DELTA

FIVE-YEAR COMPARATIVE ASSESSMENT OF ESTIMATED VS. ACTUAL EMISSIONS AND ASSOCIATED HEALTH RISKS FROM A MODERN MUNICIPAL WASTE-TO-ENERGY FACILITY

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ABSTRACT

As part of a long-term solution to the collection, transfer, and disposal of solid waste, the City and County of Spokane, WA, developed a regional solid waste management system that includes the treatment of non-recyclable solid waste at a 800 ton-per-day waste-to-energy (WTE) facility. A baseline risk assessment was performed prior to facility operations that was based on estimates of stack emissions. This paper assesses the risks posed by exposure to compounds emitted from the stack using actual stack emissions data through 1995, and compares the present risk estimates with those from the baseline risk assessment. Standard U.S EPA and Washington Department of Ecology (WDOE) guidelines current as of 1993 were used for performing the risk assessments. Risks were estimated for four receptor locations: the maximum exposed individual onsite and offsite, the actual nearest residence, and the nearest school

Mercury, total chromium, PAHs, nickel and 2,3,7,8-TCDD showed the greatest decreases in emissions from the 1991 estimates, ranging from approximately 25 to 82 times lower. Reductions in emissions of beryllium, PCBs, arsenic, cadmium, and lead were within an order of magnitude of their original estimates. Actual emissions of hexavalent chromium increased three-fold compared to estimated emissions. Total carcinogenic risks for the maximum offsite location was approximately three times lower than risks estimated in 1991. The risk to the nearest resident, $2x10^{-8}$, was 500 times lower than regulatory levels considered protective of human health (i.e., 10^{-5}).

Although mercury was determined to be the compound of greatest potential concern for non-carcinogenic effects, exposure to mercury at the nearest resident was 50,000 times lower than the allowable threshold. The reduction in risk estimates in the present study from pre-operational risk estimates can be attributed to the use of actual stack emissions rather than estimated emissions An increase in certain exposure and toxicity factors which occurred in federal guidance between 1991 and 1993 somewhat offset the reduction in risks associated with reduced emissions.

The overall conclusion was that the Spokane WTE facility poses no adverse risk to the public based on estimated and measured stack emissions and soils data, and on EPA fate and transport and risk modeling. The unique nature of this investigation, where estimated and then actual emissions data were used to estimate risks, could assist in the more realistic assessment and management of risks from combustion facilities in the future.

INTRODUCTION

In 1991, the Spokane Regional Solid Waste System (SRSWS) constructed an 800 ton-per-day solid waste WTE facility near the Spokane International Airport in Washington State. The WTE facility is supported by three recycling and transfer stations and a residue landfill for disposal of ash and noncombustible materials. As a condition of approval of the project, WDOE requested an investigation into the potential impacts to human health caused by the emissions of the WTE facility. This paper provides a summary of the final investigation.

This study is unique in that virtually all human health risk assessments (HHRA) performed for municipal waste and other combustion facilities are based on estimates of stack emissions derived from either facilities similar in design or using worst-case scenarios (1) In contrast, the present risk assessment used actual emissions and monitoring data to verify and confirm the initial estimates calculated before the facility was built These initial estimates are rarely verified once a municipal waste combustion (MWC) facility is operational, but doing so has important implications for better local understanding of facility risks and presents an opportunity to refine federal risk assessment procedures.

In order to provide an initial estimate of potential public health risks associated with the operation of this facility, a baseline health risk assessment was performed prior to its construction (2). Because that risk assessment used estimated emissions rather than actual emissions data from the WTE facility stack, the estimated health risks for the facility were based on very conservative assumptions, in accordance with U S EPA guidelines on conducting health risk assessments for MWC facilities (3,4). As a result, the baseline risk assessment tends to overestimate potential health risks This paper assesses the risks posed

by exposure to compounds emitted from the stack using actual stack emissions data, air dispersion modeling, and ambient air monitoring data for four years after start-up, and compares the present risk estimates using actual emissions data with those from the baseline risk assessment

METHODS

To assess the risks of the WTE facility, we initially used U.S. EPA and Washington Department of Ecology risk assessment guidelines current as of 1991. The U.S EPA guidelines (3,4) describe procedures for estimating direct and indirect human exposures and health risks that can result from dispersion of emitted chemicals to ambient air and their transport to soil, vegetation, animals, and water Additional guidance on general risk assessment methodology was found in references (5) and (6).

Although additional guidance was issued before the completion of the project, the risk assessment methods remained the same from 1991 through 1995, with a few exceptions as noted below. As the intent of the investigation was to compare pre- and post-start-up risks using estimated vs actual emissions data, it became more important to use consistent risk assessment methods to ensure a consistent basis of comparison rather than incorporate updated methods over the five years of the project.

