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Workers' exposures and potential health risks to air toxics in a petrochemical complex assessed by improved methodology

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Abstract *Objective*: This study was designed to comprehensively evaluate workers' potential health risks of exposure to 39 air toxics in the Ta-sher Petrochemical Complex. *Methods*: Open-Path Fourier Transform Infrared Spectroscopy (OP-FTIR) was used to measure concentrations of air toxics. We used the measured worksite concentrations between 1997 and 1999 at 11 companies in the petrochemical complex, employing 3,100 on-site workers. The 39 measured air toxics included 10 chemicals with acute reference exposure levels (*RELa*), 19 chemicals with chronic reference exposure levels (*RELc*), and 3 chemicals classified as Class 1 or 2A human carcinogens by the International Agency for Research on Cancer (IARC). We then used *RELa* to calculate the hazard index of acute health effects (HI_A) for workers in individual plants. We also calculated the hazard index of chronic health effects (*HIc*) and cancer risks for all workers in the entire petrochemical complex. *Results*: Workers in five companies had HI_A greater than 1 because of toluene, benzene, methyl ethyl ketone, chloroform and isopropanol exposures. Workers in this petrochemical complex had *HIc* greater than 1 because of acrylonitrile, 1,3-butadiene, hydrogen cyanide, and n,n-dimethylformamide exposures. Risk of hematopoietic system cancer because of benzene and ethylene oxide exposure, and respiratory system cancer because of 1,3-butadiene exposure was estimated to be 3.1–6.1×10^{-4} and 5.2–7.1×10^{-4}, respectively. *Conclusions*: Our findings indicated that workers in the petrochemical complex might have excess cancer and non-cancer risks due to acute or chronic exposures to air toxics from multiple emission sources.

Keywords Air toxics · FTIR · Risk assessment · Petrochemical · Occupational

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Introduction

A petrochemical complex is usually a consortium of many oil refinery and chemical processing factories in a large area. Depending on the numbers and types of industries located in the petrochemical complex, the toxics emitted can include benzene, styrene, toluene, xylene, ethylene, propylene, ethylbenzene, gasoline, hydrogen sulphide, jet fuel, heating oil, petroleum coke, epichlorohydrin, calcium chloride, butadiene, catalysts, and epoxy resins (Xu et al., 1998; Tsai et al., 2003). The International Agency for Research on Cancer (IARC) has defined occupational exposure in the oil refinery industry as exposure to an IARC-designated group 2A carcinogen based on epidemiological evidence (International Agency for Research on Cancer, 1998a). Studies also identified specific cancer risks stemming from exposure in a petrochemical environment to certain chemicals such as ethylene oxide, benzene, vinyl chloride monomer (VCM), and 1, 3-butadiene (International Agency for Research on Cancer, 1997; 1998b; 1998c; 1999). Under the Clean Air Act, the U.S. Environmental Protection Agency (US EPA) designated many of these chemicals (used or emitted by petrochemical plants) as hazardous air pollutants (HAPs) (Cupitt et al., 1995).

Epidemiological and health-risk assessment of potential health hazards is difficult. Changes in ambient air toxics in the petrochemical complex depend on the agents and working processes used. Most epidemiological studies of petrochemical environments (though they are complex) have focused on the assessment of single exposures without addressing the effects of mixtures (Samet and Speizer, 1993). The synergistic effect of mixtures on the etiology of disease is of increasing

concern for the public health (Samet and Speizer, 1993). Since there is an element of methodological uncertainty associated with determining the components of a mixture, measurements of the components most relevant to disease outcome may not be accomplished (Leaderer et al., 1993). Few studies have dealt with multiple exposures but they had cross-sectional designs or used surrogates for exposure measurements.

Additionally, previous efforts to characterize the potential impacts of these hazardous air pollutants usually involved numbers of limited chemicals and samples and applied inventory data, model-estimated concentrations, or air monitoring data to assess occupational and public health risks (Office of Air Quality Planning and Standards, 1990; Hassett-Sipple et al., 1991; Cote and Vandenberg, 1994; Perlin et al., 1995). As a result, potential health risks associated with all air pollutants in petrochemical environments remained undetermined.

