on emission inventories for the years 1993, 1994, and 1995, were selected for the study.[13] Table 1 provides a complete list of the 29 facilities included in the Volgograd analysis, along with an indication of each facility's primary production sector, general location in the city (central, northern, or southern zone), and facility code.[14] Facility codes are placed on Map 1 to indicate the specific location of each facility within the city. A fairly wide distribution of industrial sectors is represented by the 29 facilities, including district heating, construction materials, chemicals, ferrous and non-ferrous metals, furniture, and agricultural machinery.

Following the selection of the 29 facilities, the Working Group reviewed the facility emissions inventories for 1993, 1994, and 1995 to determine which year's data should be included in the risk assessment. The Working Group wanted to use a year for which emissions levels are likely to approximate future years' emissions levels in the city (in the base case of no major changes to environmental regulations). The year 1995 was selected as a reasonable approximation of emissions levels for the future.[15] All emissions data utilized in this study are based on engineering estimates of chemical emission rates.

The Working Group also decided to limit the focus of this risk assessment to carcinogens and noncarcinogenic particulate matter. Carcinogens are typically included in health risk assessments because the cancer endpoint requires the lowest chemical exposure and because of the concern that any exposure to carcinogens, which are considered to be nonthreshold agents, increases the lifetime risk of developing cancer. All carcinogens found in the emissions inventories of any of the 29 facilities were included in the risk assessment.

The Working Group focused on particulates as the only noncarcinogenic agent to be assessed because of the well-document health effects of total suspended particulates (TSP), and more specifically PM_{10} (particulate matter with a diameter of 10 microns or less) (see, e.g., Wilson and Spengler, 1996). It is also noted that TSP encompasses a high percentage of the total

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[12] Hazard identification was completed under the direction of Larissa Vishnavetskaya of the Agroproject Institute.

[13] The evaluation of the emissions inventories included substances such as volatile organic chemicals (VOCs), semi-volatile organic chemicals (SVOCs), polycyclic aromatic hydrocarbons (PAHs), metals, and the U.S.-designated criteria air pollutants (ozone (O_3), nitrogen oxides (NO_x), sulfur dioxide (SO_2), particulate matter, lead, and carbon monoxide (CO). Innocuous substances such as carbon dioxide (CO_2) were excluded from this consideration.

[14] Due to the large area encompassed by Volgograd's city limits and its large number of enterprises, the Working Group defined the Volgograd study area boundary as the currently defined city limits. Even though there is an industrial area located south of the city limits, facilities located outside of the city limits are not included in the Volgograd study area.

[15] It should be noted here, however, that an interested reader who does not agree with the 1995 assumption can easily change this assumption and evaluate the associated changes in risk in this study because of the air dispersion modeling approach used. This issue will be discussed in the exposure assessment section.

noncarcinogens in the emissions inventories. Particulates are a clear health concern in major cities, and given the limited resources it made sense to focus on particulates first.

The complete list of carcinogens included in the study is provided in Table 2. Table 3 provides a summary emissions inventory of total carcinogens and total particulates for the 29 facilities in the study. Note that the distribution of emissions is highly skewed even within these 29 'major' polluters. In terms of total mass, facilities 214, 252, 253, and 314 account for most emissions of carcinogens, while facilities 105, 214, 314, and 315 account for most particulate emissions.[16]

Russia does not directly regulate particulate matter as an aggregate but instead has a wide range of ambient air concentration standards for many different types of particulates. Thus, emissions inventories for regulatory purposes do not directly include 'particulates.' For this analysis, various types of particulate matter emitted by facilities were aggregated into one measure of 'total particulates.' For two facilities with high levels of total particulates, Table 4 indicates the various types of materials that are aggregated into 'total particulates' in this study. Note that for facility 314, soot is also included as a particulate, while chromium hexavalent is also a particulate for facility 315.[17] As will be discussed in the dose-response assessment section, particulate matter is included in the risk assessment directly as PM_{10} and not as total suspended particulates (TSP), with the assumption that $PM_{10} = 0.6*TSP$ (Wilson and Spengler, 1996).

Thus, to summarize Section 3.2, this study asks the following 'what if' question:

What are the estimated future health risks (individual and population risks for cancer and particulate-induced mortality via inhalation) from the identified stationary sources if their emissions remain at 1995 levels in the future?

3.3 Exposure Assessment

The first step in conducting the exposure assessment for Volgograd was to define the population of exposed individuals. For this study, the population is defined as the approximately one million individuals who live within Volgograd city boundaries and are at risk of being exposed to industrial air emissions. Population densities vary throughout the city, and residential and industrial areas are located in close proximity to one another in several parts of the city.

Based on a city map of population densities, the Working Group chose 20 receptor locations throughout the entire city.[18] Map 1 identifies the 20 receptor points in relation to the industrial facilities. Each receptor location represents 5 percent of the city's population, or 50,000 individuals. Given varying population densities, the land area associated with each receptor

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[16] Soot is included as a carcinogenic substance because of the carcinogenic polycyclic aromatic hydrocarbons (PAHs) associated with it. On a mass basis, a review of existing literature suggests that the carcinogenic PAHs represent approximately 0.5% (0.005) of the total mass of soot (IARC, 1985). Benzo(a)pyrene (B(a)P) is utilized as a surrogate for the carcinogenic PAHs. Thus, 0.5% of the mass of soot is modeled as B(a)P in this analysis.

[17] Indirect particulate emissions are not included in this study. For example, SO2 is a gas that can be converted into particulate sulfate many kilometers downwind.

[18] Population maps and other demographic information for the study area were provided by Dr. Boris Filatov of the Working Group.

point varies throughout the city. Each of the 20 receptor locations is placed at the nearest node of the air dispersion modeling grid and represents the center of the specific populated area.[19]

Following the selection of 20 receptor points throughout the city, each representing a population of 50,000 individuals, the next step was to estimate exposure point air concentrations (EPCs) at the selected receptor points based on air dispersion modeling of the emitted carcinogens and particulates. For chronic risks, such as carcinogenic risks and mortality-related particulate risks, it is necessary to estimate *annual average* EPCs. Because a goal of this study was to rely on existing Russian data and modeling technologies, estimating annual average EPCs was perhaps the most difficult part of the Volgograd risk assessment process. Several important assumptions were needed to complete the modeling stage, as described below.

The EPCs at the receptor point locations were estimated using the Ecolog air dispersion model, the standard air contaminant transport model that was developed during the Soviet period for setting emission limits in pollution permits.[20] Ecolog is thus designed to model 20-minute air concentrations relative to PDKs assuming 'worst-case' scenarios (maximum emission rates and weather conditions that lead to the highest concentrations).[21] The current version of Ecolog is not designed for estimating ambient air concentrations for long periods of time under general weather conditions.