The method for performing the HHRA proceeded through the standard EPA four steps of risk assessment: data collection and evaluation, toxicity assessment, exposure assessment, and risk characterization. The following is a brief description of some of the salient features of each step as applied to this facility.

Data Collection and Evaluation

This step involves identifying the substances present in stack emissions that may present a risk to human health In this assessment, potential chemicals of concern were identified at the outset of the study by the Environmental Health Impact Study Advisory Committee charged with oversight of the project (7). The chemicals of concern included all the primary risk drivers of risk assessments of other combustion facilities as well as compounds of interest to the advisory group

METALS	ORGANICS
Arsenic	Polychlorinated Biphenyls (PCBs)
Beryllium	Polycyclic Aromatic Hydrocarbons (PAHs)
Cadmium	Polychlorinated Dibenzo-p-Dioxins (PCDDs)
Chromium	Polychlorinated Dibenzofurans (PCDFs)
Lead	
Mercury	
Nickel	

Toxicity Assessment

The toxicity assessment defines the relationship between the dose of a chemical and the resulting health effects. This assessment results in a quantitative measure of a concentration or dose of a chemical above which humans are at risk for systemic toxicity, or an estimate of carcinogenic potency or risk. It also includes qualitative aspects of the range of target organs, toxic effects, and sensitive populations that may be affected by chemicals of concern. Key dose-response variables developed by the U.S. EPA for use in quantitative risk assessment are slope factors for evaluating cancer risks, and reference doses (RfD) and concentrations (RfC) for evaluating risks of non-carcinogenic health effects All values were taken from U S EPA's Integrated Risk Information System (IRIS) database, except for dioxin which has been removed from IRIS. The slope factor for dioxin was taken from the HEAST tables.

Exposure Assessment

The exposure assessment estimates the type and magnitude of exposures to the chemicals of potential concern that are expected to be emitted from the stack and transported offsite The results of the exposure assessment were combined with chemical-specific toxicity information, developed in the Toxicity Assessment, to characterize potential risks The exposure assessment followed EPA guidance (3,4,5,6, 8,9) and certain California State guidelines (10) for estimating potential exposure to air emissions sources.

Emission rates were measured each year from 1991 to 1995, under normal conditions at full capacity These rates are compared with the estimated rates used in the 1991 HHRA in Table I, also under normal conditions at full capacity (The scope of this investigation was limited to stack emissions data taken in 1993 and 1995, and no trend is evident from the data presented in this table. However, additional stack tests taken in 1991, 1992, and 1994 indicate the 1995 data reflect a general trend in emissions reduction over time) The estimated rates for 1991 were based on stack test data from two facilities in the U.S. of similar design by Wheelabrator Environmental Systems Inc. Using the measured emission factors and gas flow rates from those facilities, emission rates for the Spokane WTE for the 1991 HHRA were conservatively estimated assuming operations at full capacity for 49 weeks of the year.

TABLE I.
ESTIMATED VS. ACTUAL EMISSION RATES

Compound	Emission Rate (μg/s) 1991 Est	1993 Meas	1995 Meas.	1991/1995
Inorganics				
Arsenic	73	88	13	6
Beryllium	65	2	5	13
Cadmium	33	28	24	1 3
Chromium, total	1188	570	24	50
Chromium VI	59	22	170	0 3
Lead	134	1376	112	1 2
Mercury	17842	4210	218	82
Nickel	1595	3730	45	35
Organics				
2,3,7,8-TCDD TEQ	0 001	0 0005	0 00004	25
PCBs	7	1	1	7
PAH TEQ	322	153	9	36

TEQ = Toxicity Equivalent

As recommended by U.S. EPA, exposures were calculated under upper-bound conditions (4,5) The upper-bound conditions incorporate a number of conservative assumptions in determining chemical intake rates and duration of exposure in order to estimate the reasonable maximum exposure (RME), defined as the highest exposure that is reasonably expected to occur at a site for the given exposure pathway

Chemicals in stack emissions that transport offsite can deposit onto soil or water and subsequently accumulate in the terrestrial or aquatic food chains. Humans can be exposed to these compounds by direct inhalation of air impacted by facility emissions, or indirectly through dermal contact, ingestion of soil, or ingestion of foods (plant or animal) grown in impacted soil or water Both direct and indirect exposures to stack emissions were considered in this risk assessment. The exposure pathways that were evaluated:

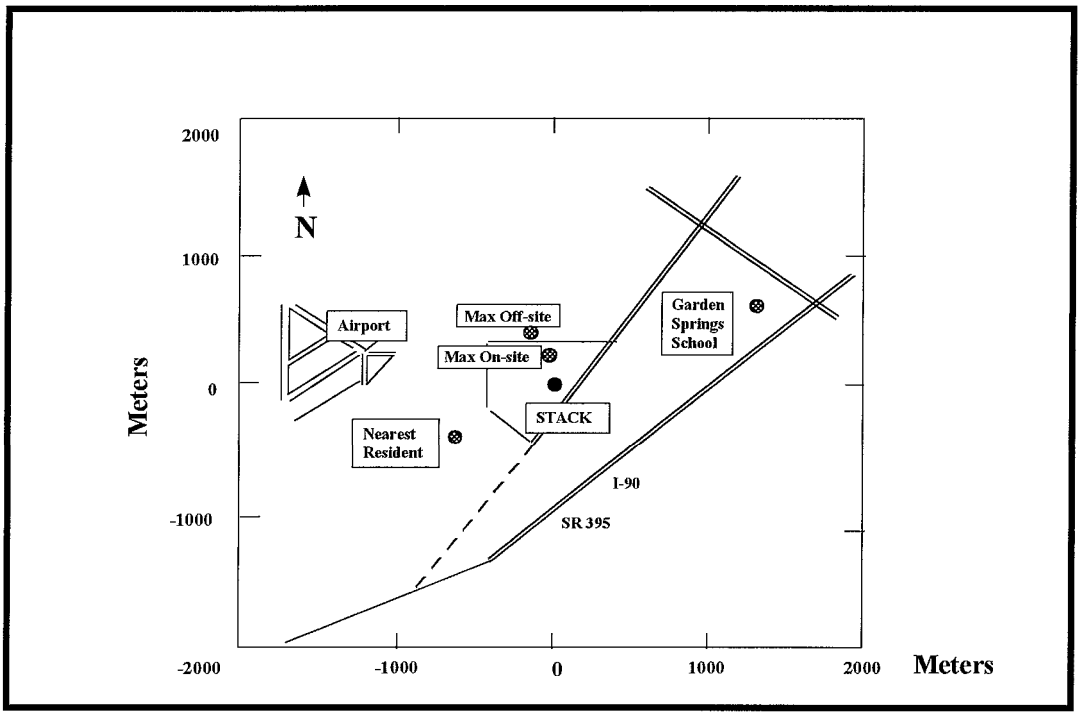
- Inhalation of emitted particulates and stack gases in ambient air
- Ingestion of compounds deposited on soil
- Direct contact with soil on which compounds have been deposited
- Ingestion of home-grown vegetables which may have been exposed to facility emissions

Other exposure pathways (ground and surface water, subsistence farming and fishing, and breast milk) were not evaluated, either because the exposure pathway was not complete, or because the pathway resulted in less exposure and risk than the above pathways and was screened from further evaluation

As defined by U.S EPA, exposures were identified for the maximum exposed individual (MEI) As the location of maximum air concentrations occurred onsite, the location of the maximum off-site impact was added. In addition, the actual nearest residence and the nearest school as a potentially sensitive population were identified as potential receptors. This resulted in a total of four receptor locations, as shown in Figure I

- Maximum air concentration (falls within facility boundary)
- Maximum off-site concentration (falls just outside facility boundary)
- Garden Springs school
- Nearest residence (ca 500 feet from facility boundary)

FIGURE I
LOCATION OF RECEPTORS



Exposure concentrations in media of concern (e g., air, soil, and garden vegetables) were determined from air dispersion modeling using actual stack emissions measured each year for four years after start-up. U S EPA exposure rates were derived from guidance manuals on public health evaluation (5) and exposure assessment factors (6,8,9,11)

Certain exposure intake and toxicity factors were modified in 1993 for the first post-start-up risk assessment to reflect updated guidance from U.S. EPA that was not available for the baseline HHRA in 1991 These included: a lower inhalation intake rate (from 30 to 20 m^3 per day), increased exposure

frequencies, increased absorption from inhaling PCBs and arsenic, an increased PAH cancer slope factor, a new reference dose for arsenic ingestion, and a new cancer slope factor for arsenic inhalation. These refinements to the methodology had the effect of increasing risk estimates somewhat over the methods used in 1991

To ensure consistency of comparison, no additional methodological changes were made in the final risk assessment completed in 1995 This allowed a more consistent comparison of estimated vs. actual risk over time without confounding the results with the effect of methodological changes, and was consistent with the scope and intent of the investigation to compare actual vs estimated risks using as consistent methods as possible Although the 1993 changes likely resulted in a somewhat more accurate estimate of risk and was thus a useful effort, incorporating further modifications in 1995 would not have had a major impact on the results and was not consistent with the scope of the effort, and was therefore not warranted

Upon derivation of the exposure intake factors, chemical and pathway-specific doses were calculated by multiplying the appropriate pathway exposure intake factor and media concentrations by chemical-specific absorption factors (see reference (2) for a more detailed description of chemical-specific absorption factors). These doses were then used to calculate carcinogenic and non-carcinogenic risks