Open-Path Fourier Transform Infrared Spectroscopy (OP-FTIR) is a very useful technique to measure various volatile organic compounds simultaneously, and has been adopted by the US EPA as reference method, TO-16, to measure air toxics (U.S. Environmental Protection Agency, 1999). In this study, OP-FTIR was used to improve assessment of workers exposed to mixtures of air toxics in a large open working environment such as a petrochemical complex. Comparing OP-FTIR-determined concentrations of air toxics to those determined by measuring reference exposure levels (REL) and cancer slope factors (Office of Environmental Health Hazard Assessment, 1999a; 1999b; 2000), we demonstrated that workers' potential health risk to all these pollutants as a whole can be more comprehensively assessed by this approach than by traditional integrated industrial hygiene sampling.

Methods

Site for evaluation

The site of our assessment is the Ta-sher Petrochemical Complex, which is located in southern Taiwan. This 120-acre petrochemical complex has 11 different manufacturing plants, produces raw petrochemical materials for further down-stream applications, and has about 3,100 on-site workers. As shown in Table 1, 23 chemicals were either processed or produced by these 11 manufacturing plants and included acetic acid, acetone, acetonitrile, acrylonitrile, ammonia, benzene, 1,3-butadiene, ethylene, ethyl benzene, ethylene oxide, ethylenediamine, formaldehyde, hydrogen cyanide, methanol, methyl ethyl ketone, methyl methacrylate, n,n-dimethylformamide, propylene, propylene oxide, styrene, sulfuric acid, toluene, and vinyl acetate. The annual inventory of these chemicals averaged between 106 tons for propylene oxide to 291,055 tons for styrene during 1997–1999.

Measurements of air toxics

We used two OP-FTIR systems (Environmental Technology Group and Bomen Instruments) to measure worksite concentrations of air toxics for each of these 11 manufacturing plants. The OP-FTIR systems were calibrated according to the TO-16 method (U.S. EPA, 1999).

Table 1 Annual chemical inventory of 11 manufacturing plants in the Ta-sher Petrochemical Complex (tons/year)

Plants Chemical	1	2	3	4	5	6	7	8	9	10	11	Total
Acetic acid		126,259			49,690							175,949
Acetone						54,000		168				54,168
Acetonitrile	13,576								9,500			23,076
Acrylonitrile	169,682											169,682
Ammonia	84,732					10,900						95,632
Benzene										205,421		205,421
1,3-Butadiene							143,630			13,092		156,722
Ethylene				63,193	30,230					75,743		169,166
Ethyl benzene										277,216		277,216
Ethylene oxide			21,508	22,160								43,668
Ethylenediamine									400			400
Formaldehyde									2,400			2,400
Hydrogen cyanide	16,990					23,000						39,990
Methanol		68,581				27,000						95,581
Methyl ethyl ketone											654	654
Methyl methacrylate						80,000						80,000
N,N-Dimethylformamide											9,870	9,870
Propylene	183,191											183,191
1,2-Propylene oxide			106									106
Styrene							31,870			259,185		291,055
Sulfuric acid						5,400			1,800	816		8,016
Toluene										5,118	290	5,408
Vinyl acetate					70,150							70,150

Up-wind and down-wind worksite concentrations of 39 air toxics were measured in each plant simultaneously using these two OP-FTIR systems. These two instruments have a resolution of 1 cm^{-1} and can detect molecules with infrared (IR) spectra at wave numbers between 400 and 4,500. We set these instruments at 4 seconds per scan. Signals with signal/noise level greater than 3 and IR intensity greater than 100, acquired in 20 min, were used to calculate the average concentration and hourly average concentration. The path height and path length used to measure path-integrated concentration were 1.6–1.8 m and 100–300 m, respectively, depending on the size of the plant. Wind speed and wind direction during air sampling were measured using the RM Young Wind Monitor (Model 05103, Campbell Scientific Corp, Edmonton, Canada), which was operated side-by-side with the OP-FTIR system. Continuous and representative monitoring of air toxics was conducted for 2–8 days each year at each plant for three years between 1997 and 1999. The exact locations of these monitoring sites are shown in Figure 1.