The 29 facilities in this study include several thousand individual stacks and fugitive sources. Given the rather small quantity of carcinogens and particulates emitted by many of the 29 facilities, and to save time and financial resources during the analysis, each facility was modeled as if it had one aggregate stack. The Working Group defined the characteristics of the aggregate stack (e.g. height and location within the facility) as a rough average across all sources in the facility. Using this 'aggregate-stack' assumption, a base modeling run to estimate EPCs at the 20 receptor points was completed assuming an emission rate normalized to 1 g/sec. It is important to emphasize that just one Ecolog run was needed for each facility using this modeling approach.[22 23 24]

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[19] The figure is set up as a Cartesian coordinate matrix.

[20] The basic reference in Russia for air dispersion modeling is OND 86.

[21] In addition to total tons per year emitted in 1995, emission *rates* for the carcinogenic chemicals and PM_{10} for each of the 29 facilities were needed for the exposure assessment step of the risk assessment. Two types of emission rates are used in this study: (1) annual average emission rates in units of g/sec; and (2) 'maximum' emission rates in units of g/sec assumed to occur for a maximum 20-minute duration. Average emission rates are based on estimates of total tons per year. Maximum emission rates, based on maximum capacity utilization and efficient operation of production equipment and pollution control technologies, are commonly available in Russia because they are parameters used to determine pollution permit limits. Emission rate estimates exist as individual point estimates. No summary statistics regarding the variability of the chemical emissions during the year are available for Volgograd industrial emissions (as is commonly the case in Russia).

[22] This modeling approach has important implications for risk management analyses because policy options that are designed to alter emission rates can be evaluated in terms of their risk impacts directly, without having to re-run the air dispersion models. This greatly simplifies the policy evaluation process, especially since policy makers do not always have direct, inexpensive, and fast access to air dispersion modeling capabilities.

[23] The Working Group developed the aggregate stack approach in the following way. First, a sample modeling run was set up to investigate how best to aggregate the various sources to produce minimum errors compared to running the model using individual sources at the single facility. To test its model, the Working Group randomly picked a single facility and five receptor points in the Volgograd community and ran the model in four ways: (1) as individual sources utilizing the individual source characteristics for each stack and then summing the impacts across each receptor for all of the stacks; (2) as a single area source using ground level as the stack height; (3) as a single

EPCs were not modeled for all receptor points for all facilities. Since Volgograd is a large (long) city, air concentrations at receptors located great distances from the specific emission sources are negligible. Thus, receptors located mainly in the same area of Volgograd (northern, central, southern) as the source were included in the modeling run.

Some sample results of the normalized emission modeling runs are presented in Tables 5.1 - 5.7. In each table, the first column titled 'receptor points' denotes the receptor points as identified in Map 1, while the second column is the results of the normalized emission run for each receptor point modeled for that facility.

The main complication with using Ecolog for this risk assessment is that it estimates 20-minute concentrations under maximum emission conditions. The emissions data for each of the 29 modeled facilities, which were developed primarily on the basis of mass balance chemical engineering estimates, represent the maximum short-term estimates of chemical emissions expected to occur throughout an entire calendar year. The time duration for these emissions is assumed as approximately 20-30 minutes. The data can therefore be interpreted as the maximum emission levels expected to result, at sometime during the year, from either normal facility operation at full capacity or an anticipated emissions upset condition (a short-term condition at a facility that results in higher than normal emissions).

These short-term maximum emission estimates are useful in the Russian context to compare modeled ambient air concentrations with short-term regulatory criteria (i.e. 20-minute PDKs). Such short-term maximum emission estimates are not useful for estimating either cancer health risk or chronic noncarcinogenic air pollutant impacts, however, because these health problems result from longer-term chemical exposures. Long-term emissions estimates, such as annual average emissions, are more appropriate for conducting risk assessment calculations based on U.S.EPA methodologies.

To convert Ecolog 20-minute maximum emissions to annual average emissions, a method was developed that requires two weighting factors. The first factor is defined as the fraction of time the facility operates throughout an entire year, based on the number of shifts per day (1,2, or 3), each representing eight-hour work schedules, and the number of weeks in the year the facility operates (52 weeks maximum). The second factor, which reflects the steadiness of emissions throughout the entire year, is defined as the ratio of annual average emissions of the facility (based on total tons/year for all pollutants converted to an average g/s) and the maximum 20-minute emission rates.

For example, assume that facility X operates 2 shifts per day for 8 months of the year, has total emissions of 500 tons/year, and has a maximum 20-minute emission rate for all pollutants of 150 g/sec. The first weighting factor is (2/3)*(8/12) to account for 2 shifts out of 3 per day for 8 out of 12 months. To compute the second weighting factor, note that 500 tons/year equates to

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stack using a weighted height based on the mean of the individual stack heights; and (4) as a single stack using the top of the highest stack as the source height. Receptor modeling results, based on a unit emission of 1 g/sec for each source run, were prepared. The results from modeling runs #2, #3, & #4 were compared with the full run #1, and it was concluded that the weighted source model from run #3 agreed best with the full model #1. Therefore, a weighted source term was utilized for all 29 of the individual facility modeling runs.

[24] This normalized emissions rate approach to air dispersion modeling has been used elsewhere for assessing cancer risks from multiple facilities in a region. For example, see "Screening Analysis of Air Pollution", prepared for the Technical Workgroup, Project Silesia, Katowich, prepared by Industrial Economics, Inc. and Sullivan Environmental Consulting, Inc., January 1994.

14.4 g/sec assuming the facility operates throughout an entire year. Thus, the second weighting factor is 14.4/150. The product of these two weighting factors, 0.04, must be multiplied by the Ecolog results to derive an annual average emission estimate.

Table 6 provides the two weighting factors used for each facility in this study. The "steadiness of emissions" factor ranges from 0.09 to 1, while the "operating time" factor ranges from 0.14 to 1. For reference, these weighting factors for the relevant facility are also contained in columns 3 and 4 of Tables 5.1 - 5.7.

To estimate annual average concentrations for each pollutant from each facility, it is necessary to calculate:

$$AAC = a * WF1 * WF2 * d \qquad (2)$$

where

AAC	=	average annual concentration estimate for any included chemical ($\mu g/m^3$)
a	=	normalized 20-minute exposure point concentration from Ecolog ($\mu g/m^3/g/s$)
WF1	=	weighting factor 1 representing fraction of facility operating time
WF2	=	weighting factor 2 representing steadiness of emissions
d	=	maximum emission rate of chemical (g/s).

For example, in Table 5.1, column 5 provides the annual concentration estimate for cadmium at each modeled receptor point.

To estimate inhalation exposure to airborne carcinogens, chronic daily intake (CDI in mg/kg-day) estimates are derived using the following equation:

$$CDI = \frac{ACC \times CF \times IR \times EF \times ED}{BW \times AT} \qquad (3)$$

where:

CDI	=	chronic daily intake in units of milligrams per kilogram body weight per day (mg/kg-day)
AAC	=	average annual exposure point concentration (from equation (2))
CF	=	units conversion factor (0.001 mg/μg)
IR	=	inhalation rate, assumed as 20 m^3/day
EF	=	exposure frequency, assumed to be daily, 365 days/year
ED	=	exposure duration, assumed as an entire lifetime of 70 years for residential adults
BW	=	average body weight during the exposure period, assumed to be 70 kg (adult)
AT	=	averaging time (period over which exposure is averaged in days), assumed to be 25,550 days for adults (365 days x 70 years).