Risk Characterization

For chemicals with carcinogenic effects, the cancer risk associated with a dose from a given route of exposure is calculated by multiplying the dose by the cancer potency factor for the given chemical. The predicted cancer risk is an upper-bound estimate of the potential risk associated with exposure For example, a risk of 10^{-5} means that a person has a one-in-100,000 upper-bound probability of developing cancer over a lifetime as a result of exposure to the given dose of the chemical at a given location A total pathway risk was calculated by summing the cancer risk estimates of the individual chemicals relevant to each pathway. Total carcinogenic risk is then estimated by summing carcinogenic risks for all chemicals for all pathways according to guidelines set forth in the U S EPA Guidance (5) Current U S. EPA guidance for remediating hazardous waste sites suggests a range of risks of 10^{-4} to 10^{-6} (one in 10,000 to one in a million) as an "acceptable" range of excess upper-bound lifetime cancer risks (12)

Non-carcinogenic risks are presented as a hazard quotient, which is the ratio between the calculated dose or concentrations in air and the RfD or RfC, respectively. Doses or concentrations for each chemical and exposure pathway are compared with the route-specific RfD or RfC If the hazard quotient is less than unity (one), then no adverse health effects are expected. If the hazard quotient exceeds unity, then the potential may exist for non-carcinogenic health risks As a screening procedure, summation of the hazard quotients with like biological endpoints resulting in a hazard index of less than one suggests no adverse health effects would occur from non-carcinogenic chemical exposures

RESULTS OF RISK CHARACTERIZATION

The following section discusses the results of carcinogenic and non-carcinogenic risk calculations for the chemicals of concern in air, soil, and vegetables for the present study using actual stack emissions data

Carcinogenic Risks

Total carcinogenic risks for the 1995 evaluation fell well below risk values currently considered acceptable for protection of human health by the U.S. EPA (12), ranging from $5x10^{-9}$ to $5x10^{-7}$ for reasonable upper-bound exposure conditions at the point of maximum offsite exposure (Table II) Using actual emissions data, the pathway contributing the most to total risk was generally inhalation, with the indirect pathways of ingestion of soils and garden vegetables and dermal contact each contributing up to two orders of magnitude less than inhalation to the total risk for these two receptors (maximum offsite and nearest resident) Because of low rates of measured emissions from the stack, dioxins and furans consistently contributed the lowest carcinogenic risks Risks were one to two orders of magnitude lower at other offsite receptor locations, and lower still for average instead of maximum exposure scenarios

Risks for dermal contact of $2x10^{-10}$ were associated with the nearest resident receptor location, whereas the maximum impact receptor locations were associated with risk levels of $5x10^{-9}$. These risks are based on the evaluation of organic chemicals only. Exposure to metals via dermal contact was not considered a pathway of concern due to the physical and chemical characteristics of inorganic compounds (10)

TABLE II
CARCINOGENIC RISK – 1991 VS 1995

	Maximum Offsite			Closest Resident		
Pathway	1991	1993	1995	1991	1993	1995
Inhalation	7E-	2E-7*	5E-7*	5E-8	1E-8*	2E-8*
Ingestion						
Soil	4E-8	1E-8	1E-8	4E-9	2E-9	2E-10
Vegetable	9E-7*	3E-8	1E-7	7E-8*	3E-9	1E-9
Dermal Contact	3E-9	1E-9	5E-9	4E-10	1E-10	2E-10
Total Risk	*2E-6*	*2E-7*	*6E-7*	*1E-7*	*2E-8*	*2E-8*
Reduction 1991-1995			*3 3x*			*5x*
Amount Below 10^{-5}			*17x*			*500x*

* = *Highest value for this combination of year/receptor/pathway*

Non-Carcinogenic Risks

Both the chemical and pathway-specific hazard quotients and the overall hazard index for non-carcinogenic risks were lower than unity (i.e., one). (Because they were collectively lower than one, the hazard quotients were not grouped by common biological endpoint. Doing so would have resulted in several smaller hazard indices that are individually and collectively well below unity) These results indicate that none of the chemical doses exceeded the non-carcinogenic toxicity criteria for any of the receptor locations, and that the likelihood of adverse non-carcinogenic health effects due to facility emissions is well below applicable regulatory thresholds.

A toxicity criterion for non-carcinogenic effects via inhalation was only available for mercury As mercury was the main driver of non-carcinogenic risks (in part due to the significant contribution of the inhalation pathway for mercury risk), data are presented in Table III only for that element The hazard quotient for exposure to mercury via inhalation was 0 0003 at the maximum offsite location and 0 00002 for the closest resident, representing 100- to 150-fold reductions from 1991 estimates

TABLE III.
MERCURY HAZARD QUOTIENTS – 1991 VS. 1995

Pathway	Maximum Offsite 1991	1993	1995	Closest Resident 1991	1993	1995
Inhalation	0.02	0 003	0.0002	0.001	0.0002	0.00001
Ingestion						
Soil	0 003	0.001	0.00005	0.0003	0.0001	0.000004
Vegetable	0.01	0 003	0.0001	0 002	0 0004	0 00001
Total Risk	*0 03*	*0 007*	*0.0004*	*0 003*	*0.0007*	*0.00002*
Reduction 1991 to 1995			*100x*			*150x*
Amount Below Unity			*3,300x*			*50,000x*