Non-cancer and cancer benchmark risk estimates

Benchmark estimates of carcinogenic and noncarcinogenic risk for each air toxic were taken directly from the California Environmental Protection Agency (Cal-EPA) and the United States Environmental Protection Agency (USEPA) (Office of Environmental Health Hazard Assessment, 1999a; 1999b; 2000; Office of Air Quality Planning and Standards, 1990). The acute and chronic reference exposure levels (REL) for airborne toxicants proposed by the Cal-EPA were used for comparison purposes (Office of Environmental Health Hazard Assessment, 1999a; 2000). The hazard index of acute effects for the j manufacturing plant (HI_{Aj}) was calculated as follows. First, we divided individual air toxics'

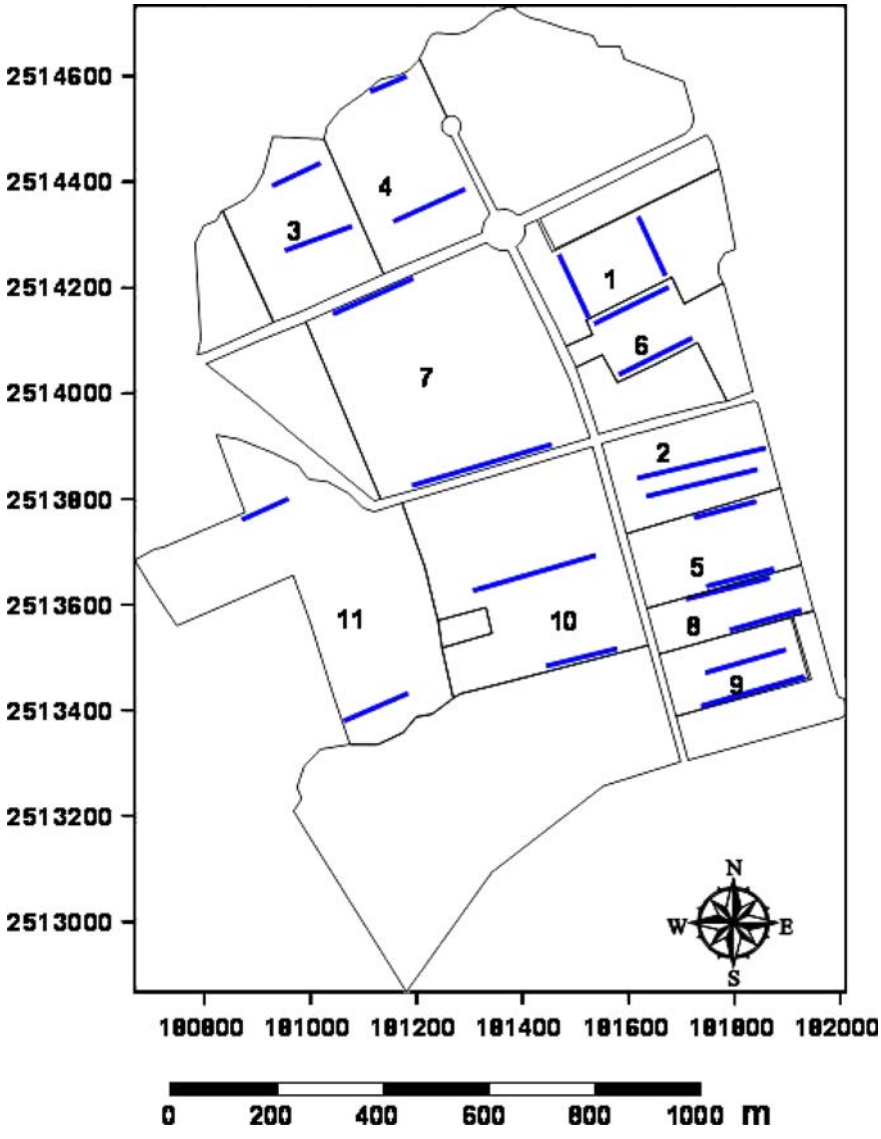

Fig. 1 The OP-FTIR sampling sites (blue bold lines) of 11 plants in Ta-sher Petrochemical Complex, Taiwan

hourly concentration (C_{ij}) over the three-year measuring period by its respective acute reference exposure level ($RELa_i$). Second, we summed the $C_{ij}/RELa_i$ measurements to obtain HI_{Aj} for the j plant, as shown in the following equation. Only air toxics with REL were used to calculate HI_{Aj} for each plant. Workers in the j manufacturing plant have potential acute health risks if the calculated HI_{Aj} was greater than 1.

$$HI_{Aj} = \frac{C_1}{RELa_1} + \frac{C_2}{RELa_2} + \cdots + \frac{C_{ij}}{RELa_i}$$

where i represents one of 23 air toxics with measurable REL and j (which varies from 1 to 11) identifies one of 11 petrochemical companies.