The CDI estimate from equation (3) assumes that the Volgograd population is stationary. Individuals are assumed to remain at the same location throughout their entire lifetimes. The CDI estimation also uses a representative, average individual to represent each person in the

population. This individual is modeled as an adult who is assumed to breathe 20 m^3 of air per day throughout an entire lifetime of 70 years. These two assumptions are likely to insure that the estimated chemical exposures are conservative estimates; that is, the exposures are unlikely to be exceeded by anyone residing in the study area.[25]

3.4 Dose-Response Assessment

3.4.1 Carcinogens

For carcinogens, slope factors (SFs) are used to convert chronic daily intakes into additional cancer risks. The slope factors used in this study were derived by the U.S. EPA for the inhalation exposure route. Inhalation slope factors for all carcinogens included in this study, which are provided in Table 7, are presented in units of $(mg/kg\text{-}day)^{-1}$, but are also published by the U.S. EPA as Unit Risk Factors (URFs) as described in Section 2. The U.S. EPA publishes SFs and URFs in several sources, the most up-to-date being the on-line Integrated Risk Information System (IRIS) data base (U.S. EPA, 1997).

3.4.2 Particulates

Particulate matter, and specifically PM_{10}, is the noncarcinogenic substance included in this study. The U.S. EPA has not published a slope factor or reference dose for PM_{10} because it is a U.S. criteria pollutant regulated under the U.S. National Ambient Air Quality Standards. The Working Group thus had to develop a noncarcinogenic health criterion for PM_{10}.

Existing literature on the health effects of air pollution provides clear evidence of mortality and several kinds of morbidity effects from inhalation exposure to particulates, and this information is regularly used in international health impact studies. To develop a PM_{10} mortality coefficient comparable to a unit risk factor for a carcinogen, the Working Group relied on a review of the literature on acute exposure to ambient air particles (e.g. Wilson and Spengler, 1996). It is reasonable to use acute, i.e. short-term, exposures for this study because exposure is assumed to occur every day. Short-term effects summed over longer time periods can thus be used to project mortality risks from long-term exposure to PM_{10}.

Acute-exposure epidemiology studies, conducted under wide ranging environmental, climatic, demographic, and geographic conditions, consistently indicate that an approximate 1 percent increase in daily mortality rates occurs for every 10 $\mu g/m^3$ of PM_{10} in ambient air (Wilson and Spengler, 1996). Based on this relationship, an individual particulate coefficient describing the additional deaths per person per 10 mg/m^3 (assuming linearity) can be developed as follows.

Assume that there are 2,100,000 deaths in the U.S. annually. If the entire U.S. population of 250,000,000 individuals were exposed to 10 $\mu g/m^3$ of PM_{10} daily, the rate of daily deaths attributable to air pollution in this population would be estimated as: 2,100,000 deaths/year x 0.01 mortality increase/365 days/year x 250,000,000 U.S. population = .00000023 deaths/day-person-10 $\mu g/m^3$ PM_{10}. The individual particulate coefficient (IPC) is then estimated as:

[25] Enough information is provided in this report so that the interested reader can make changes in these assumptions and evaluate the change in estimated health risks. For example, it is well known that average Russian male life expectancy is not 70 years.

IPC	=	.00000023	x	365	x	70	(4)
(units)		(deaths/day-person-10 $\mu g/m^3$ PM_{10})		(days/yr)		(years)	
IPC	=	.0059					
(units)		(deaths/person per 10 $\mu g/m^3$ PM_{10} lifetime exposure)					

Using the IPC developed in equation (4), and knowing the size of the exposed population and their average lifetime PM_{10} exposure point concentration, the additional number of deaths attributable to PM_{10} exposure can be estimated. In this study, each receptor point is assumed to have an identical population of 50,000 individuals. Therefore, a Volgograd receptor particulate mortality coefficient for lifetime exposure to 10 $\mu g/m^3$ PM_{10}, denoted here as VRPC(lifetime), can be defined as:

$$\text{VRPC(lifetime)} = \text{IPC} \times 50{,}000 = 295 \quad (5)$$

where IPC is defined in equation (4). Thus, 295 deaths are estimated to occur at each receptor in Volgograd per lifetime exposure for each 10 $\mu g/m^3$ of PM_{10}.

For calculation of annual effects, an annual average Volgograd receptor particulate mortality coefficient, denoted as VRPC(annual), can be defined as:

$$\text{VRPC(annual)} = \text{VRPC(lifetime)}/70 = 295/70 = 4.2 \quad (6)$$

which implies that approximately 4.2 deaths/year would be expected in a receptor point population of 50,000 exposed to an average PM_{10} air concentration of 10 $\mu g/m^3$.

For example, assuming linearity for the PM_{10} coefficient, a lifetime exposure to 100 $\mu g/m^3$ of PM_{10} would result in an estimated 2,950 additional deaths over a 70-year lifetime per 50,000 exposed individuals (using equation 5), or 42 deaths/year (using equation 6). These particulate mortality coefficients are utilized to estimate the impacts of PM_{10} in the Volgograd study area for each of the receptor point populations.

3.5 Risk Characterization and Results

Risk characterization combines health criteria (carcinogen slope factors and the PM_{10} mortality coefficient), exposure point concentrations (EPCs), and chronic daily intakes (CDIs) for carcinogens to estimate additional cancer risks and additional mortality risks from PM_{10}. A discussion of the uncertainties inherent in the health risk assessment process is also included in the conclusion to this report. This discussion is an essential part of the analysis and should be considered carefully when interpreting the risk characterization results.

Tables 5.1-5.7 provide examples of individual facility risk tables for each receptor location for carcinogens and particulates, while Table 8 and Table 9 provide a complete summary of results for all facilities in this study. Consider first Tables 8 and 9. In Table 8, the first column indicates the 29 facilities in the study (see Table 1 for the set of facility codes and facility names). The top row in Table 8 indicates the 20 receptor points for the study. Each cell in the matrix represents annual population cancer risk (estimate of additional cases of cancer in the receptor population of 50,000) from a facility at a specific receptor point. Thus, for example, Table 8 reports that emissions of all carcinogens from Facility 214 creates a risk of an additional 0.2580 cancer cases per year at receptor point 1. The last column of Table 8 reports the sum of all population cancer risks created by each facility across all receptor points, while the last row of Table 8 reports the sum of all such risks created by all facilities at each receptor point. The final number in the lower right corner of the table is the annual population risk across all 29 facilities and all 20 receptor points in the city. Table 9 is organized in an identical way for particulates.

As indicated in Table 8, this risk assessment estimates that 13 additional cancer cases per year are expected to result in the Volgograd population due to the reported levels of 1995 stationary source industrial air emissions. Seven of the 29 industrial facilities contribute 97 percent of the total population cancer risk, and just four facilities (214, 314, 122, and 253) contribute 84 percent of this additional cancer risk. These cancer risks vary considerably across the city. Individuals living in the areas represented by receptors 1-5, the southern third of the city, experience approximately 53 percent of the estimated additional cancer risk for the total city population.