COMPARISON OF PRE-OPERATIONAL HEALTH RISK ESTIMATES WITH RISKS UNDER CURRENT OPERATING CONDITIONS

This section compares the results of the baseline risk assessment conducted in 1991 with the results of the current study This study was performed using actual stack emissions data taken through 1995 and the risk assessment methodology presented in (13). To summarize the results, concentrations of compounds in air and soil from air dispersion and deposition modeling using actual stack emissions data were less than those using estimated emissions based on similar operating facilities As shown in Table I, measured emissions of mercury, total chromium, PAHs, nickel and 2,3,7,8-TCDD decreased the most from the 1991 estimates, ranging from approximately 25 to 82 times lower. Reductions in emissions of beryllium, PCBs, arsenic, cadmium, and lead were within an order of magnitude of their original estimates Actual emissions of hexavalent chromium increased three-fold compared to estimated emissions, the only compound to show an increase over its 1991 estimate

The soil sampling data offered an opportunity to measure the concentration of the compounds of concern in the soil before the facility was built ("background") versus the expected increase in these compounds resulting from deposition of facility emissions as estimated by the modeling. Table IV shows the incremental increases in risk for two receptor locations, the maximum offsite location and the closest resident. At the maximum offsite location, for example, the existing background concentrations of arsenic were 7,429 times higher than the additional arsenic estimated to be deposited at this location from stack emissions. Of the compounds of concern, mercury was estimated to contribute the largest incremental increase relative to background concentrations However, given modeling uncertainties and background variability, it is difficult to estimate the significance of these modeled data and to determine which increases are distinguishable from background concentrations.

TABLE IV
BACKGROUND SOIL VS. MODELED CHEMICAL CONCENTRATIONS (mg/kg)

Compound	Maximum Offsite			Closest Resident		
	Bkgrnd*	Modeled**	*Diff*	Bkgrnd	Modeled	*Diff*
Inorganics						
Arsenic	2 6E+0	3 5E-4	*7,429*	2 6E+02	68E-8	*97,000,000*
Beryllium	4 0E-1	1 6E-5	*25,000*	4 1E-1	2 0E-4	*2,050*
Cadmium	4 5E+0	1 1E-4	*40,909*	1 3E+0	8 6E-4	*1,512*
Chromium	4 3E+1	2 9E-3	*14,828*	3 2E-1	3 0E-2	*11*
Lead	6 6E+1	6 9E-3	*9,565*	8 0E+1	7 1E-2	*1,127*
Mercury	5 0E-2	1 7E-2	*3*	5 0E-2	1 3E-1	*4*
Nickel	5 3E+1	2 42E-2	*2,190*	8 2E+0	3 0E-1	*273*
Organics						
2,3,7,8-TCDD TEQ	1 1E-6	3 8E-10	*2,895*	1 7E-6	2 9E-9	*586*
PAH (B[a]P) TEQ	2 0E-2	3 8E-8	*526,316*	9 2E-2	2 9E-7	*317,241*

TEQ - Toxicity Equivalents

Source * Sampling
** Modeling

Comparison of risk estimates for the pre- and post-start-up operations are presented in Tables II, III and V. Total carcinogenic risks for all four receptor locations are approximately three times lower than risks estimated in the 1991 HHRA (Table II) The post-start-up total risk estimates for the maximum offsite location are 17 times lower than regulatory levels considered protective of human health (i.e , 10^{-5}), and 500 times lower at the location of the nearest resident

For non-carcinogenic risks, hazard indices for mercury were 100 to 150 times less in 1995 than hazard indices estimated in the 1991 HHRA for these two receptors (Table III) Exposures to mercury at these locations were 2,500 to 50,000 times lower than levels considered potentially of concern. A summary of the differences is provided in Table V These reductions are largely a reflection of the significant decreases in mercury emissions from 1991 to 1995, which more than offset the increased risks due to the refinements in the risk assessment methods

RESULTS OF OTHER MWC HEALTH RISK ASSESSMENTS

There have been several health risk assessments done for MWC facilities, including the 21 facilities described in (1) and the more recent study of the Robbins Resource Recovery facility near Chicago (14) These studies show that risk assessments of proposed facilities, relying heavily on estimated emissions and computer-based models, consistently result in risks just below regulatory thresholds of concern (i.e., generally 10^{-5} to 10^{-6} for cancer risk) Due to the high level of uncertainty inherent in the risk assessment process default values, reaching risk estimates below levels of regulatory concern can be readily accomplished by replacing default assumptions with more site-specific data until the uncertainty is reduced to below the applicable threshold.