To calculate the hazard index of chronic health effects for individual air toxics throughout the petrochemical complex (HIc_i), we first used average concentrations of individual air toxics ($\overline{Ci}$) measured in all manufacturing plants over a three-year sampling period to represent chronic exposures for all workers in the petrochemical complex. Average concentration of individual air toxics ($\overline{Ci}$) was calculated by dividing the sum of all C_{ij} by the number of manufacturing plants with measurable air toxic levels. The average concentrations of these air toxics ($\overline{Ci}$) were then divided by their respective chronic reference exposure levels ($RELc_i$) in order to obtain HIc_i for the entire petrochemical complex. If HIc_i was greater than 1, there was a potential chronic health risk attributable to one specific air toxic in the petrochemical complex.

$$HIc_i = \frac{\overline{Ci}}{RELc_i}$$

We assumed a respiration rate of 10 m^3 per day at work, which was one-half of one person's respiration rate in one day. We also assumed 5.5 working days per week, which is the mandatory work requirement in Taiwan. Three-year average concentrations of air toxics were used to calculate risks of chronic health effects for all workers throughout the petrochemical complex.

For cancer risks, our first step was to use the International Agency for Research on Cancer (IARC) classification system to group the measured air toxics. Then we used the unit risk factor values proposed by the CalEPA for respective air toxics in the IARC Class 1 or 2A to calculate the potential cancer risks (Office of Environmental Health Hazard Assessment, 1999b). Again, three-year average concentrations of air toxics were used to calculate cancer risks in specific target organs for all workers throughout the petrochemical complex. Because volume concentrations (ppb) were used in our cancer risk calculation, conversion factors were used to transform unit risk factor values from $(\mu g/m^3)^{-1}$ to $(ppb)^{-1}$. We estimated a worker's lifetime exposure on the assumption that a worker is on site 8 hours per day, 270 days per year for 45 years—an estimate which assumes that a worker enters the workforce at age of 20 and retires at age of 65.

Results

Environmental concentrations and effects

The spatial distribution of air toxics was not uniform throughout the petrochemical complex. Only 3 of 39 chemicals (ammonia, ethylene, and methanol) were detected in all 11 manufacturing plants. Four (*N*,*N*-dimethylformamide, methyl ethyl ketone, propylene, and vinyl acetate) were detected in 10 plants. The others were not homogeneously distributed.

Table 2 summarizes measurements of 39 air toxics at the Ta-sher Petrochemical Complex. The total sampling duration was 77 days and the number of valid path-integrated measurements was 29,664 over three years. The percentage of measurements lower than the limit of detection (LOD) ranged from 29.1% to 99.9% for different air toxics. On the basis of percentages of measurements above the LOD, the five most frequently detected air toxics were (in order of their detection frequency) ethylene (61.9%), ammonia (53.4%), cyclohexane (41.2%), methanol (37.8%), and propylene (25.6%). All but 4 (acetonitrile, ethylenediamine, formaldehyde, and sulfuric acid) of the 23 chemicals used or produced by the 11 manufacturing plants could be detected by our measurements. By contrast, another 20 air toxics not listed in the chemical inventory of the manufacturing plants were detected. These 20 air toxics were acetylene, n-butane, butyl acetate, chlorodifluoromethane (HCFC-11), chloroform, cyclohexane, dichlorodifluoromethane (CFC-12), dimethyl ether, ethyl acetate, ethyl acrylate, formic acid, n-hexane, isopropanol, methyl acetate, 2-methyl 2-butene, 2-methyl pentane, octane, n-pentane, propane, and trichlorodifluoromethane (CFC-13).

In order to summarize our OP-FTIR data and deal with the missing data, we used the values of 0 and $LOD/\sqrt{2}$ to calculate the lowest and highest possible concentrations, respectively (Helse et al., 1990). As shown in Table 2, hourly concentrations for each air toxic varied widely over the three-year sampling period. The standard deviation was always greater than the mean concentrations for each air toxic. Our measurements also showed difference in mean concentrations of the 39 air toxics, with the highest being about three orders of magnitude greater than the lowest.

The 39 air toxics were classified into three categories based on their potential effects on health (i.e., acute, chronic, and carcinogenic effects). Short-term excess exposures to the 10 air toxics in the acute effects category (namely, ammonia, benzene, chloroform, hydrogen cyanide, isopropanol, methanol, methyl ethyl ketone, propylene oxide, styrene, and toluene) can potentially harm various organs (such as eyes) and systems (such as the respiratory, nervous, alimentary, reproductive and hematopoietic systems) (EPA, 1999a). High short-term concentrations of these 10 air toxics, ranging from 230 ppb of propylene oxide to 4,234 ppb of isopropanol, were observed during the study period.