Tables 5.1-5.4 provide detailed risk estimates for the four facilities that generate 84 percent of estimated additional cancer risk in the city. Cancer risks come mainly from these four facilities and are also limited to just a few chemicals. Essentially all additional cancer risks are from chromium VI from Facility 122, vinyl chloride and carbon tetrachloride from Facility 214, soot from Facility 314, and vinyl chloride from Facility 253.

As with Table 8 for carcinogens, Table 9 provides a complete summary risk table for all facilities and receptor points for particulates. As indicated in the lower right corner of Table 9, this study estimates approximately 2,700 additional deaths per year in Volgograd from particulate emissions from the 29 facilities. This overall mortality impact by PM_{10} on the health of the Volgograd residential population is approximately 200 times greater than the impact of the potential human carcinogens (even assuming that all cancers cause death).

Two industrial facilities (314 and 315) contribute 85 percent of the mortality impacts attributable to PM_{10} exposure. Detailed results for these two facilities are provided in Tables 5.5 and 5.6. Not surprisingly, since these two facilities are located in the northern part of the city, the health impact due to PM_{10} exposure is also disproportionate within the city's northern residential population. However, since the five designated residential receptors (16-20) in the north represent 59 percent of the mortality impact attributable to PM_{10}, a significant level of particulate risk is created even at some distance from these two facilities.

It is interesting to compare the detailed results for Facility 214 with those for Facility 315, as can be done using Table 5.5 and Table 5.7. The emissions inventories in Table 3 show that Facility 214 emitted 2,480 tons of particulates in 1995, while Facility 314 emitted 2169 tons, about 300 tons less than Facility 214. From a simple look at total particulate emissions, it is tempting to conclude that these two facilities' emissions are of equal importance for environmental policy. The risk assessment concludes, however, that mortality risks from

particulates are more than ten times higher for Facility 314 (about 470 additional deaths per year) as compared to Facility 214 (about 40 additional deaths per year in the city).

3.6 Uncertainties in the Volgograd Study

Health risk assessment is a multi-step, multi-disciplinary process that estimates the potential human health risks that result from exposure to chemicals in the environment. This process requires both scientific analysis and professional judgment to capture the complexity of the variables and input parameters required to quantify health risk, and this complexity gives rise to considerable uncertainty. The numerical risk estimates derived from a risk assessment should be viewed in light of these uncertainties.

There are several sources of uncertainty in the Volgograd study. Some of these sources are inherent to the risk assessment process, while others are specific to the data and methodology used in the Volgograd study. Some of the assumptions used to implement the Volgograd risk assessment are likely to overstate risks and some are likely to understate risks. The overall effect is unclear.

Regarding basic data, emissions estimates used in the analysis are highly uncertain, only maximum emissions are presented, and no distribution of emissions is available. There is no way to verify the accuracy or completeness of the 1995 emissions inventory. While the study was based on the assumption of 1995 emissions levels continuing into the future, future emission levels will of course depend on the level and types of production and technologies used in the future.[26]

There is also uncertainty in the approach used to model ambient concentrations. Ecolog is a model designed primarily to establish emission limits within the existing Russian regulatory framework based on 20-minute PDKs. This study uses Ecolog as the foundation for predicting annual average exposure point concentrations. Information on the underlying accuracy and precision of this model, as well as the accuracy of the approach used to convert 20-minute Ecolog results into annual average concentrations, is not available. Even the best air quality dispersion models (which are Gaussian plume models except for long-range transport) are only accurate within a factor of two or three, however (U.S. EPA 1992a;1986). The overall impact of this uncertainty on the Volgograd study is that the modeled exposure point concentrations are likely to be overestimates of the actual concentrations at the modeled receptor locations.

Chemical specific toxicity factors are another source of uncertainty in this analysis. As discussed in Section 2, methods used to derive carcinogen slope factors often make conservative assumptions, which imply that predicted risks may be greater than real risks by orders of magnitude. Chromium VI, benzene, and vinyl chloride represent the only known (Group A) human carcinogens known to be emitted by the Volgograd industries. Cadmium is a Group B1 probable human carcinogen based on human epidemiology evidence. For the remaining potential carcinogens (carbon tetrachloride, chloroform, etc.), the slope factors represent upper-bound estimates of the 95th percentile potencies as derived from laboratory animal data. The lower-bound cancer potency values for these chemicals may be as low as zero (U.S. EPA 1988). The

[26] While the issue of future emission levels, and even the basic viability of certain facilities, is not included here, it is easy to conduct a 'sensitivity analysis' of the existing risk estimates using Tables 5.1 - 5.7 and Table 6. Thus, an interested reader who thinks future emissions will be larger or smaller than 1995 levels can adjust relevant weighting factors (WF1 or WF2 from Table 6) or the maximum emission rates reported in Tables 5.1-5.7.

use of upper-bound cancer potency values may therefore result in overestimates of potential carcinogenic risk to the potentially exposed population.

Chemicals which are simultaneously metabolized can also interact with one another and influence overall toxicity. These effects can be either synergistic or antagonistic, enhancing or impeding toxic effects. These potential effects have not been evaluated in the Volgograd study, and the impact of chemical interaction on the final risk estimates is not clear.

A related source of uncertainty for this study is the assumption that soot is 0.5% B(a)P for the carcinogen calculation. Based on a review of available information, this is a reasonable assumption, but it is still uncertain. Similar uncertainty may exist in the calculation of the coefficient used for estimating PM_{10} mortality in Russia. It is not known whether the general composition of particulates and the population mix in Volgograd differ substantially from those found in areas where previous PM_{10} epidemiology studies have been conducted.

Regarding demographic and exposure factors, only ambient exposures have been considered. Human mobility patterns and indoor/outdoor differences in exposures may bias the results differentially depending on age, residence location, building characteristics, and occupational and personal characteristics.

4. CONCLUSIONS

4.1 Health Risks in Volgograd

The main objective of this study was to show how existing quantitative health risk assessment methods can be implemented using existing data and air dispersion modeling capabilities that are commonly available in Russia and other parts of the former Soviet Union. The Volgograd study did fulfill this objective. Health risk estimates were derived for both carcinogenic chemicals and noncarcinogenic particulate matter (PM_{10}) via the inhalation exposure route. The mortality risks posed by PM_{10} emissions are estimated at 2,700 additional deaths per year in the city (population of about 1 million), while health risks posed by carcinogenic emissions are estimated at 13 additional cancer cases per year.

Even with the substantial uncertainties associated with the risk assessment methods and underlying data, particulate mortality risks appear to be unacceptably high in Volgograd and to warrant closer consideration by environmental and health authorities. At a time of tight budgets for Volgograd city and its enterprises, existing carcinogen emission levels probably do not warrant priority action on the part of the city, however.