The published literature contains only one other assessment of a MWC facility using actual measurements (15) Due to space constraints, only the highlights of this study can be given here, although the parallels

with this study are evident. It can be described as broader in scope but less in depth than the Spokane study This study of the Rutland, Vermont, MWC facility was sponsored by EPA and published in 1991 Its purpose was to determine the levels of compounds in the air, soil, sediment, water and agricultural products (milk, carrots, potatoes, forage) attributable to the MWC, and whether there were human health risks attributable to the operation of the facility. A total of 135 ambient air samples and an unknown number of other samples of various media were collected at four locations at or near the point of maximum estimated exposure (MEI) between October 1987 and February 1989 Milk, carrot, potato and forage samples were collected from farms in the area surrounding the incinerator Water, sediment, soil and milk samples were taken twice prior to and once after facility startup Potatoes and forage were sampled once, and a carrot was sampled once before facility startup.

TABLE V
COMPARISON OF RISK – 1991 VS 1995

	1991	1995	Difference
Pathway of Greatest Concern	Ingestion	Inhalation	
Carcinogens			
Highest Max Offsite Risk	2E-6	6E-7	3x less
Highest Nearby Res Risk	1E-7	2E-8	5x less
Highest School Risk	3E-7	1E-7	3x less
Mercury			
Highest Max Offsite Risk	3E-2	3E-4	100x less
Highest Nearby Res Risk	3E-3	2E-5	150x less
Highest School Risk	6E-3	7E-9	857,143x less

The MEI was less than 2 km from the plant, as determined by air dispersion modeling (ISCLT and LONGZ) in this rural area characterized by complex terrain The samples were analyzed for arsenic, beryllium, cadmium, chromium, mercury, nickel, lead, benzo(a)pyrene, PCBs, and dioxins/furans Most concentrations were below the analytical limits of detection, and lead was the most frequently detected compound. (detected in 108/122 sampled, DL of 0.0061 $\mu g/m^3$) Mercury concentrations were not reported "because of problems associated with precision" (15) Chromium and lead concentrations were detected in milk samples taken before facility startup, but were below the detection limit during facility operation.

The EPA concluded that "the objective of the study could not be attained because the majority of pollutants in the ambient air and environmental media were not present in concentrations that could be detected by the analytical methods employed This made a direct determination of the contribution of the incinerator to the measurable concentration of pollutants not possible " Further, "the measured pollutant concentrations in the ambient air or environmental media cannot be correlated with the emissions or operation of the MWC The MWC does not appear to be the primary source of these pollutants " Based on the above results, similar conclusions could be made of the Spokane MWC

An outside firm was subsequently contracted to critique the EPA study (16). Their conclusions.

- The analytes were present at levels within background variability (especially in soils).
- Annual average concentrations at the MEI were less than or equal to the analytical detection limit for all compounds except dioxins and furans.
- It would take 100 years to double most of the background metals concentrations, assuming the maximum rate of deposition
- Air concentrations of arsenic would have to be 1,000,000 times higher to exceed the detection limit in urine, a common measure of human exposure to arsenic
- For dioxins and furans, human exposure is dominated by sources other than the MWC.

DISCUSSION

Risk assessments completed for this WTE facility before it was built (2,17) both showed greater carcinogenic and non-carcinogenic risk estimates using estimated emissions compared to subsequent risks using actual measured emissions. As described in (2), risks calculated using estimated emissions in 1991 were all lower than 10^{-6} for carcinogenic risks and below regulatory thresholds for non-carcinogenic risks. The net reduction in risk estimates from pre-operational risk estimates can be attributed to at least two sources use of actual stack emissions rather than estimated emissions, and refinement of receptor exposure estimates.

In addition, toxicity criteria (i e , slope factors and RfDs) for some of the chemicals of concern changed between 1991 and 1995. For example, the estimates of the potency of arsenic for non-carcinogenic effects and of PAHs to cause cancer have increased since 1991. Both of these increases in potency estimates have been incorporated into the risk assessment and somewhat offset the reductions in risk estimates that were due to reduced stack emissions. Furthermore, pre-operational health risks for the WTE facility were estimated using emissions data from two other facilities similar in design (2) This approach was intentionally conservative and was recognized to likely overestimate actual emissions at the Spokane WTE facility All three facilities (i.e., the Spokane WTE and the two facilities used for emissions estimates in 1991) have similar air pollution control devices Stack parameters, operating loads, and gas flow rates used to estimate emission rates in the 1991 HHRA were similar to those during actual emission measurements for the current HHRA.

The unique nature of this investigation, using actual emissions data to estimate risks, will assist in making more realistic assessments of future risks from combustion facilities, enhance risk management decisions, and improve local understanding about the health impacts of these facilities. This investigation particularly demonstrates how the use of estimated stack emissions tends to overestimate risks to human health.