Table 2 Concentrations of 39 air toxics measured by the OP-FTIR method at the Ta-sher Petrochemical Complex during 1997–1999 (in ppb)

Chemical	% measurements > LOD	Low estimate[a]		High estimate[b]		Maximum concentrations
		Mean	SD	Mean	SD	
Acetic acid	6.8%	2.5	13.9	7.1	13.2	557
Acetone	5.6%	7.7	49.3	17.4	47.8	1,180
Acetylene	11.1%	1.0	3.7	3.4	3.1	80
Acrylonitrile	18.9%	43.5	178.9	47.8	177.8	4,198
Ammonia	53.4%	21.2	62.4	21.5	62.3	1,476
Benzene	3.5%	3.0	27.5	14.6	26.2	990
n-Butane	2.6%	0.2	7.4	7.6	7.2	740
1,3-Butadiene	15.2%	7.7	37.3	10.5	36.7	3,080
Butyl acetate	3.8%	0.1	1.7	2.9	1.7	140
Chlorodifluoromethane	3.3%	0.3	3.4	1.0	3.4	286
Chloroform	1.0%	0.4	7.4	1.3	7.3	270
Cyclohexane	41.2%	33.0	643.6	33.4	643.6	60,830
Dichlorodifluoromethane	< 0.1%	< 0.1	0.1	0.7	0.1	9
Dimethyl ether	0.4%	0.1	1.2	3.2	1.1	107
Ethyl acetate	16.5%	0.6	6.0	3.4	5.7	259
Ethyl acrylate	1.8%	0.2	3.7	3.0	3.6	180
Ethyl benzene	0.3%	0.3	6.0	12.3	5.4	436
Ethylene	61.9%	179.7	577.3	180.6	577.0	23,596
Ethylene oxide	7.7%	9.0	67.1	12.6	66.6	4,705
N,N-Dimethylformamide	17.6%	53.4	211.7	59.2	210.2	3,488
Formic acid	1.4%	2.4	32.6	5.2	32.4	2,808
n-Hexane	0.5%	0.2	4.2	1.8	4.1	205
Hydrogen cyanide	2.3%	2.9	24.7	26.3	22.1	1,200
Isopropanol	1.5%	5.4	83.7	6.2	83.7	4,234
Methanol	37.8%	23.4	78.8	24.3	78.5	2,449
Methyl acetate	1.4%	0.5	9.1	3.2	9.0	316
2-methyl 2-butene	< 0.1%	< 0.1	< 0.1	< 0.1	< 0.1	14,000
Methyl ethyl ketone	19.1%	55.1	200.3	64.7	197.7	6,265
Methyl methacrylate	12.7%	8.1	30.8	10.1	30.3	660
2-Methyl pentane	0.5%	0.2	5.5	4.1	5.3	335
Octane	< 0.1%	< 0.1	3.5	0.9	3.5	433
n-Pentane	1.2%	0.4	5.0	3.4	4.8	234
Propane	2.3%	1.8	25.5	8.4	25.1	1,600
Propylene	25.6%	31.4	119.6	35.0	118.7	3,202
1,2-Propylene oxide	1.7%	0.1	3.0	7.9	2.9	230
Styrene	10.2%	9.6	41.1	13.3	40.2	526
Toluene	6.1%	35.7	214.4	47.0	212.5	3,630
Trichlorofluoromethane	0.5%	< 0.1	0.7	0.7	0.7	46
Vinyl acetate	20.0%	18.4	92.9	20.5	92.5	4,298

[a] '0' replaced measurements less than LOD as low estimate.
[b] ' $LOD/\sqrt{2}$' replaced measurements less than LOD as high estimate.

Nineteen chemicals had various chronic effects. Chronic exposures to these 19 air toxics in the chronic effects category (namely, acrylonitrile, ammonia, benzene, 1,3-butadiene, chloroform, ethyl benzene, ethylene oxide, ethylene, hexane, hydrogen cyanide, isopropanol, methanol, methyl ethyl ketone, n,n-dimethylformamide, propylene, 1,2-propylene oxide, styrene, toluene, and vinyl acetate) can damage various organs (such as eyes, kidney, and liver) and systems (such as respiratory, hematopoietic, nervous, reproductive, alimentary, endocrine, and cardiovascular systems) (EPA, 2000). Of these 19 air toxics, ethylene was present in the highest average concentration (179.7–180.6 ppb), while chloroform was present in the lowest average concentration (0.4–1.3 ppb) as shown in Table 3.