While no other quantitative health risk assessments of PM_{10} are known to have been conducted in Russia, studies from the U.S. can be used to put the Volgograd results in context. A recent study by the Natural Resource Defense Council (NRDC, 1996), for example, estimated mortality health risks from PM_{10} for 48 locations in the U.S. Average annual mean concentrations of PM_{10}, based on directly measured air quality, ranged from about 60 to 30 micrograms per cubic meter (the U.S. annual standard is currently 50 micrograms per cubic meter) (NRDC, 1996, p. 78). Based on such concentrations, annual mortality was estimated in the range of 1,230 deaths per million to 450 deaths per million (NRDC, 1996, p. 79). At a couple of local 'hot spots,' average annual concentrations were between about 80-90 micrograms per cubic meter.

In Volgograd, average annual PM_{10} levels are estimated at over 100 micrograms per cubic meter for ten receptor points for facility 314 and for three receptor points for facility 315 (see tables 5.5 and 5.6). Just from emissions from these two facilities, average annual estimated concentrations are 100 percent higher than the U.S. PM_{10} annual ambient air quality standard (the concentration at receptor 19 for facility 314 is more than 1,000 micrograms per cubic meter).

4.2 Implications for Russian Environmental Policy

For the city of Volgograd, the logical next step for environmental policy makers would be to undertake an analysis of potential cost-effective reductions in mortality risks from particulates. While such a risk management analysis is not included here, an indication is provided below of how this analysis could proceed. Regarding particulates and the two main facilities (314 and 315) causing PM10 mortality risks in Volgograd, analysis of abatement options has already been completed by the Russian Air Management Project (RAMP), which is financed by the U.S. government and implemented by the U.S. EPA, Volgograd city officials, and various local and foreign consultants. Fortunately, the RAMP project has identified several low- and high-cost particulate control options for these two facilities, including capital and operating/maintenance costs.

A follow-up risk management analysis, which will be completed as part of the same HIID project that implemented the Volgograd risk assessment, will be to evaluate these particulate control options in terms of cost effectiveness. Initial analysis indicates that the marginal cost of reducing PM_{10} mortality risks remains quite low up to some important level of risk reduction (perhaps up to a 30 percent reduction), after which marginal costs increase substantially.

Regarding particulate risks, environmental and health authorities in Volgograd and elsewhere in Russia may be tempted to ask the following question: *since the facilities are operating within their PDV emission levels, why are mortality risks from particulates so high?*

There are two parts to the answer. First, Russia does not regulate "particulates" as a single substance. Recall from Table 4 that "particulates" includes many different substances. Russian has a PDK for each individual substance, but not a PDK for particulates as a group. Based on Ecolog air dispersion modeling, proposed emission rates for individual substances could thus all satisfy the individual PDK levels, but the summed ambient concentrations representing all particulates could be enormous. Russia needs to develop a regulatory framework for managing particulates as an aggregate category, such as PM_{10} . Environmental authorities may need additional technical training to allow them to implement and enforce such a new approach to particulates.

Second, Russia does not currently consider chronic health effects when setting facility emission limits. The current study provides one simple framework for evaluating chronic health effects from annual particulate emission levels within the current Russian regulatory framework. Calculations of chronic risk would provide important additional information for environmental authorities to use when setting PDV levels.

Rather than continuing to calculate chronic risk using existing models such as Ecolog, however, the capacity to model air dispersion over longer time periods and under more complex weather conditions is needed. Work in this area is currently underway in Russia. Since there are many existing air dispersion models and computer programs that can perform such analysis, including models already translated into Russian, improving local modeling methods does not

need to be a complicated or time consuming process. A main constraint for implementing better models will be the accessibility and quality of the meteorological information residing mainly with the Hydromet organization. A useful project for foreign assistance financing would be to support the creation of a meteorological modeling data base (wind rose, stability classes, etc.), beginning with major cities and industrial areas in Russia, to facilitate better air dispersion modeling. In some locations, it may be necessary to establish new, reliable meteorological monitoring stations for state-of-the-art air quality modeling.

Abbreviations used in the report

CDI	chronic daily intake
EPC	exposure point concentration
PDK	ambient air concentration limit
PDV	facility emission limit
RfC	reference concentration
RfD	reference dose
SF	slope factor
TSP	total suspended particulates
URF	unit risk factor

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Table 1. List of Volgograd enterprises included in the case study

Code	Name	Industry Type
CENTRAL ZONE		
105	Prefab Blocks Construction Plant-1	Construction materials
117	Asphalt-Concrete Plant under Road Constructing Co	Construction materials
122	Drilling Machines Plan	Machine building
136	Silica-Building Materials Plant	Construction materials
137	Silica-Insulation Materials	Construction materials
144	Plant TD&N (Tractor Spares)	Agricultural machinery
168	Experimental Van Plant	Furniture, armored kiosks
169	Volgogradytftemash (Oil Production and Refinery Equipment Plant)	Machine building
174	"Gips" Joint Stock Co (Gypsum)	Construction materials
180	"Bioden" JSCo	Food industry
SOUTH ZONE		
211	Volgo hydro-power Plant	Energy sector
213	Volgograd Furniture - Woodworking Production Co	Wood processing
214	RPA "Chimprom" (Chemical Industry Plant)	Chemical industry
218	Locomotive shop, station Sarepta	Locomotive shop
220	Techuglerod Plant (Technical Carbon Plant)	Oil-organic synthesis
237	Steel-wire- rope Plant	Ferrous metallurgy
239	Heat Power Plant N 3	District heating
241	Heat Power Plant N 2	District heating
249	Shipbuilding Plant	Shipbuilding plant
25 0	Production Co Vtorchermet (Recycling ferrous Metal	Ferrous metallurgy
252	Production Co Lukoil (All Russian Refinery Co)	Oil-organic synthesis
253	Production Co Caustic, JSCo " Plastcard"	Chemical industry
254	Ceramic Plant	Ceramics
NORTH ZONE		
303	Tool Plant Production Co, Volgograd Tractor Plant"VGTZ"	Agricultural machinery
310	Volgograd Tractor Plant	Agricultural machinery
312	Reinforced Concrete Articles Plant N2, Ltd	Construction materials
314	Aluminum Plant	Non-ferrous metallurgy
315	"Red October "Plant	Ferrous metallurgy
341	Production Co "Barricady"	Military

Table 2. Substances Included in the Volgograd Health Risk Assessment

Carcinogens

Benzene
Benzo(a)pyrene
Benzyl chloride
Cadmium
Carbon tetrachloride
Chloroform
Chromium
Formaldehyde
Nickel
Polyvinyl chloride dust
Soot (modeled as 0.5 % B(a)P)
Vinyl chloride

Noncarcinogens

Particulate matter (modeled as PM_{10})

Table 3. Summary Emission Inventories for Selected Facilities, 1995

Facility Code	Total Carcinogens*, t/yr	Total Particulates (TSP), t/yr
105		1523
117		10
122	0.060	20
136	0.028	280
137	0.004	272
144	0.009	8
168	0.004	4
169	0.000	150
174		13
180	0.055	0
211		809
213	10.662	100
214	552.714	2480
218	9.233	12
220	25.693	22
237		6
239	31.255	42
241		22
249	0.050	43
250	4.850	199
252	197.018	45
253	626.106	264
254	0.000	334
303	0.000	6
310	5.102	228
312		0
314	916.947	10003
315	0.007	2169
341	9.907	102

* Total carcinogens include soot directly, not 0.5% Benzo(a)pyrene as modeled.