In summary, reductions in carcinogenic and non-carcinogenic risk values between 1991 and 1995 resulted primarily from the use of actual stack emissions data in the current study These reductions were somewhat offset by changes in exposure assumptions and toxicity criteria since 1991 that tended to increase the risks. The greatest change in risk estimates from 1991 can be attributed to the greatly lower stack emissions that were measured at the Spokane WTE compared to the conservative estimates used prior to its operation in 1991

CONCLUSIONS

We conclude that the Spokane WTE facility risks are significantly below regulatory standards for acceptable exposure to the public, based on estimated and measured stack emissions and soils data, and on EPA fate and transport and risk modeling. The differences between pre- and post-start-up risk estimates are primarily due to the use of measured stack emissions data for the post-start-up risk assessments The measured emissions were significantly less than originally estimated before the facility was built and generally continued to reduce over time

This study represents the most comprehensive (of two) MWC health risk assessments to be conducted using actual emissions and soils data. Using actual data (emissions, air, soil, vegetables, or other media) in risk assessments in place of default assumptions has important implications for both national risk assessment methodology and for local understanding of health impacts of solid waste combustion. The study also concluded that USEPA risk assessment methodology did not underestimate risk to surrounding residents, and that the facility met or exceeded original expectations established by its permit conditions

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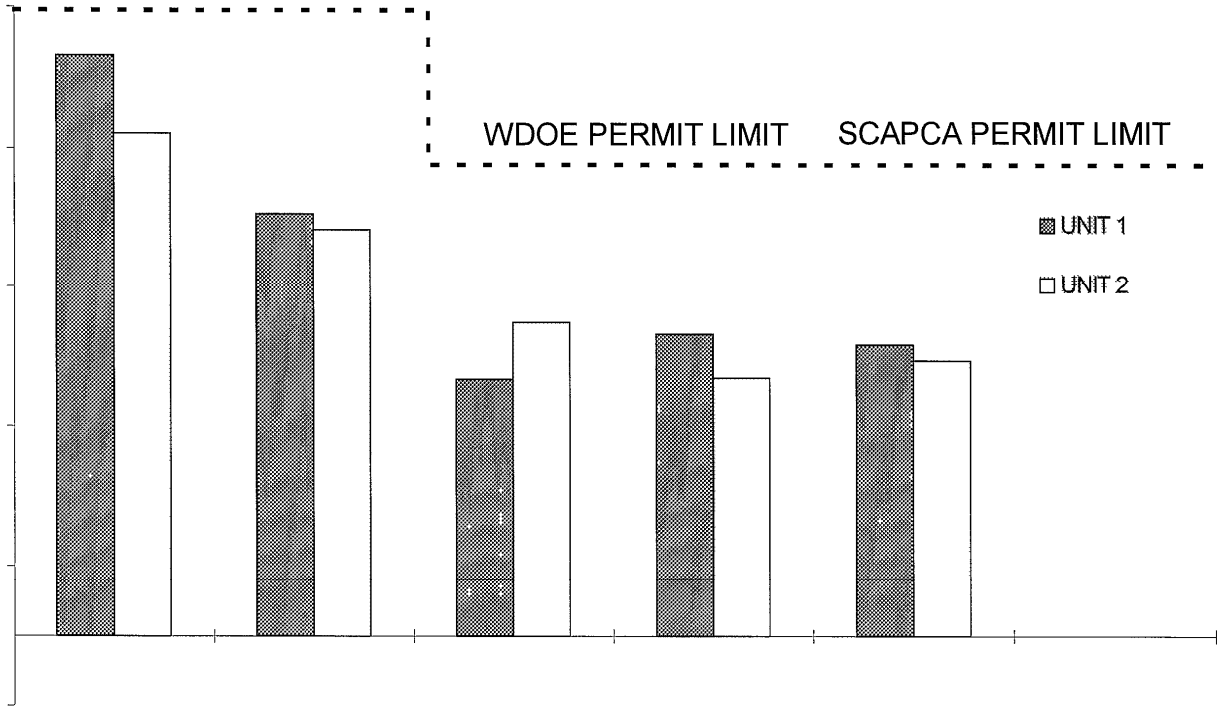
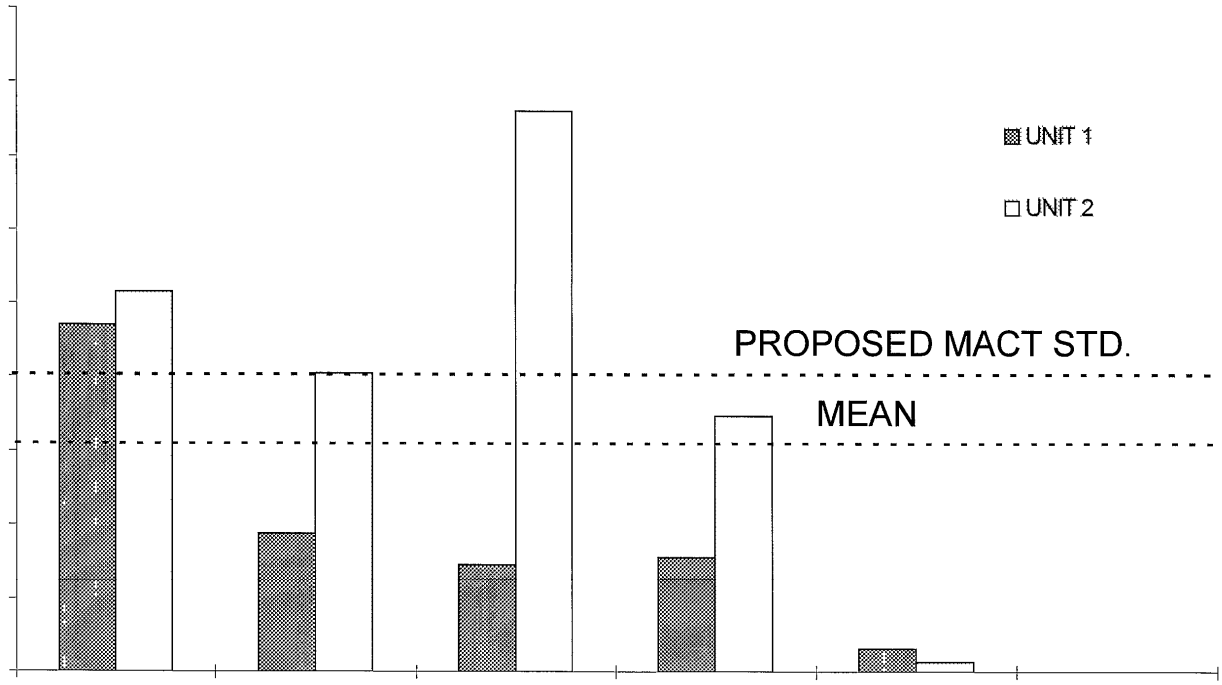
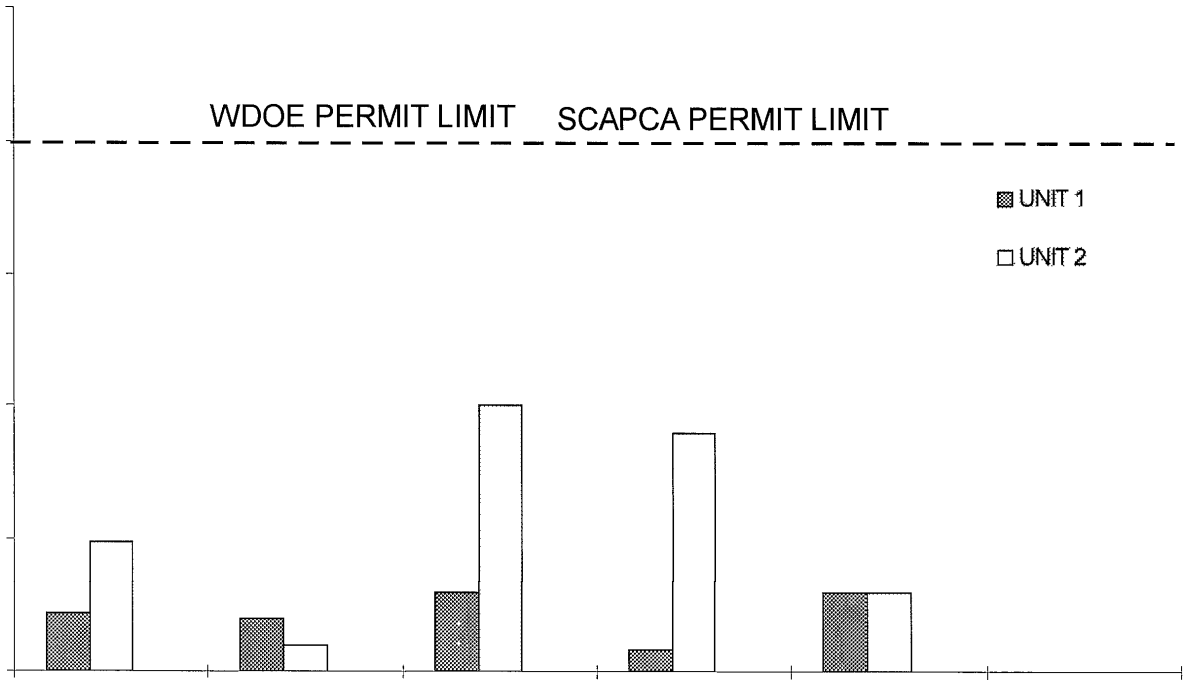
Oxides of Nitrogen



Mercury



Total Particulate



Lead