Lifetime exposures to benzene, 1,3-butadiene, and ethylene oxide (the 3 monitored air toxics classified as the Class 1 or 2A human carcinogens by the IARC) may increase workers' risks of developing cancers of the hematopoietic or respiratory systems according to the EPA (EPA, 1999b). The average concentrations were 3.0–14.6 ppb for benzene, 9.0–12.6 ppb for ethylene oxide, and 7.7–10.5 ppb for 1,3-butadiene (Table 4).

Acute health risks

The percentage of HI_{Aj} (i.e., the hazard index attributable to acute health effects) measurements greater than 1 for workers in each of the 11 manufacturing plants during 1997–1999 are shown in Figure 2. Five manufacturing plants (companies 1, 6, 7, 10, and 11) had HI_A greater than 1, which was mainly due to toluene, benzene, methyl ethyl ketone, and chloroform. The percent

Table 3 Hazard index of chronic health effects (HI_c) for workers exposed to 19 air toxics in Ta-sher Petrochemical Complex during 1997–1999

Chemical	RELs (ppb)	Mean (L)[a]	Mean (H)[b]	HI_c	Target organs or system
Acrylonitrile	1	43.5	47.8	43.5 – 47.8	Respiratory system
Ammonia	300	21.2	21.5	0.1	Respiratory system
Benzene	20	3.0	14.6	0.2–0.7	Hematopoietic system; development; nervous system
1,3-Butadiene	8	7.7	10.5	1.0–1.3	Reproductive system
Chloroform	50	0.4	1.3	< 0.1	Alimentary system; kidney; development
Ethyl benzene	400	0.3	12.3	< 0.1	Alimentary system (liver); kidney; endocrine system
Ethylene oxide	18	9.0	12.6	0.5–0.7	Nervous system
Ethylene	20,000	179.7	180.6	< 0.1	Nervous system
Hexane	2000	0.2	1.8	< 0.1	Nervous system
Hydrogen cyanide	8	2.9	26.3	0.4 – 3.3	Nervous system; endocrine system; cardiovascular system
Isopropanol	2,970	5.4	6.2	< 0.1	Kidney
Methanol	3,000	23.4	24.3	< 001	Development
Methyl ethyl ketone	4,000	55.1	64.7	< 0.1	Alimentary system; kidney
Methyl tert-butyl ether	800	0.0	3.4	< 0.1	Kidney; eyes; alimentary system
N,N-Dimethylformamide	30	53.4	59.2	1.8 – 2.0	Alimentary system; respiratory system
Propylene	2,000	31.4	35.0	< 0.1	Respiratory system
1,2-Propylene oxide	9	0.1	7.9	0 – 0.9	Respiratory system
Styrene	300	9.6	12.3	< 0.1	Nervous system
Toluene	100	35.7	47.0	0.4–0.5	Nervous system; alimentary system; development

[a] Mean (L): mean concentrations using 0 to substitute measurements of < LODs (unit: ppb)
[b] Mean (H): mean concentrations using $LOD/\sqrt{2}$ to substitute measurements of < LODs (unit: ppb)

of $HI_A > 1$ measurements over the three-year monitoring period varied between about 0.05% and 1.8%. The highest HI_A of 9.0 occurred at company 6 where chloroform was the main contributing air toxic.

Chronic health risks

The values of the hazard index of chronic health effects (*HIc*) are shown in Table 3. The *HIc* values were consistently greater than 1 for acrylonitrile (range, 43.5–47.8), n,n-dimethylformamide (1.8–2.0), and 1,3-butadiene (1.0–1.3). We also found some *HIc* values greater than 1 for hydrogen cyanide. Chronic exposures to these four air toxics could potentially increase the risk of respiratory, reproductive, alimentary, nervous, endocrine, and cardiovascular system damage. Acrylonitrile could pose the most observable non-cancer health risk because its *HIc* was most obviously greater than 1.