Table 4. Sample Definition of Total Suspended Particles (TSP)

TSP Composition for Facility 314

Aluminum Oxide
Barium Chloride
Vannadium Pentoxide
Iron Oxide
Manganese and its compounds
Sodium hydroxide
Soot
Fluoride non-organic compounds
Naphthalene
Antracene
Phenantrene
Pyrene
Stearine
Suspended solids
Boiler oil soot in terms of Vanadium
Non-organic dust with Silicon oxide content 20-70%
Non-organic dust with Silicon oxide content more than 70%
Asbestos dust
Wood dust

TSP Composition for Facility 315

Iron Oxide
Potassium Oxide
Manganese and its compounds
Sodium hydroxide
Chromium Hexavalent
Colophony
Dust non-differentiated
Non-organic dust with Silicon oxide content 20-70%
Non-organic dust with Silicon oxide content more than 70%
Wood dust

Table 5.1 Cancer Risk for Facility 122

				Cadmium		Nickel		Chromium VI		Carbon Tetrachloride		Formaldehyde		Total
Receptor Points	Normalized Modeling Results, (ug/m3)/(g/s)	Fraction of Operating Time in 1995	Steadiness of Emissions Factor	Annual Concentration, ug/m3	Population Cancer Risk	Annual Concentra-tion, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	
1														
2														
3														
4														
5														
6														
7	1.0	0.46	0.34	0.00001	1.24E-05	0.00001	2.43E-06	0.001	1.09E-01	0.001	1.08E-05	0.0005	4.59E-06	1.09E-01
8	1.8	0.46	0.34	0.00001	1.24E-05	0.00001	2.43E-06	0.023	1.92E-01	0.002	2.16E-05	0.0009	8.27E-01	1.92E-01
9	1.4	0.46	0.34	0.00001	1.24E-05	0.00001	2.43E-06	0.018	1.51E-01	0.002	2.16E-05	0.0007	6.33E-06	1.51E-01
10	1.0	0.46	0.34	0.00001	1.24E-05	0.00001	2.43E-06	0.013	1.09E-01	0.001	1.08E-05	0.0005	4.59E-06	1.09E-01
11	4.3	0.46	0.34	0.00002	2.49E-05	0.00003	7.29E-06	0.054	4.52E-01	0.005	5.41E-05	0.0021	1.93E-05	4.52E-01
12	1.8	0.46	0.34	0.00001	1.24E-05	0.00001	2.43E-06	0.023	1.92E-01	0.002	2.16E-05	0.0009	8.27E-06	1.92E-01
13	0.9	0.46	0.34			0.00001	2.43E-06	0.011	9.20E-02	0.001	1.08E-05	0.0004	3.67E-06	9.21E-02
14	4.0	0.46	0.34	0.00002	2.49E-05	0.00003	7.29E-06	0.05	4.18E-01	0.005	5.41E-05	0.0019	1.47E-05	4.18E-01
15	1.7	0.46	0.34	0.00001	1.24E-05	0.00001	2.43E-06	0.021	1.76E-01	0.002	2.16E-05	0.0008	7.35E-06	1.76E-01
16	0.8	0.46	0.34			0.00001	2.43E-06	0.01	8.37E-02	0.001	1.08E-05	0.0004	3.67E-06	8.37E-02
17	0.6	0.46	0.34					0.008	6.69E-02	0.001	1.08E05	0.0003	2.76E-06	6.70E-02
18	0.1	0.46	0.34					0.001	8.37E-03					8.37E-03
19	0.1	0.46	0.34					0.001	8.37E-03					8.37E-03
20														
Nearest	8.3	0.46	0.34	0.00004		0.00006		0.104		0.001		0.004	3.67E-05	
Total					1.24E-04		3.40E-05		2.06E+00		2.49E-04		8.63E-05	2.06E+00

Table 5.2 Cancer Risk for Facility 214

				Benzyl Chloride		Vinyl Chloride		Chloroform		Carbon Tetrachloride		Formaldehyde		Total
Receptor Points	Normalized Modeling Results, (ug/m3)/(g/s)	Fraction of Operating Time in 1995	Steadiness of Emissions Factor	Annual Concentration, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	
1	0.3	0.73	0.74	0.018	6.24E-04	3.479	2.09E-01	0.141	2.33E-03	4.273	4.62E-02	0.003	2.76E-05	2.59E-01
2	0.5	0.73	0.74	0.029	1.01E-03	5.799	3.49E-01	0.235	3.88E-03	7.122	7.70E-02	0.005	4.59E-05	4.31E-01
3	1.1	0.73	0.74	0.064	2.22E-03	12.757	7.68E-01	0.516	8.53E-03	15.668	1.69E-01	0.012	1.10E-04	9.48E-01
4	1.7	0.73	0.74	0.099	3.43E-03	19.715	1.19E+00	0.797	1.32E-02	24.214	2.62E-01	0.019	1.74E-04	1.47E+00
5	0.7	0.73	0.74	0.041	1.42E-03	8.118	4.89E-01	0.328	5.42E-03	9.97	1.08E-01	0.008	7.35E-05	6.03E-01
6	0.4	0.73	0.74	0.023	7.98E-04	4.639	2.79E-01	0.188	3.11E-03	5.697	6.16E-02	0.004	3.67E-05	3.45E-01
7	0.2	0.73	0.74	0.012	4.16E-04	2.319	1.40E-01	0.094	1.55E-03	2.849	3.08E-02	0.002	1.84E-05	1.72E-01
8	0.2	0.73	0.74	0.012	4.16E-04	2.319	1.40E-01	0.094	1.55E-03	2.849	3.08E-02	0.002	1.84E-05	1.72E-01
9	0.2	0.73	0.74	0.012	4.16E-04	2.319	1.40E-01	0.094	1.55E-03	2.849	3.08E-02	0.002	1.84E-05	1.72E-01
10	0.2	0.73	0.74	0.012	4.16E-04	2.319	1.40E-01	0.094	1.55E-03	2.849	3.08E-02	0.002	1.84E-05	1.72E-01
11														
12														
13														
14														
15														
16														
17														
18														
19														
20														
Nearest	5.8	0.73	0.74	0.338		67.263		2.721		82.612		0.063		
Total					1.12E-02		3.84E+00		4.27E-02		8.47E-01		5.42E-04	4.74E+00

Table 5.3 Cancer Risk for Facility 314

				Soot		Benzene		Benzo(a)pyrene		Total
Receptor Points	Normalized Modeling Results, (ug/m3)/(g/s)	Fraction of Operating Time in 1995	Steadiness of Emissions Factor	Annual Concentration, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	
1				0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	
2				0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	
3				0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	
4				0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	
5				0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	
6				0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	
7	0.2	1.0	0.76	4.6960	0.0350	0.6610	0.0039	0.0001	0.0001	
8	0.2	1.0	0.76	4.6960	0.0350	0.6610	0.0039	0.0001	0.0001	0.0391
9	0.2	1.0	0.76	4.6960	0.0350	0.6610	0.0039	0.0001	0.0001	0.0391
10	0.2	1.0	0.76	4.6960	0.0350	0.6610	0.0039	0.0001	0.0001	0.0391
11	0.4	1.0	0.76	9.3930	0.0700	1.3220	0.0078	0.0002	0.0003	0.0391
12	0.4	1.0	0.76	9.3930	0.0700	1.3220	0.0078	0.0002	0.0003	0.0781
13	0.4	1.0	0.76	9.3930	0.0700	1.3220	0.0078	0.0002	0.0003	0.0781
14	0.5	1.0	0.76	11.7410	0.0875	1.6530	0.0098	0.0002	0.0003	0.0976
15	0.6	1.0	0.76	14.0890	0.1049	1.9840	0.0117	0.0002	0.0003	0.1170
16	1.4	1.0	0.76	32.8740	0.2449	4.6280	0.0274	0.0006	0.0009	0.2730
17	1.3	1.0	0.76	30.5260	0.2274	4.2980	0.0254	0.0005	0.0007	0.2530
18	3.1	1.0	0.76	72.7930	0.5422	10.2490	0.0607	0.0013	0.0019	0.6050
19	3.5	1.0	0.76	82.1860	0.6122	11.5710	0.0685	0.0014	0.0021	0.6830
20	2.0	1.0	0.76	46.9630	0.3498	6.6120	0.0391	0.0008	0.0012	0.3900
Nearest	8.8	1.0	0.76	206.6390		29.0930		0.0036		
Total					2.5188		0.2817		0.0089	2.8090

Table 5.4 Cancer Risk for Facility 253

				Benzene		Vinyl Chloride		PVC Dust		Total
Receptor Points	Normalized Modeling Results, (ug/m3)/(g/s)	Fraction of Operating Time in 1995	Steadiness of Emissions Factor	Annual Concentra-tion, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	Annual Concentration, ug/m3	Population Cancer Risk	
1	1.3	0.99	0.62	5.308	3.14E-02	9.142	5.50E-01	0.447	2.69E-02	6.10E-01
2	0.8	0.99	0.62	3.267	1.93E-02	5.626	3.39E-01	0.275	1.66E-02	3.76E-01
3	0.4	0.99	0.62	1.633	9.66E-03	2.813	1.69E-01	0.137	8.25E-03	1.88E-01
4	0.2	0.99	0.62	0.817	4.84E-03	1.406	8.46E-02	0.069	4.15E-03	9.39E-02
5	0.1	0.99	0.62	0.408	2.41E-03	0.703	4.23E-02	0.034	2.05E-03	4.69E-02
6	0.1	0.99	0.62	0.408	2.41E-03	0.703	4.23E-02	0.034	2.05E-03	4.69E-02
Nearest	1.9	0.99	0.62	7.758		13.361		0.653		
Total					7.01E-02		1.23E+00		6.00E-02	1.36E+00

Table 5.5 Annual PM10 Mortality for Facility 315

Receptor Points	Normalized Modeling Results, (ug/m3)/(g/s)	Fraction of Operating Time in 1995	Steadiness of Emissions Factor	Annual Concentration, ug/m3	Population Mortality Risk
1					
2					
3					
4					
5					
6					
7	0.2	1	0.71	12.60	5.25
8	0.3	1	0.71	18.89	7.87
9	0.5	1	0.71	31.49	13.12
10	0.7	1	0.71	44.09	18.37
11	0.9	1	0.71	56.68	23.62
12	1.7	1	0.71	107.06	44.61
13	6.7	1	0.71	421.96	175.82
14	0.8	1	0.71	50.38	20.99
15	1.4	1	0.71	88.17	36.74
16	1.7	1	0.71	107.06	44.61
17	1.4	1	0.71	88.17	36.74
18	0.7	1	0.71	44.09	18.37
19	0.8	1	0.71	50.38	20.99
20	0.2	1	0.71	12.60	5.25
Nearest	4.6	1	0.71	289.70	
Total					472.34

Table 5.6 Annual PM10 Mortality for Facility 314

Receptor Points	Normalized Modeling Results, (ug/m3)/(g/s)	Fraction of Operating Time in 1995	Steadiness of Emissions Factor	Annual Concentration, ug/m3	Population Mortality Risk
1					
2					
3					
4					
5					
6					
7	0.2	1	0.76	58.9880	24.58
8	0.2	1	0.76	58.9880	24.58
9	0.2	1	0.76	58.9880	24.58
10	0.2	1	0.76	58.9880	24.58
11	0.4	1	0.76	117.9760	49.16
12	0.4	1	0.76	117.9760	49.16
13	0.4	1	0.76	117.9760	49.16
14	0.5	1	0.76	147.4700	61.45
15	0.6	1	0.76	176.9640	73.74
16	1.4	1	0.76	412.9170	172.05
17	1.3	1	0.76	383.4230	159.76
18	3.1	1	0.76	914.3160	380.97
19	3.5	1	0.76	1032.2930	430.12
20	2.0	1	0.76	589.8820	245.78
Nearest	8.8	1	0.76	2595.4790	
Total					1769.74

Table 5.7 Annual PM10 Mortality for Facility 214

Receptor Points	Normalized Modeling Results, (ug/m3)/(g/s)	Fraction of Operating Time in 1995	Steadiness of Emissions Factor	Annual Concentration, ug/m3	Population Mortality Risk
1	0.3	0.73	0.74	5.154	2.15
2	0.5	0.73	0.74	8.590	3.58
3	1.1	0.73	0.74	18.897	7.87
4	1.7	0.73	0.74	29.205	12.16
5	0.7	0.73	0.74	12.025	5.01
6	0.4	0.73	0.74	6.872	2.86
7	0.2	0.73	0.74	3.436	1.43
8	0.2	0.73	0.74	3.436	1.43
9	0.2	0.73	0.74	3.436	1.43
10	0.2	0.73	0.74	3.436	1.43
11					
12					
13					
14					
15					
16					
17					
18					
19					
20					
Nearest	5.8	0.73	0.74	99.640	
Total					39.37

Table 6. Weighting Factors Used to Estimate Difference between 20-minute and Annual Averaging Times

Facility Code	Fraction of operating time throughout 1995	Factor wich reflects steadyness of the emissions
105	0.23	0.45
117	0.17	0.09
122	0.46	0.34
136	0.96	0.3
137	0.99	0.63
144	0.14	0.47
168	0.23	0.18
169	0.68	0.21
174	0.18	0.32
180	0.47	0.67
211	0.74	0.27
213	0.23	0.41
214	0.73	0.74
218	1	0.24
220	1	1
237	0.95	0.27
239	0.74	0.44
241	0.74	0.51
249	0.23	0.24
250	0.23	0.39
252	1	0.9
253	0.99	0.62
254	0.73	0.46
303	0.14	0.38

Table 7. U.S. EPA Slope Factors (SFs) for the Identified Carcinogens for the Inhalation Exposure Route (SF units = $(mg/kg\text{-}day)^{-1}$) .