Cancer risks

The estimated lifetime cancer risks due to occupational exposures to the 3 carcinogens are shown in Table 4. For the two Class 1 hematopoietic system carcinogens, the cancer risk was $0.5–2.5\times10^{-4}$ for benzene and $2.6 – 3.7\times10^{-4}$ for ethylene oxide. For the Class 2A respiratory system carcinogen 1,3-butadiene, the cancer risk was $5.2 – 7.1\times10^{-4}$. Total risk of hematopoietic system

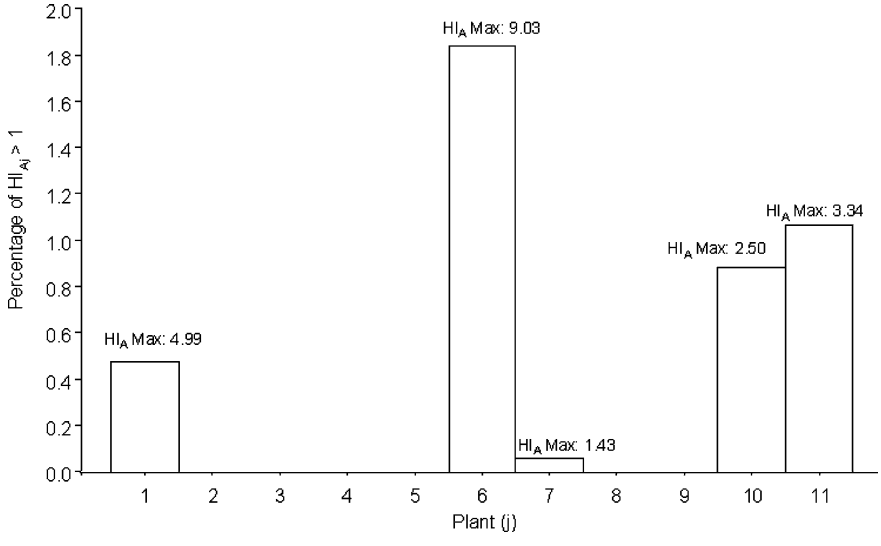

Fig. 2 Percentage of measurements of hazard index due to acute health effects (HI_A) greater than 1 for 11 plants in the Ta-sher Petrochemical Complex, Taiwan

Table 4 Estimated worker's lifetime cancer risks due to occupational exposures to 3 carcinogenic air toxics in the Ta-sher Petrochemical Complex

Chemical	EPA class	IARC class	Unit risk $(\mu g/m^3)^{-1}$	Conversion factor	C^a_{OBS1} (ppb)	C^b_{OBS2} (ppb)	Cancer risk		Target organs
							Low	High	
Benzene	A	1	2.9E-05	3.2	3	14.6	5.0E-05	2.5E-04	Hematopoietic system
Ethylene oxide	B1	1	8.8E-05	1.8	9	12.6	2.6E-04	3.7E-04	Hematopoietic system
1,3-Butadiene	B2	2A	1.7E-04	2.2	7.7	10.5	5.2E-04	7.1E-04	Respiratory system
Benzene and ethylene oxide combined							3.1E-04	6.1E-04	Hematopoietic system

[a] C_{OBS1}: mean concentrations using 0 to substitute measurements of < LODs
[b] C_{OBS2}: mean concentrations using $LOD/\sqrt{2}$ to substitute measurements of < LODs

cancer for workers exposed to both benzene and ethylene oxide was about $3.1-6.1\times10^{-4}$. The risk of respiratory system cancer due to occupational exposure to 1,3-butadiene was about $5.2-7.1\times10^{-4}$.

Discussion and conclusions

Air toxics can contribute to outdoor air cancer risk. Previous studies have shown that volatile organic chemicals (VOCs) typically account for 35–55% of nationwide outdoor air cancer risk in the United States (US EPA, 1990). A unit risk factor is defined as the estimated probability that an individual will develop cancer as a result of exposure to a pollutant at an ambient concentration of 1 μgm^{-3} for 70 years. These are not absolute values but rather upper-limit estimates and are used here primarily to illustrate the relative importance of the potentially carcinogenic species measured (Cheng L., et al. 1997).

Our study estimated the cancer and noncancer risks due to acute or chronic exposures to 39 air toxics measured using OP-FTIR systems and emitted from multiple emission sources in a petrochemical complex. Of these 39 air toxics, 19 chemicals (acetic acid, acetone, acrylonitrile, ammonia, benzene, 1,3-butadiene, ethylene, ethyl benzene, ethylene oxide, hydrogen cyanide, methanol, methyl ethyl ketone, methyl methacrylate, n,n-dimethylformamide, propylene, 1,2-propylene oxide, styrene, toluene, and vinyl acetate) were raw materials or products in the annual chemical inventory of the 11 manufacturing plants and the other 20 were detected by the OP-FTIR monitoring. However, 4 (including acetonitrile, ethylenediamine, formaldehyde, and sulfuric acid) of 23 chemicals identified by emission inventory alone were not assessed to be health risks in this study. The health risks of these 4 chemicals should be listed in the potential exposure list in any future study pertaining to petrochemical workers. Our findings have shown that appropriate health risk assessment cannot rely solely on review of the chemical inventory or air toxics monitoring. Both are needed to comprehensively identify the potential occupational hazards for workers in the petrochemical industry.

Using the OP-FTIR monitoring method, we were able to detect several air toxics (not easily measured by other air sampling and analysis methods) at ubiquitously high concentrations throughout this petrochemical complex. The OP-FTIR method not only measured concentrations of 19 air toxics emitted from all emission sources, but also identified 20 air toxics not routinely reported in the emission inventory. Therefore, we conclude that characterization of workers' exposures to air toxics can be greatly enhanced by the use of this monitoring method. Our OP-FTIR method also reduced the problem (reported in previous studies characterized by limited monitoring data) of underestimating environmental concentrations of air toxics (Woodruff et al., 1998; Rosenbaum et al., 1999). In addition, we also measured spatial and temporal peak concentrations of air toxics, which can be used to estimate potential acute health effects. Simultaneous measurements of various air toxics by OP-FTIR allowed us to estimate potential health risks posed by multiple pollutants in a complex industrial setting such as a petrochemical industrial park. By considering the potential health impact of multiple pollutants in combination, we can fully evaluate the potential health risks for an area even when individual air toxics are at concentrations below their respective toxicity benchmarks.

With the help of available toxicity information, we were able to estimate the potential health risks of many air toxics determined by OP-FTIR in this study. Our results showed that workers in the Ta-sher Petrochemical Complex were exposed to air toxics that could increase their risks of suffering various adverse acute, chronic, and carcinogenic health effects. We found acute and chronic hazard indices that were greater than 1. The risks of hematopoietic and respiratory system cancers were both greater than one in ten thousand (1×10^{-4}), which is the benchmark risk for occupational exposures. These findings support the notion that workers in such intensive petrochemical industry environments may have higher potential health risks than the general population.

The key air toxics causing 1) acute effects were toluene, benzene, methyl ethyl ketone, and chloroform, 2) chronic effects were acrylonitrile, n,n-dimethylformamide and 1,3-butadiene, and 3) carcinogenic effects were benzene, ethylene oxide, and 1,3-butadiene. These air toxics posed the highest potential health risk to workers in the petrochemical complex we studied. Future studies

should focus on the possible acute, chronic, and carcinogenic effects of these pollutants on workers' eyes, and their respiratory, nervous, alimentary, reproductive and hematopoietic systems.

The uncertainty of location and duration of FTIR monitoring to assess workers' exposures to air toxics may have led us to under- or overestimate health risks. Since the FTIR system monitored area concentrations only rather than personal exposures, we may have underestimated acute health risks because workers' actual exposures may be higher near emission sources, which greatly enhance their exposure to a few specific compounds through their skin. We may have overestimated chronic health effects and cancer risks since we relied on 3-year measurements of air toxics to extrapolate lifelong 45-year occupational exposure risks. We may also have underestimated both acute and chronic effects because some chemicals in this petrochemical complex may not be measured by our OP-FTIR system, such as vaporous compounds adsorbed on particles. However, the OP-FTIR monitoring system in our study has generated considerably more exposure data than previous spot sampling approaches, which could not be used to measure exposures of multiple air toxics simultaneously. Our study approach and findings provide industrial hygienists with a useful tool and information to conduct risk management and risk communication in petrochemical companies, which may have multiple emission sources of chemicals that pose health risks to workers.

In conclusion, an improved exposure assessment methodology (using OP-FTIR) is described that comprehensively identifies the potential health risks for workers in a petrochemical industry setting. We found that exposure to toluene, benzene, methyl ethyl ketone, and chloroform with potential acute effects, acrylonitrile, n,n-dimethylformamide and 1,3-butadiene with potential chronic effects, and benzene, ethylene oxide, and 1,3-butadiene with potential carcinogenic effects was higher among petrochemical workers than among the general population. Future epidemiological studies should focus on the association between exposure to these air toxics and their possible acute, chronic, and carcinogenic effects on workers.

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