Benzene	0.029
Benzo(a)pyrene	7.3
Benzyl chloride	0.17
Cadmium	6.1
Carbon tetrachloride	0.053
Chloroform	0.081
Chromium	41
Formaldehyde	0.045
Nickel	1.19
Polyvinyl chloride dust	0.095
Soot (0.5% BAP)	7.3
Vinyl chloride	0.095

Table 8. Annual Population Cancer Risk in Volgograd by Facility and Receptor Points (sum of individual carcinogens)

	Receptors 1-20. Each receptor represents 50,000 individuals.																				
Facility #	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	Annual Mortality
105																					
117																					
122							0.1090	0.1920	0.1510	0.1090	0.4520	0.1920	0.0920	0.4180	0.1760	0.0837	0.0669	0.0084	0.0084		2.0583
136							0.0000	0.0001	0.0001	0.0001	0.0004	0.0003	0.0001	0.0013	0.0008	0.0004	0.0002	0.0002	0.0002	0.0000	0.0041
137						0.0005	0.0009	0.0012	0.0012	0.0010	0.0048	0.0023	0.0013	0.0160	0.0034	0.0014	0.0010	0.0009	0.0012		0.0369
144							0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000		0.0001
168						0.0008	0.0009	0.0000	0.0000												0.0017
169				0.0000	0.0009	0.0027	0.0018	0.0009	0.0009	0.0000											0.0071
174																					
180			0.0002	0.0005	0.0013	0.0027	0.0061	0.0022	0.0022	0.0016	0.0006	0.0006	0.0005	0.0005	0.0003						0.0194
211																					
213	0.0006	0.0008	0.0013	0.0059	0.0253	0.0079	0.0016														0.0434
214	0.2580	0.4300	0.9460	1.4700	0.6020	0.3440	0.1720	0.1720	0.1720	0.1720											4.7380
218	0.0063	0.0106	0.0658	0.0058	0.0030	0.0023	0.0015														0.0953
220	0.0338	0.0169	0.0085	0.0085	0.0085																0.0761
237																					
239	0.0013	0.0013																			0.0025
241																					
249	0.0284	0.1100	0.0502	0.0084	0.0050	0.0034															0.2053
250	0.0590	0.0927	0.3630	0.1520	0.0590	0.0420															0.7677
252	0.3960	0.1350	0.0730	0.0313	0.0261	0.0209															0.6823
253	0.6120	0.3770	0.1880	0.0942	0.0471	0.0471															1.3654
254	0.0019	0.0004	0.0001	0.0001	0.0001																0.0026
303																					
310						0.0004	0.0006	0.0006	0.0008	0.0010	0.0012	0.0015	0.0023	0.0014	0.0021	0.0043	0.0355	0.0232	0.0091	0.0027	0.0865
312																					
314							0.0391	0.0391	0.0391	0.0391	0.0781	0.0781	0.0781	0.0976	0.1170	0.2730	0.2530	0.6050	0.6830	0.3900	2.8093
315								0.0008	0.0008	0.0010	0.0018	0.0037	0.0126	0.0018	0.0027	0.0037	0.0027	0.0010	0.0018		0.0343
341							0.0002	0.0002	0.0002	0.0002	0.0002	0.0004	0.0004	0.0002	0.0004	0.0005	0.0014	0.0014	0.0004	0.0005	0.0067
Ann. mort.	1.3973	1.1747	1.6960	1.7766	0.7782	0.4746	0.3337	0.4091	0.3683	0.3250	0.5391	0.2788	0.1873	0.5368	0.3026	0.3668	0.3606	0.6400	0.7041	0.3932	13.0430

Table 9. Mortality Impacts due to Estimated PM10 Emissions in Volgograd, by Facility and by Receptor Points

	Receptors 1-20. Each receptor represents 50,000 individuals.																				
Facility #	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	Annual Mortality
105							0.93	1.70	1.55	1.08	12.07	3.72	1.55	18.33	4.80	1.39	0.77	0.16			48.04
117							0.04	0.05	0.05	0.05	0.24	0.19	0.10	1.34	0.48	0.14	0.06	0.06	0.09		2.90
122							0.40	0.72	0.56	0.40	1.71	0.72	0.36	1.59	0.68	0.32	0.24	0.04	0.04		7.76
136							0.71	0.95	1.07	1.07	4.39	3.56	2.14	14.96	8.79	3.80	1.66	1.42	2.61	0.59	47.73
137						0.28	0.51	0.68	0.68	0.57	2.72	1.30	0.74	9.13	1.93	0.79	0.57	0.51	0.68		21.08
144							0.09	0.11	0.19	0.31	0.35	1.28	1.41	0.21	0.38	0.15	0.12	0.09	0.10		4.79
168				0.04	0.08	0.22	0.36	0.11	0.12	0.08	0.04	0.04	0.03								1.12
169			0.24	0.48	1.68	4.86	3.36	1.32	1.26	0.90	0.42	0.36	0.30	0.30	0.24						15.73
174			0.18	0.27	0.94	2.54	2.41	0.89	0.89	0.63	0.27	0.27	0.22								9.51
180			0.01	0.28	0.07	0.16	0.35	0.13	0.13	0.09	0.04	0.04	0.03	0.03	0.02						1.35
211	1.61	1.61	4.04	8.07	4.04	2.42	0.81	0.81	0.81												24.22
213	0.32	0.40	0.64	2.95	12.60	3.91	0.80														21.61
214	2.15	3.58	7.87	12.17	5.01	2.86	1.43	1.43	1.43	1.43											39.37
218	0.50	0.85	5.26	0.46	0.24	0.18	0.12														7.62
220																					0.00
237	3.58	1.85	0.30	0.12	0.09	0.09	0.06														6.08
239	0.15	0.15																			0.31
241	0.14	0.14	0.05	0.05	0.05																0.42
249	0.71	2.79	1.27	0.21	0.13	0.09															5.21
250	0.29	0.48	1.87	0.80	0.32	0.21															3.97
252	25.21	8.62	4.64	1.99	1.66	1.33															43.45
253	4.78	2.94	1.47	0.74	0.37	0.37															10.66
254	52.21	10.67	2.29	1.14	1.14	0.76															68.21
303											0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.03	0.02	0.10	0.19
310						0.16	0.24	0.24	0.32	0.40	0.48	0.64	0.97	0.56	0.89	1.77	11.27	9.66	3.78	1.13	32.53
312								0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.02	0.02	0.04	0.06	0.03	0.22
314							24.58	24.58	24.58	24.58	49.16	49.16	49.16	61.45	73.74	172.05	159.76	380.97	430.12	245.78	1769.64
315							5.25	7.87	13.12	18.37	23.62	44.61	175.82	20.99	36.74	44.61	36.74	18.37	20.99	5.25	472.34
341							0.04	0.04	0.04	0.04	0.04	0.06	0.06	0.04	0.06	0.08	0.10	0.10	0.06	0.08	0.81
Annual mort.	91.66	34.09	30.12	29.76	28.42	20.46	42.48	41.64	46.79	50.00	95.55	105.95	232.88	128.94	128.74	225.14	211.32	411.44	458.55	252.96	2666.88

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